Dialysis Access Management

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Editors

Second Edition

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Brief Introduction of Hemodialysis and Vascular Access

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Ru Yu Tan and Chieh Suai Tan

Introduction

Three main treatment options exist to replace the kidney function of patients with end-stage renal disease (ESRD): hemodialysis (HD), peritoneal dialysis (PD) and renal transplantation. Hemodialysis is an extracorporeal renal replacement therapy that uses counter-current dialysate flow across a semipermeable membrane to achieve uremic toxins and fluid removal. It is now established as a mature therapy worldwide and has been the mainstay of treatment for ESRD patients. In the United States, a steady increase in ESRD prevalence was observed between 1980 and 2016, and the majority of patients used HD as their renal replacement therapy [1].

The First Hemodialysis

The first successful HD was described and performed by Kolff in 1943 [2]. Kolff's rotating drum dialyzer was made of cellulose membrane tubes which were filled with blood and wrapped around a wooden drum that rotated through the dialysate for removal of uremic toxins during treatment. Since then, the concept was further refined and improved to provide a safe, reliable and effective therapy for patients suffering from ESRD. Modern dialyzers are made of more biocompatible or synthetic membranes with clearly defined characteristics and the ability to provide quantitative clearance of uremic toxins.

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The First Arteriovenous Access

Maintenance HD is only possible in the presence of a vascular access. There are three main types of vascular accesses used to perform maintenance HD: arteriovenous fistula (AVF), arteriovenous graft (AVG) and central venous catheter (CVC). The initial form of vascular access was called the Scribner shunt. It was implanted for the first time in a human in 1960 [3]. The surgical technique to create the autogenous AVF was described by Brescia and Cimino in 1966. The use of synthetic graft for arteriovenous (AV) access creation began in the 1970s while hemodialysis catheters started in the 1980s [4, 5].

Trends in Hemodialysis

Hemodialysis is now established as a mature therapy worldwide and has been the mainstay of treatment for patients with ESRD. In 2017, 86.9% of incident ESRD patients began renal replacement therapy with HD in the United States. Of all prevalent ESRD patients, 62.7% were treated with HD [1].

AVF provides the best outcome when compared to AVG and CVC. Patients with AVF have the lowest rates of infection, morbidity and mortality. However, the use of AVF in hemodialysis patients was extremely low in the 1990s. To increase the appropriate use of AVF, the Fistula First Initiative was started in 2003 [4]. Although there was a substantial increase in the rate of AVF placement, the primary failure rates remained high. Driven by the changing demographics in patients with ESRD, placement of AVF is increasingly challenging as ESRD patients entering dialysis are older and more likely to suffer from multiple comorbidities including diabetes mellitus, coronary artery disease and peripheral arterial disease. This highlights the need to individualize patient care when planning for vascular access placement.

Over the past decade, there has been a growing interest in home HD and intensive HD where patients receive either long-hours nocturnal dialysis or short daily dialysis. Home HD encourages patients' independence and allows patients the freedom to schedule dialysis at their own convenience. Intensive HD is demonstrated in many studies to result in a better quality of life, cost-saving in healthcare and better clinical outcomes [6]. Although home HD was only used by 2% of all prevalent patients receiving maintenance hemodialysis in 2017, the proportion was increased by 120% compared to 2007 [1]. A known adverse outcome to more frequent hemodialysis is the competing risks of vascular access-related complications.

Research is currently underway to develop lightweight, easy to use wearable dialysis devices and implantable hemodialysis devices that would allow patients to receive dialysis without restricting their daily activities. The successful development of these new dialysis devices could potentially improve the outcomes and quality of life of patients with ESRD. Similarly, improvement in AVF creation techniques and the use of novel devices such as drug-coated balloons for endovascular intervention are exciting developments in dialysis access management.

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Angiographic Imaging Equipment

2

Chieh Suai Tan, Robert M. Sheridan, and Steven Wu

Introduction

Since the accidental discovery of X-rays in 1895, technology has evolved so rapidly that minimally invasive endovascular interventions are routinely performed under radiological guidance.

Having high-quality fluoroscopic imaging is pivotal for endovascular intervention. Hence, it is essential to know your machine well and understand some of the common terminologies.

Angiographic Imaging System

Interventional suites may be equipped with either a stationary (Fig. 2.1) or a mobile fluoroscopic imaging system (Fig. 2.2). The common features of these systems are the presence of a C-arm, an angiographic procedure table and a console or computer system to process and project the images for viewing on a screen. As the name suggests, C-arm consists of a C shaped metal mount equipped with an X-ray generator

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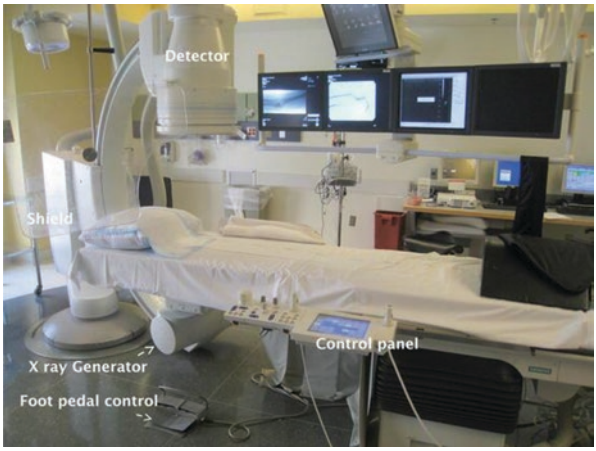

Fig. 2.1 The layout of an angiography suite with a stationary fluoroscopic imaging system

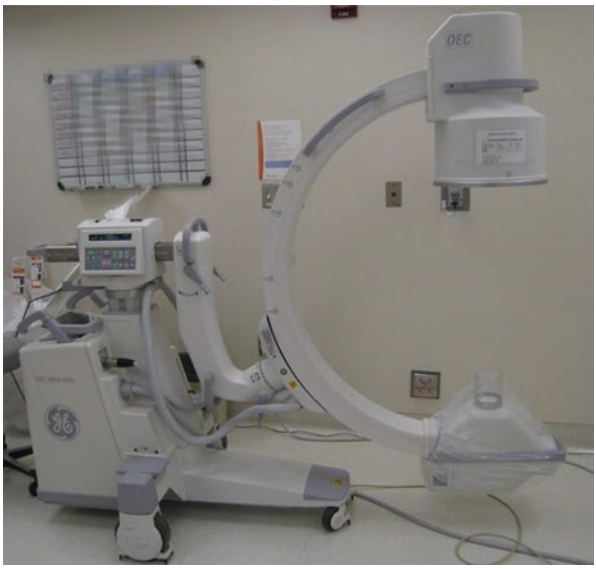

Fig. 2.2 The portable C-arm of a mobile fluoroscopic imaging system. The radiographer has to manually position the C-arm over the area of intervention

at one end and an X-ray receptor at the opposite end of the C-arm. The patient is placed on a radiolucent procedure table, between the X-ray tube and the receptor.

A stationary fluoroscopy system consists of a ceiling or floor mounted C-arm, ceiling mounted monitors and floor mounted procedure table. The entire set up is fully mechanized and the patient is positioned within the fluoroscopy field by either moving the motorized procedure table or the C-arm. The stationary system usually has a larger generator that can provide higher image resolution than a mobile system. In addition, the stationary system has excellent C-arm mobility to allow imaging of the target area at different angulations.

A mobile fluoroscopy system consists of a portable C-arm and monitor that can be moved from room to room. The angiographic procedure table is usually stationary and the radiographer manually positions the C-arm over the area of intervention. They are usually less expensive and have smaller X- ray generators and lower heat capacity than stationary systems. Some new generation portable C-arm systems can produce high quality images and have image processing capability similar to that of the stationary systems.

C-arm

The C-arm consists of an X-ray generator and an X-ray receptor (Fig. 2.3a, b). The X-ray beam that is generated travels through the patient and is captured by the receptor, which is either an image intensifier or a digital flat-panel detector. The current that is required to generate X-rays is measured in milliampere/second (mAs). It ranges from 0.5–5 mA for fluoroscopy and is triggered when the fluoroscopy pedal

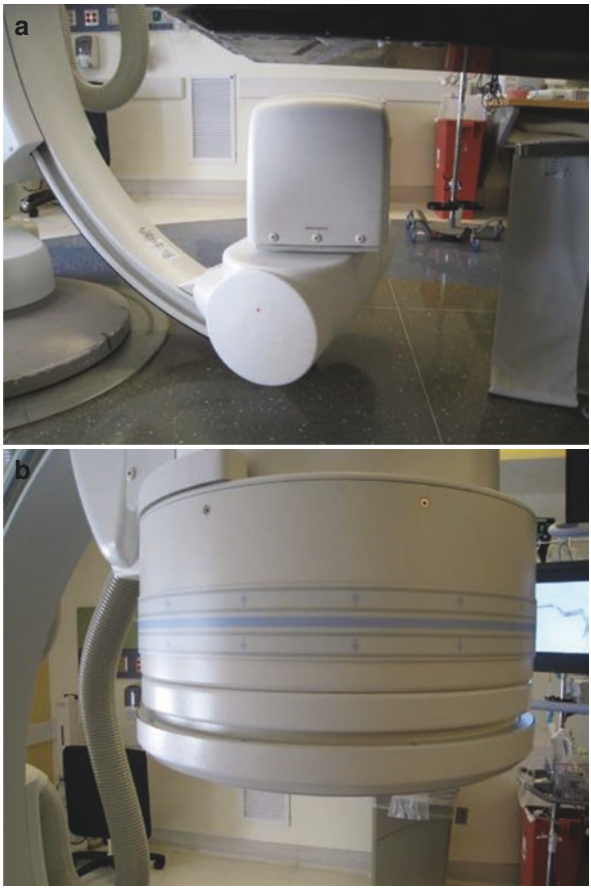

Fig. 2.3 (**a**) The generator is mounted on the lower end of the C-arm and is located under the table. (**b**) The X-ray detector is mounted on the top end of the C-arm

is pressed. The current determines the density of the image. Peak kilo-voltage (kVp), which is a measure of the potential difference across the anode and cathode, determines the maximum kinetic energy of the X-ray beam. The kinetic energy of the X-ray beam impacts the penetrability of the X-ray beam and the contrast of the image. In an automated system, the interaction between the mAs and kVp is determined by the computer to provide the best image quality at the lowest radiation dose to the patient.

Foot Switch

A foot switch control is used to start the generation of X-rays by the C-arm (Fig. 2.4). The pedals are programmed to begin imaging using fluoroscopy or digitally subtracted angiography when depressed respectively. X-rays are generated once the pedal is depressed and continued until the pedal is released.

Monitor Console

The monitor console usually has two or more computer screens to display the images (Fig. 2.5). The screen on the left shows the "active" or "live" images, while the one on the right displays the last recorded image frame or replay the image sequences.

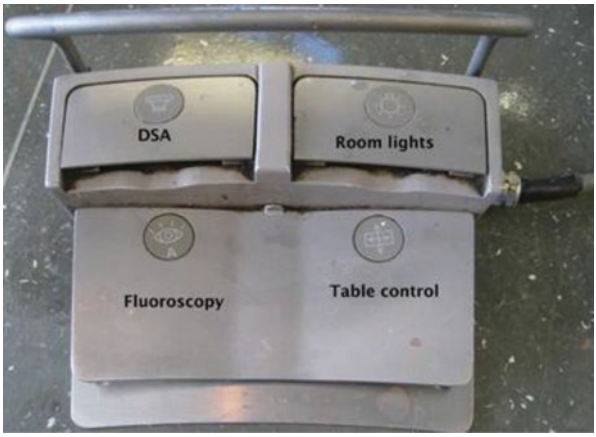

Fig. 2.4 The pedals on the foot switch can be programmed based on user preference

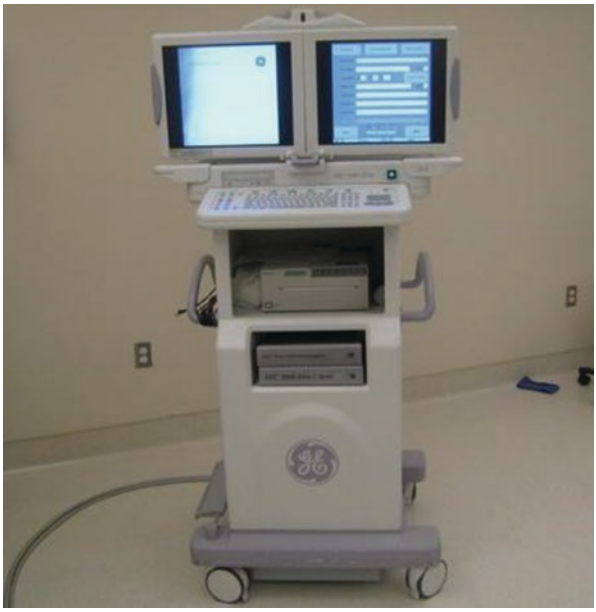

Fig. 2.5 The monitor console in a mobile fluoroscopic imaging system is mounted on wheels and can be moved together with the mobile C-arm from room to room

Table and Control Panel

The procedure table is made of carbon fiber to allow easy penetration of the X-ray beam. The stationary system may be equipped with two control panels; one located at the side of the procedure table, and another may be found within the control room (Fig. 2.6a–c).

Imaging Options

Pulsed Fluoroscopy

Variable rated pulsed fluoroscopy is an important feature in a digital angiographic imaging system (Fig. 2.7a, b). In pulsed mode, the X-ray beam is not generated continuously but delivered intermittently in synchrony with the image display to produce the appearance of a smooth continuous image. The use of pulsed fluoroscopy can significantly reduce X-ray dose, but "flickering" of the images can occur when it is set too low. The default setting in our center is 15 pulses per second although in general, 4 pulses per second is sufficient for dialysis access intervention. The X-ray dose at 30 pulses per second is equivalent to that of continuous fluoroscopy.

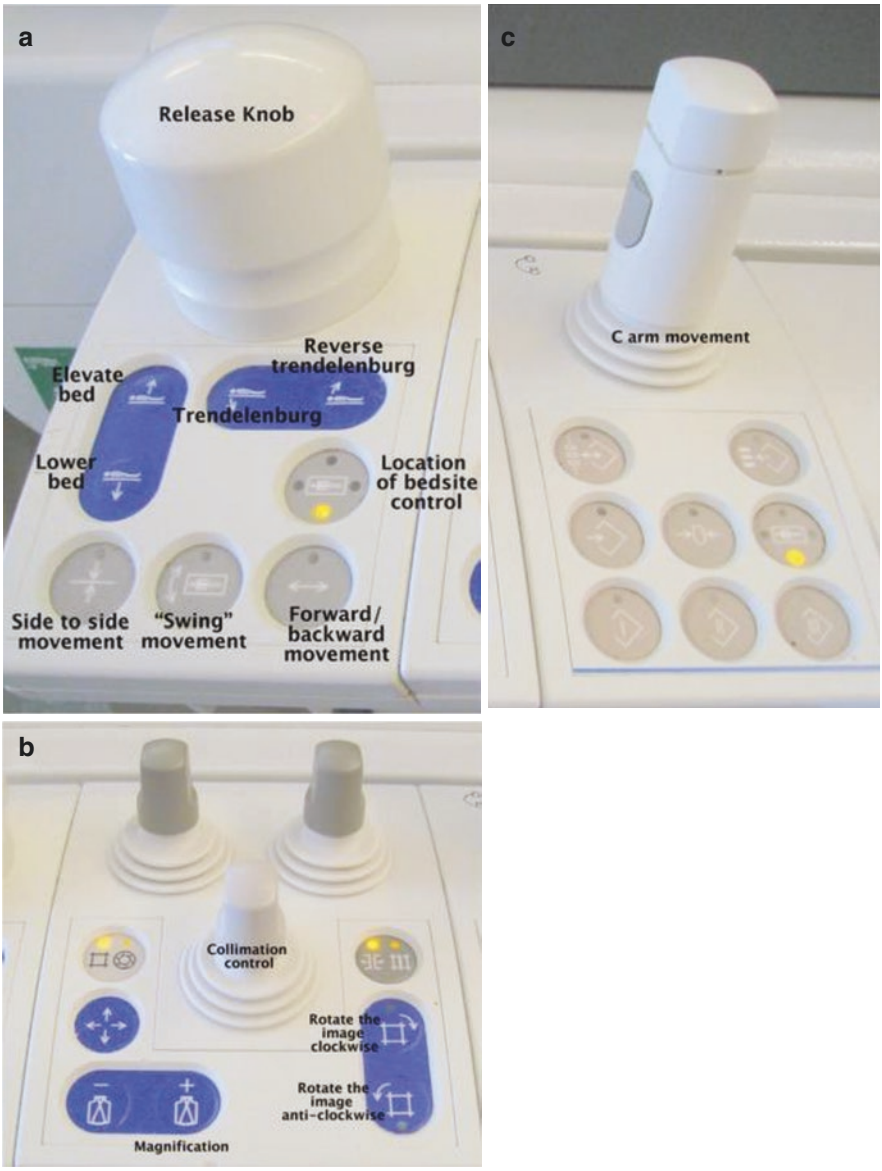

Fig. 2.6 (**a**) Horizontal movement of the table in all directions is possible once the release knob is depressed. The three grey colored buttons are used when the restriction in a particular direction of the table movement is required. (**b**) This panel controls the generation of images by the C-arm. Collimation is used to restrict the field of view to the area of interest. Magnification is used to magnify the area of interest for detailed examination. The images can also be rotated clockwise or anti-clockwise. (**c**) This control stick controls the movement of the C-arm. It is used to move the C-arm around the patient

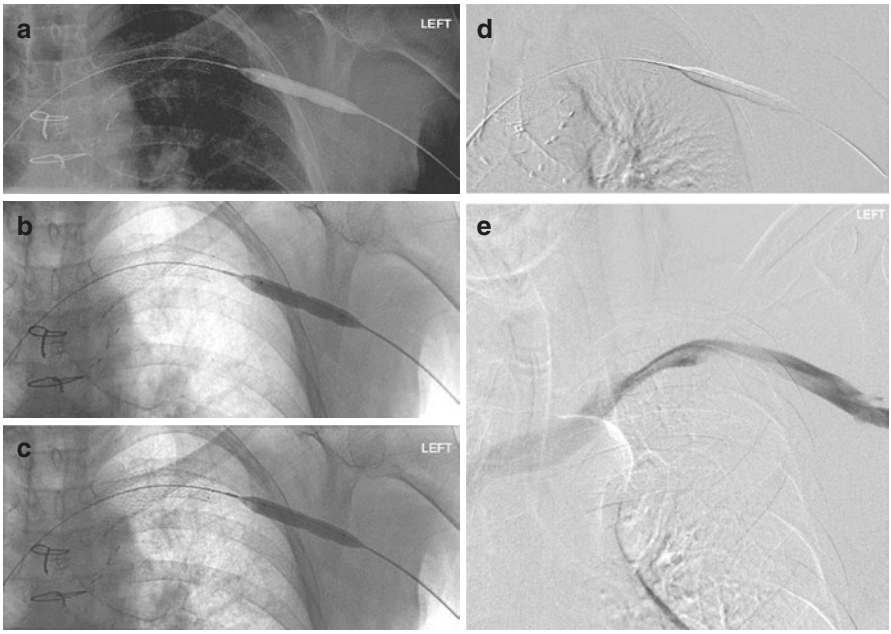

Fig. 2.7 (**a**) Fluoroscopy with a "white on black" setting. The angioplasty balloon which is filled with radiopaque contrast material will appear white. (**b**) Fluoroscopy with a "black on white" setting. The angioplasty balloon which is filled with radiopaque contrast material will appear black. (**c**) In digital subtraction angiography, a "mask" of the area is first created. (**d**) Using the "mask" that was initially acquired, background tissues or structures are then digitally removed from the subsequently acquired images. (**e**) The injected contrast will appear black on a "white-out" background that has been digitally subtracted or modified using the "mask" image as the reference image

Fluoroscopy Versus Digital Subtraction Angiography (DSA)

In standard fluoroscopy, electron dense objects such as bones and iodinated contrast materials absorb more energy and appear white on a black background. This is usually reversed digitally such that bone and contrast will appear black on a white background.

In digital subtraction angiography (DSA), a "mask" of the area of interest is first taken and used as a reference to digitally remove or subtract the "background" tissues or structures from the images that are subsequently acquired during contrast material administration. Vessels that are filled with the contrast material appear black on a "white-out" background. Subtraction angiography improves the contrast resolution of the images (Fig. 2.7c–e).

Acquisition of DSA images is described in "frames per second". The image acquisition frame rate can be adjusted as per the target vasculature. While a slow acquisition frame rate may not adequately capture the flow of contrast material, a high frame rate may be unnecessary and may result in a high radiation dose. In

general, 3 frames per second is sufficient when imaging the central veins (to compensate for chest movement artifact), while 1–2 frames per second is adequate for peripheral dialysis access interventions.

Collimation versus Magnification

Collimation is used to limit the size of the field of view to the area of interest. It helps to decrease the radiation dose to the patient and improve image quality by reducing scattered radiation.

Magnification is used to magnify or enlarge the area of interest. Magnification results in an increase in the patient's radiation dose and should be used only when fine detail is needed.

Optimizing Image Quality

The quality of the images will have an impact on the ability to make an appropriate interpretation. While obtaining the best image possible is essential, one must be mindful of the potential adverse effects of radiation. Some of the techniques to improve image quality are as follows:

1. Minimize the distance between the X-ray detector (image receptor) and the patient. This improves image quality and decreases scatter radiation.
2. Remove radiopaque objects such as oxygen tubing and ECG leads from the field of view (Fig. 2.8).

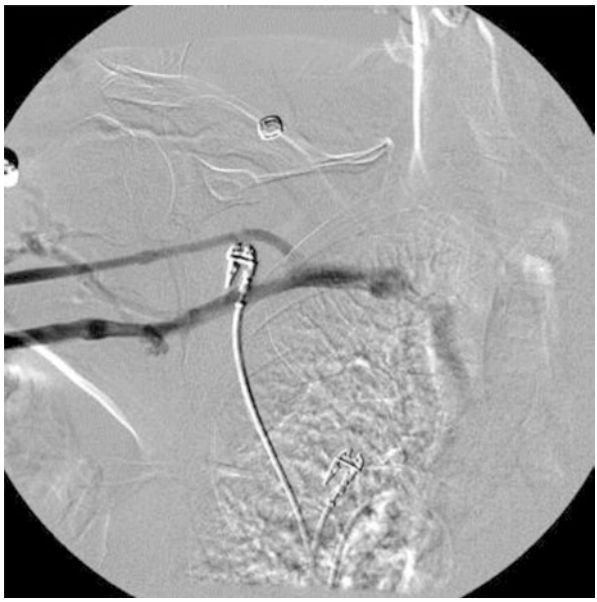

Fig. 2.8 ECG leads should not be placed within the fluoroscopic field

3. Minimize patient's movements to decrease movement artifacts, e.g., instructing the patient to breath-hold during imaging of the central veins will improve image quality.
4. Position the patient and X-ray detector before starting imaging.
5. Keep the area of interest in the center of the image.
6. Use collimation to "remove" unnecessary areas.
7. Use magnification to see details in a specific area when necessary.
8. Increase the number of pulses per second or frames per second where necessary.
9. Use full strength iodinated contrast material rather than diluted contrast material, especially when imaging the proximal or central vessels.
10. Oblique views may be necessary to delineate overlapping vessels and detecting eccentric vascular disease.

Endovascular Tools

3

Chieh Suai Tan, Zubin D. Irani, and Steven Wu

Introduction

The Seldinger technique, first described in 1953, revolutionized the way angiography was performed. It overcomes the traditional need for surgical exposure of a blood vessel before catheterization by using a guidewire to introduce devices into a blood vessel via a percutaneous puncture. The technique involves percutaneous puncture of a blood vessel with a hollow needle, introduction of a guidewire through the needle into the blood vessel lumen, removal of the needle while maintaining the guidewire in position, followed by advancement of a catheter over the guidewire.

The refinement of this technique by the placement of a sheath over the puncture site allows devices to be introduced via the same vascular access site without the need for multiple punctures. The tools for endovascular interventions are outlined in this chapter.

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Access Needle

All endovascular intervention begins with the insertion of a vascular access needle. There is a great variety of access needles that can be used. Examples include the micropuncture needle, introducer needle, sheath needle and angiocath (Fig. 3.1a–c). Their common feature is the presence of a central channel for the introduction of a guidewire. The diameter of a needle is described using the stubs iron wire gauge system in "gauge" or "G". The maximum guidewire diameter that an 18-G and

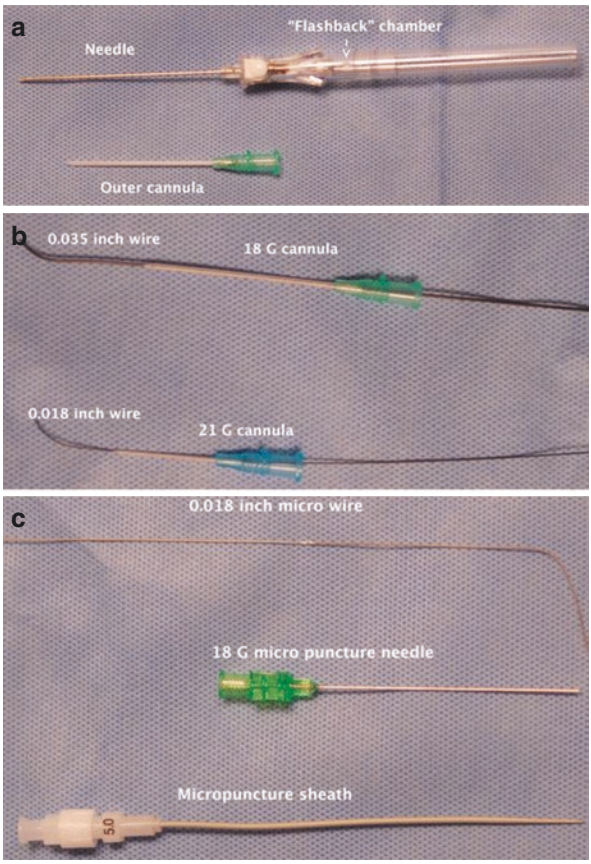

Fig. 3.1 (**a**) An angiocath consists of a hollow core needle with an outer sheath. The "flashback" chamber allows visualization of blood once the needle punctures the vessel. (**b**) An 18G cannula can accommodate a 0.035 in. guidewire while a 21G cannula can accommodate a 0.018 in. guidewire. (**c**) A micropuncture set consists of a micropuncture needle, a guidewire and a transitional sheath. The transitional sheath consists of an inner 3 Fr sheath and an outer 5 French sheath. The needle is used to puncture the vessel. The wire is threaded through the needle after a successful puncture. The needle is then removed, and the sheath inserted over the guidewire. The inner 3 Fr sheath can accommodate the 0.018 system while the outer 5 Fr sheath is able to accommodate the 0.035 system. The design of the transitional sheath permits upsizing from the 0.018 to 0.035 system when required

21-G needle can accommodate is 0.035 and 0.018 in. respectively. An 18G angiocath is routinely used to obtain access to an arteriovenous fistula or a graft in our institution. A fistulogram or graftogram can also be performed through the 18G angiocath via injection of contrast material. The 18G cannula can be exchanged for a vascular sheath over a 0.035 in. guidewire.

Sheath

Sheaths are used to secure the puncture site for vascular intervention. They are plastic tubes that are open on one end and capped with a hemostatic valve at the other (Fig. 3.2a). The hemostatic valve prevents bleeding and air embolism during the procedure and allows wires, catheters and other devices to be introduced into the vessel. The valve end usually has a short sidearm that can be used for flushing, contrast material administration and medications.

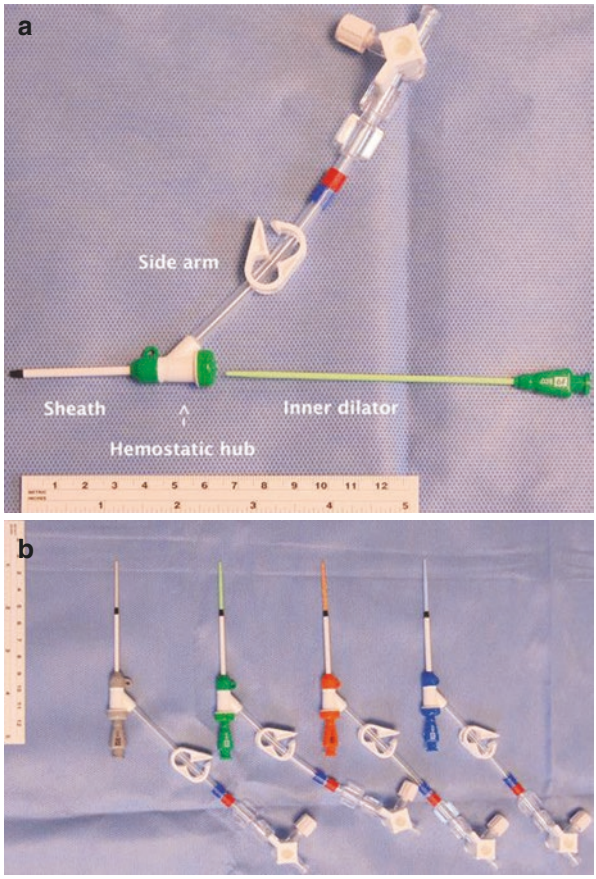

Fig. 3.2 (**a**) Components of a vascular sheath. (**b**) In general, short sheaths (4 cm) are used for dialysis access interventions. Sheaths are described by their inner diameters. The different sheath sizes are shown here. The 6 F sheath is frequently used as the routine sheath for dialysis access interventions

Sheaths are sized by their inner diameter described using the "French" (Fr) system, which is based on "π". The diameter of the sheath is obtained by dividing the "Fr" by "π" or approximately 3. For example, a 6-Fr sheath is approximately 2 mm by the inner diameter. The outer diameter for a sheath is 1.5–2 Fr larger; hence a 6 Fr (2 mm) sheath will create an 8 Fr (2.5 mm) hole in the vessel wall.

The size of the sheath to be inserted is determined by the diameter of the catheter or angioplasty balloon or device to be used (Fig. 3.2b). The product insert of the catheter or angioplasty balloon or device will specify the size of the sheath that is required.

Sheaths also come in different lengths. For AV access interventions, a short sheath (4 cm) is routinely used. In general, a 4 cm 6 Fr short sheath is often used as the routine sheath for intervention. The sheath can be "up-sized" over a guidewire for a larger sheath if a larger angioplasty balloon or stent deployment is required.

Dilator

Dilators are used to enlarge the puncture tract to facilitate the placement of sheaths, catheters or devices. Dilatation is done by sequentially passing larger dilators over a guide wire till the tract is adequately sized to accept the intended sheath, catheter or device (Fig. 3.3). Unlike sheaths that are sized by their inner diameter, dilators are sized by their outer diameter. Hence, a 7–8 Fr dilator is needed to enlarge the

Fig. 3.3 Dilators come in different diameters and lengths. The common feature is the presence of a tapered end

tract for a 6 Fr sheath. In general, sheaths come together with their appropriately sized dilators in a pack and extra dilators are not required unless you are planning to upsize the sheath by 2 Fr or more.

Catheter

Similar to dilators, catheters are sized by their outer diameter using the Fr system. Again, there is a huge variety of catheters available for diagnosis and intervention. Broadly, catheters can be classified based on their intended use. The material, shape of the catheter tip, end hole diameter, configuration of side holes (location, size and number) of each catheter are designed to fulfill its specific purpose.

Non Selective or Flush Catheter

A flush catheter is used for diagnostic angiography. It has an end hole and multiple smaller side-holes to allow for uniform dispersion of contrast material during administration. The "pigtail" catheter is a typical flush catheter with a curled tip that is used for aortography.

Selective Catheter

Selective catheters are used to seek the orifice of vessels and direct guidewire into a specific location. For this reason, they come in many different shapes. They have less or no side holes and generally have end hole design for angiography to perform angiography. The tip of the catheter may be angled like a hockey stick, such as a Kumpe catheter (Cook Medical, Bloomington, Ind) or have complex curvatures such as the "Cobra" catheters (Cook Medical, Bloomington, Ind).

Guiding Catheters

These are hybrid between diagnostic catheter and sheath. They are typically used in place of sheaths to access vessel of interest and to deliver tools for intervention. They are non-tapered and come in different tip configurations and lengths. Their French size refers to their outer diameter. Some guiding catheters have special hydrophilic coatings to enhance the trackability of the catheter over occluded vessels.

In summary, every catheter has its own unique characteristics and purpose. The best catheter is one that you are familiar with and is versatile enough to meet your expectations and requirements most of the time. A 65 cm long 4F Berenstein catheter or Kumpe catheter is routinely used for AV access intervention in our institution (Fig. 3.4a–d).

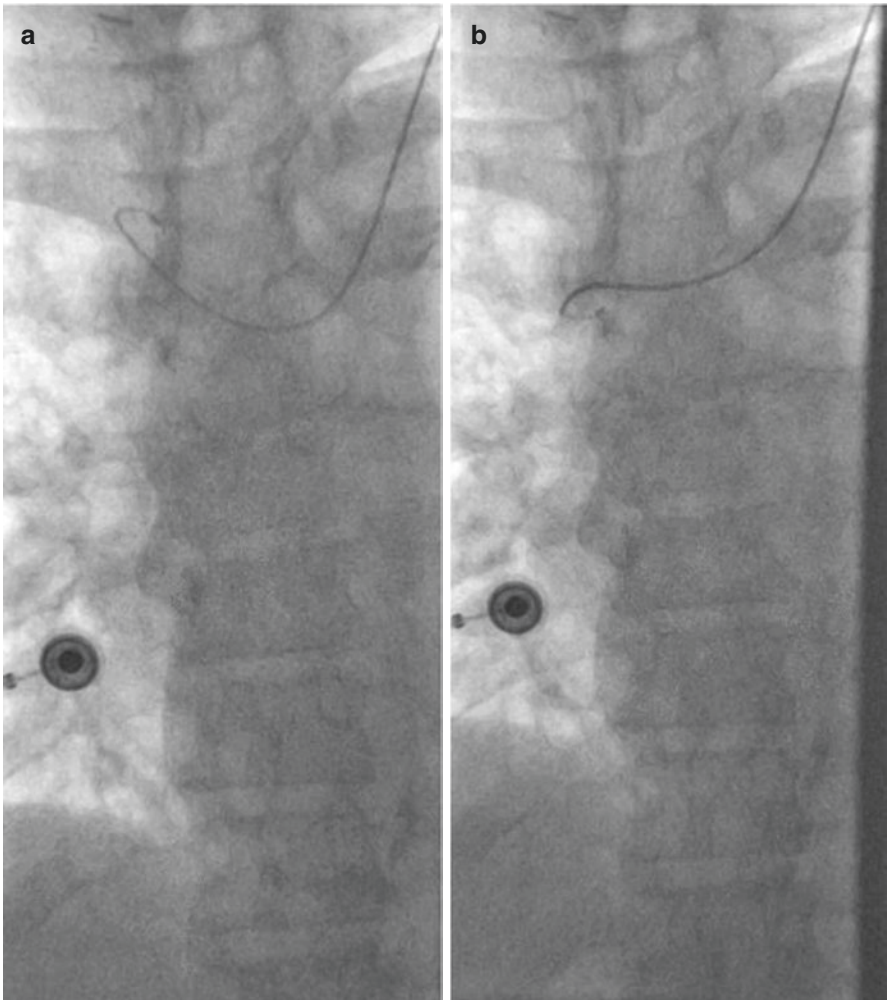

Fig. 3.4 (**a**) The guidewire repeatedly curled upward while attempting to maneuver it into the inferior vena cava. (**b**) A Kumpe catheter was introduced to "stiffen" the wire and exert more control over its movement. (**c**) The guidewire was navigated towards the IVC with the aid of the Kumpe catheter. (**d**) The guidewire finally passed into the inferior vena cava

Guidewire

Guidewires come in different thicknesses, lengths, stiffness, coating and tip configurations (Fig. 3.5). The diameters of a guidewire are measured in inches and are available in sizes ranging from 0.008 to 0.038 in.. The common sizes in everyday use are the 0.018 and 0.035 in. wires. Guidewires may have a

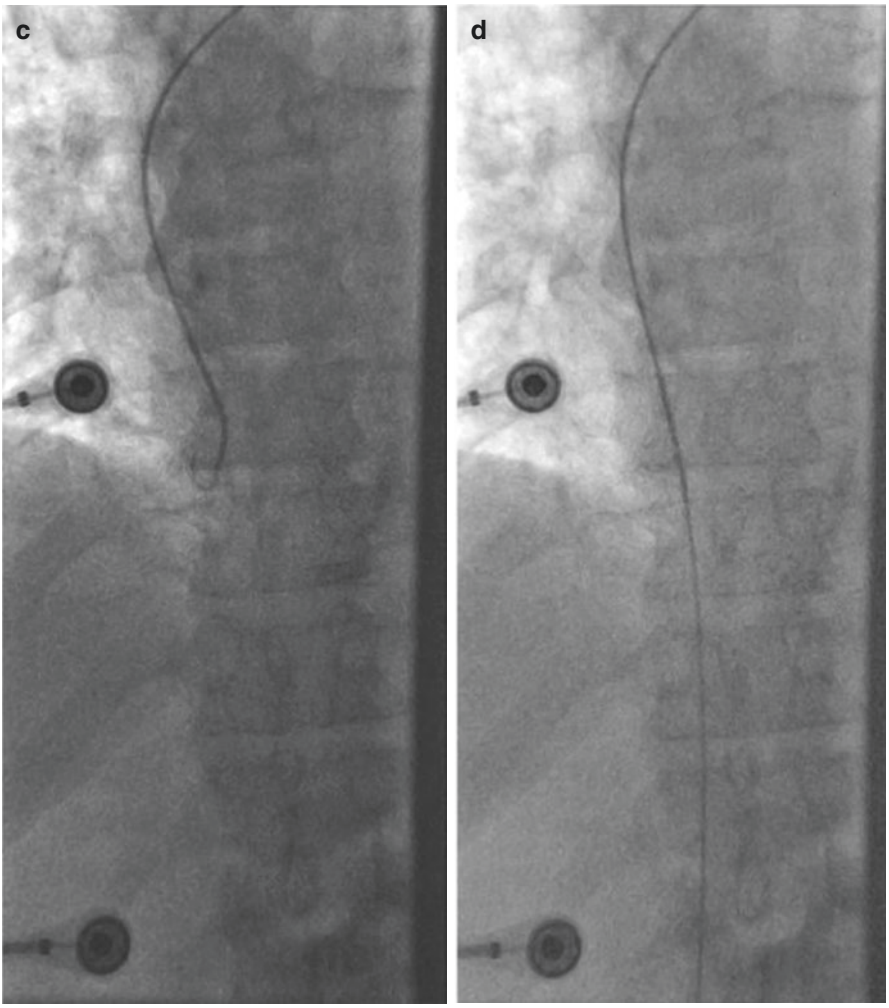

Fig. 3.4 (continued)

hydrophilic coating to enhance their maneuverability. The hydrophilic or "water seeking" coating enables the wire to be advanced easily within the vessels. Hydrophilic wires with steerable or angled tip designs are especially useful in crossing critically stenotic lesions. However, the hydrophilic coating makes the wire feel slippery and can give an impression of movement when it is stationary. A 180 cm 0.035 in. regular angled tip hydrophilic wire is routinely used for dialysis access intervention in our institution.

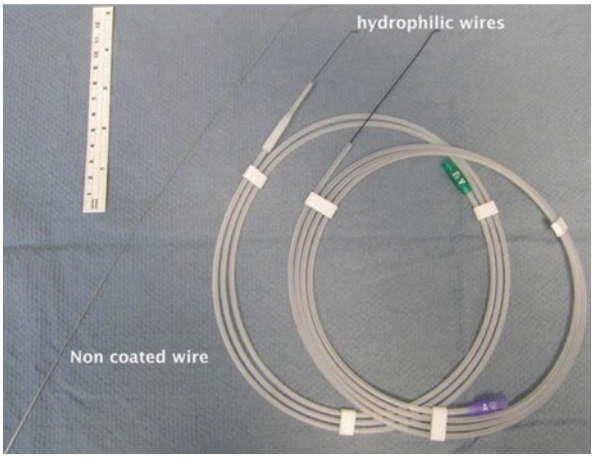

Fig. 3.5 Guidewires can come in different lengths, tip designs and coating

Balloon Catheter

There are two basic types of balloons. A non-compliant balloon is used for angioplasty while a compliant balloon (e.g. Fogarty balloon) is used for embolectomy or temporary vascular occlusion. These balloons are mounted on catheters and the shaft of the balloon catheter is described using the Fr system (Fig. 3.6a–g) and the length of the shaft in centimeters.

The size of the balloon, on the other hand, is described by its diameter in millimeters when inflated; followed by its length in centimeters. For example, an "8 by 4" balloon has an 8 mm diameter and a 4 cm length when inflated and may be mounted on a 5 Fr shaft. The balloon is tightly wrapped around the balloon catheter before inflation. After use, the deflated balloon will not return to its original size and can be larger in diameter than the shaft that it is mounted on. As such, an 8 mm balloon that is mounted on a 5 Fr balloon catheter will require a 6 Fr sheath to permit smooth removal of the balloon catheter. The size of the sheath required to permit the passage of the balloon catheter is usually described in the product insert.

A "cutting" or "scoring" balloon is a non-compliant balloon with atherotomes or blades mounted on its surface to "cut" or "score" the stenotic lesion during inflation. It is useful in the treatment of stenotic lesions that are resistant to balloon angioplasty.

The angioplasty balloons that are used for dialysis access intervention are usually non-compliant and can be inflated to "high pressure". It is important to know the nominal pressure and the rated burst pressure of the balloon when performing an angioplasty procedure. These pressures are usually indicated on the product insert of the angioplasty balloon. The nominal pressure is the inflation pressure at which the stated diameter of an angioplasty balloon is achieved. The rated burst pressure (RBP) is the pressure at which 99.9% of balloons can maximally withstand before rupture with 95% confidence. It is advisable not to inflate beyond the rated burst pressure as rupture of the angioplasty balloon can result in embolism of the balloon fragments and retrieval of a ruptured balloon may be difficult.

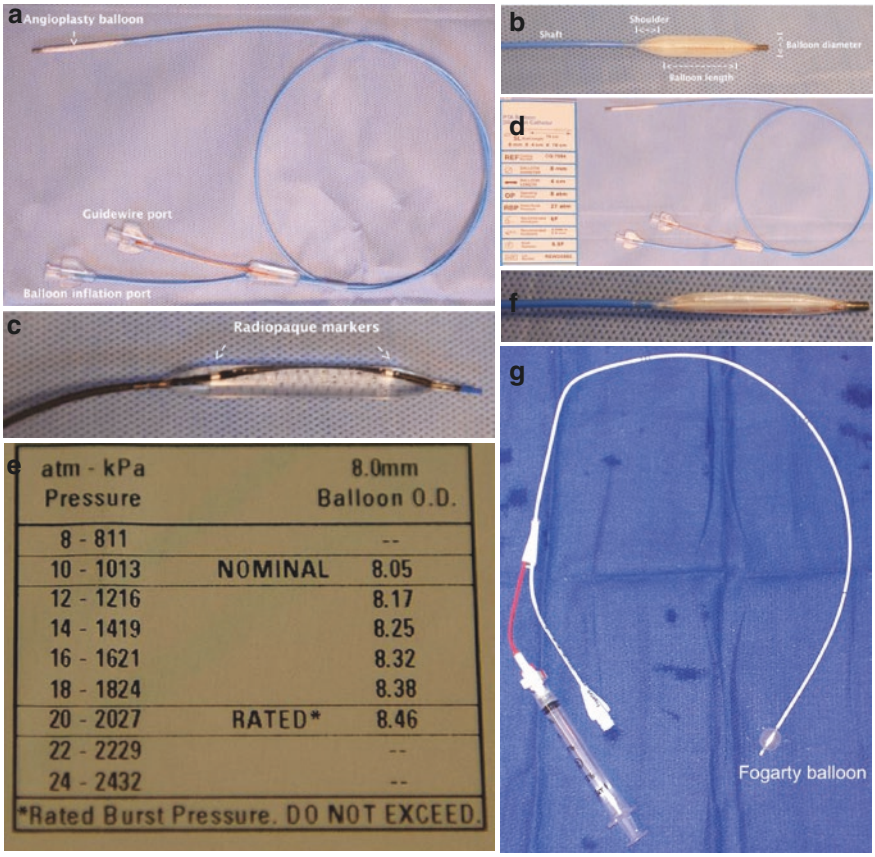

Fig. 3.6 (**a**) Components of an angioplasty balloon catheter. This is an example of a high pressure, non-compliant balloon. (**b**) The size of an angioplasty balloon is described by its diameter in millimeters when inflated; follow by its length in centimeters. The balloon is mounted on the tip of a catheter. (**c**) Radiopaque markers are present to mark the position of the balloon during fluoroscopy. (**d**) The characteristics of the angioplasty balloon catheters are described in the product insert. Information on the recommended sheath size, guidewire and rated burst pressure of the balloon is indicated. (**e**) The nominal pressure and rated burst pressure are usually indicated in the product insert. The nominal pressure is the inflation pressure at which the stated diameter of an angioplasty balloon is achieved. The rated burst pressure (RBP) is the pressure at which 99.9% of balloons can maximally withstand before rupture with 95% confidence. (**f**) The deflated angioplasty balloon will not return to its pre-inflated size after being used and is bigger than the diameter of the balloon catheter that it is mounted on. Therefore, even though it is mounted on a 5 Fr balloon catheter, a 6 F sheath is required to permit smooth removal of the entire balloon catheter after use. (**g**) The Fogarty balloon (Edwards Lifesciences, Irvine, CA) is an example of a low pressure, compliant balloon

The angioplasty balloon can be inflated either using an inflation device or a syringe assembly. The inflation device looks like a huge syringe with an attached manometer. It has a locking mechanism to maintain pressure and allow inflation of an angioplasty balloon to a precise pressure. These devices are designed for one-time use and may be costly. An alternative is to manually inflate the balloon using a syringe assembly consisting of a 3 and 10 mL syringe connected via a 3-way stopcock. The balloon is first inflated using the 10 mL syringe. The 3-way stopcock is then turned and pressure is maintained using the 3 mL syringe. The pressure should be maintained for approximately 3 min to ensure adequate dilatation and effacement of the stenosis.

Stents

Stents are broadly classified into balloon-expandable or self-expanding balloon stents. Balloon expandable stents require balloon dilatation to increase their diameter from the compressed state. They have greater radial strength and can be over dilated but they will not return to their deployed shape when crushed or compressed; hence they are less suitable to be used in peripheral vessels in the limbs. However, they are preferred when accurate positioning of the stent is of paramount importance. One such example is during the treatment of subclavian artery ostial stenosis, where the stent has to be positioned accurately across the stenosis with minimal protrusion of the stent into the aortic lumen. Self-expanding stents will conform to the vessel wall and expand to its designed diameter. They have greater flexibility and will return to the original shape after bending or compression.

The flexibility and strength of a stent are dependent on the construction material, design and configuration of its "cells". A stent is made up of multiple cells that are connected by struts. A closed-cell design is one where struts support every cell. In comparison, in a stent with open-cell design, some cells are not in contact with any struts at all. By varying the cell designs and number of struts, the flexibility and radial strength of a stent can be altered.

Stents can be constructed with stainless steel or alloys such as Nitinol and Elgiloy (Fig. 3.7a). Nitinol exhibits shape memory, allowing it to regain its shape after compression. Stents that are constructed using Nitinol provide better long-term patency compared with stainless steel stents in the treatment of hemodialysis graft related stenosis [1].

Stents may also be "bare" or "covered" with relatively inert polymeric covering such as expanded polytetrafluoroethylene (PTFE) (Fig. 3.7b). The intention of the covering is to decrease the high restenosis rates associated with bare-metal stents that are caused by neointimal hyperplasia. The covering is postulated to work by providing a barrier to prevent the migration of smooth muscle cells and separate the thrombogenic wall surfaces from the luminal blood flow. The characteristics of the four types of self-expanding covered stents that are available in the United States are summarized in Table 3.1 [2]. Covera and viabahn stents are approved by the FDA for the treatment of venous anastomotic stenosis in arteriovenous grafts.

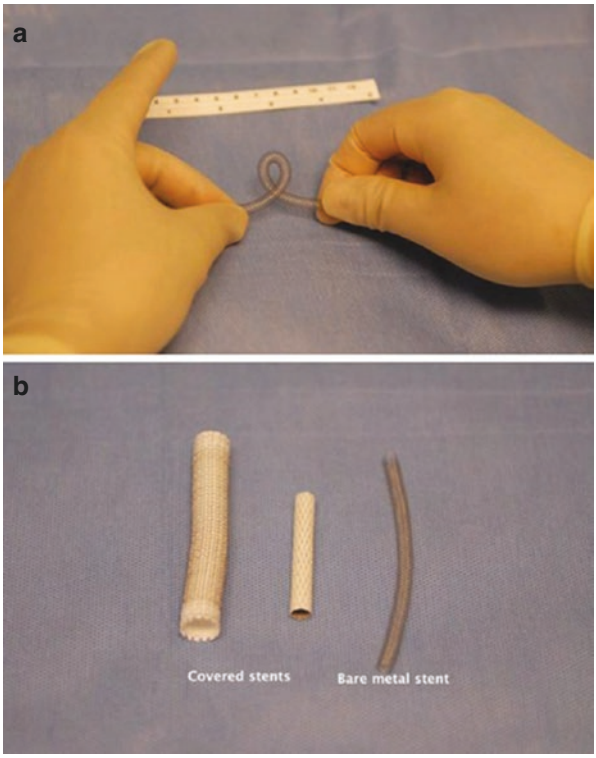

Fig. 3.7 (**a**) Bare nitinol stents exhibit great flexibility and return to their original shape after compression. (**b**) Different types of stents

Table 3.1 Types of stents used in dialysis vascular access intervention

Name of stents	Wallgraft	Viabahn	Fluency plus	Covera
Stent material	Elgiloy	Nitinol	Nitinol	Nitinol
Covering	Polyethylene terephthalate	Expanded PTFE	Expanded PTFE	Expanded PTFE
Characteristics	Relatively rigid with poor contourability	Very flexible	Stiffer than Viabahn	Very flexible, available in a straight or flared configuration

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Basic Endovascular Skills and Techniques

4

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Introduction

Performing interventional procedures requires a different skillset not traditionally associated with nephrology training. It involves developing cognitive skills and understanding indications for different interventional procedures, their limitations and potential complications, mastering fluoroscopic eye-hand coordination, understanding the behaviors and properties of various guidewires and catheters and acquiring fine motor skills in manipulating these devices. These skills are essential to perform interventional procedures safely and effectively. Competencies can be achieved with the right attitude and adequate hands-on procedural and clinical training.

Preparation for Intervention

1. History and physical examination aid in determining the site of lesions and treatment planning. Always examine the patient, understand the indications for intervention and review the ultrasound images and images from any previous

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interventions and operations. Plan the approach in advance and identify the initial puncture site. Anticipate any problems that you might come across during the procedure (Fig. 4.1a).

2. Ultrasound is an essential tool to confirm the findings of your physical examination. Start scanning the AVF or AVG from the AV or arterial-graft anastomosis respectively. Move the probe along the entire length of the AVF or AVG to visualize the site of stenosis (Fig. 4.1a).

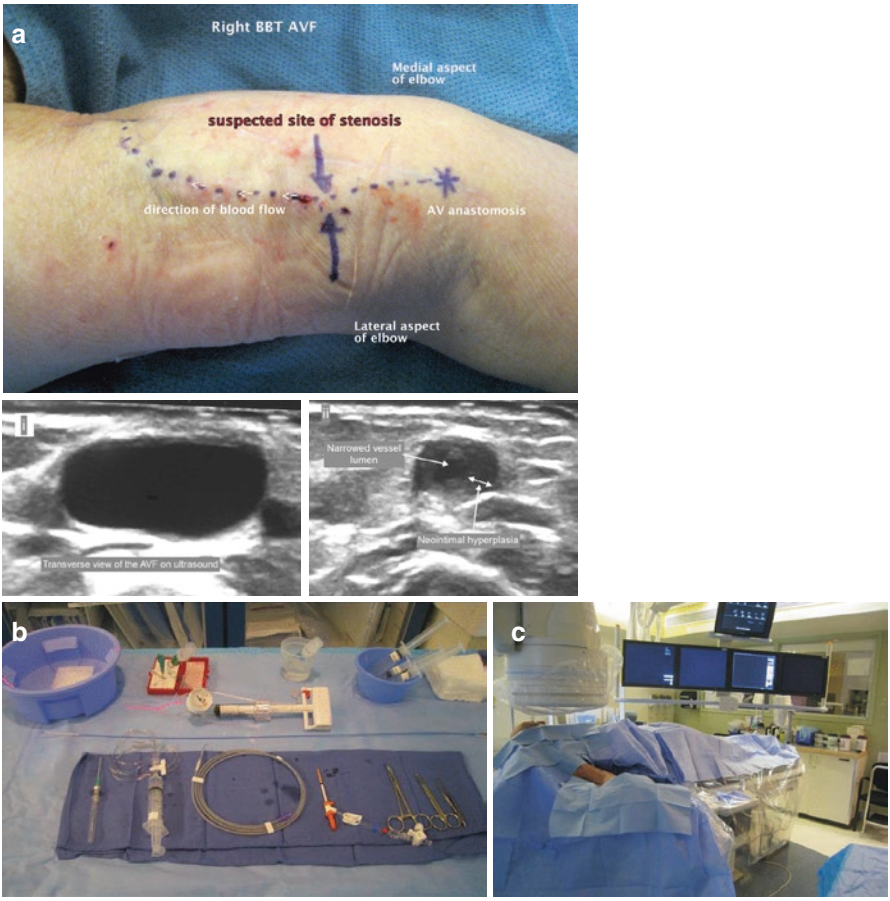

Fig. 4.1 (**a**) Right arm of the patient. Note the needling sites on the medial side of the arm and the presence of a scar that runs along the AVF. This patient has a right brachiobasilic arteriovenous fistula. Assess the direction of blood flow and the possible site of stenosis. Confirm the site of stenosis using ultrasound. (**i**) shows the appearance of a non-stenotic segment on transverse view. (**ii**) shows the appearance of the stenotic segment on ultrasound (**b**) Prepare your equipment on a table in a sterile fashion. (**c**) Clean and drape the patient, leaving only the area of interest exposed. Cover the sides of the table and image detector with transparent sterile sheets

3. The equipment needed for the intervention should be prepared and laid out on the procedure table in a sterile fashion. Flush the lumens of all the sheaths and catheters with normal saline solution (Fig. 4.1b).
4. The patient should be positioned such that it enables you to work forehand with your master hand most of the time.
5. Clean and drape the patient in a sterile fashion. Only the area of intervention should be exposed. The rest of the body and equipment surfaces should be covered by sterile drapes (Fig. 4.1c). Proper drapes are required as the length of wires and catheters can be long, and the wires or catheters can easily get contaminated when they fall on an uncovered area.

The Initial Puncture

The initial puncture can be done blindly or with ultrasound guidance. Ultrasound is particularly useful when cannulating an immature or severely stenosed vascular access. We routinely use an 18G angiocath (intravenous catheter) for the initial puncture of the vascular access. The angiocath consists of a needle and a plastic cannula (Fig. 4.2). The plastic cannula does not cover the needle completely. The cannula ends just before the tip of the needle becomes beveled. Hence, the angiocath needs to be advanced an extra few millimeters after seeing the initial flashback of blood. Advancing the angiocath is necessary as the needle tip has entered the lumen of the vessel but the cannula may still be within the vessel wall. Premature withdrawal of the needle from the cannula will lead to an inability to thread the cannula into the AV access. The alternative would be to do the initial puncture with a sheath needle to exchange it for a vascular sheath over the guidewire. The initial puncture can be carried out using ultrasound to provide real-time visualization of the passage of the needle as shown in Fig. 4.3.

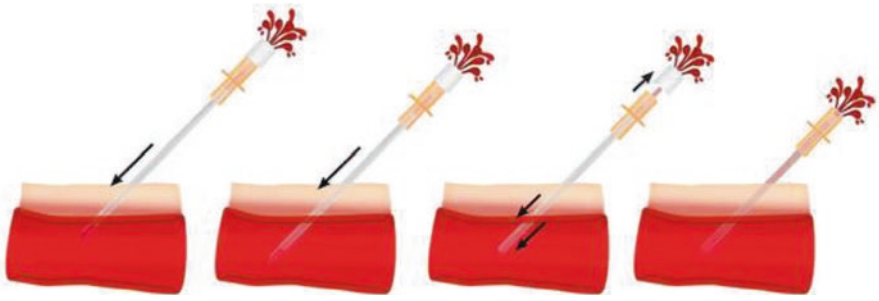

Fig. 4.2 The plastic cannula of the angiocath does not cover the needle completely. Therefore, even if there is "flashback" of blood when the needle tip enters the vessels, the cannula may not yet be within the vessel. There will be difficulties threading the cannula into the vessel. Hence, advance the angiocath a few millimeters more after the initial "flashback" before pushing the cannula into the vessel

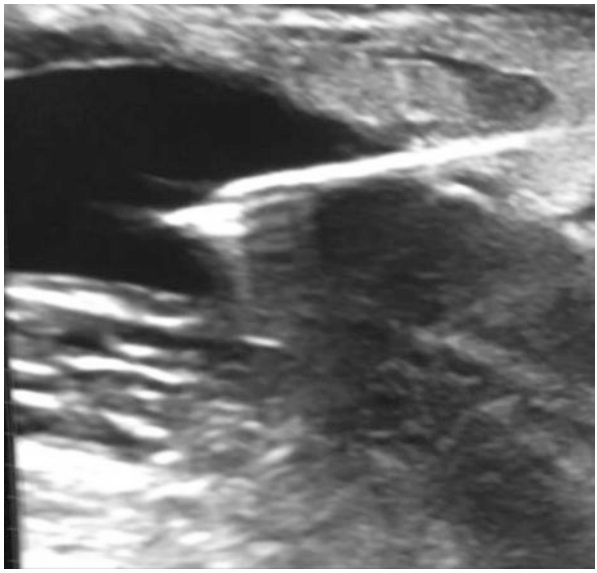

Fig. 4.3 Real-time ultrasound guided cannulation of the AVF using a sheath needle

1. Locally anesthetize the area with lidocaine before placing angiocath (Fig. 4.4a). Hold the vessel or graft between the left thumb and index finger.
2. Hold the angiocath in your right hand like a pen (Fig. 4.4b).
3. The angle of the puncture should usually be about 40–45 degrees. It should not be too steep in order to allow for smooth passage of the guidewire.
4. Advance the angiocath gently. Once a flashback of blood is seen, advance the angiocath by a few millimeters more. Hold the needle in position while advancing the cannula (Fig. 4.4c). Remove the needle from the cannula and attach a syringe to the cannula (Fig. 4.4d). An initial diagnostic fistulogram or graftogram can be performed by injecting contrast material through the cannula. Adding an extension tubing between the cannula and the syringe can allow the operator to inject the contrast material from a distance, thus decreasing the operator's radiation dose.
5. A "flashback" will not be seen when puncturing a thrombosed graft or fistula. Instead, you will feel a "give" when the needle enters the graft or fistula. Advance the cannula and remove the needle. Jiggle the cannula in and out of the graft. If you are within the graft, some blood (dark-colored) may appear within the cannula. Confirm the position by passing a 0.035 in. guidewire into the cannula gently and checking under fluoroscopy or ultrasound.

Sheath Placement

The puncture site is secured by exchanging the cannula for a vascular sheath over the guidewire.

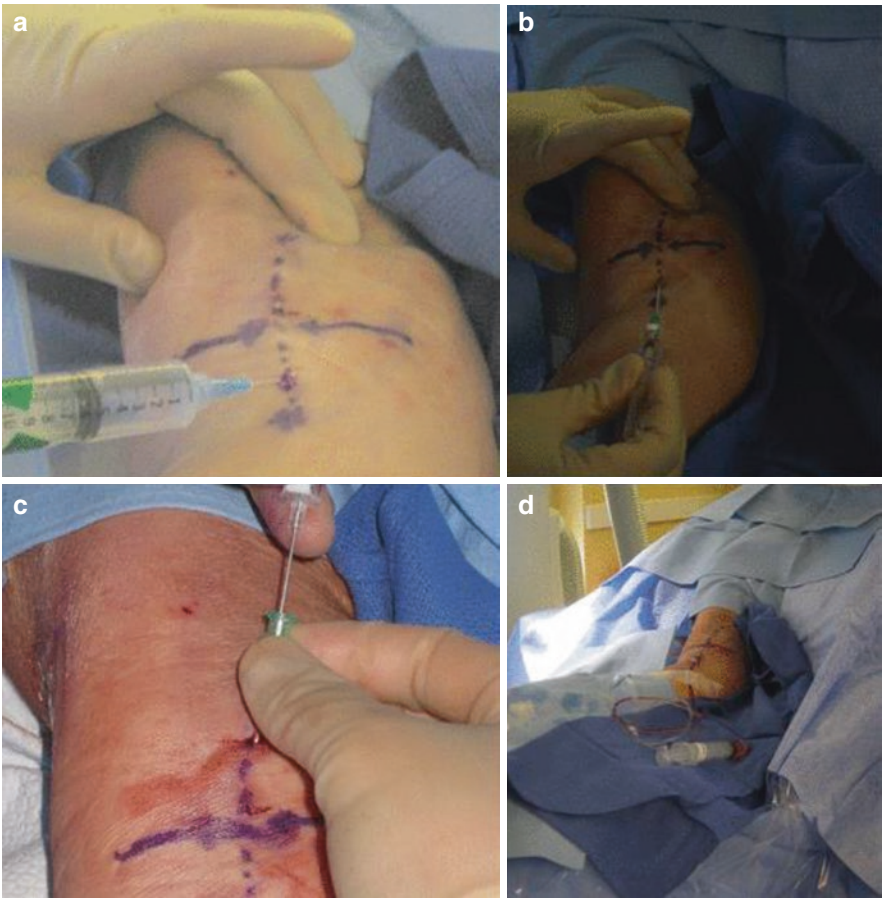

Fig. 4.4 (**a**) Anesthetize the puncture site with lidocaine. (**b**) Hold the angiocath like a pen. (**c**) Hold the needle and push the cannula into the vessel. (**d**) Connect the syringe to the cannula to do a diagnostic fistulogram

1. Insert a guidewire into the cannula. The floppy tip of the guidewire can sometimes be difficult to pass through the hub of cannula or needle. Grasp the wire near its leading edge between the thumb and the index finger while pinning the wire against the palm with the rest of the fingers. Straighten the wire tip by applying an upward traction force using the thumb and index finger. Occasionally, the wire may not pass as the cannula/ access needle is abutting against the vessel wall. Pull back the cannula/access needle to advance the wire (Fig. 4.5).
2. The guidewire should be advanced a few centimeters at a time (Fig. 4.6a). If the distance between the catheter hub and location where the guidewire is grasped is too far apart, the guidewire will buckle during advancement (Fig. 4.6b).

3. The insertion of the guidewire should be under fluoroscopic guidance (Fig. 4.6c, d). The tip of the guidewire should be advanced up the vessel as far as possible. Once in position, pin down the wire while removing the cannula (Fig. 4.6e).
4. The vascular sheath comes coupled to an inner dilator. Thread the vascular sheath with the inner dilator over the guidewire and advance it into position (Fig. 4.6f). The tapered leading edge of the inner dilator will help to dilate the path for the sheath. Once the sheath sits snugly over the initial puncture site, remove the inner dilator from the sheath.

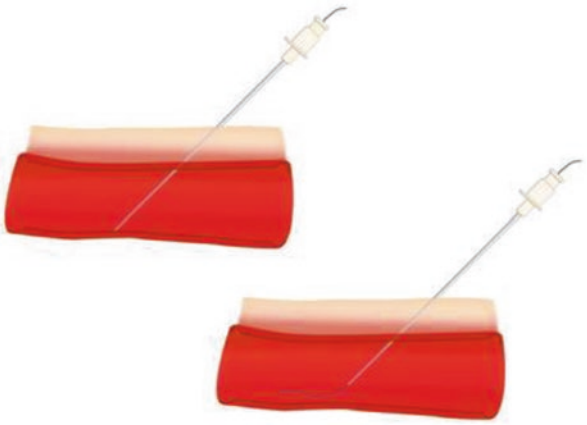

Fig. 4.5 The wire may appear to stuck while passing through a cannula or access needle. This occurs when the angle of the needle entering the vessel is too steep or if the tip of cannula is abutting the vessel wall. Pull back the cannula slightly to advance the wire

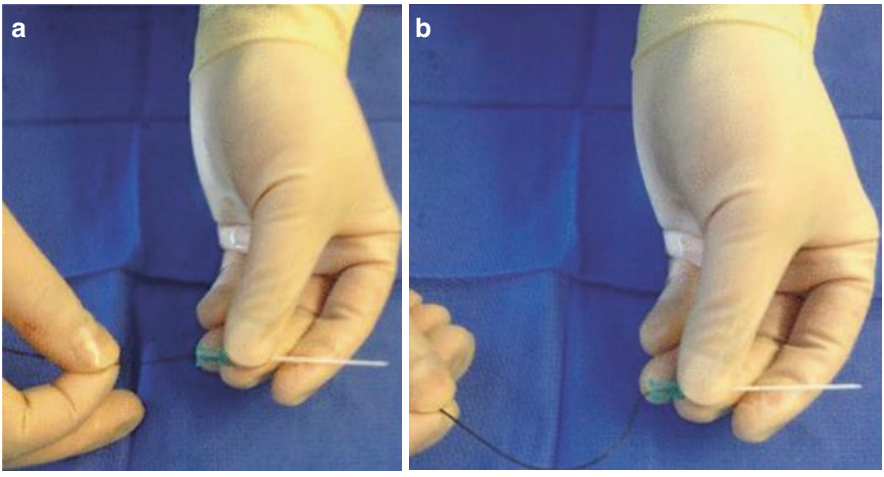

Fig. 4.6 (**a**) Hold the wire near the hub to advance it. (**b**) If the wire is grasped too far from the hub, it will buckle when you try to push. (**c**) Insert the wire into the cannula. (**d**) Push in the wire under fluoroscopic guidance. (**e**) To minimize bleeding, compress the venotomy site when removing the cannula. (**f**) Thread the sheath into the vessel over the guidewire, while holding and using the guidewire as a rail

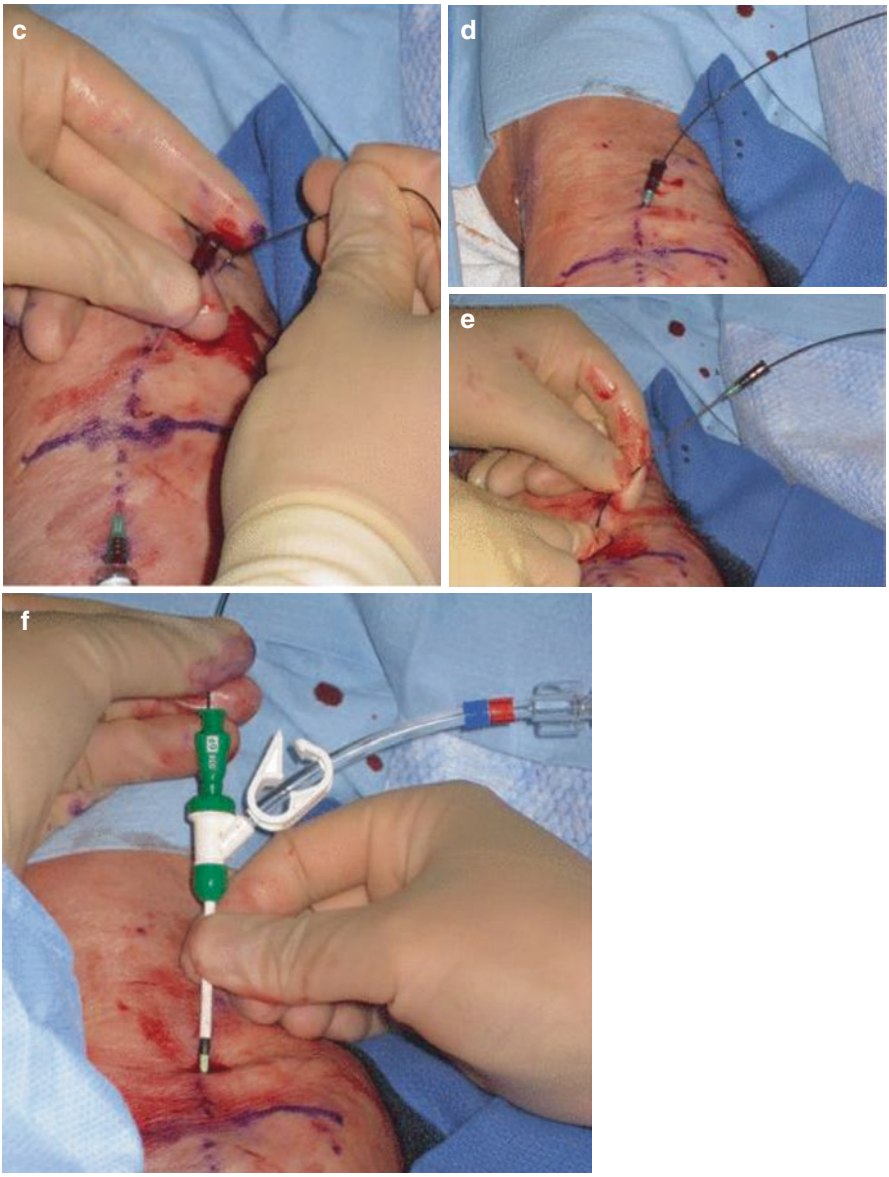

Fig. 4.6 (continued)

5. With the sheath in place, catheters, angioplasty balloons and stents can be threaded over the guidewire into the vascular lumen for endovascular interventions.

Contrast Material and Fluoroscopy Tips

1. "Half strength" contrast material is used when imaging the peripheral vessels. Dilute the contrast material with normal saline in a 50/50 mixture. "Half strength" contrast material is also used to fill up the inflation device to inflate angioplasty balloons.
2. "Full" strength (undiluted) contrast material is used when imaging the central veins. The patient should be instructed to hold his breath while imaging the central veins to decrease movement artifacts and improve image quality. The digital subtraction angiography (DSA) pedal should only be depressed after the chest has stopped moving.
3. For DSA, inject the contrast material only after the "mask" has been created, i.e., after the screen has changed to a "white-out" appearance.

Sheath Removal

1. To secure hemostasis after sheath removal, apply a purse-string suture around the sheath (Fig. 4.7a, b).
2. Tighten the two ends of the purse-string suture while removing the sheath (Fig. 4.7c).
3. Compress the venotomy site while tying down the suture (Fig. 4.7d, e).
4. Examine the thrill of the AV access after hemostasis is secured.
5. Stitches can be removed in 1–2 days.

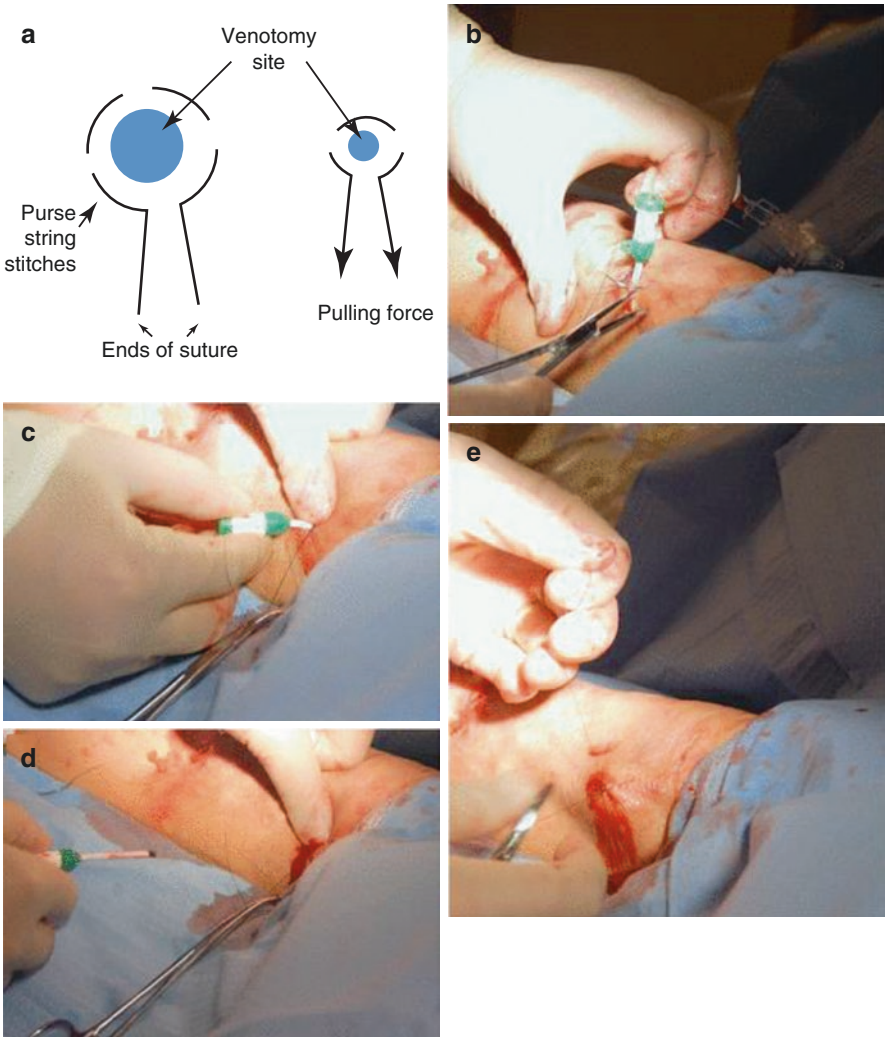

Fig. 4.7 (**a**) The purse-string suture is performed by running the stitches in and out along the edge of a wound in circular fashion such that when the ends are drawn tight, the wound is closed like a purse. (**b**) Apply a purse-string suture around the sheath, beginning at the 6 o'clock position. Keep a close distance to the sheath as you apply the stitches. Encircle the sheath with the purse-string suture. (**c**) Pull the purse-string suture as you remove the sheath. (**d**) Compress the venotomy site once the sheath has been removed. (**e**) Tie down the suture to secure hemostasis

Radiation Safety

5

Patrick D. Sutphin, Steven L. Hsu, and Sanjeeva Kalva

Introduction

At the end of 2013, 421,000 persons with end stage renal disease (ESRD) were treated with hemodialysis, and the number of ESRD individuals requiring hemodialysis will continue to increase in the foreseeable future [1]. The mortality rate for ESRD patients receiving dialysis has been declining since 2002 [1]. The combination of increasing prevalence of ESRD patients requiring hemodialysis and their improved survival will continue to fuel the growth in the number of fluoroscopically-guided hemodialysis access interventions [2].

Recognizing the serious injuries arising from prolonged radiation exposure during fluoroscopically-guided procedures, the United States Food and Drug Administration issued a Public Health Advisory in 1994, which not only raised the level of awareness and concern of physicians utilizing fluoroscopy, but also prompted investigations for improvements in reduction and documentation of radiation exposure.

In addition to acute radiation exposure injuries, hemodialysis patients are at a greater risk of all-cause mortality as well as an increased risk for cancer and cardiovascular disease. These patients tend to have multiple comorbidities and risk factors that contribute to the risk of cancer and cardiovascular disease, but the traditional risk factors may not account for all of the increased risk [3, 4]. A recently proposed risk factor in hemodialysis patients for both cancer and cardiovascular disease is the cumulative exposure to ionizing radiation. Kinsella et al. performed a retrospective

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study of 100 maintenance hemodialysis patients with a median follow up of 3.4 years. Review of patient records found a median annual dose of 6.9 mSv per patient year and a median cumulative effective dose (CED) of 21.7 mSv over the study period. Thirteen of the 100 patients studied had a CED greater than 75 mSv. Approximately14% of the CED was related to dialysis access procedures [5]. Additional studies confirmed the elevated CED in dialysis patients [6, 7]. As survival of patients on hemodialysis improves, the elevated CED for some patients may have significant clinical impact. This chapter will focus on methods to minimize radiation exposure during fluoroscopy guided dialysis access interventions.

Definitions and Units

Radiation Exposure

Radiation exposure is the amount of electrical charge produced by ionizing electromagnetic radiation in a unit mass of air. Exposure is expressed in coulombs per kilogram or roentgens [8, 9]. The quantity of ionization of air can be correlated to absorbed dose.

Air Kerma

Kerma is an acronym for **k**inetic **e**nergy **r**eleased in matter. Kerma is measured in the clinical setting as air kerma, which is the kinetic energy released into air and expressed in units of gray (Gy) [8, 9].

Absorbed Dose

Absorbed dose (D) is amount of radiation energy absorbed per unit mass of matter. The absorbed dose can also be expressed in units of Gray, which facilitates comparison of air kerma and absorbed dose. An air kerma of 1 mGy is deemed to be approximately equivalent to an absorbed dose of 1 mGy [8, 9].

Peak Skin Dose

The peak skin dose is the highest radiation dose at a point on the patient's skin and expressed in units of Gray [10, 11].

Kerma-Area Product (KAP)

Kerma-area product is also known as roentgen-area product or dose-area product. KAP is computed by multiplying the entrance skin dose to the area of the radiation

beam. KAP is expressed in Gy·cm^2. Temporal summation of KAP provides an estimate of the skin dose [8, 9, 11, 12].

Effective Dose

Performance of a radiologic examination emphasizes targeted radiation exposure for the patient. For example, when a patient with an upper extremity arteriovenous (AV) fistula presents with elevated venous pressures and prolonged bleeding at the cannulation sites following hemodialysis, fluoroscopic images should be limited to the patient's upper extremity and chest. Not all of the tissues in the upper extremity and chest have the same sensitivity to the stochastic effects of radiation. Therefore a radiation-weighting factor for each organ has been computed to take into account the risk to each exposed organ. The effective dose is the weighted sum of the doses to all exposed organs. The effective dose provides a total estimated risk to the patient from radiation exposure [8, 11, 12].

Effects of Radiation

Deterministic Effects

The deterministic effects of radiation exposure occur when a threshold radiation dose is exceeded. The severity of deterministic effects increases with the dose. An example of a deterministic effect is radiation-induced skin erythema, which occurs when a skin dose of 2 Gy has been surpassed [8, 11–13]. When the skin dose exceeds 5 Gy, then permanent partial epilation can occur, and when the skin dose exceeds 10 Gy, then permanent epilation occurs along with dermal atrophy or induration [13].

Stochastic Effects

Stochastic effects are not related to a threshold dose. The probability of occurrence of a stochastic effect increases with increasing radiation dose. Radiation-induced cancer is the most concerning stochastic effect. Although radiation exposure may not engender cancer for all individuals, increasing the radiation exposure will increase the probability of inducing cancer.

Dose Limits

The International Commission on Radiological Protection (ICRP) was founded in 1928 and has published recommendations to limit the detrimental effects of radiation for all individuals [14]. ICRP has published the recommended dose limits for radiation workers and members of the public. The following are occupational dose limits and do not pertain to planned exposure of patients.

Whole Body Dose

The ICRP recommends a whole body dose limit equal to an effective dose of 20 mSv per year averaged over a 5-year period. Thus, the total effective dose should not exceed 100 mSv during the 5-year time interval. Furthermore, within any single year, the effective dose should not exceed 50 mSv [14].

Extremity Dose

The majority of radiation exposure in hemodialysis interventions is directed at the extremity. Skin and bone are relatively insensitive to the stochastic effects of radiation, thus the ICRP dose limit for extremities is correspondingly higher compared to the average whole body effective dose. Although hemodialysis fistulas and grafts are more durable than tunneled hemodialysis catheters, fistulas and grafts typically require repeat interventions to optimize their function and prevent access loss, thus the interventional radiologist should be mindful of one's occupational exposure and also the patient radiation exposure and deterministic effects which can occur. The recommended dose limit for extremities is 500 mSv per year [14].

Methods to Reduce Radiation Exposure During Dialysis Access Interventions

Pre-procedure Planning

Reduction of patient radiation exposure begins during the pre-procedure planning phase. The details of a patient's prior interventions and associated images should be reviewed to familiarize the interventional radiologist with the patient's vascular anatomy, identify appropriate sites of vascular access, and anticipate problematic locations. Meticulous review can reduce the procedure time, utilization of the angiography suite, and dramatically lower radiation exposure.

Prior to performance of a procedure, the cumulative radiation dose should be aggregated and the dates of prior procedures should be noted. The effects of radiation exposure as it relates to skin injury are considered additive when acquired within a 60-day period [11, 15]. Any poorly functioning or completely nonfunctional hemodialysis access should be managed expeditiously. Although the cumulative radiation dose acquired within the 60-day timeframe is taken into consideration, this should not thwart prompt performance of hemodialysis access interventions. Prior recent radiation exposure should guide interventional radiologists to inform patients of the potential for skin injury.

Once the patient arrives to the angiography suite, a confirmatory ultrasound of the arteriovenous graft or fistula should be performed to verify the planned sites of access and to further elucidate the locations of the graft or fistula that may require intervention.

Procedural Techniques for Patient Radiation Dose Reduction

The principle of ALARA (as low as reasonably achievable) must be a priority when imaging patients for diagnostic or therapeutic purposes. The following are techniques which minimize patient radiation exposure and permit adherence to the ALARA principle.

Collimation

Collimation involves defining the boundaries of radiation exposure. Only the immediate location where clinical information is required should be imaged. Not only does collimation reduce radiation dose to the patient, collimation also improves image contrast and quality by reducing the scatter radiation incident on the detector (Figs. 5.1 and 5.2).

Exposure Time

Being cognizant of the radiation exposure time and making active attempts to reduce the exposure times help adhere to the ALARA principle. For a given pulse dose, reducing the exposure time, will reduce the overall patient radiation exposure. At our institution, interventional radiologists are routinely notified when the exposure time exceeds 60 min. Following 60 min of exposure time, our technologists have been instructed to communicate when an additional 5 min of exposure time has transpired. Our institutional policies adhere to the guidelines for patient radiation

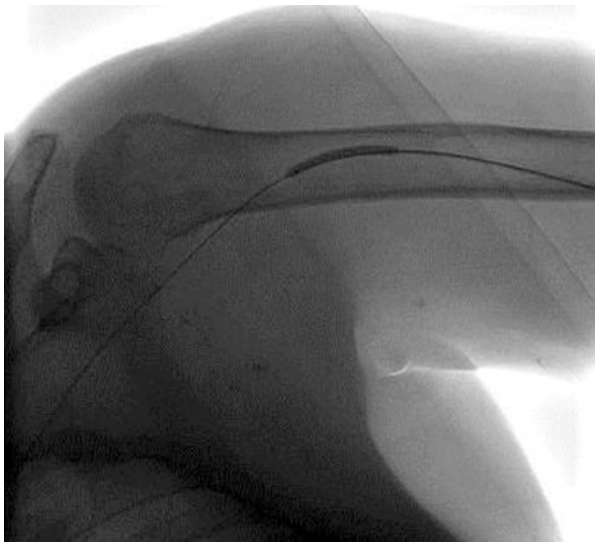

Fig. 5.1 Lack of collimation: Angioplasty performed within the cephalic vein at the site of outflow vein stenosis without consideration of collimation

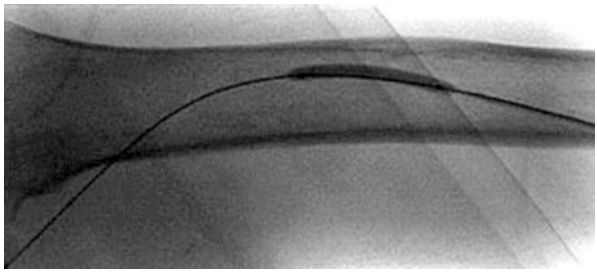

Fig. 5.2 Collimated image: Angioplasty performed within the cephalic vein at a second site of stenosis with collimation demonstrates a corresponding improvement in image contrast and quality while reducing radiation dose

dose management jointly established by the Society of Interventional Radiology (SIR) and the Cardiovascular and Interventional Radiological Society of Europe (CIRSE) [11]. The guidelines recommend informing the operator when any one of several conditions occur. These conditions include exceeding a fluoroscopy time of 60 min, surpassing an air kerma of 5000 mGy, exceeding a final peak skin dose of 3000 mGy, and accumulating a kerma-area product of greater than 500 Gy·cm^2 [11]. Knowledge of the exposure time should not prompt an interventional radiologist to cancel or inadequately complete a procedure, however, knowledge of increasing exposure times should guide the physician toward alternative procedural approaches or seek consultation from more experienced colleagues.

Object-Detector and Source-Detector Distances

The distance from the patient to the image detector should be minimized. Minimizing the distance of the patient to the detector reduces scatter and beam intensity. Conversely, the source-detector distance should be maximized. The inverse-square law states that the radiation dose to an object is inversely proportional to the square of the distance from the radiation source to the object. Thus, the procedural table on which the patient is positioned should be elevated as much as possible from the radiation source, however, patient positioning should not limit the ability of the interventional radiologist access to the patient [16] (Figs. 5.3 and 5.4).

Last Image Hold

The last image hold option should be utilized routinely to document and assist with procedural planning rather than acquisition of additional spot fluoroscopic images or performance of digital subtraction angiograms [17]. As an example, prior to stent deployment, a hand contrast injection through the access sheath can be performed to confirm appropriate positioning of the stent. The last image hold option permits the operator the ability to select the appropriate fluoroscopic image, transfer this image to a second monitor, and utilize the image to assist with accurate stent deployment.

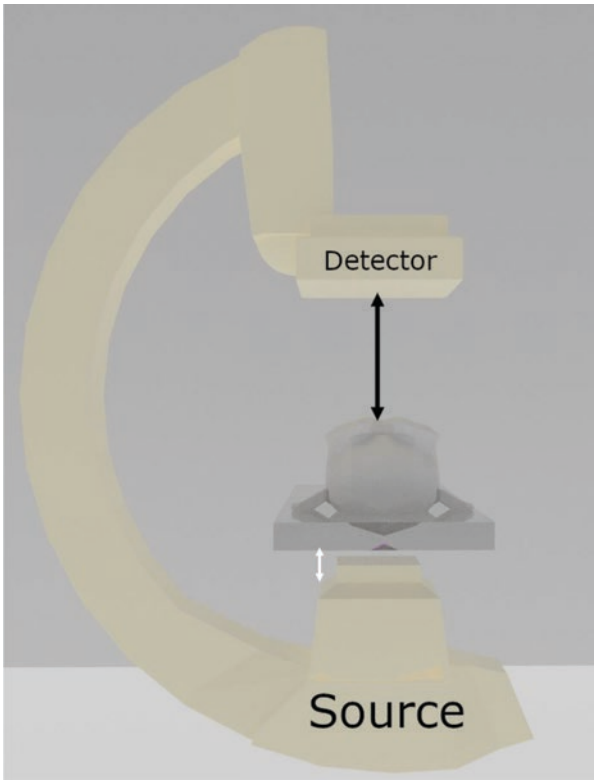

Fig. 5.3 Flat panel fluoroscopic unit. Image detector, radiation source, distance of the radiation source to the patient (*white arrow*), and distance of the patient to the image detector (*black arrow*) are identified

Reduction of Pulse Rate

The number of pulses of radiation delivered per second should be reduced to the lowest rate possible and balanced with acquisition of images of adequate quality. The default pulse rate on fluoroscopy units had been 30 pulses per second for many years [15]. At our institution, the default pulse rate has been established at 4 pulses per second, which has been deemed adequate for acquisition of quality images. However, procedures such as catheter placements and tube exchanges, which do not require complex catheter and wire manipulations can be performed with pulse rates of 2 per second. Reduction of the fluoroscopic pulse rate has been shown to reduce radiation dose [18].

Digitally Subtracted Angiography

Digitally subtracted angiography (DSA) is an image processing technique in which a radiographic digital image of the area of interest is acquired prior to the delivery of contrast material. This image serves as the mask image. The mask image is

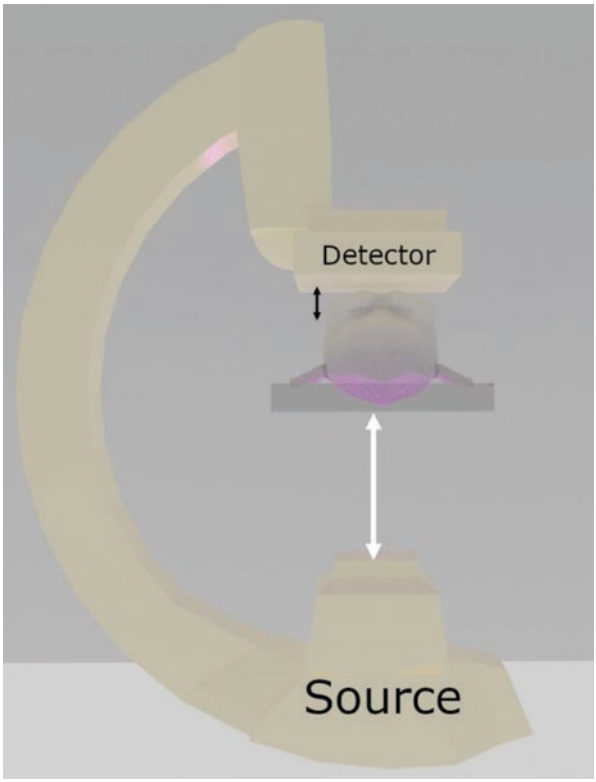

Fig. 5.4 Flat panel fluoroscopic unit illustrating minimization of distance from the image detector to position of the patient on the procedural table (*black arrow*) and maximizing the distance from the radiation source to the patient on the procedural table (*white arrow*)

subsequently subtracted from subsequent digital radiographic images obtained following the arrival of contrast material. The resulting subtracted image is then enhanced through the expansion of the dynamic range [19]. DSA provides high quality images with the trade-off of higher radiation exposure [20]. A review of 764 vascular procedures over a one year period, revealed that 70% of the DAP was secondary to the acquisition of radiographic images. The authors concluded that the DAP could be significantly reduced through the use of fluoroscopic scenes to document findings, when feasible, compared to DSA images [20].

Road Mapping

A road map can be created through contrast material injection into the hemodialysis graft or fistula or through performance of a digital subtraction angiogram. The image following contrast material injection that delineates the outflow vessels can be displayed overlying real-time fluoroscopy images. This permits the interventional radiologist with a vascular map—"road map", to navigate through vessels without additional contrast enhanced images or digital subtraction angiograms, thus minimizing the patient's radiation dose [8].

Documentation of Radiation Exposure

An essential component of an effective radiation safety program within healthcare facilities where fluoroscopy-guided procedures are performed is documentation of patient radiation exposure. Designing an appropriate workflow for the documentation to ensure the uniform and accurate documentation of radiation exposure either through automated or manual means. As stated before, the radiation dose from prior interventions will need to be rapidly retrieved, reviewed, and aggregated as part of the pre-procedural planning phase. Radiation dose monitoring software is available to facilitate the collection and storage of radiation dose information.

Ultrasound

Alternative modalities to radiographic evaluation and treatment may be employed to reduce radiation exposure. Ultrasound-guided dialysis vascular management has been described as a method to guide balloon angioplasty in the treatment of dysfunctional dialysis access [21–23]. In a study of 189 ultrasound-guided balloon angioplasties of dialysis access, 127 (67%) were performed without the use of fluoroscopy. The reason for procedural failures included difficulty in transversing aneurysmal segments and anastomotic stenoses [22]. Ultrasound-guided dialysis access management has also been performed in the office setting without immediate fluoroscopy backup [23].

Image Noise Reduction

The available equipment for procedures also plays an important role in reducing radiation exposure. The perception of image quality is inversely proportional to image noise. Noise reduction algorithms had previously been applied to photographs, particularly those obtained in low light settings. Söderman et al. adapted the concept of noise reduction to radiographic images. The noise reduction algorithm they designed reduced radiation exposure by 75% in digitally subtracted angiograms in neuroradiology without loss of image quality [24].Though this technology has not been specifically studied in the context of dialysis access interventions, this technology has the potential to reduce radiation exposure in multiple vascular beds [25, 26].

Patient Follow-Up

The SIR guidelines recommend follow-up clinic visits for patients who have received a significant radiation dose. A significant radiation dose can be implied when conditions arise whereby the operator is alerted per SIR/CIRSE guidelines. This includes attaining a peak skin dose of greater than 3000 mGy, a reference point

air kerma of greater than 5000 mGy, a kerma-area-product greater than 500 Gy·cm^2 or when the exposure time has exceeded 60 min [11, 15]. A follow-up visit can be set approximately 2 weeks from the date of the procedure to correspond to the time when transient erythema and epilation will manifest [15].

Radiation Exposure to the Interventionist

Interventionists are also at risk from the cumulative effects of ionizing radiation over a career of performing fluoroscopy-guided interventions (Fig. 5.5). The standard radiation shielding apparel should apply in dialysis interventions including lead apron, thyroid shield, leaded glasses with lateral protection, as well as radiation shields which may be floor, table, or ceiling mounted. Strategies to minimize the use of radiation may also be employed, such as the use of ultrasound described above to both evaluate dialysis access and guide balloon angioplasty.

Manual-injection DSA is often performed in dialysis access management. In a review of procedures performed with manual-injection DSA, Hayashi et al. found that greater than 90% of operator exposure was related to manual injection. Based on these findings it was recommended to either use a power injector to avoid radiation exposure or maintain a position as far away from the patient as possible while performing manual injection DSA [27].

The anatomic nature of dialysis access procedures predisposes hands to higher levels of radiation. A retrospective study by Stavas et al. found that radiation exposure to the hands was relatively high during restoration of flow in clotted dialysis access grafts [28]. Radiation exposure to both the right and left hands was tracked through the use of thermoluminescent ring dosimeters on each hand of five interventional radiologists over a total of 62 synthetic graft declot procedures. The mean right hand exposure was found to be 0.78 mSv, and the mean left hand exposure was 0.55 mSv. No patient-related factors such as position of the graft, age, sex, previous thrombosis or number of previous interventions were found to be significant factors in hand dose. On the other hand, technical factors such as fluoroscopic time and the number of angiographic runs were significant factors in total hand dose. In comparison, a multicenter study of radiation exposure found the median exposure of one hand per procedure to be 0.075 mSv over a wide variety of procedure types [29]. Similarly, a prospective single institution study found the average hand dose to be 0.0996 mSv over a variety of endovascular procedures including coronary angiography, pelvic angiography, and lower and upper extremity angiography [30].

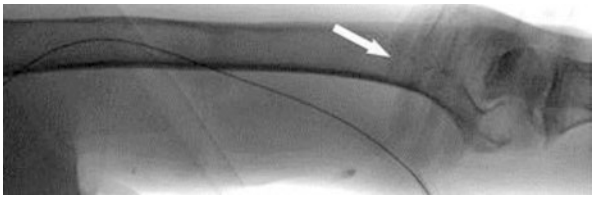

Fig. 5.5 Fluoroscopic image taken from a fistulogram with interventionist's hand (*arrow*) in the field of view

The recommended annual occupational limits to the hand are 500 mSv by both (IRCP) and the National Council on Radiation Protection and Measurements (NCRP) [30]. Although it would take greater than 600 declot procedures to exceed the recommended exposure limits of 500 mSv, it is important to recognize the increased exposure during declot procedures and develop strategies to minimize exposure. Several strategies have been explored in addition to reducing fluoroscopic time and the number of angiographic runs. These strategies include the use of leaded shields, leaded gloves, and radioprotective drapes. The use of a disposable radioprotective bismuth drape demonstrated a marked reduction of hand exposure by 29-fold [31]. A relatively new development is the introduction of an x-ray attenuating lotion which contains bismuth oxide (Bi_2O_3) ceramic powder (UltraBlox by Bloxr, Salt Lake City, UT) and can be applied to the hands [32].

Dialysis access thrombectomy tends to be the procedure associated with the greatest radiation dose both to the patient as well as the interventionist. One additional technique to reduce both the procedure time and radiation exposure in thrombectomy is the use of tissue plasminogen activator (tPA). One study compared the use of mechanical thrombectomy versus mechanical plus "no-wait lysis" on the procedure time and radiation exposure. The no tPA group had an average procedure time of 55.5 min and the "no-wait lysis" group had a procedure time of 27.2 min and fluoroscopy times were reduced to 159 seconds in the "no-wait lysis" group from 243 seconds in the no tPA group [33].

Conclusion

Given the potential for serious patient injuries and long-term ill effects resulting from cumulative radiation exposure, meticulous pre-procedural planning should be undertaken and techniques for radiation reduction must be optimized. The ALARA principle is the guiding principle for all proceduralists utilizing fluoroscopy. Although much attention was been made toward patient radiation dose reduction, it should be mentioned that optimizing patient dose management translates into optimal operator dose management and provision of high quality patient care.

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Conscious Sedation and Anesthesia Care

6

Lee-Wei Kao, Chieh Suai Tan, and Jason Qu

Endovascular interventions for arteriovenous fistulas (AVF) and grafts (AVG) can cause significant pain and discomfort to patients. Adequate sedation and analgesia are often required, hence it is important for the interventionist to understand and appreciate some of the basic principles of sedation and anesthesia. In particular, many patients with ESRD have co-morbidities that may put them at risk of cardiac events during sedation and anesthesia. As such, proper assessment of the patient and putting in place a robust crisis management plan are critical in the smooth operation of the intervention suite.

Pre-procedural Evaluation

The objectives of the pre-procedural evaluation include establishing a doctor-patient relationship, reviewing the patient's overall health condition with an emphasis on their cardiopulmonary function, and identifying risk factors for sedation. The following classification system proposed by The American Society of Anesthesiologists

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(ASA) is a widely used guide for describing a patient's baseline functional capacity [1]:

ASA Physical Status 1: A normal healthy patient
ASA Physical Status 2: A patient with mild systemic disease that does not cause substantive functional limitations
ASA Physical Status 3: A patient with severe systemic disease that causes substantive functional limitations
ASA Physical Status 4: A patient with severe systemic disease that is a constant threat to life
ASA Physical Status 5: A moribund patient who is not expected to survive without the operation

Patients with ASA physical statuses 3 and 4, especially with a history of sleep apnea, poor heart function, COPD, difficult airway, and complications from previous sedation and anesthesia should have an anesthesia consult and be evaluated prior to monitored anesthesia care(MAC) or general anesthesia. The modified Mallampati classification is widely used by the anesthesiologist to predict the difficulty of an airway (Fig. 6.1). The assessment is made with the patient sitting upright, with the head in the neutral position, the mouth open as wide as possible, and the tongue protruded maximally without phonation.

Class I. Faucial pillars, soft palate, and uvula are visible.
Class II. Faucial pillars and soft palate may be seen, but the uvula is masked by the base of the tongue.
Class III. Only soft palate is visible. Intubation is predicted to be difficult.
Class IV. Soft palate is not visible. Intubation is predicted to be very difficult.

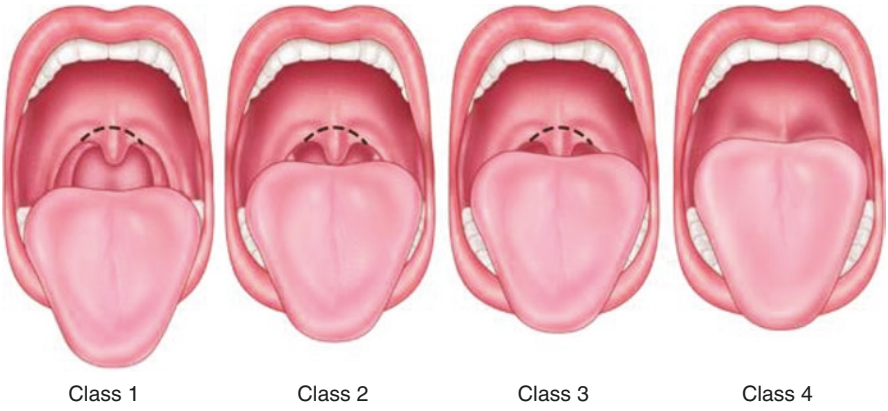

Fig. 6.1 Modified Mallampati airway classification

Patients should be instructed to abstain from solid food for 6 h and clear fluids for 2 h prior to the procedure. Most routine drugs should be taken on the day of the procedure with a minimal amount of water. Exceptions include diabetic medications and ACE inhibitors. Guidelines for management of anticoagulants are institution-dependent, and an anesthesiologist as well as the prescribing physician should be consulted for any patient taking an anticoagulant for whom a peripheral nerve block is being considered. A procedure and sedation consent form should be obtained. Patients should be advised not to drive for at least 24 h after receiving sedation.

Safety in Sedation

The procedure room should be large enough to accommodate a portable resuscitation cart and personnel in the event of resuscitation. The physician and the nurse involved in the procedure should be trained in the monitoring of patients under sedation and be certified in advanced cardiac life support (ACLS). A qualified RN in sedation or anesthetist should be present the entire time and should have training in basic environmental safety, prevention of cross-infection, and crisis management skills such as basic and advanced cardiac life support [2].

Monitoring, Airway Devices and Anesthesia Machine

Monitoring should be consistent with ASA standards, irrespective of the depth of sedation or anesthesia [3] (Fig. 6.2). The monitoring devices must give an audible signal when their alarm thresholds are exceeded. Hemodynamic monitoring includes ECG and noninvasive blood pressure (NIBP) measurements at a minimum interval of 5 min. Oxygenation is monitored with pulse oximetry. Continuous monitoring of End-tidal carbon dioxide ($EtCO_2$) should be used during any procedure requiring moderate or deep sedation. Standard monitoring for general anesthesia involves oxygenation (oxygen analyzer and pulse oximetry), ventilation (capnography and minute ventilation), circulation (ECG, blood pressure, and perfusion assessment), and temperature.

When a nasal cannula or facial mask cannot maintain adequate oxygen saturation, advanced airway management is warranted (Fig. 6.3). For mask ventilation, the mask can be connected to an Ambu bag or anesthesia circuit to provide positive pressure ventilation. An oral airway is inserted into the mouth to eliminate obstruction from the tongue while a nasopharyngeal airway is inserted through a nostril to bypass the airway obstruction. A laryngeal mask airway is inserted orally to align its opening with the vocal cords. Endotracheal intubation is the ultimate way of controlling ventilation and securing the airway. The endotracheal tube is inserted into the trachea through the vocal cords under direct vision or using a video-imaging device.

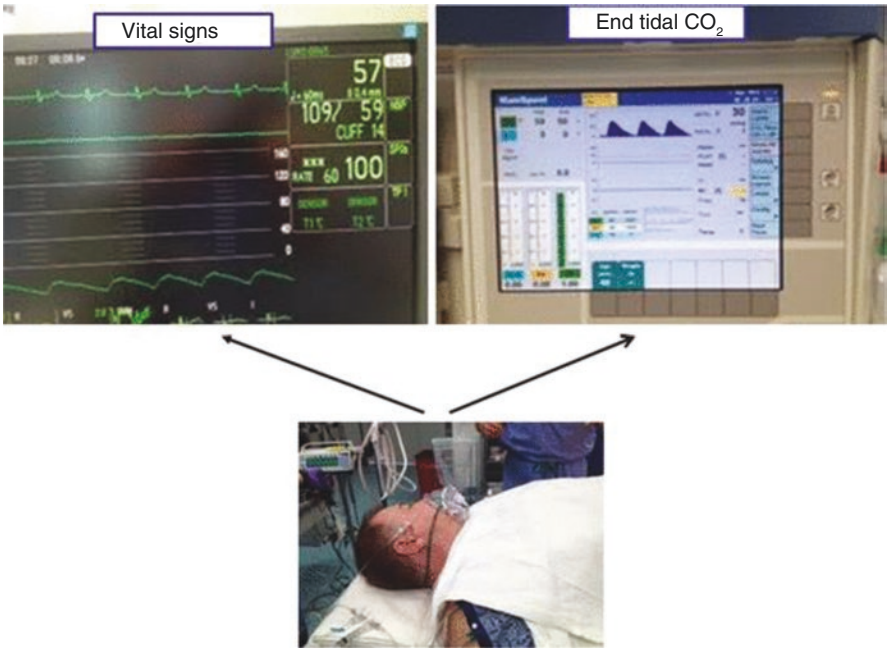

Fig. 6.2 ASA standard monitoring

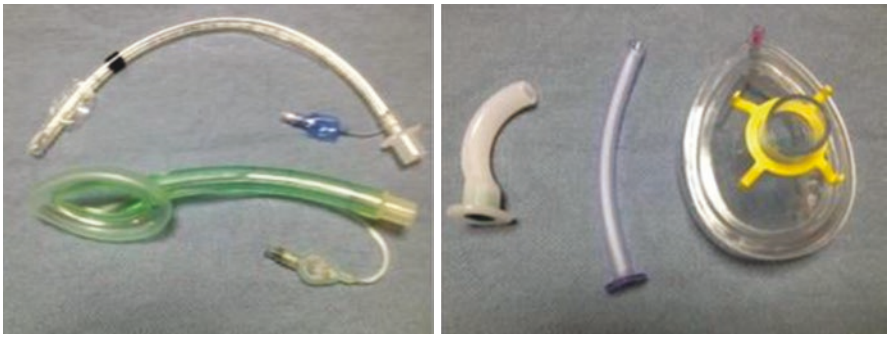

Fig. 6.3 Airway devices: endotracheal tube, laryngeal mask airway; oral airway, nasal airway and mask

Sedation Levels and General Anesthesia [4]

Moderate Sedation

"Moderate Sedation/Analgesia" ("Conscious Sedation") is a drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. No interventions

are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.

Deep Sedation

"Deep Sedation/Analgesia is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained."

General Anesthesia

"General Anesthesia is a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patients often require assistance in maintaining a patent airway and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired."

Monitored Anesthesia Care

Monitored Anesthesia Care (MAC) is a specific anesthesia service for a diagnostic or therapeutic procedure. MAC may include varying levels of sedation, analgesia, and anxiolysis as necessary. The provider of monitored anesthesia care must be prepared and qualified to convert to general anesthesia when necessary [5].

Local Anesthetics and Intravenous Sedation

Local anesthetics block nerve conduction by impairing propagation of the action potential in axons. There are two categories of local anesthetics by their chemical structures. The commonly used Amides include lidocaine, bupivacaine, mepivacaine, and ropivacaine; Esters include procaine, chloroprocaine, and tetracaine. The choice of local anesthetics must take into account the rate of onset, duration, and the potential for local or systemic toxicity [6].

Adding epinephrine to local anesthetics can prolong the duration of action, decrease systemic toxicity, assist in the detection of intravascular injection, provide local vasoconstriction, and decrease surgical bleeding. The typical concentration of epinephrine used in local anesthetic solutions is 1:200,000, or 5 μg/ml. Raising the pH of the local anesthetic solution by adding sodium bicarbonate will speed up the

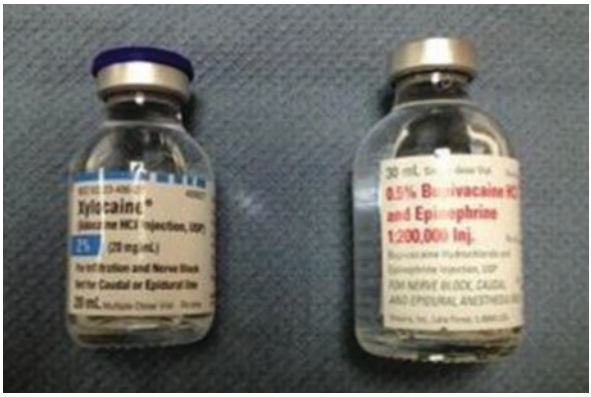

Fig. 6.4 Local anesthetics

onset of action and increase the rate of diffusion. The commonly used local anesthetics are 0.5–5% lidocaine with 1% solution being most frequently used, and 0.25–0.75% bupivacaine with 0.25% being most frequently used (Fig. 6.4).

Regional Anesthesia

Peripheral nerve blocks can be used as the primary anesthetic or as an adjunct for AVF procedures. Single-shot blocks can provide sensory and motor blockade for up to 24 h. Regional anesthesia can significantly decrease the amount of sedation required for the procedure which in turn decreases the risk of adverse events such as respiratory depression and hypotension. Absolute contraindications for regional anesthesia include lack of patient consent and skin infection at the site of needle insertion. Coagulopathy and pre-existing neuropathy are considered relative contraindications [7]. Risks of regional anesthesia that should be discussed with the patient include infection, bleeding, damage to surrounding structures, local anesthetic toxicity, and ineffective or incomplete nerve blockade.

Premedication with anxiolytics may be needed to ensure patient comfort for peripheral nerve blocks. A commonly used regimen is 1 mg of midazolam and 50 mcg of fentanyl, with additional incremental doses titrated to effect. The patient should remain alert enough to inform the provider if they experience paresthesias or warning signs of local anesthetic toxicity. Standard ASA monitors should be used during and after any nerve block, and supplemental oxygen may be needed if anxiolytics are given. The patient should be monitored closely for oversedation or complications related to the nerve block.

Regional anesthesia for upper extremity procedures target the brachial plexus at various points. The most common nerve blocks for procedures distal to the shoulder are the supraclavicular, infraclavicular, and axillary nerve blocks. Of these blocks, the supraclavicular nerve block has the highest risk of phrenic nerve dysfunction and pneumothorax; as such, it may be less desirable in patients with pre-existing pulmonary disease such as severe COPD or lung dysfunction on the contralateral

side. These blocks may need to be supplemented with blockade of the intercostobrachial and medial cutaneous nerves which innervate the medial upper arm. The use of ultrasound guidance can decrease the risk of adverse events such as intravascular injection, nerve injury, and pneumothorax from pleural puncture. Other techniques to prevent complications include aspiration prior to injection of local anesthetic and incremental injections.

When utilized as a primary anesthetic, regional nerve blocks are commonly performed with a mixture of local anesthetics. A longer-acting local anesthetic is used for lasting pain relief but has a slower onset. In order to ensure adequate surgical anesthesia for the procedure, another local anesthetic with a faster onset but shorter duration is used. One example of this is the use of mepivacaine, a quicker but shorter-acting local anesthetic, along with ropivacaine, a slower but longer-acting anesthetic.

The adequacy of sensory and motor blockade should be tested prior to beginning the procedure. Sensory blockade can be tested to pinprick and/or cold sensation. If there is inadequate blockade in any nerve distribution, the nerve block may be supplemented with an additional block. However, care should be taken not to exceed the maximal recommended dose in order to reduce the risk of local anesthetic toxicity (Table 6.1).

Intravenous Agents

A large number of sedatives, opioids, and adjunctive agents can be used for AVF procedural sedation. The general pharmacology and characteristics for each drug class should be thoroughly understood. The ideal sedation regimen in AVF procedures should provide adequate coverage for anxiety, sedation, and pain.

Table 6.1 Local anesthetics

Anesthetics	Onset	Concentration (%)	Duration (hours)	Toxicity	Usual dose (ml) (70 kg patient)	Maximal dose (mg)
Amides						
Lidocaine (Xylocaine)	Rapid	0.5–1.0	0.5–2.0	Moderate	1–50	300 (500)
Bupivacaine (Marcaine, Sensorcaine)	Slow	0.25–0.5	2–4	High	1–45	175 (225)
Mepivacaine (Carbocaine)	Moderate	0.5–1.0	0.25–2.0	Moderate	1–50	300 (500)
Esters						
Procaine (Novocaine)	Rapid	0.5–1.0	0.25–0.5	Low	1–60	400 (600)
Chloroprocaine (Nesacaine)	Very rapid	2–3	0.25–0.5	Very low	1–100	800 (1000)

Dose in epinephrine-containing solution in parenthesis. Adding epinephrine prolongs the analgesia duration of lidocaine, mepivacaine, procaine and chloroprocaine but not bupivacaine or ropivocaine

Benzodiazepines are the conventional drugs of choice for any kind of sedation. The sedative, anxiolytic, and amnestic effects of benzodiazepines are attributed to their ability to potentiate the inhibitory influences of GABA—the principal inhibitory neurotransmitter in the brain [8]. Midazolam has largely replaced the commonly used benzodiazepines such as diazepam and lorazepam because of its shorter distribution and elimination half-life. The advantages of Midazolam include a rapid onset with short duration of action and a greater degree of amnesia. It is water soluble and therefore essentially painless during injection. The onset of intravenous midazolam is 1–3 min with a duration of action of 1 h. It is administered in 0.5–2 mg increments. Intravenous diazepam has an onset time of 2–3 min with duration of action of 6 h. Lorazepam has an even slower onset time and longer duration of action.

Opioids act by binding to endogenous opioid receptors in the central and peripheral nervous system. In addition to producing sedation, the primary effects of opioids are analgesia and inhibition of autonomic reflexes. As they depress catecholamine release and obtund sympathetic reflexes to noxious stimuli, opioids are also "cardioprotective". The sedative effect of opioids is synergistic with that of most sedatives. Fentanyl and meperidine are the most frequently used opioids. Despite its historical popularity in sedation regimens, meperidine has several properties that render it less attractive than fentanyl. Its anticholinergic properties can increase heart rate and depress myocardial contractility. Its active metabolite, normeperidine, is a CNS stimulant with a half-life of 18 h and accumulates in patients with renal insufficiency. Unlike meperidine, fentanyl does not promote histamine release. It acts rapidly with an onset time of 2–3 min and has a redistribution time of 30 min. It is administered in 25–50 ug increments. Fentanyl has a greater potential to produce skeletal muscle rigidity and apnea. It can accumulate following repeated doses, and the respiratory depressant effect can last much longer than its analgesic effects. Morphine is rarely used in AVF procedures because of its long-acting properties and its active metabolite, morphine-6-glucuronide, which is cleared renally. Remifentanil is an ultra-short acting opioid that is metabolized by plasma esterases. Because of this, its elimination half-life is not dependent on renal or hepatic function. Remifentanil when used for procedural sedation is generally used as a temporizing measure for brief periods of stimulation such as injection of local anesthetic.

Antihistamines including diphenhydramine and promethazine, are less effective sedatives and anxiolytics than benzodiazepines. They are useful adjuncts in potentiating the sedative effects of benzodiazepines but cannot be used as primary agents for procedural sedation. Antihistamines also have some antiemetic properties which may be useful in longer procedures.

Propofol is typically administered by anesthesiologists or CRNAs (Certified Registered Nurse Anesthetist) for general anesthesia and MAC. It is a hypnotic agent that facilitates inhibitory neurotransmission by enhancing the function of Υ-aminobutyric acid type A ($GABA_A$) receptors in the CNS. It has a rapid onset time of 30–45 s and rapid termination due to redistribution. It is a direct cardiac depressant and causes vasodilation, leading to dose-dependent bradycardia and hypotension.

Dexmedetomidine is an agent that acts by agonism of alpha-2 adrenergic receptors. It causes a state of "cooperative" sedation during which the patient is arousable and able to follow commands. One key advantage of dexmedetomidine is that it is able to produce sedation without causing respiratory depression. This is particularly useful in patients for whom respiratory compromise is a concern such as patients with obstructive sleep apnea. However, it can still cause airway obstruction at higher doses and has synergistic effects with other sedatives. Dexmedetomidine is also useful as an anesthetic adjunct in opioid-tolerant patients due to its opioid-sparing effects. Side effects include bradycardia and hypotension that are more pronounced when administered as a bolus. It has a long elimination half-life of 2 hours which may make it less desirable than other shorter-acting agents.

Sedation and Analgesia Related Complications and Treatment

Sedation for AVF patients can be challenging because most of the patients have concomitant pulmonary and or cardiac illnesses. Titrating medications with small doses and early recognization of cardiopulmonary compromise are the keys to avoiding devastating complications.

Local Anesthetics

True allergic reactions to local anesthetics are uncommon. It is important to differentiate them from common nonallergic responses such as vasovagal episodes and responses to intravascular injection of local anesthetics and/or epinephrine. Systemic toxicity usually results from intravascular injection or overdose. Aspiration before injection, use of epinephrine-containing solutions, and small incremental volumes are techniques that will minimize intravascular injection. Clinical features of central nervous system toxicity include complaints of a metallic taste, numbness of the tongue and lips, light-headedness, tinnitus and visual disturbances. These may progress to muscle twitching, loss of consciousness, tonic-colonic seizures, and even coma.

Cardiovascular toxicity is rare but can be severe and difficult to treat. It can present as decreased ventricular contractility, refractory cardiac arrhythmias, and loss of peripheral vasomotor tone. Intravascular injection of bupivacaine may cause cardiovascular collapse, which is often refractory to therapy because of its high affinity for sodium channels. Ropivacaine is similar to bupivacaine in potency and duration of action but has less cardiac toxicity. At the first sign of toxicity, injection of local anesthetic should be discontinued, and oxygen should be administered. If seizure activity is prolonged or interferes with ventilation, anticonvulsant treatment is indicated. Midazolam (1–2 mg), lorazepam (2–4 mg), or thiopental (50–200 mg) may be given. Endotracheal intubation should be performed to provide adequate oxygenation. Local anesthetic-induced cardiac arrhythmias and collapse are difficult to treat. The treatment includes intravenous amiodarone, electric cardioversion, 20%

intralipid infusion, and potentially cardiopulmonary bypass. Intralipid is the definitive treatment for local anesthetic systemic toxicity, and its administration should not be delayed. Cardiac depressants such as propofol, calcium channel blockers, and beta blockers should be avoided.

Intravenous Agents

Hypoxia or desaturation is the primary complication of sedation and analgesia. Continuous oxygen supply via nasal cannula or face mask should be used. The oxygen supply should be checked immediately when the pulse oximetry reading starts to drop. A backup oxygen supply such as an oxygen cylinder should be used if there is any suspicion that the wall source has failed. If the patient does not respond to verbal stimulation or sternal rub, jaw thrust should be applied to stimulate the patient and open the airway which may be obstructed. An oral or nasal airway can also help in this situation. Positive pressure ventilation with an Ambu bag may be indicated if these measures are ineffective. The anesthesia team should be called for advanced airway support if all the above efforts fail.

Hypotension and hypertension are also common concerns during sedation. While hypertension may be related to agitation and pain, it may also be caused by hypoxia or hypercarbia. Hypotension is most likely related to cardiovascular depression. Common intravenous agents to treat intraprocedural hypotension include phenylephrine and ephedrine. Inotropes such as norepinephrine may be needed in patients with severe cardiovascular disease.

Inadequate sedation may be secondary to anxiety or pain. If necessary, additional benzodiazepines may be given to relieve anxiety. Additional local anesthetics or opioids can be given for pain relief if required. Injection of local anesthetic at the procedural site can be very painful and stimulating for the patient; if additional medications are needed for this brief period, very short-acting medications such as remifentanil and propofol should be used as temporizing measures. If longer-acting agents such as midazolam and fentanyl are used, there is a risk of oversedation after the stimulus is either no longer present or blunted by local anesthetics.

Reversal Agents or Antagonists

If the patient does not respond to repeated verbal or painful stimulation after the procedure is completed or if the patient remains hypotensive or in respiratory depression, benzodiazepine and/or opioid overdose should be considered. Pharmacologic antagonists such as naloxone for opioids and flumazenil for benzodiazepines can reverse the above effects. These antagonists must be used with caution, with careful titration to effect. Flumazenil can precipitate acute benzodiazepine withdrawal which may lead to seizures. The recommended dose for flumazenil is 0.2 mg/min. Naloxone can be titrated with 0.04 mg every 2–3 min as needed.

Postprocedure Recovery

Patients should be continually monitored in the recovery room as in the procedure room. Discharge criteria should follow the recommendations of the ASA [9]. Patients should have returned to their baseline level of consciousness with stable vital signs that are within acceptable limits. If a reversal agent was required at the end of the procedure, the patient should be monitored for an adequate amount of time to ensure that re-sedation does not occur after the reversal agent wears off. The patient should receive specific written instructions including management of pain, relevant postprocedural complications, and routine and emergency follow-up. If a peripheral nerve block was performed, follow-up evaluation should include adequacy and duration of the block, presence of residual sensory or motor blockade, and paresthesias or other side effects. Patients should not drive for at least 24 h, and a chaperone is often recommended.

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Vascular Anatomy for Hemodialysis Access

7

Shahbaj Ahmad, Chieh Suai Tan, Steven Wu, and Gerald A. Beathard

Introduction

Good vascular access is of utmost importance to provide optimum dialysis in patients with End Stage Renal Disease (ESRD). Therefore, the creation of a good vascular access requires proper planning and strategies. These include preserving venous vasculature during the pre-ESRD care of the patient, construction of the AVF before the anticipated need and maintenance of the access by providing interventions to prevent malfunctioning of the vascular accesses. A good knowledge of the normal vascular anatomy and its variant in the upper limb, thorax and thigh is therefore crucial in the management of dialysis vascular access.

The dialysis circuit can be considered as a closed-loop system that begins and ends at the heart. Any obstruction or stenosis within this circuit can lead to access failure.

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Arterial Supply of the Upper Limb (Fig. 7.1a)

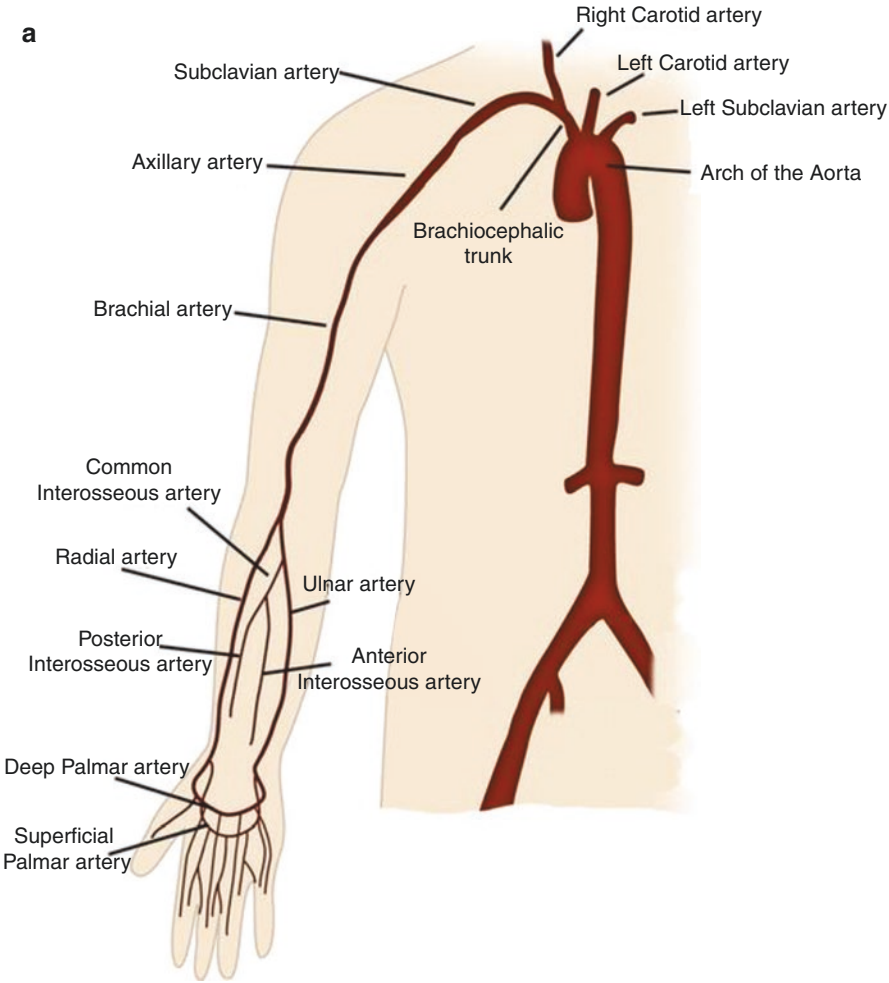

Fig. 7.1 (**a**) The main vessels of the arterial system of the upper limb and trunk are illustrated here. All interventionists should be familiar with the nomenclatures of the vessels. (**b**) The three main branches from the aortic arch are the brachiocephalic, left carotid, and left subclavian artery. (**c**) The left subclavian artery becomes the axillary artery after crossing the first rib. The axillary artery continues as the brachial artery after crossing the lower border of the teres major. (**d**) The brachial artery runs medial to the humerus initially but it gradually turns to the front of the bone as it runs down the arm. Note the presence of a stenosis in the axillary artery. (**e**) The brachial artery ends approximately 1 cm below the level of the elbow, where it divides into the radial and ulnar arteries. (**f**) Arterial anatomy of the forearm and hand. Note the presence of a radiocephalic fistula and the presence of a juxta-anastomotic stenosis

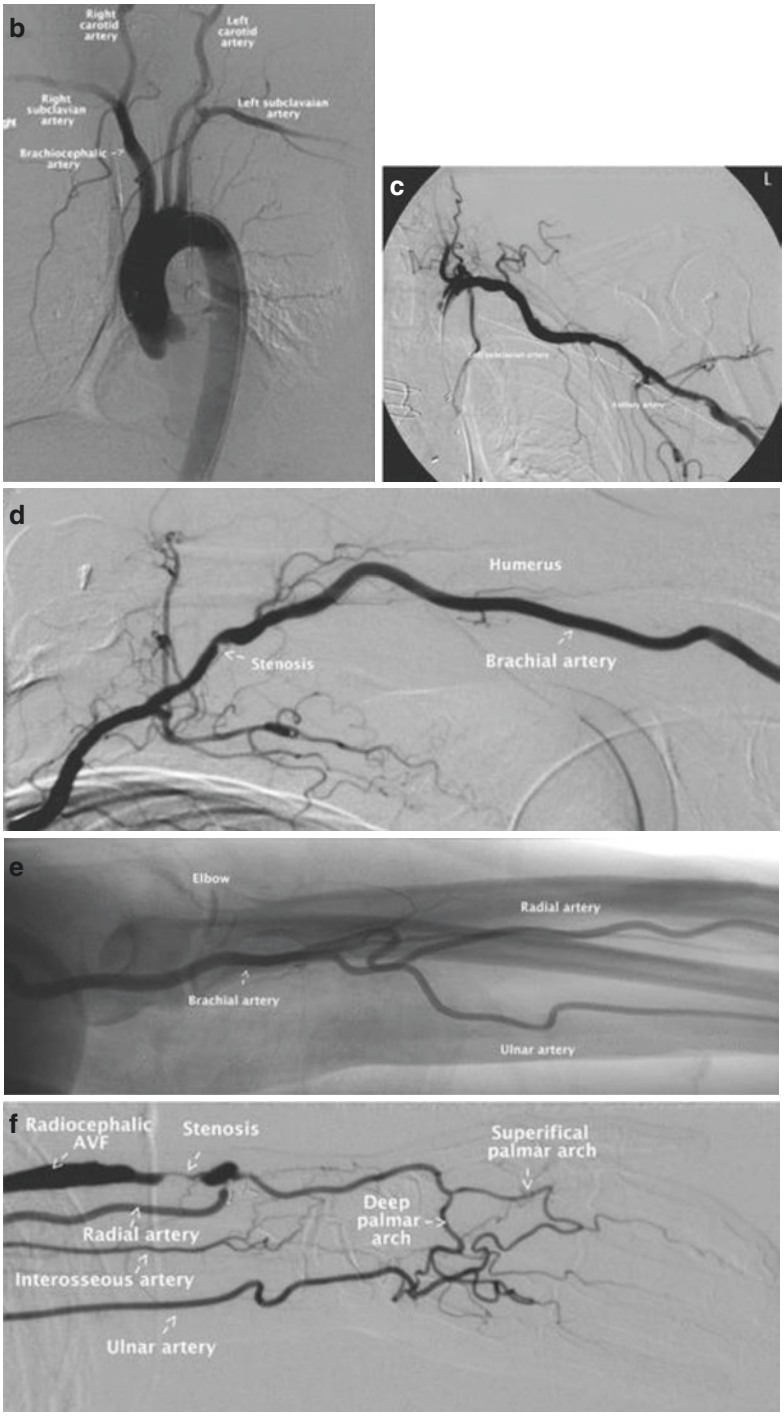

Fig. 7.1 (continued)

The aortic arch has three main branches: brachiocephalic artery, left common carotid artery and the left subclavian artery. In turn, the brachiocephalic artery gives off two main branches, namely the right subclavian artery and the right common carotid artery (Fig. 7.1b).

The subclavian artery continues to become the axillary artery after crossing the border of the first rib (Fig. 7.1c). The axillary artery passes through the axillary fossa and becomes the brachial artery after crossing the lower border of the teres major muscle, which cannot be seen radiologically (Fig. 7.1d).

The brachial artery continues down the arm and bifurcates into the ulnar and radial artery at the level of the elbow (Fig. 7.1e). The bifurcation may occur proximal to the elbow in some patients. Moreover, an accessory brachial artery may be present in 0.52% of the patients [1]. This accessory branch, when present, is given off at the proximal 1/3 of the brachial artery and rejoins the main brachial artery proximal to the elbow.

The ulnar artery runs on the medial aspect of the forearm and terminates within the palm to form the superficial palmar arch with the superficial palmar branch of the radial artery. The common interosseous artery branches off from the ulnar artery below the radial tuberosity and further divides into the anterior and posterior interosseous artery in the forearm.

The radial artery runs on the lateral aspect of the forearm, passes through the anatomical snuff-box to terminate within the palm by forming the deep palmar arch with the deep branch of the ulnar artery.

Within the hand, digital arteries arise from both deep and superficial arches to supply the fingers (Fig. 7.1f). The superficial palmar arch is more distal than the deep palmar arch and may be "incomplete" (absence of anastomosis between the ulnar and radial branches) in approximately 15% of the population. This may cause a problem during the instrumentation of the radial artery. The Allen test is used to test the patency of the palmar arches clinically.

Venous Drainage of the Upper Limb and Thorax

The venous drainage of the upper limb consists of both superficial and deep veins (Fig. 7.2a). The deep veins accompany the arteries and are connected to the superficial system by perforating veins. Dialysis vascular access is created by anastomosing one of the superficial veins to an artery.

The perforators play an important role in shunting the blood from the deep venous system to the superficial veins in endovascularly created arteriovenous fistula around the elbow [2]. Ligation of the perforator around the elbow may hasten the maturation of a brachial arteriovenous fistula that is created in a side to side anastomosis with the cephalic vein [3].

Considerable variation in the anatomy of the superficial veins exists but three prominent veins: the basilic, cephalic and median antebrachial veins can be identified in most patients (Fig. 7.2b). The smaller and unnamed veins are usually referred to as accessory veins.

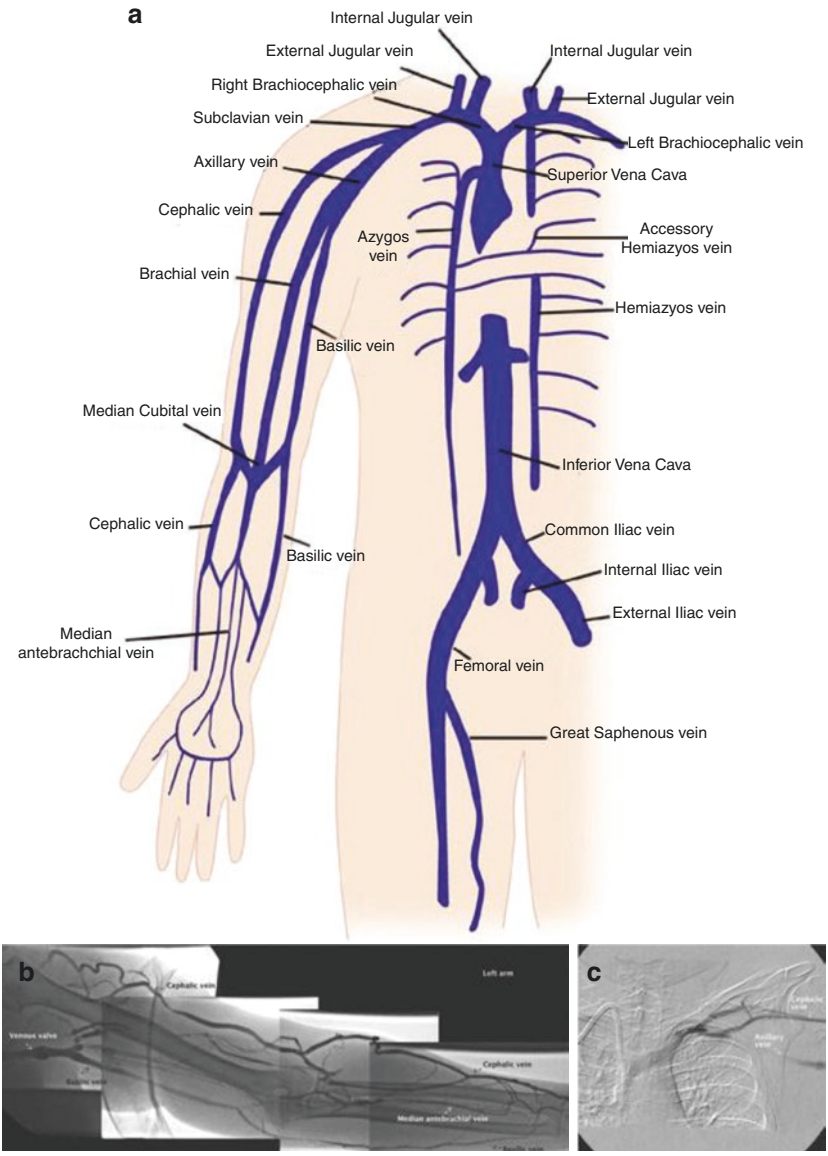

Fig. 7.2 (**a**) Venous drainage of the upper limb and body. (**b**) Considerable variations of the superficial veins exist but in general, these are the main vessels that can be used for AV access creation. (**c**) A double cephalic arch is seen here. It is a normal variant. (**d**) Anatomy of the cephalic arch. (**e**) The basilic and brachial veins join at the inferior border of the teres major, which is not visible radiologically, to form the axillary vein. The axillary vein becomes the subclavian vein after the cephalic arch. The subclavian vein becomes the brachiocephalic vein (on the right) or the innominate vein (on the left) after joining with the internal jugular vein. The brachiocephalic vein and innominate vein join to form the superior vena cava. (**f**) The catheter is lying within a left-sided superior vena cava. (**g**) The azygos veins are usually not visible unless there is an obstruction of the vena cave

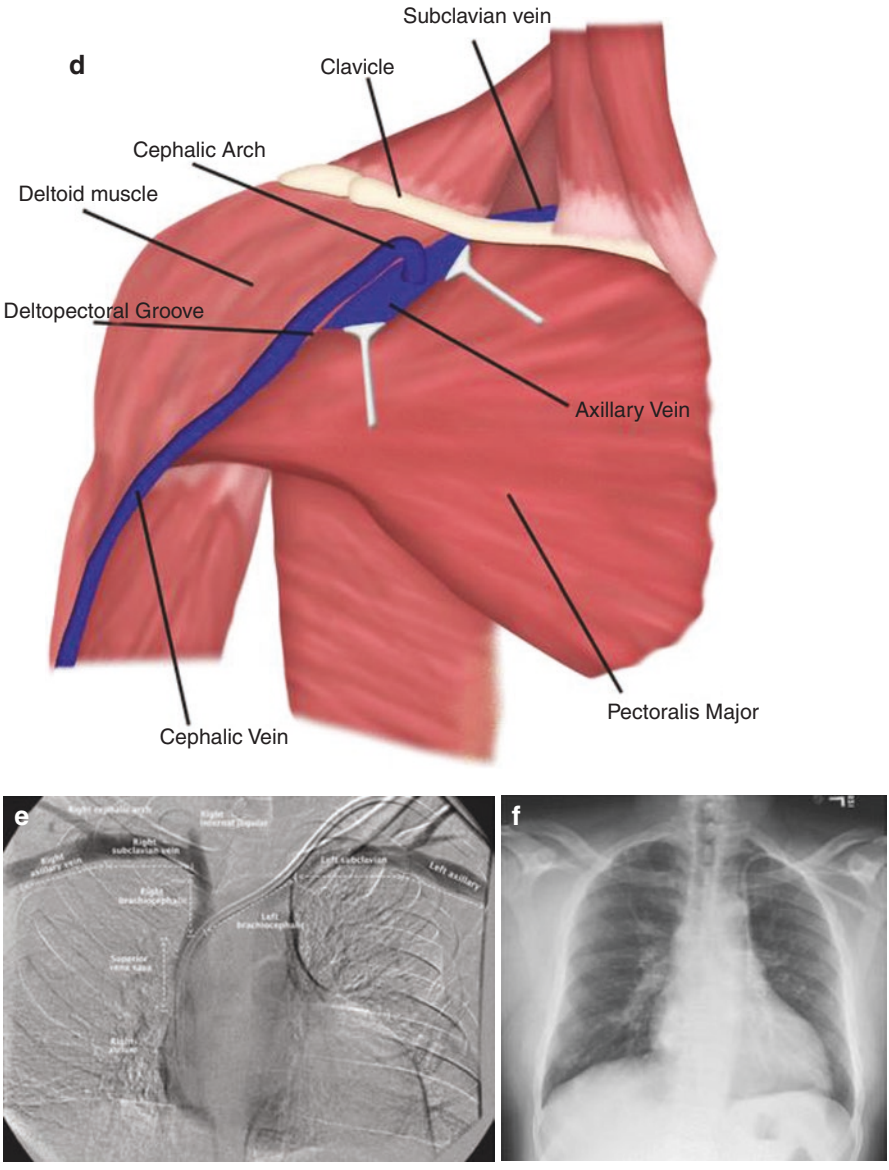

Fig. 7.2 (continued)

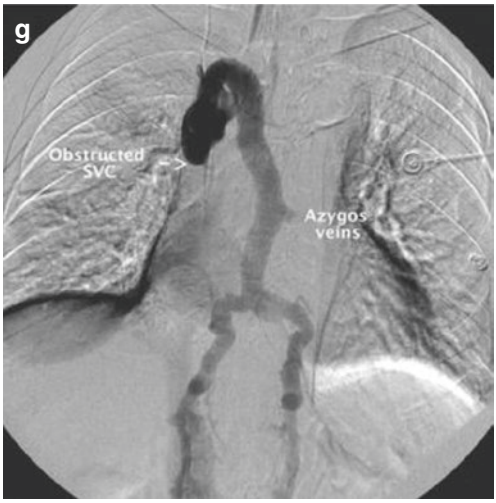

Fig. 7.2 (continued)

The basilic vein begins medially at the dorsal aspect of the hand and lies on the medial or posterior-medial aspect of the forearm. It crosses the elbow anteriorly and ascends obliquely in the grove between the bicep brachii and pronator teres where it runs across the brachial artery. It then runs along the medial border of the biceps brachii, pierces the brachial fascia to join up with the brachial vein to form the axillary vein. As it is fairly deep in the upper limb, transposition of the vein is often necessary after brachiobasilic arteriovenous fistula creation.

The median antebrachial vein ascends on the ulnar aspect of the forearm and drains into the basilic vein or the median basilic vein.

The cephalic vein begins in the radial part of the dorsal venous network and crosses the anatomical snuff-box to run along the radial border of the forearm. A radiocephalic arteriovenous fistula may be created by anastomosing the cephalic vein to the radial artery in the snuff-box or in the forearm. Also, percutaneous endovascular AVF may be possible in the snuff-box as the radial artery and the cephalic vein are in close proximity [4].

Just below the anterior aspect of the elbow, the cephalic vein gives off the median antecubital vein to join the basilic vein. Around the elbow, the cephalic vein or its branch may be anastomosed with the proximal radial artery or distal brachial artery to create a proximal radial artery arteriovenous fistula or brachiocephalic arteriovenous fistula. The proximal radial artery arteriovenous fistula has a lower risk of steal phenomenon when compared to the brachiocephalic fistula [5].

The cephalic vein then crosses the elbow to ascend along the lateral border of the Biceps Brachii. It then passes between the pectoralis major and deltoid muscle to go below the clavicle where it turns sharply to pierce the clavipectoral fascia to drain into the axillary vein. Various anatomic variants have been described. These include

direct drainage of the cephalic vein into the external and internal jugular veins, subclavian veins or the presence of a double arch (Fig. 7.2c).

The region where the cephalic vein drains into the axillary vein is called the cephalic arch (Fig. 7.2d). It is vulnerable to the development of stenosis, especially in patients with brachiocephalic fistulas. The pathogenesis has been postulated to be secondary to the higher blood flow associated with brachiocephalic fistulas, the presence of valves that restrict flow or a restrictive clavipectoral fascia that impede the dilatation of the cephalic arch [6].

The axillary vein continues as the subclavian vein, which is then joined by the internal jugular vein to form the brachiocephalic vein on the right and innominate vein on the left. Radiologically, the cephalic arch on one end and the internal jugular vein at the other demarcates the subclavian vein.

The brachiocephalic vein together with the innominate vein forms the superior vena cava and drains into the right atrium (Fig. 7.2e). Occasionally, a left-sided superior vena cava may be present and it may drain directly into the right atrium or via the coronary sinus (Fig. 7.2f).

The azygos system consists of the azygos vein on the right and the hemiazygos and accessory hemiazygos veins on the left (Fig. 7.2g). They arise from the posterior aspect of the vena cava to provide an alternate route for blood to drain into the right atrium. They are interconnected to drain the intercostal, subcostal, mediastinal, esophageal and lumbar veins. The arch of the azygos drains the azygos vein into the superior vena cava. The azygos system is usually not visualized unless in the presence of superior or inferior vena cava obstruction.

Arterial Supply and Venous Drainage of the Lower Limbs

The arterial supply of the lower limb is derived from the femoral artery, which is a continuation of the external iliac artery. It is most superficial and palpable at the inguinal region where it lies just behind the midpoint of the inguinal ligament (Fig. 7.3a). The femoral vein is the main draining vein of the lower limb. It is a continuation of the popliteal vein and is most superficial at the inguinal region. It lies medial to the femoral artery and continues as the external iliac vein after passing under the inguinal ligament (Fig. 7.3b).

Placing a loop graft between the femoral artery and femoral vein creates a thigh arteriovenous graft (Fig. 7.3c). The femoral vein can also be used for the placement of a dialysis catheter.

Venous Anatomy of the Neck

The internal and external jugular veins provide venous drainage of the head and neck.

The external jugular vein begins within the substance of the parotid gland and runs down the neck towards the midpoint of the clavicle. It courses across the

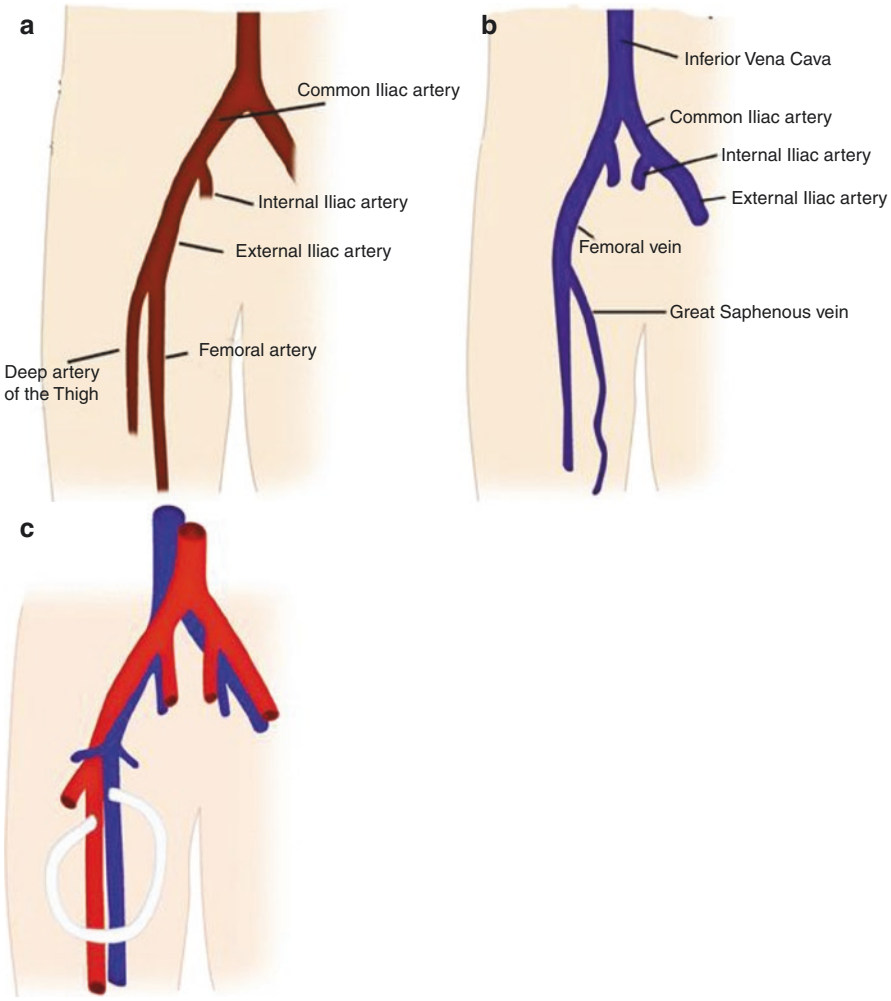

Fig. 7.3 (**a**) The main arterial supply of the lower trunk and thigh region is shown. (**b**) The main venous drainage of the thigh and lower trunk are shown here. (**c**) A thigh arteriovenous graft can be created by placing a loop graft between the femoral artery and the femoral vein

sternocleidomastoid muscle and turns to perforate the deep fascia approximately 4 cm above the clavicle. The external jugular vein then drains into the subclavian vein. The right external jugular vein when dilated may be an alternative to the left internal jugular vein for tunneled dialysis catheter insertion with cumulative patency better than the latter in the patients having unsuitable right internal jugular vein [7].

The internal jugular vein begins at the jugular foramen and runs lateral and parallel to the common carotid artery in a straight-line direction from the mastoid process to the medial side of the clavicular head of the sternocleidomastoid muscle. For this reason, the anatomic triangle formed by the two head of the sternocleidomastoid

muscle and the medial 1/3 of the clavicle is often used as a landmark for blind catheter insertion. However, in a study by Lin et al., the internal jugular vein was found to be anterior to the carotid artery in approximately 20% of the ESRD patients [8]. This highlighted the importance of using ultrasound to facilitate catheter insertion.

The internal jugular vein joins the subclavian vein to form the brachiocephalic vein.

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Hemodialysis Access: Types

8

Shahbaj Ahmad, Chieh Suai Tan, Robert M. Schainfeld, and Steven Wu

Introduction

Hemodialysis is an extracorporeal mode of renal replacement therapy. Therefore, there is a need for a "bridge" to connect the patient to this extracorporeal circuit. Dialysis vascular access acts as a bridge for this purpose. Efficient dialysis requires not only an excellent extracorporeal machine, but also an excellent vascular access. That is why vascular access is often considered the "Achilles Heel" of hemodialysis [1]. Construction and maintenance of vascular accesses contribute a tremendous cost towards the management of ESRD patients. The annual cost borne by US Medicare for three consecutive years from 2011–2013 was about 2.8 billion USD, which accounted for approximately 12% of the total payment for the care of ESRD patients [2].

The ideal vascular access should be easy to construct, immediately usable after construction, and should have the lowest maintenance cost. Sadly, none of the hemodialysis vascular access meets all these criteria. Native arteriovenous fistula comes closest to the ideal vascular access [3–5], although it lacks the quality of being ready for use immediately after construction. Fistula takes a minimum of about 4–6 weeks to

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Table 8.1 Characteristics of different vascular access

	AVF	AVG	Catheter
Lead time between creation and utilization	Usually between 4 and 6 weeks	Depending on the material used; may be used immediately or within 2 weeks	Can be used immediately
Ease of creation	Technically demanding	Technically demanding	Easy
Initial success rate	Low	High	High
Long term patency rate	High	Worse than AVF but better than catheter	Low
Infection rate	Low	Worse than AVF but better than catheter	High
Blood flow rate	High	High	Low
Overall maintenance cost	Low	High	High

mature before it can be used for cannulation for dialysis. So, till the fistula is ready, the patient will have to receive hemodialysis via a temporary access.

Broadly, dialysis vascular access can be classified as temporary vascular access and permanent vascular access (See Table 8.1). AVF & AVG are permanent vascular accesses, and a non-tunneled catheter is a temporary vascular access. The tunneled cuffed catheter may be used either as a temporary or permanent vascular access. The pros and cons of each type of dialysis vascular access are summarized in Table 8.1.

Due to the high complication rates associated with catheters (including but not limited exclusively to infection and malfunction), their routine use should not be encouraged. Compared to AVG, AVF offers superior long-term primary patency and lower infection rates and as such is the dialysis vascular access of choice. As time is required for AVF to mature after creation, patients should be referred to a surgeon for evaluation of an AVF creation by Stage 4 (glomerular filtration rate (GFR) less than 30 mL/min/1.73 m^2) Chronic Kidney Disease. This will help alleviate the need for catheter placement at the time of dialysis initiation and avoid the complications associated with dialysis catheter placement.

Arteriovenous Fistula

An AVF is created by anastomosing a vein to an artery. However, finding optimal vessels for an AVF creation can be challenging, especially in elderly and obese patients. The usual practice of choosing the non-dominant over the dominant arm for access creation is applicable only if both arms have vessels with a similar chance of success. In general, AVFs are created in the most distal vessels first to preserve the vascular "real estate". In order of preference, the radiocephalic AVF is preferred, followed by the brachiocephalic AVF and the transposed brachial basilic vein fistula. Arms ipsilateral to pacemaker wires are usually not used, as there is a high risk of associated central vein stenosis from these wires. If necessary, a diagnostic contrast venogram can be performed to check the patency of the central veins in patients with a pacemaker before ipsilateral AVF creation.

As recommended by the Fistula First Breakthrough Initiative, vessel mapping should be performed in all patients prior to access creation unless suitable vessels

can be readily identified on physical examination. Mapping of the vessels can be performed using Doppler ultrasound or venogram. Doppler ultrasound has the advantage of being a noninvasive test and avoids the risk of contrast-induced nephropathy. On the other hand, a venogram permits direct visualization of the entire venous system of the limb up to the central veins.

Regardless of the modality, for vessels to be suitable for access creation, the inner diameter of the artery and veins should be more than 2.0 and 2.5 mm respectively. Measurement of the diameter of the vein should be made with a tourniquet around the arm. The veins for AVF creation should also have a minimum straight segment length of 8–10 cm for cannulation and should preferably be less than 0.5 cm from the skin for easy cannulation.

Figures 8.1, 8.2, 8.3 and 8.4 show the configuration of three commonly created upper limb AVFs. The characteristics, pros and cons of the four most common AVFs are summarized in Table 8.2.

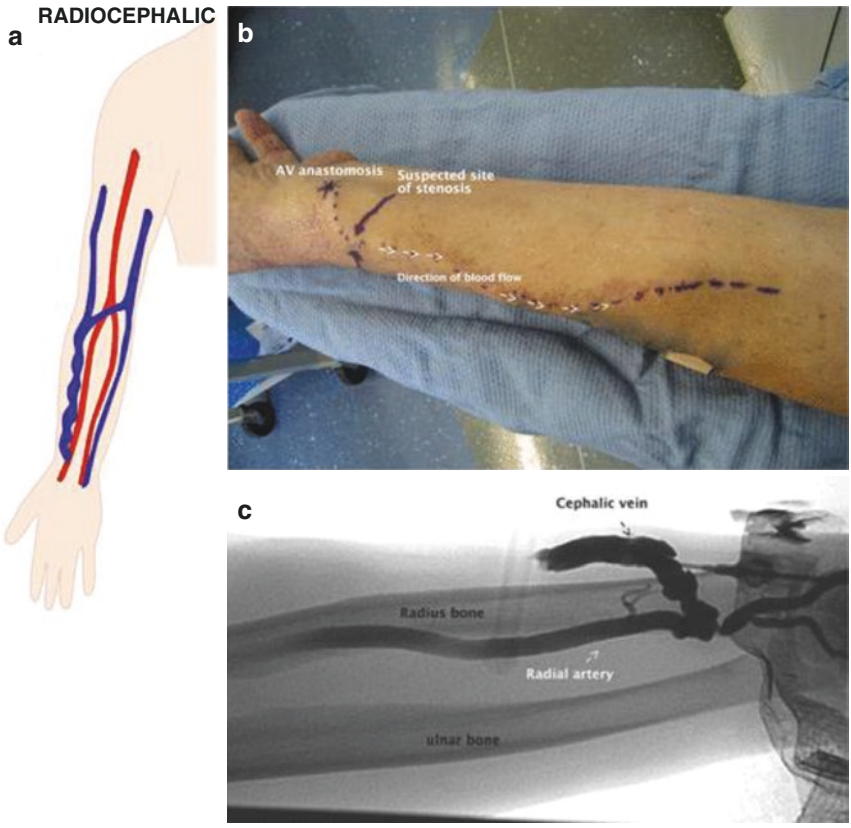

Fig. 8.1 (**a**) Radiocephalic arteriovenous fistula. (**b**) The radiocephalic fistula is created by anastomosing the cephalic vein to the radial artery at the wrist. The arteriovenous anastomosis is near the wrist and the direction of blood flow is as indicated. (**c**) "Reflux" fistulogram was performed by occluding the outflow vein while injecting contrast into the cephalic vein. The radial artery and the juxta-anastomotic segment of the left radiocephalic AVF can be clearly visualized here

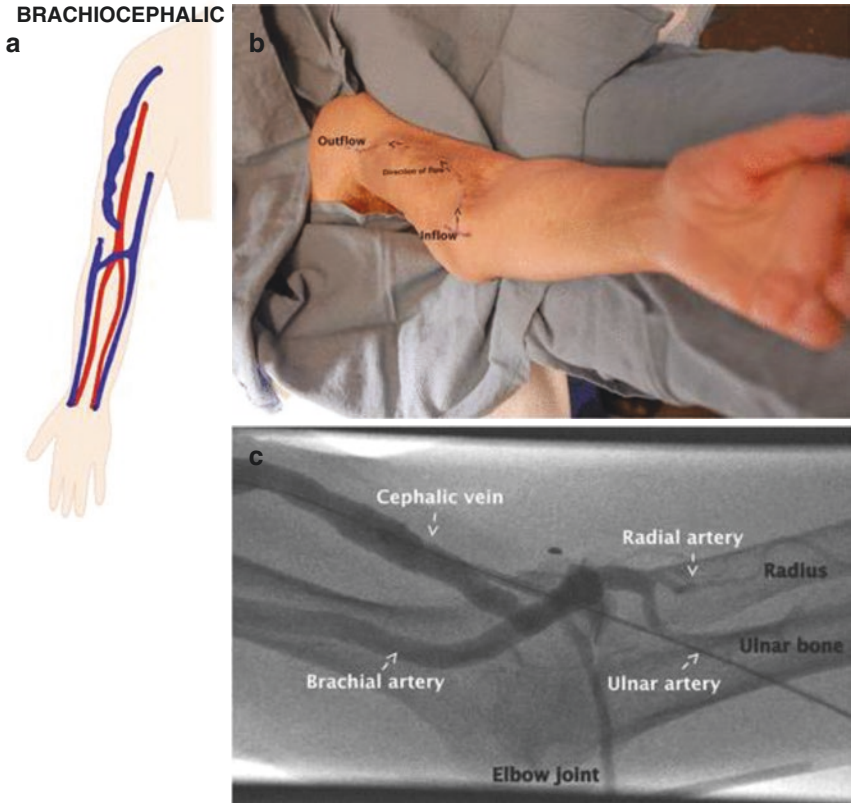

Fig. 8.2 (**a**) Brachiocephalic arteriovenous fistula. (**b**) The brachiocephalic fistula is created by anastomosing the cephalic vein to the brachial artery at the elbow. The cephalic vein runs on the lateral aspect of the arm and is usually superficial enough for easy cannulation. The brachiocephalic arteriovenous fistula can be identified by its location. The surgical scar is at the elbow where the inflow is located. It runs on the lateral aspect of the arm towards the axilla. (**c**) The appearance of a brachiocephalic fistula on fluoroscopy

Proximal radial artery AVF and ulnobasilic fistula are uncommon types of fistulae. Proximal radial artery arteriovenous fistula is created by joining the median antecubital vein or cephalic vein with the radial artery immediately after its origin from the brachial artery at the elbow. It has the advantage of having a lower chance of distal hand ischemia syndrome compared to brachial arteriovenous fistula [10]. Ulnobasilic fistula may be a primary native AVF in the forearm if a radiocephalic AVF is not possible. This is because unlike the cephalic vein, the basilic vein in the forearm is usually patent as it is not commonly used for venepuncture. This is constructed by connecting the ulnar artery with the basilic vein on the medial side of the forearm near the wrist. In some patients, a transposed radiobasilic AVF may be possible if the ulnar artery is of small caliber at the wrist.

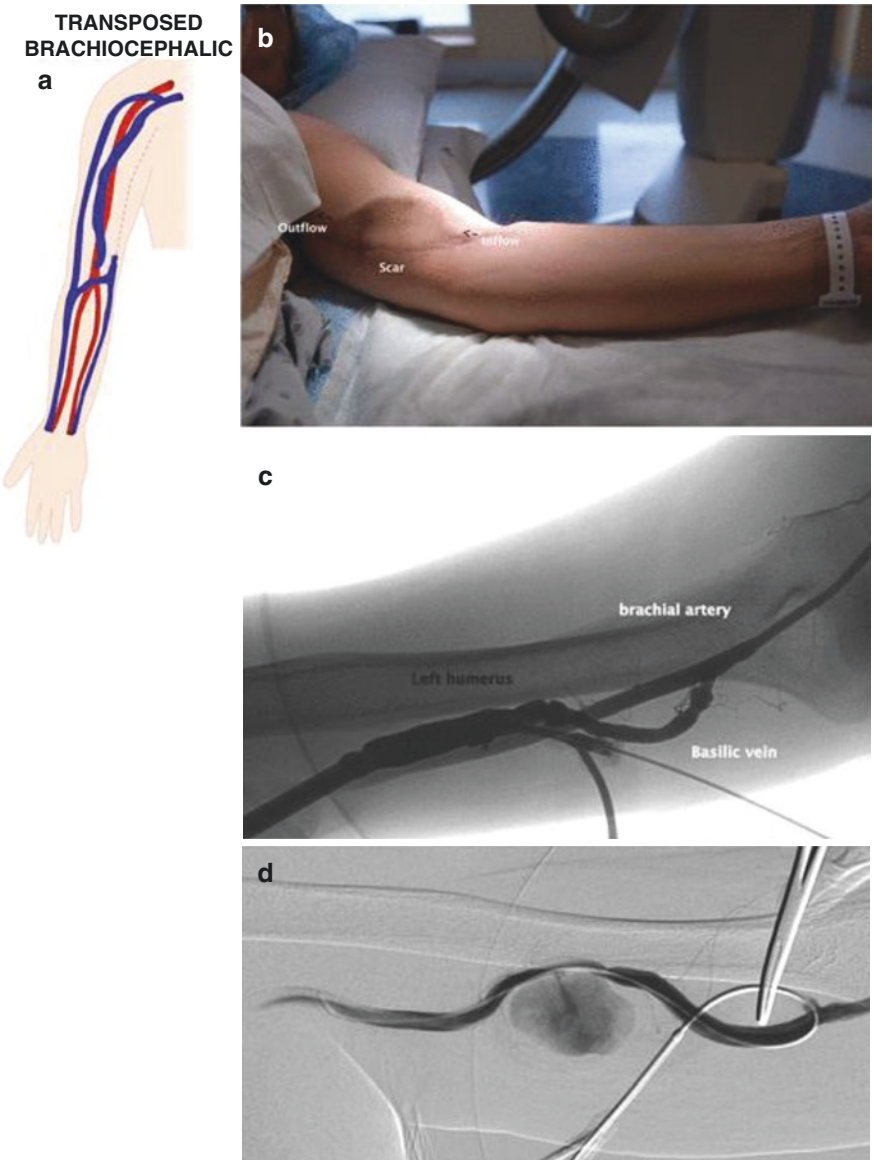

Fig. 8.3 (**a**) Brachiobasilic arteriovenous fistula. (**b**) The brachiobasilic arteriovenous fistula is created by anastomosing the basilic vein to the brachial artery at the elbow. The basilic vein lies on the medial aspect of the arm and is usually too deep and medial to be cannulated for dialysis, thus requiring additional "transposition" surgery to bring it nearer to the skin surface. The BBT AVF can be identified by its location and the presence of a transposition scar which runs on the medial aspect of the arm. The inflow is located near the elbow while the outflow is towards the axilla. (**c**) The basilic vein lies in close proximity to the brachial artery. Occasionally, the brachial artery may be inadvertently injured during attempts to cannulate the basilic vein for dialysis. (**d**) Pseudoaneurysm of the brachial artery as a result of inadvertent injury during placement of dialysis needle in a patient with left BBT AVF

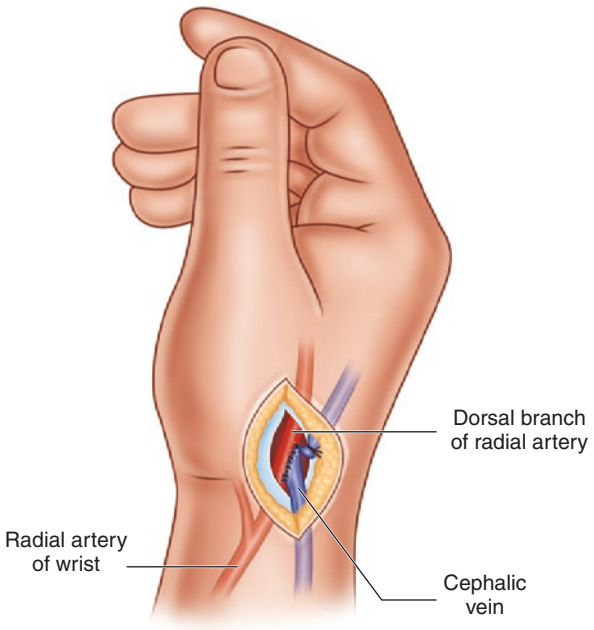

Fig. 8.4 Diagram showing the snuff-box arteriovenous fistula in the right hand. Note that the anastomosis is end to side. However, side to side anastomosis is also possible because of the close proximity of the dorsal branch of the radial artery to the cephalic vein here

Table 8.2 Characteristics of different upper limb AVF [3, 6–9]

Types of AVF	Brescia-Cimino/ radiocephalic (RC)	Brachiocephalic (BC)	Brachiobasilic (BB)	Gracz	Snuff - Box
Vessels used	Cephalic vein to radial artery	Cephalic vein to brachial artery	Basilic vein to brachial artery	Median cubital vein to brachial artery	Cephalic vein to posterior branch of radial artery
Types of anastomosis	End vein to side artery or side to side	End vein to side artery or side to side	End vein to side artery	End vein to side artery	End vein to side artery or side to side
Advantage	Simple anastomosis, preserves upper arm vessels for future use, low risk of upper limb ischemia	Higher blood flow rate than RC AVF	Higher blood flow rate than RC AVF	Simultaneous arterialization of both the cephalic and basilic veins, low risk of upper limb ischemia	Minimal mobilization of the vessels. Simple anastomosis, preserves upper arm vessels for future use, low risk of upper limb ischemia

Table 8.2 (continued)

Types of AVF	Brescia-Cimino/ radiocephalic (RC)	Brachiocephalic (BC)	Brachiobasilic (BB)	Gracz	Snuff - Box
Disadvantage	Lower blood flow rate, high primary failure rates	Increased risk of developing dialysis access steal syndrome and higher incidence of cephalic arch stenosis compared to RC AVF	Technically challenging, increased risk of developing dialysis access steal syndrome, need to transpose basilic vein	Lower flow compared to BC and BB AVF	Lower blood flow rate, high primary failure rates
Patency rate	1 year patency rate: 48–69%	Mean 2 years patency rate: 70–85%	Mean 2 years patency rate: 55–70%	3 years patency rates: 80%	1 year patency rate: 65–76%.
Usual sites of stenosis	Juxta-anastomotic	Cephalic arch stenosis, juxta-anastomotic	"Swing point" stenosis, juxta-anastomotic	Venous outflow, juxta-anastomotic	Data unavailable.

Arteriovenous Graft

Although AVF is the preferred type of vascular access, some patients with small veins that have a low likelihood for AVF maturation may benefit from AVG creation. An AVG is created by joining a vein to the artery using a synthetic (e.g. PTFE) or biosynthetic (e.g. Bovine vein) material. The forearm loop graft and the upper arm curved graft are the two preferred types of access. The forearm loop grafts are used to anastomose either the cephalic, antecubital or basilic veins to the brachial artery. For the upper arm AVG, a curved graft is usually used to join the axillary or basilic vein to the brachial artery. Some examples of AVG are shown in Figs. 8.5 and 8.6.

Dialysis Catheter

Dialysis catheters are needed to provide access for hemodialysis in the absence of a functioning AVF or AVG. They can be further categorized as tunneled or non-tunneled dialysis catheters.

1. Tunneled dialysis catheters are characterized by the presence of a cuff to anchor the catheter. The presence of the cuff and the subcutaneous tunnel help decrease the risk of infection. A tunneled catheter is preferred if prolonged use (>1 week) is anticipated [3] Contraindications for tunneled dialysis catheter insertions are active septicemia, platelet count <50 X 10^9/L and/or INR >1.5 [11] The preferred site for tunneled dialysis catheter insertion is the right internal jugular vein [3].

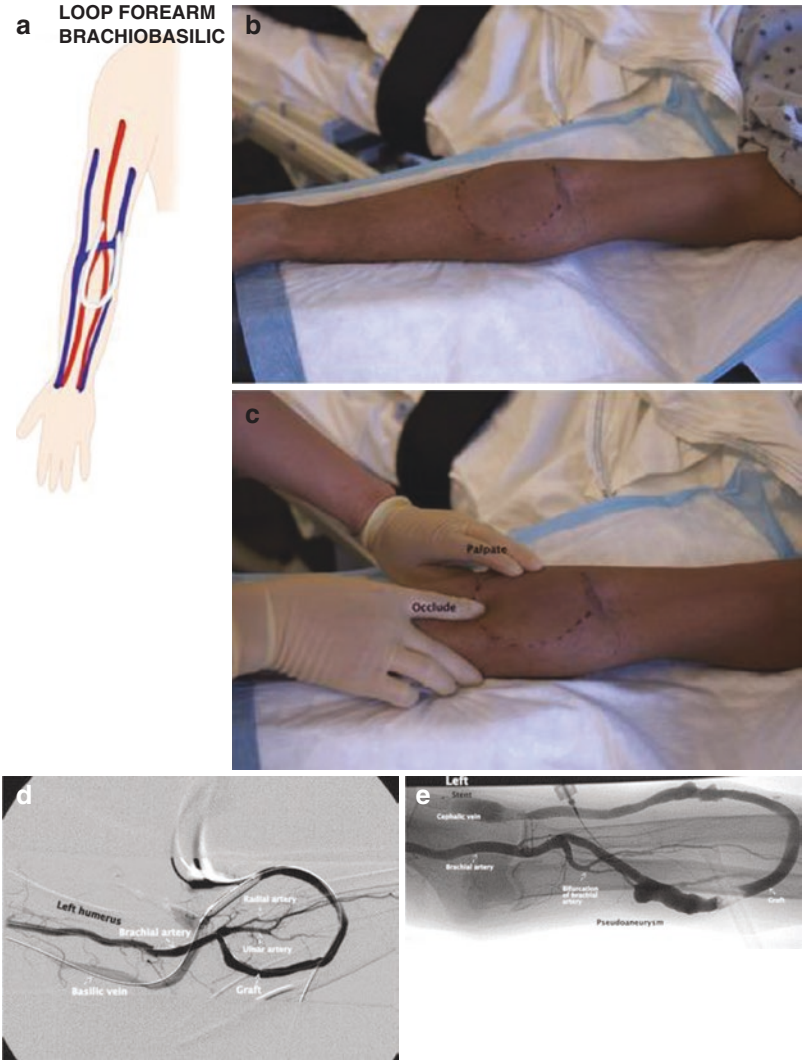

Fig. 8.5 (**a**) Forearm Brachiobasilic loop graft. (**b**) The forearm loop graft connects the brachial artery to either the cephalic, antecubital or basilic vein at the elbow. All three forearm loop grafts appear similar and it may be challenging to differentiate the two on physical examination. Regardless, the key point in approaching a patient with a forearm loop graft is identifying the inflow and outflow of the graft. (**c**) To determine the inflow of a forearm loop graft, occlude the apex of the loop graft with your right index finger and palpate the medial aspect of the graft. If the medial aspect of the graft becomes pulsatile, then the medial aspect of the graft is the inflow of the graft. If the thrill on the medial aspect of the graft disappears after occlusion at the apex of the graft, then the medial aspect of the graft is the outflow of the graft. (**d**) In a brachio-basilic forearm loop graft, the basilic vein is joined to the brachial artery using a loop graft in the forearm. The basilic vein runs on the medial aspect of the arm. Hence, the thrill may be palpable on the medial aspect, compared to the brachiocephalic graft where the thrill may be palpable on the lateral aspect of the arm. (**e**) A brachiocephalic forearm loop graft is created by joining the cephalic vein to the brachial artery through a loop graft in the forearm. Note the presence of a pseudoaneurysm on the graft and a stent at the graft vein junction. The cephalic vein runs on the lateral aspect of the arm

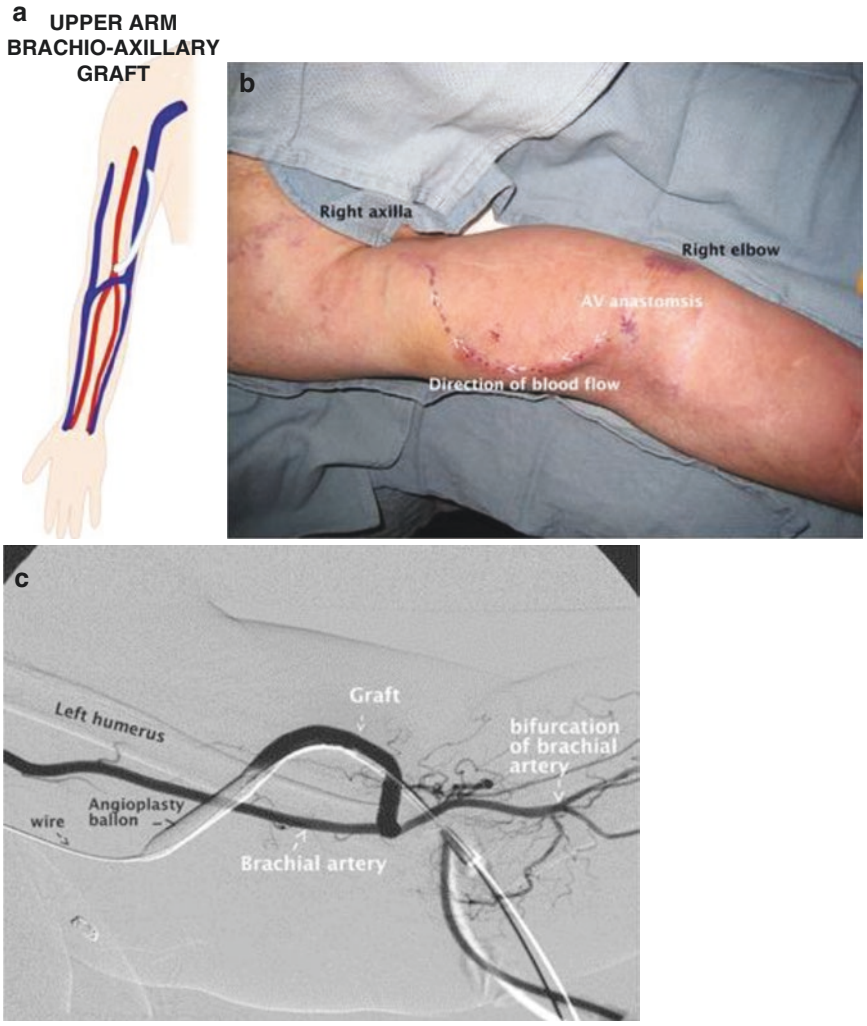

Fig. 8.6 (**a**) Brachio-axillary graft. (**b**) A right brachio-axillary arteriovenous graft is shown here. The AV anastomosis is at the elbow, and the direction of blood flow is as indicated. Note that the graft curved medially to join the axillary vein. (**c**) A left upper arm brachio-axillary arteriovenous graft is created by joining the axillary vein to the artery using a "curved" graft. Note that the graft needs to be curved medially to join the axillary vein. The wire is lying within the graft. This is a retrograde graftogram, which is performed by occluding the graft with an angioplasty balloon during contrast administration

The other veins used are the left internal jugular vein, femoral vein and subclavian vein. The subclavian vein should be avoided as it has a very high rate of stenosis following catheterization. This complication would preclude the construction of vascular access in the ipsilateral limb in the future.

2. Non-tunneled dialysis catheters have shorter extra-vascular segment and are inserted for short term (<1 week) use or when placement of a tunneled dialysis catheter is contraindicated. It is noted that the KDIGO suggests initiation of dialysis in AKI patients through the non-tunneled dialysis catheter rather than the cuffed catheter [12].

The use of a dialysis catheter is a double-edged sword. The insertion of a dialysis catheter is a relatively simple procedure and the catheter can be used immediately after insertion. On the other hand, they do not last as long as AVF and AVG and are prone to infection and thrombosis. Long term use of catheters is also associated with central vein stenosis. Specifically, the use of tunneled hemodialysis catheters for chronic hemodialysis is associated with increased hospitalization, morbidity and mortality. Hence, the use of tunneled hemodialysis catheters for chronic hemodialysis should not be encouraged.

Conclusion

Establishing functioning vascular access is critical in the delivery of hemodialysis. Timely referral to a surgeon for AVF creation by Stage 4 (glomerular filtration rate (GFR) less than 30 mL/min/1.73 m^2) Chronic Kidney Disease will alleviate the need for catheter placement at the time of dialysis initiation.

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9 Physical Examination of Dialysis Vascular Access and Vascular Access Surveillance

Ru Yu Tan, Chieh Suai Tan, David J. R. Steele, and Steven Wu

Introduction

Failure to detect dysfunctional vascular access may result in morbidity and mortality in patients receiving maintenance hemodialysis. Hence, routine monitoring is recommended by the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF KDOQI) guidelines [1]. Vascular access monitoring is performed by physical examination to detect signs that would suggest the presence of pathology. Surveillance refers to periodic evaluation of the vascular access by utilizing invasive or non invasive tests to detect the presence of pathology [1].

Thrombosis can occur in both arteriovenous grafts (AVG) and arteriovenous fistulas (AVF), although it is more common in AVG. Despite the proven advantages of AVF over AVG, AVF may still eventually fail. Declotting a thrombosed AVF is more time consuming, tedious, and has a lower success rate compared to declotting an AVG [2]. Vascular access thrombosis occurs due to stasis of blood in the presence of a flow-limiting stenosis [3]. It is important to intervene before thrombosis occurs as patency rates after declotting a thrombosed graft are reduced compared to that of an angioplasty of a patent arteriovenous graft [4]. It is

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postulated from population studies that regular monitoring and surveillance can detect a decrease in flow within a dialysis access, and hence, the development of stenosis. Through this, vascular access patency rates can be improved by intervening before vascular access thrombosis occurs [5]. While controversies remain over the benefits of vascular access surveillance in improving long-term vascular access patency [6], we believe that vascular access monitoring and surveillance complement each other in detecting failing access that may compromise the delivery of adequate dialysis and prevent thrombosis through preemptive interventions when clinically indicated.

Physical Examination

Physical examination of the dialysis vascular access is an essential skill for the interventional nephrologist. It can accurately detect and localize stenoses in a great majority of vascular access, is easy to perform and provides other important information in the evaluation of AVF and AVG. [7]

Inspection

Begin the physical examination by putting the patient's arms side by side on a pillow (Fig. 9.1a–d). The size of the two arms should be similar. Swelling of the arm and edema on the side of the dialysis access is suggestive of venous hypertension and possibly resistance to outflow locally or centrally. Look at the fingers and nails for any discoloration or dystrophic changes to suggest steal syndrome.

Focus your attention on the dialysis access (Fig. 9.2a–f). Determine if it is an AVF or an AVG, the type of anastomosis and the anastomotic site. Note the presence of collateral veins and look for signs of infection, hematoma or thinning of skin. Look for the presence of aneurysmal dilatation or pseudoaneurysm (Fig. 9.3a–i) Aneurysmal dilatation or aneurysm of AVF can occur as a consequence of outflow stenosis or weakened vessel wall from repeated cannulation of the same AVF segment. It differs from pseudoaneurysm as all layers of the vessel wall are present. Pseudoaneurysm occurs when the leak in the vessel is contained by the surrounding tissue instead of the vessel wall. It may occur in AVG due to the degeneration of the graft material or in AVF from arterial trauma.

Palpation

Begin palpation of the AVF or AVG from the anastomotic site. The AV anastomotic site is where the vein or graft is anastomosed to the artery. The direction of the blood flow within the AVF or AVG is away from the anastomosis and towards the chest. Place your fingers over the AV access to feel for the pulse and thrill. In general, there should be very little pulse in the AV access and it should feel soft and compressible

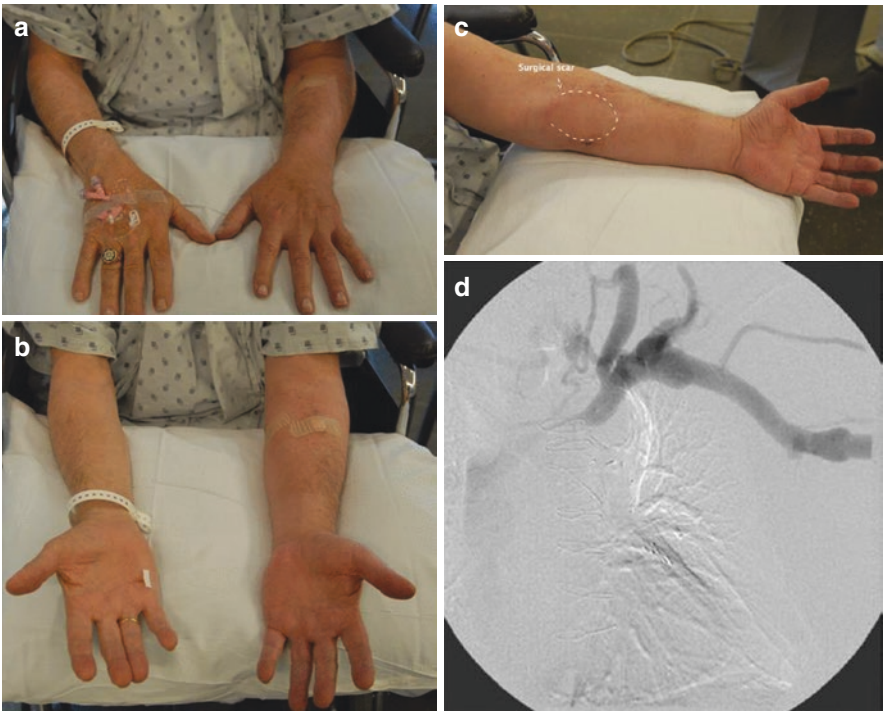

Fig. 9.1 (**a**) Inspection is a key component of the physical examination. Begin by placing both arms on a pillow. The AV access is on the left forearm and the left arm appears bigger than the right. (**b**) Turn the hand over and inspect the arm. The left arm is clearly larger than the right. The left hand also appears redder than the right. The bandage on the forearm indicates recent cannulation of the AV access. (**c**) On closer inspection, this patient has a forearm loop graft. Note the presence of a transverse surgical scar at the elbow and the loop configuration of the graft. (**d**) The cause of the arm swelling was secondary to severe stenosis of the left brachiocephalic vein

in an AVF. In the presence of downstream stenosis, the pulse will become strong and the AV access may feel "pulsatile". The thrill of the AV access is related to flow and is most obvious at the anastomosis. It has both a systolic and diastolic component and should be continuous over the entire course of the AV access. In the presence of a significant downstream stenosis, the thrill may only be felt during the systolic component. Move the finger(s) along the AV access; the pulse and thrill may suddenly disappear at the stenotic segment. For an AVF, assess the diameter and depth of the fistula to determine if it can be easily cannulated for dialysis. Specifically, assess if the length of the AVF is long enough to accommodate the placement of two dialysis needles. By convention, there should be a minimum of 5 cm between the arterial and venous needles to avoid access recirculation. The arterial needle should be placed at least 1 inch from the AV anastomosis to avoid causing damage to the anastomosis.

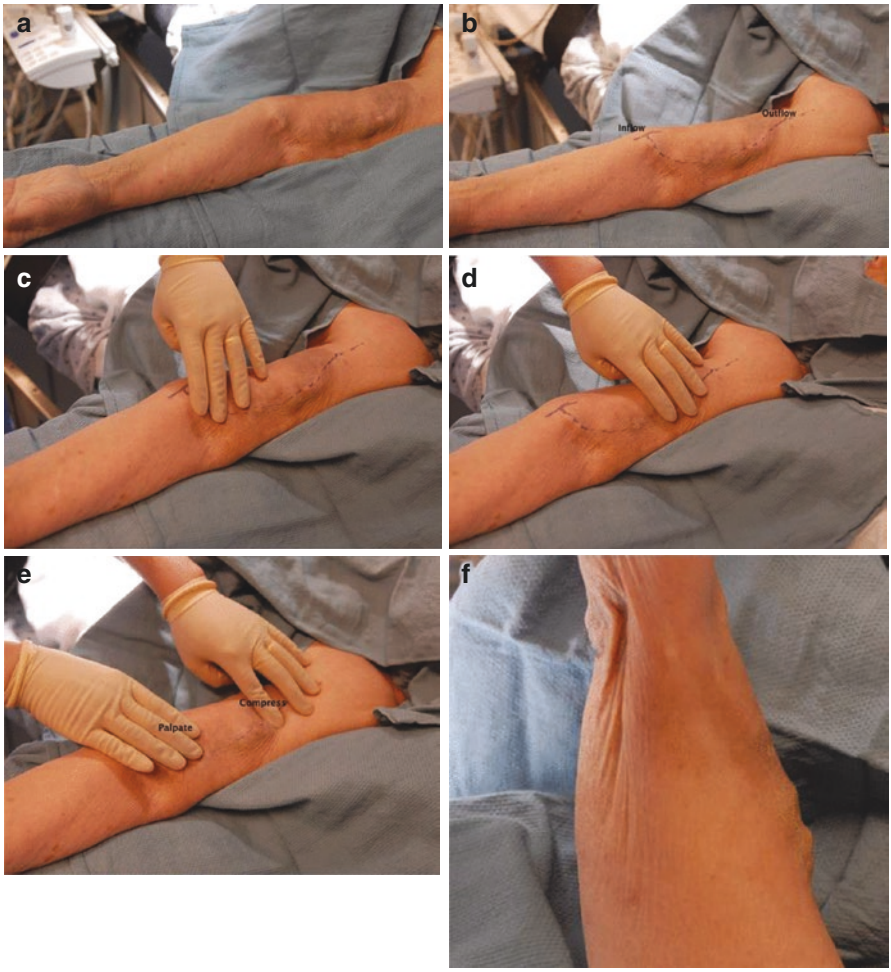

Fig. 9.2 (**a**) Identify the type of AV access that the patient has placed. This patient has a left brachiocephalic AVF. (**b**) Identify the inflow and outflow of the AVF. (**c**) Begin palpation from the inflow of the AVF. (**d**) Palpate the entire length of the AVF to feel the thrill and pulse. (**e**) Augmentation: Palpate the pulse of the AVF with your right fingers, then compress the AVF with your left index finger. The pulse intensity over the right fingers should increase. Failure to increase the pulse intensity with compression of the AVF would suggest inflow stenosis. (**f**) Arm elevation: Raise the arm above the level of the heart. The AVF should collapse or become flaccid. In the example shown, the brachiocephalic fistula remained distended after arm elevation, suggestive of an outflow stenosis

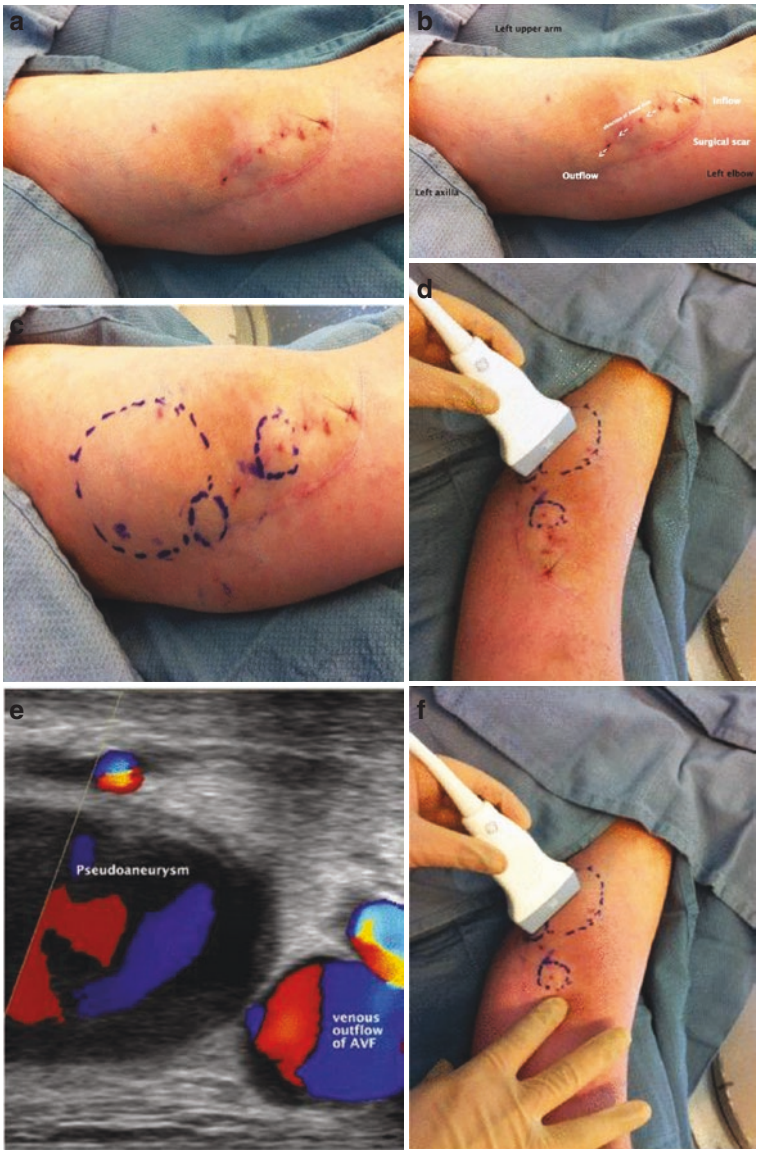

Fig. 9.3 (**a**) The patient complained of left arm pain and swelling after dialysis. (**b**) This is a left transposed brachiobasilic fistula. The direction of blood flow is as indicated below. (**c**) A large area of swelling was noted near the outflow of the fistula. (**d**) Ultrasound was performed to assess the area. (**e**) Color doppler showed flow within the area of swelling, suggestive of a pseudoaneurysm. (**f**) Doppler study of the pseudoaneursym was repeated with occlusion of the inflow of the fistula. (**g**) No flow was seen in the venous outflow of the fistula after occlusion of the inflow but flow continued within the pseudoaneurysm, suggestive that the pseudoaneurysm is arising from the arterial system. (**h**) Angiography confirmed that the pseudoaneursym was arising from the brachial artery. The basilic vein was in close proximity to the brachial artery and the brachial artery was probably inadvertently injured during placement of the dialysis needle. (**i**) The pseudoaneurysm was treated with thrombin injection with good results

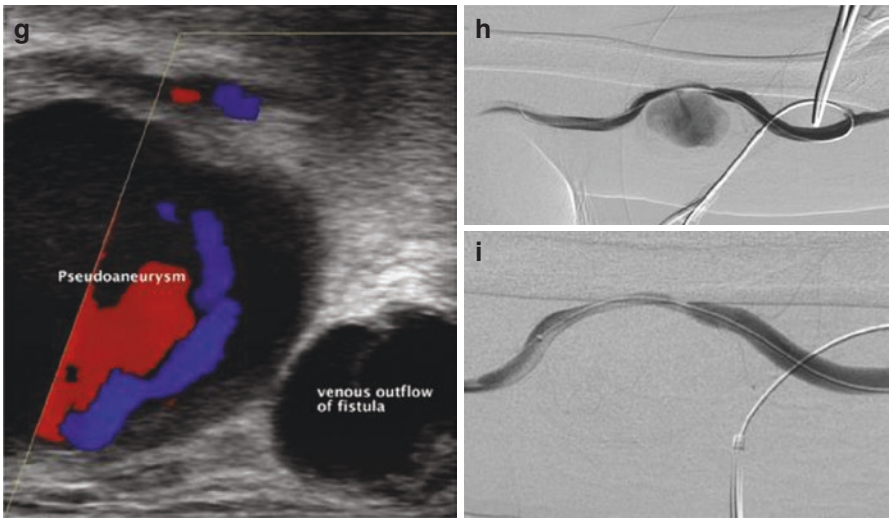

Fig. 9.3 (continued)

Maneuvers

Pulse Augmentation

This maneuver works better in AVF than AVG and is useful to evaluate the inflow of the AV access. Feel the pulse of the AV access with your right fingers and occlude the AV access at some point distant from the arterial anastomosis with your left index finger. The pulse intensity should feel stronger (augmented) on the right fingers. Failure to augment suggests the presence of an inflow stenosis. If the pulse is already pulsatile before augmentation, failure to augment after occlusion is suggestive of a severe outflow stenosis.

Arm Elevation

This maneuver is useful when examining the AVF. Raise the arm above the level of the heart. The AVF should collapse or become flaccid. In the presence of venous stenosis, the segment of the AVF before the stenosis will remain distended while the segment after the stenosis will collapse. This is a good preliminary screening test for evaluation of the access outflow.

Auscultation

The auditory manifestation of the thrill, or the bruit, can be easily auscultated over the vascular access. It has both a systolic and diastolic component and has a low pitch, rumbling character. It is especially useful to confirm the presence of flow

when the thrill is not very well palpable. In the presence of a downstream stenosis, the diastolic component may be lost and the pitch becomes higher as the stenosis increases in severity.

Surveillance

Dialysis access flow surveillance is performed by using devices to measure the access blood flow (Qa) and venous pressure (VP). A detailed description of the various techniques and protocols is beyond the scope of this chapter. Briefly, Qa can be measured by using doppler ultrasound, ultrasound dilution technique using Transonics hemodialysis monitor (Transonics, Inc., Ithaca, NY), conductance or thermal dilution technique. Some dialysis machines have in-built conductivity cells to allow real-time or "on-line" measurement of dialysate conductance. Propriety software and algorithm have been incorporated into these dialysis machines to compute changes in conductance for Qa measurement during dialysis, therefore eliminating the need for additional access monitoring equipment.

VP is the pressure required to infuse blood back into the access and is routinely recorded during hemodialysis. It may be elevated due to malposition of the needle or the presence of outflow stenosis and is less sensitive and specific than direct measurements of access flow rates. It is only meaningful if reading is obtained at the beginning of dialysis or at low pump speed as much of the resistance arises from the needle rather than the vascular access when the blood flow rate is high. On the other hand, intra-access pressure (IAP), which is a component of VP, can be sequentially measured for surveillance of AVG. IAP can be measured directly as static IAP using a pressure measuring device or indirectly calculated using computerized algorithm as an equivalent IAP from the VP recorded using dialysis. The IAP is usually less than 50% of the mean arterial blood pressure in an AVG and the ratio increases with the development of outflow stenosis.

It is important to monitor trends and correlate with clinical findings rather than relying on a single measurement when planning for intervention. The 2019 NKF KDOQI vascular access update also stated that action should not be based solely on surveillance findings [1]. For example, intervention is usually indicated when a decrease in Qa is accompanied by difficulties in cannulation, recurrent triggering of the arterial alarm (from low pressure) during dialysis or when dialysis adequacy is affected. On the other hand, an increased VP accompanied by prolonged bleeding from the cannulation sites after dialysis; enlarging AV access, arm swelling or recurrent triggering of the venous alarm (high pressure) during dialysis also warrant further evaluation. The location and positioning of the needles on the AV access can affect the readings. It is therefore important to check if there is any change in the cannulation sites when there is a sudden variation in the Qa and VP trends.

Hemodialysis Dose and Adequacy

The advent of hemodialysis therapy has converted end-stage renal disease from a terminal illness to a chronic disease state. Similar to the management of many other

chronic diseases, it is essential to monitor the adequacy of treatment to ensure the patients' well-being.

Blood investigations are done at least once a month to monitor the adequacy of diaysis. While the presence of persistent hyperkalemia, or metabolic acidosis is suggestive of insufficient clearance, urea has always been used as the conventional marker for dialysis clearance.

The Urea Reduction Ratio (URR) or the Clearance of Urea (Kt/V) can be used to assess the adequacy of dialysis. For access surveillance, it should be noted that there are "variants" of Kt/V of urea such as the single pool Kt/V (spKt/V), double pool kt/V(dpKt/V) and equilibrated Kt/V (eKt/V); therefore, one should use the same formula for assessment each time when computing trends in an individual patient.

Access Dysfunction and Recirculation

When the delivered dialysis dose is much lower than what is prescribed, vascular access dysfunction and recirculation should be considered as a possible cause. Assessment of recirculation can be performed using the ultrasound-based dilution technique or blood-based urea measurement. A recirculation value greater than 10% should be considered abnormal and warrants consideration for a diagnostic angiogram.

Hemodialysis access recirculation occurs when dialyzed blood returning through the venous needle reenters the dialysis circuit through the arterial needle instead of the systemic circulation (Fig. 9.4a–c). This is commonly due to the presence of high-grade venous stenosis, which obstructs the venous outflow and causes the backflow of blood into the arterial needle. Occasionally, it may also be caused by inflow stenosis, which results in the backflow of blood from the venous limb of the access to meet up with the demand generated by the arterial blood pump of the machine.

Recirculation may also be caused by inadvertent reverse placement of the arterial and venous needles on the AV access. In particular, the direction of blood flow in a loop graft needs to be determined accurately to ensure correct needle placement. One easy way to determine the direction of flow is to temporarily occlude the loop AVG at its apex and palpate the graft at either side of the occlusion. The thrill on the venous limb should diminish while the arterial limb will become more pulsatile.

The consequence of mixing dialyzed or cleansed and undialyzed/uncleansed blood within the dialysis circuit is a lower solute concentration within the blood compartment of the dialyzer. The lower solute concentration results in a lower diffusion gradient across the dialyzer membrane and decreases dialysis efficiency. Therefore, regular monitoring and surveillance protocols are important as inadequate dialysis from access recirculation can affect the well being of dialysis patients and results in life threatening electrolyte imbalances such as hyperkalemia and metabolic acidosis.

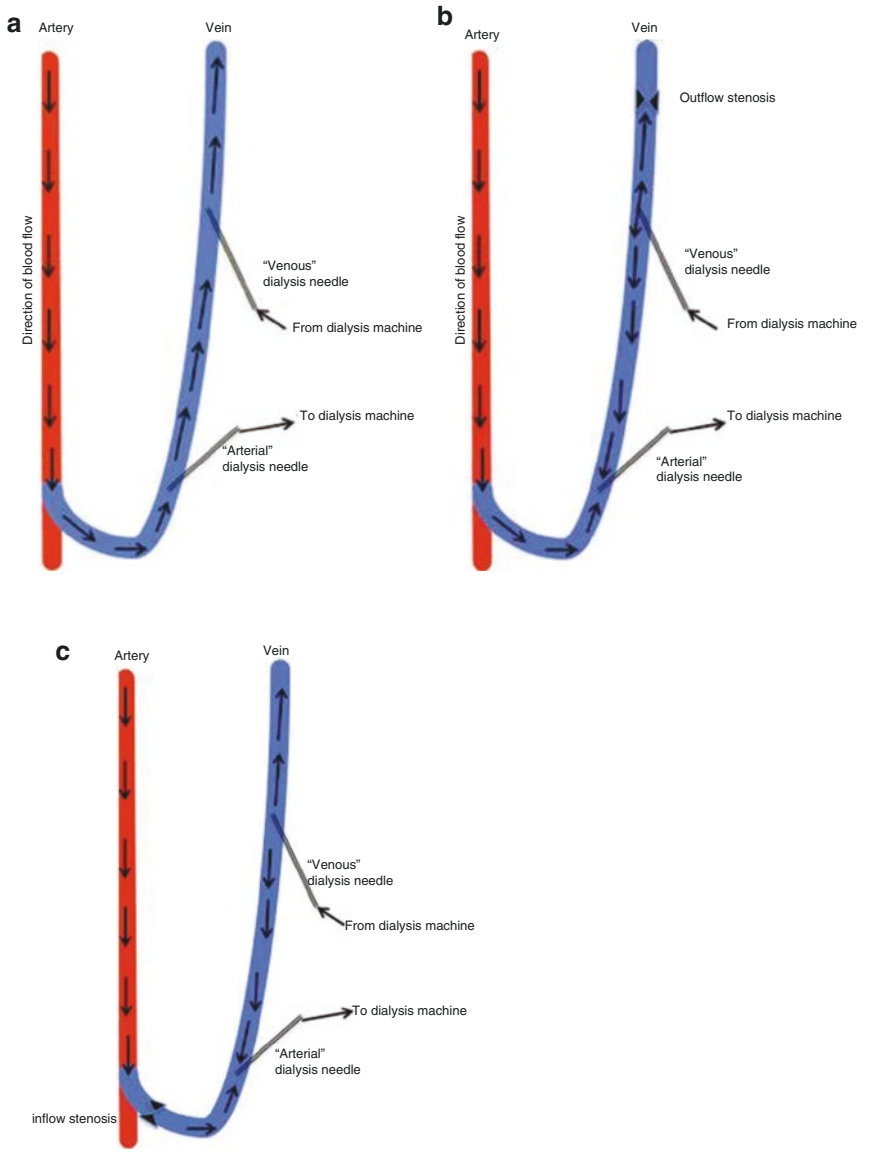

Fig. 9.4 (**a**) The direction of blood flow is shown by the arrows. The "arterial" dialysis needle is typically placed against the direction of blood flow while the "venous" dialysis needle is placed in the opposite direction to minimize access recirculation. The typical blood flow rate within an AV access is usually above 600 mL/min. The blood pump of the dialysis machine will generate a negative pressure to pull blood into the dialysis circuit. The usual blood flow rate within the dialysis circuit is around 300 mL/min. (**b**) In the presence of an outflow stenosis, the venous pressure within the AV access will increase and obstructs the return of blood from the dialysis circuit. This may cause the back flow of blood into the arterial needle, resulting in an increase in recirculation. (**c**) In the presence of inflow stenosis, the blood flow rate within the AV access will decrease. The inflow of blood into the AV access may not be able to meet up with the demands of the dialysis machine. The negative pressure generated by the arterial blood pump of the dialysis machine will "pull" blood from the venous limb into the dialysis circuit to maintain the flow, causing an increase in recirculation

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Non-invasive Imaging of Dialysis Access Circuit

10

Mark Reddick and Sanjeeva Kalva

Introduction

A well functioning dialysis access is the lifeline for patients with end stage renal disease (ESRD) on hemodialysis. Dialysis access circuits include both arterio-venous fistulas (AVF) and arterio-venous grafts (AVG). An AV fistula is created by connecting a vein to an artery, resulting in one anastomosis. An AV graft is created when a biologically acceptable tube is connected to an artery at one end and to a vein at the other end, resulting in two anastomoses. The upper extremities are preferred over the lower extremities for dialysis access creation and fistulas have been shown to out-perform grafts in terms of durability and infection rates [1]. Maintaining a given dialysis access requires early detection of access dysfunction. The goal is to keep the dialysis access functioning such that adequate dialysis is achieved, as well as to prevent thrombosis of the access. Once dialysis access thromboses, its lifespan declines dramatically [1–3].

Duplex ultrasound continues to be the mainstay imaging modality for evaluation of dialysis access circuits. Dialysis access circuits are typically superficial and allow excellent evaluation by ultrasound. Ultrasound imaging can augment the physical exam in detecting dialysis circuit problems by characterizing and localizing problem areas within the access circuit and help identify patients at high risk for future circuit thrombosis [4]. The use of pre-operative ultrasound for mapping prior to access has been shown to improve patency of dialysis access and identify situations

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in which alternative access sites should be utilized [5–7]. The use of routine surveillance ultrasound of already existing dialysis access circuits remains controversial but is typically done in conjunction with some evidence of access dysfunction from parameters measured at dialysis or from clinical exam findings [8]. The information gathered from a thorough ultrasound evaluation of a dialysis circuit is unique relative to other vascular beds. This is because the creation of a graft or fistula results in unique flow dynamics including increased peak systolic and end diastolic velocities, high flow rates, turbulence and spectral broadening. A thorough understanding of these unique flow dynamics is therefore critical when interpreting these studies.

Indications

Indications for dialysis access ultrasound include: low flow, difficult cannulation, clot aspiration, elevated venous pressures, prolonged bleeding, elevated recirculation time, and diminished urea reduction rate. These indications are encountered during dialysis. There are several clinical indicators of fistula/graft dysfunction that can also be used to initiate an ultrasound evaluation including: poor maturation, evidence of infection, perigraft mass (hematoma, pseudoaneurysm), symptoms of distal limb ischemia or "steal", loss of thrill or diminished thrill, pulsatility and collapse of the fistula/graft. Some of these indications suggest a problem within a particular segment within the circuit (Table 10.1). For example, a history of low flow suggests an inflow or arterial anastomotic or juxta-anastomotic stenosis peripheral

Table 10.1 Common indications for ultrasound evaluation of dialysis access circuits and their associated ultrasound findings

Indication for ultrasound study	Common findings on ultrasound
Low flow, poor maturation, diminished or absent thrill, collapse of the vein	Stenosis of inflow artery, arterial anastomosis, or juxta-anastomotic segment peripheral to the cannulation zone
Difficult cannulation	Cannulation zone too deep (> 6 mm), short segment of superficial cannulation zone, immature fistula (< 4 mm, flow <500 mL/min), tortuosity
Clot aspiration	Non specific
Elevated venous pressure, prolonged bleeding, pulsatility	Stenosis central to the cannulation zone (outflow or central veins)
Elevated recirculation time, diminished urea reduction rate	Competing outflow, stenosis peripheral or central to cannulation zone
Suspected infection	Perigraft fluid collection, soft tissue edema, aneurismal degeneration of anastomosis
Perigraft mass	Abscess, hematoma, pseudoaneurysm
Steal symptoms	High flow in graft/fistula with augmentation of plethysmographic waveform in digits with graft/fistula compression

to the cannulation zone. A history of elevated venous pressure, however, suggests a stenosis that is central to the cannulation zone.

Ultrasound Acquisition, Display and Transducers

In addition to acquiring excellent anatomical images, modern ultrasound machines are also capable of acquiring important hemodynamic information such as the presence, direction and location of flow, as well as the velocity of blood flow and the presence of turbulence. Also, quantitative flow measurements can be obtained with the use of appropriate software packages that can be incorporated into the ultrasound unit.

There are a few key terms regarding the modes of ultrasound data acquisition and display that are fundamental to understand when interpreting dialysis access ultrasound studies. B-mode or gray scale ultrasound generates a two dimensional image of anatomical structures in shades of gray. This mode is useful to evaluate the anatomy of the circuit, measure vessel diameters and depths (Fig. 10.1) and to assess the presence and extent of perigraft hematomas, abscesses, and pseudoaneurysms (Fig. 10.2).

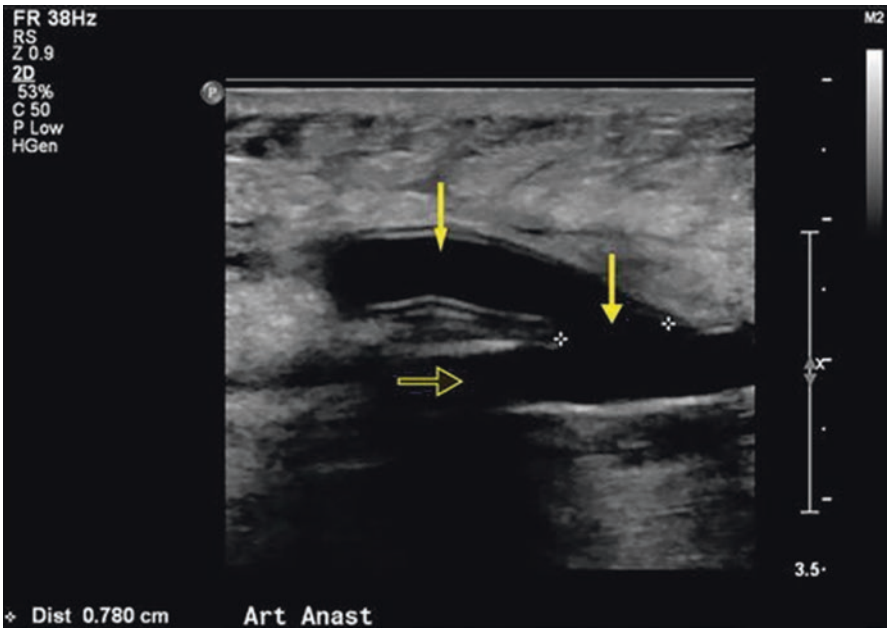

Fig. 10.1 Gray scale image demonstrating the arterial anastomosis of a dialysis access graft. The anastomosis can be accurately measured in gray scale imaging. Open arrow points to the inflow artery. Thin arrow points to the anastomosis which measures 7.8 mm as indicated by the calipers. The thicker arrow points to the arterial limb of the synthetic graft

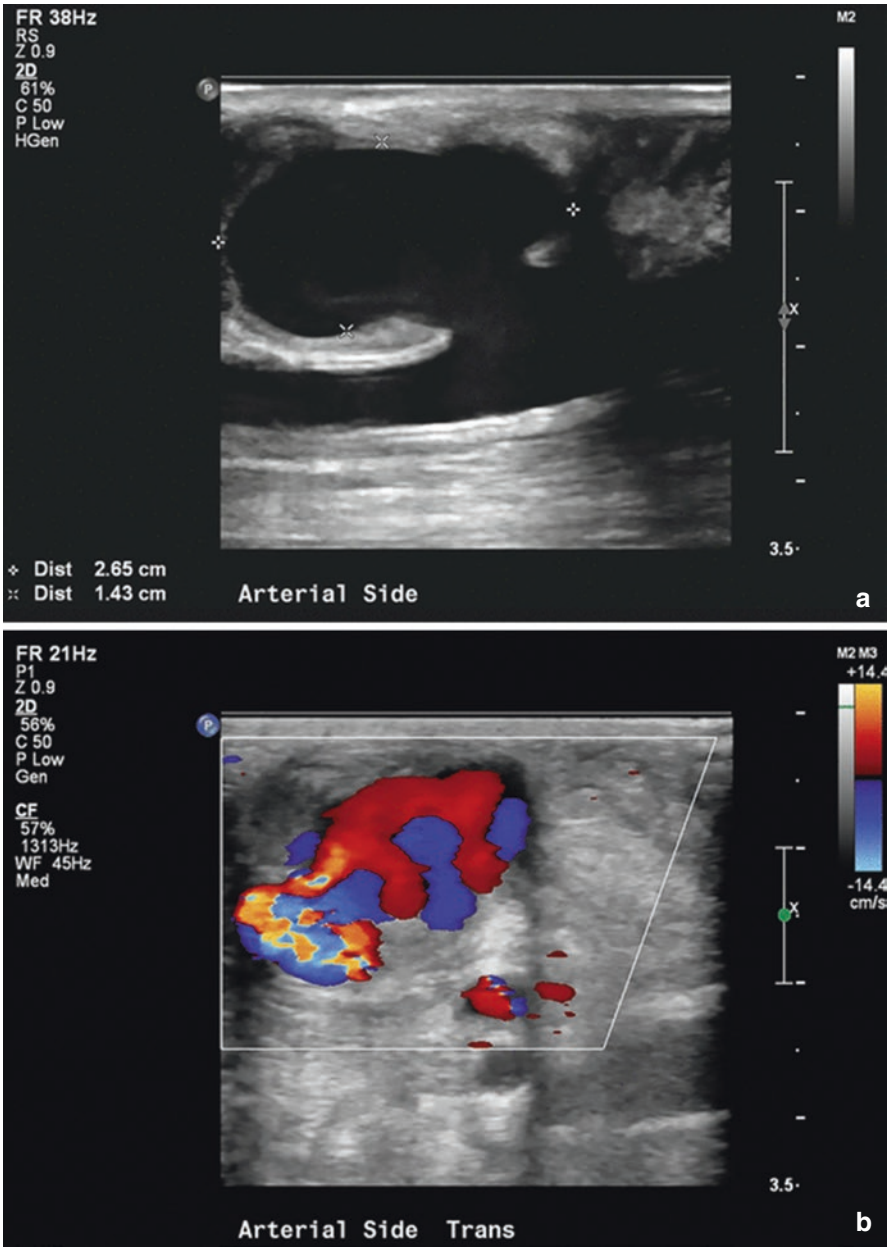

Fig. 10.2 Gray scale or B-mode ultrasound image of an access site pseudoaneurysm. The aneurysm is lobulated and measures 2.7 cm × 1.4 (**a**). This pseudoaneurysm demonstrates flow within it on color Doppler in a different scan plane (**b**)

Pulse wave Doppler is a technique in which the transducer emits ultrasound in pulses. Blood flow velocity measurements are limited to those in the physiological range, but the depth of the recorded velocity measurement can be determined. Color flow Doppler is a type of pulsed Doppler that adds information regarding the direction of flow (Fig. 10.3). In general, flow towards the transducer is assigned a red color while flow away from the transducer is assigned a blue color. Turbulent flow can be detected by the presence of admixing of red and blue indicating that flow is going both toward and away from the transducer at such a location.

Another important mode of ultrasound acquisition and display is spectral Doppler. This mode of ultrasound displays flow velocities as a spectrum on the Y-axis along a time line on the X-axis. The term spectral broadening refers to a greater range of flow velocities. This often correlates with turbulent flow detected with color flow Doppler and can be seen at sites of stenosis.

Finally, duplex Doppler refers to a form of image display in which both color flow and spectral Doppler waveforms are displayed simultaneously. This allows for accurate localization of velocity information (Fig. 10.4).

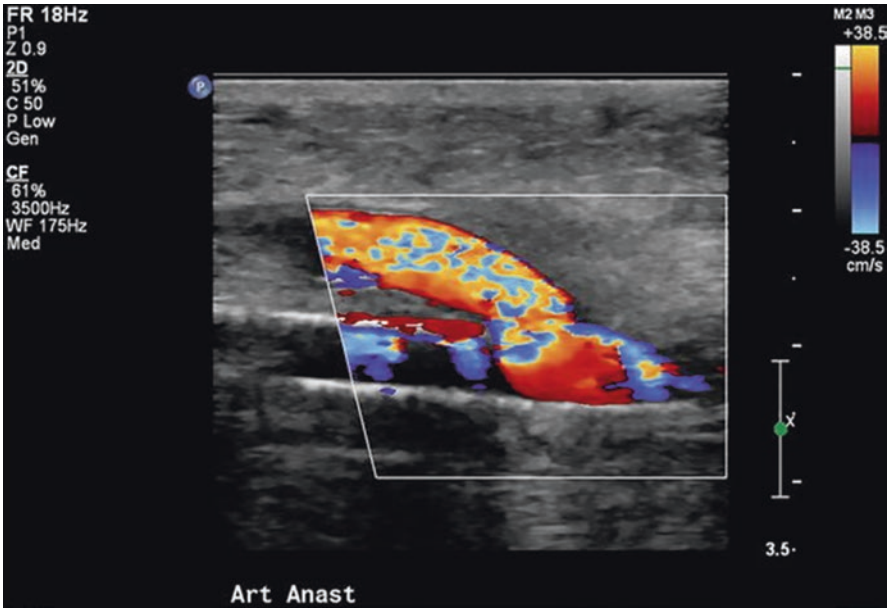

Fig. 10.3 Color Doppler image of the same arterial anastomosis as in Fig. 10.1 demonstrating turbulent flowwithin the arterial limb of the graft. Note that there are areas of both blue and red, indicating turbulence. This is a normal finding at a dialysis access arterial ansatomosis but would be considered abnormal in a native artery

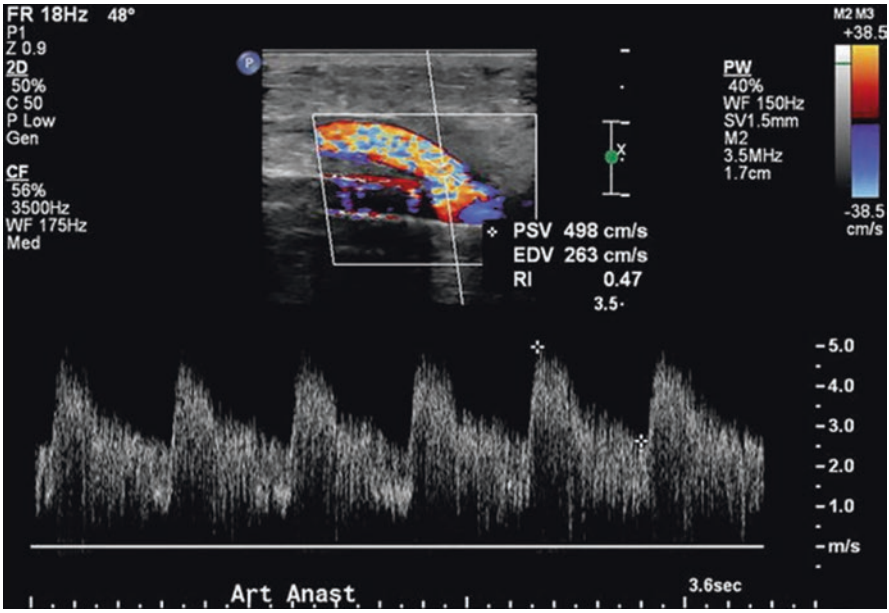

Fig. 10.4 Duplex Doppler image of the same arterial anastomosis as seen in Figs. 10.1 and 10.3. The waveform is velocity on the Y-axis and time on the X-axis. Note the spectral broadening or filling in of the waveform. This correlates with the mixing of the blue and red colors on color Doppler. The angle of incidence is 48 degrees as indicated in the upper left hand corner and the sample volume is very small in within the central flow lumen of the sampled area. The peak systolic velocity and end diastolic velocities are 498 cm/sec and 263 cm/sec, respectively

The types of transducers that can be used for dialysis access evaluation include curved, phased array and linear transducers. Frequencies range from 2 MHz to 10 MHz. In general, curved and phase array transducers are helpful in evaluating deeper structures while linear transducers are better for superficial structures. Higher frequency transducers are more sensitive for detecting low flow and have better spatial resolution whereas low frequency transducers allow better penetration. Aliasing is an artifact that occurs when the sampled blood is flowing too fast for the system to accurately obtain its velocity. This happens more commonly with higher frequency transducers.

It is usually possible to gather the appropriate information necessary to make access management decisions based on a thorough physical exam and ultrasound evaluation of a given dialysis access. However, there are occasionally patient-specific factors that can limit the study. These factors include the presence of catheters or lines, edema, hematoma, wounds, surgical dressings, contractures and calcification.

Doppler Imaging

In order to interpret the velocity and waveform information obtained from Doppler evaluation of dialysis access circuits, it is important to understand the Doppler shift equation, the angle of incidence, and the Doppler sample volume, and how these are interrelated. One critical part of interpreting these studies is to ensure that the angle of incidence and sample volume parameters are appropriately selected.

The Doppler equation is as follows:

$$\Delta F = 2\, F_0\, v\, (\cos\theta)/C$$

- Delta f is the Doppler frequency shift
- f0 is the ultrasound emission frequency
- V is the mean velocity of blood flow
- C is the speed of sound in tissue (1450 m/sec)

This equation demonstrates that the Doppler shift is proportional to the frequency of the probe that is used, the velocity of the blood flow, and the cosine of the Doppler angle. The Doppler angle is the angle between the central flow lumen of the vessel and the ultrasound beam. This angle can be manipulated by the ultrasound user. The cosine θ portion of this formula dictates the optimum angle of incidence when measuring velocity. Since Cos 90 equals 0, there will be no detectable velocity when the transducer is perpendicular to the direction of flow. Since Cos 0 equals 1, the highest velocity will be acquired when the ultrasound beam is parallel to the direction of flow. However, gray scale image quality is compromised at this angle and such an angle is often not technically feasible. Angles above 60 degrees result in increasing magnification of velocity error. In order to standardize and optimize the acquisition and reporting of Doppler velocities, an angle of incidence as close to 60 degrees is used. If the Doppler angle is improperly set, it can manifest as an apparent stenosis when there is not a stenosis present (Fig. 10.5).

Another important parameter to consider is the sample volume. This sets the volume of flowing blood from which the velocity data and waveforms are obtained. In the Doppler equation, v is the mean velocity of blood flow. This mean velocity changes with the size of the sample volume. This is due to the normal distribution of velocities within a vessel where centrally there is more uniform laminar flow and the velocities drop off with distance from the central flow lumen to the vessel wall. In general, the sample volume should be as small as possible and as central as possible (Fig. 10.4).

The longitudinal plane allows for greater ease of setting the Doppler angle at 60 degrees. Areas of stenosis will demonstrate characteristic decreased lumen diameter on gray scale imaging, increased peak systolic velocity, spectral broadening and turbulence, as well as decreased velocity downstream from the stenosis (Fig. 10.6).

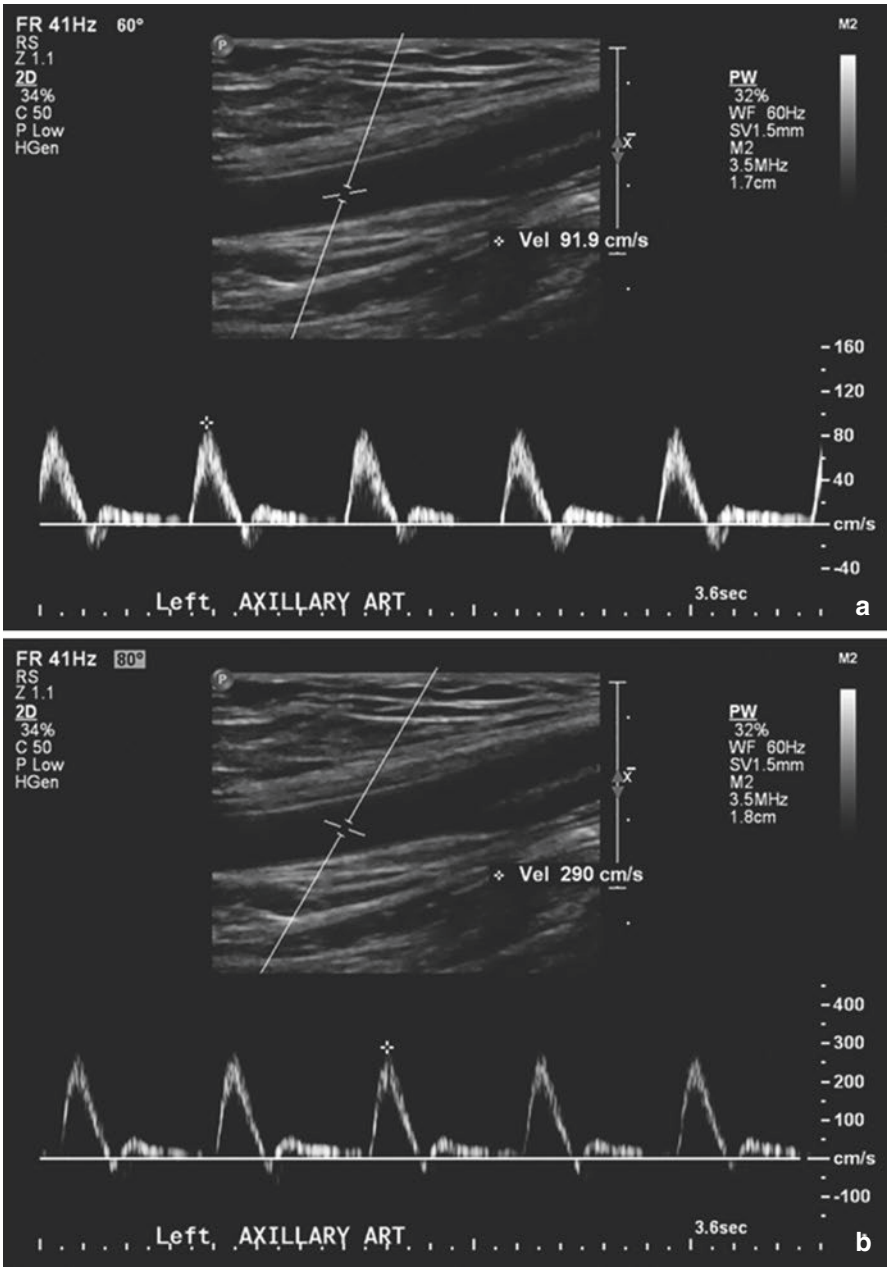

Fig. 10.5 The Doppler angle can be chosen by the ultrasound user. In (**a**), the Doppler angle is set to 60 degrees (upper left corner). This gives a velocity measurement of 91.9 cm/s. In (**b**), the Doppler angle is incorrectly chosen at 80 degrees. This gives a velocity of 290 cm/s which suggests a possible stenosis in the axillary that is being interrogated. In order to optimize and standardize vascular ultrasound reporting, an angle as close as possible to 60 degrees is chosen

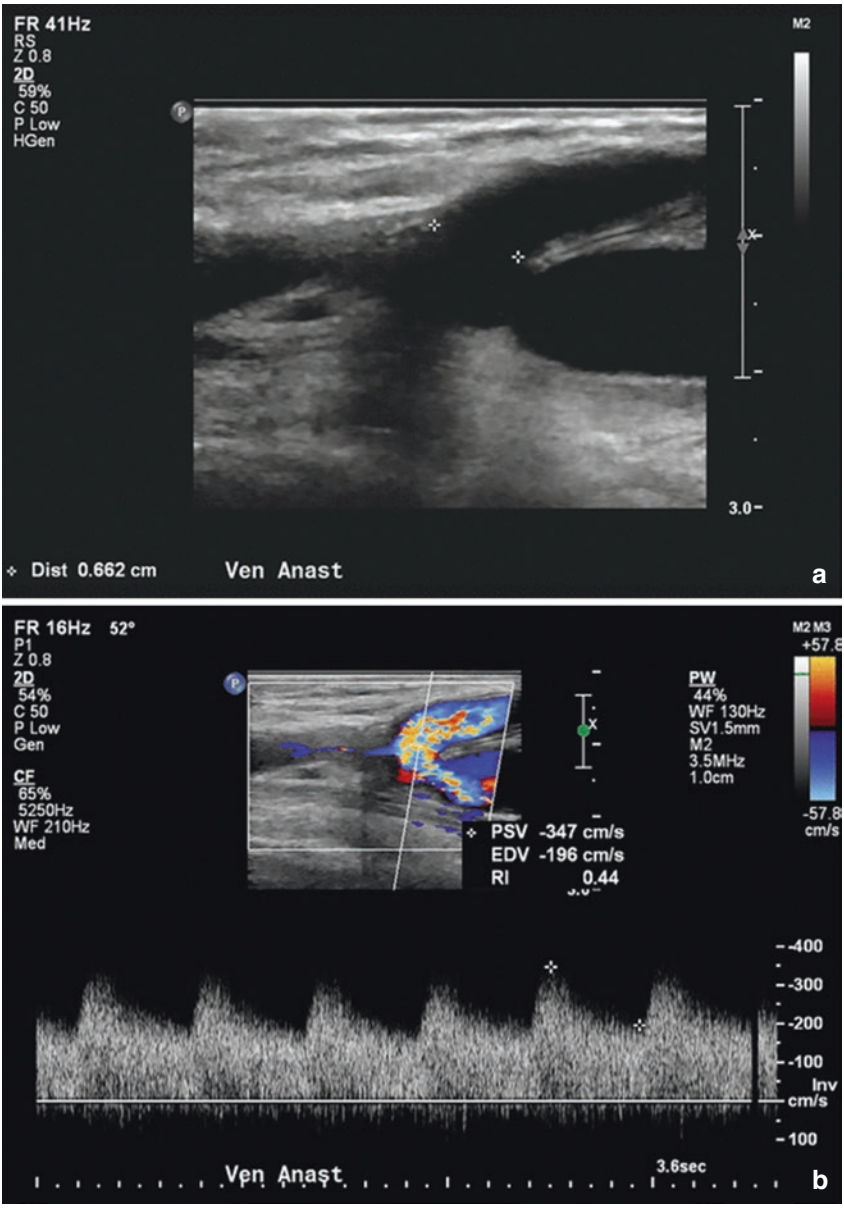

Fig. 10.6 (**a**) Gray scale image demonstrating the venous anastomosis measuring 6.6 mm, as well as a high grade juxta-anastomotic stenosis with marked reduction of luminal diameter. (**b**). Duplex Doppler image of the venous anastomosis demonstrating PSV of 347 cm/s and EDV of 196 cm/s. (**c**) Magnified color Doppler image demonstrating the high grade juxta-anastomotic stenosis with only a trace of color flow through the stenosis along with elevated velocity of 450 cm/s. Note the neointimal hyperplasia seen as a smudgy gray area on either side of the stenotic flow lumen (yellow arrow) and elevated velocity at the site of stenosis of 450 cm/s (**d**). (**e**) Duplex Doppler image of the outflow vein immediately central to the stenosis demonstrating a marked reduction in velocity of 50 cm/s

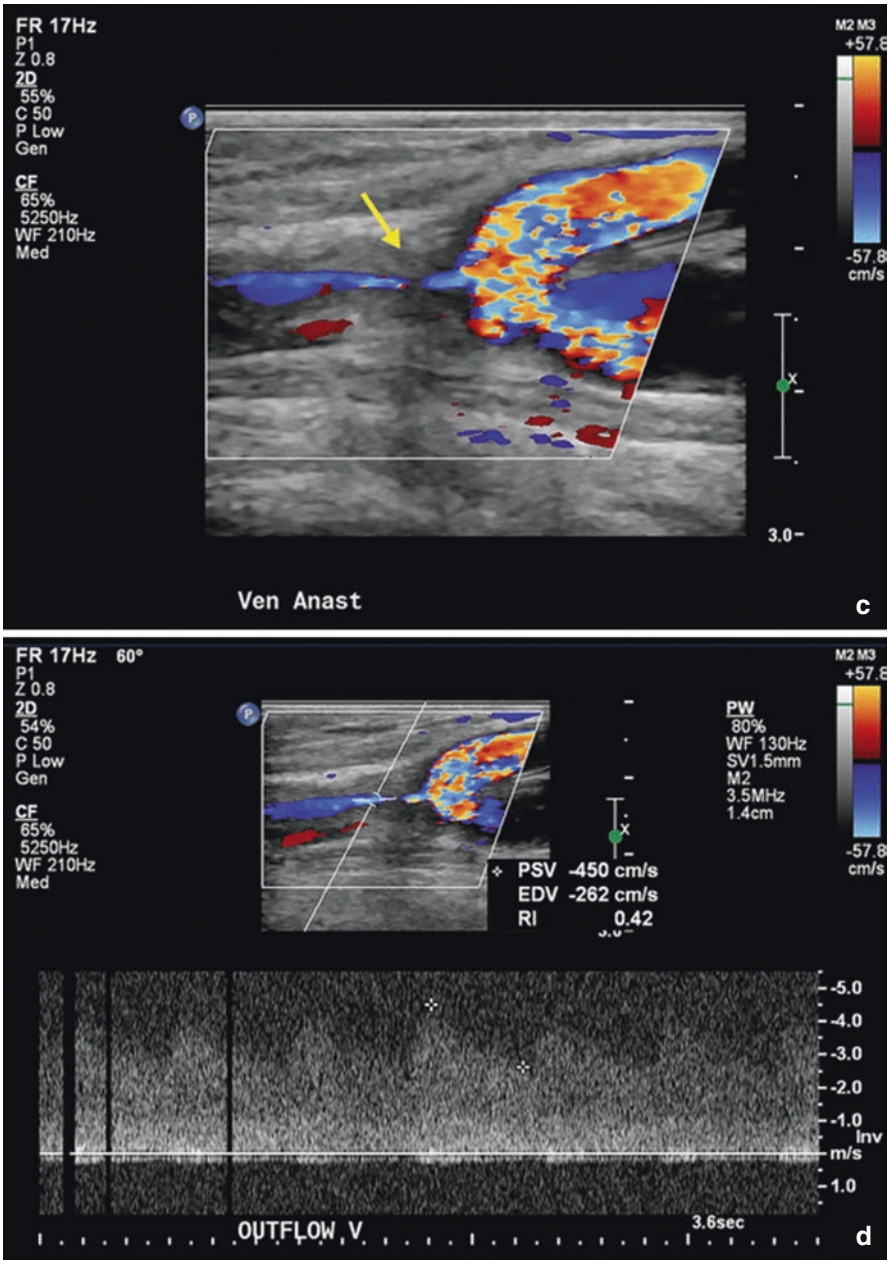

Fig. 10.6 (continued)

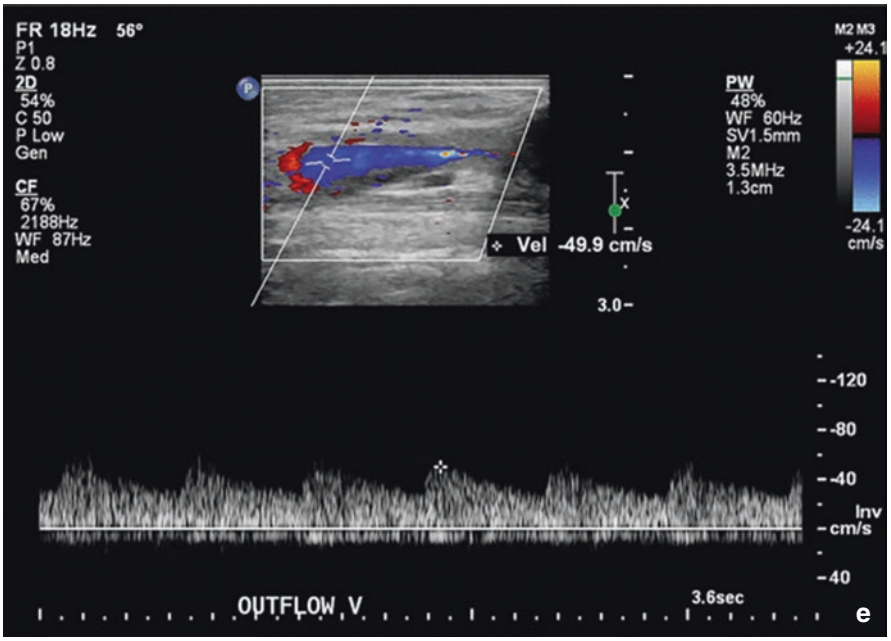

Fig. 10.6 (continued)

The typical range of blood flow velocity shifts is from approximately 0.2 KHz to 8 KHz, which is in the audible range. This allows the imager to simultaneously listen to the flow dynamics and correlate this important aspect of vascular ultrasound imaging with the data that is acquired.

Another useful capability of Doppler ultrasound is calculation of flow volume. This is accomplished by the use of a mathematical software package that is incorporated into the ultrasound unit. To measure flow volume, the imager places the sample volume gate as done with velocity measurements but the gate is extended to include the entire vessel lumen The diameter of the vessel is also demarcated with cursors. It is important for the diameter to be demarcated in gray scale as this eliminates any overestimation of diameter caused by bleed through on color Doppler (Fig. 10.7). Flow rates within dialysis fistulas/grafts are typically in the range of 500–1500 mL/min. At these flow rates, adequate dialysis is usually achieved. In patients with peripheral arterial disease, however, higher flow may result in vascular steal symptoms (see Chap. 15).

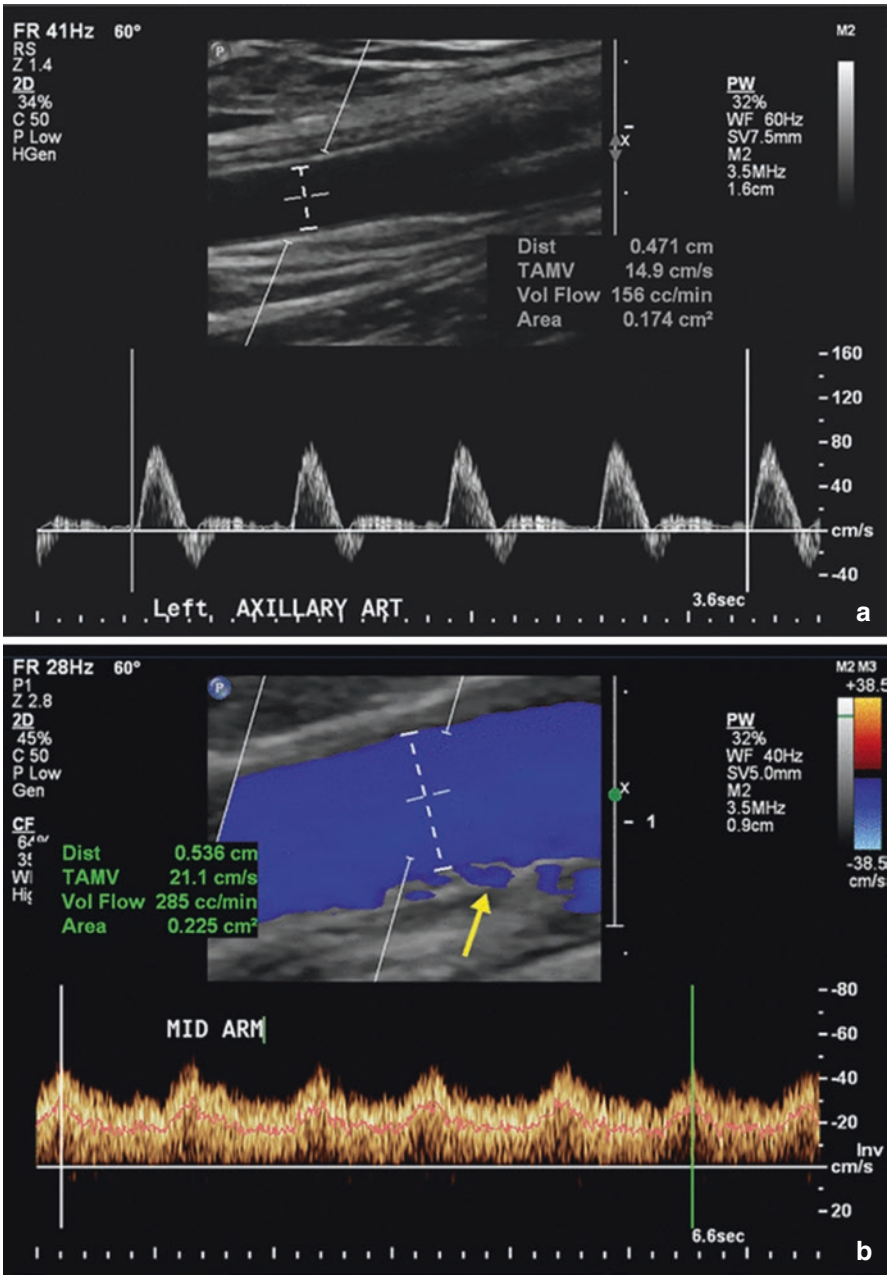

Fig. 10.7 (**a**) Calculation of flow volume in the axillary artery. This requires the diameter of the vessel to be demarcated which is most accurately done in gray scale. The angle of incidence is set at 60 degrees as indicated in top left hand corner. Mathematical software calculates the flow volume based on a prescribed formula utilizing the mean velocity. (**b**). Segment of dialysis access circuit in a different patient from (**a**). Note the "bleed through" in the color image (yellow arrow). This could result in overestimation of flow by demarcating too large of a diameter

Procedure

When performing ultrasound dialysis access circuit evaluation, the patient should be in the supine position, slightly reclined, with the access extremity extended approximately 45 degrees and externally rotated. A detailed protocol for Duplex Doppler evaluation of hemodialysis access circuits was published in 2012 by Teodorescu et al. [9]. We concur with this protocol, which starts with evaluation of the entire circuit in the transverse plane from the inflow artery to the outflow veins and central venous structures if they are amenable. This gives the imager an overview of the anatomy of the circuit and preliminarily identifies potential areas of stenosis. Next, the inflow artery is evaluated in the longitudinal plane using pulsed color and spectral (duplex) Doppler. The PSV, EDV and flow volume should be recorded. The proximal anastomosis is then interrogated. Gray scale diameter measurement, color Doppler and spectral PSV, EDV and waveforms should be recorded. If the access is a graft, the venous anastomosis should also be interrogated with similar imaging documentation. PSV, EDV and flow volume should be measured within the cannulation zone. The remainder of the access should then be scanned in the longitudinal plane using color flow Doppler to identify areas of turbulence. Once identified, an area of turbulence should be further evaluated with color and spectral Doppler documenting PSV, EDV and waveforms. For any stenosis, the same imaging data should be acquired at an area 2 cm upstream and 2 cm downstream. The axillary, subclavian and innominate veins should be imaged in gray scale, color and spectral Doppler (Fig. 10.8). It is often not possible to image the central veins. Normal findings in the central veins include: continuous flow with mild phasicity in the axillary and subclavian veins and respirtatory phasicity in the more central inominate veins. There should be increased flow towards the heart during inspiration and decreased flow during exhalation. Abnormal findings in the central veins include lack of flow in keeping with acute or chronic occlusion. In chronic central venous occlusive disease, the parent veins will demonstrate lack of flow, absence of respiratory phasicity in the peripheral veins and presence of numerous tortuous, dilated collateral vessels (see CT image from Fig. 10.10).

Other important images and measurements should be obtained depending on the indication. For example, if the problem is difficult cannulation, then the depth of the access from the skin surface should be documented. In general this should be 6 mm or less. If only a small segment of the access is superficial, measurement of the length of this segment should be documented. If the problem is poor maturation, then the diameter of the cannulation zone should be measured. If this measurement is less than 4 mm or the flow is less than 500 mL/min, there is a high likelihood that the access will not mature [10].

In addition to assessing the adequacy of the access for dialysis, other things to consider and document are the presence of perigraft hematomas, pseudoaneurysms or other perigraft fluid collections (Fig. 10.2). These findings alert the dialysis team and treating physicians to additional problems with patient's access beyond the adequacy for dialysis.

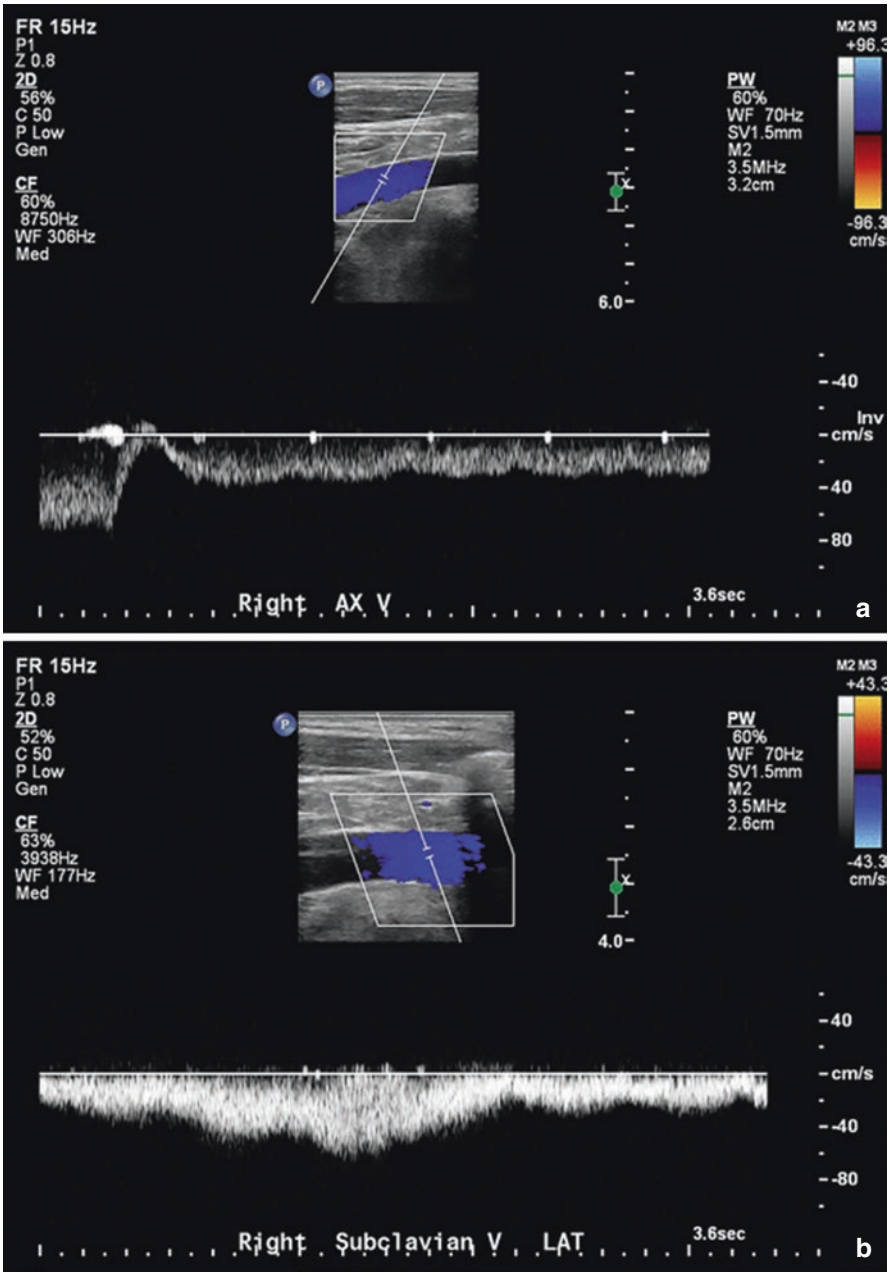

Fig. 10.8 Normal duplex Doppler images of more central venous structures including the axillary (**a**), subclavian (**b** and **c**) and inominate veins (**d**). These are frequent areas of stenosis in patients with a history of prior dialysis access or other chronic indwelling catheters

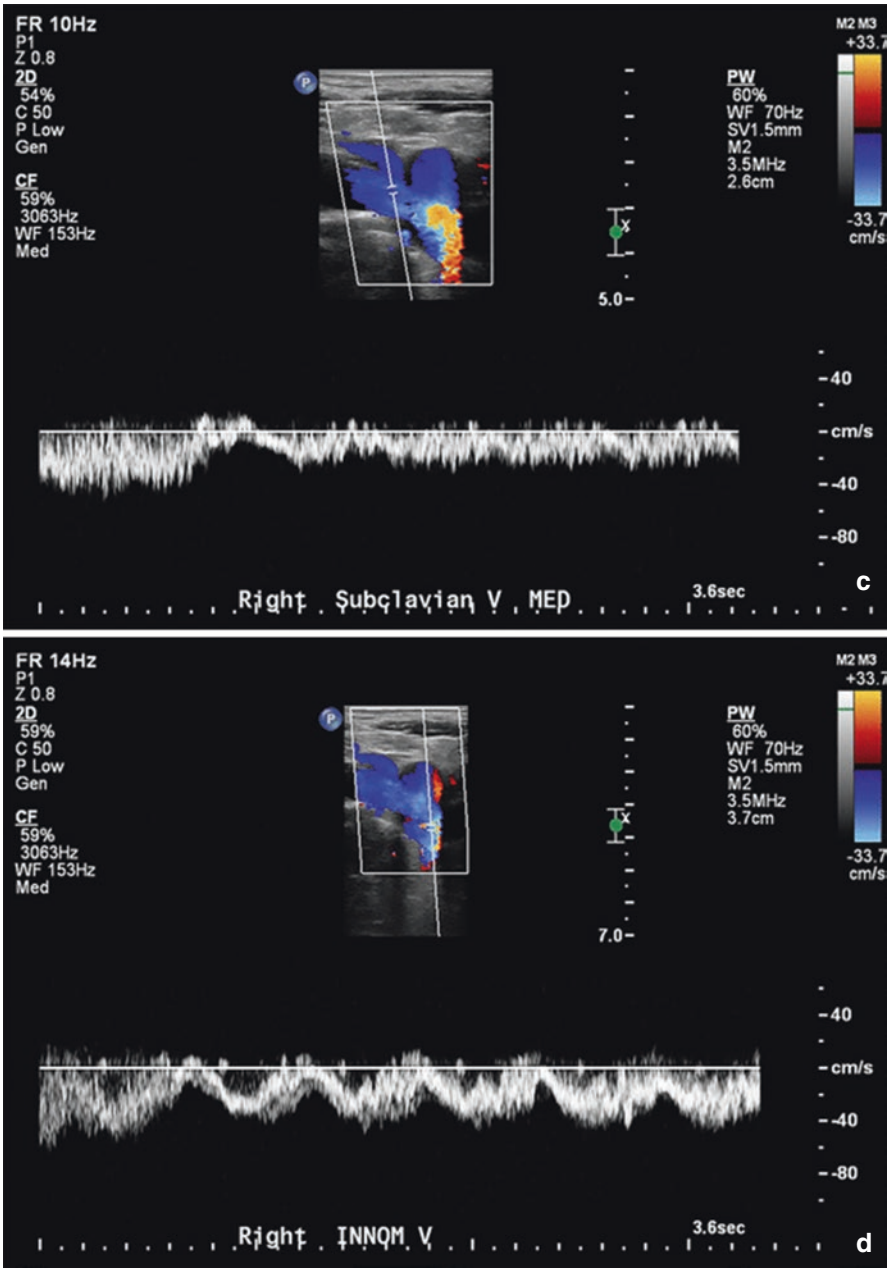

Fig. 10.8 (continued)

Diagnostic Criteria

Normal findings as well as findings associated with moderate and severe stenosis are listed in Table 10.2. Also, those findings that are associated with inflow and outflow stenosis and access occlusion are listed.

Pitfalls

Several potential pitfalls can limit the integrity of the ultrasound study. These include chronic occlusion of outflow or central veins that are well collateralized, low systemic blood pressure, and poor Doppler angle or sample volume selection. Also, discrepancies can arise such as a focal velocity > 300 cm/s with no apparent luminal diameter reduction or absent velocity acceleration in the presence of a flow

Table 10.2 Classification of stenoses and their associated ultrasound imaging findings. From Teodorescu et al. [9]

Classification	Velocity (cm/sec)	Imaging characteristics
Normal	Mid graft PSV > 150 cm/sec Anastamosis PSV > 300 cm/sec, chaotic, disorganized flow	No visible narrowing Distended outflow veins Aneurysms, puncture sites, perigraft fluid may be visible
Moderate stenosis	Ratio of PSV at stenosis to PSV at 2 cm beyond anastamosis if normal-appearing <3	Decrease in lumen diameter Echogenic narrowing Wall abnormalities
Severe stenosis	Marked velocity acceleration at stenotic area Ratio of PSV at stenosis to PSV 2 cm beyond anastamosis if normal-appearing >3	Intraluminal echogenicity with <2 mm Lumen or > 50% diameter reduction Marked reduction in lumen diameter with color Doppler
Inflow stenosis	Increased PSV at the site of the stenosis with monophasic and diminished waveforms distally Flow acceleration with graft compression at the outflow anastamosis	Intraluminal echogenicity <2 mm lumen at velocity acceleration
Outflow stenosis	Mid graft PSV < 100 cm/sec Distal vein PSV > 300 cm/sec Velocity at the proximal anastamosis will diminish in proportion to severity of venous outflow stenosis	Intraluminal echogenicity with <2 mm lumen at velocity acceleration Prominent collateral veins around outflow
Occlusion	No Doppler signal	Intraluminal echogenicity Graft walls collapsed Occluded vein may not be visible

PSV peak systolic velocity

lumen diameter reduction. This can be seen with inflow disease (see Chap. 15) or low systemic blood pressure. In these situations, a correlative study such as CTA or catheter based angiography may be useful.

Computed Tomography Angiography

Axial imaging such as computed tomography angiography (CTA) may be useful when there is discrepant data acquired from ultrasound. Also, CTA is useful if there is concern for graft infection or abscess, aneurismal degeneration of an anastomosis, or an extensive fluid collection. In this setting, the graft may be extracted and the CT will also document the patency of the central venous structures prior to creation of a new circuit access.

A few studies have been done evaluating the efficacy of CTA for evaluation of the failing dialysis access [11–15]. These studies include a relatively small number of patients. CTA was shown to have a relatively low sensitivity for detecting central venous stenoses, particularly in veins that are in close proximity to osseous structures such as the subclavian vein. This study also showed that all plane evaluation resulted in greatest sensitivity in detection of venous stenoses [13]. Dialysis circuits containing stents are generally not amenable for evaluation by MRI/MRA due to associated artifact. CTA, however, gives relatively minimal associated artifact (Fig. 10.9). One area where CTA has been shown to be of value is in the assessment of aneurysms. Unlike conventional angiography, CTA shows both the flow lumen through the aneurysm as well as areas of thrombosis. CTA was also shown to demonstrate the existence, extent and anatomy of venous collaterals (Fig. 10.10) [13].

At this time, we recommend the use of CTA in instances where there is discrepant data from the physical exam and/or ultrasound. It may also be used to evaluate the central venous structures prior to access creation or planned recanalization and to evaluate aneurysms and extent of venous collaterals.

MRI/MRA

Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) play a limited role in the evaluation of dialysis circuits. Adequate evaluation of dialysis circuits requires the use of Gadolinium as contrast material. The use of Gadolinium based contrast materials in this population has been linked to the development of nephrogenic systemic sclerosis (NSF). NSF is a devastating diffuse sclerosing disease that affects the skin, eyes, joints and internal organs. Newer Gadolinium-based contrast materials, (group 2 agents), are now available for use in this patient population. Non-contrast MR imaging of dialysis access circuits is challenging and not routinely performed. One significant advantage of MRA over CTA is that signal is obtained only from vascular structures. This limits the effect of bone

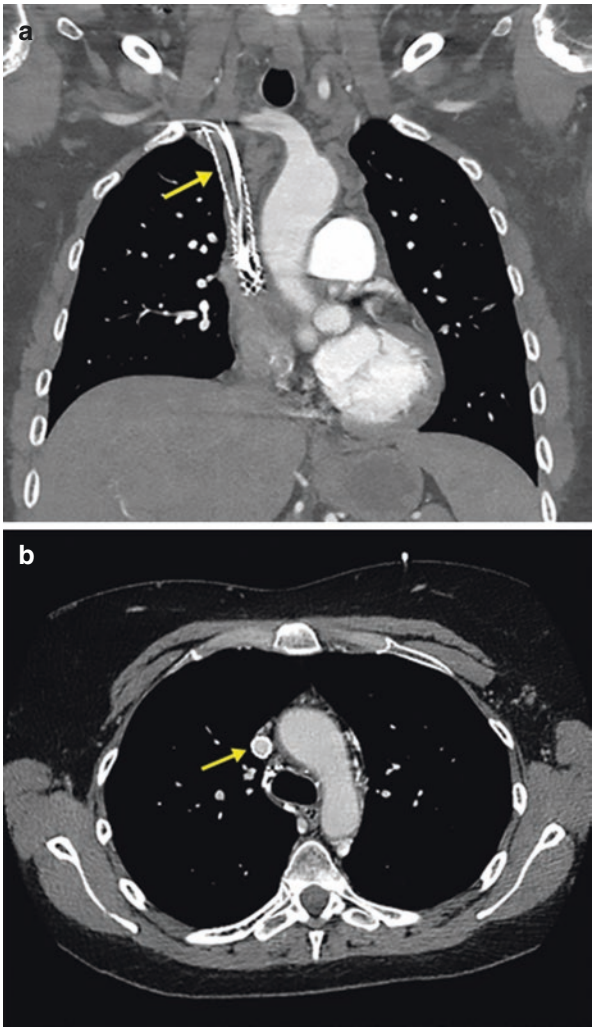

Fig. 10.9 (**a**) CTA demonstrating an occluded SVC stent (yellow arrow). Very little artifact is seen related to the stent. Most of the artifact is related to the mediport catheter occupying a large portion of residual flow lumen in this undersized stent performed at an outside institution. In (**b**), the patency of the stent is well established with CTA (yellow arrow). This is after mediport removal and recanalization of the SVC

and calcium related artifact. However, unlike CTA, artifact related to graft material and/or circuit stent limits evaluation of graft/stent patency and may overestimate or underestimate stenosis [16].

Conclusions

Dialysis access ultrasound evaluation is useful for early detection of access problems. This chapter discussed the indications for dialysis access evaluation using ultrasound. We provided a brief overview of ultrasound, a detailed protocol for performing dialysis access ultrasound studies, as well as the diagnostic criteria for

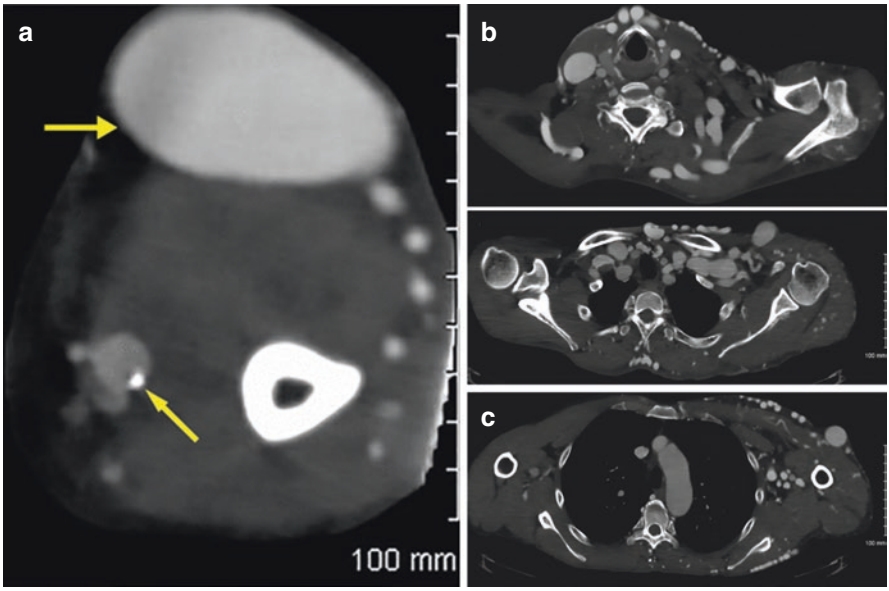

Fig. 10.10 (**a**) CTA showing a brachial artery catheter (thin yellow arrow) placed for CTA evaluation of a failing left arm radio-cephalic dialysis access circuit. Note the large, aneurismal outflow vein (thick yellow arrow). (**b–d**). Extensive collaterals in the upper arm, axilla, chest wall, mediastinum due to bilateral subclavian vein chronic occlusion

access dysfunction and some pitfalls. The utility and limitations of axial imaging such as CTA and MRA were discussed.

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Angiogram and Angioplasty

11

Suh Chien Pang, Chieh Suai Tan, Steven Wu, and Arif Asif

Introduction

It is important to use the correct terminology for radiological procedures. An angiogram is a diagnostic procedure to visualize blood vessels using contrast material. Hence, angiograms of arteriovenous fistula (AVF) and arteriovenous graft (AVG) are called fistulogram and graftogram respectively.

Angioplasty is a term used to describe a procedure to dilate stenosed or occluded blood vessels. In radiology context, it is synonymous with percutaneous transluminal angioplasty (PTA), which involves entry through the skin (percutaneous) and going through the vessels (transluminal) to the site of the lesion and dilate the narrowed or occluded blood vessel (angioplasty).

These two techniques (angiogram and angioplasty) are valuable in the management of arteriovenous (AV) access as stenosis secondary to neointimal hyperplasia is often the cause of access thrombosis and failure in a dialysis patient. It is

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important to note that a clinically significant AV access stenotic lesion is one that is accompanied by clinical signs and symptoms and angiographic evidence of more than 50% narrowing.

Venous Intervention

Although both arterial and venous interventional procedures require similar endovascular skill set, there are distinct dissimilarities between the two procedures. The knowledge of these differences is crucial to ensure successful endovascular interventions. The differences are summarized in Table 11.1.

The site of lesion in an AVF is broadly divided into "inflow" or "outflow" stenosis as summarized in Fig. 11.1. The stenosis may occur at the arterial, artery-vein anastomosis, juxta-anastomotic segment, body (needling sites), draining and central veins. It is often possible to delineate the site of stenosis based on the clinical findings and physical examination. These are as summarized in Table 11.2.

AVFs have a primary non-functional rate of about 20% (range,10–50%) that varies among centers and requires a maturation time between 1 and 4 months [1].

Table 11.1 Differences between venous and arterial interventions during dialysis access management

	Venous intervention	Arterial intervention
Complications	Rupture, thrombosis, pulmonary embolism	Dissection, thrombosis, rupture
Balloon length	Oversize	Cover the lesion and a little beyond
Balloon diameter	Oversize	Do not oversize
Duration of inflation	Long, minimum 3 min	Short
Type of balloon	High pressure balloon	Choice depends on type of lesion

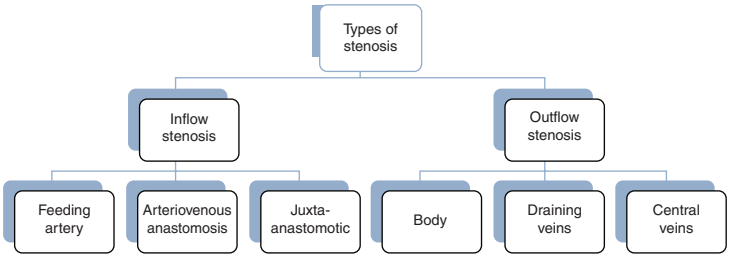

Fig. 11.1 The juxta-anastomotic (JA) segment is defined as the first 2 cm of the vein after the artery-vein anastomosis. The body of the AVF is defined as the segment of the venous limb where cannulation for dialysis is made while the draining veins (inclusive of the cephalic arch) are segments that drain the AV access. The central veins include the axillary, subclavian, brachiocephalic veins and the superior vena cava

Table 11.2 Summary of possible sites of stenosis in an AVF in relation to symptoms and physical examination

Clinical findings	Physical examination	Possible sites of stenosis
High dynamic or static venous pressure	Pulsatile AVF	Outflow stenosis
Prolonged bleeding after removal of dialysis needle	Pulsatile AVF	Outflow stenosis
Upper limb swelling	Swollen arm	Central vein stenosis
Decrease thrill	Flat AVF	Inflow stenosis and body
Difficult cannulation	Flat or difficult to palpate	Inflow stenosis and body
	Good thrill	No stenosis, AVF may be too deep for cannulation
"Failure to mature"	Flat AVF	Inflow stenosis
	Multiple dilated veins	Presence of accessory veins or collaterals

Failure of maturation may be due to the presence of an inflow stenosis caused by neointimal hyperplasia at the juxta-anastomotic segment [2], which can be treated with angioplasty. Late fistula failure is caused primarily by neointimal hyperplasia that results in venous stenosis. The most common site of stenosis in the radiocephalic fistula is at or around the juxta-anastomotic region. In contrast, most stenoses occur in the swing segment for brachiobasilic fistula and the cephalic arch for brachiocephalic fistula respectively [3–6].

AVG has lower primary failure rates but inferior long-term patency when compared to AVF. The site of stenosis is usually at the graft-vein anastomosis or within 6–10 cm of the anastomosis [1].

Equipment

Prepare the following equipment on a sterile trolley (Fig. 11.2).

1. Normal saline for flushing of sheaths and catheters
2. Lidocaine
3. 18 Gauge (G) intravenous catheter
4. 0.035 in. wire
5. 6 Fr or 7 Fr 4 cm sheath
6. Kumpe catheter
7. Angioplasty balloon
8. Balloon inflator
9. 2/0 non-absorbable suture (Ethilon)
10. Gauze
11. Hemostat
12. Needle holder
13. Syringes and needles

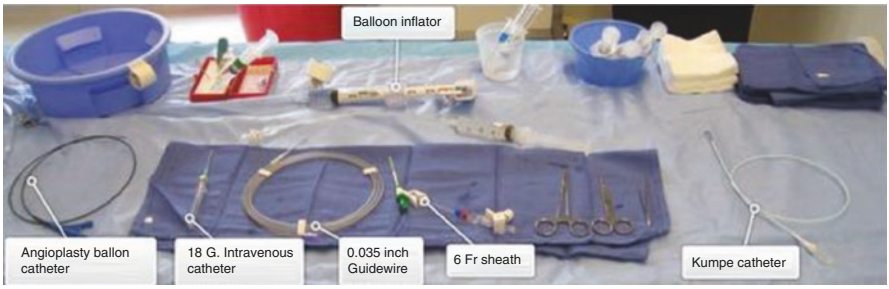

Fig. 11.2 Equipment for fistulogram and angioplasty

Fistulogram and Angioplasty

1. Review the indications for the procedure. The indications for the procedure often provide clues to the site of stenosis. Bedside ultrasonography is a useful tool to confirm the site of stenosis.
2. Examine the AVF to identify (Fig. 11.3a, b)
   (a) "Parts" of the AVF,
   (b) Needling sites for dialysis,
   (c) Direction of flow,
   (d) Aneurysmal segment,
   (e) Contraindication to intervention such as the presence of infection,
   (f) Thrill, pulse and bruit of the AVF as baseline for comparison after the procedure.
3. Determine the site of sheath placement
   (a) Place it close to and towards the direction of the stenotic site.
   (b) Avoid placing it over the aneurysmal segment or directly over the stenotic segment as it will impede angioplasty.
4. Anesthetize the skin with 1% lidocaine (Fig. 11.4).
5. Cannulate the AVF with a 18G intravenous catheter. In this case example, the suspected lesion is at the JA segment. Hence, the catheter is placed in a retrograde direction towards the inflow (Fig. 11.5).
6. Advance the needle till you feel a "give" and see "flashback".
7. Hold the needle in position while pushing in the cannula (Fig. 11.6).
8. Do an initial diagnostic fistulogram using the cannula (Fig. 11.7).
9. Inject the contrast material through the cannula to visualize the AVF and draining veins all the way through the superior vena cava.
10. Note the presence of any stenotic lesions (Fig. 11.8a–d).
11. Do a "reflux" fistulogram by occluding the outflow vein while injecting the contrast material (Fig. 11.9a, b).
12. The stenosis in this case example is around the JA region.

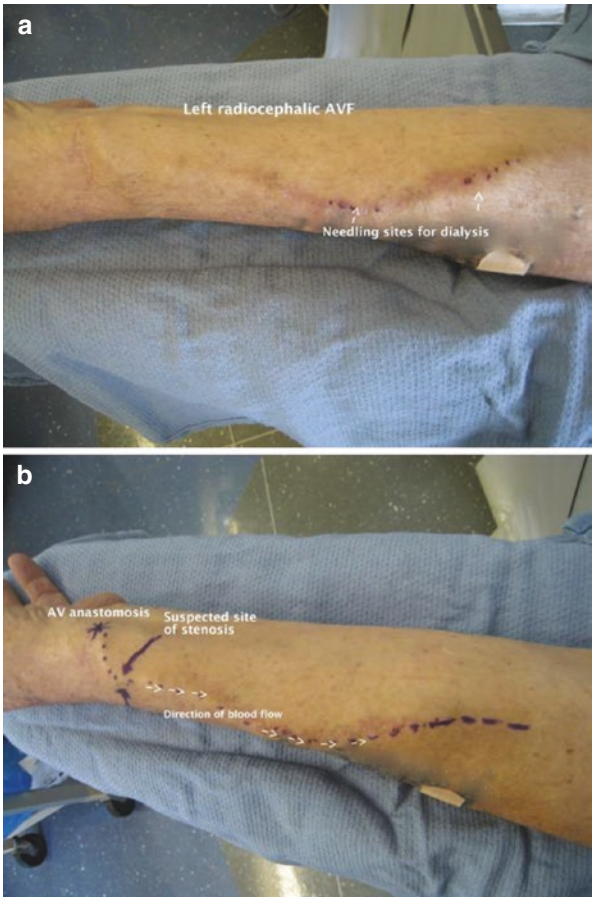

Fig. 11.3 (**a**) Physical examination of the AVF. (**b**) Identify the area of stenosis

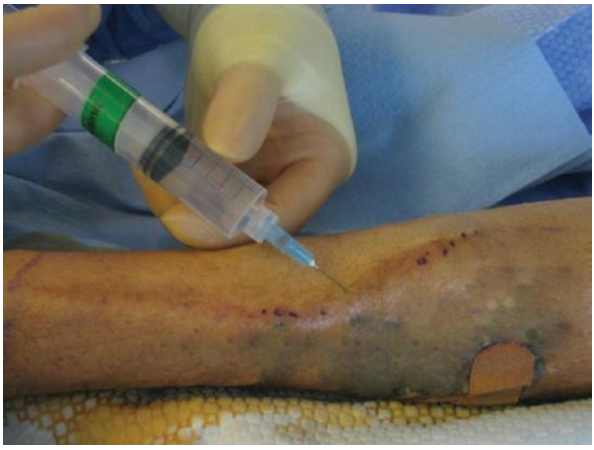

Fig. 11.4 Anesthetize the skin

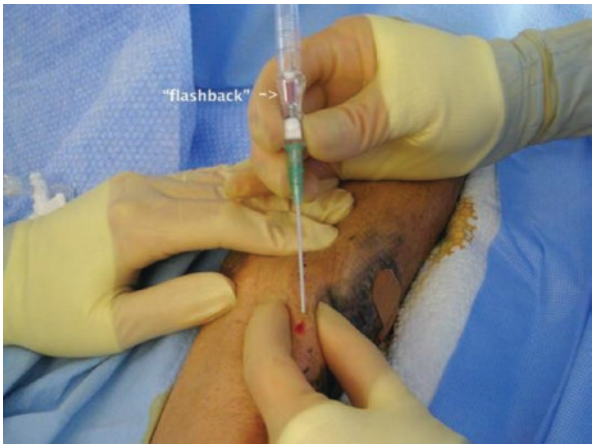

Fig. 11.5 Push in the IV catheter till "flashback" is seen

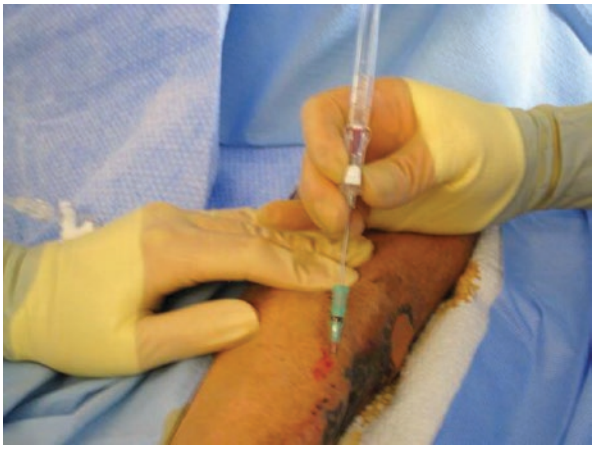

Fig. 11.6 Remove the needle and push in the cannula

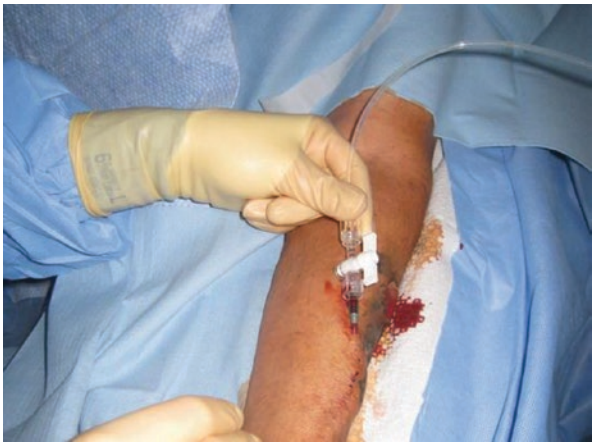

Fig. 11.7 Inject contrast via the cannula

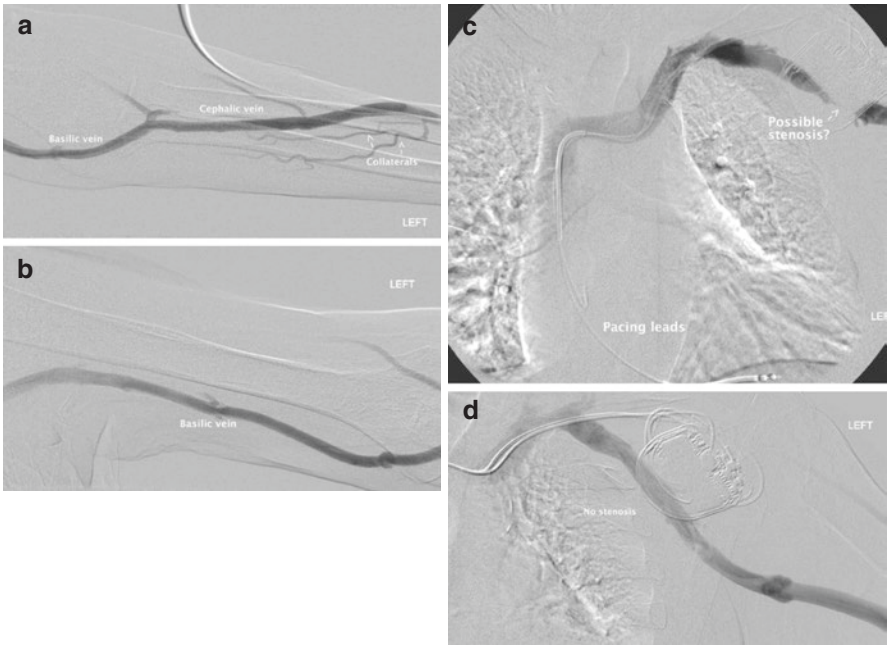

Fig. 11.8 (**a**) Free flow of contrast within the AVF. (**b**) No evidence of stenosis in the basilic vein. (**c**) Possible stenosis seen during imaging of the central veins. (**d**) No stenosis was seen after adjusting the imaging angle

13. Insert a 0.035 in. wire into the 18G cannula. Remove the cannula and insert a 6 Fr sheath over the guidewire (Fig. 11.10a–d).
14. Manipulate the guidewire tip across the JA stenosis into the feeding artery with the help of a Kumpe catheter under fluoroscopy (Fig. 11.11a, b).
15. After crossing the stenosis, remove the guidewire and inject the contrast material via the Kumpe catheter to visualize the site of stenosis (Fig. 11.11c).
16. After confirming the position, reinsert the guidewire and remove the Kumpe catheter.
17. Review the images to decide on the size of angioplasty balloon.
18. Prime the balloon catheter by aspirating back on the inflation port with a balloon inflation device.
19. Insert the angioplasty balloon catheter over the guidewire. The inflatable portion of the balloon catheter is marked with radio-opaque markers. Position the balloon across the lesion (Fig. 11.12a, b).
20. Balloon inflation (Fig. 11.12c–f)
    (a) A balloon inflation device is often used for this purpose.
    (b) The pressure rating for each angioplasty balloon is listed on the package label. This pressure rating indicates the amount of pressure that the balloon will tolerate before rupture.

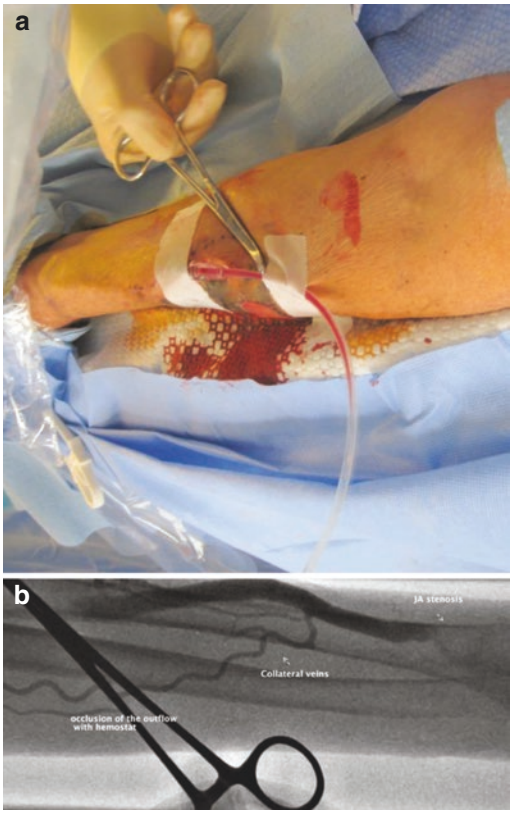

Fig. 11.9 (**a**) Compress the AVF with a hemostat to "reflux" the contrast material to demonstrate the inflow. (**b**) Poor flow of contrast material, demonstrating stenosis of the juxta-anastomotic area (JA)

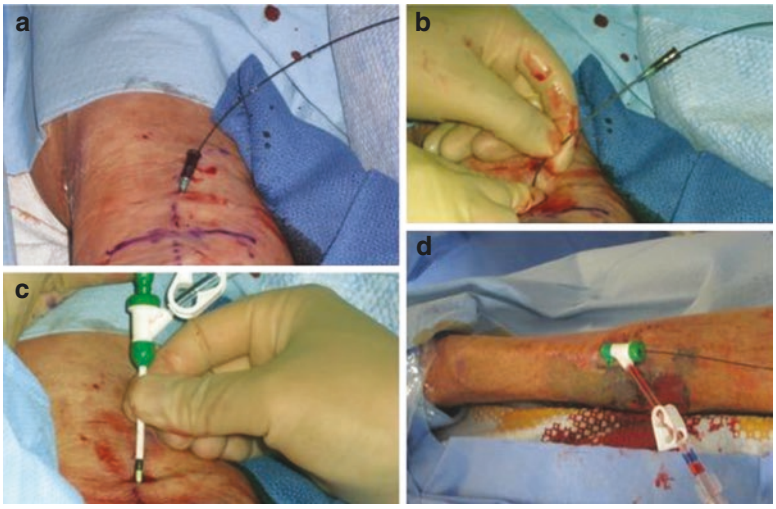

Fig. 11.10 (**a**) Insert the guidewire into the cannula. (**b**) Remove the cannula while pressing on the venotomy site. (**c**) Insert the sheath via the guidewire. (**d**) Sheath is placed over the guidewire

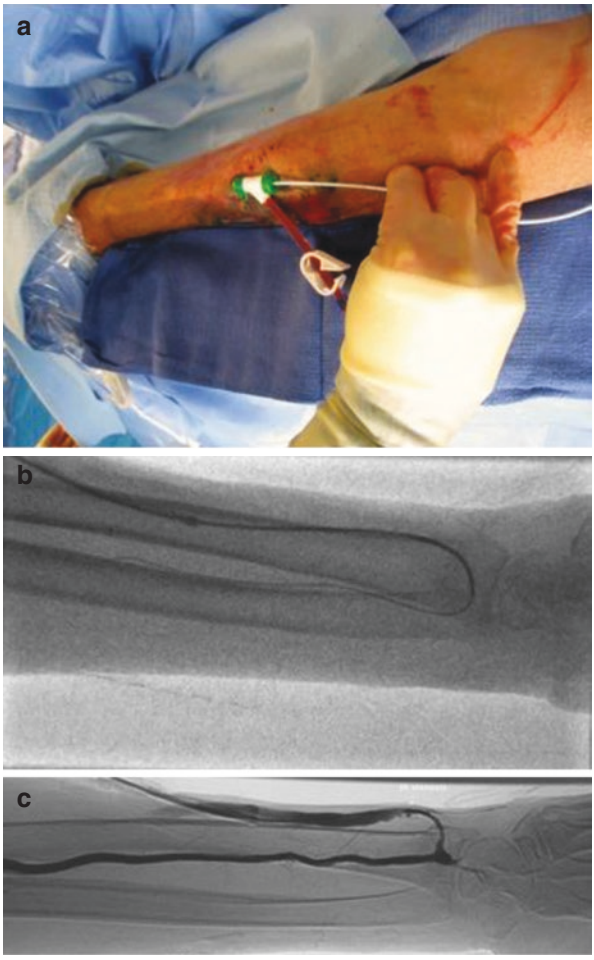

Fig. 11.11 (**a**) Insert Kumpe catheter over the guidewire. (**b**) Steer the guidewire across the stenosis into the feeding artery with the help of the Kumpe catheter. (**c**) After removing the guidewire, inject contrast material through the Kumpe catheter to assess the stenotic area well

(c) Within the balloon inflator, contrast material is used to permit the visualization of the balloon during inflation and deflation.
(d) Inflate the balloon while maintaining traction on the shaft of the balloon catheter to prevent it from slipping off the lesion. Inflation is stopped when the lesion is completely effaced or when the pressure rating is reached. Keep the balloon inflated for at least 3 min.
(e) Deflate the balloon completely and move it to the next downstream lesion if any. If not, remove the balloon catheter but leave the guidewire in situ.

21. Reinsert the Kumpe catheter over the guidewire then remove the guidewire.
22. Inject contrast material through the Kumpe catheter to determine the effectiveness of treatment and presence of any complications such as extravasation (Fig. 11.12g).

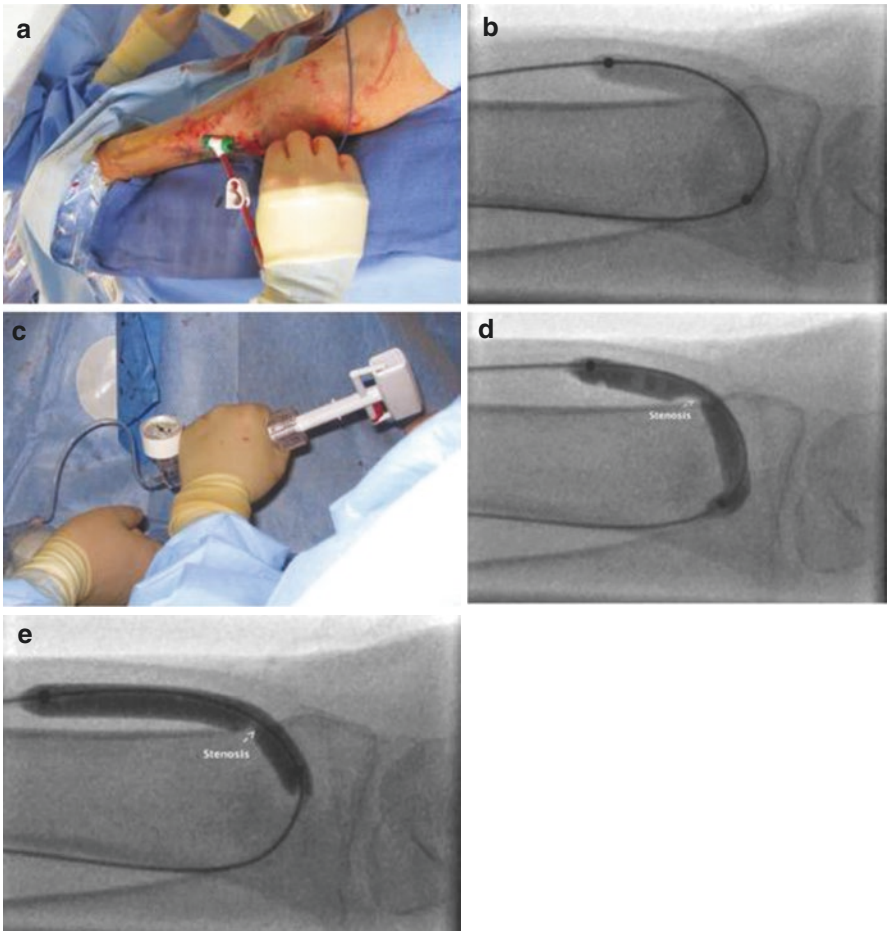

Fig. 11.12 (**a**) Insert the balloon catheter over the guidewire. (**b**) Position the balloon catheter across the stenosis and inflate the balloon. (**c**) Inflate the balloon using an inflator. (**d**) The stenotic lesion is demonstrated during balloon inflation. (**e**) Repositioning of the balloon may be necessary. (**f**) Complete effacement of the stenosis. (**g**) Fistulogram showed a decrease in severity of stenosis

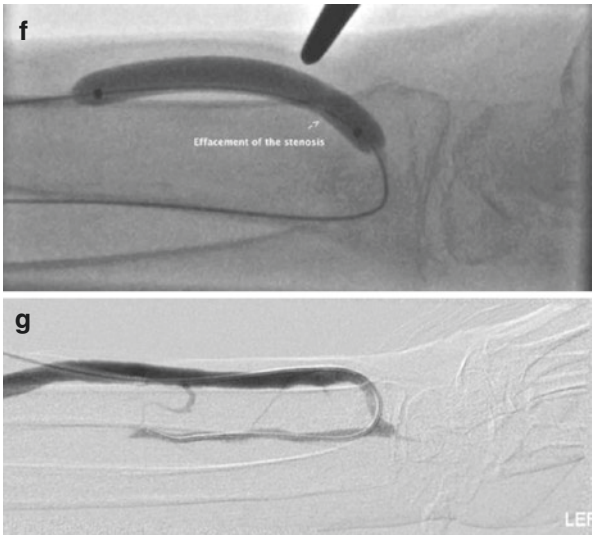

Fig. 11.12 (continued)

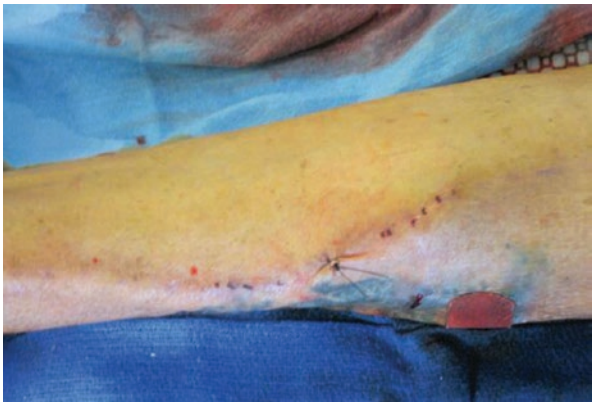

Fig. 11.13 Final appearance of the AVF after completion of the procedure

23. Once all the lesions are treated, remove the Kumpe catheter and examine the AVF for thrill and the hand for any complications.
24. Apply a purse string suture around the sheath and tighten it immediately after pulling out the sheath (Fig. 11.13).

Graftogram and Angioplasty

The procedure for a graftogram is similar to that of a fistulogram. In comparison to an AVF, the stenotic lesion is usually in the outflow. Before puncturing the graft, it is important to find out the direction of blood flow and cannulate the graft in the

correct direction. To determine the inflow and outflow of the AV graft, temporarily occlude the midsection of the graft with your fingers. The inflow should become pulsatile after occlusion while the "thrill" should disappear in the outflow segment of the AV graft.

1. Examine the AVG to identify the site of stenosis (Fig. 11.14a, b).
2. Clean and drape the patient.
3. Anesthetize the skin.
4. Cannulate the AVG with an 18G intravenous catheter and direct it towards the suspected site of stenosis. In this case example, the suspected site of stenosis is within the central veins. Hence, the catheter is placed in an antegrade direction, towards the central veins (Fig. 11.14c).
5. Do a graftogram to delineate the site of stenosis.
6. Check the outflow and central veins (Fig. 11.15a, b).
7. Insert the guidewire into the intravenous cannula. Exchange the cannula of the intravenous catheter for a sheath over the guidewire (Fig. 11.16).
8. Navigate the wire tip across the stenosis into the inferior vena cava. A Kumpe catheter may be used to steer the wire into the inferior vena cava if needed (Fig. 11.17a).

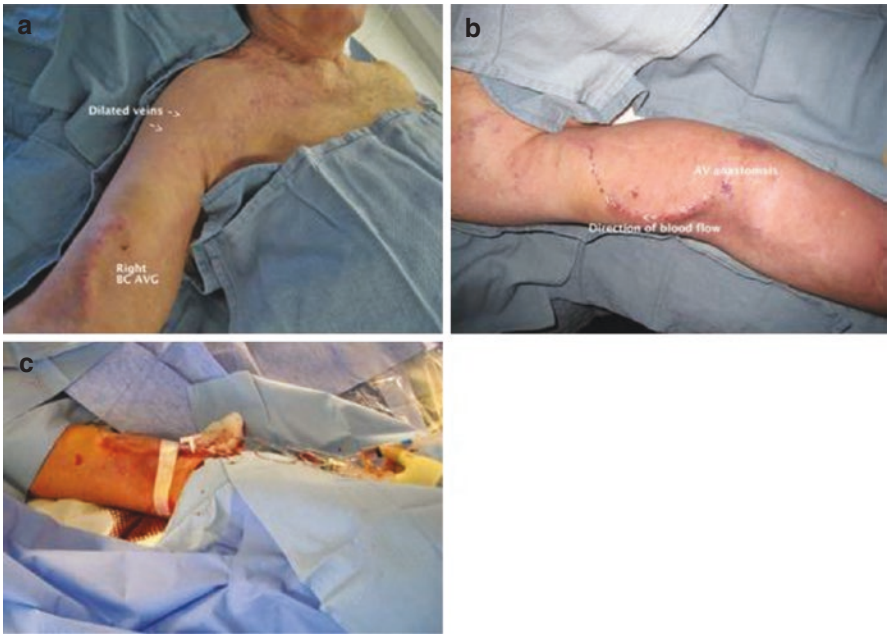

Fig. 11.14 (**a**) Examine the AVG. The presence of dilated arm and chest veins suggest the presence of a central vein stenosis. (**b**) Note the direction of blood flow and plan the site of sheath placement. (**c**) Do an initial graftogram using a 18G cannula

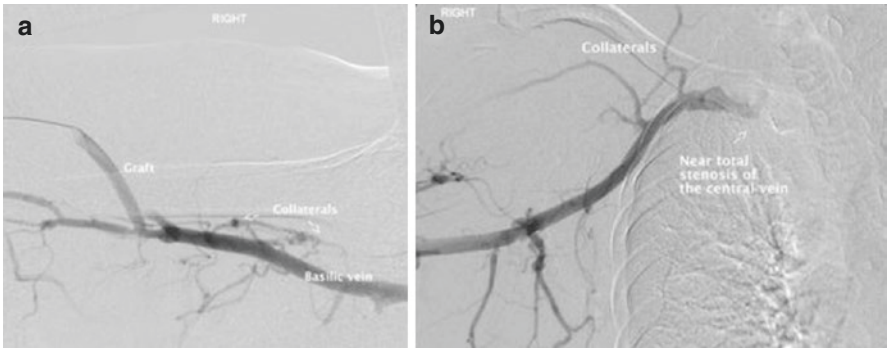

Fig. 11.15 (**a**) Initial graftogram showed multiple collaterals, suggestive of outflow stenosis. (**b**) Central venogram showed near total stenosis of the central vein

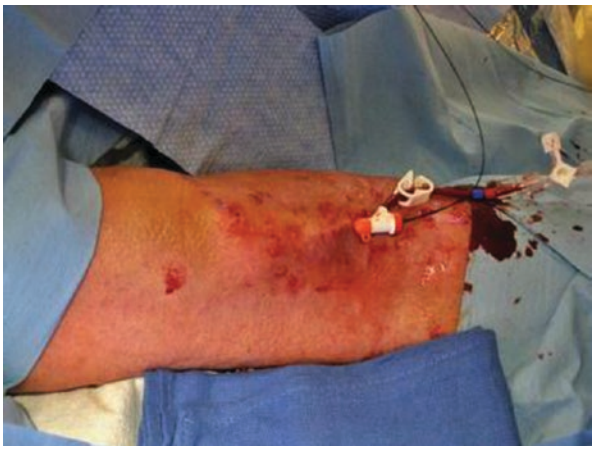

Fig. 11.16 Exchange the cannula for a sheath over the guidewire

9. Insert the angioplasty balloon to the site of stenosis and inflate the balloon to treat the stenosis (Fig. 11.17b–d).
10. Assess the outcome of angioplasty by doing a DSA run (Fig. 11.17e).
11. Check the inflow of the graft by doing a "reflux" angiogram by injecting contrast material via the sidearm of the vascular sheath while occluding the outflow of the graft with either the angioplasty balloon or by manual compression with a hemostat.
12. Remove the guidewire.
13. Apply purse string suture and remove the sheath (Fig. 11.18a, b).

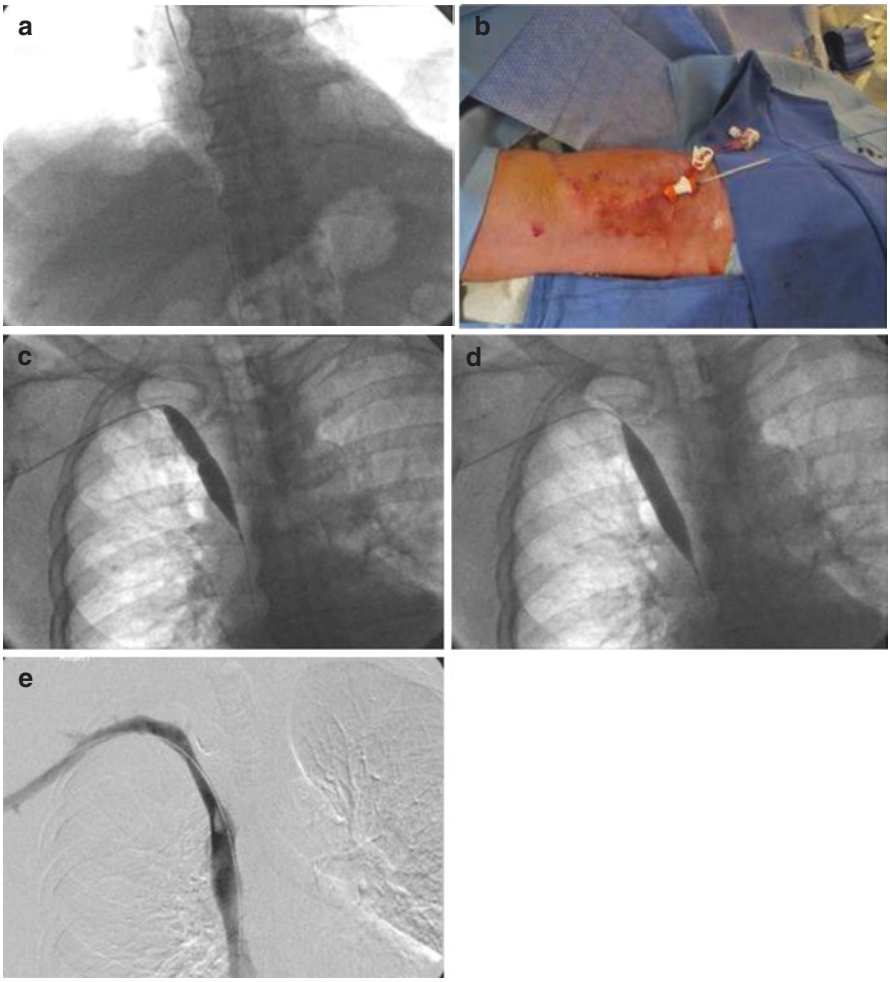

Fig. 11.17 (**a**) Steer the guidewire tip across the stenosis into the inferior vena cava. (**b**) Insert the balloon catheter over guidewire. (**c**) Inflate the balloon. (**d**) Complete effacement of the stenosis with balloon inflation. (**e**) Improvement in flow after balloon angioplasty

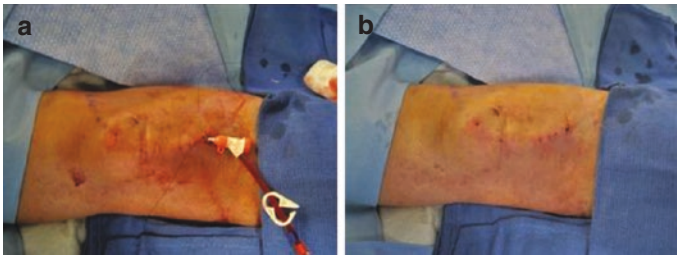

Fig. 11.18 (**a**) Apply a purse string suture around the sheath. (**b**) Remove the sheath and tighten the purse string suture

Tips and Troubleshooting

Some of the common problems and complications that you may come across while performing a fistulogram and angioplasty are covered in Table 11.3.

Table 11.3 Common problems and complications

Problem	Suggestions
Inability to cannulate AV access	1. Occlude the venous outflow to engorge the veins
	2. Cannulate the AV access under real-time ultrasound guidance
	3. Find an alternative site which may be remote from dialysis cannulation sites
Inability to pass guidewire through a stenosis (Fig. 11.19)	1. Use an angled catheter to "stiffen" the wire and rotate the wire across the stenosis
	2. Use a curved tip guidewire or modify the curvature of the wire tip by running a hemostat over the tip while holding the guidewire between the hemostat and thumb
	3. Change to a smaller caliber guidewire
	4. Apply external compression to the site of stenosis using a hemostat to change the configuration of the stenosis
Inability to advance the balloon catheter over the guidewire at the site of stenosis	1. Change to a smaller angioplasty balloon to pre-dilate the lesions before using a larger balloon
	2. Use a balloon catheter that has a better trackability
	3. Wedge the balloon catheter into the lesion and gently inflate it repeatedly to dilate the lesion till it passes through
"Watermelon seed" effect: Balloon repeatedly slipping off site of stenosis during inflation. (Fig. 11.20)	1. Hold onto the balloon catheter during inflation
	2. Reposition the balloon catheter to "center" it at the site of stenosis
	3. Use a longer balloon to cover the length of stenosis
	4. Use a smaller balloon to pre-dilate the stenosis
Persistent "waist" at maximal pressure inflation (Fig. 11.21)	1. Change to an ultra high pressure balloon
	2. Use a scoring balloon, e.g. AngioSculpt (AngioScore, Fremont, CA)
	3. Use a cutting balloon, e.g. peripheral cutting balloon (Boston Scientific, Natick, MA)
	4. Extend balloon inflation time
"Recoil": Lesions effaced during pressure inflation but recur after deflation of balloon (Fig. 11.22)	1. Exclude external compression
	2. Reassess hemodynamic significance. If intervention is necessary,
	(a) Prolong the balloon inflation time, or
	(b) Use larger balloon, or
	(c) Use an ultra high pressure balloon, or
	(d) Use an angioscoring balloon, or
	(e) Use a cutting balloon, or
	(f) Consider using a stent

(continued)

Table 11.3 (continued)

Problem	Suggestions
Venous rupture (Fig. 11.23)	1. Control the extravasation by compressing the inflow of the AV access
	2. It is critical to maintain the wire across the site of rupture at all time
	3. Reposition the balloon to the rupture site and gently re-inflate the angioplasty balloon to a lower pressure to tamponade the bleed for 3–5 min
	4. To prevent thrombosis of the AV access during the prolonged tamponade, periodically deflate the balloon for a few seconds to allow flow to continue
	5. Most of the bleeding will stop. If persistent, deploy a stent graft across the ruptured segment
	6. If stent deployment is necessary, the sheath would need to be upsized to allow the passage of the stent
Rupture of angioplasty balloon	1. Exclude venous rupture which may have occurred concurrently
	2. Gently withdraw the balloon through the sheath to prevent further fragmentation of balloon material
	3. If balloon is stuck while pulling it out from the sheath, apply a purse string suture around the sheath and remove the sheath together with the balloon
	4. After removal, check the balloon to ensure that all fragments are removed
Wire fragments or foreign body is inadvertently created during procedure	1. Remove the fragments with a snare device (EN snare or goose neck snare)
Sheath is dislodged during procedure	1. Re-insert the inner dilator into the sheath before replacing the sheath, do not push in the sheath without the inner dilator in place
Hematoma after sheath removal	1. Tends to occur more frequently in non matured AVFs
	2. Compress the puncture site
	3. Redo the purse string

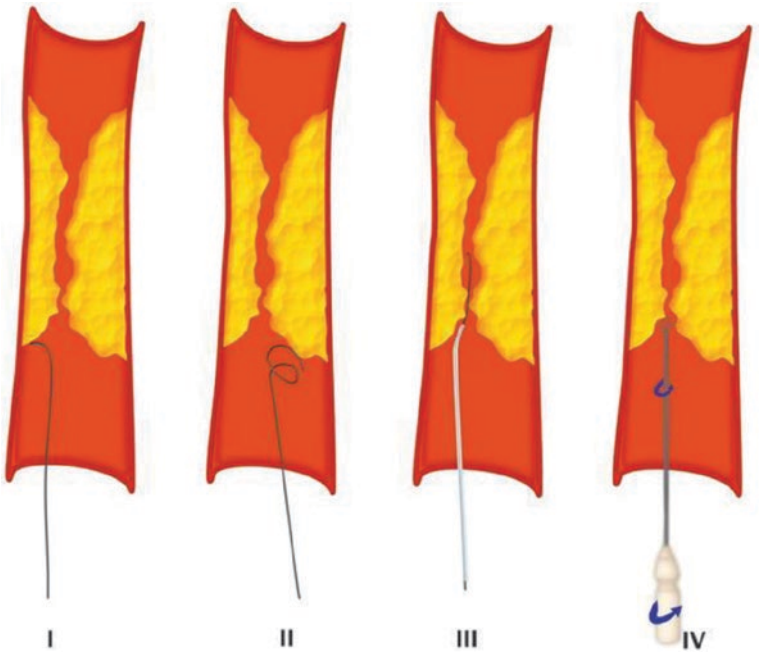

Fig. 11.19 Difficulties may be encountered while trying to cross a stenotic lesion. The wire may tangle up when you try pushing the wire forward (**I**, **II**). An angled catheter may be passed over the wire to stiffen and rotate the tip of the wire across the lesion (**III**). A torque device may also be attached to the wire to "spin" it across the lesion (**IV**)

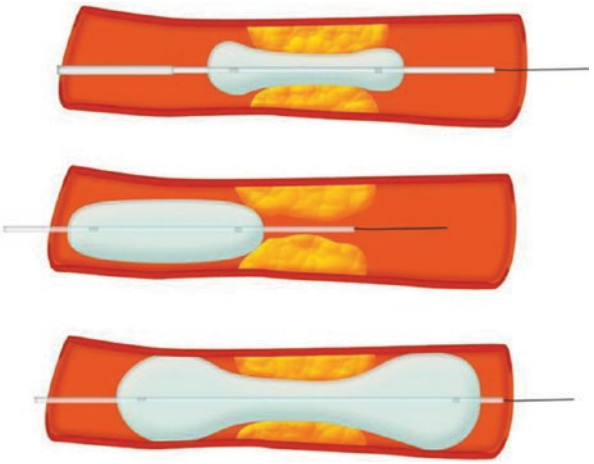

Fig. 11.20 The angioplasty balloon may slip off the site of stenosis during inflation. This can be managed by repositioning the balloon and holding on to the balloon catheter (near the sheath) during inflation to keep it in position. A longer balloon may be needed if the stenosis is long

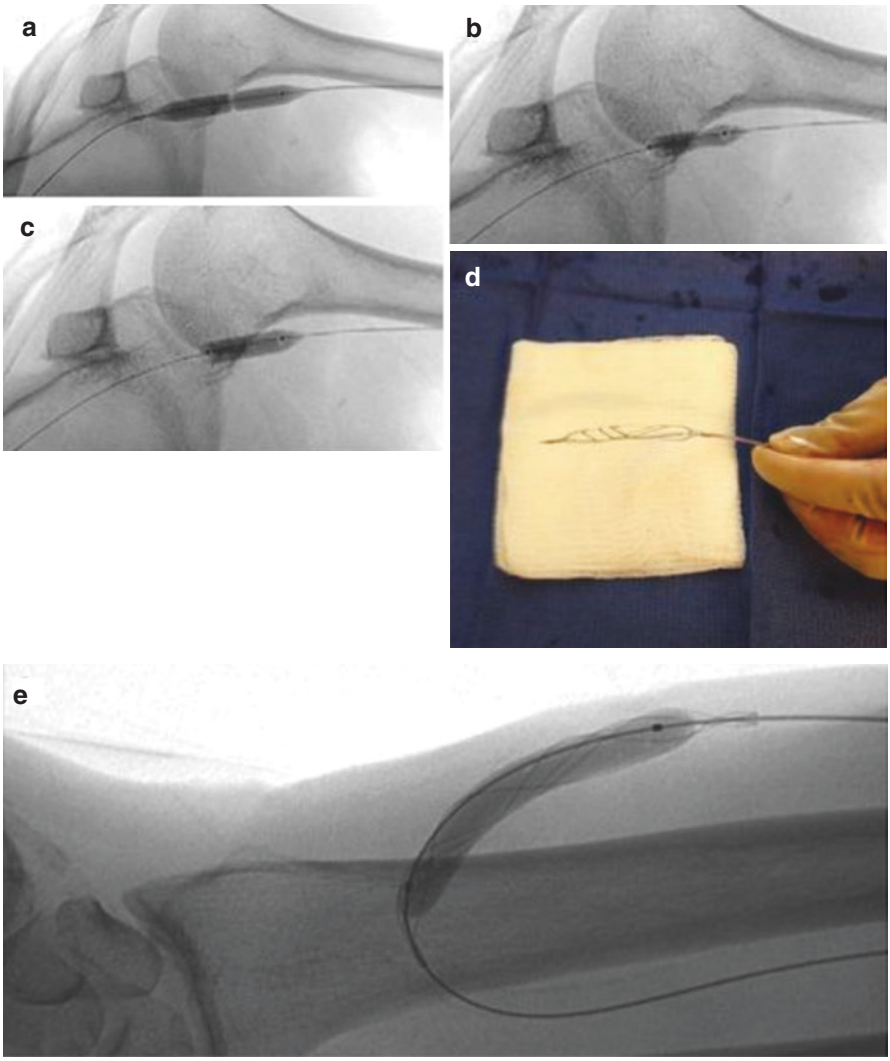

Fig. 11.21 (**a**) Persistent "waisting" despite using a high pressure balloon at maximal balloon inflation. (**b**) The same lesion treated with a cutting balloon. (**c**) Effacement of the stenosis with cutting balloon. (**d**) An Angiosculpt balloon has three or four flexible nitinol spiral struts attached to a semi-compliant balloon. (**e**) Inflation of the balloon will create a radial force to push the edges of the nitinol struts into the target lesion to "score" it

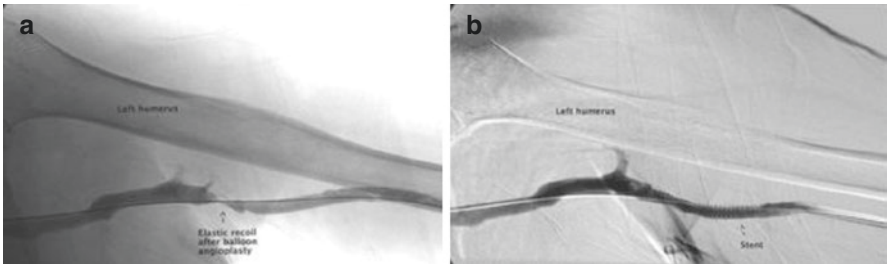

Fig. 11.22 (**a**) Stenotic lesion at the graft vein junction "recoiled" after balloon angioplasty. (**b**) A stent was placed to maintain the patency of the AVG

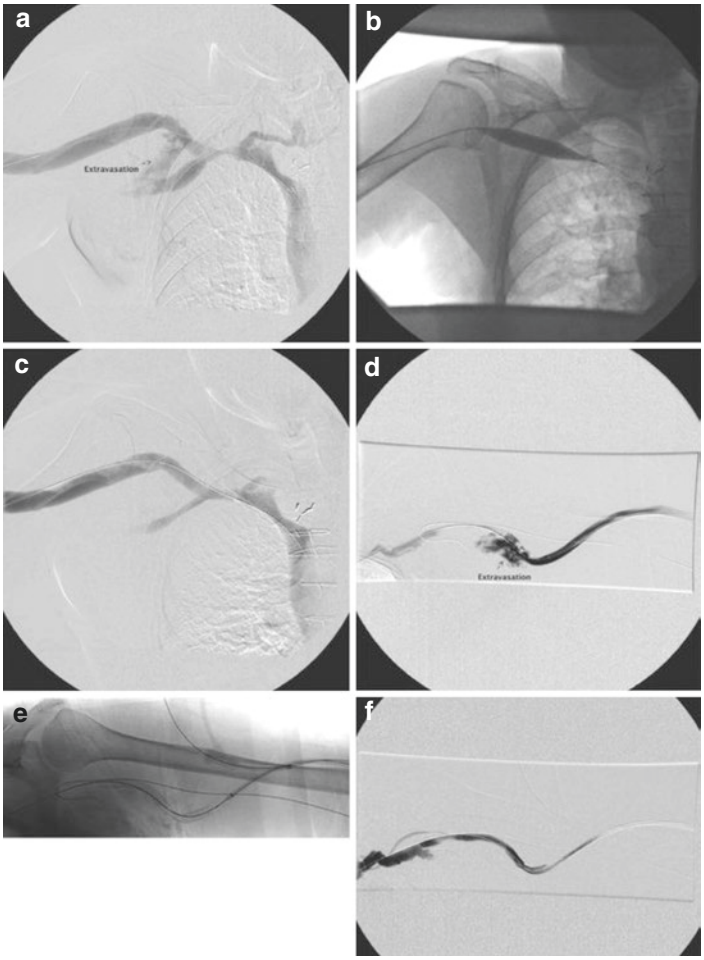

Fig. 11.23 (**a**) Extravasation of contrast after angioplasty of the cephalic arch. (**b**) The angioplasty balloon was gently re-inflated at lower pressure to tamponade the site of bleeding for 3–5 min. (**c**) Extravasation of contrast material stopped. (**d**) Venous rupture after balloon angioplasty of the graft vein junction of a left upper arm BB AVG. (**e**) Covered stent was deployed to control the bleeding. (**f**) Extravasation of contrast material stopped after stent deployment

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Declotting of Dialysis Vascular Access

12

Ru Yu Tan, Chieh Suai Tan, Steven Wu, and Sanjeeva Kalva

Introduction

Thrombosis of arteriovenous (AV) access can be considered a renal "emergency" that requires urgent intervention. Percutaneous declotting of AV access is minimally invasive, and well tolerated by most patients. It can be easily arranged and performed in an outpatient setting. This renders it the preferred technique for salvaging thrombosed AV access compared to traditional open surgical thrombectomy. With the advent of interventional nephrology as a subspecialty, declotting of the AV access can now be performed swiftly with minimal disruption to the dialysis schedule of the patients.

Thrombosis occurs due to stasis of blood secondary to flow-restricting stenoses within the circuit. Therefore, treatment of a thrombosed AV access requires thrombolysis and angioplasty of the underlying stenosis. In arteriovenous grafts (AVG), the sites of stenosis are most commonly at the graft-vein junction or in the draining vein. On the contrary, the site of stenosis may be within an outflow vein for an upper arm arteriovenous fistula (AVF) or the inflow vessels for a lower arm AVF [1, 2].

Although similar techniques are employed for declotting of AVF and AVG, there are some subtle differences in the approach and etiology. In particular, declotting of

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a thrombosed fistula requires more time and expertise than declotting of a graft. The differences are as summarized in Table 12.1.

Methods of Declotting

Broadly, the two methods of declotting as summarized in Fig. 12.1. The differentiation is arbitrary as often a combination of both pharmacological and mechanical thrombolysis are needed to ensure technical success.

Table 12.1 Differences between declotting of an AVF and AVG

	AVF	AVG
Configuration	Native vein to artery	Vein to synthetic material to artery
Flow requirement for patency	Usually remain patent at low blood flow	Likely to thrombose at low blood flow
Incidence of thrombosis	Low	High
Etiology	Inflow stenosis more than outflow stenosis	Graft vein junction stenosis
Urgency of intervention	High (within hours to few days)	Lower than AVF (within days to weeks)
Clot burden	Variable, usually low but can be high in the presence of aneurysm	Moderate, usually confined to the graft
Technical difficulties	High. May be difficult to cannulate the main outflow vein	Low. Graft is visible and can be cannulated easily
Success rate	Lower than AVG	Higher than AVF

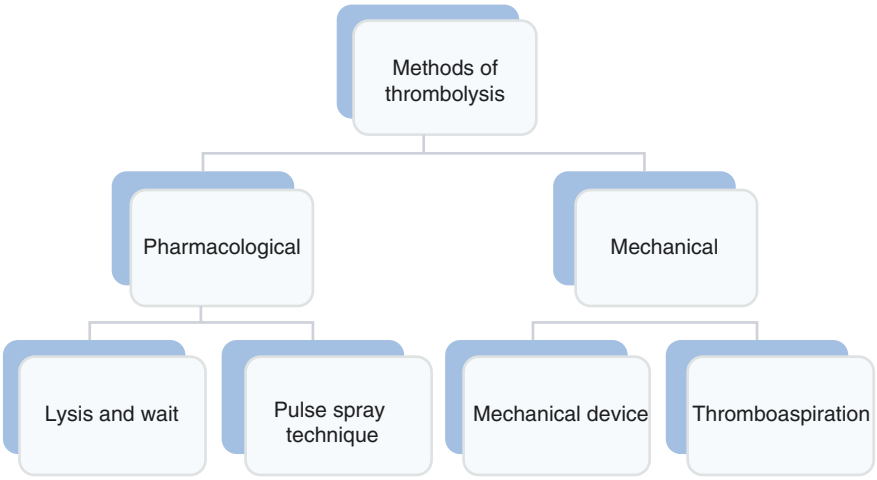

Fig. 12.1 Methods of thrombolysis

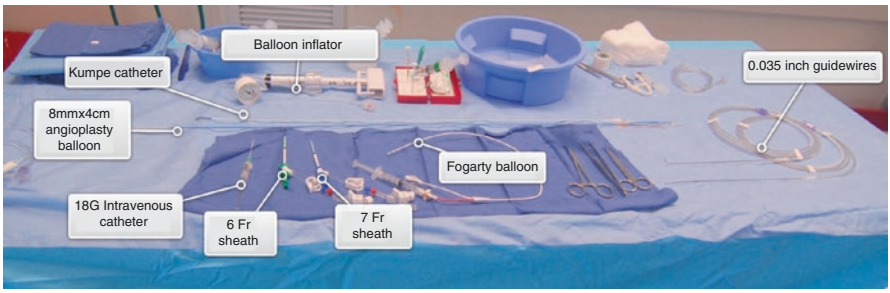

Fig. 12.2 Equipment for declotting

Equipment (Fig. 12.2)

1. Normal saline for flushing of sheaths and catheters
2. Lidocaine
3. Two 18 Gauge (G) intravenous catheters
4. Two 0.035 in. guidewires
5. One 6 Fr 4 cm sheath
6. One 7 Fr 4 cm sheath
7. One Kumpe catheter
8. One Angioplasty balloon
9. One 5.5 Fr Fogarty balloon
10. One balloon inflator
11. 4 mg of tissue plasma activator (tPA) in 4 mL of sterile water
12. 2000 units of heparin (1000 units/mL concentration)
13. Two 3/0 non absorbable sutures (Ethilon)
14. Gauze
15. Hemostat
16. Needle holder
17. Syringes and needles

Declotting of AVG

1. Two puncture sites are required to access the entire circuit to remove all the clots and the "arterial plug" (Fig. 12.3a).
2. The first puncture should be placed close to the arterial anastomotic site in an antegrade direction towards the venous outflow. The direction of blood flow can be determined by:
   (a) Physical examination: Palpate for arterial pulsations along the graft. It should get more distinct as you approach the AV anastomosis.
   (b) History: Enquire about the placement of needles during dialysis. The "A" needle is directed towards the inflow while the "V" needle is directed towards the outflow.

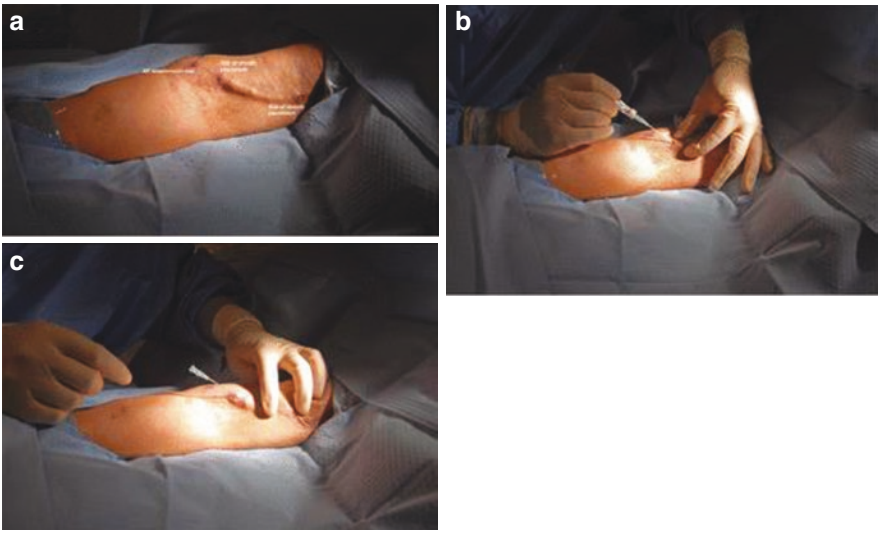

Fig. 12.3 (**a**) Note the AV anastomotic site and mark the site of sheath placement. (**b**) Hold the IV cannula like a pencil with your right hand and advance it gently while feeling for a "give". (**c**) Stale blood should be seen in the cannula if it is in the right position

(c) Surgical notes.
(d) Images from previous access intervention.

3. Cannulate the AVG with an 18G angiocath (Fig. 12.3b, c).
   (a) Advance the needle till you feel a "give". "Flashback" is usually not seen as the blood is clotted within the AVG.
   (b) Hold onto the needle while pushing in the cannula.
4. Gently instill 4 mg of tPA into the thrombosed AVF via the cannula (Fig. 12.4a).
5. Insert a 0.035 in. wire into the 18G cannula (Fig. 12.4b) and push the tip of the wire up to the subclavian vein.
6. Remove the cannula over the guidewire (Fig. 12.4c) and thread a 4 cm long 6 Fr sheath along with its dilator over the guidewire to insert the sheath into the cannulation site. Remove the dilator from the sheath (Fig. 12.4d).
7. Pass a 5 Fr Kumpe catheter up to the level of the thoracic cage. Remove the guidewire and perform a venogram to check the central veins via the Kumpe catheter (Fig. 12.5a).
8. Pull back the catheter towards the AVF while injecting the contrast to image the draining veins (Fig. 12.5b, c).
9. Treat significant stenotic lesions with balloon angioplasty: Reinsert the guidewire over the Kumpe catheter, then remove the Kumpe catheter and insert the balloon catheter over the guidewire (Fig. 12.6a–c).
10. After treatment of the stenotic segment, the next step is to declot the AVG mechanically.
11. Pull back the deflated angioplasty balloon into the thrombosed AVG and inflate the balloon (Fig. 12.7a). The aim is to "macerate" the thrombus within the

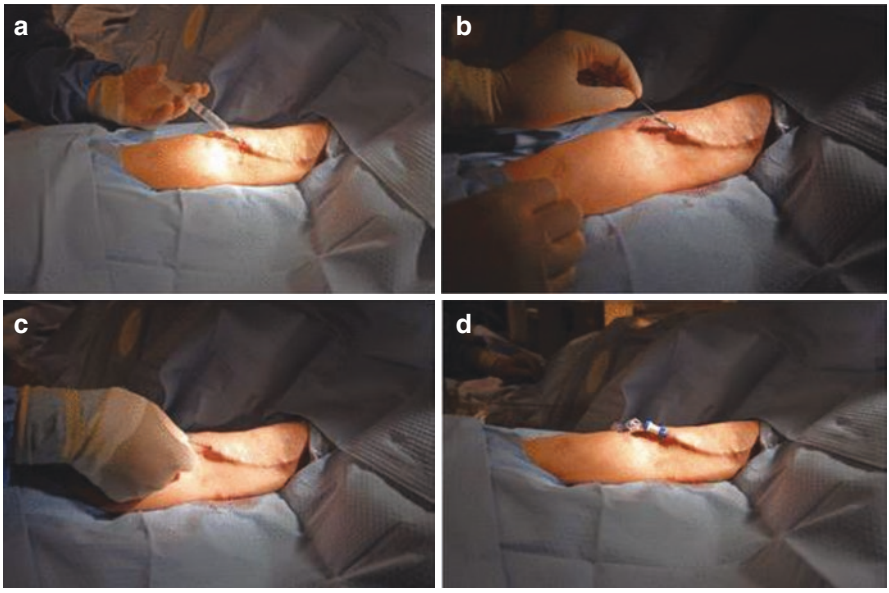

Fig. 12.4 (**a**) Instill tPA into the graft via the cannula. (**b**) Pass the guidewire into the graft via the cannula. (**c**) Remove the cannula from the graft but leave the wire in situ. (**d**) Insert the sheath into the AVG over the guidewire

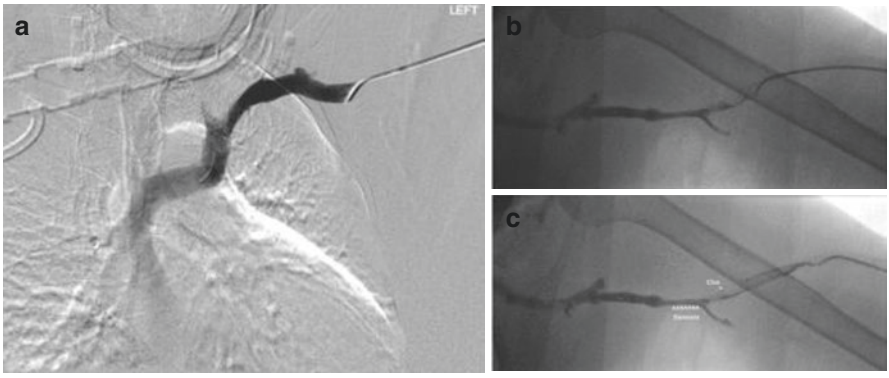

Fig. 12.5 (**a**) Do a central venogram with the catheter placed just outside the thoracic cage. (**b**) Inject the contrast material while pulling the catheter towards the sheath. (**c**) There is a stenosis at the graft vein junction and a thrombus within graft

AVG. There is no need to keep the balloon inflated for 3 min. Deflate the balloon and move it towards the sheath before inflating it again. Repeat these till you reach the tip of the sheath (Fig. 12.7b, c).

12. Aspirate clots from the side-port of the sheath and perform a graftogram to check the results of thrombolysis (Fig. 12.7d–f). If necessary, repeat the step 11 and 12.

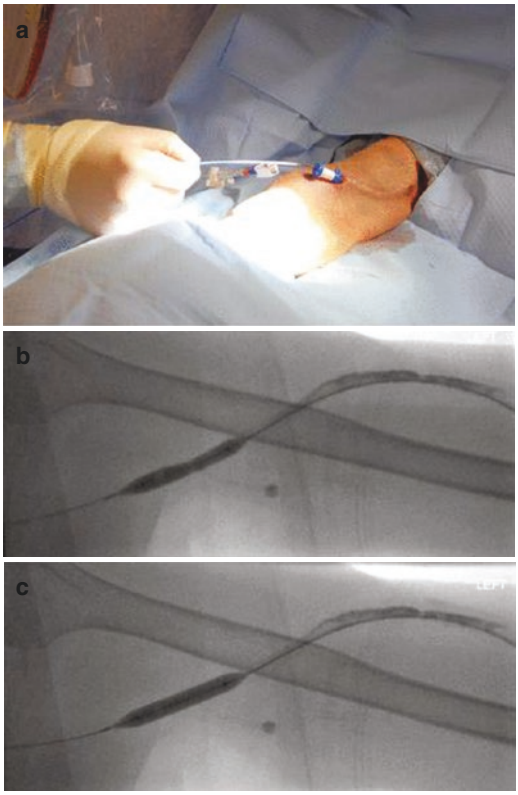

Fig. 12.6 (**a**) Insert the angioplasty balloon catheter over the guidewire. (**b**) Stenosis causing "waisting" of the balloon during inflation. (**c**) Complete effacement of stenosis at maximal balloon inflation

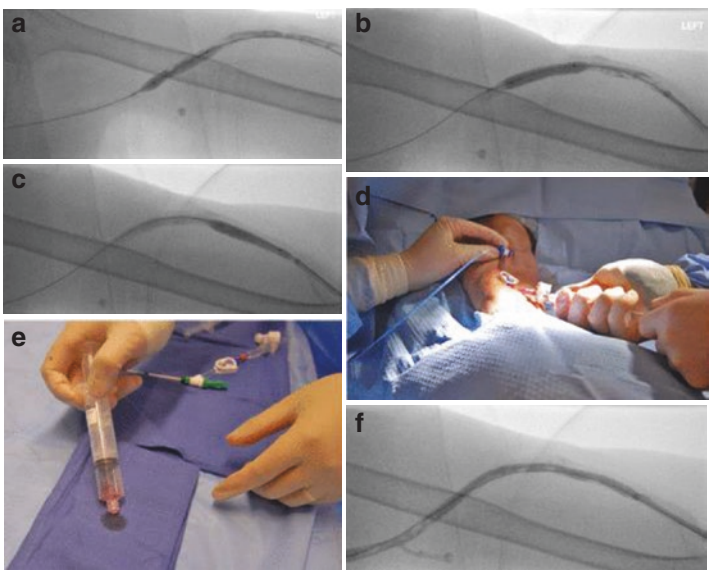

Fig. 12.7 (**a**) Inflate the angioplasty balloon to macerate the thrombus. (**b**) Deflate, pull back and re-inflate the balloon. (**c**) Repeat the steps still you reach the tip of the sheath. (**d**) Aspirate from the side port of the sheath. (**e**) Look for the presence of clots that are aspirated. (**f**) Graftogram to check the result of thrombolysis

13. Once the venous outflow is cleared, make a second puncture on the venous limb and direct it in a retrograde direction towards the arterial inflow (Fig. 12.8a–c). Ensure there is sufficient distance between the first and second puncture for sheath placement so that the two sheaths do not overlap. Instill 2000 units of heparin intravenously.
14. Gently pass the guidewire tip into the feeding artery (Fig. 12.9a). Do not use excessive force as it might push the "arterial plug" or thrombus into the artery and result in distal embolization.
15. Insert a 5.5 Fr Fogarty balloon catheter over the guidewire into the artery. Inflate the Fogarty balloon with 1.5 mL of diluted contrast (Fig. 12.9b–d). Pull back the balloon towards the sheath to dislodge the arterial plug (Fig. 12.9e, f). Repeated attempts may be needed to clear the arterial plug.
16. Perform a check angiogram to ensure adequacy of thrombolysis (Fig. 12.10a).
17. A retrograde graftogram can be performed by compressing the graft while injecting contrast via the sidearm of first sheath (Fig. 12.10b).
18. Do a final run of the outflow veins. Any significant stenosis that is discovered after thrombolysis should be treated with balloon angioplasty.
19. Apply purse-string suture around the sheath (Fig. 12.10c).
20. Pull out the sheath and immediately tighten the purse-string suture (Fig. 12.10d, e).
21. Apply dressing over the wound.
22. Check the distal pulses.
23. Watch out for any hematoma formation.
24. Stitches can be removed after 1–2 days.

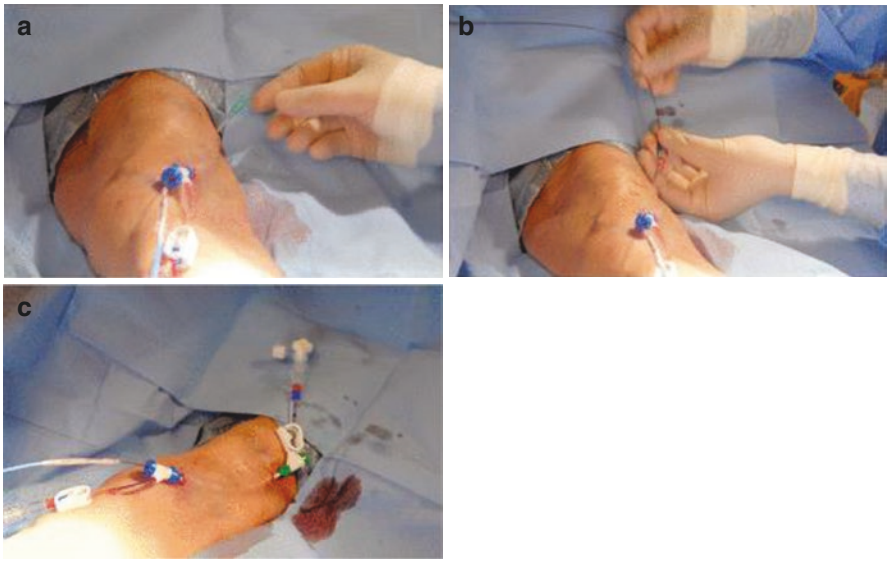

Fig. 12.8 (**a**) Cannulate the AVG using the IV cannula as described previously. (**b**) Insert the wire through the cannula. (**c**) Remove the cannula and insert the sheath into the graft over the guidewire

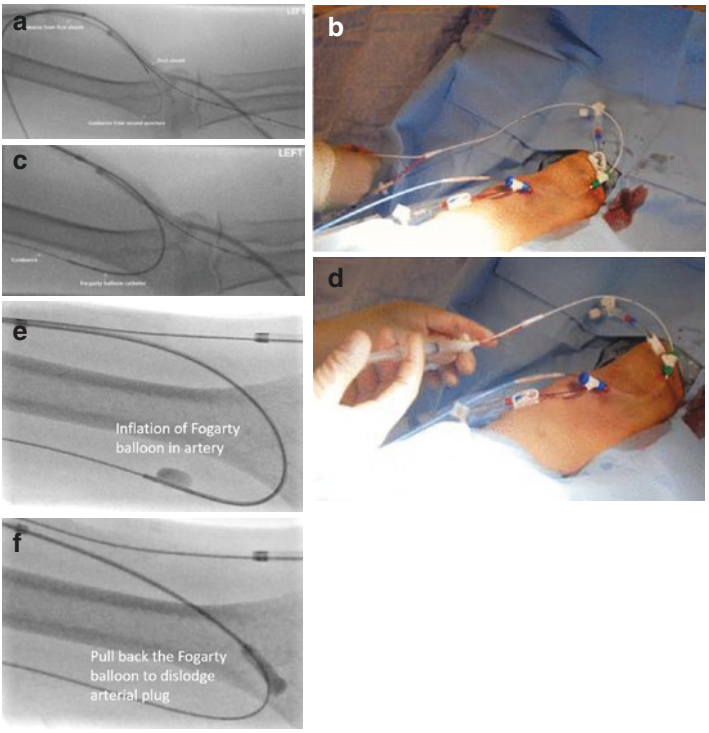

Fig. 12.9 (**a**) Insertion of guidewire into the feeding artery. (**b**) Insert the Fogarty balloon catheter over the guidewire. (**c**) Ensure that the guidewire is within the feeding artery. Insert the Fogarty balloon catheter into the feeding artery under fluoroscopy. (**d**) Inflate the Fogarty balloon with 1.5 mL of diluted contrast. (**e**) Inflate the Fogarty balloon in the feeding artery. (**f**) Pull back the inflated balloon catheter towards the sheath to dislodge the "arterial plug"

Declotting of AVF

The steps for thrombolysis in an AVF are similar to that of an AVG. However, compared to the AVG, declotting a thrombosed AVF can be more challenging for the following reasons:

1. Greater variation of the venous anatomy. Specifically, the presence of accessory or collateral veins can confound the placement of the initial puncture. The clotted fistula may be draining via the collateral veins which may confuse the operator. It is crucial to get access into the main draining vein to successfully declot it.
2. The stenosis can be much tighter and impossible to cross.
3. The venous wall can be much thinner than the synthetic graft and is much more difficult to feel and cannulate.
4. Higher risk of rupture, especially in recently created AVF that has clotted off.

Tips and Troubleshooting

Some of the common problems and complications that you may meet while performing a declotting procedure are covered in Table 12.2.

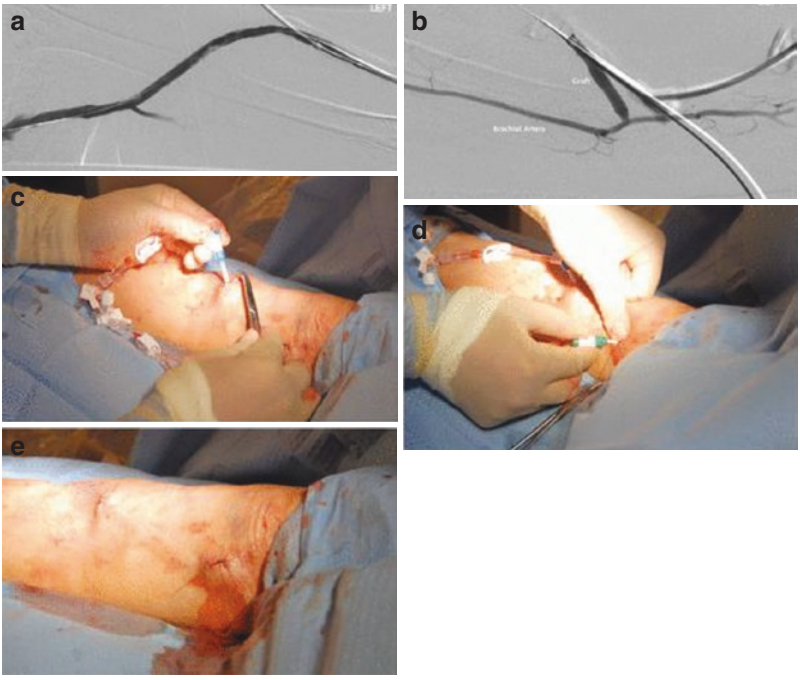

Fig. 12.10 (**a**) Check adequacy of thrombolysis. Note the free flow of contrast through the graft. (**b**) Retrograde graftogram to visualize the inflow of the AVG. (**c**) Apply purse-string suture around the sheath. (**d**) Tighten the purse-string while pulling out the sheath. (**e**) AVG after sheath removal

Table 12.2 Common problems and complications

Problem	Trouble shooting
Venous embolism	In the presence of high thrombus load, significant pulmonary embolism can occur. This could result in respiratory compromise especially in patients who have known poor respiratory reserve. Furthermore, in patients with known right to left cardiac shunt, such as atrial septal defects, declotting is contraindicated due to the risk of paradoxical embolism causing cerebrovascular accident. Open thrombectomy should be considered in patients with high thrombus load to reduce their risk of venous embolism.

continued

Table 12.2 (continued)

Problem	Trouble shooting
High thrombus burden expected	Use thrombectomy device for clot removal to decrease risk of significant pulmonary embolism. Open surgical thrombectomy could also be considered.
Clots within the aneurysmal segment	Clearing of clots from large aneurysmal segment can be challenging as they typically contain chronic thrombi which are hard, adherent and difficult to remove. If the thrombus load is high, use an thrombectomy device such as the Angiojet (Fig. 12.11a–j) or Arrow-Trerotola device (Fig. 12.12a–d). Chronic thrombus that cannot be cleared are sometimes left alone if adequate flow can be re-established. For small aneurysms, press down on the aneurysm while pulling the Fogarty balloon across it. Several passes are often necessary to clear out the clots completely.
Arterial embolism (See Fig. 12.13)	One of the most feared complications of declotting. Possible treatment strategies are:
	1. Balloon catheter embolectomy
	(a) Pass guidewire and inflate the Fogarty balloon beyond the level of embolus
	(b) Pull back the clot into the AV access
	2. Catheter thromboaspiration
	(a) Pass the guidewire beyond the embolus and insert a 7Fr catheter up to the level of the embolus
	(b) Attach a 50 ml syringe to the catheter and apply strong aspiration pressure to remove the clot
	3. Back bleeding
	(a) Only works if there is sufficient collaterals in the arterial supply of the upper limb
	(b) Occlude the artery proximal to the AV anastomosis using a blood pressure cuff or angioplasty balloon
	(c) Ask the patient to repeated clench and unclench the fists to increase flow to the hand and retrograde flow towards the AV access
	(d) the retrograde flow will push the emboli into the AV access and the outflow veins
	4. Surgical embolectomy
Residual thrombus on final check angiogram	"Polishing" is done by using the Fogarty balloon. Insert the Fogarty balloon catheter to the site of the residual thrombus. Inflate the balloon and push it forward to dislodge the thrombus. Deflate the balloon, pull back the balloon and repeat the steps till the residual thrombus is cleared

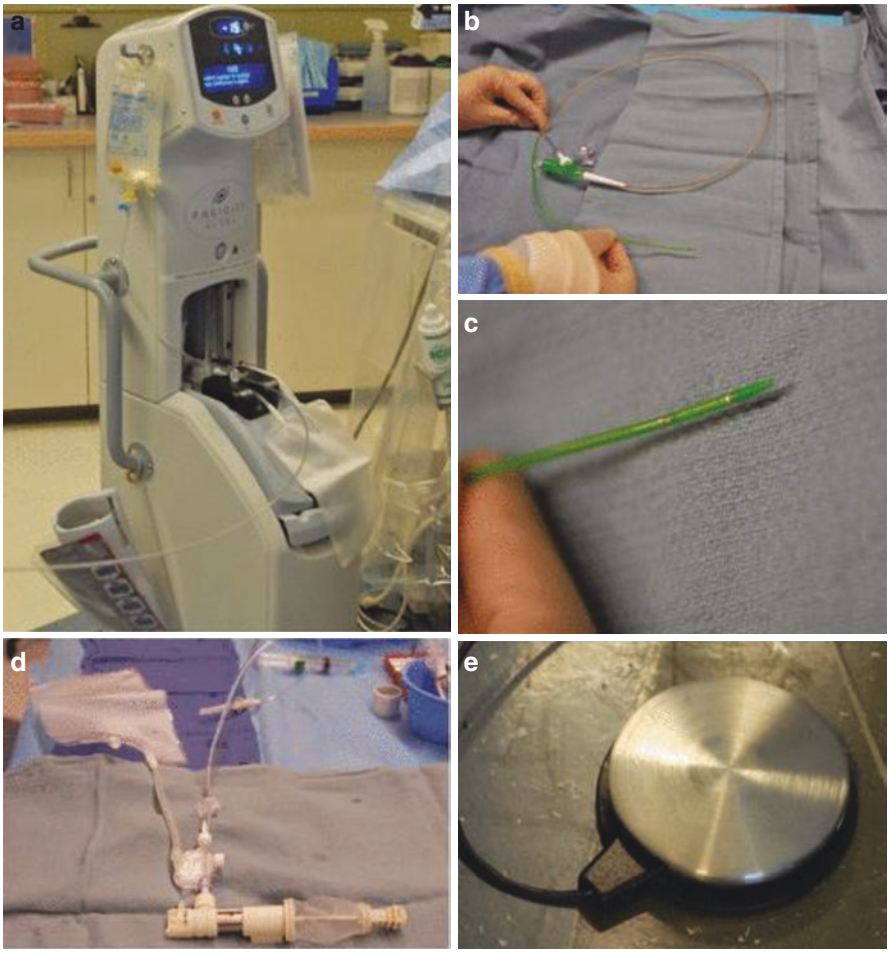

Fig. 12.11 (**a**) The Angiojet thrombectomy system (Boston Scientific, Malborough, Massachusetts) consists of three components: the machine (as shown), disposable Angiojet catheter and disposable pump set. The machine can generate high pressure saline jets and suction at an alternating rate of 60 times per minute. (**b**) The catheter has an over-the-wire catheter tip design. It contains 2 lumens: one for the inflow of high velocity saline jets, one for the passage of guidewire and evacuation of thrombotic debris. (**c**) The Angiojet catheter has side holes (marked by radio-opaque markers) near its tip. High velocity saline jets exit through the proximal side holes while suction occur at the distal side holes to aspirate the thrombus into the catheter, where it is fragmented by the high pressure saline jets and removed from the body. (**d**) A disposable pump set connects the Angiojet catheter to the Angiojet machine. It has a collection bag for thrombotic debris and a spike connector to saline bag. (**e**) The operation of the Angiojet thrombectomy system is controlled by a foot pedal. (**f**) After connecting all three components of the system, the Angiojet catheter is primed by submerging the catheter in saline and activating the machine to purge air out of the system. (**g**) This patient presented with a thrombosed AVG. A stent was previously placed at the graft vein junction to maintain patency of the graft. A large thrombus was seen just distal to the stent. Decision was made to use the angiojet thrombectomy system to remove the thrombus. (**h**) The Angiojet catheter was passed into the sheath via a guidewire to the location of interest. (**i**) Radio-opaque markers are present on the Angiojet catheter to mark the location of the catheter. Advance the Angiojet catheter to the distal end of the thrombus. Start the machine and pull back the catheter through the thrombus. On average, 3 to 5 passes are needed to clear the thrombus. (**j**) Thrombotic debris mixed with normal saline are collected within the bag at the end of procedure

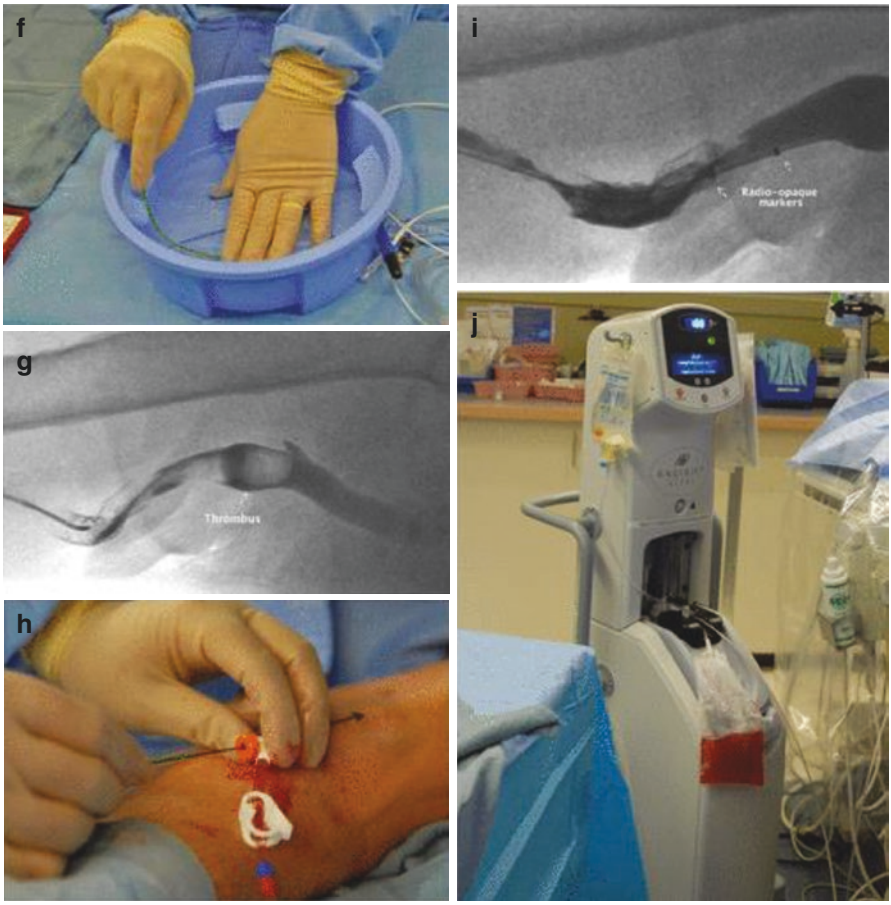

Fig. 12.11 (continued)

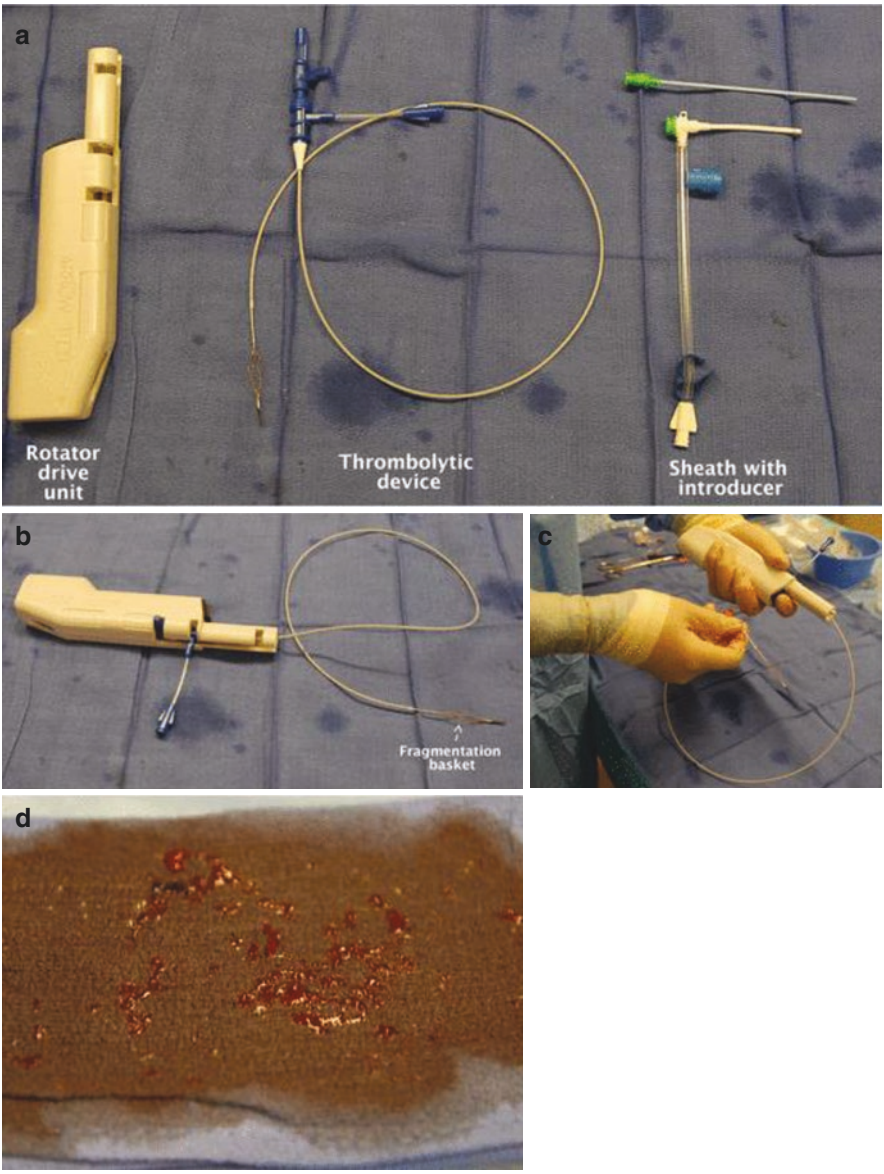

Fig. 12.12 (**a**) The Arrow-Trerotola Percutaneous Thrombolytic Device comes in three components: The rotator drive unit, thrombolytic device and the introducer sheath. (**b**) The thrombolytic device has a unique 9 mm self expanding fragmentation basket that will conform to the wall of the vessels. The 5 Fr. standard basket thrombolytic device is shown here. The device also comes in a 7 Fr. over-the-wire configuration. The setup is completed by fitting the thrombolytic device to the rotator drive unit. (**c**) Once activated, the disposable hand-held rotator drive unit will spin the basket at a rate of 3000 rpm to macerate the thrombus. (**d**) Macerated clots were aspirated from the side-arm of the sheath after the device was pulled through the thrombus

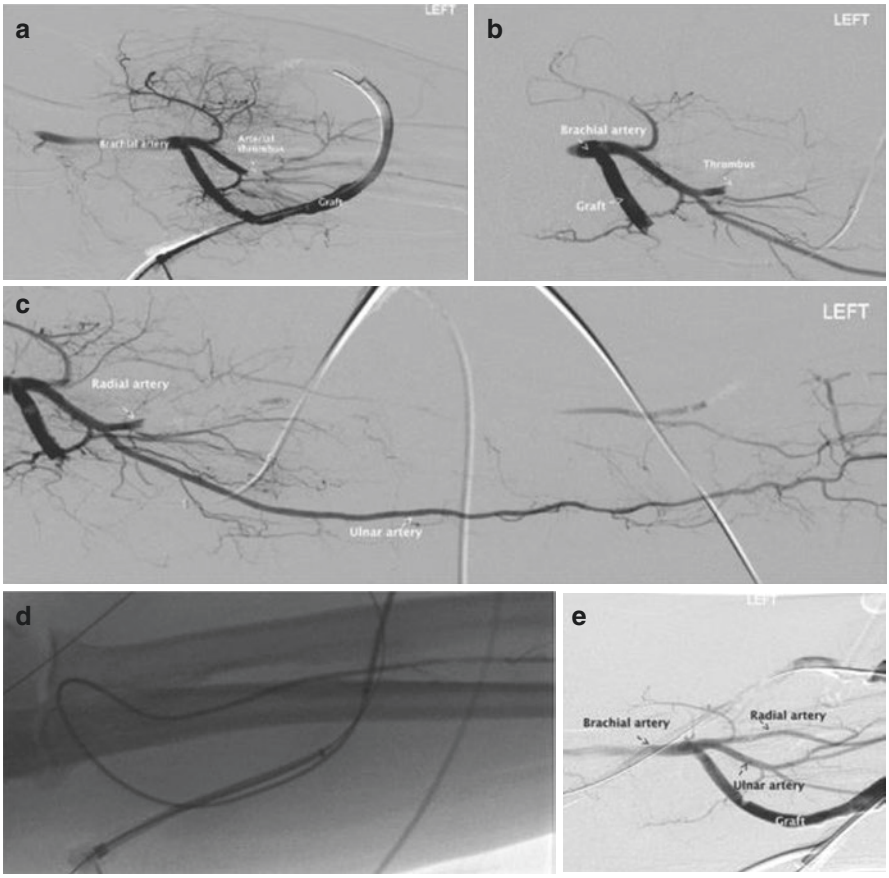

Fig. 12.13 (**a**) Post de-clotting graftogram is performed by doing a retrograde graftogram while occluding the graft using a Fogarty balloon. It showed an arterial thrombus at the bifurcation of the brachial artery with absence of flow in both the radial and ulnar artery. (**b**) A repeat retrograde graftogram showed migration of the thrombus into the radial artery. Note the reestablishment of flow in the ulnar artery and the absence of flow in the radial artery. (**c**) Absence of contrast within the radial artery. (**d**) A Fogarty balloon was used to pull the thrombus back into the AVG. (**e**): Reestablishment of flow down the radial artery is seen

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Endovascular Stent Placement

13

Ru Yu Tan, Chieh Suai Tan, Steven Wu, and Harold Park

Introduction

Although most dysfunctional dialysis access can be successfully salvaged via percutaneous intervention, restenosis is common and repeated interventions are often required. The use of stents, specifically covered stents or stent grafts, has emerged to be a viable therapeutic option in treating dialysis vascular access dysfunction over the past decade [1, 2]. The increase in stent grafts utilization is partly driven by the lack of durability of angioplasty alone in maintaining access patency and the brisk intimal hyperplasia associated with the bare metal stents. Furthermore, the patency benefits from stent grafts have been demonstrated in graft vein junction stenosis and cephalic arch stenosis in randomized control trials, although these studies have not demonstrated long-term improvement in patency rates. In the study published by Shemesh et al. [3], stent grafts were found to be superior in maintaining venous patency in the treatment of recurrent cephalic arch stenosis when compared to the bare metal stents. Haskal et al. [4] demonstrated the beneficial effect of the stent grafts compared to angioplasty alone in maintaining access patency in the treatment of AVG associated venous anastomotic stenosis.

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However, the placement of stents in the AV access circuit has remained controversial. Stent graft should be placed in an appropriate anatomical location based on its indication following a shared decision between the patient, vascular surgeon and nephrologist. The following should be considered when deciding for a stent graft:

1. Recurrent stenosis of graft-vein junction in AVG
2. Recurrent stenosis of cephalic arch
3. Angioplasty-induced vascular rupture
4. AV access aneurysm or pseudoaneurysm
5. Recurrent stenosis at surgically inaccessible location or contraindication to surgery.

Careful planning and great care should be used when deploying stent grafts. Not only are stent grafts expensive, they also carry the risk of migration, infection, and can compromise future access options. As such, their use should be limited to salvage situations, approved indications, and clinical scenarios described above.

Equipment

1. 0.035 in. guidewire
2. 9 Fr introducer sheath
3. Sterile syringes
4. Contrast material
5. Saline solution
6. Appropriate diagnostic catheter and accessories
7. Appropriate sized angioplasty balloon

Steps for Stent Deployment

1. Ensure that the stenotic segment is fully effaced with angioplasty balloon. A lesion that cannot be effaced with an angioplasty balloon should not be stented.
2. Sizing
   (a) *Diameter*
   Appropriate sizing is critical for stent deployment. Measure the diameter of the adjacent normal vein segment where the stent will be deployed. The stent should be oversized by approximately 10% from the measured diameter. For stent graft, it should not be oversized by more than 1 mm as it can cause infolding of the graft material. As self-expanding stents cannot be expanded beyond their maximal diameter, an undersized stent will not anchor well and run the risk of migration.

(b) *Length*
The length of the stent should overextend the lesion by approximately 1 cm. If the stent is to be placed near the bifurcating junction of a vessel, it should not extend beyond the bifurcation point.
(c) *Sheath*
A larger sheath is usually required for stent deployment. Remember to upsize the sheath to the appropriate size as recommended by the stent manufacturer.

3. Considerable variation of the stent delivery system exists between stent manufacturers and it is advisable to read the instruction for use provided by the stent manufacturer. The delivery systems of two different FDA approved covered stent for use in dialysis access are shown in Figs. 13.1a–g and 13.2a–e. The common feature in most of the delivery system is that the stent is crimped onto the shaft of the catheter by a covering layer. The stent is deployed when this layer is retracted. The tip of the delivery system is radiopaque to facilitate navigation under fluoroscopy. Radiopaque markers may be present to mark the two ends of the compressed stent within the delivery system. An additional radiopaque marker may also be present on the covering layer. This marker acts as a visual guide during retraction of the covering layer.
4. Prime the delivery system with normal saline.
5. The stent and the shaft of the delivery system should be aligned in a straight line to allow precise placement of the stent.
6. Insert the delivery device over the guidewire to the site of stenosis
7. Center the stenotic or target segment between the two markers under fluoroscopy.
8. Once the markers are in position, fix its position and deploy the stent according to its delivery system.
9. The initial deployment should be slow. The distal end of the stent will begin to open up like a flower. After deployment of approximately 15 mm of the covering layer, wait for the distal end of the stent to fully expand before continuing deployment.
10. After the stent is fully deployed, wait for the stent to fully expand before removing the delivery system.
11. Dilate the stent graft with an angioplasty balloon that is equal to the size of the stent graft that is placed.

Troubleshooting

1. Stent embolism
Vein diameter progressively increase as they drain centrally. Hence, there is a possibility that the stent may emboli to the heart and lungs during deployment. Therefore, it is important to have the guidewire tip in the inferior vena cava before stent deploy-

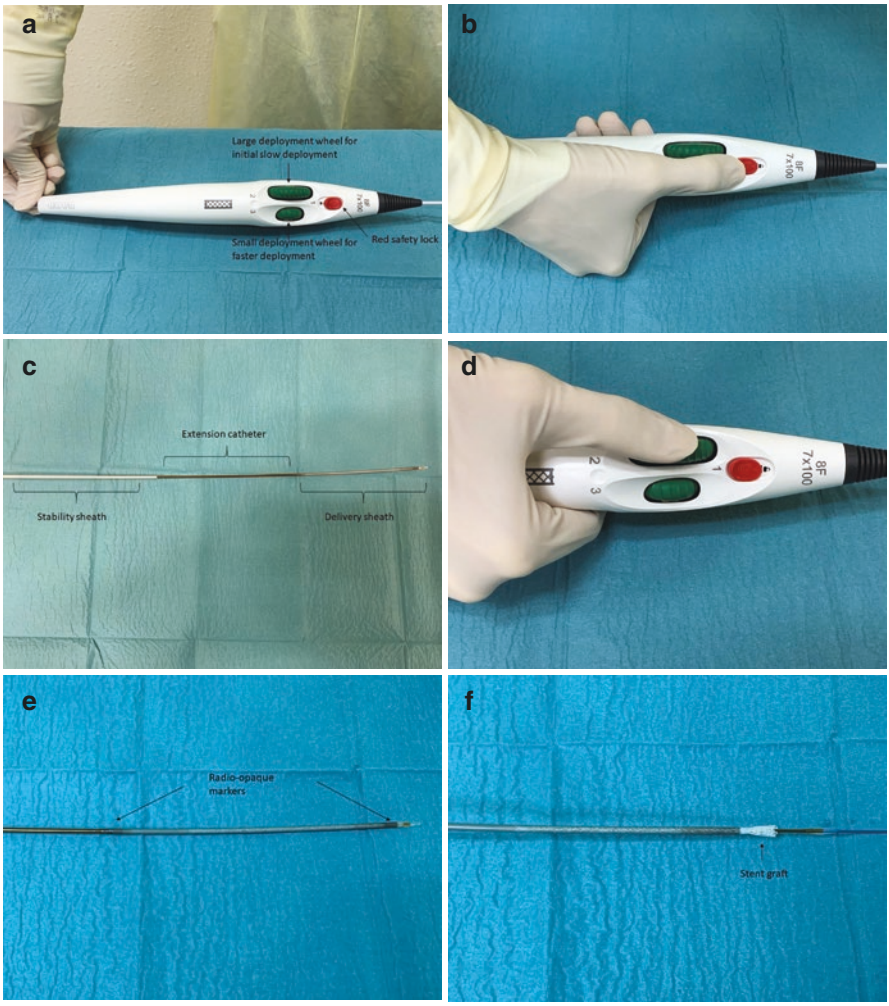

Fig. 13.1 (**a**) The delivery system for Covera stent graft (Becton Dickinson, Tempe, Arizona). (**b**) Unlock the red safety lock by pressing it down and pulling it backward. (**c**) Once the stent is positioned over the site of stenosis, straighten the catheter, use one hand to maintain a stationary hold of the white stability sheath and adjust for placement accuracy. (**d**) With the other hand, activate the stent release mechanism by rotating the large wheel (**e**) Radiopaque makers are present on the delivery sheath of the catheter to serve as a visual guide during the process. (**f**) After deployment of approximately 15 mm, wait several seconds to allow the distal end of the covered stent to expand fully. Ensure the covered stent has wall apposition before completing deployment with either the large or small wheel

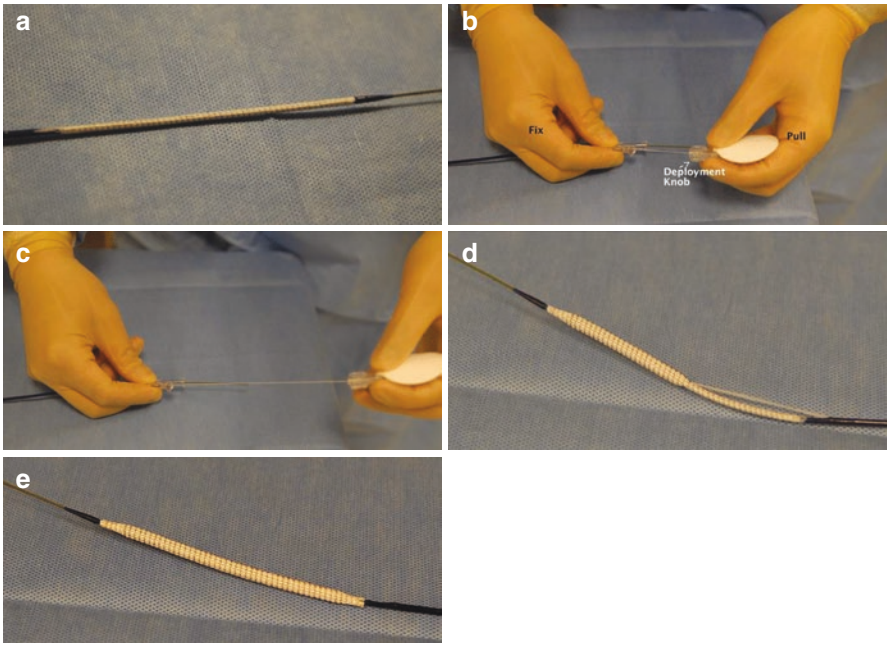

Fig. 13.2 (**a**) The stent is tightly crimped onto the shaft of the delivery catheter by a covering layer. Radiopaque markers are present to mark the tip of the catheter and the position of the stent on the catheter. (**b**) The delivery system for the Viabahn stent graft (W.L Gore & Associates, Flagstaff, Arizona) consists of a Y shape hub assembly. Once the stent is positioned over the site of stenosis, unscrew the deployment knob. A release string is attached to the knob. Fix down the Y shape hub and pull the knob away from the hub. (**c**) Keep the Y shape hub fixed with one hand while pulling the deployment knob away from the hub assembly. (**d**) The deployment of the stent will occur from the tip of the delivery catheter towards the hub. (**e**) The entire stent is released when the string is fully unwound

ment. In the event of stent embolism, the stent will end up in the inferior vena cava rather than the heart where it may trigger off life-threatening arrhythmias.

Depending on the location of the migrated stent, one can chose to remove it or place a larger stent within the migrated stent to "fix" it in place. If the stent gets lodged within the central veins, it would be advisable to remove it. To remove the stent, place a large sheath in the femoral vein, at least 1–2 Fr larger than the stent you are trying to retrieve. Grab one end of the stent with a tulip snare or a biopsy forceps and pull it into the sheath. Remove the sheath together with the stent.

2. Malpositioning of stent
   It is important to be precise during stent placement as salvage or repositioning can be a tedious process. Due to the direction of blood flow, the stent tends to be malpositioned distal to the lesion. If the distance between the malpositioned stent and the lesion is short, two strategies can be used:

(a) Inflate an angioplasty balloon (same size or 1 mm larger than the stent) within the stent to capture the stent. Pull the angioplasty balloon together with the stent towards the lesion and deflate the balloon to reposition the stent. This may not work if the self-expanding stent has fully expanded and is much larger than the proximal lesion.
(b) Place a longer stent to cover both the lesion and the malpositioned stent.

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Minimally Invasive Banding Procedure

14

Chieh Suai Tan, Zubin D. Irani, and Steven Wu

Introduction

The creation of a hemodialysis access is a non-physiological process that joins the high flow arterial circuit to the low-flow, low-resistance venous pathway. The systemic vascular resistance decreases immediately post anastomosis and the cardiac output increases to accommodate the shunting effect of the arteriovenous (AV) access. This creates the potential for a spectrum of problems, including high-output cardiac failure and vascular access-associated distal hypoperfusion ischemic syndrome (DHIS) or steal syndrome. High-output cardiac failure occurs when the cardiac function is unable to meet up with the demands created by the shunting effects of the AV access while DHIS results when the AV access diverts an excessive amount of blood away from the distal artery, resulting in tissue hypoperfusion.

Banding of the AV access is a procedure used to reduce the blood flow to the access by creating a high resistance band to restrict the flow of blood into the AV access. Specifically, the MILLER (Minimally Invasive Limited Ligation Endoluminal-Assisted Revision) banding procedure is a technique that can be used

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to reduce blood flow to the access with the aid of fluoroscopy and some basic endovascular tools [1]. The indications for the MILLER banding procedure are

1. Vascular access-associated distal hypoperfusion ischemic (DHIS) or steal syndrome. Before performing the MILLER procedure, it is crucial to exclude any arterial disease that may be causing the distal hypoperfusion or steal syndrome. The baseline blood flow rate of the access should also be measured. Banding is most suitable for AV access that has high flow. Banding a low or normal flow access that is causing steal syndrome will decrease the AV access flow rate further, resulting in access thrombosis or AV access flow rate that is too low for dialysis treatment. Such AV access should be ligated or revised. The algorithm for patients with DHIS is as shown in Fig. 14.1.
2. Vascular access causing high output cardiac failure

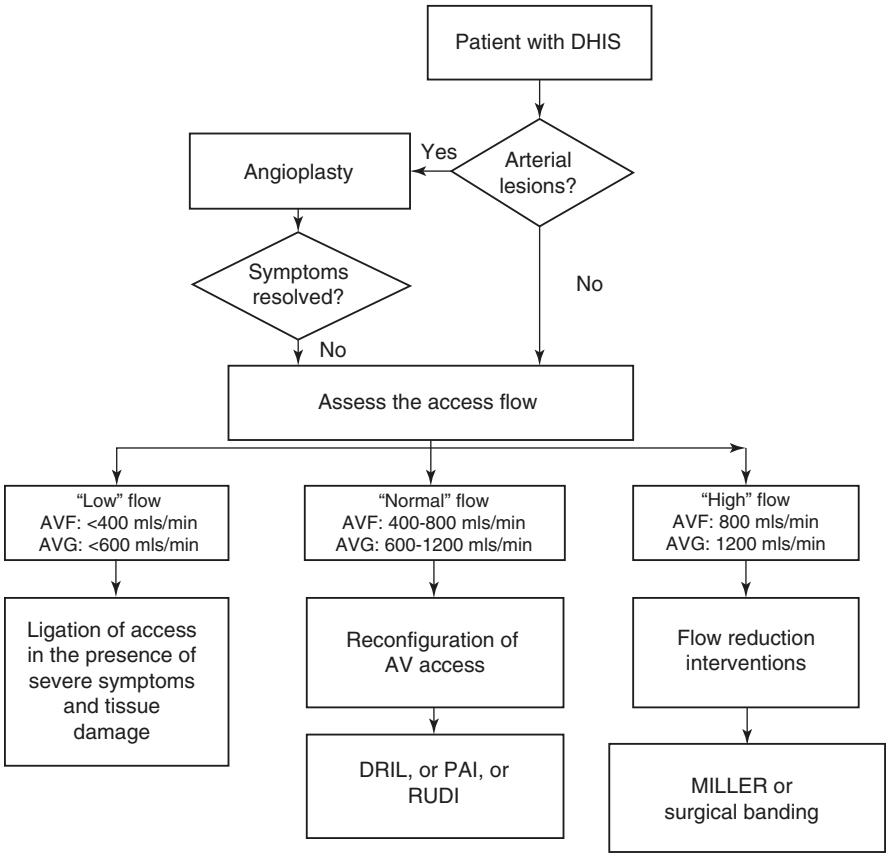

Fig. 14.1 Approach to patients with Distal Hypoperfusion Ischemic Syndrome (DHIS). *DRIL* Distal Revascularization and Interval Ligation, *PAI* proximalization of the arterial inflow, *RUDI* revision using distal inflow, *DRIL* PAI and RUDI are surgical procedures

Equipment for MILLER Procedure

1. Lidocaine
2. 18G intravenous catheter
3. 6 Fr vascular sheath
4. 0.035 in. guidewire
5. Angioplasty balloon
6. Surgical blade
7. 2/0 Prolene suture
8. 3/0 Vicryl suture
9. Hemostat
10. Catheter-based thermodilution system to measure flow

Steps for MILLER Procedure

1. Clean and drape the patient. Cannulate the AV access in the retrograde direction with a 18G intravenous catheter at a reasonable distance from the AV anastomosis. Image the outflow and central veins to exclude the presence of any stenosis. Any significant stenosis within the outflow and central veins should be treated with balloon angioplasty before the MILLER banding procedure.
2. Image the inflow of the AV access by doing a "reflux" angiogram (Fig. 14.2a). This is done by injecting the contrast while compressing the outflow of the AV access.
3. Insert a 0.035-in. guidewire over the 18G intravenous catheter and exchange it for a 6 Fr vascular sheath. Make a flow measurement of the vascular access using the catheter-based thermodilution system.
4. Selection of the banding site. Palpate the vein adjacent to the arterial anastomosis site to find an area in which the banding site would be as close to the anastomosis as possible (1–3 cm) and superficial enough for dissection. Ultrasound may be used to determine the depth of the vein and the presence of adjacent vascular structures (Fig. 14.2b).
5. Do an angiogram to confirm the location of the arterial anastomosis and the banding site. Measure the diameter of the inflow artery and the vein at the banding site (Fig. 14.2c).
6. Make two 1 cm lateral incisions parallel to the vein at the banding site. Using a hemostat, create a tunnel under the vein between the two lateral incision sites by blunt dissection. Pull two strands of 2/0 Prolene through the tunnel that was created (Fig. 14.3a–e).
7. Create a second tunnel just below the skin but above the vein between the two lateral incisions. Pull the suture across the tunnel so that it is now looped circumferentially around the vein (Fig. 14.4a, b).
8. Pass the guidewire into the inflow artery. Inflate an angioplasty balloon over the banding site and tie the Prolene sutures over the inflated angioplasty balloon. The size of the angioplasty balloon to be used is dependent on the diameter of

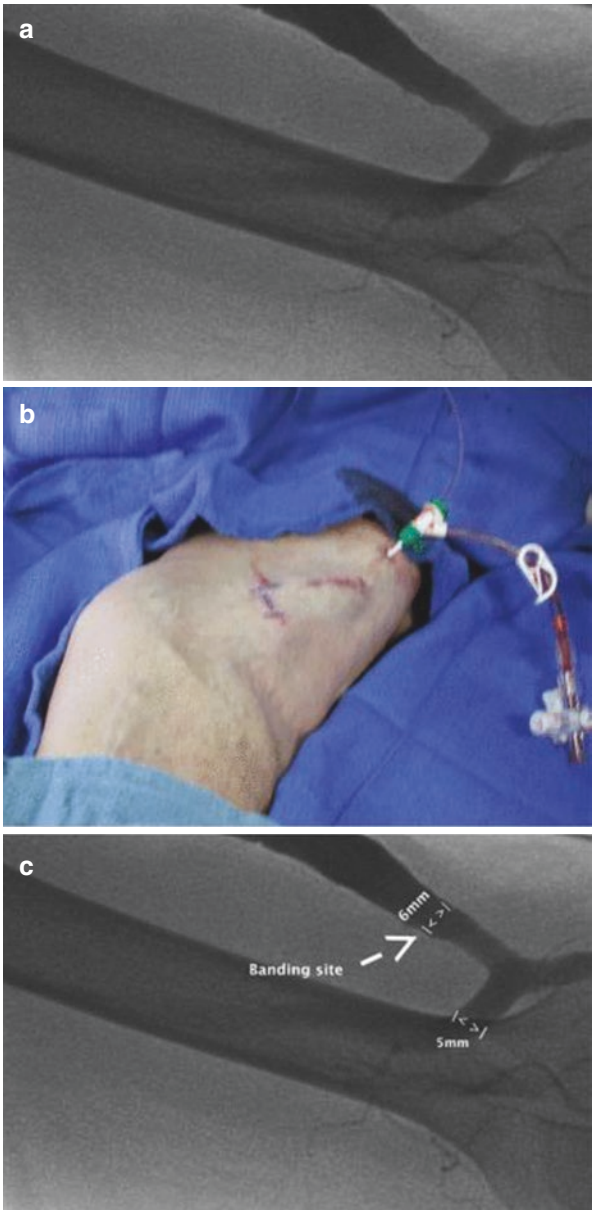

Fig. 14.2 (**a**) This patient has a high flow left brachiocephalic fistula. A reflux angiogram was done to visualize the inflow of the fistula. (**b**) After insertion of the vascular sheath, palpate and mark out the banding site. (**c**) Confirm the location of the banding site and measure the diameter of the inflow artery and the vein at the banding site

the inflow artery. In general, the size of the balloon used should be equal to or smaller than the size of the artery. The typical balloon sizes are between 4 and 6 mm (Fig. 14.5a–c).

9. Once the ligature is secured, deflate the balloon and do an angiogram of the AV access to document the results. Make a flow measurement of the vascular access

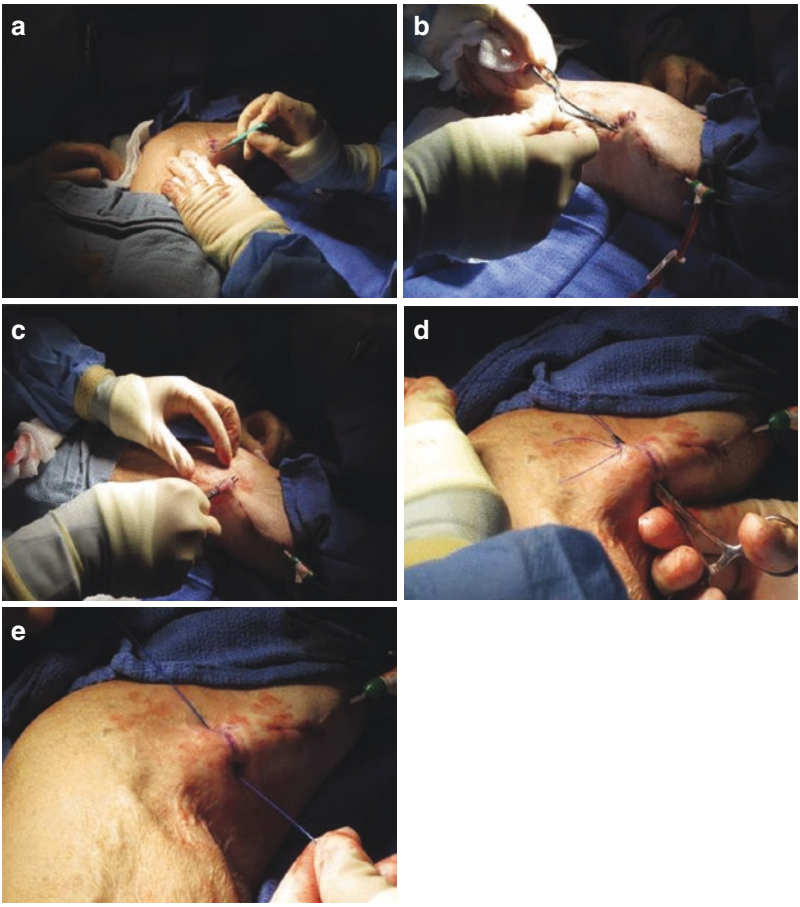

Fig. 14.3 (**a**) After administrating local anesthesia, make two incisions parallel to the vein at the banding site. (**b**) Enlarge the incision sites by blunt dissection. (**c**) Through the process of blunt dissection, create a tunnel beneath the vein between the two lateral incision sites. (**d**) Grab the ends of the prolene suture with the hemostat. (**e**) Pull the sutures into the tunnel beneath the vein

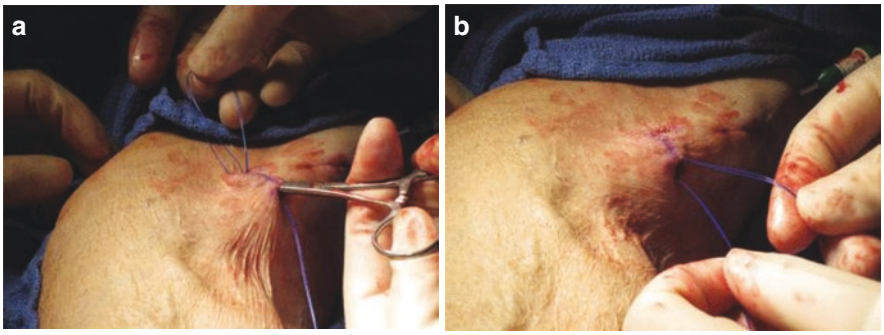

Fig. 14.4 (**a**) Create a second tunnel just below the skin but above the vein between the lateral incisions. (**b**) Pull the sutures across the superficial tunnel. The sutures are now looped circumferentially around the vein

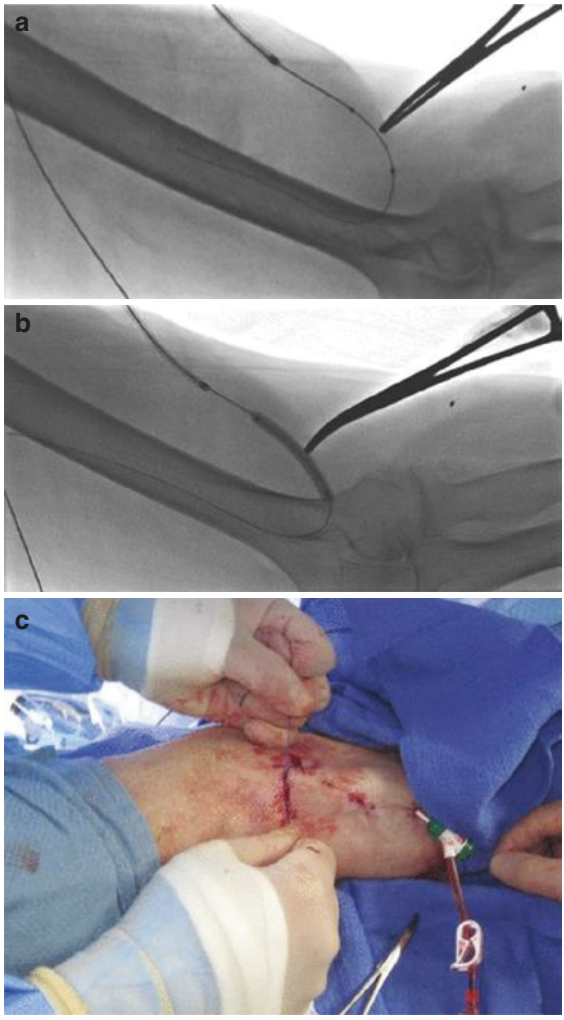

Fig. 14.5 (**a**) Position the angioplasty balloon over the banding site. (**b**) Inflate the angioplasty balloon over the banding site. (**c**) Tie the prolene sutures over the inflated angioplasty balloon. Hold the angioplasty balloon catheter while tying the suture

using the catheter-based thermodilution system to document the reduction in flow within the vascular access (Fig. 14.6a, b).

10. Palpate the AV access to ensure that flow is adequate for dialysis. In patients where banding is done for steal syndrome, check if the symptoms are better.
11. Close up the incision site with absorbable suture and remove the vascular sheath (Fig. 14.7a–e).

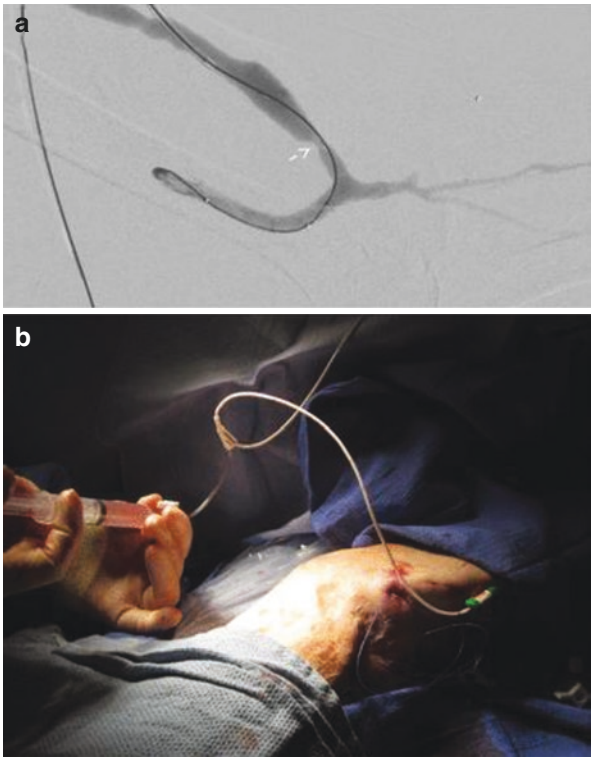

Fig. 14.6 (**a**) Post banding fistulogram showed the stenosis created by the prolene suture. (**b**) Blood flow rate of the fistula is measured using a thermodilution catheter after banding

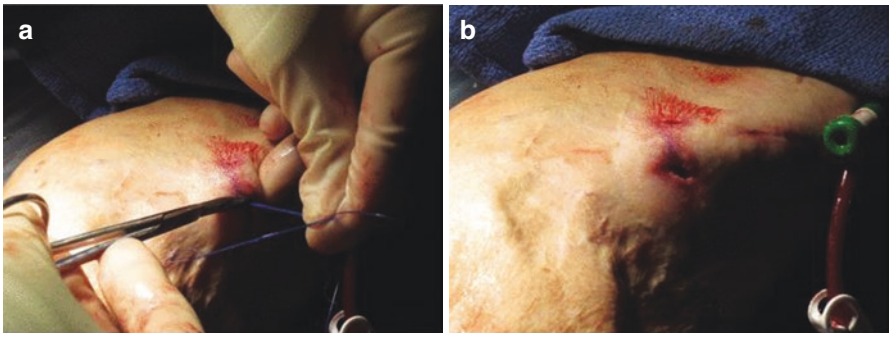

Fig. 14.7 (**a**) Cut the prolene suture and bury the ends within the subcutaneous tissue. (**b**) Appearance of the incision site after the procedure. (**c**) Close the incision sites with non-absorbable sutures. (**d**) Remove the vascular sheath after placing a purse-string suture around it. (**e**) Final appearance of the fistula after the banding procedure

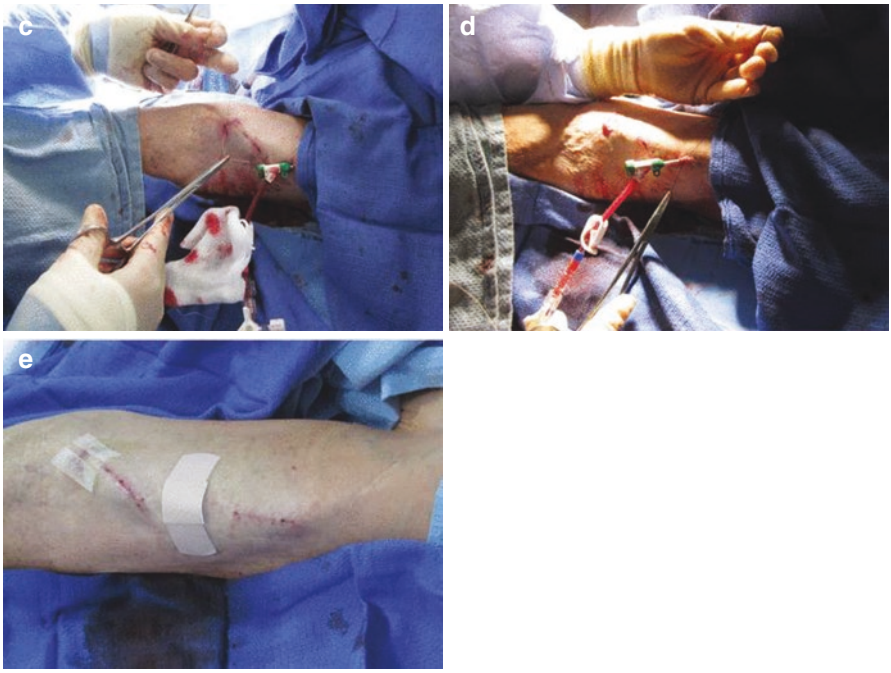

Fig. 14.7 (continued)

Tips and Troubleshooting

1. Patient selection
   Patients with aneurysmal juxta-anastomotic junction may not be suitable for the banding as the area of blunt dissection to free the vein would be extensive. Surgical revision of the inflow may be a better option in such patients.
2. Banding site management
   The angioplasty balloon should be inflated to the rated burst pressure before tying the sutures around the banding site. Excessive forces should not be used when tightening the suture as the balloon might be indented by the sutures, resulting in overcorrection of the banding procedure.
3. Poor flow within the AV access after banding
   If the flow within the AV access is too low to be used for dialysis after banding (overcorrection), inflate a balloon with a diameter that is 1 mm larger than the one that is used to stretch the band.
4. Persistent symptoms despite banding
   If the flow is still elevated post banding, a repeat procedure with a second ligature may be attempted. A balloon with a diameter that is 1 mm smaller than that of the balloon used for the first banding procedure should be used for the second attempt. If the patient persists to have steal syndrome after banding, ligation of the fistula should be considered.

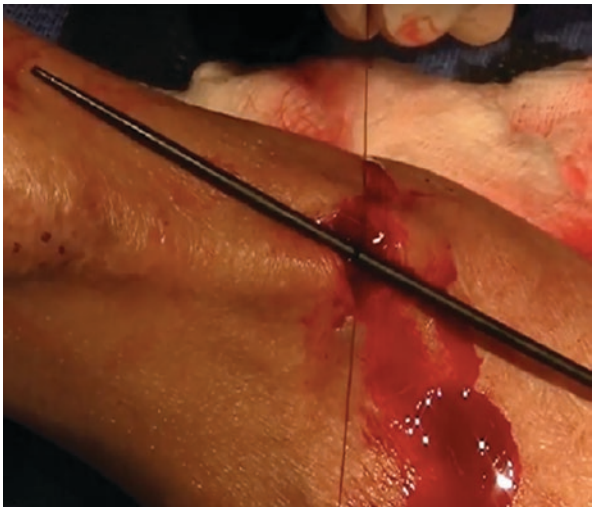

Fig. 14.8 A strand of suture is used to create the band by tying it around the dilator. The dilator is then removed and the band is created around the juxta-anastomotic segment [2]

Modifications of the MILLER Procedure

1. There are descriptions of modifications of the MILLER procedure for banding of AVF in the literature. Modifications include
   (a) Use of a 10 Fr dilator instead of an angioplasty balloon as support to tie the prolene suture [2]. The procedure is performed as described from step 1 to 7 above. The retrograde vascular sheath is removed and exchanged for a 10 Fr dilator over a 0.035 in. wire. The 10 Fr dilator is passed gently over the guidewire to the juxta-anastomotic segment under fluoroscopy guidance. The prolene suture is then tied over the dilator to create a band to restrict flow to the AVF. The dilator is then removed and the sheath entry site is closed with prolene suture.
   (b) The dilator can be used as an external scaffold to tie the prolene suture [3]. The procedure is performed as described from step 1 to 7 as above. The dilator is used to tie the suture externally as shown in Fig. 14.8. The dilator is then removed and the cutdown site is repaired.

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Peripheral Arterial Disease in Hemodialysis Access

15

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Introduction

Peripheral artery disease (PAD) is a major health concern and its incidence increases with age. End stage renal disease (ESRD) is a significant risk factor for developing PAD and is associated with a remarkably high incidence of cardiovascular morbidity and mortality [1]. In patients with ESRD, the estimated prevalence of PAD ranges between 17% and 48% [2, 3]. Two pathological processes have been identified in PAD associated with ESRD namely medial arterial calcification(MAC) and occlusive arteriosclerotic disease [4].

Hemodialysis is the major renal replacement technique used in the United States. Per the 2013 statistics published by the National Kidney and Urological disease information clearinghouse, 93% of patients on dialysis in the United States are on hemodialysis [5]. The hemodialysis accesses are of three types, arteriovenous fistulas (AVF), arteriovenous grafts (AVG) and hemodialysis catheters. AVF is considered the gold standard for access based on its superior patency and low complication rate [6]. Per the Dialysis outcome and practice pattern study (DOPPS) practice monitor (accessed April 2020), 71.5% of the hemodialysis in the US occurs via a native arteriovenous fistula, whereas 17.8% use synthetic arteriovenous grafts [6]. Irrespective of the type of vascular access, a robust inflow to maintain the circuit and

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also perfuse the upper extremity is essential for access patency and limb perfusion. Even in the best-case scenario of having an AVF, there is a 30% abandonment rate after 1 year [7]. The most common cause of failure for AVF is perianastomotic stenosis compromising the arterial inflow [8]. The other major problem with compromised arterial inflow is dialysis access associated steal syndrome (DASS), which occurs in 3.7%–5% of dialysis patients [9]. Thus, the focus of the current chapter is on arterial inflow disease affecting patients on hemodialysis.

Normal and Variant Anatomy

The upper extremities derive their blood supply from the aorta via the subclavian arteries. On the right side, the subclavian artery arises from the brachiocephalic artery whereas the left subclavian artery originates directly off of the aortic arch. The subclavian artery continues as the axillary artery after crossing the lateral margin of the first rib. The axillary artery supplies branches to the shoulder girdle and lateral chest wall and subsequently continues as the brachial artery beyond the inferior lateral margins of the teres minor muscle. The brachial artery gives rise to the deep brachial artery and additional branches around the elbow (Fig. 15.1a, b).

In the cubital fossa, the brachial artery divides into the radial and ulnar arteries. The radial recurrent artery and the anterior and posterior ulnar recurrent arteries arise immediately beyond the origin of their respective arteries to anastomose with

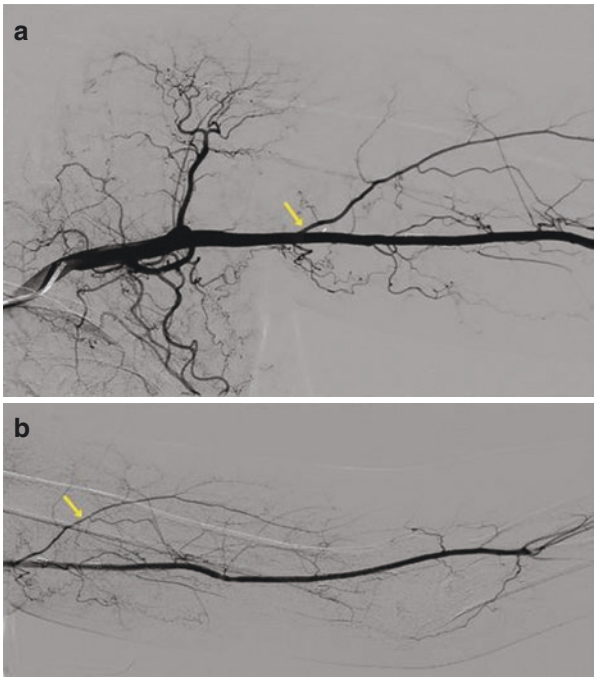

Fig. 15.1 (**a**) Digital subtraction angiogram of the left upper extremity demonstrates the origin of the deep brachial artery (*arrow*). (**b**) Digital subtraction angiogram of the left upper extremity delineates the course of the deep brachial artery (*arrow*)

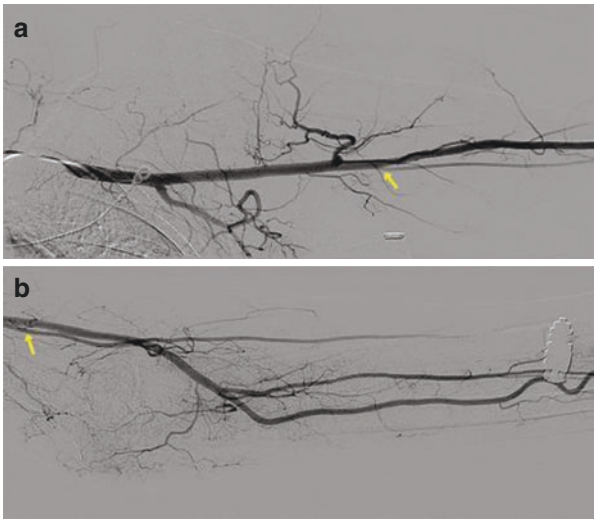

Fig. 15.2 (**a**) Digital subtraction angiogram of the left upper extremity demonstrates high origin of the radial artery (*arrow*). (**b**) Digital subtraction angiogram of the left upper extremity demonstrating the course of the radial artery with high origin in the forearm (*arrow*)

the branches of the brachial and deep brachial arteries around the elbow. The radial artery courses along the radial aspect of the forearm to the wrist, traverses the anatomical snuff box, and gives rise to the deep palmar arch. The ulnar artery courses along the ulnar aspect of the forearm and continues as the superficial palmar arch. The common interosseous artery arises from the ulnar artery and gives rise to the anterior and posterior interosseous arteries. The terminal branches of these arteries anastomose with branches of the radial and ulnar arteries to form the dorsal and volar carpal arches. The two carpal and two palmar arches form a rich collateral pathway between the radial and ulnar arteries.

A high origin of the radial artery from the axillary artery (2.7%–5%) or the upper brachial artery (5.9%–12.1%) is an important anatomical variant [10] (Fig. 15.2a, b). A high origin of the ulnar artery is much less common (0.17–2.0%) [10] (Fig. 15.3a, b). Duplication of the brachial artery and hypoplasia or aplasias of the radial/ulnar arteries are rare variants. A persistent median artery results from lack of regression of the embryonic median branch arising from the common interosseous artery and is found in 2–4% of the population [11].

Pathophysiology

Creation of an arteriovenous (AV) shunt causes a significant local and systemic change to the blood flow. There is bypassing of the resistance vessels in the distal extremity and creation of a parallel fixed low resistance return pathway to the heart. As the flow within the fistula increases there is diminished perfusion of the tissues distal to the fistula. To compensate for this loss of tissue perfusion, there is arterial vasodilatation. Mean arterial blood flow in the brachial artery at rest is around 50 ml/min, whereas the mean blood flow in the radial artery at rest is 25 ml/min [12]. To achieve dialysis

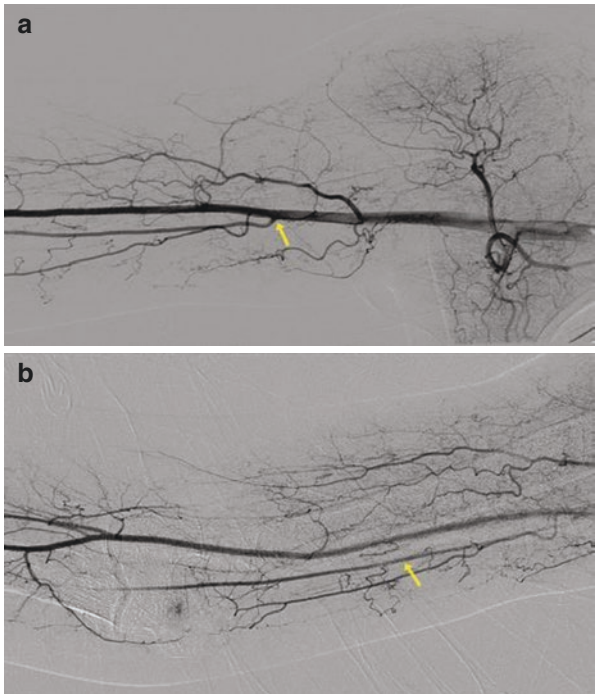

Fig. 15.3 (**a**) Digital subtraction angiogram of the right upper extremity demonstrating high origin of the ulnar artery (*arrow*). (**b**) Digital subtraction angiogram of the right upper extremity demonstrating high origin of the ulnar artery (*arrow*) as it courses into the forearm. Note the origin of the radial and interosseous arteries in the cubital fossa

compatible flow (which averages 500 ml/min) the blood flow in the artery must increase 10–20 fold. Poiseuille's Law states that the volume of a homogenous fluid passing per unit time (flow) through a capillary tube is directly proportional to the pressure difference between its ends and to the fourth power of its internal radius and inversely proportional to its length and the viscosity of the fluid. Thus if the blood flow is steady and the viscosity and pressure gradient down the artery are constant, then the brachial artery needs to dilate approximately 80% to achieve a tenfold increase in flow [12].

In reality this is not the case, the pressure gradient changes pre and post creation of the AV fistula. A "steal phenomenon" occurs due to the change in pressure gradient. The term "steal" refers to the phenomenon that occurs when there is a connection between two vascular beds. The difference in resistance causes arterial blood to be diverted from the higher resistance vascular circuit to the lower resistance vascular circuit. In the case of dialysis vascular access, the two vascular beds are the extremity distal to the AV access and the AV access itself. This results in retrograde flow in the artery distal to the anastomosis [13](Fig. 15.4a, b). Blood flow assessment reports in the AVF with steal phenomenon have shown that the blood flow in the AVF actually exceeds that of the feeding artery by 15–20% due to retrograde contribution [14, 15]. The steal phenomenon is a physiological response to resistance differences and occurs in the majority of patients with AV access. By itself, steal phenomenon, does not lead to hand ischemia or dialysis access dysfunction.

Substantial arterial dilatation is still required to achieve good dialysis flow. There is evidence that this arterial vasodilatation is mediated by nitric oxide release from

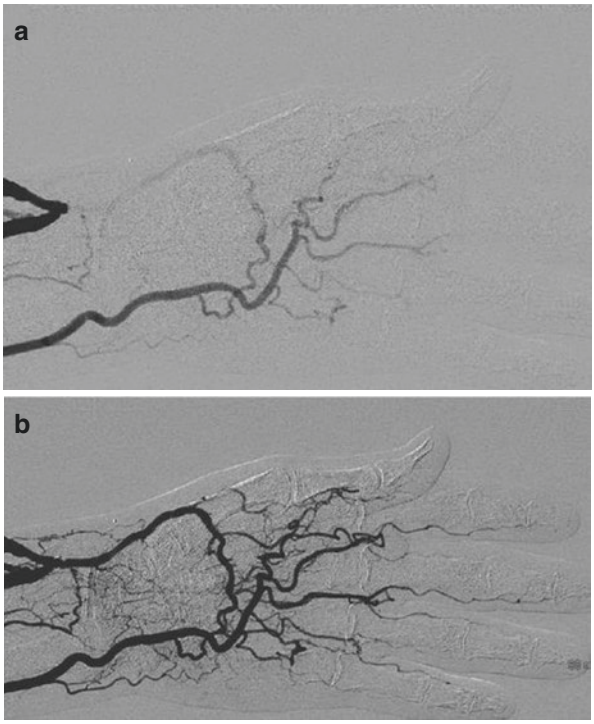

Fig. 15.4 (**a**) Digital subtraction angiogram of the right upper extremity demonstrating flow into a dominant ulnar artery downstream from an upper extremity dialysis access. (**b**) Digital subtraction angiogram of the right upper extremity in a more delayed frame demonstrating retrograde flow into the radial artery distal to the anastomosis, consistent with steal phenomenon

the endothelial cells in response to wall shear stress. In experimental studies of fistula maturation there is evidence of fragmentation of the arterial internal elastic lamina in order to facilitate vasodilatation. The vasodilatation is mediated by metalloproteases, which are activated by nitric oxide [12, 16]. The arterial diameter continues to increase as long as the sheer stress persists, which may take up to 1 year. Any condition that prevents arterial remodeling leads to poor flow through the shunt. For example, poor collateralization distal to the AV access site can result in tissue ischemia, which is further accentuated by the steal phenomenon.

Risk Factors

Recognizing the risk factors at the time of access planning is critical for successful creation of a durable AV access. Major predisposing risk factors which will limit the inflow include female sex (OR 2.77), age > 60 years (OR 1.03), diabetes (OR 6.04) peripheral artery disease (OR 2.70) and the use of brachial artery as the inflow (OR 8.42) [17]. These conditions could be focal or diffuse. Focal stenosis in the inflow circuit can occur anywhere in the aorta, subclavian (Fig. 15.5a, b), axillary, brachial (Fig. 15.6a, b) and radial arteries (Fig. 15.7a, b) or at the arterial anastomosis. Diffuse vessel wall calcification due to MAC leads to limited ability of the artery to dilate [4].

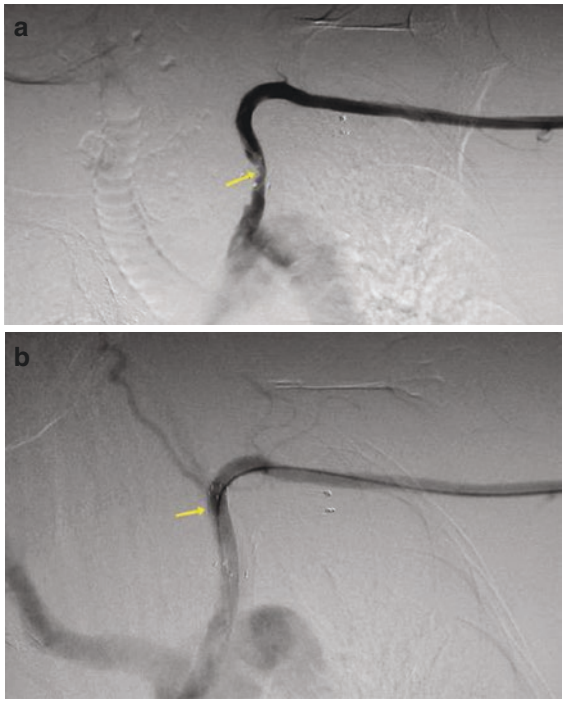

Fig. 15.5 (**a**) Digital subtraction angiogram of the subclavian artery demonstrates focal stenosis (*arrow*). The vertebral artery is not opacified. (**b**) Digital subtraction angiogram of the subclavian artery after stent (*arrow*) placement. There is resolution of the subclavian stenosis and flow in the vertebral artery

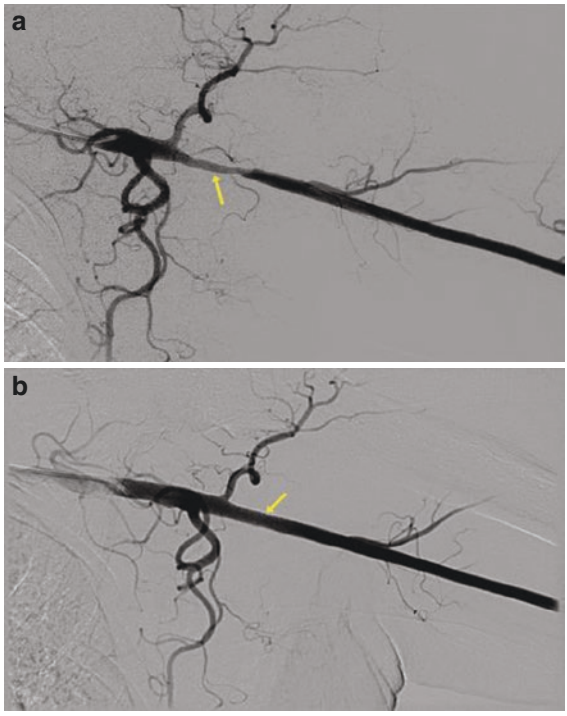

Fig. 15.6 (**a**) Digital subtraction angiogram demonstrates focal stenosis (*arrow*) in the brachial artery. (**b**) Digital subtraction angiogram demonstrates resolution of stenosis after angioplasty and stent (*arrow*) placement

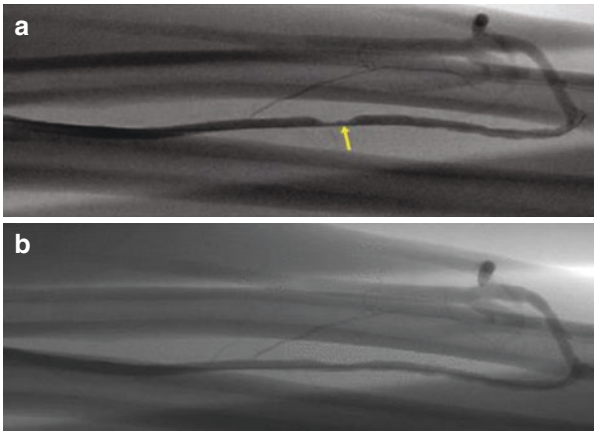

Fig. 15.7 (**a**) Digital angiogram demonstrates a left upper extremity radio-cephalic fistula with severe stenosis (*arrow*) of the radial artery. (**b**) Digital angiogram demonstrates resolution of radial artery stenosis following successful angioplasty with a 3 mm balloon

Signs and Symptoms

Clinical presentations vary based on the site of stenosis. In general, AV access dysfunction/non-maturation occurs due to juxta anastomotic disease (JXA). More proximal arterial inflow disease presents with dialysis access associated steal syndrome (DASS) and/or ischemic monomelic neuropathy (IMN).

Juxta-anastomotic disease presents with early fistula failure (within 3 months) or access dysfunction. This specific type of stenosis has a typical appearance; the lesion occurs in the segment of the vein that is immediately adjacent to the anastomosis (Fig. 15.8). The etiology of this phenomenon is unclear. It is thought to be due to trauma during surgical manipulation resulting in ischemic injury to the vasa venosum supplying the vein [18]. Clinical examination will reveal a very accentuated pulse at the arterial anastomosis with a water-hammer character. As one moves up the vein from the anastomosis the pulse goes away abruptly at the site of stenosis. Above this level, the pulse is very weak and the vein is poorly developed [19].

DASS results from arterial insufficiency distal to the AV access site (Fig. 15.9a, b). The incidence of hand ischemia after placement of a hemodialysis shunt is 5% [20]. Clinical features of DASS have been classified into four classes akin to the Rutherford classification for lower limb ischemia [21, 22]. These are as follows:

- Stage I: No clinical symptoms, discrete signs of mild ischemia are present. Hand may be cold compared to the opposite side. Numbness, paresthesia, absent or diminished pulses may be noted. Management is conservative.
- Stage II: Divided into two categories based on whether the symptoms are tolerable (IIa) or not tolerable (IIb). The pain is not present at rest and is brought on only during dialysis or exercise. Stage IIb usually requires invasive therapy.
- Stage III: Pain at rest and/or loss of motor functions; urgent invasive therapy is recommended.

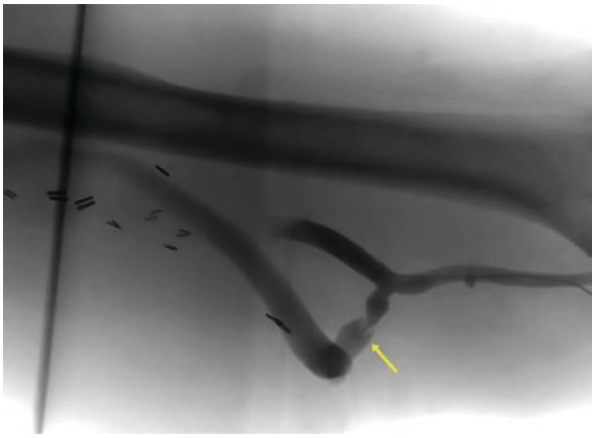

Fig. 15.8 Digital angiogram demonstrates a brachial artery to basilica vein fistula with venous stenosis (*arrow*) approximately 1 cm distal to the arterial anastomosis

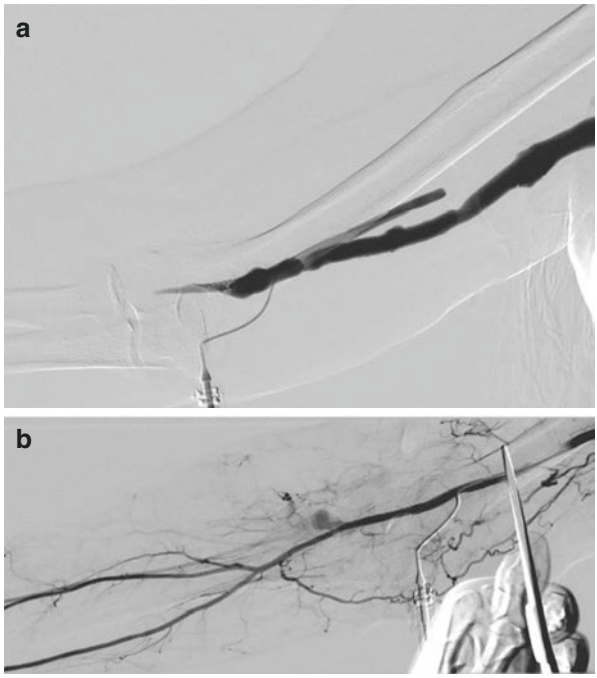

Fig. 15.9 (**a**) Digital subtraction angiogram demonstrates a right upper extremity brachiobasilic fistula with decreased flow downstream to the fistula in a patient with clinical symptoms of ischemia consistent with steal syndrome. (**b**) Digital subtraction arteriogram in the same patient demonstrates increased downstream arterial flow after manual compression of the venous outflow of the fistula

- Stage IV: Presence of tissue loss. This stage is further classified into reversible (IVa) and irreversible (IVb). Both require urgent intervention. In the former any intervention to improve blood flow should be undertaken. In the latter, significant loss of function may require more invasive measures such as amputation.

Ischemic monomelic neuropathy is caused by focal ischemic axonal nerve injury involving the sensory and motor branches in the distal portions of the affected limb, usually occurring in diabetic hemodialysis patients. Signs of DASS may be entirely

absent [23].Typically IMN involves the three nerves of the upper limb and occurs immediately after creation of the access. Management is mainly symptomatic.

Diagnosis

An arterial stenosis is defined as 50% or greater decrease in the luminal diameter as compared to an adjacent normal appearing artery [24]. Inflow stenosis can occur anywhere from the ascending aorta to the AV anastomosis [25]. In the setting of an AVF, stenosis that develops up to 4 cm from the anastomosis is termed as juxta-anastomotic (JXA) stenosis [26]. Imaging studies that are commonly used to evaluate AV access inflow vessels include Doppler ultrasound, CT/MR angiography and conventional angiography.

Doppler ultrasound is performed using 5–7.5 mHz linear array probes. Transverse and longitudinal B-mode and color flow images are obtained along the arterial inflow from the subclavian artery, the arterial anastomosis, and into the JXA segment (Fig. 15.10a, b). The artery is thick walled, less compressible, deeply situated and has long straight segments with a similar diameter. Waveforms are recorded from a small sampling volume placed in the central flow stream at attempted angles of 60° relative to the vessel walls. Based on the velocity of flow and vessel diameter, the flow is calculated electronically. Unfortunately, there is no general agreement on the objective diagnostic criteria for hemodynamically significant access stenosis. A proposed criterion for diagnosis of arterial stenosis include [27]:

- Arterial luminal narrowing of ≥50% on the B-mode scan
- >two-fold increase in peak systolic velocity at the site of stenosis compared to normal appearing proximal arterial segment
- plus one of the following additional criterion:
  - Flow reduction by 20% during dialysis
  - Flow <600 ml/min during dialysis
  - Residual luminal diameter of <2 mm on B-mode scan

Brachial artery resistive indices have been shown to correlate with graft dysfunction [28]. In DASS, the blood flow distal to the arterial anastomosis may be reversed or bidirectional [29]. Digital blood pressure evaluation using both blood pressure (DBP) and digit brachial index (DBI: ratio of the DBP to contralateral brachial blood pressure) has been used to evaluate patients with DASS. Normal DBI varies between 0.8 and 1.1. A DBI below 0.4 and a DBP of less than 60 mmHg is considered indicative of DASS [30]. The accuracy of a DBP of below 60 mmHg for determining hand ischemia was 92% (sensitivity 100%, specificity 87%) compared with 94% for DBI of less than 0.4 (sensitivity 92%, specificity 96%) [13].

The role of CTA/MRA in the diagnosis of arterial inflow has been studied and proven to be superior to doppler ultrasound [31]. However, due to the expense associated with these tests and the fear of nephrogenic systemic sclerosis with MRA, these tests are utilized only in problem solving situations.

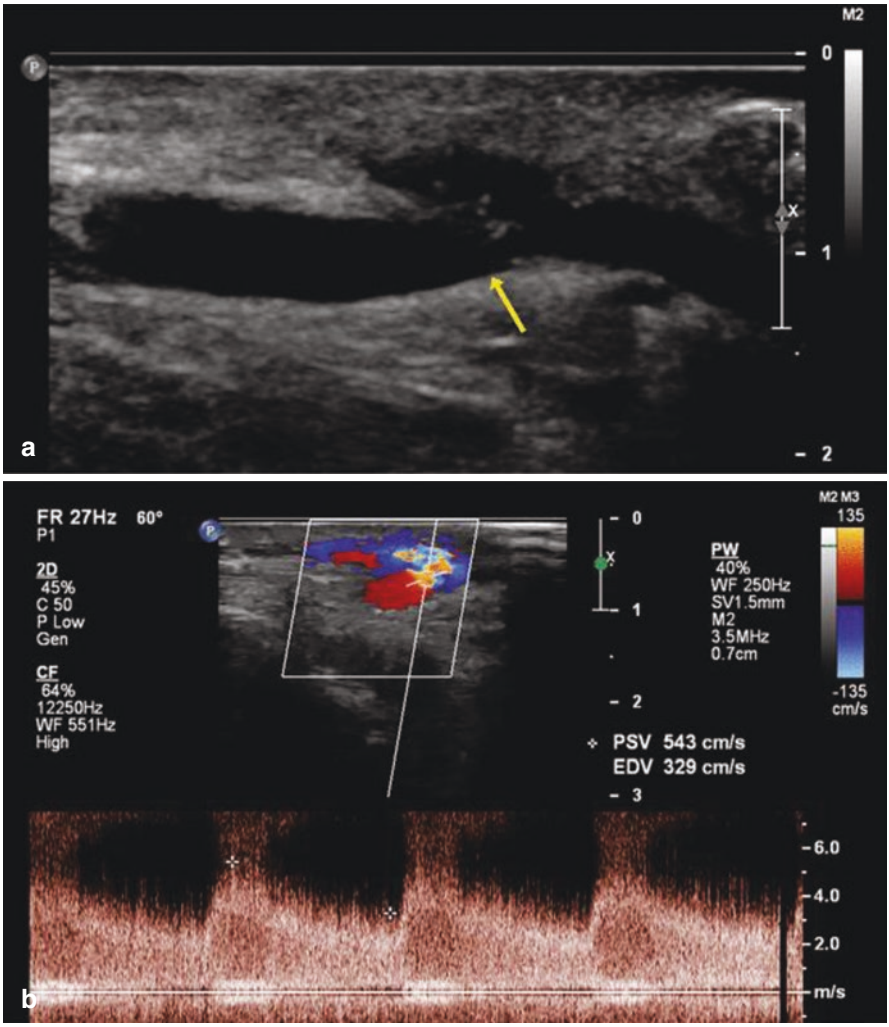

Fig. 15.10 (**a**) Grayscale ultrasound demonstrates juxta-anastomotic stenosis (*arrow*) of the radial artery. (**b**) Color Doppler ultrasound at the same location demonstrates increased velocity at the site of stenosis consistent with hemodynamically significant stenosis of the radial artery

Angiographic evaluation is the gold standard for arterial inflow evaluation. Angiographic evaluation should be performed for the entire arterial inflow from the aortic arch to the outflow veins. The key is to distinguish physiological steal from an obstructing lesion, such as stenosis or occlusion, in the feeding artery that is causing the ischemia. An angiogram of the arterial tree distal to the anastomosis should be performed both with the access open and occluded. The status of the distal circulation can then be evaluated.

Management

Preoperative evaluation is critical in avoiding access failures and complications. Arterial system evaluation should begin with bilateral blood pressure measurement. A difference of greater than 20 mm is suggestive of subclavian stenosis [32]. Further, all peripheral pulses should be present and strong. A modified Allen's test should be performed as a screening test for evaluating the competency of the palmar arch. Ultrasound imaging of the proposed arterial inflow should be performed. The optimal diameter of the brachial artery should be more than 1.6 mm [33] with a peak systolic velocity > 50 cm/s. A resistive index ≥0.7 is also proposed [34].

In patients who are at high risk for developing DASS, it is prudent to evaluate further with DBI and DBP measurements. There is no established numerical threshold value for DBI and DBP for development of DASS.

The incidence of arterial stenosis in an AV access is about 5–13% [35]. Traditionally, JXA stenosis has been treated by surgical revision using a jump graft or by creating a proximal neoanastomosis. JXA stenosis has a high recurrence rate after balloon angioplasty. The primary patency rates vary between 41 and 55% at 1 year [36, 37]. Recently there has been interest in using drug eluting balloons for treating JXA stenosis. A recent report using a paclitaxel coated balloon used after the standard balloon angioplasty for JXA showed a primary patency of 81% at 1 year [38]. A more recent meta-analysis comparing angioplasty versus drug eluting balloon(DEB), cutting balloon and stents demonstrated that DEB is more effective in treating JXA stenosis compared to PTA [39]. Further, patency rates of 81%, 60%, 53% respectively has been reported with DEB angioplasty at 12, 24 and 48 months respectively [40]. Standard balloon sizes for the arterial anastomosis are 4–5 mm whereas the venous side of the JXA is treated with 6–7 mm balloons. Placement of a stent in the JXA region is controversial.

The management of DASS is based on the clinical classification. Once the patient has stage IIb symptoms, endovascular/surgical therapy should be considered. Management further depends on the location of the access. Access at the wrist, with retrograde flow in the radial artery and hand ischemia can be remedied by ligating or endovascular coil embolization of the radial artery beyond the anastomosis. Management of access at the brachial artery requires more work up. A Doppler ultrasound to determine the flow will dictate the management options. A low to normal flow where the flow in a fistula is less than 800 ml/min or less than 1000 ml/min in a graft should be surgically corrected by using a technique referred to as proximalization of the arterial inflow (PAI) [41, 42]. During PAI, a bypass is created from a proximal artery (axillary artery) to the proximal portion of the fistula. This procedure is considered superior to the distal revascularization with interval ligation (DRIL) procedure for low-normal flow DASS. The primary patency of the bypass graft at 2 years ranges between 45 and 70% [43]. More proximal arterial disease affecting the subclavian or right brachiocephalic arteries are best diagnosed on CTA/MRA and treated with either endovascular stenting or surgical bypass.

In patients with high flow DASS, another surgical procedure, revision using distal inflow (RUDI), is the procedure of choice. An additional procedure to reduce the

flow includes banding. Banding can be done surgically without pressure measurement (blind banding), with pressure measurement (precision banding), or endovascular-surgical combined.

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Swing Point Stenosis

16

Gerald A. Beathard

Introduction

The most common complication associated with AV access is venous stenosis. A subgroup of venous stenotic lesions referred to as "swing point stenosis" are extremely problematic. These lesions develop where the vein that constitutes the arteriovenous fistula (AVF) or its drainage makes a sharp, curved angle.

The primary mechanism by which venous stenosis occurs is a derangement in wall shear stress (WSS). Based primarily upon data derived from arterial studies, it has been shown that wall shear stress (WSS) plays an important role in regulating the function of endothelial cells [1]. As soon as the AVF is opened there is an immediate pressure drop within the distal artery, due to the presence of the low resistance bypass outlet created by the access. As a result, blood flow increases several-fold immediately and continues to increase at a slower pace long-term [2–4]. This results in abnormal WSS at the blood vessel wall interface, stimulating remodeling of the artery characterized by an increase in vessel diameter and wall thickness. This process returns WSS toward normal [5–7]. However, because of the curved configuration of the swing points, low blood flow pockets and flow turbulence can develop [8, 9]. Low blood flow is associated with low WSS, the magnitude of which has a significant linear relationship to the increase in blood flow velocity. Neointimal hyperplasia, having an inverse relationship with WSS develops [10, 11]. As the low WSS induced neointimal hyperplasia progresses, it aggravates the problem by causing even more flow disturbance. With time, this process leads to the development of a stenotic lesion (Fig. 16.1).

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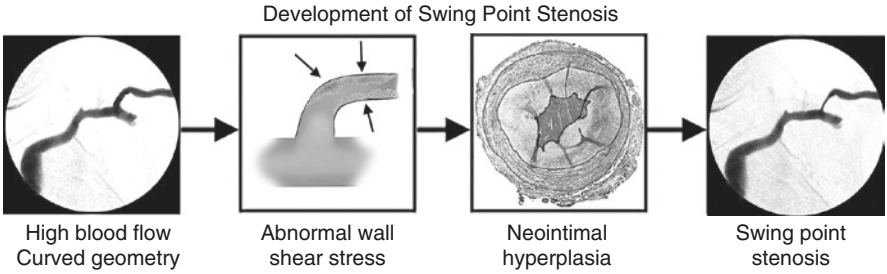


Fig. 16.1 Development of swing-point stenosis

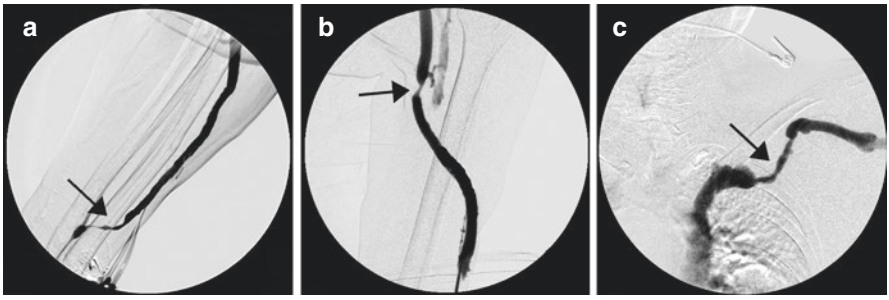


Fig. 16.2 Swing-point stenosis spectrum. (**a**) Juxta-anastomotic stenosis (arrow), (**b**) Brachial-basilic angle of transposition stenosis (arrow), (**c**) Cephalic arch stenosis (arrow)

Swing point lesions are very common. In a study of 278 patients with an arteriovenous fistula (AVF) associated venous stenosis, 45.7% (127/278) fell into this category [12]. Three swing point lesions have been characterized—(1) juxta- anastomotic stenosis (JAS) (Fig. 16.2a), (2) stenosis which develops at the angle of transposition (BATS) (Fig. 16.2b) of a brachial basilic AVF, and (3) cephalic arch stenosis (CAS) (Fig. 16.2c). Each of these lesions has a predilection for a type of fistula. Although JAS can occur with any type of AVF, when presented with a radial-cephalic fistula, the first lesion to consider is that of JAS. With a brachial-basilic transposition AVF, BATS should be the first suspect, and with a brachial-cephalic it should be CAS.

Juxta-Anastomotic Stenosis (JAS)

General

Although it is also seen later during the functional life of an AVF, the most common lesion resulting in failure to mature (primary failure) of an AVF is JAS [12–20]. This is defined as stenosis occurring within the first 3 to 4 cm of the AVF, immediately adjacent to the arterial anastomosis. The lesion results in luminal narrowing, decreased AVF blood flow leading to problems of maturation, and at times, early

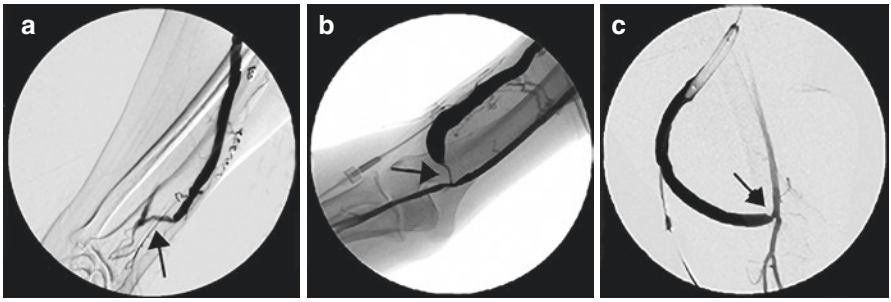


Fig. 16.3 Variants of Juxta-anastomotic lesion. (**a**) Juxta-anastomotic only (arrow), (**b**) Both JAS and anastomotic stenosis (arrow), (**c**) Anastomotic only (arrow)

thrombosis. Regardless of the etiology, the pathologic evaluation of this lesion is always consistent with neointimal proliferation. JAS is seen more commonly in with radial-cephalic AVFs (64–77%) [21–23] than in upper arm AVFs (39–56%) [21, 23]. Three lesion variations may be observed (Fig. 16.3) in the access in the zone adjacent to the anastomosis: juxta-anastomotic stenosis only, anastomotic stenosis only, and a combination of the two.

Etiology

Although it seems apparent that there is something related to either the way the anastomosis is created or its configuration that results in vascular injury culminating in juxta-anastomotic stenosis, the exact etiology is not clear. Possible mediators of vascular injury in this setting include: (1) direct hemodynamic injury due to non-laminar flow and oscillatory WSS, (2) surgical injury from suture site inflammation, and (3) twisting and torqueing of the peri-anastomotic venous segment at the time of AVF creation [24]. It is possible that the exact cause may vary from case to case and be a combination of causes. The possibility of surgical trauma related to mobilization and manipulation of the vein at the time of surgery has prompted some surgeons to advocate a microsurgical technique utilizing a operative microscope for AVF creation [25].

Using a computer model of the AVF, a study was conducted comparing the flow dynamics of a side-to-side versus end-to-side anastomosis at flow rates ranging from 600 to 1200 mL/min [26]. It was found that the most optimal WSS profile was generated by the side-to-side anastomosis. It has also been noted that the anatomical characteristics of the cephalic vein and radial artery predispose the radial-cephalic AVF to juxta-anastomotic stenosis due to torsion of the vein when the anastomosis is created. These two vessels are in different tissue planes. The radial artery lies below the deep fascia while the cephalic vein is at the level of the superficial fascia (Fig. 16.4). In addition, the vein is situated at a variable distance lateral to the artery. In the typical surgical procedure, this anatomical configuration necessitates significant movement of the vein in more than one plane to bring it into position for the

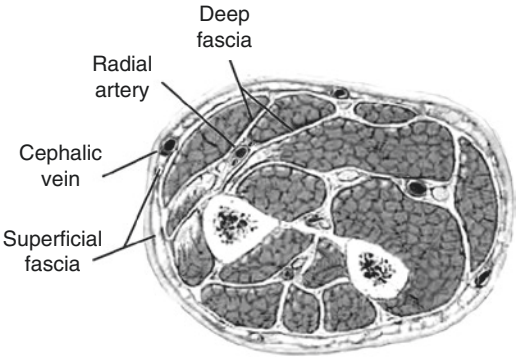


Fig. 16.4 Relationship of cephalic vein and radial artery at site commonly used for creation of radial-cephalic AVF

creation of the anastomosis. It has been theorized that this mobilization results in torsion which is made worse by the high blood flow of the newly created AVF and that this torsion is a major etiologic factor for the development of stenosis [27].

Prevention

In order to take advantage of the flow benefit of a side-to-side anastomosis and to avoid the torsion resulting from the typical technique for vein mobilization, an approach to the creation of the anastomosis referred to as the pSLOT (piggyback Straight Line On-lay Technique) was investigated [27]. In the initial study, this technique (n = 54) was compared with a cohort of cases where the anastomosis was created using the end-to-side technique (n = 57). The incidence of juxta- anastomotic stenosis was 16.7% and 40.3% in these two groups, respectively ($p = 0.01$) [27]. Although this was an observational study, the results obtained suggest that addressing the theorized etiological factors of anastomosis configuration and vein manipulation may be significantly beneficial.

Another approach to avoiding the adverse hemodynamics associated with the standard surgical procedure involves moving the artery rather than the vein to make the anastomosis. In a report of 53 cases in which radial artery deviation and reimplantation (RADAR) was performed to create a radial-cephalic AVF, the results obtained were compared with 73 controls in which the classical surgical procedure was utilized [28]. AVFs created with the RADAR technique had superior rates of maturation of 75% vs 45% and 92% vs 71% at six weeks and three months, respectively. Primary patency and cumulative patency were 93% vs 53% ($p < 0.0001$) and 100% vs 89% ($p = 0.001$) at six months. The incidence of juxta-anastomotic stenosis in the RADAR group was 2% vs 41% in the standard group ($p = 0.001$) and the incidence of re-intervention was 10% vs 74% ($p = 0.001$), respectively.

Diagnosis by Physical Examination

JAS can be easily diagnosed by physical examination of the anastomosis and adjacent vein [29, 30]. Normally, a very prominent thrill is present at the anastomosis (Fig. 16.5). In the absence of abnormalities, the pulse is soft and easily compressible. With juxta-anastomotic stenosis, a very forceful pulse is felt at the anastomosis. The thrill, which is normally continuous, is present only in systole. In some instances (severe lesion) it may be very short and even difficult to detect. As one moves up the vein from the anastomosis with the palpating finger, the pulse goes away rather abruptly as the site of stenosis is encountered. Above this level, the pulse is very weak and may be difficult to detect. The stenosis itself can frequently be felt as an abrupt diminution in the size of the vein, almost like a shelf. These findings are very typical for this lesion.

Management

This lesion is amenable to treatment (Fig. 16.6). There are two alternatives for therapy, percutaneous angioplasty (PTA) [13–16, 21, 23, 31–39] or surgery [36, 37, 40–46]. Unfortunately, there are no randomized controlled studies to determine which of these two modalities represents the better choice for therapy. Reports that have compared the two are primarily from surgical groups and have concluded that surgery provides a better primary patency then does angioplasty [44–46]. The

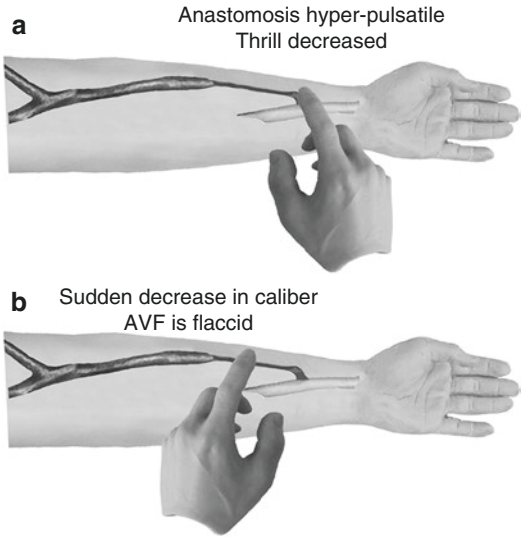


Fig. 16.5 Diagnosis of juxta-anastomotic stenosis by physical examination. (**a**) palpate anastomosis, (**b**) palpate remainder of AVF starting proximal to the anastomosis

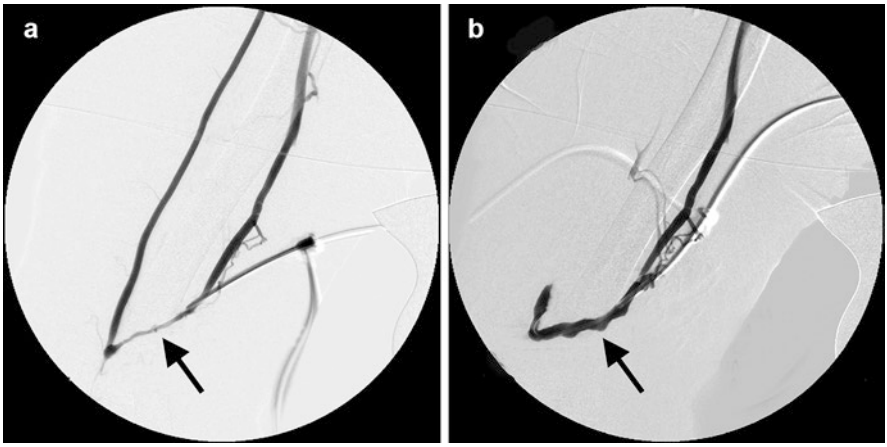


Fig. 16.6 Juxta-anastomotic stenosis. (**a**) Lesion, (**b**) Post PTA

surgical approach has been to either create a neo-anastomosis higher on the artery or to interpose a segment of expanded polytetrafluoroethylene (ePTFE) graft.

A retrospective study [46] compared the outcome and cost of surgery and PTA in the preemptive repair of juxta-anastomotic stenosis in distal forearm AVF. Sixty-four AVFs with >50% venous juxta-anastomotic stenosis were considered: 21 were treated surgically (11 with proximal neo-anastomosis and 10 with PTFE interposition graft) and 43 by PTA. After treatment, the cases had quarterly access blood flow measurements. End points were restenosis and procedure failure rate (either re-intervention by another technique or access loss). Initial procedural success was 100% for surgery and 95% for PTA ($P = 0.539$). Restenosis rate was 0.168 and 0.519 events/AVF-year for surgery and PTA, respectively ($P = 0.009$). The type of procedure was the only variable that was significantly associated with restenosis, the adjusted relative risk being 2.77-fold higher (95% confidence interval 1.07 to 7.17; $P = 0.036$) after PTA than following surgery. There was no significant difference in the assisted primary patency between the two groups. The procedure failure rate was 0.110 and 0.097 events/AVF-year for surgery and PTA, respectively ($P = 0.736$). The cost profile also was similar for the two procedures.

The authors of this study [46] concluded that while the restenosis rate was higher after PTA, with strict surveillance for restenosis, the two procedures showed similar assisted primary patency and cost, suggesting that they should be considered equally valid, complementary alternatives in the preemptive treatment of juxta-anastomotic stenosis in forearm AVF. The authors of another study [44] concluded that a reasonable approach to juxta anastomotic stenosis would be to perform a PTA first, reserving the surgical approach as a back-up in case of failure. This was based upon their findings that although the primary patency following surgical treatment was

superior, the similar assisted patency and the fact that PTA does not exclude a later surgical correction if required.

Basilic Angle of Transposition Stenosis (BATS)

General

The basilic vein is commonly used for creation of AVFs in the upper arm, with anastomosis to the brachial artery. The normal position of the basilic vein is not optimal for use as a dialysis access (Fig. 16.7). At the level of the elbow, the basilic vein is superficial, coursing along the medial aspect of the upper arm, where it lies within the median bicipital sulcus. It continues up to about the middle third of the sulcus where it pierces the brachial fascia and continuous centrally at a deeper level. The brachial fascia varies in thickness, but over the medial aspect of the upper arm it is relatively thick and fibrous. Because of its medial location and the depth of its most proximal portion, superficialization and transposition are necessary to make it easily accessible for use as a clinically functional AV access.

The angle created when the vein is moved to its optimal position for use as a hemodialysis access can lead to the development of venous stenosis. Since the upper portion of the vein is bound down by the brachial fascia, a sharp angle (angle of transposition) can be created when the lower portion is moved superficially and laterally (Fig. 16.8a). This is the point at which stenosis occurs due to turbulence, although surgical manipulation and mechanical factors related to the brachial fascia may also be contributory. A more gradual transition (Fig. 16.8b) at the time of transposition results in less blood flow turbulence and a decreased tendency for the formation of stenosis.

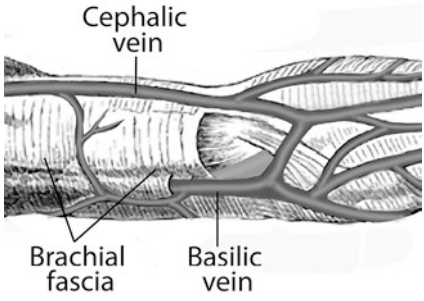


Fig. 16.7 Relationship of basilic vein to brachial fascia

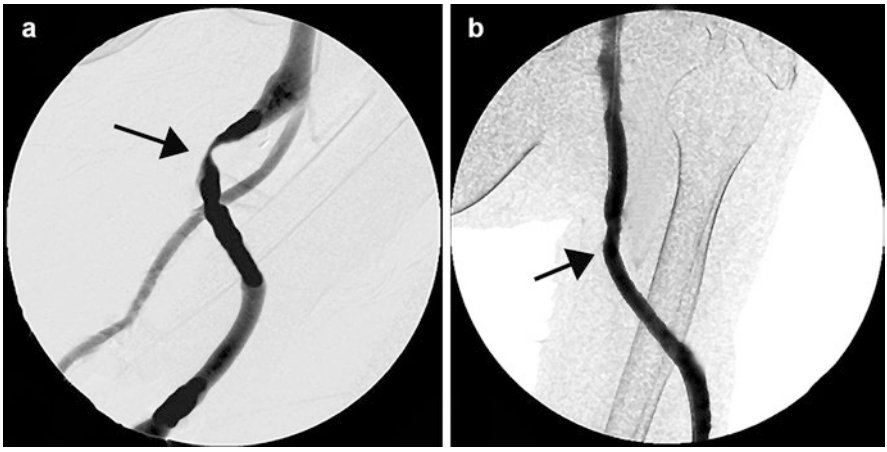


Fig. 16.8 Comparison of angles at the mobilization point for brachial-basilic AVF. (**a**) Acute angle (arrow), (**b**) gradual transition with more obtuse angle (arrow)

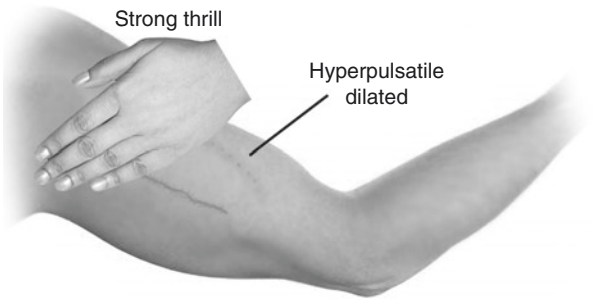


Fig. 16.9 Diagnosis of BATS by physical examination

Stenosis at this site is the most common complication associated with a brachial-basilic AVF [47, 48]. However, the frequency with which it is observed varies depending upon local practices, since it is at least in part iatrogenic. In one series, the incidence of BATS in failing brachial-basilic AVFs was 60% [21]. In another, the incidence was 74% and accounted for 87% of all interventions on this type of access [47]. When the BATS lesion occurs, it is frequently a high-grade stenosis associated with a marked increase in intra-luminal pressure. This is followed by a progressively decreasing blood flow which promotes the development of thrombosis. The increased AVF intra-luminal pressure also causes the vessel to dilate, resulting in aneurysm formation.

Diagnosis by Physical Examination

BATS can also be diagnosed with a reasonable degree of confidence by physical examination (Fig. 16.9). On palpation using the palm of the hand, the distal vein and cannulation zone of the AVF is hyperpulsatile. A prominent thrill can be felt over

the upper portion of the AVF transposition scar (the site of the angle of transposition) on palpation with fingertips. Auscultation over the site reveals a loud systolic bruit.

Management

The primary treatment for BATS has been angioplasty (Fig. 16.10). However, there are very few studies related specifically to the treatment of this problem. In one series [47] of 93 patients with a transposed brachial- basilic AVF, the incidence of stenosis was 54%. In 74% of these, the lesion was a BATS. These cases were treated with angioplasty and had a primary patency at 1- year of 42%. Cumulative patency at 1, 2 and 3- years was 68%, 58% and 53%, respectively. Repeat angioplasties were required in 65% of the cases with 50% requiring 3 or more and 29% requiring 4 or more interventions. The median time between angioplasties was 75-days.

Only a single study dealing with the treatment of BATS by the insertion of a stent has been reported. In this study [49], 37 cases with BATS were treated with stent grafts. Cases served as their own control by analyzing lesion and access patency pre- and post-stent placement. Lesion primary patency at 6 and 12-months pre-stent was 29% and 3%, respectively and post-stent was 57% and 40% respectively. Lesion assisted primary patency at these same intervals was 39% and 13% pre-stent and 91% and 80% post stent, respectively. Access primary patency at 6 and 12-months pre-stent was 29% and 3% and post-stent was 26% and 19%, respectively. Assisted primary patency for the access pre-stent was 36% and 10% at the same time intervals and post stent was 82% and 75%, respectively. The intervention rate for the lesion decreased from 0.47/month to 0.13/month following stent placement. The intervention rate for the accesses went from 0.50/month prior to stent placement to 0.15/month afterwards.

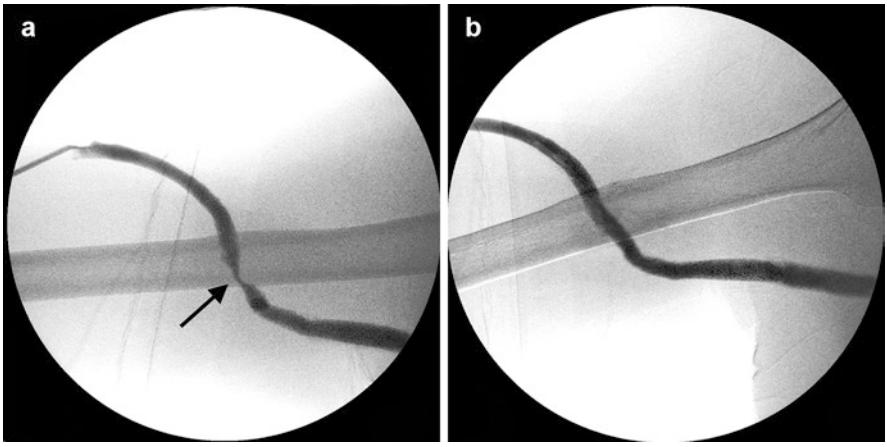


Fig. 16.10 Angioplasty of BATS lesion. (**a**) Lesion (arrow), (**b**) Result of treatment

Cephalic Arch Stenosis (CAS)

General

The third segment of the dialysis access circuit included under the heading of swing point is a naturally occurring segment of the cephalic vein known as the cephalic arch. This structure is very important to dialysis access maintenance due to the high incidence of cephalic arch stenosis (CAS). Because of its frequency, compounded by its resistance to treatment and frequency of complications of treatment, problems in this area are a common cause of AVF failure making it the most problematic of the swing point stenosis group.

After ascending along the lateral surface of upper arm, the cephalic vein transitions from a superficial position to a deep subcutaneous one. It passes beneath the clavicle and pierces the clavipectoral fascia to join the axillary vein to form the subclavian vein. Due to its curvature, this segment of the vein has been referred to as the cephalic arch. It is the most proximal (central) segment of the cephalic vein just before its confluence with the axillary vein, and is approximately 5 cm in length [50].

The terminal portion of the cephalic vein may have several anatomical variation. A single channel that joins the axillary vein to form the subclavian vein is the most common variant (Fig. 16.11a); however, a double (Fig. 16.11b) and even a triple arch is occasionally encountered. The limbs of these multiple-arch variants may join the axillary vein or combinations of the axillary vein and the external or internal jugular veins. Rarely, a single arch can drain directly into the external jugular, internal jugular, or subclavian vein.

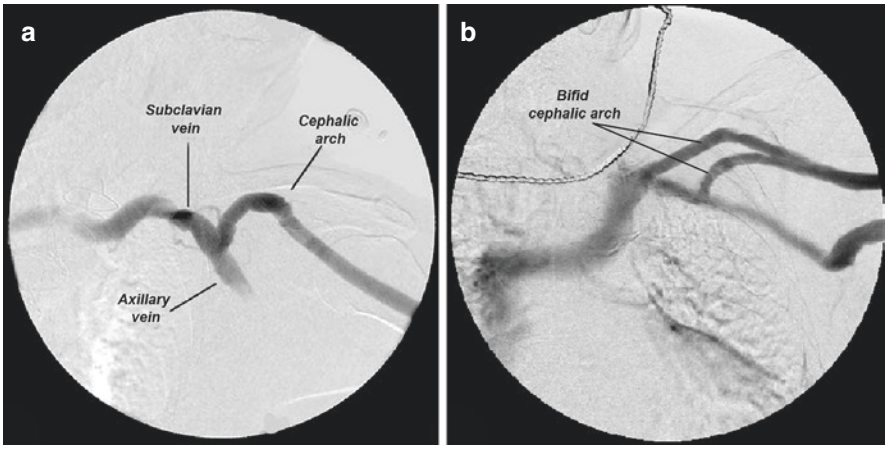


Fig. 16.11 Variants of cephalic arch. (**a**) Single arch, (**b**) Bifid cephalic arch

Unique Nature of the Cephalic Arch

The cephalic arch is a unique vascular structure because of three features. First, its anatomical configuration—the curved arch. With the increased blood flow that occurs with dialysis access creation, this configuration results in areas of decreased WSS leading to neointimal hyperplasia and stenosis. Second, one or more valves are located in the cephalic arch. It is well known that venous stenosis has a predilection for developing at valve sites [51], and while the location of valves within veins in general is somewhat variable, their location in this segment of the cephalic vein is rather constant. There are, on average, two valves in the cephalic arch in most individuals [52, 53]. The most common location of a valve in the cephalic vein (92%) is just distal (3 mm) to the confluence with the axillary vein [52, 53]. Third, the cephalic arch is mechanically limited in its ability to dilate in response to the increased blood flow of the developing AVF because of anatomical relationships associated with the clavipectoral fascia and associated structures (Fig. 16.12). The clavipectoral fascia is a dense membrane occupying the space between the pectoralis minor muscle and the subclavian vessels. It lies over the axillary vessels and nerves and is pierced by the cephalic vein, thoracoacromial artery and vein, and lateral pectoral nerve. As it passes through the foramen, the density of this membrane can inhibit the vein's ability to dilate. In addition, it is possible for the vein to be compressed by neighboring anatomic structures.

Although these unique anatomical features increase the risk for turbulent blood flow in this portion of the cephalic vein, hemodynamic studies using computational fluid dynamic modeling have shown that an increased volumetric flow rate plays a major role in the deranged hemodynamics within the cephalic arch [9, 54].

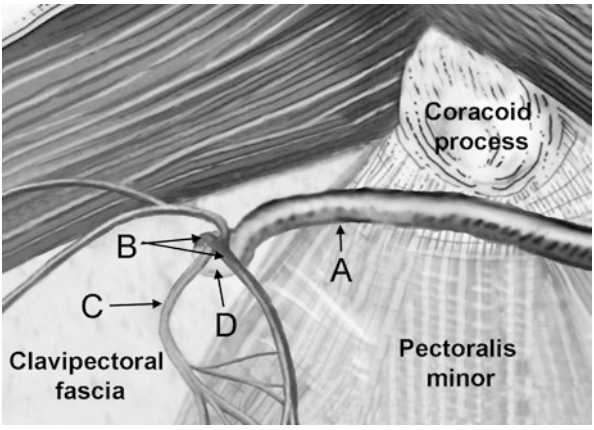


Fig. 16.12 Structures passing through clavipectoral fascia orifice. (**A**) Cephalic vein, (**B**) Thoracoacromial artery (vein not shown), (**C**) Lateral pectoral nerve, (**D**) Subclavian vein

Etiology of CAS

High access blood flow is a significant problem in the hemodialysis patient. A high blood flow rate has been shown to result in congestive heart failure, pulmonary hypertension and the development of a mega-fistula (diffuse aneurysmal dilatation). It also plays a major role in the etiology of CAS. Although its unique anatomical features increase the risk for turbulent blood flow in cephalic arch, hemodynamic studies using computational fluid dynamic modeling have shown that an increased volumetric flow rate plays a major role in the deranged hemodynamics that occur within the cephalic arch resulting in the development of neointimal hyperplasia [9, 54]. High blood flow appears to be important both in the development [8, 9, 54–56] and recurrence [55, 57] of CAS.

Incidence of CAS

Although CAS was found to account for 50% of stenoses in upper arm AVFs in one study [23], it has been reported in 15 to 18% of cases of AVF failure [12, 58]. Since the cephalic arch contributes to the drainage of both the brachial-cephalic and the radial-cephalic AVF, CAS can be seen with either type of access. However, it is seen more frequently with the former [12, 56, 58, 59]. Long-term patency of a brachial-cephalic AVF is significantly shorter than a radial-cephalic AVF [60, 61]. This difference in access longevity has been attributed at least in part to the difference in the incidence of CAS [59]. In one report (n = 177) [58], a 39% CAS incidence was documented in brachial-cephalic AVFs compared to 2% in radial-cephalic AVF. In another report (n = 127) [59], the difference was 77% and 20% and in a third report (n = 241) [62], 97% of CAS cases occurred with a brachial-cephalic and 3% occurred with a radial-cephalic AVF. The explanation for this difference in incidence appears to be related to differences in blood flow in the cephalic arch [56]. Blood flow through this venous segment is generally higher with a brachial-cephalic than a radial-cephalic AVF for two reasons. First, a brachial artery AVF has a higher blood flow rate than a radial artery AVF. Second, all the blood flow of a brachial-cephalic fistula goes through the cephalic arch, while in a radial-cephalic AVF there is the possibility for a division of blood flow into both the basilic and the cephalic veins. In some instances, all or a large portion of the blood flow completely misses the cephalic arch (Fig. 16.13). Since the cephalic vein is not involved in its drainage, CAS is not seen in association with a brachial-basilic AVF.

CAS in Diabetics

A special relationship between diabetes and CAS has been documented in a number of reports: (1) the time from access creation to the development of the lesion does not differ between diabetics and nondiabetics [54], (2) lesion recurrence following treatment is higher in diabetics [62], and (3) there is a decreased incidence of CAS in diabetics [56, 58, 59, 63].

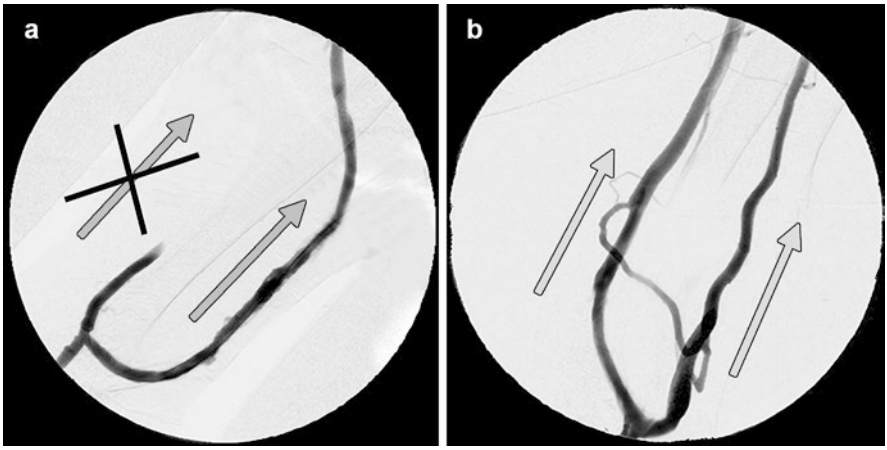


Fig. 16.13 Differences between potential for cephalic arch blood flow. (**a**) Brachial-cephalic AVF, flow is only through cephalic and cephalic arch, (**b**) radial-cephalic AVF with potential for blood flow divided between basilic and cephalic veins

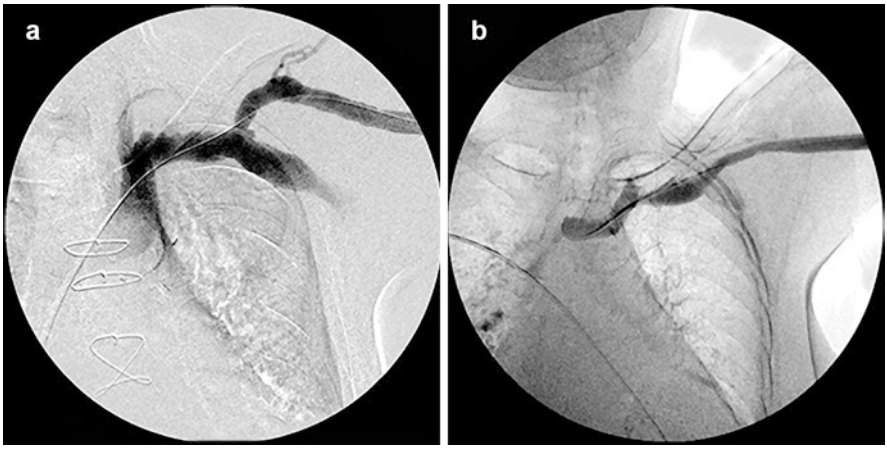


Fig. 16.14 Variations in cephalic arch geometry. (**a**) Small R/d ratio, (**b**) Large R/d ratio

The explanation for the lower incidence of CAS in diabetics is not clear. Cephalic arch geometry has been suggested as a possible factor. In a study of 45 cases [63], 12 with diabetes and 33 without, two distinct types of cephalic arch geometries were noted. These two types were defined by differences in the ratio between the radius of the cephalic arch and the diameter of the vein (*R/d ratio*). In addition, there was a corresponding difference in the overall angle created by the cephalic arch (arch angle) (Fig. 16.14). Patients having diabetes mellitus showed a significant probability of having a larger *R/d* ratio and wider arch angle (Fig. 16.14b). In

addition, the direct relationship between increased blood flow velocity and low WSS has been reported to be less significant in the diabetic patient [8].

It has been suggested that the difference in cephalic arch geometry in these patients might be due to differences in blood flow dynamics [63]. This possibility was suggested by reports that diabetic patients frequently have smaller arterial diameters, higher arterial pressures and higher blood flow velocities [64, 65]. Studies have shown that the angle of the cephalic arch can change in response to blood flow velocity. In one study [9] in which serial measurements of cephalic arch geometry were made in relationship to blood flow hemodynamics, the average arch angle was found to decrease by approximately 16° by the third month in conjunction with a tenfold increase in blood flow velocity.

Variability in Location of Stenosis

There is significant variability in the exact location of stenotic lesions that occur in the cephalic arch. The most common site for CAS is the point of confluence of the cephalic arch and axillary vein to form the subclavian vein [54] (Fig. 16.15a). However, it may also develop more peripherally (Fig. 16.15c), and in some cases the lesion may extend into the subclavian vein (Fig. 16.15b).

In one study of 69 cases with CAS, the cephalic arch was divided into four domains starting immediately distal (peripheral) to the beginning of the arch and extending through the point where the cephalic joins the axillary to form the subclavian vein [54] (Fig. 16.16). Frequently, lesions occurred in more than one domain; however, the most frequent site of occurrence (single or in combination) was the terminal zone of the cephalic arch, immediately adjacent to its confluence with the axillary vein to form the subclavian, referred to as domain IV. With total occlusion, collaterals form as with other types of venous stenosis. In most cases the primary collateral drainage involves the jugular system, as shown in Fig. 16.17.

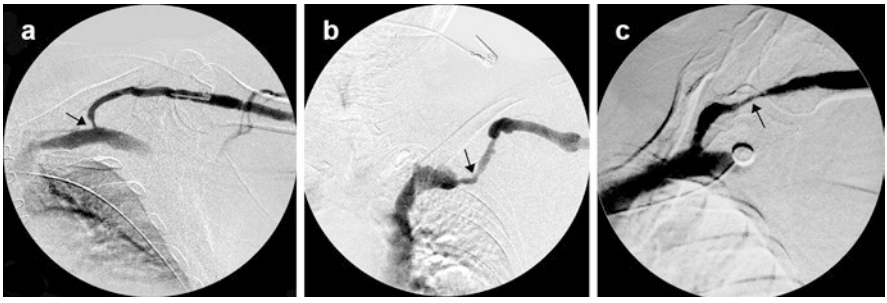
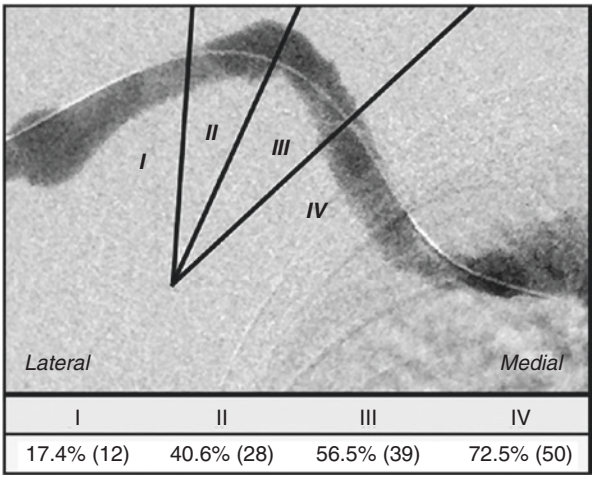


Fig. 16.15 Three variations in lesion distribution (arrows). (**a**) Lesion at the junction with axillary vein, (**b**) Lesion extending into subclavian, (**c**) Lesion peripheral to junction with axillary vein



I	II	III	IV
17.4% (12)	40.6% (28)	56.5% (39)	72.5% (50)

Fig. 16.16 Domains of cephalic arch showing incidence of CAS in each

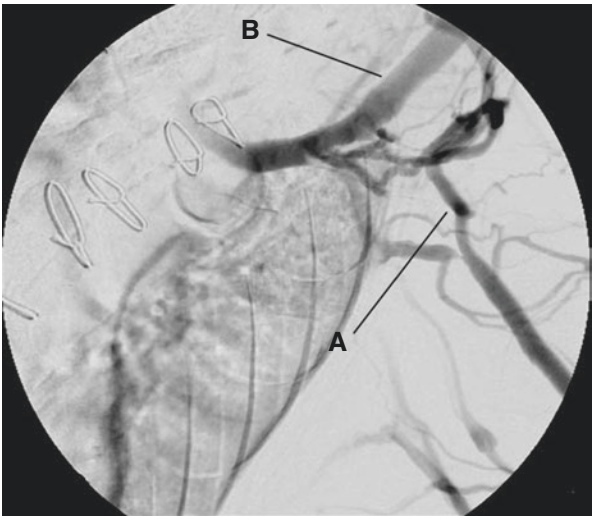


Fig. 16.17 Occluded cephalic arch (**a**). Drainage through collaterals to internal jugular (**b**)

Diagnosis by Physical Examination

When presented with the patient with a dysfunctional brachial- cephalic AVF, CAS should be strongly considered due to the frequency with which this lesion occurs. By physical examination (Fig. 16.18), the fistula is hyperpulsatile to palpation with the palm of the hand. In addition, the fistula is dilated and may be aneurysmal. The diastolic component of the normal thrill and bruit present at the anastomosis is shortened. Palpation with the tips of the fingers of the infra-clavicular area often

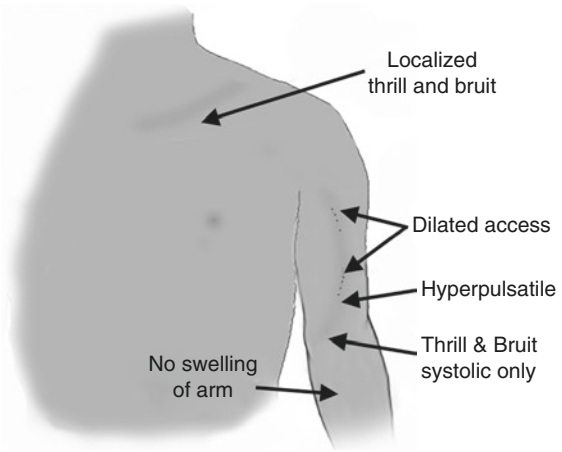


Fig. 16.18 Diagnosis of cephalic arch stenosis by physical examination

reveals the presence of a thrill. Auscultation this area reveals a loud bruit which is primarily systolic. The same physical findings may be found in a patient with subclavian stenosis. If there is edema of the arm, CAS can be ruled out since edema only occurs with a central venous lesion. In the absence of arm edema, these two lesions are difficult to distinguish from each other by physical examination.

Prevention of CAS

The relationship between high blood flow and CAS offers an opportunity for incorporating prevention into individual patient access planning. An upper arm AVF created using the brachial artery for the anastomosis is associated with a high-flow volume because of the size and pressure of the associated artery. This increases the risk for development of CAS. This risk is reduced by using the proximal radial artery which is of smaller size with a lower pressure and blood flow rate [66–68]. In a study in which patients with a proximal radial-cephalic AVF (upper arm) was compared to those with a brachial-cephalic AVF, blood flow volumes were 735 ± 344 mL/min versus 1060 ± 578 mL/min [68]. AVF maturation and primary, assisted primary and cumulative patency rates did not differ significantly between the two groups. The incidence of CAS in the brachial-cephalic group was twice of that noted in the proximal radial-cephalic group.

Management of Cephalic Arch Stenosis

The general indication for treatment of any venous stenosis lesion is the presence of ≥50% decrease in the luminal diameter of the vessel. However, no lesion should be treated based only upon an anatomical criterion. The stenosis must be shown to be clinical-hemodynamically significant (Table 16.1). Multiple primary and secondary

Table 16.1 Criteria for judging clinical-hemodynamic significance

Physical examination
• Hyperpulsatility of fistula
• Abnormal thrill palpated in subclavicular area
• Abnormal bruit heard in subclavicular area
• Failure of fistula collapse when the arm is elevated
Clinical parameters
• Inability to achieve the target dialysis blood flow
• Prolonged bleeding from needle puncture sites for three consecutive dialysis sessions
• Unexplained (>0.2 units) decrease in the delivered dialysis dose (Kt/V) on a constant dialysis prescription without prolongation of dialysis duration
• Elevated venous pressure recorded during hemodialysis for three consecutive dialysis sessions

Table 16.2 Management options for CAS

Primary
Angioplasty
Flow reduction
Secondary
Intravascular Stent placement
Surgical intervention
Central transposition of cephalic vein
Bypass

modalities have been used to treat CAS. These include percutaneous balloon angioplasty using conventional and cutting balloons, endovascular stent insertion using both bare-metal stents and stent-grafts, and surgical interventions (Table 16.2). Despite multiple publications on the topic, it is not possible to draw any evidence-based conclusions related to best practices for management of CAS, due to the poor quality of studies that have been reported [69]. Unfortunately, most studies demonstrate a marked degree of heterogeneity in their initial design and their presentation of data. Very few prospective studies have been reported, and randomized clinical trials are rare and uniformly underpowered. Some reports have been novel proof-of-concept studies as opposed to assessments of efficacy compared to other forms of treatments. A lack of uniformity in the reporting of outcomes (patency data) makes comparison among studies difficult, if not impossible.

Treatment Modalities

The goal of CAS management should be to restore and maintain access function for the longest possible period of time with the least number interventions. However, it is difficult to draw any evidence-based conclusions related to best practices for management of CAS due to the poor quality of studies that have been reported [69, 70]. As a result, multiple modalities have been used to treat CAS without defining a single unified management strategy. These modalities can be roughly divided into primary, ancillary and secondary categories (Table 16.2).

Primary Treatment

Percutaneous Balloon Angioplasty (PTA)

PTA with either a high-pressure or an ultrahigh-pressure balloon has become the standard of practice for treatment of venous stenosis, particularly for CAS (Fig. 16.19). However, treatment of CAS with PTA has been reported to have problems when compared to other sites. Although generally successful, the results reported for CAS have varied considerably [50, 58, 62, 71–74]. CAS lesions are resistant to dilatation (less so with an ultrahigh pressure balloon) and venous rupture is more frequently seen, with an incidence of 5 to 6% [23, 50, 57, 58]. A high recurrence rate has resulted in shorter long-term patency for these lesions with repeated intervention ranging from 1.5 to 3.5 episodes per year [58, 75]. Efforts should be made to assure optimal results with PTA since residual stenosis has been shown to be associated with lesion recurrence. Proper balloon sizing and use of an ultrahigh pressure balloon if necessary in order to avoid residual stenosis are recommended [57]. The severity of the lesion at first encounter, vessel wall diameter proximal to the stenosis and average venous pressure have also been shown to increase the risk of recurrence [55]. As discussed below, there is reason to avoid angioplasty in many cases of CAS as the primary treatment.

Blood Flow Reduction

Access blood flow rate correlates with both the development [8, 9, 54–56] and recurrence of the CAS lesion [55, 57]. Therefore, all patients with CAS should have their access blood flow evaluated as a guide for management planning. If excessive, blood flow reduction should be considered as a primary treatment [76] or at least as an adjunct procedure [77]. "Precision-banding" refers to banding performed in conjunction with techniques for lumen reduction and change in access blood flow. To assure success, accurate intra-operative flow measurements to judge the degree of

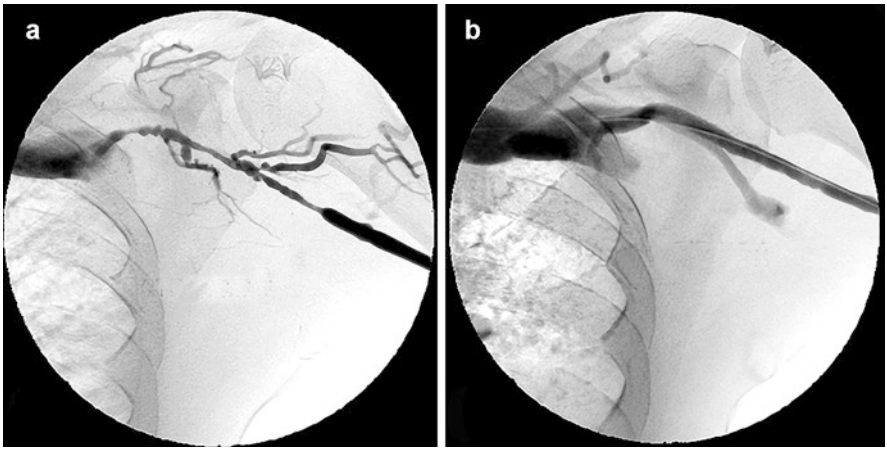


Fig. 16.19 Angioplasty of cephalic arch stenosis, pre and post

access flow restriction should be considered mandatory [78]. In one report, significant changes in access blood flow were noted with only 0.5 mm reduction in lumen diameter achieved with banding [79].

In a study of patients who had undergone two or more prior interventions for CAS [76], blood flow reduction by precision banding reduced the recurrence rate from 3.34 episodes to 0.9 per access per year ($p < 0.001$). The average follow-up time was 14.5-months (range, 4.8 to 32-months). Lesion patency at 3-, 6-, and 12-months post-procedure was 91%, 76% and 57%, respectively.

Precision banding can be performed using either a surgical or an endovascular approach. Both techniques are minimally invasive procedures [79–82]. Although successful endovascular banding has been reported in patients with an AVG, it can be problematic. The walls of a prostetic graft are not flexible and may fold into the lumen, creating a potential problem. Since many of these cases are managed by interventionalists, endovascular precision banding has become the standard of practice in many locations. Endovascular precision-banding is achieved by using some type of precise sizing dowel placed either intraluminally or extraluminally to assure that a controlled degree of luminal restriction has been achieved. An angioplasty balloon, either 4 or 5 mm [76], a vascular dilator ranging in size from 10 to 14 French (3.3 to 4.2 mm) [83] have been used as an intraluminal sizing dowel. Extraluminal precision banding (Fig. 16.20) using a vascular dilator or a coronary dilator of 4 mm size [79] has also been shown to be effective in reducing access blood flow [79, 81]. Use of a vascular dilator has a distinct economic advantage by voiding the expense of an angioplasty balloon that will only be used as a sizing dowel. Extraluminal dilator-assisted banding also offers several advantages over intraluminal. Fluoroscopy is not required; it can be done with ultrasound guidance. It is more time efficient and economical than intraluminal banding. This plus the fact it is equally successful makes it the more attractive procedure.

Banding should be accompanied by ultrasound access blood flow measurement immediately post-banding to assess the effect and adjust the degree of banding using objective blood flow criteria. Currently, there is no generally accepted definition for normal blood flow or when it is too high in a brachial-cephalic AVF. Generally, the access flows in an upper arm AVF are in the range of 900–1500 mL/min

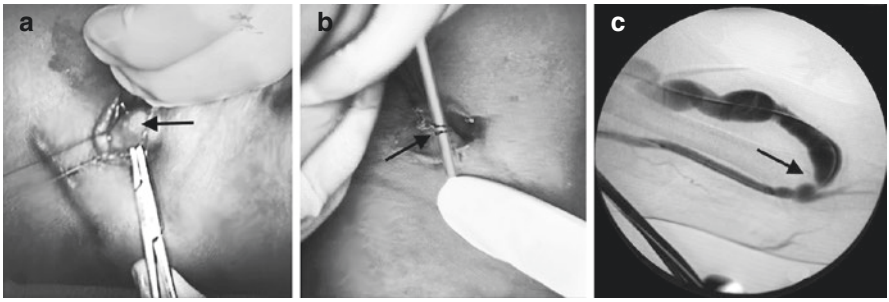


Fig. 16.20 Extraluminal precision banding of CAS. (**a**) Exposure of juxta- anastomotic zone of AVF, (**b**) Extraluminal ligation over vascular dilator, (**c**) Radiographic appearance of final results

[84–86]. Normal blood flow has been defined by some as 800 mL/min for an AVF and up to 1000 mL/min for an arteriovenous graft (AVG) for a patient with a blood pressure in the normal range [87, 88]. Clinical judgment is required to adjust these normal levels for a case with either low or high blood pressure. Based upon information available it seems reasonable that if AVF blood flow exceeds 1000 to 1200 mL/min, precision banding should be considered to reduce the level to a range of 500 to 700 mL/min. Post-banding, access patency rate is good when blood flow is reduced to levels no lower than 500–600 ml/min for AVFs and 600–700 mL/min for AVGs [78, 89]. Using Doppler flow ultrasound (DUS) to assess blood flow volume, the measurement should be made from the brachial artery at least 5 cm proximal to the anastomosis regardless of whether or not one is dealing with a radial or a brachial artery-based AVF [70]. In cases of high bifurcation of the brachial artery, the measurement should be made above the bifurcation.

Secondary Treatment

There are two choices for secondary therapy, stent placement and surgical intervention. The indications for these alternatives should be individualized, especially for the surgical alternative which is more invasive.

Intravascular Stent Placement

Although studies relating specifically to the treatment of CAS are limited in number, both bare-metal stents (BMS) and stent-grafts (SG) have been used for treatment failures or recurrences [50, 74, 90–93] (Fig. 16.21). In-stent stenosis is common in venous stenosis lesions treated with a BMS. In one study of CAS treated with a BMS, 70% of cases developed restenosis within three months of the index intervention [90]. Although small numbers of cases have been involved, studies using a SG have reported a decreased reinvention rate attributed to the graft material covering of the stent preventing neointimal hyperplasia ingrowth. In one study [90], interventions per patient year were 1.9 in the BMS and 0.9 in the SG cohort. In a second study [74], the SG cohort required 1 reintervention per access year in

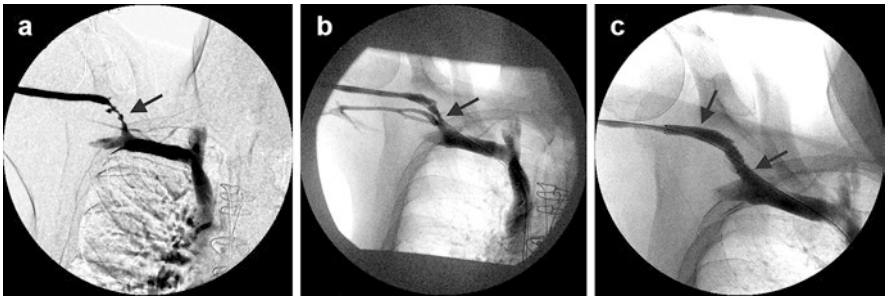


Fig. 16.21 Stenting of elastic cephalic arch lesion. (**a**) The lesion (arrow), (**b**) Lesion after PTA with large residual (arrow), (**c**) Lesion after stent placement (the stent lies between the two arrows)

comparison to 2.54 for PTA and 2.74 for BMSs based upon historical data. The decision to place a stent for the treatment of CAS should also take into consideration its effects on a future surgical option.

Surgical Management of CAS

Surgical interventions for the management of CAS include central transposition (outflow relocation) and bypass (Table 16.2). The use of patch angioplasty has been used in the past, but its use has decreased considerably because the procedure involves major surgery and the success rate of other approaches is equal or better. Even though appearing to be a more definitive therapy, the surgical approach to CAS has not been widely employed. While endovascular procedures are easily performed with sedation/analgesia, surgical intervention generally requires general anesthesia [50]. Hence, surgical options are usually considered later when CAS is recurrent.

Central Transposition Central transposition of the cephalic vein (outflow relocation, or "turn-down") is a surgical procedure in which a venovenostomy is created between the proximal end of the cephalic vein and the axillary or upper brachial vein (Fig. 16.22). Fortunately, CAS lesions are generally short and focal and it is unusual to have a problem with available vein length necessary for the transposition. In most cases, the procedure can be performed even it involves a long segment of stenosis [94]. Once freed, the vein is transposed by passing it through a subcutaneous tunnel to the upper basilic/axillary vein where an end-to-side venovenostomy is created to restore central blood flow.

Although all reported studies are based on a small cohort of cases, the success rates of this procedure has been superior compared to PTA [50, 71, 73, 77, 94–98]. However, when comparing these two modalities, it is important to note that the two groups being treated are not equal. While PTA is generally the primary treatment, central transposition is reserved for PTA failures. The transposition procedure is not without problems. The development of venous stenosis at the venovenostomy site can occur. However, its frequency is less than the CAS recurrence rate with PTA and the response to treatment is better. Reintervention rates per year following translocation have been significantly reduced in comparison to those recorded before the procedure. Reduction of reintervention rates from 3.5 to 1 [71], 2.5 to 1.5 [99], and 1.9 to 0.4 [77] per patient per year have been reported. The majority of these reinterventions are required for stenosis that develops at the venovenostomy site.

It has been reported that primary patency rates are diminished in cases in which the CAS lesion has been previously treated with PTA. In one study [97], the primary patency rates of the transposed cephalic vein at 12-month was 58% among those without and 12% in those with prior PTA of the proximal cephalic vein ($p = 0.003$).

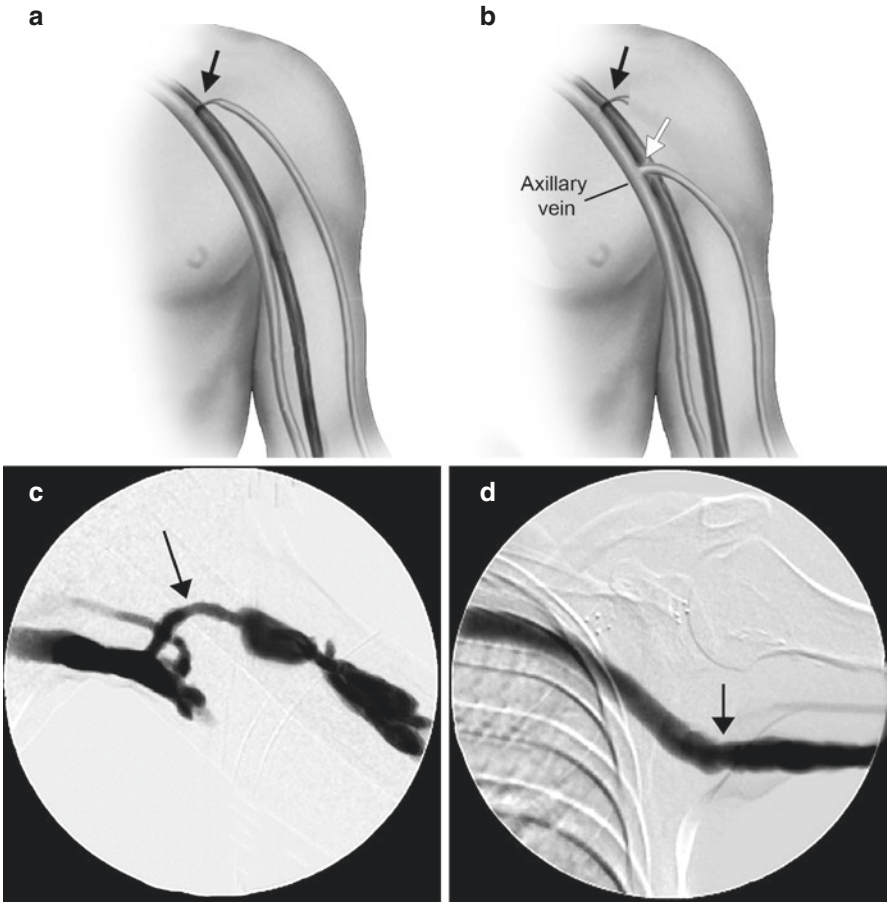


Fig. 16.22 Translocation procedure. (**a**) Drawing of lesion (black arrow), (**b**) Drawing of procedure (Old site of cephalic arch—black arrow, site of new venovenostomy—white arrow), (**c**) Lesion in cephalic arch (black arrow), (**d**) Site of venovenostomy (black arrow)

Bypass Surgical bypass is performed by tunneling graft material, either saphenous vein or expanded polytetrafluoroethylene (ePTFE), from the proximal uninvolved end of the cephalic vein to the jugular, axillary, or subclavian veins. A study including 31 cases in which a bypass procedure was performed with multiple treatment modalities [50]. The conduit used for the bypass was the saphenous vein in 70% of the cases and expanded polytetrafluoroethylene in the remainder, while the axillary vein was the outflow conduit in 81% and the internal jugular or subclavian vein was utilized in the remainder. Primary patency, assisted primary patency and cumulative patency in this cohort at 6 and 12-months were 95% and 91%, 99% and 91%, and 98% and 96%, respectively. As is the case with transposition, stenosis at the venous anastomosis can occur. This event was noted in 16% of cases.

Proposed Algorithm for the Management of CAS

There is inadequate evidence to construct a single unified management strategy for the treatment of CAS. However, the first consideration in management should be directed toward the pathophysiology if possible. The pathophysiology of CAS is intimately related to the access blood flow rate. Hence, it is recommended that the first step in management for any patient with CAS begin with an assessment of this parameter.

CAS should not be considered for treatment unless it has >50% stenosis and is clinical-hemodynamically significant (Table 16.1). However, there is a significant variability with what is considered to be clinical-hemodynamically significant. Cases eligible for treatment can generally be classified into two categories, (1) those with normal or low blood flow, 800 mL/min for an AVF and up to 1000 mL/min for an AVG and (2) those with high blood flow, i.e., greater than these levels. While flow measurement using DUS is necessary for accurate distinction between the two groups, cases can be recognized with reasonable confidence by physical examination (Fig. 16.23). The approach to primary treatment for these two categories of CAS will be different. It is recommended that angioplasty should be the first

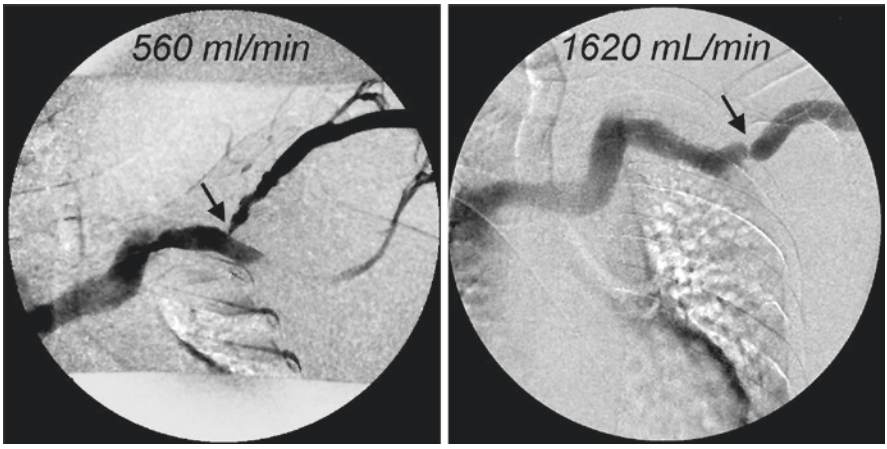


Fig. 16.23 Differentiating between low-normal blood flow CAS in high blood flow CAS by physical examination

intervention for a normal or low blood flow case. Precision banding should be considered as primary treatment for the high blood flow category (Fig. 16.24). Secondary treatment modalities should be reserved for angioplasty failures and early recurrence of the lesion after primary treatment. Whether to use a stent-graft or a surgical procedure for secondary treatment should be based upon the treating team's clinical judgment, selecting an appropriate alternative for each individual patient taking in to account the patient's clinical status, individual practice expertise and the goal of maintaining access function for the longest time possible with the least number of interventions.

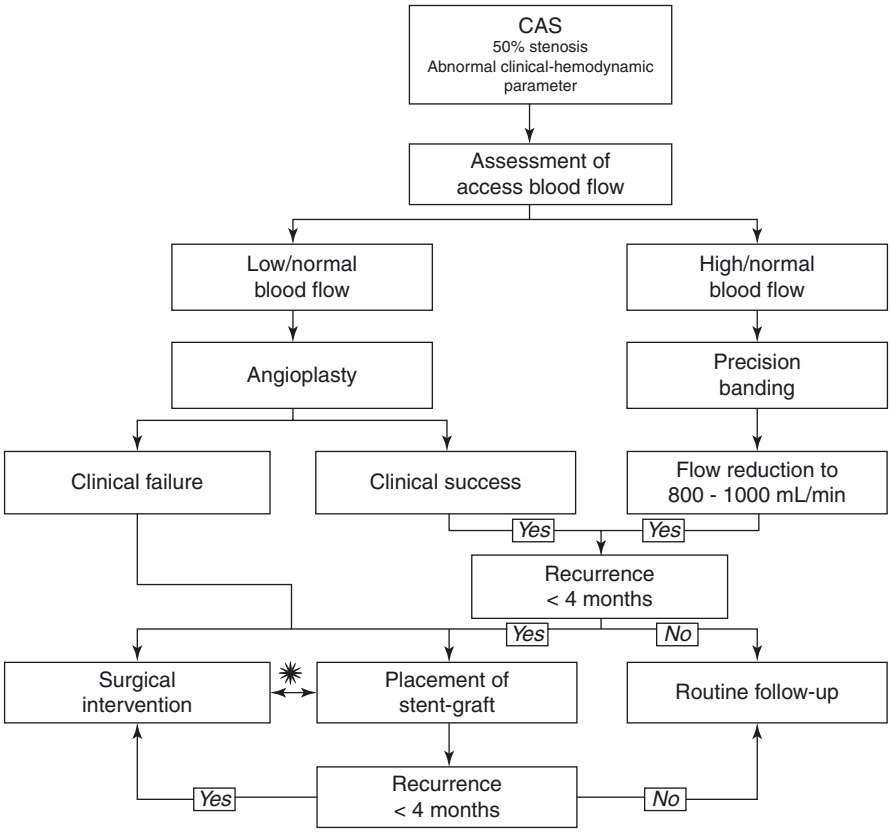


Fig. 16.24 Proposed algorithm for treatment of CAS

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Thoracic Central Vein Obstruction

17

Gerald A. Beathard

Introduction

Stenotic and obstructive lesions of thoracic central veins (TCV) i.e., thoracic central vein obstruction (TCVO), represent a unique category of issues related to dialysis vascular access. Although there is some overlap with problems that affect the peripheral venous system, TCVO not only affects the patient's current arteriovenous (AV) access but can also adversely affect future access creation and associated with significant patient's morbidity [1].

Definition of Thoracic Central Venous System

Although the terms 'central' and 'peripheral veins' are used commonly in dialysis AV access, standard anatomical terms do not offer specific definitions. For our purposes, central veins are those that serve as the final common pathway for venous drainage from a group of peripheral veins and lie either within the bony thorax (Fig. 17.1, Table 17.1) or the bony pelvis. Although the azygos vein is not typically included as a component of the thoracic central venous system, we include it here because it meets the definition and is an important component of the system. The veins that lie outside of the stated bony anatomical boundaries are referred to as peripheral veins.

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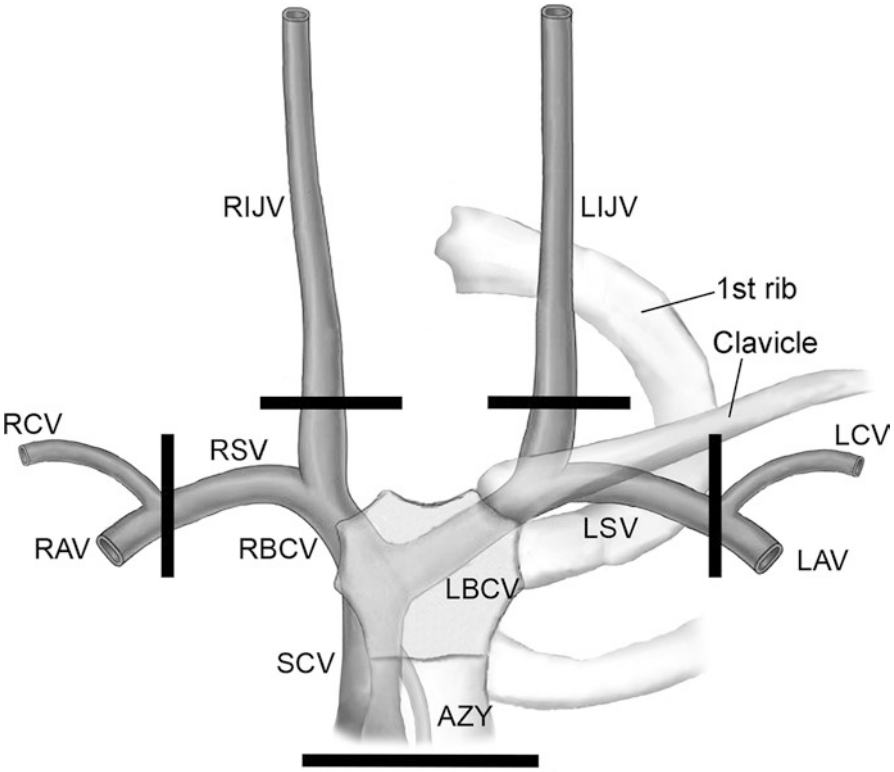

Fig. 17.1 Components of thoracic central venous system within the limits of the space outlined with black marks. (*R* right, *L* left, *IJV* internal jugular vein, *CV* cephalic vein, *AV* axillary vein, *SV* subclavian vein, *BCV* brachiocephalic vein, *SVC* superior vena cava, *AZV* azygos vein

Table 17.1 Thoracic central vein landmarks

Internal jugular vein	Extends from the superior thoracic aperture (C7–T1 intervertebral disc) to T1/T2 disc
Subclavian vein	Extends from the lateral margin of first rib to confluence with IJV (center of sternal head of clavicle)
Brachiocephalic vein	Extends from the confluence of SV and IJV (center of the sternal head of the clavicle) to where BCVs merge (point halfway between center of sternal head of right clavicle and center of AZY shadow)
Superior vena cava	Extends from where BCVs merge to superior cavoatrial junction (point 2 vertebral bodies inferior to carina)
Azygous vein	Extends from the 12th thoracic vertebra in the posterior mediastinum to the level of the fourth thoracic vertebra where it joins the superior vena cava

IJV Internal jugular vein, *SV* Subclavian vein, *BCV* Brachiocephalic vein, *AZY* Azygos vein

Incidence of TCVO

The literature reports a frequency ranging from 3 to 41% [2–5]; however, the true incidence of TCVO is not entirely clear. Only symptomatic cases are generally recognized. If the patient does not have an AV access in the affected extremity, the problem may never come to light. Its variable occurrence is related to the frequency of the use of central venous catheters in the population being studied [2, 6]. In one study, the odds ratio for the number of catheters and the development of TCVO was 2.69 [2].

Etiology of TCVO

In general, three identifiable mechanisms of TCVO can be considered – (1) venous wall thickening, (2) endoluminal obstruction, and (3) extrinsic compression [7] (Table 17.2). While the pathophysiology involved is different, there is significant overlap.

Venous Wall Thickening

Venous wall thickening secondary to neointimal hyperplasia or fibrosis is the most common mechanisms resulting in TCVO. These changes generally represent a response to the introduction of an indwelling foreign object into the central venous system.

Central Venous Catheters

In the dialysis patient population, the most common offending indwelling object is a central venous dialysis catheter. In general, the frequency of TCVO is directly proportional to the frequency of dialysis catheter usage; however, it varies considerably with the vein utilized [2].

Table 17.2 Mechanisms of TCVO in dialysis patients

Venous wall thickening
Central venous dialysis catheter
Peripherally inserted central catheter
Cardiac implantable electronic device
Bare-metal stents and stent-grafts
De novo (no identifiable cause)
Endoluminal obstruction
Thrombosis
External compression
Ectatic aortic arch
Costoclavicular junction compression

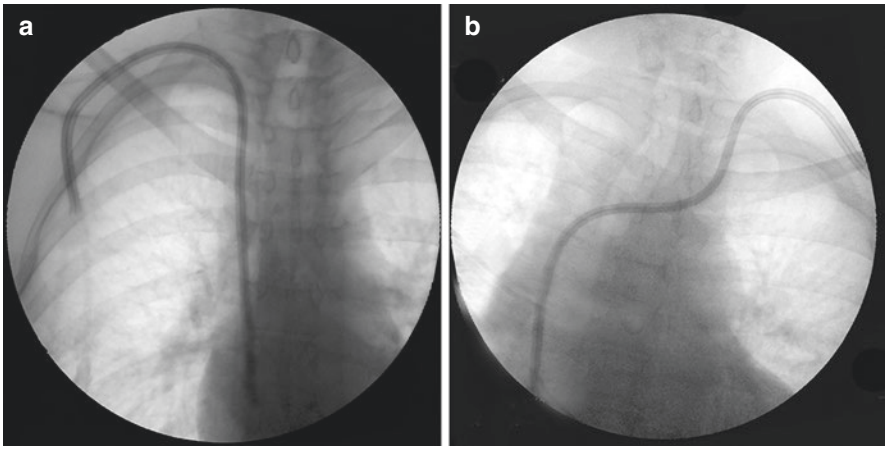

Fig. 17.2 Comparison of right (**a**) and left (**b**) internal jugular veins

The right internal jugular vein (RIJV) is the preferred site for the insertion of a dialysis access catheter. This site is associated with the lowest incidence of TCVO, reported in the range of 10% [8–12]. The lower incidence has been attributed to the straight path as RIJV catheter passes through the right brachiocephalic vein (RBCV) and superior vena cava (SVC) to reach the right atrium (Fig. 17.2a), minimizing mechanical irritation from movement of the mediastinale and cardiovascular structures.

A catheter inserted through the left internal jugular vein (LIJV) is associated with appreciably more complications than are observed on the right side [9–12]. In addition to being longer and having a smaller cross-sectional area than the right [13], a left side catheter must traverse three anatomic angulations (Fig. 17.3): (1) between the LIJV and left brachiocephalic vein (LBCV), (2) between the LBCV and SVC, and (3) an angle created as the BCV crosses over the brachiocephalic artery and aorta [14]. Despite these problems, the LIJV is the usual site selected as a second choice when cannulation of the right side is not an option.

The incidence of subsequent TCVO is approximately 30 to 50 percent when the subclavian vein (SV) is used for central venous dialysis catheter placement [8, 15–17]. The primary cause for this high incidence is thought to be due to mechanical effects of the sharp curve created at the junction of the SVC and BCVs (Fig. 17.4), along with the movement of these structures with the beating of the heart and respiration. An increased incidence of SV stenosis is also observed with increasing number of catheter insertions, indwelling time, number of dialysis sessions, and incidence of catheter-related infections [8, 16, 18].

To minimize the risk of developing central vein stenosis, the use of a short-term temporary catheter is frequently advocated. However, these catheters are relatively stiff, resulting in increased mechanical trauma to the vein. In one study, 14% of patients with a temporary catheter develop stenosis although a mean dwell time for

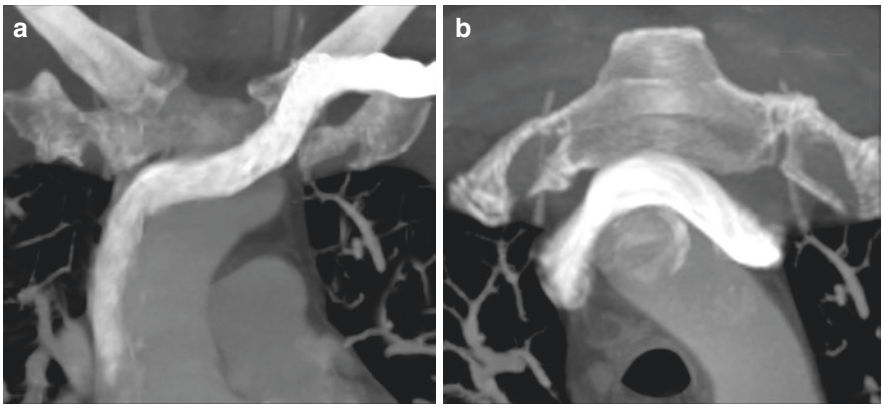

Fig. 17.3 Reconstituted computerized tomography (CT) image of brachiocephalic vein showing angles that must be traversed by a left sided catheter. (**a**) Frontal view, (**b**) Transverse view

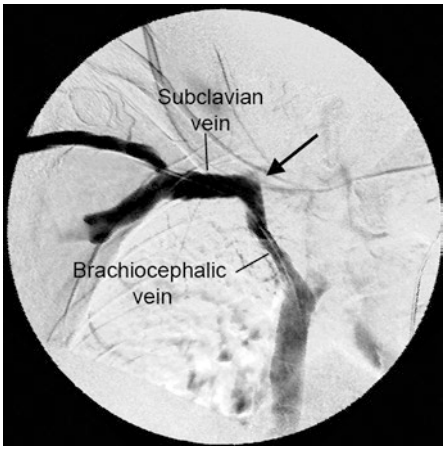

Fig. 17.4 Subclavian—brachiocephalic junction angle (arrow)

the catheters was only 21 days [19]. In two cases, stenosis was greater than 50%. The incidence of stenosis with SVC venous catheters is also greater with temporary than with tunneled catheters [20].

Peripherally Inserted Central Catheter

The widespread use of the peripherally inserted central catheter (PICC) has contributed to the incidence of TCVO [21]. This was demonstrated in a study 150 patients in whom a PICC was inserted [22]. It was found that 7.5% of patients with previously normal central venograms developed subsequent angiographic abnormalities after PICC placement; 4.8% developed central vein stenosis and 2.7% had central venous occlusion.

Cardiac Implantable Electronic Device

Because of the high incidence of cardiovascular disease in the dialysis patient population, there is a frequent need for the placement of a cardiac implantable electronic device (CIED) such as a cardiac pacemaker, implantable cardioverter defibrillator or cardiac resynchronization device. The precise prevalence of CIED usage in ESRD is largely unknown, although a range of 8 to 10% has been quoted [23, 24].

Traditionally, the method of CIED insertion has been transvenous placement of the electrical leads and the implantation of an impulse generator in a subcutaneous pocket. In the general population, this approach has been associated with a relatively high frequency of TCVO (Fig. 17.5) [20, 25, 26]. The pathogenesis of the development of stenosis in association with the transvenous leads of a CIED is thought to be mechanical irritation as with a transvenous catheter. These leads are generally introduced through the SV, causing that structure to be the most frequently affected in the development of a stenotic lesion. However, the BCV and SVC may also be involved.

In addition to the mechanical effects of TCVO, transvenous CIED leads can also induce or aggravate tricuspid insufficiency, which is commonly seen in dialysis patients [27–29]. Because the problems associated with transvenous leads, it has been suggested that transvenous access be avoided if a patient is on dialysis or at risk of requiring dialysis in the future. In these cases, a CIED with either epicardial or subcutaneous leads is recommended [30, 31].

Bare-metal Stents and Stent Grafts

Although used for the treatment of stenosis, bare-metal stents and stent-grafts devices can also promote its development, with the potential of producing lesions

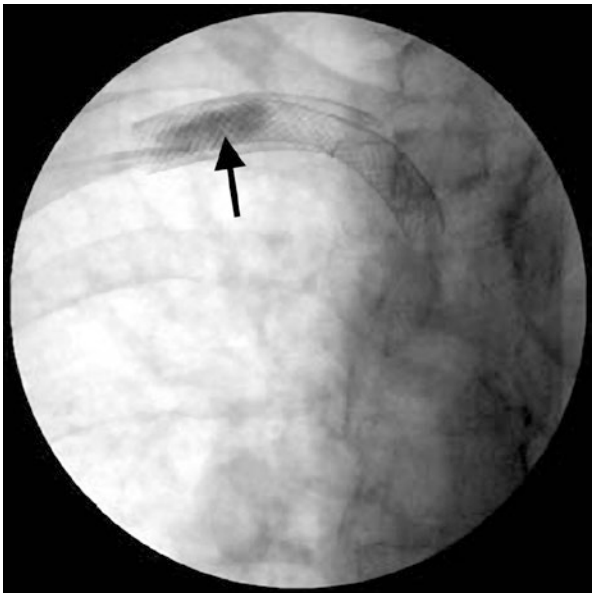

Fig. 17.5 Stenosis within the subclavian stent. Black arrow—lumen containing radiocontrast remainder of unopacified stent indicates occlusion due to stenosis

that are more severe and require more frequent intervention. This is especially true for the bare-metal stent [32]. With this device, the development of neointimal hyperplasia within the stent is a common occurrence and at times can be aggressive requiring repetitive intervention and even leading to complete obstruction (Fig. 17.5). With a stent-graft, intra-stent stenosis is prevented by the graft-material covering the device [33], but stenosis can occur at the ends of the stent (candy-wrapper stenosis). This is more likely to occur with a SG having bare metal struts at the end of the device [34].

De novo Stenosis

Although vessel wall thickening is generally secondary to an identifiable instance in which an indwelling foreign object was introduced into the central venous system, it can develop *de novo* without an identifiable antecedent [2, 6, 35, 36]. It should be noted that when considering cases such as these, a lack of an antecedent central venous intervention simply means that there is no record of such. The age and comorbidity burden of patients with chronic kidney disease (CKD) and the nature of the disease are such that it is unlikely that a patient would arrive at the point of starting dialysis without having had at least a temporary catheter placed, despite there may be no documentation of the event.

Endoluminal Obstruction

Devices such as the central venous catheter and CIED leads can also represent an obstruction, especially if the vessel lumen is already narrowed by neointimal hyperplasia. However, the most frequently observed cause of endoluminal obstruction is thrombus or the placement of an inappropriate stent by size or location.

Central Vein Thrombosis

Central vein thrombosis can occur in association with a central venous catheter (Fig. 17.6a) although the frequency with which this occurs is unclear. In one study [37] involving 143 patients with RIJV dialysis catheters, the incidence of thrombosis was 25.9%. Since most of these thrombi are asymptomatic, the discovery of central vein thrombosis is underestimated due to the lack of symptom in many patients.

Thrombosis of an AV access is generally limited to the peripheral vessels; however, in cases in which there is a central venous lesion, thrombosis may occur within or extend into a central venous structure (Fig. 17.6b). A thrombus that develops within the central venous system can generally be cleared if addressed early. However, over time, thrombus in contact with the vein wall becomes attached and organized, creating a permanent obstruction.

Stents

Inappropriate stent placement can result in endoluminal obstruction in two situations—(1) a stented vessel enlarges to a size greater than the stent within it

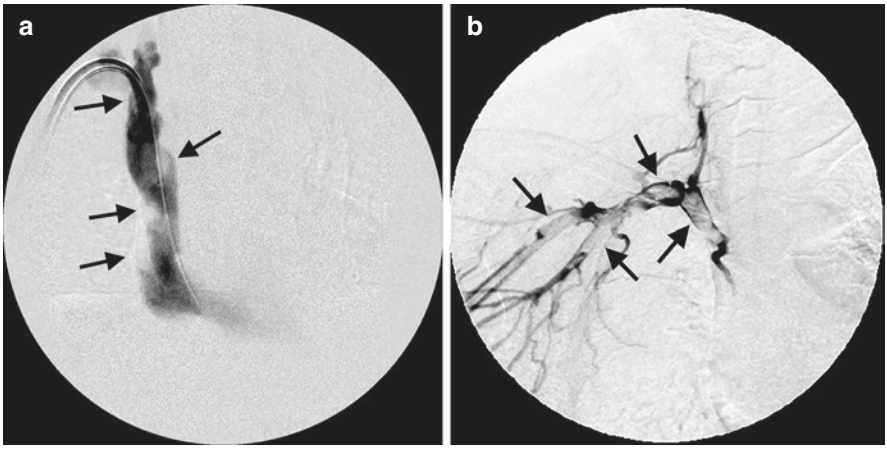

Fig. 17.6 Endoluminal obstruction due to thrombus. (**a**) Endoluminal obstruction due to thrombus associated with catheter (arrows), (**b**) Endoluminal thrombus associated with brachiocephalic vein obstruction (arrows)

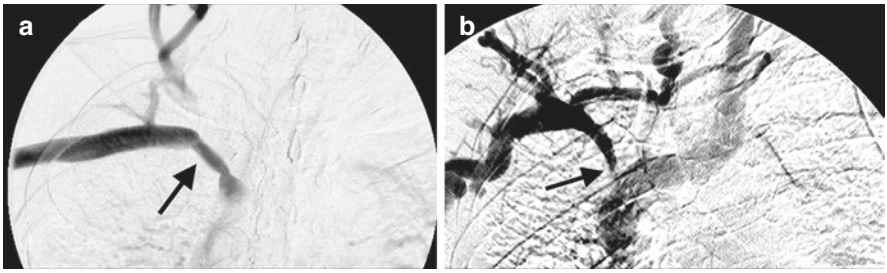

Fig. 17.7 Endoluminal stenosis associated with stent. (**a**) Under sized stent causing obstruction (arrow), (**b**) Poor placement of stent-graft in left brachiocephalic vein causing obstruction of right brachiocephalic vein (arrow)

(Fig. 17.7a) and (2) if the stent is placed so that it occludes the lumen of an associated vessel (Fig. 17.7b). Proper sizing of a stent is of considerable importance.

If a stent is deployed at the confluence of two veins and bridges across one of the lumens ("jails" the vessel), it creates an endoluminal obstruction. There are two sites where the anatomy of the central veins presents a risk for this adverse occurrence—(1) the confluence of the internal jugular and SV, and (2) the confluence of the right and LBCVs. In a study [38] of 52 cases in which a central venous stent-graft was placed for TCVO, a review of angiograms revealed that the internal jugular vein was covered in 40 cases (77%), and the contralateral BCV was covered in 3 cases. Jailing of the internal jugular vein generally does not result in a major problem. With a bare-metal stent, the open construction of the device allows blood to flow freely. However, when the confluence of a BCV is covered with a stent-graft

(Fig. 17.7b) it can render the placement of an AV access in ipsilateral extremity impossible.

A stent diameter that is either too large or too small can create problems. If a stent-graft is oversized, compression of the fabric covering of the stent can form pleats (definition—a fold in cloth made by doubling the material upon itself) which can obstruct the lumen of the device, resulting in TCVO. If a stent is smaller than the adjacent normal vessel's diameter, it can create a mechanical obstruction. In addition, enlargement of the vein over time after the stent has been placed can also result in or worsening of a mechanical obstruction. This endoluminal obstruction is not amenable to dilatation and represents a permanent problem.

External Compression of Central Veins

There are instances in which TCVO is the result of external compression from adjacent anatomical structures rather than intraluminal pathology. The two most commonly encountered one in the dialysis patient are (1) arterial compression and (2) musculoskeletal compression.

Ectatic Aortic Arch

As the patient ages, the aortic arch can become ectatic and along with its associated arteries can compress the enlarged BCV against the sternum (Fig. 17.8). This creates a characteristic compression type of deformity of the LBCV characterized by its typical location and configuration. This is recognized as extrinsic compression because the edges of the deformity are straight and abrupt rather than the typical tapered configuration commonly associated with an intraluminal lesion. Frequently,

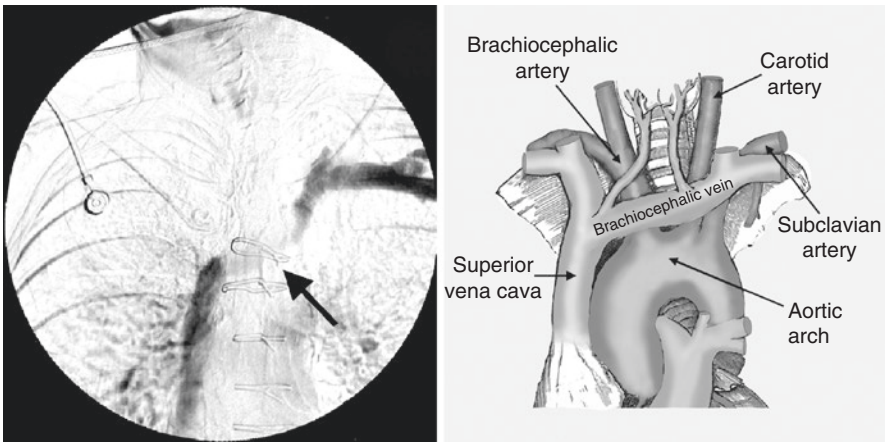

Fig. 17.8 Extrinsic compression of left brachiocephalic vein (arrow). Compare defects in vein with diagram of anatomy on right

collaterals may be seen peripheral to the defect confirming that it is a true obstruction. It is important that this situation be recognized as dilatation with an angioplasty balloon will have no benefit and has the potential for causing additional problems.

In one study of 48 cases [39], some degree of extrinsic compression was observed in 21 (44%). Twelve (25%) were characterized as mild, six (13%) as moderate and three as (6%) severe compression. Collateral veins were seen in 11 cases with compression (52%). All three patients with severe extrinsic compression were symptomatic and were treated with stent placement.

Costoclavicular Junction Compression

As the SV enlarges due to the increased blood flow associated with a vascular access, it can be mechanically compressed as it passes through the costoclavicular space which lies beneath the clavicle. This is a triangular space (Fig. 17.9) whose upper boundary is the clavicle and the subclavius muscle which runs along the inferior aspect of the clavicle and attaches to the first rib. At the point of attachment, this muscle forms a dense connective tissue band, the costoclavicular ligament. Attached below to the superior and medial aspect of the cartilage of the first rib, this ligament forms the medial boundary of the costoclavicular space. The anterior scalene muscle which is inserted into the first and second ribs forms the lateral border.

Expansion of the costoclavicular space is restricted because the clavicle and first rib are both securely anchored to the sternum and to each other anteriorly. As the SV enlarges with the development of the AV access it becomes increasingly susceptible to constriction. In addition, it has been postulated that turbulent blood flow occurs at this unique area promoting the development of neointimal hyperplasia [40]. Treatment of TCVO occurring at this site is problematic. Angioplasty (PTA) generally results in a poor result, and stents placed in this zone frequently become compressed (Fig. 17.9) and can actually be detrimental [40, 41]. Surgical treatment provides the best results by relieving the bony compression [40, 42, 43] (see discussion below under management).

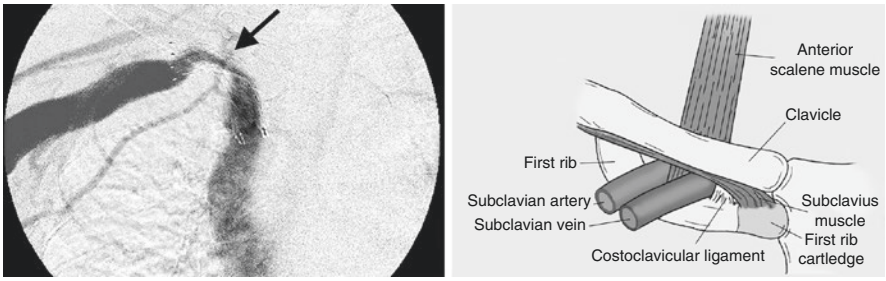

Fig. 17.9 Costoclavicular compression. Left—Angiogram of subclavian vein with stenosis at the costoclavicular junction (arrow), Right—Drawing of costoclavicular area showing subclavian vein relationships

Collateral Circulation Associated with TCVO

With the development of any venous obstruction, collateral vessels develop providing an alternative circulation around the blockage either through the recruitment of pre-existing vascular structures or as result of the development of new vessels, neovascularization. The collateral circulation associated with TCVO is important because it can serve to preserve patency of the AV access and in some instances can even be used to create a new unobstructed pathway for blood to flow from an obstructed AV access. In addition, collateral vessels serve as an indication of a significant obstruction.

Collateral Pathways

There is considerable variation in the spectrum of venous structures that participate in the development of collateral circulation resulting from TCVO. Venous collateral pathways have been defined in order to categorize the changes [44]. Four are of particular importance to hemodialysis access—(1) the jugular pathway, (2) the lateral thoracic pathway, (3) the anterior thoracic pathway and (4) the azygous pathway. Other pathways that are occasionally involved have also been described. These include the systemic-to-pulmonary venous, cavo-portal, intrahepatic, and cardiac vein pathways [45, 46]. The pathway(s) involved in the development of collaterals depends upon the location of the obstruction within the TCV system. In addition, each of these pathways communicate with other pathways resulting in overlap in the clinical manifestations that are observed.

Jugular Collateral Pathway

The network of veins providing drainage of the head and neck are associated with the jugular venous system. This system is composed of three paired veins—the internal jugular which lies deep and is the dominant component of the group, and the external and anterior jugular veins (anterior is sometimes single) which are superficial. There are anastomotic connections between these vessels and with both the subclavian and BCVs. The anterior jugular arch connects the right and left anterior jugular veins and provides a communication between the two sides of the jugular venous system. In addition, there are connections between the jugular veins and the veins associated with the thyroid gland and with the lateral vertebral veins of the paravertebral venous plexus.

Lateral Thoracic Collateral Pathway

The paired lateral thoracic veins provide collateral circulation over the anterior-lateral aspect of the thorax and between the SVC and inferior vena cava (IVC). This system communicates with the anterior thoracic collateral pathway through connections with the anterior thoracic veins via the anterior intercostal veins. The system also communicates with the azygos system via intercostal veins. The lateral thoracic veins communicate with the thoracoepigastric and superficial epigastric veins and

through them, with the epigastric veins providing a potential blood flow channel to the IVC.

Anterior Thoracic Collateral Pathway

The anterior thoracic veins which form the basis of this pathway are also paired and drain into the BCVs. They contribute to the thoracic venous network by communicating with one another across the midline and with the lateral thoracic veins via the intercostal veins. In addition, they communicate with both the superficial epigastric and inferior epigastric veins as well as both the SVC and IVC.

Azygous Pathway

The basis for this pathway is the azygos system which is composed of the azygos, hemiazygos, accessory hemiazygos, and right and left superior intercostal veins. The azygos vein lying on the right side of the mediastinum is formed by the union of the ascending lumbar veins and right subcostal veins and drains into the SVC immediately superior to the pericardial reflection. The lower intercostal veins drain into the azygos on the right and into the hemiazygos and accessory hemiazygos on the left. The upper intercostal veins drain into the right and left superior intercostal veins. The hemiazygos and accessory hemiazygos drain into the azygos vein by communications across the midline. This system represents an important communication between the SVC and IVC.

Collaterals Associated with Site of Occlusion

The overall pattern of collateral veins that develops in response to TCVO is dependent upon the vein that is obstructed and the site at which the obstruction occurs.

Internal Jugular Vein (IJ V)

Collateral flow resulting from obstruction of the IJV can be very robust, involving the jugular collateral pathway which allows for communication with the contralateral jugular veins through the anterior jugular arch. The paired internal jugular veins have rich anastomotic channels over the thyroid gland. The vertebral veins and the paravertebral venous plexus may also be involved (Fig. 17.10).

Subclavian Vein (SV)

Collateral flow associated with SV occlusion frequently involves the jugular venous pathway (Fig. 17.11a) as well as the lateral thoracic collateral pathway (Fig. 17.11b). In addition to named vessels, a profusion of unnamed tortuous venous collaterals often develops.

Brachiocephalic Vein (BCV)

With obstruction of the BCV, collateral blood flow may develop through deep and superficial veins of the back chest and neck into the contralateral IJV, SV and BCV. In addition, collateral flow through the communications between the anterior

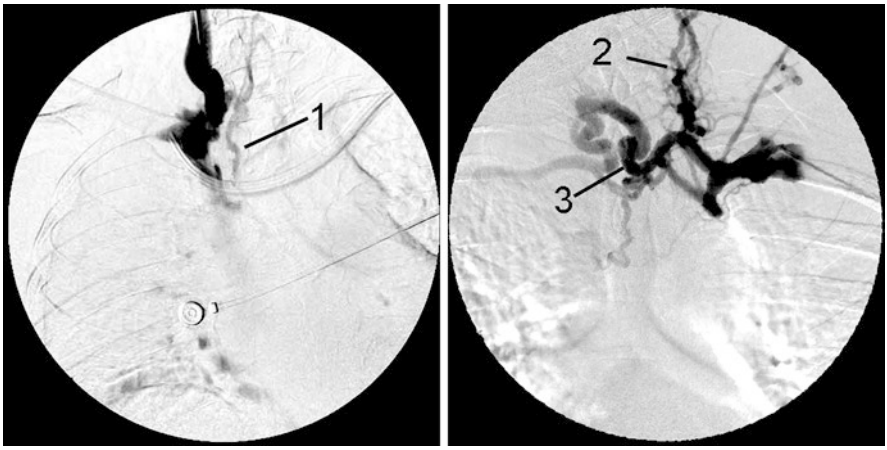

Fig. 17.10 Occlusion of internal jugular vein (1—Anterior thyroid vein, 2—Paravertebral venous plexus, 3—Anterior jugular arch)

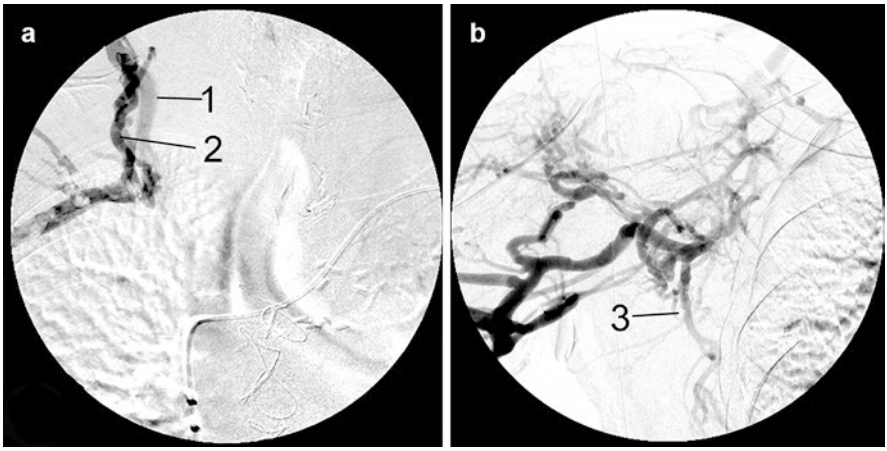

Fig. 17.11 Occlusion of subclavian vein. (**a**) Both the internal (1) and external (2) jugular veins are visible (note thrombi in subclavian vein due to thrombosed access), (**b**) Lateral thoracic vein (3)

and posterior thoracic pathways to the azygos pathway frequently occurs. (Fig. 17.12).

Superior Vena Cava Occlusion Above Azygos Vein

When the SVC is occluded at a level above the azygos vein, TCV drainage takes place through both the lateral thoracic and anterior thoracic pathways into the azygos system. This lesion is primarily caused by previous central venous catheters. The collateral circulation from this level of obstruction is generally recognized

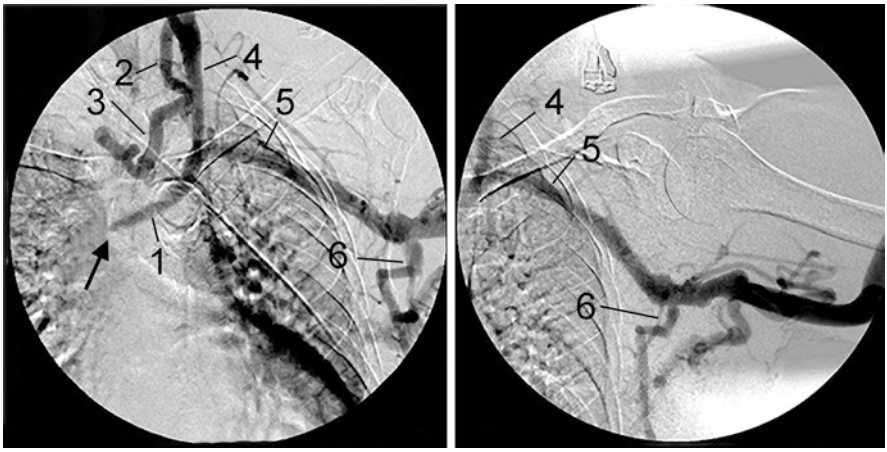

Fig. 17.12 Occlusion of brachiocephalic vein, the two images are from the same patient (1—Left brachiocephalic vein, 2—Left internal jugular vein, 3—Anterior jugular arch, 4—Left internal jugular vein, 5—Subclavian vein, 6—Left lateral thoracic vein)

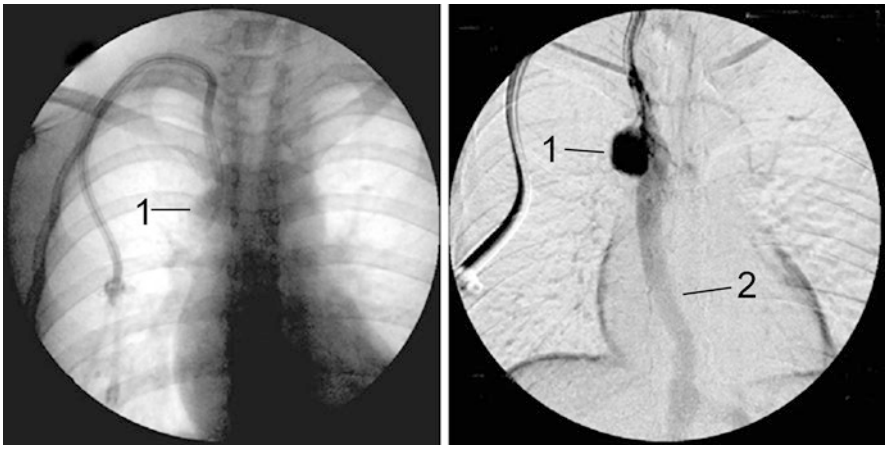

Fig. 17.13 Occlusion of superior vena cava above insertion of azygous, note the nodular enlargement in the right lateral upper mediastinum in the radiograph on the left, with radiocontrast injection its identity becomes apparent (1—Arch of azygos, 2—Azygos vein)

radiographically during a catheter procedure as a markedly increased prominence of the azygos vein and its arch at the point of attachment to the SVC (Fig. 17.13).

Superior Vena Cava Occlusion Below Azygos Vein

When the SVC is occluded at a level below the azygos vein, the thoracic central venous collateral drainage is achieved by thoracic blood flow draining into the azygos system and then flowing retrograde through the azygos pathway into abdominal central veins that communicate with the IVC.

Collateral Decompression of TCVO

In the patient with a dialysis vascular access, TCVO involving the ipsilateral access drainage results in increased pressure within the peripheral veins. They frequently manifest as swelling of the access extremity (see below). Occasionally, the development of collaterals increases the effective cross-sectional area of venous drainage. This result in sufficient decompression of the peripheral veins giving either no clinical signs and symptoms or disappearance of swelling. In these cases, the access is often able to function adequately and provide effective dialysis. If the AV access develops thrombosis, thrombectomy can generally be successfully accomplished. However, in some cases problems are encountered due to occlusion of collateral vessels by thrombus or small emboli resulting from the thrombectomy procedure.

Clinical Consequences of TCVO

The spectrum of clinical manifestations associated with TCVO is relatively broad due to the fact that obstruction may be partial or complete and different components of the TCV system may be involved alone or in combination. In the absence of an ipsilateral dialysis AV access, there is generally no obvious evidence of the problem. When an AV access is present, the case may be asymptomatic and recognized only when diagnostic studies are performed [47].

Although AV access arm edema (Fig. 17.14) is the most typical manifestation of this condition, the clinical consequences of TCVO represent a spectrum ranging from loss of a dialysis catheter insertion site, progressive edema of the access arm, to a total loss of AV access creation sites in the affected extremity. As stenosis begins and progresses, the associated signs and symptoms begin in a mild form and progress to the more severe one. In our discussions, we will dwell primarily upon the consequences of the advanced lesion which represents the extreme of the clinical spectrum.

Mechanism of Arm Edema

The pathogenesis of edema in the arm ipsilateral to the AV access is related to the fact that the TCV complex is the final common single pathway for venous drainage from the extremity. The presence of the AV access results in a high level of blood flow into its venous drainage. With a progressive increase in diameter of the vessels, pressure remains low in the system unless there is an obstruction. As an obstructing lesion begins to develop, resistance begins to increase resulting in a progressive increase in pressure within the affected vein(s). When these changes occur in a peripheral vein, an alternative pathway can develop through the development of collateral veins and relieve the pressure. However, since the central veins are the final common single drainage pathway for tributary veins of the extremity, there is no alternative pathway. The venous pressure within the extremity increases resulting in extravasation of fluid recognized as edema. Collateral veins also develop with

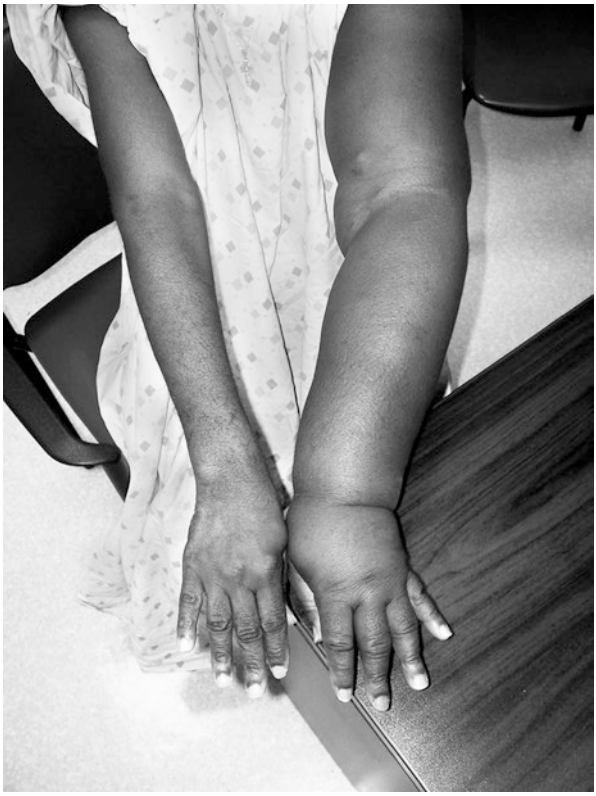

Fig. 17.14 Swollen AV access arm due to TCVO

TCVO and in some cases, even with total venous obstruction, their development may be so robust that clinical symptoms do not develop.

Edema of the AV access arm can be a major problem symptomatically for the patient and functionally for their AV access. As the access arm swells, cannulation becomes more challenging. Increased pressure from the downstream obstruction causes the draining veins to enlarge resulting in both aneurysmal changes and increasing tortuosity. The increased resistance from the obstruction leads to elevated dialysis venous pressures, prolonged bleeding from needle sites after dialysis and results in a decrease in access blood flow adversely affecting the efficiency of the dialysis treatment. Decreased blood flow also places the access at risk for thrombosis. Untreated, the access can eventually become unusable. If the causative TCVO lesion cannot be effectively treated (or bypassed), the access will require closure.

The increased severity of the swollen arm is often associated with tenderness and pain. Skin discoloration, lymphatic weeping, stasis ulcers, cellulitis and non-healing wounds can occur. Depending upon the TCV vessel involved, pleural effusion and edema of the face, shoulder, and breast can develop (Fig. 17.22) [20, 48–51]. Superior vena cava syndrome with unusual manifestations such as esophageal varices [52], unilateral conduction hearing loss [53], secondary glaucoma and papilledema [54], and myoclonic epilepsy [55] may be seen.

Level of TCVO Involvement

Depending upon the component of the TCV that is affected, the clinical picture observed, and its consequences will vary. This spectrum has been classified into four levels of involvement starting peripherally and progressing centrally and characterized by progressively increasing complexity of signs and symptoms [7] (Table 17.3).

TCVO Level 1

Typically, Level 1 has four major components, the RIJV and LIJV and the right and left SV. A variety of one and two vessel involvements can be seen (Fig. 17.15), and the clinical consequences of each is somewhat different. If the ipsilateral IJV and SV are involved, the resulting clinical picture of TCVO Level 2 develops (see below).

Although no specific clinical symptoms are associated, obstruction of either IJV affects decisions related to central venous catheter placement and planning for AV access creation. This is especially important in catheter-dependent cases in which

Table 17.3 Clinical levels of TCVO

Level 1	(a) Unilateral IJV or SV obstruction with patency of the contralateral IJV, SV, and BCV (b) Bilateral obstruction of IJVs, SVs, or combined IJV and SVs, with both BCVs patent.
Level 2	(a) Unilateral BCV obstruction (b) Ipsilateral obstruction of the IJV
Level 3	Bilateral BCV obstruction, with flow to the right atrium passing through the SVC
Level 4	SVC obstruction that prevents or impedes direct thoracic venous flow to the right atrium with any combination of BCV, IJV, or SV obstruction

IJV Internal jugular vein, *SV* Subclavian vein, *BCV* Brachiocephalic vein, *SVC* Superior vena cava

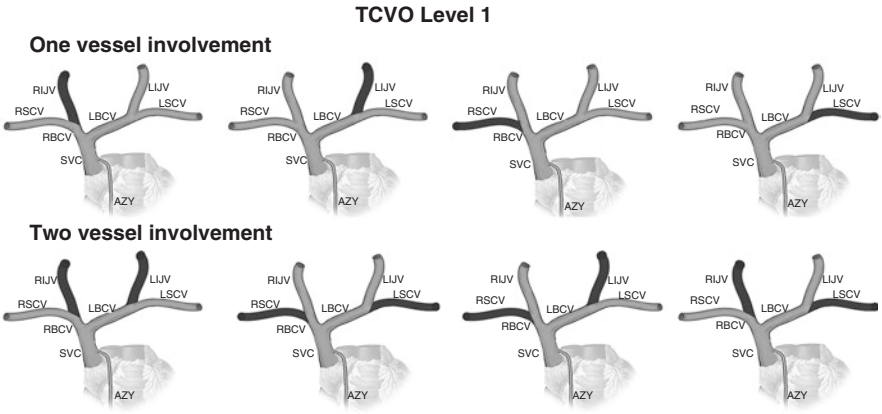

Fig. 17.15 TCVO Level 1 variations that can occur involving either one or two vessels at this level (*R* Right, *L* Left, *IJV* Internal jugular vein, *SV* Subclavian vein, *BCV* Brachiocephalic vein, *SVC* Superior vena cava, *AZY* Azygos vein)

the only anatomical alternative is to create an AV access ipsilateral to the catheter. In this instance, the AV access can be significantly jeopardized because of risk of further involvement of TCVs due to the presence of the catheter. If both IJVs are obstructed, an alternative venous site (collateral vein or femoral vein) is necessary for central venous dialysis catheter placement.

Obstruction of the SV can create major problems for an ipsilateral AV access. The increased resistance due to the lesion and its associated increase in pressure in the AV access can lead to aneurysmal changes and increasing tortuosity in tributary venous structures. As a SV lesion progresses, there is an increased risk of thrombosis and for the development of ipsilateral arm edema due to the presence of the access. Obstruction of the SV has a major effect on AV access planning, a problem made worse if the obstruction is bilateral.

TCVO Level 2

Typical involvement at Level 2 presents a more complex clinical pattern due to the involvement of TCVs at a more central location, the RBCV and LBCV. However, ipsilateral occlusion of both the IJV and SV can also present the same clinical picture. Because of the vessels involved, several anatomical variations are possible at this Level (Fig. 17.16).

Since vessels of Level 2 are the channels receiving the tributary vessels of Level 1, the clinical consequences of occlusion at that level are also observed. In addition to edema of the AV access extremity, Level 2 occlusion can also result in edema of the ipsilateral face, shoulder and breast [51]. Level 2 involvement with complete obstruction results in the loss of an ipsilateral AV access.

TCVO Level 3

The vessel typical of Level 3 involvement is the SVC; however, several anatomical variations are also possible (Fig. 17.17). As the final common pathway for all blood conveyed by the tributary TCV vessels, obstruction of the SVC results in all of the consequences attributed to problems occurring at both Levels 1 and 2, with the addition of the adverse effect of obstructing blood flow from the entirety of the thorax. The result of this problem, is some variant of superior vena caval syndrome.

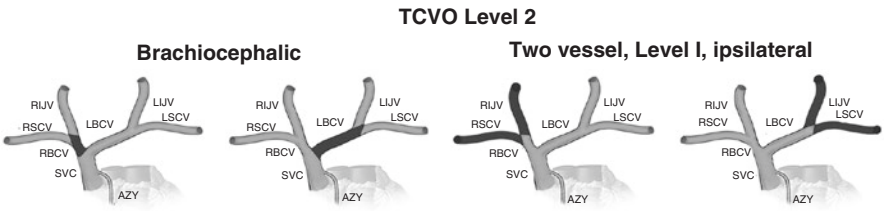

Fig. 17.16 TCVO Level 2 variations that can occur including Level 1 ipsilateral two vessel involvement (*R* Right, *L* Left, *IJV* Internal jugular vein, *SV* Subclavian vein, *BCV* Brachiocephalic vein, *SVC* Superior vena cava, *AZY* Azygos vein)

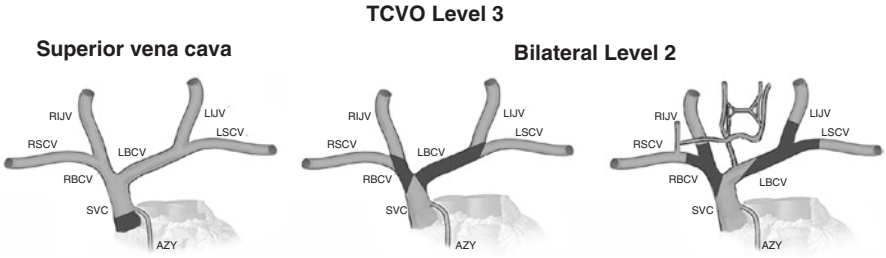

Fig. 17.17 TCVO Level 3 variations that can occur including Level 2 bilateral (*R* Right, *L* Left, *IJV* Internal jugular vein, *SV* Subclavian vein, *BCV* Brachiocephalic vein, *SVC* Superior vena cava, *AZY* Azygos vein)

Table 17.4 Classification of superior vena cava (SVC) syndrome

Type I	Partial obstruction of SVC (≤ 90%) with patency of azygous and antegrade flow
Type II	Near complete to complete obstruction of SVC (> 90%) with patency of azygous and antegrade flow
Type III	Near complete to complete obstruction of SVC (> 90%) with patency of azygous and retrograde flow
Type IV	Complete obstruction of SVC and one or more of the major tributaries including the azygos

Superior Vena Caval Syndrome

The incidence of superior vena caval syndrome is not clearly defined but has been reported to be in the range of 1% to 3% in patients with central venous catheters [56]. Based upon the superior vena caval site and extent of the involvement, a classification system for superior vena caval syndrome has been described [57] (Table 17.4). According to this classification system, the three Level 3 variations in Fig. 17.17 would be Types II, III, and II, in that order. In the third example, blood flow to the SVC above the azygos vein is preserved due to connections between the brachiocephalic and the jugular collateral pathway.

Superior vena caval syndrome is a very serious condition characterized by edema of both upper extremities, face and neck along with multiple dilated collateral veins over the neck and chest. Patients may complain of a feeling of "fullness" of the head, lightheadedness, chest pain, hoarseness, coughing, dyspnea and orthopnea. Pleural effusion, frequently bilateral, is often present and can be large. Unlike its association with neoplasia, superior vena caval syndrome in the dialysis patient generally develops relatively slowly allowing for the proliferation of collateral vessels making an acute life-threatening presentation less likely, but not impossible. With total obstruction of the SVC and upper extremity AV access is not possible.

TCVO Level 4

The SVC is also involved TCVO Level 4; however, in this instance thoracic venous blood flow to the right atrium by way of the SVC is not possible (Fig. 17.18). All thoracic venous blood must pass through collateral pathways that communicate

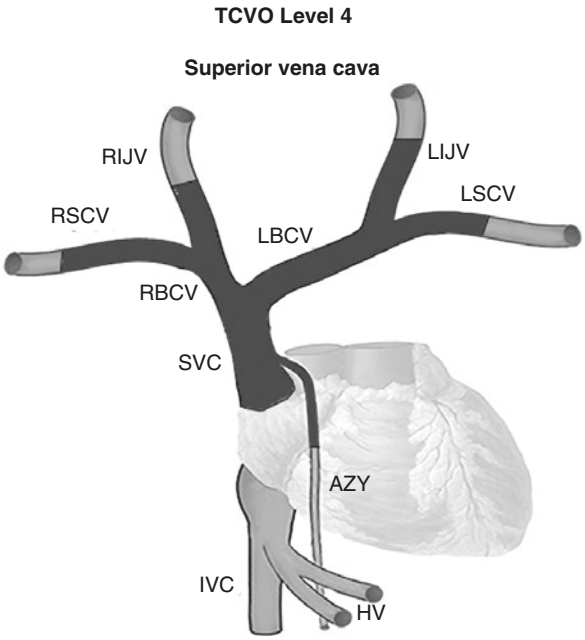

Fig. 17.18 TCVO Level 4 (*R* Right, *L* Left, *IJV* Internal jugular vein, *SV* Subclavian vein, *BCV* Brachiocephalic vein, *SVC* Superior vena cava, *AZY* Azygos vein)

with the IVC and then to the right atrium. Fortunately, although it can occur, this is not a common occurrence in the dialysis patient.

Management of TCVO

The approach to the management of TCVO varies considerably depending upon the spectrum of clinical signs and symptoms associated with the various Levels of obstruction.

Asymptomatic TCVO

Not all cases require treatment, some cases are asymptomatic. In these cases, treatment is not indicated unless dictated by the need to create a functional AV access for dialysis. NKF–K/DOQI practice guidelines state that in order to qualify for angioplasty treatment a venous stenosis lesion should cause a greater than 50% decrease in the luminal diameter of the vessel and be associated with clinical/physiological abnormalities [58]. Whether are not TCVO is symptomatic is an important distinction. Multiple studies have examined the consequences of treating asymptomatic TCVO and have shown that treatment is frequently followed by increasing stenosis and symptom escalation [36, 47, 59].

There is evidence indicating that an asymptomatic central venous lesion present before AVF creation can become symptomatic in association with the increased blood flow resulting from the placement of an AV access [51, 60, 61].

Symptomatic TCVO

When presented with a patient with symptomatic TCVO, there are three categories of management alternatives—endovascular, surgical and closure of the access (Table 17.5).

Endovascular Management

Although the primary patency rate has not been high, endovascular treatment has become the standard of care in the dialysis patient [62].

Percutaneous Transluminal Angioplasty (PTA)

The first-line treatment for TCVO is PTA [63]. Unfortunately, the studies reporting patency data specific for this entity have used criteria that are not uniform in reporting the description of lesions, their severity, or the outcome of treatment. This makes a direct comparison between reports difficult. A compilation of 12 reports representing a total of 520 cases [4, 64–74] from 1988 to 2014 shows a technical success rate from 77% and 96%. At 6-months primary patency, assisted primary patency and cumulative patency were 23% to 84%, 45% to 100%, and 88% to 100%, respectively. The primary patency, assisted primary patency and cumulative patency at 12-months were 12% to 77%, 28% to 82% and 63% to 100%, respectively.

There is a subgroup of TCVO lesions that are less responsive to PTA due to elasticity (Fig. 17.19). The incidence of elastic lesions has been reported in the range of 20% to 60% [75, 76]. In addition to elastic recoil, a high rate of early recurrence is

Table 17.5 TCVO management options

Endovascular
Angioplasty
Stent placement
Bare-metal stent
Stent-graft
Advanced techniques
Sharp needle recanalization
Radiofrequency recanalization
Surgical
Banding
Bypass graft
Decompression
Primary repair of obstruction
Closure of AV access

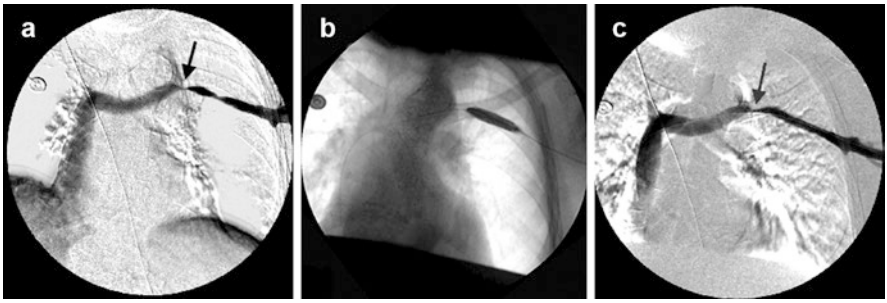

Fig. 17.19 Elastic lesion in subclavian vein. (**a**) Initial appearance of lesion, (**b**) Full effacement of angioplasty balloon, (**c**) Appearance of lesion following angioplasty demonstrating marked elastic recoil

commonly seen following an initial acceptable result [76]. Patients with TCVO associated with transvenous CIED leads also demonstrate a poor response to PTA in comparison to routine TCVO cases [50, 64].

Stent Placement

Practice guidelines published by NKF-K/DOQI [77] state that stenting of TCVO lesions should be considered in the following situations—(1) acute elastic recoil (> 50%) following PTA, or (2) stenosis that recurs within a three-month period. Although, it has generally been concluded that stent usage is advantageous, they do not provide a permanent solution. In some studies, results have shown no improvement in patency for stent placement in TCVO [69, 72, 78] and multiple re-interventions are required in view of recurrent stenosis in most patients [69, 72, 79]. In addition, stent placement may cause potential technical problems. Stents may migrate at the time of deployment or post-deployment. This can lead to major complications, especially if the device enters the heart. Stent development has evolved through three iterations—stainless steel bare-metal stents, nitinol bare-metal stents stent-grafts (covered stents). Although the evidence to support the conclusion is not substantial, most interventionist feel that the effectiveness of the device has improved with each iteration.

Bare-Metal Stents (BMS)

There are two types of BMSs, stainless-steel and nitinol. Most studies have involved the stainless-steel variety. Some reports in which stainless steel and nitinol stents have been compared in advantage for the nitinol device [80] while others have failed to detect a difference [70, 78, 81].

Most of reports utilizing BMS have been relatively small a variety of different stent types have been used and in some, a mixture of stainless-steel and nitinol stents was used and reported as a group with no distinction as to type [3, 32, 65–67, 69, 70, 72, 78–80, 82–94]. Many studies are deficient in that a full range of patency data was not reported and most have been observational with no control group for

comparison. However, based upon the data that are available, the primary, primary assisted and cumulative patency at 6-months were 11% to 84%, 62% to 91%, and 54% to 100%, respectively. The same values at 12-months were 11% to 75%, 45% to 78%, and 33% to 100%, respectively.

A major problem associated with the use of the BMS is the development of neointimal hyperplasia, which in some cases is aggressive (Fig. 17.20). This problem is characterized by neointimal tissue ingrowth through the open struts of the BMS. In order to prevent this problem, stents covered with either ePTFE or Dacron were developed with the expectation that this material would form a barrier to prevent this ingrowth. These devices have come to be referred to as stent-grafts (SG).

Stent-graft (SG)

Only a small number of studies specifically dealing with TCVO treated with a SG have been reported [32, 34, 38, 51, 95, 96]. Unfortunately, these suffer from the same deficiencies as the studies on BMSs. Primary, primary assisted and cumulative patency at 6-months were 40% to 100%, 100%, and 55% to 100%, respectively. The same parameters at 12-months were 29% to 100%, 80% to 100%, and 39% to 100%, respectively.

Even though the covering of the SG serves as a barrier to tissue ingrowth through the struts of the stent, it is not totally immune to the development of neointimal hyperplasia. Stenosis can develop at the ends of the stent creating a "candy-wrapper" lesion. These lesions are more likely to develop with a stent which has a bare-metal element at either end.

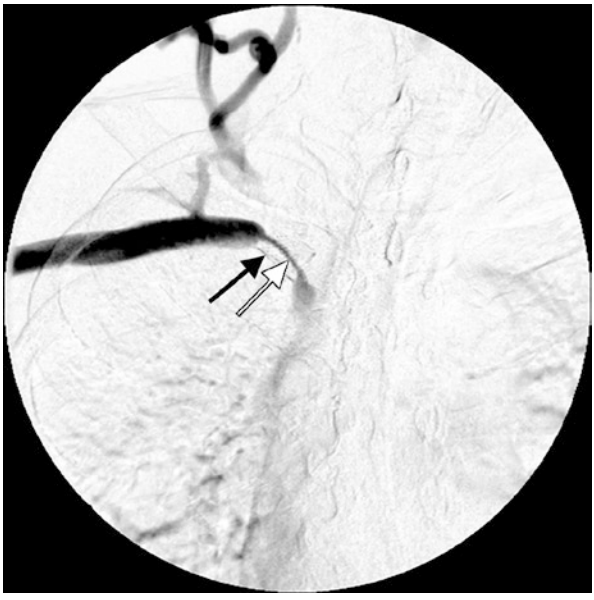

Fig. 17.20 Bare metal stent with in-stent stenosis due to neointimal hyperplasia (Black arrow—edge of stent, White arrow—radiocontrast in lumen of the vessel)

PTA Versus Stenting

While there are no randomized control trials to date comparing PTA and stent use in the setting of TCVO, retrospective studies showed varying differences in patency between the two groups depending upon the type of stent used [32, 70, 72].

Caution is advised in making comparisons based upon reported studies of PTA and stent usage because of the poor quality of the studies that have been reported as discussed above. In addition, stenting of TCVO is a salvage procedure, assuming that it is done for the recommended indications. Thus, comparison with PTA is not appropriate. In comparisons of PTA alone versus PTA plus stent placement for the "problematic" cases, it appears that the latter is superior (enables salvage of the case). This should be viewed as an endorsement of stent placement for "problematic" cases in that they provide results that are at least equivalent to "non-problematic" cases.

Some investigators have used PTA and primary stent placement in an attempt to limit recurrence rates associated with TCVO [65, 69, 78, 79, 85, 86, 89, 92, 93, 97]. However, secondary interventions are usually necessary, and as a result, assisted primary patency rates have generally been comparable to that of PTA alone.

Advanced Endovascular Techniques

The conventional techniques (PTA and stent placement) for establishing access across an occluded TCV segment require the passage of a guidewire as the first step. However, these lesions are often progressive leading to eventual total occlusion, TCVO. In these cases, a guidewire cannot be passed and standard techniques for treatment are not possible. If no TCV available for the creation of a vascular access, there are alternatives such as a femoral graft or catheter and as a last resort, translumbar and transhepatic catheters. However, these choices are often less than optimal. They are associated with an increased incidence of complications, may be poorly tolerated and are frequently associated with a poor quality of life. In these cases, a better choice may be to salvage a TCV site either with surgical reconstruction [98–102] or one of the approaches described here as an advanced endovascular technique.

Sharp Recanalization

Sharp recanalization is a specialized endovascular procedures that has been used effectively to salvage total vascular obstruction [103–115]. This technique involves approaching the lesion from opposite sides either with two catheters or a catheter and a needle device. In some cases, especially those in which the distance is short, the lesion can often be perforated with the back-end of a hydrophilic guidewire followed by the introduction of an angioplasty balloon to open the lesion, which can then be stented if necessary [109]. In other instances, a needle device can be used to cross the obstruction and penetrate a target such as an angioplasty balloon [113] (Fig. 17.21) or endovascular snare [114]. This is followed by the use of standard guidewire and catheter techniques to perform PTA and frequently, the placement of a stent to re-establish blood flow.

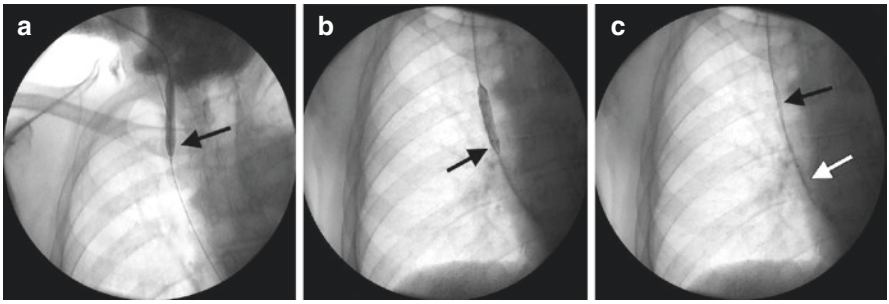

Fig. 17.21 Sharp recanalization used to access right internal jugular vein utilizing technique referred to as balloon-guidewire-entrapment. (**a**) Cannulation and passage of guidewire into angioplasty balloon introduced through extra thoracic portion of jugular vein, (b) Guidewire being advanced into superior vena cava by advancing angioplasty balloon, (**c**) Guidewire extracted from balloon by arresting its advancement while continuing to advance angioplasty balloon (Black arrow—guidewire, White arrow—angioplasty balloon)

Several different needle devices have been used, the choice of needle generally is decided by the complexity of the procedure. In the least complex cases, a 21-gauge micro - puncture or a Chiba needle (similar to micro puncture but has an obturator and comes in a variety of sizes) is generally used to penetrate the target (Fig. 17.21). In more complex cases, other types of needles have been used, such as a TIPS needle (Rosch-Uchida) [104, 106–108, 110] or a transseptal needle [114].

Procedures involving most TCVs when performed with care are rarely associated with significant complications. However, lesions involving the RBCV and/or SVC pose a significant risk as the pericardial reflection extends a significant distance up and around the SVC. Perforation of the vein into the pericardium can lead to pericardial tamponade [113].

Radiofrequency Recanalization

Ablation of myocardial arrhythmogenic foci using radiofrequency (RF) energy is well-recognized. By using higher voltage, RF can be used to perforate an obstructing lesion by vaporizing target cells in contact with the transducer [116]. This technology has been shown to be effective at recanalizing TCVs [117–122]. While RF recanalization is an option for the treatment of cases refractory to the passage of a guidewire, it requires a skilled, experienced operator and is associated with significant risk of serious complications [122].

Surgical Management

Although not often required, surgical management of TCVO in selected cases can play an important role. Surgical procedures for TCVO falls into 4 general categories: banding, by-pass of the stenotic lesion, decompression, and primary repair.

Banding

There is a relationship between clinical signs and symptoms of TCVO and the rate of access blood flow [2, 35]. It has been shown that banding the access to decrease blood flow (Fig. 17.22) can result in improvement or elimination of the clinical manifestations [123]. This can be especially important in cases of technical failures or in those in whom symptoms reappear after a short time period. In a report involving 22 cases with failed attempts to correct TCVO by PTA and stent placement, banding was performed by tying sutures over an inflated angioplasty balloon (generally four-mm in diameter) using the balloon as a sizing dowel (balloon-assisted banding). Mean access blood flow prior to banding was 1640 mL/min and decreased to 820 mL/min following the procedure. Symptoms resolved promptly in 20 patients and were markedly improved in the remaining two. No patient experienced a loss of their access in the early period following the procedure.

Surgical Bypass

A variety of surgical bypass procedures have been used to treat hemodialysis patients with TCVO. If the obstruction is within the SV central to the confluence of the internal jugular, the simplest approach is to do an internal jugular vein turn-down procedure. In doing this, the cranial end of the internal jugular vein is detached, turned down and connected to the subclavian or axillary vein [98, 124, 125] in order to bridge the obstruction. It is also possible to bridge from the cephalic or axillary vein to the internal jugular vein using a polytetrafluoroethylene graft in order to bypass an obstructed subclavian segment [100, 124]. With a more central obstruction within the BCV, this ipsilateral option is not possible. However, it may be possible to create a bypass to the contralateral jugular system provided it is patent.

In more complex cases, axillary-to-axillary, axillary-to-femoral and axillary-to-iliac venous bypass, and direct bypass to either the SVC, IVC or right atrium have been utilized to provide vascular access. In addition, an all-arterial graft, such as axillary-to-axillary arterial loop and the creation of a graft access from an artery using the heart as venous outflow have been described [124, 126–129].

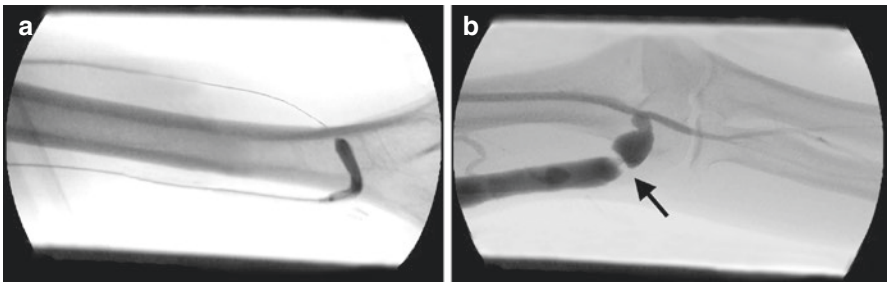

Fig. 17.22 Balloon-assisted banding. (**a**) Angioplasty balloon in place across anastomosis, (**b**) Banding procedure completed (arrow)

Costoclavicular Junction Decompression

The subclavian venous lesion which occurs at the costoclavicular junction (see above, Fig. 17.9) is resistant to PTA and stent placement. These lesions are best treated by decompression; however, this approach to management should be reserved for those cases in which symptoms are severe. There has been significant resistance to other forms of therapy, and alternative access sites is limited [130]. The procedure involves the resection of either the first rib or clavicle followed by a thorough external venolysis to ensure that the adjacent muscle and other tissues are not compressing the access draining vein [40, 42].

Closure of Access

The last resort of treatment is closure of the AV access. This will result in a quick resolution of the signs and symptoms of venous hypertension in the extremity of the patient with TCVO. However, the patient is left without a dialysis access. Access closure may be accomplished in several ways. It can be done surgically or by simply occluding the access for 45 to 60 minutes with an endovascular balloon or manually. This should be done on a non-dialysis day so that the patient will not be anticoagulated.

Creation of AV Access in Patients with TCVO

While not all cases are symptomatic, TCVO can be a devastating problem for the hemodialysis patient. It affects the patient's quality of life, and can render the extremity unusable for dialysis vascular access. Although peritoneal dialysis and kidney transplantation should always be a consideration, a functional AV access can be created for most patients with TCVO. This is especially true in cases in which complete obstruction of the SVC is absence and if the obstruction in more peripheral vessels of the TCV is incomplete. The availability of a device such as the HeRO (Hemodialysis Reliable Outflow) device allow for the creation of a peripheral AV access in patients who are catheter -dependent.

The creation of an AV access in the extremity of a patient with some degree of TCVO, even asymptomatic cases, is frequently thought to be contraindicated because of fear of precipitating or exacerbating the patient's symptoms. As long as the obstruction is not complete, a successful AV access can be created in these patients with careful planning [131]. The key to success in these cases is vascular mapping and avoiding the creation of a high blood flow vascular access.

In cases with complete obstruction, a functional AV access may be possible by using sophisticated techniques to restore patency of an occluded vessel.

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Vascular Steal

18

Gerald A. Beathard

Introduction

Blood flow is determined by pressure and resistance with flow moving in the direction of the lower pressure. If a pressure gradient exists between two vascular beds that are interconnected by collaterals, blood flows from the high to low pressure system. This phenomenon can also occur involving a single artery if there is a significant pressure drop at some point along the course of the vessel such as occurs when there is a stenotic lesion within the artery. Blood flow in collateral arteries interconnecting the high- pressure (pre-stenosis) and the low-pressure (post stenosis) zones is determined by the hemodynamic differences between the two. Blood flows from high pressure to low pressure. Vascular steal was first described and the term "steal" was introduced in an editorial in 1961 [1]. It described a case in which an occluded subclavian artery caused a decrease in downstream perfusion pressure in the vertebral artery originated proximal to the occlusion, resulting in a reversal of blood flow in that vessel because of the interconnecting collateral vessels with higher pressure [2]. Central nervous system symptoms resulted from the vascular steal.

When a dialysis vascular access is created, blood flow is immediately diverted into the low pressure, low resistance venous connection resulting in a decrease in perfusion pressure distal to the anastomosis. Distal hypoperfusion is prevented primarily by collateral blood flow. This pressure gradient can also result in a reversal of blood flow in the artery distal to the access e.g., vascular steal. Although vascular steal can occur in other situations, most of those that are clinically recognized are in association with a dialysis vascular access. If this hemodynamic abnormality is asymptomatic it is referred to as the "steal phenomenon." If it is symptomatic, is referred to as "steal syndrome."

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There are four clinical steal syndromes associated with dialysis vascular access. These have been referred to as: (1) subclavian-coronary artery steal syndrome associated with coronary artery symptoms, (2) subclavian-vertebral artery steal syndrome associated with central nervous system symptoms, (3) ischemic monomelic neuropathy (IMN) associated with neurological symptoms affecting the hand, and (4) dialysis access steal syndrome (HAIDI) associated with ischemic symptoms affecting tissues of the hand. This latter syndrome is the most frequently occurring of the group. Although the term HAIDI has been used extensively to describe the syndrome, hemodialysis access-induced distal ischemia (HAIDI) is a more descriptive term since most patients with a dialysis vascular access have vascular steal and it is asymptomatic (steal phenomenon) [3, 4]. When distal ischemia occurs, it is primarily due to distal hypoperfusion rather than vascular steal as such. In addition, some patients with HAIDI do not have vascular steal [5].

Hemodialysis Access-Induced Distal Ischemia (HAIDI)

Of the two variants of hand ischemia associated with dialysis vascular access, HAIDI is the most common. This syndrome has been reported to have an incidence ranging from 1%–8% [6]. However, what is generally reported are cases requiring surgical intervention. The clinical manifestations of HAIDI vary across a broad spectrum, ranging from mild to severe. Mild symptoms frequent resolve and do not require surgical intervention. In a study which utilized a questionnaire to assess the incidence and severity of symptoms, it was found that mild to moderate symptoms were relatively common [7]. Depending upon the access type, at least one symptom of decreased hand perfusion was present in 38% to 79% of cases.

Risk Factors

The recognition of risk factors (Table 18.1) is important for dialysis access planning. In cases in which there is a significant risk, preventive measures should be taken. The most important patient characteristic predisposing to the development of HAIDI is peripheral vascular disease, both macro- and microvascular. With the creation of an AV access, there is a tenfold or greater increase in blood flow [8]. In order to accommodate this increase, maturation of the feeding artery is necessary. The presence of peripheral vascular disease can interfere with this process and place

Table 18.1 Risk factors for Dass

Peripheral artery disease
Use of brachial artery for anastomosis
High access blood flow
Female gender
Age > 60 years
Multiple previous access procedures

distal tissues at risk. Use of the brachial artery is a major risk factor for the development of HAIDI. Simply moving the anastomosis to the proximal radial artery reduces the risk considerably. Additional risk factors are similar to those found in patients with peripheral arterial disease (PAD), such as diabetes and older age. Female gender and a history of multiple previous access procedures also present an increased risk of HAIDI [9–12]. The incidence of HAIDI is higher in association with an AVF, and especially those with high blood flow [13, 14].

Pathogenesis

Five vascular beds are important in the pathogenesis steal (Fig. 18.1): (1) the proximal artery which feeds into two competing circuits, (2) the AV access including its draining veins and side branches, (3) the artery extending beyond the anastomosis toward the distal extremity, (4) collateral arteries arising from the axial artery proximal to the anastomosis, providing perfusion to the circulation distal to the anastomosis, and (5) the vascular bed of the hand. These five components make up what has been referred to as the hand/vascular access complex [6]. Each of these vascular components can play a role in the pathogenesis of HAIDI. The degree of involvement of each of these components can vary from patient to patient.

Role of the AV Access and Draining Veins

The placement of an AV access creates an abnormal, non-anatomic vascular circuit where a high-pressure low-volume flow artery (typically 50 to 100 mL/min) suddenly becomes the critical element in producing the steal phenomenon, i.e., a

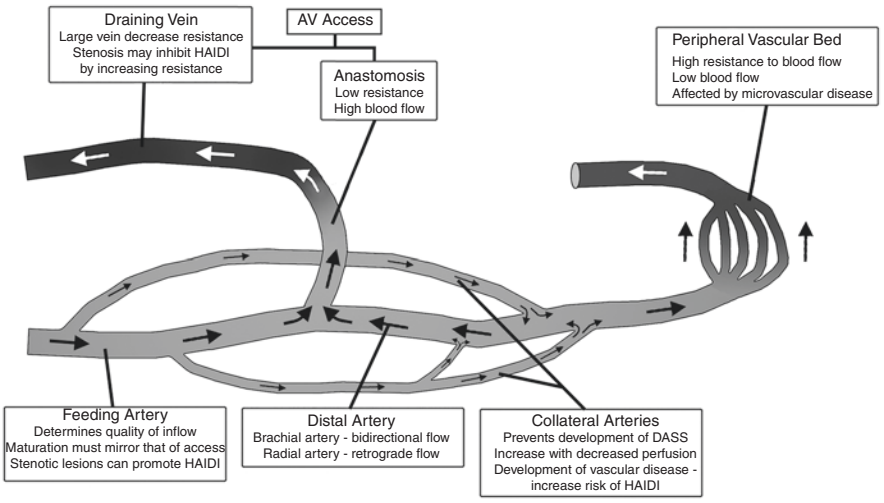

Fig. 18.1 Schematic diagram of arteriovenous access showing components of hand/vascular access complex showing the role that each can play in the pathogenesis of HAIDI

high-flow, low-resistance system. The presence of the AV access results in a reduction in distal arterial pressure and perfusion and promotes retrograde flow in the distal artery. There is a correlation between access blood flow volume and the development of HAIDI [7, 15]. Acute cases tend to occur more often with an AVG due to the immediate high blood flow associated with the large diameter of the graft relative to the feeding artery. In the case of an AVF, progressive maturation with increasing levels of blood flow leads to an increased incidence of HAIDI over time.

As stated above, the effects of the steal phenomenon are generally offset by other components of the hand/access complex. It is only when these compensatory mechanisms are inadequate that ischemia occurs.

The location of the arterial inflow for the AV access is an important factor in the development of HAIDI. A brachial artery access is much more likely to develop problems than a radial one [10, 16] due to a significantly higher blood flow than an access created using a smaller artery.

The draining veins of the AV access can also play an important role in the pathogenesis of HAIDI and in mitigating its symptoms. A large collateral vein associated with an AVF serves to decrease resistance even further resulting in a greater shunting of blood from the distal circulation [17]. The development of venous stenosis within the access or its drainage results in an increase in resistance to flow and a decrease in the steal phenomenon. This can ameliorate or prevent the development of ischemic symptoms. This situation is generally appreciated only when HAIDI develops or worsens immediately following a successful angioplasty which removes the stenosis.

Role of the Feeding Artery

With the creation of an AV access, blood flow in the feeding artery must increase 10- to 20-fold. Initially, this increase in blood flow is the result of the sudden decrease in resistance to blood flow with the opening of the AV access. However, this change in blood flow also results in abnormal wall shear stress resulting in the elaboration of mediators that promote vascular remodeling. These changes represent maturation of the feeding artery, a process that mirrors the maturation of the vein in the case of an AVF. In both the vein and the artery, this maturation phenomenon is characterized by an increase in both blood flow rate and internal diameter (Fig. 18.2). Although the size of the access is constant with an AVG, arterial maturation and increased blood flow are necessary to offset the shunting that occurs through the access. This remodeling of the artery is mandatory in order to supply the demands of the AV access and the needs of distal tissue.

The presence of peripheral vascular disease (atherosclerosis) can prevent this maturation process. This in concert with the blood flow demands of the AVF exerts an adverse effect on distal tissue perfusion and can result in HAIDI. In cases in which a critical degree of arteriopathy is already present, arterial maturation failure exerts its effect early during the period of rapid change in the AVF blood flow, leading to the early development of HAIDI. With an AVG the problem is manifest immediately. Adverse changes that affect arterial blood flow may also occur over time, as pre-existing arteriopathy progresses or as a manifestation of

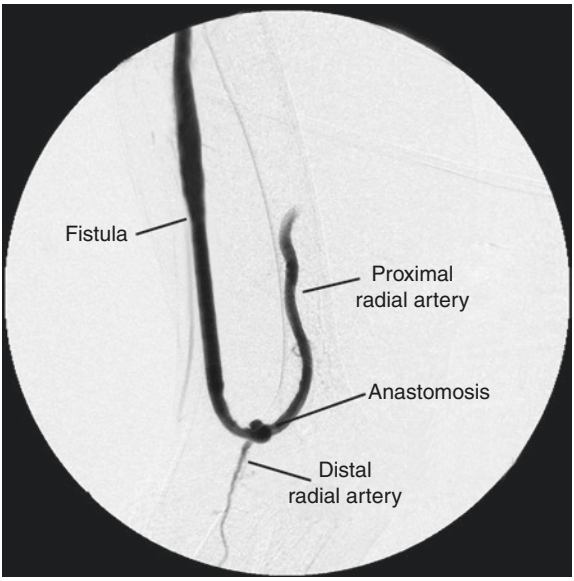

Fig. 18.2 Radial-cephalic fistula. Note the difference in size of the proximal and distal radial arteries. The distal radial artery represents the normal size, the proximal radial artery reflects maturation due to the presence of the fistula

newly-developed arterial disease. These changes result in progressive reduction of tissue perfusion, increasing the risk of hand ischemia [11, 18]. Reports have shown the presence of a proximal arterial stenosis in 20% to 30% of patients who present with distal hand ischemia [19, 20], and in one small series the incidence was even higher [21].

Role of Collateral Arteries

Branches of the major vessels of the upper extremity and hand provide collateral circulation to these areas when blood flow in the major vessels is compromised [22–24]. Two changes occur in the collateral circulation after the creation of an AVF, vasodilatation which appears early and an increase in blood flow. These two events stimulate the development of a robust compensatory collateral network [11, 25]. This change acts to off-set the deleterious effects of the steal phenomenon and prevent hand ischemia. These vessels also experience the phenomenon of arterial maturation which mirrors that of the access. This mechanism becomes even more critical if there is a restriction of blood flow in the primary feeding artery.

In the case of an upper arm access, branches of the brachial artery communicate with branches of both the ulnar and radial arteries across the elbow to provide collateral circulation to the forearm when blood flow in the brachial artery is compromised. With an access originating from the radial artery, the ulnar artery through the palmar arch plays an extremely important role in providing collateral circulation to the hand (Fig. 18.3).

In addition to the radial and ulnar arteries which provide most of the blood supply to the hand, there is also a significant contribution from the median artery and the interosseous arterial system.

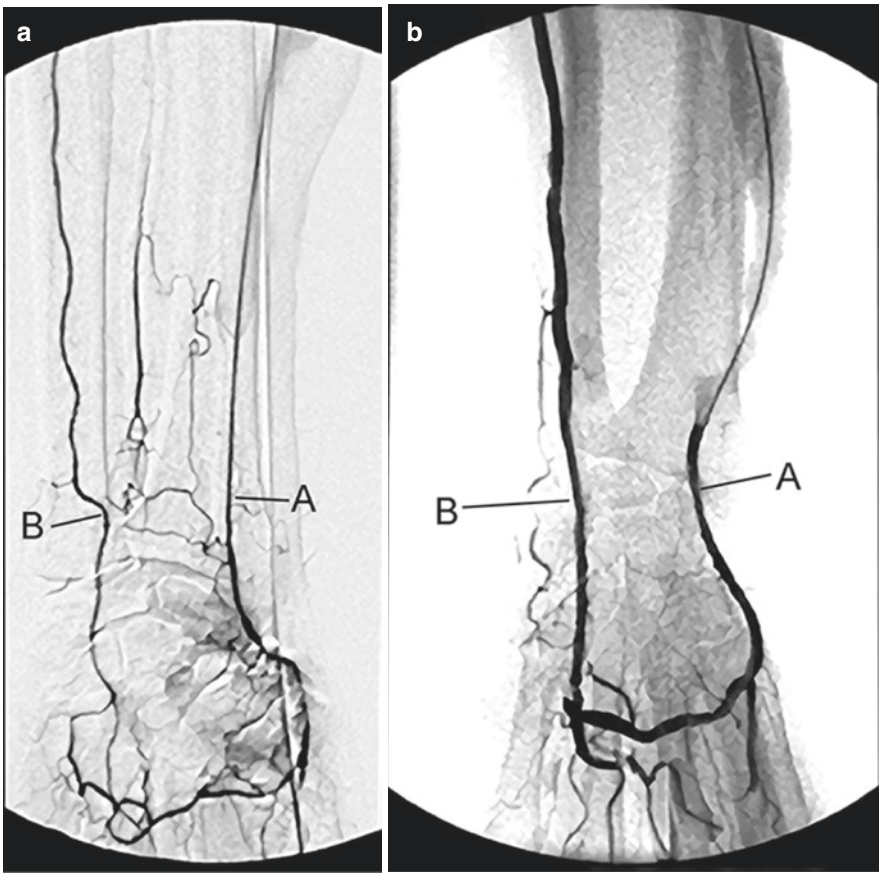

Fig. 18.3 Maturation of collateral vessels. (**a**) normal appearance of radial artery (**A**), ulnar artery (**B**) and palmar arch. (**b**) appearance of vessels in patient with radial-cephalic AVF. Marked enlargement of the distal radial artery (**A**), ulnar artery (**B**) and palmar arch is apparent. These changes represent maturation of these vessels due to increased collateral blood flow

Role of Artery Distal to Anastomosis

The role of the artery distal to the anastomosis in the pathogenesis of HAIDI varies according to the site of the anastomosis used for the creation of the access. With a forearm access using the radial artery, blood flow in the distal artery is retrograde [3, 6]. In this instance, the artery serves as a shunt allowing blood to bypass the hand by coursing through the palmar arch and travel retrograde up the distal artery to the anastomosis. By doing this it plays a major role in the pathogenesis of the syndrome. As discussed below, occlusion of the distal artery will result in a resolution of the ischemic symptoms.

With an upper arm access using the brachial artery for the anastomosis, blood flow in the distal brachial artery and its major branches is typically thought to be bidirectional, antegrade during systole and retrograde during diastole [6, 14].

However, some investigators have found that it is consistently antegrade, i.e., there is no steal present [26, 27]. In these cases, it is proposed that HAIDI results from regional hypotension as a result of either insufficient remodeling of the feeding artery or severe atherosclerosis leading to a loss of perfusion pressure in the distal tissues [5, 28]. It has also been demonstrated that there is a blood pressure drop associated with turbulent blood flow at the arteriovenous anastomosis which adds to the distal hypotension [5]. In addition to bidirectional and antegrade blood flow in the distal artery, cases with large collateral bypassing the point of anastomosis, blood flow in the distal artery may be retrograde above the insertion of the collateral [26].

Role of Peripheral Vascular Bed of Hand

The peripheral vascular bed of the hand is the low flow, high resistance component of the hand/vascular access complex. With the creation of an AV access there is an immediate drop in the pressure in the digital arteries. This can be appreciated by assessing the digital-brachial pressure index (DBI)—the ratio of the digital blood pressure to the contralateral brachial blood pressure (the normal value for this test is a ratio greater than 0.75).

In one study [29], the DBI in 35 patients with end-stage renal disease was evaluated before and after access creation. Values were obtained preoperatively, on the day of surgery, and 1 month postoperatively. After access creation the DBI dropped in 28 (80%) of the 35 patients. Six patients (17%) developed symptomatic HAIDI, 3 of whom (9%) eventually required access revision. In those patients without ischemic steal symptoms ($n = 29$) the mean DBI decreased from 0.9 to 0.7 ($p < 0.01$) immediately after surgery and stabilized at 1 month. For those with symptomatic HAIDI, the DBI decreased from 0.8 to 0.4 ($p < 0.01$) immediately after surgery and decreased no further at 1 month. Utilizing a DBI < 0.6 as a diagnostic value, sensitivity was 100%, specificity was 76%, the positive predictive value was 46%, and the negative predictive value was 100%.

The presence of microvascular disease involving the peripheral vascular bed results in increased resistance to blood flow and creates a major risk for the development of hand ischemia. A vascular resistance that is higher than normal at the time of access creation can cause difficulties immediately or very early. Since this arteriopathy is generally progressive, problems can worsen or develop over time.

Signs and Symptoms

The spectrum of signs and symptoms associated with HAIDI range from minor—cold hand to devastating—tissue loss (Table 18.2). A patient may have very cold hands with no or only minimal complaints of pain. These two functions are not necessarily synchronous. Normal microvascular blood flow in the digits is characterized by two components: (1) thermoregulatory flow, which is modulated by arteriovenous anastomoses and contributes to body temperature control; and (2) nutritional blood flow, which is provided by papillary capillaries and maintains

Table 18.2 Signs and symptoms of DASS in order of increasing severity

Nail changes
Coolness of hand and fingers
Tingling and numbness in hand and fingers
Muscle weakness in hand
Pale or cyanotic fingernail beds
Hand pain with exercise
Hand pain at rest
Sensory and motor function deficit in hand
Fingertip ulcerations
Tissue loss

tissue viability [30, 31]. Thermoregulatory flow is more sensitive; therefore, when hand ischemia develops, sensory loss will generally present before motor loss [20]. In some instances, symptoms of HAIDI may become evident only after an unrelated minor trauma, when a fingernail or fingertip becomes a non-healing infection leading to gangrene and tissue loss.

As stated above, mild and moderate symptoms associated with HAIDI are experienced on a much larger scale than generally reported. The clinical diagnosis is dependent upon the symptom threshold required by the patient to voice a complaint and the physician performing the evaluation. The frequency of the symptoms was shown in a study involving patients with three different types of AV access using a detailed hand ischemia questionnaire [7]. In all instances, the access had been used for six months or more. In patients with a brachial-cephalic AVF, 79% (22/28) gave a positive response on the questionnaire for at least one symptom of HAIDI compared with 38% (25/65) of those with a radial-cephalic AVF and 52% (14/27) with a loop AVG. None of these cases with mild to moderate symptoms require intervention. Examination of the contralateral arm (not have an AV access) in those cases with symptoms of HAIDI found that pallor/cyanosis was present in 14%, trophic lesions in 14% and prolonged capillary refill in 7%. These findings underscore the systemic nature of the macrovascular and microvascular disease present in patients with HAIDI.

Classification of HAIDI

HAIDI may be classified in two ways, (1) according to signs and symptoms and (2) according to time of appearance.

Clinical Classification

Because the signs and symptoms associated with HAIDI represent a relatively broad spectrum, a mechanism for clinical classification of individual cases is of value. The classification most frequently used is based upon 4 grades denoting increasing levels of symptom severity (Table 18.3) [3]. This classification system offers the benefits of being based upon the individual patient's signs and symptoms and is applicable as a guide for planning a treatment strategy. However, it is

Table 18.3 Clinical grades of DASS

Grade 1—Pale/livid hand and/or cool hand without pain
Grade 2a—Tolerable pain during exercise and/or dialysis
Grade 2b—Intolerable pain during exercise and/or during dialysis
Grade 3—Rest pain and/or loss of motor function
Grade 4a—Limited tissue loss, potential for preservation of hand function
Grade 4b—Irreversible tissue loss, significant hand function is lost

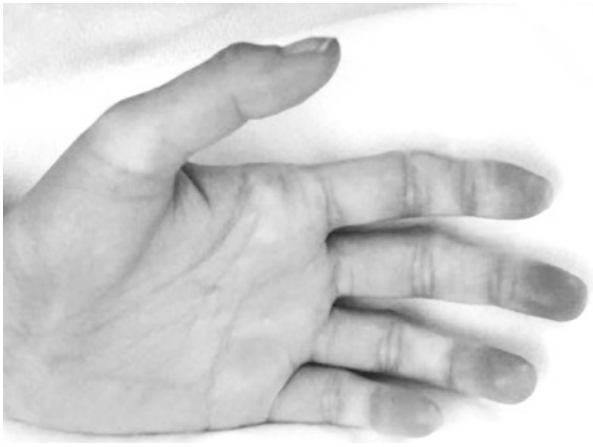

Fig. 18.4 Ischemic hand with pale areas and cyanotic fingertips. May be seen with HAIDI Grades 1–3

important to realize that the signs and symptoms complex that characterizes HAIDI represent a continuous spectrum, and placing patients into the defined categories is somewhat subjective.

Grade 1

Signs: Pale or cyanotic nail beds, mild coldness of skin of hand (Fig. 18.4). The arterial pulse at the wrist is often reduced as is the systolic finger pressure.

Symptoms: None or mild symptoms—numbness and paresthesia.

Grade 2

Grade 2 cases are subdivided into two categories having management implications.

Grade 2a

Signs: Pale or cyanotic nail beds, coldness of skin of hand, reduced arterial pulsations at wrist.

Symptoms: Tolerable pain (cramps, paraesthesia, numbness, coldness) with intense use of hand or during dialysis treatment.

Grade 2b

Signs: Pale or cyanotic nail beds, coldness of skin of hand, reduced arterial pulsations at wrist.

Symptoms: Intolerable pain (cramps, paraesthesia, numbness, coldness) with intense use of hand or during dialysis treatment. This determination is based *entirely upon the patient's definition.*

Grade 3

Signs: Pale or cyanotic nail beds, coldness of skin of hand, reduced arterial pulsations at wrist.

Symptoms: Rest pain or motor dysfunction of fingers or hand.

Grade 4

Grade 4 cases are subdivided into two categories that have management implications.

Grade 4a

Signs: Tissue loss (ischemic ulceration, necrosis). Nail changes, motor and sensory loss (Fig. 18.5a).

Symptoms: Rest pain or motor dysfunction of fingers or hand.

Grade 4b

Signs: Extensive tissue loss (Fig. 18.5b).

Symptoms: Rest pain or motor dysfunction of fingers or hand. Irreversible tissue loss of the hand.

Classification Based Upon Time of Onset

HAIDI may appear acutely within hours to days following AV access surgery and may either worsen or subside over time. It may also occur more subtly in a chronic form which develops later and is often progressive. Because of these differences, the natural history of HAIDI has been classified based upon time of development

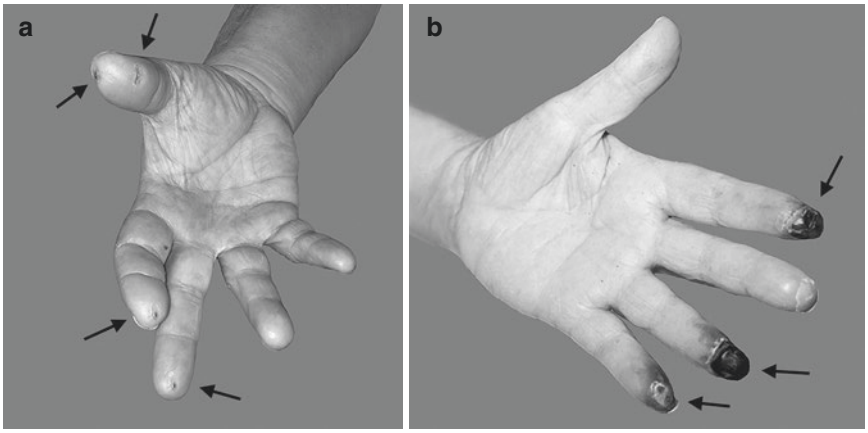

Fig. 18.5 Grade 4 HAIDI. (**a**) trophic changes at fingertips (arrows). (**b**) dry gangrene of fingertips (arrows)

after AV access creation as acute (within 24 hours), sub-acute (within 1 month) or chronic (>1 month) [13, 16, 32].

In a meta-analysis of 21 reports of cases treated for HAIDI (surgical or endovascular) which included 464 cases [16], the time of onset and the type of access were tabulated (Fig. 18.6). Acute HAIDI was diagnosed in 22% (104/464) of the cases. Most of these acute patients (87%, 91/104) had an AVG; the remaining 13%, (13/104) had an upper arm AVF. Subacute HAIDI was present in 21% (97/464) of the cases, and 79% (77/97) had an upper arm AVF compared to 21% (20/97) with an AVG. A total of 263 patients (57%) had chronic HAIDI which developed 16 ± 3 months after the AV access construction. Of these chronic patients, 88% (232/263) had an AVF compared to 12% with an AVG (31/263). Chronic HAIDI was not commonly associated with an access in the forearm (9%, 20/232) compared to elbow level (91%, 212/232).

The pathogenic mechanisms affecting the time of onset of HAIDI are related to the hemodynamic changes that occur in the access following its creation and how they are affected by the patient's vascular pathology (Fig. 18.7) [6]. Both acute and subacute HAIDI are related to pre-existing pathology. With an AVG, which cannot dilate, blood flow is maximal at the onset. The greater incidence of acute HAIDI with an AVG is most likely due to the artery-to-graft size mismatch, plus a combination of macrovascular and microvascular disease, resulting in an immediate overload of the collateral circulation and failure of compensatory mechanisms. In the case of an AVF, remodeling as part of the maturation process results in progressive vessel hypertrophy and higher levels of blood flow, increasing the risk of chronic HAIDI as the process progresses. In addition to underlying arterial disease, subacute and chronic HAIDI can occur due to disproportionate vessel maturation.

It should also be noted that steal can also occur immediately after an angioplasty procedure to treat a stenotic outflow lesion. In these cases, relief of the stenosis results in a sudden decrease in resistance within the access. This causes more blood to be shunted away from the distal circulation.

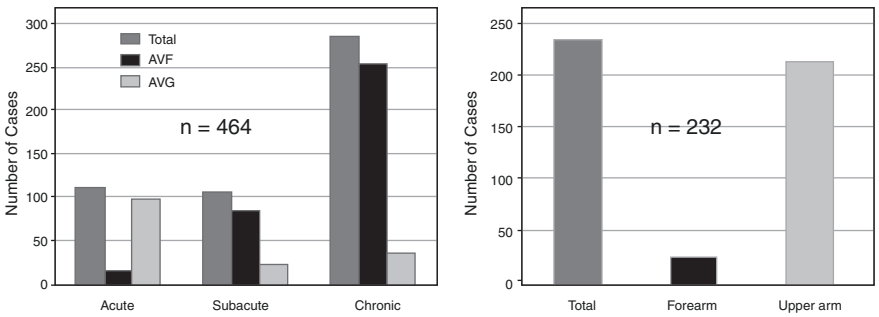

Fig. 18.6 Timing and location of HAIDI cases

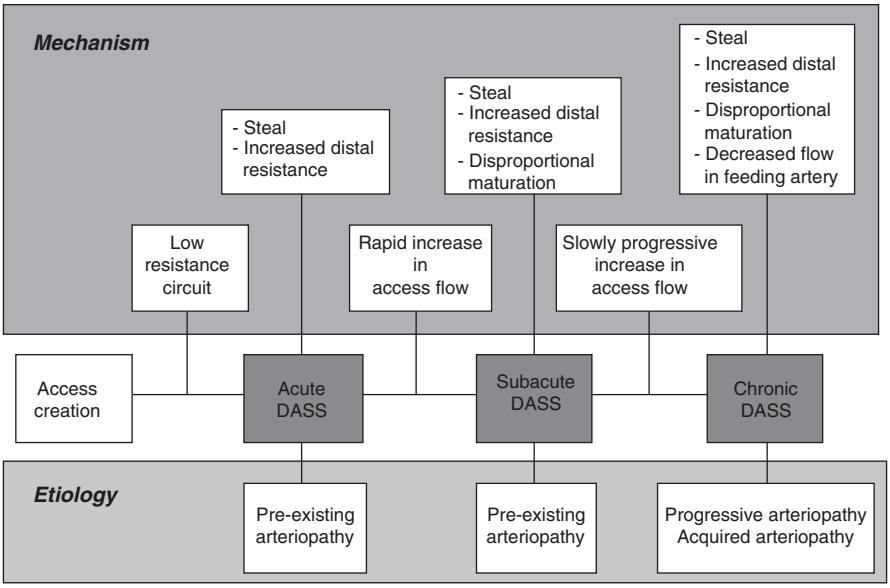

Fig. 18.7 Pathophysiology of HAIDI by time of onset

Diagnosis

Early diagnosis of HAIDI is important in order to facilitate timely treatment, and making an early diagnosis is dependent upon maintaining a high level of suspicion for the condition on the part of all healthcare professionals caring for the hemodialysis patient. Because of the critical importance of early diagnosis, patients should be instructed to report immediately any coldness, loss of motion or significant reduction in sensation in their access hand.

HAIDI can generally be diagnosed clinically based upon signs and symptoms presented by the patient [3, 33–35]. Physical examination of the hand will reveal cold skin, pallor, cyanosis, diminished sensation, and, ultimately, ulceration and gangrene. The radial pulse is usually diminished or absent, but in some cases, the volume of the radial pulse may be normal on palpation although the patient's symptomatic [21]. Symptoms of HAIDI can generally be relieved, at least to a degree, by manual occlusion of the AV access. This is considered to be a compelling sign supporting the diagnosis of HAIDI. Manual occlusion of the access also augments the distal pulse.

Initial clinical evaluation should always include blood pressure measurement in both arms performed sequentially. The purpose of this aspect of the examination is to test for an arterial lesion above the level of the blood pressure cuff, i.e., the subclavian artery. A difference of 10 mm Hg or less is considered to be normal, a difference in the range of 10 to 20 mm Hg is marginal, and 20 mm Hg or greater. is suggestive of subclavian artery stenosis in the low-pressure arm [36].

If the diagnosis is not readily obvious based upon the clinical features presented, there should be a low threshold for performing additional testing. These tests are of value not only in confirming the presence of HAIDI but also in defining the underlying cause of the problem, guiding management decisions, and monitoring the results of therapy. Some tests such as Doppler ultrasound and angiographic evaluation are considered mandatory. Additional tests are available that are valuable in questionable cases or in following the progress of mild cases, include digital-brachial index (DBI), basal digital blood pressure (BDP), and nerve conduction studies.

Ultrasound Evaluation

Doppler ultrasound (DUS) is an important tool in the evaluation of the patient with suspected or proven HAIDI. Using this modality, the direction of blood flow in the distal arterial tree can be easily determined. Although this examination is not diagnostic of HAIDI, it can demonstrate the presence of the steal phenomenon in suspected cases. Whether it is physiologic or pathologic steal depends upon the patient's signs and symptoms. Blood flow in the distal artery may be retrograde [37] or bidirectional [11, 38] with or without the presence of HAIDI. With the AVF occluded it may be possible to assess the run-off in the collateral arteries. In addition, arterial assessment using DUS should identify an inflow arterial stenosis, although this technique may be limited in very proximal (subclavian) stenosis. The presence of low blood flow rates associated with HAIDI, combined with severe arterial calcification, can reduce the diagnostic accuracy of Doppler ultrasound [39].

Ultrasound is also important in the determination of AVF blood flow volume, which is critical for the planning of appropriate patient management. This measurement should be made from the brachial artery regardless of whether or not one is dealing with a radial or a brachial artery-based AVF [40–48]. In the case of a brachial artery access the measurement should be made at least 5 cm proximal to the anastomosis. Because the incidence of high bifurcation of the brachial artery has been reported to be in the range of 12% to 19% [49, 50], care must be taken to ensure that the measurement is actually being made from the brachial artery.

Angiographic Evaluation

Since three of the four components of the hand/vascular access complex involve the arterial system, angiographic evaluation of the patient with HAIDI is very important. This evaluation should include imaging from the aortic arch to the palmar arch to evaluate for both proximal and distal arterial disease. These studies are critical not only to aid diagnosis but also to plan surgical management. If arterial lesions are identified, treatment may solve the ischemic problem [21, 51].

Although not diagnostic of HAIDI, angiography with and without AVF compression can demonstrate the character of the distal arterial flow associated with the steal phenomenon. In most instances, these patients will have minimal, if any, radiocontrast flowing into the artery distal to the anastomosis. In some cases, radiocontrast will be seen to flow into the downstream artery with the pressure of the injection and then reflux back into the arteriovenous access as the pressure resolves. Slow,

sluggish flow to the palmar arch from the ulnar arterial may be observed. With the access occluded, these changes revert back toward normal.

Digital Blood Pressure Evaluation

Because the major determinant of ischemia of the hand and digits characteristic of HAIDI is perfusion blood pressure in the peripheral vascular bed, digital blood pressure measurements have a strong correlation with the disease. It is recommended that digital blood pressure be used in cases where diagnosis is not clear [20, 35, 51–53]. These pressure readings have been utilized in three ways—(1) basal digital pressure (BDP), i.e., blood pressure measured in the digit under basal conditions; (2) digital- brachial index (DBI), the ratio of the digital blood pressure to the contralateral brachial artery blood pressure; and (3) the change in digital blood pressure (CDP) with AV access compression.

A BDP <60 mmHg or a DBI <0.4 in a symptomatic patient with an AV access is generally accepted as supporting the diagnosis of HAIDI. In a study [54] in which both of these metrics were assessed, it was found that both BDP and DBI were significantly lower in patients with HAIDI than those without—30 mmHg versus 102 mmHg ($p < 0.001$), and 0.3 versus 0.8 ($p < 0.001$), respectively. Using these data, the cut-off of BDP of <60 mmHg was shown to have an accuracy of 92% (sensitivity of 100%, specificity of 87%) in diagnosing HAIDI. Using a cut-off level of <0.4 DBI, accuracy was 94% (sensitivity 92%, specificity 96%).

In patients with HAIDI, manual occlusion of the access should return the BDP back toward normal [7, 55]. A CDP to near-normal levels with manual compression of the AVF suggests that the steal phenomenon in these patients is reversible and will be responsive to corrective surgery if required [19]. Although CDP is not as reliable diagnostically as BDP and DBI, it does play a worthwhile role in the evaluation of a patient with suspected AV access-induced hand ischemia. Rarely, a patient may have hand ischemia secondary to the progression of native arterial disease unrelated to their AV access. In these cases, the patient will have a low BDP and DBI indicative of poor digital blood pressure but will not have a significant CDP, because steal is not playing a role in the pathology of the ischemia. These cases will not benefit from surgical intervention directed toward the AV access.

Nerve Conduction Evaluation

Nerve conduction studies have been used to evaluate the nerve damage that occurs with HAIDI. These studies generally consist of assessing amplitude and latency of evoked action potentials in the ulnar and median nerves using surface recordings from the thenar and hypothenar eminences, and index and little fingers, respectively. Abnormal studies correlate well with ischemia related to HAIDI and have been advocated as a means of following patients whose symptoms are not severe enough to require intervention. It has been recommended that a deterioration of nerve conduction, even in the face of symptoms that are mild, be taken as an indication for surgical treatment [35].

Differential Diagnosis

The most important condition to be ruled out is IMN, but others include carpal tunnel syndrome, diabetic neuropathy, iatrogenic nerve damage and arthropathy. Pre-existing conditions such as arthropathy and diabetic neuropathy can generally be ruled out as etiologies of symptoms because of their presence prior to access surgery. Formulating a differential diagnosis for newly developed hand pain should take into consideration whether symptoms develop early or late after access creation. In the early setting, hand pain begins immediately after access creation, and numbness and tingling of the hand can occur secondary to surgery due to soft tissue swelling or the formation of a hematoma compressing a nerve. This is not unusual and typically resolves within the first 2 to 4 weeks [56].

IMN (see below) occurs acutely and can be diagnosed clinically on the basis of an immediate onset of excruciating pain after AV access creation, most commonly observed when the brachial artery has been used. All three nerves (median, radial and ulnar) are involved. Involvement of a single nerve in the setting of vascular access surgery should prompt a search for a local nerve compression secondary to a complication of the surgery [57]. Neurologic symptoms and signs predominate, and the hand is warm and shows no evidence of ischemic changes. Nerve conduction studies can be used to confirm the diagnosis. IMN should not be a consideration for hand pain that develops late following AV access creation.

Carpal tunnel syndrome is due to median nerve compression at the wrist as it passes through the carpal tunnel. This syndrome is common in dialysis patients. It may be recognized as a pre-existing condition, become manifested or be exacerbated by access surgery [58, 59], or develop chronically in relation to AV access creation. Although carpal tunnel syndrome is generally bilateral, it has been reported to be worse in the hand ipsilateral to the AV access [60]. An electromyelogram showing reduction of motor conduction can help to establish the diagnosis [61].

Prevention

Unfortunately, there is no uniformly accepted, objective approach to the prevention of HAIDI. The recommended approach may be considered under four headings—(1) demographic characteristics (risk factors), (2) clinical evaluation (vascular mapping and digital blood pressure assessment), (3) presurgical planning, and (4) intraoperative evaluation.

Demographic Characteristics

A great deal has been written about risk factors (see Table 18.1) associated with the development of HAIDI, and it is generally accepted that the likelihood of developing HAIDI is proportional to the number of risk factors that are present. These factors have been identified based upon their prevalence in patients who have the

condition. Unfortunately, this is problematic, especially for chronic HAIDI. Predictability based upon risk factors has not been validated in all published studies [7, 54, 55, 62]. The fact that the development of HAIDI is multifactorial also adds to the uncertainty of predicting its development in an individual patient.

In some instances, patients may present with the need for creation of a new dialysis access having already had a prior episode of HAIDI, or even having had ischemia necessitating access ligation or an amputation. Additionally, cases with a history of amputations due to primary vascular disease may present with the need for dialysis access creation. Some investigators have suggested that certain demographic characteristics identify a patient has "high-risk" [10, 63, 64].

- Patients with two or more risk factors
- Patients who have had previous amputations due to peripheral arterial disease
- Patients who have had HAIDI with a prior dialysis access

All such patients should be considered to be at greater risk for HAIDI development after construction of a new vascular access [64]. These higher risk patients should be counseled preoperatively, their operative plans should be designed to reduce the risk of hand ischemia, and they should be observed closely postoperatively [65]. Nevertheless, a "high-risk patient" profile is not totally clear, and unless definitive evidence from ultrasound, digital pressure assessment or angiography is found, the creation of an AV access should not be automatically avoided.

Clinical Evaluation

The clinical evaluation of the patient's vascular anatomy and digital blood pressure plays an important role in access planning in the prevention of HAIDI.

Vascular Mapping

Every patient considered for an AV access should have a detailed and complete evaluation, including vascular mapping, before surgery [66]. The arterial aspect of this evaluation is particularly relevant to the prevention of HAIDI.

Digital Blood Pressure Assessment

DBI has been shown to be of definite value for making the diagnosis in patients with symptoms suggestive of HAIDI. Although it has been advocated as a tool to predict the development of HAIDI for patients who are at increased risk [9, 67, 68], there is no absolute value for DBI that can accurately predict who will develop HAIDI. Studies suggest that the lower the DBI threshold used, the greater the sensitivity and positive predictive value (PPV), but there is no value at which the development of HAIDI is inevitable [9, 67, 68]. In one investigation [67] involving intra-operative measurements taken at the time of access construction, a DBI threshold of 0.6 was found to have a specificity and PPV of 43% and 21%, respectively. However, a DBI of 0.45 was a more accurate predictor of HAIDI with a sensitivity of 80% and a PPV of 30%.

Surgical Planning

Surgical considerations are important in the planning of hand ischemia prophylaxis. There are several surgical approaches that have been recommended. For an upper arm access, these recommendations include using the proximal radial artery for access inflow instead of the brachial artery, limiting the size of a brachial artery anastomosis, and proximalization of the brachial artery inflow (see PAI below) [11, 64, 69, 70]. If either the distal radial or ulnar artery is not suitable for forearm access construction, a "middle-arm" AVF created utilizing the proximal radial artery should be considered. This AVF is bidirectional and has been shown to function well with a low incidence of hand ischemia [70].

Intraoperative Evaluation

The loss of a preoperatively palpable radial arterial pulse immediately after access construction is considered to be an ominous sign and should generate a high index of suspicion for HAIDI [71]. If Doppler signals are obtainable at the wrist and palm in the operating room following access construction, one can simply observe the patient closely in the postoperative period, with intervention indicated if significant ischemic symptoms develop. If no Doppler signals are obtainable at the wrist and palm at the time of access construction, the first step should be to optimize patient hemodynamics and environmental temperature. If this fails to return perfusion to the hand, further assessment should be considered to investigate for arterial pathology, such as spasm, dissection, or thrombus. If it is obvious that the patient is experiencing acute HAIDI, immediate surgical revision based upon access blood flow measurement should be undertaken.

An intraoperative DBI can help predict the risk of hand ischemia, a level less than 0.45 being critical [9, 67]. There are several surgical alternatives available when this occurs. The choice should be based upon blood flow measurements.

Treatment

It should be recognized that not all cases of HAIDI require intervention. When treatment is indicated, the goal of treatment should be twofold: (1) correct the underlying pathology in order to improve distal perfusion, and (2) preserve the patency of the AV access. Of these two, the first takes precedence; however, both are generally possible. In addition to finding and treating primary arterial lesions which can present as HAIDI [21, 51], management of HAIDI should be individualized based upon five clinical features (Fig. 18.8)—(1) the clinical status and prognosis of the individual patient, (2) the grade of the disease, (3) the level of blood flow in the access, (4) the status of the arteries proximal and distal to the arterial anastomosis, and (5) the location of the arterial anastomosis.

Clinical Status and Prognosis

A first step in planning for HAIDI management should be to evaluate the patient's clinical condition including comorbidity burden and degree of frailty (a loss of

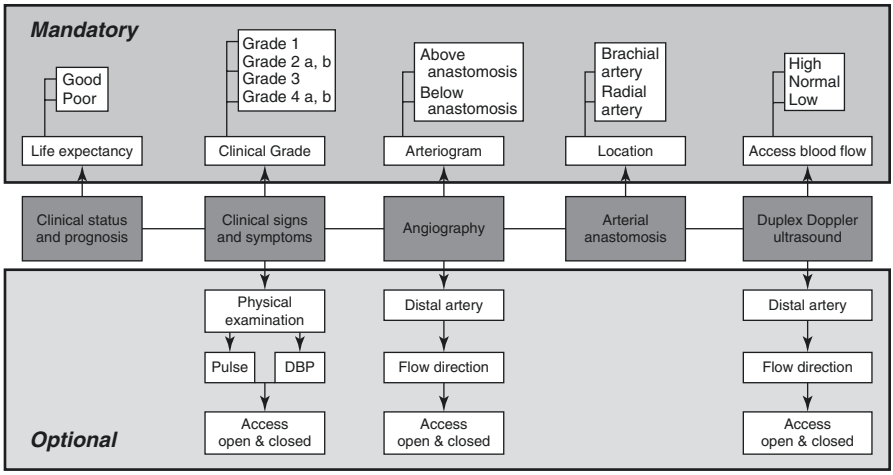

Fig. 18.8 Algorithm for clinical evaluation prior to planning therapy

cognitive, functional, and health reserves leading to increased vulnerability). Many of the comorbidities that define mortality risk also place the patient at risk for distal hypoperfusion. As result, patients who develop HAIDI tend to have a shortened life expectancy [72]. Patient frailty is also common in the dialysis patient population [73–75]. This factor has been shown to limit life expectancy and is not age-specific [73–75]. If the patient's clinical condition is poor suggesting a poor life expectancy, the best approach would be to ligate the AV access and insert a tunneled dialysis catheter and continue with palliative dialysis.

Clinical Grade of HAIDI

Determining the clinical stage of the disease is important in determining the level of treatment indicated in the individual patient [16].

Grade 1

Many dialysis patients with a newly created access have mild symptoms that could be interpreted as Grade 1 HAIDI, since this is basically a subjective determination. Treatment of these cases is not indicated. Mild symptoms generally abate within a few weeks as tissue trauma associated with surgery resolves and compensatory mechanisms such as the development of collaterals takes place. Conservative and symptomatic treatment may be beneficial—various hand warming techniques such as wearing a glove, reducing antihypertensive agents if appropriate, performing hand exercises, and using vasodilatory medication [76]. In some instances, Grade 1 cases are progressive. Frequent follow-up evaluation is important, with special attention to subtle neurologic changes and evidence of muscle wasting. Deterioration of nerve conduction, even in the face of symptoms that are mild, should be taken as an indication for surgical treatment [35].

Grade 2

Grade 2 HAIDI includes the symptoms of stage I with the addition of hand pain. Since pain is patient-specific, and there are definite differences in a patient's threshold and perception of pain, classification as 2a or 2b is based entirely upon the patient's subjective experience of pain.

With Grade 2a HAIDI immediate treatment is not indicated. The first step should be conservative management as outlined above. Additionally, in cases that either have occurrence or worsening of pain only on dialysis, holding antihypertensive medications before dialysis may be helpful. With early postoperative appearance (< 30 days) and no neurologic deficits present, 80% of patients will have spontaneous and significant pain relief [33, 35, 77]. Treatment for patients with Grade 2a HAIDI is indicated only when: (1) significant pain or other symptoms fail to ameliorate after 1 month and symptomatic treatment is not effective, (2) neurologic deficits are present [78], or (3) the ischemic changes appear to be advancing [33, 79]. If repeated nerve conduction studies demonstrate deterioration, surgical treatment of HAIDI should be offered, even with mild clinical signs and symptoms [35]. If the pain is intolerable (Grade 2b) after conservative management measures, then immediate surgical intervention is indicated.

Even in Grade 2 HAIDI cases, blood flow should be assessed. If found to be low, an etiology should be sought and treated. If improvement in blood flow is not possible, consideration should be given to ligating the access, since the problem is likely to progress. In cases of high blood flow, a flow reduction procedure should be considered [3].

Grade 3

Either motor impairment or pain at rest occurring any time postoperatively is a clear indication for surgical intervention [35].

Grade 4

These patients are in a critical state. With more severe signs and symptoms of hand ischemia such as ulcers or gangrene, there is a risk for loss of digits or even the hand. In these cases, immediate evaluation and treatment are essential in order to reverse the ischemia and minimize the degree of disability while saving the access, if possible. Occasionally the functional deficit is acute and rapidly progressive, closure of the access may represent the best alternative in these cases.

Status of the Arteries Proximal and Distal to Anastomosis

As has been emphasized, an important aspect of the evaluation of every patient with suspected HAIDI should be a radiologic evaluation of the entire arterial inflow from the aortic arch to the anastomosis and from the anastomosis to the hand, including the palmar arch. Arterial stenosis is frequently present, and its treatment may solve the ischemic problem [21, 51, 80, 81].

Location of Arterial Anastomosis

The location of the arterial anastomosis, whether it is at the wrist (radial artery) or the elbow (brachial or proximal radial artery), is an important determinant for the approach to therapy once a decision has been made to treat HAIDI.

Forearm

HAIDI is not seen as frequently with a forearm AVF as with an upper arm AVF, especially those associated with the brachial artery. The radial-cephalic AVF is unique in that the distal radial artery serves only to permit retrograde blood flow, which facilitates the steal phenomenon. Occluding the artery and interrupting retrograde flow from the palmar arch will resolve the steal [28, 67, 82, 83]. Its loss in this situation has no adverse effect; however, it is critical to confirm that an intact ulnar artery and palmar arch provide perfusion of the hand prior to radial artery occlusion.

Retrograde flow in the radial artery distal to the arterial anastomosis of a radial-cephalic AVF is easily interrupted. This can be done with an endovascular approach by placing an embolization coil in the vessel [84–86] or by surgical ligation [87] (Fig. 18.9), a procedure referred to as DRAL (distal radial artery ligation).

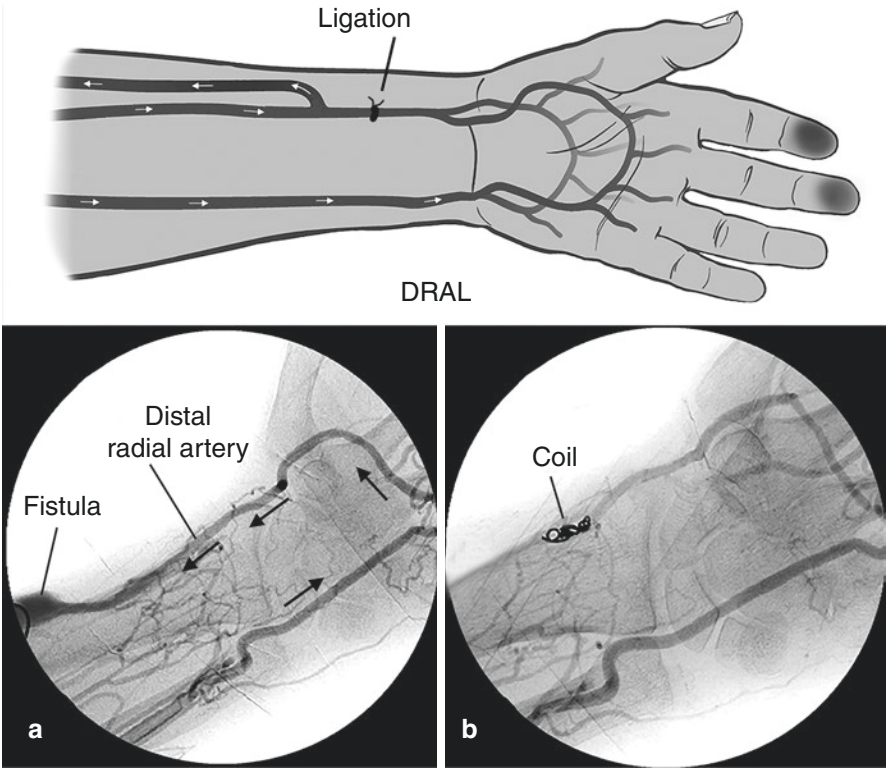

Fig. 18.9 Treatment of HAIDI in radial cephalic AVF. Top panel—distal radial artery ligation (DRAL), (**a**) distal radial artery with retrograde blood flow, (**b**) coil placed in distal radial artery to obstruct retrograde blood flow

Interruption of flow prevents retrograde flow and eliminates the steal [28, 67, 82, 83]. Because it avoids the potential complications of surgery and the ease with which it can be done, the endovascular approach using an embolization coil is the preferred treatment [84].

It should be noted that a distal radial artery associated AVF with an abnormally high blood flow and aneurysmal dilatation can be treated in the same manner as described below for an upper arm AVF.

Upper Arm

This category includes both brachial artery and proximal radial artery-based accesses. However, as has been indicated, a brachial artery-based access is at higher risk for the development of HAIDI. No single technique is suitable for all cases of HAIDI associated with the upper arm AV access. Four procedures have been described. Three are referred to by their acronyms—PAI, DRIL and RUDI and the fourth is referred to as precision banding. It is important to recognize that these procedures are not equally effective in restoring adequate distal perfusion. Preoperative access blood flow measurements are indispensable in planning the optimal treatment of HAIDI. For treatment purposes, normal access blood flow for an AVF is considered to be 800 mL/min, and 1000 mL/min for an AVG. Flows below these values are classified as low blood flow and above as high blood flow. For management purposes, a differentiation is made between (1) low to normal and (2) high categories (Fig. 18.10).

Low to Normal Access Blood Flow

This category is defined as blood flow that is equal to or less than 800 mL/min for an AVF and 1000 mL/min for an AVG. The treatment goal in this group of patients is to either maintain or increase perfusion to the hand without losing the AV access.

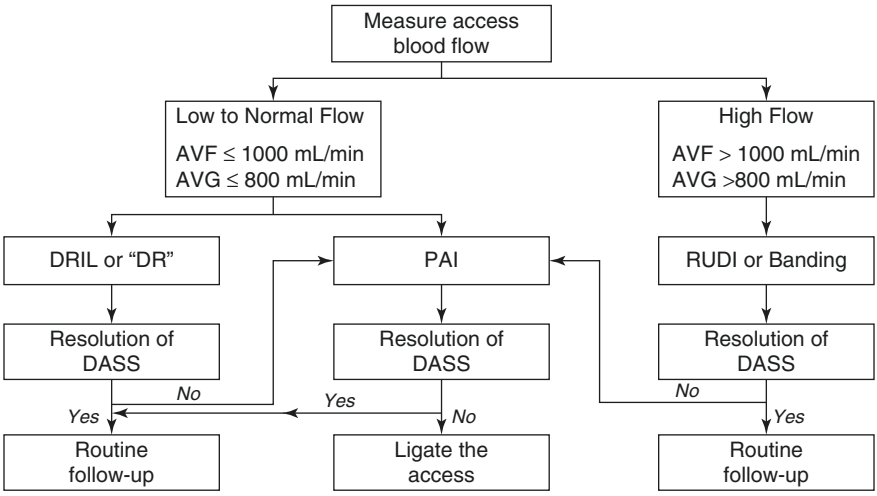

Fig. 18.10 Algorithm for the treatment of HAIDI in upper arm based on access blood flow

Primary arterial disease is a major contributor to hand ischemia in a significant number of these cases, especially those at the low end of the spectrum for blood flow. In some cases, the low blood flow access may actually be marginal and not salvageable.

Proximalization of the Arterial Inflow (PAI)

PAI is the procedure of choice for this category. This procedure is performed by closing the original arterial anastomosis and moving the inflow of the AV access to a more proximal level on the feeding artery by using expanded ePTFE as an interposition graft [52, 88] (Fig. 18.11). In a study which used a silicon model to simulate the hemodynamics of an AVF, it was demonstrated that the more proximally an arteriovenous anastomosis is located, the higher the distal arterial pressure at any given fistula blood flow [89]. This relationship serves as the basis for the PAI procedure. In addition, because of the more proximal anastomosis, collateral arterial flow is increased, which also improves distal blood pressure and flow to the hand. Many surgeons prefer the PAI procedure since it is less invasive than the alternative procedure—distal revascularization-interval ligation (DRIL) (see below) [52, 64, 69, 90].

In a review [91] of three articles (total of 57 cases) reporting the results of PAI, the pooled rate of symptomatic relief was 98.2% (range 91% to 100%) and early thrombosis of the vascular access was 7% (range 2% to 17%). An alternative to the PAI and DRIL procedures is the "DR" portion of the DRIL procedure (see below) the end result of which is in essence very similar to the PAI.

Distal Revascularization with Interval Ligation (DRIL)

The DRIL procedure [77] (Fig. 18.12), was the first surgical procedure described for the treatment of HAIDI both to be based upon an understanding of pathophysiology and to predictably accomplish the dual goals of correction of hand hypoperfusion and preservation of the dialysis vascular access. Because of its success, it rapidly became the standard for treatment of all HAIDI patients regardless of blood flow level.

The DRIL procedure consists of (1) the creation of an arterial bypass from the artery proximal to the anastomosis to the artery distal to the ligation (DR) using either expanded polytetrafluoroethylene or a reversed saphenous vein and, (2) ligation of the artery just distal to the anastomosis (IL). Conceptually, the DRIL

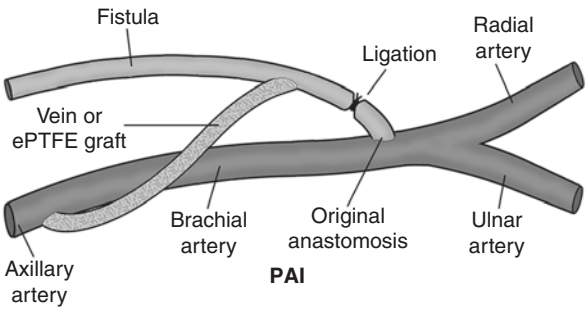

Fig. 18.11 Proximalization of the arterial inflow (PAI)

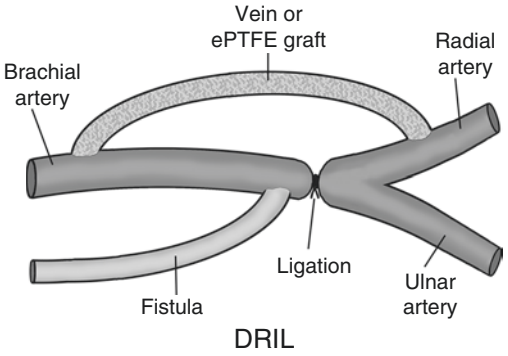

Fig. 18.12 Distal revascularization-interval ligation (DRIL)

procedure provides an added low-resistance collateral artery which reduces the total peripheral resistance in the distal extremity. This serves to increase the total peripheral perfusion to the hand. It does this while simultaneously blocking retrograde flow to the access.

In a review [91] of 15 articles dealing with the DRIL procedure dating from 1997 to 2013, data on 590 cases were presented. The pooled rate of symptomatic relief in these cases was 83.7% (range 80% to 87%), early thrombosis of the vascular access occurred in 0.9% (range 0.3% to 2.3%), and bypass graft primary patency was 86.3% (83% to 89%). It should be pointed out that many of these cases had high-volume blood flow rather than low to normal levels. This exerts a definite effect on the results obtained making it impossible to compare with the currently recommended low to normal blood flow indication.

Although initially considered to be the standard for treatment of HAIDI, in recent years the complete DRIL procedure has been used less often. Ligation of the major artery to the forearm, the DL step of the procedure, is something that many surgeons are reluctant to do. In addition, with the increasing age of the dialysis population, patients presenting with HAIDI are more likely to be elderly with advanced peripheral arterial disease, making distal revascularization more difficult and sometimes impossible to perform. DRIL is a complex multi-step procedure which often includes harvesting the saphenous vein for the bypass. Because of this complexity, general anesthesia is needed, which increases the risk for patients with a large comorbidity burden [92]. Because of these issues and the fact that the hand-perfusion advantage offered by this procedure is not great, some surgeons prefer to do only the DR (distal revascularization, Fig. 18.13) portion of the procedure [93, 94]. The result of this modification makes the procedure similar to the PAI procedure as described above.

High Access Blood Flow

Blood flow rates well above the accepted normal range are common with AVFs and also occur with some AVGs. It is not unusual with an upper arm AVF to see blood flow rates in excess of 2000 mL/min. It has been estimated that about two-thirds of the HAIDI cases that present for treatment have high access blood flow. Patients

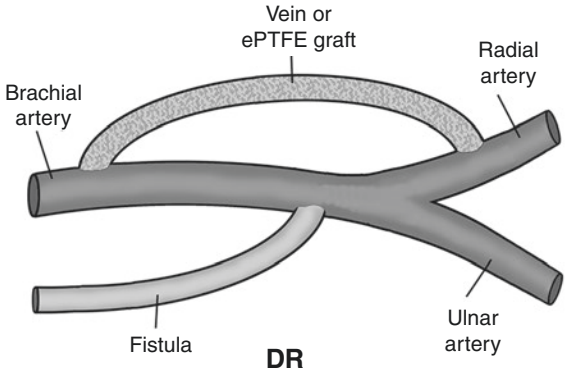

Fig. 18.13 Distal revascularization only (DR)

with high blood flow-associated HAIDI generally have later onset of ischemic symptoms in contrast to those with normal flow [32, 53, 88, 95]. In these patients, HAIDI is the result of the increasing AVF blood flow that occurs over time (increasing the demand) in combination with arterial disease which is also generally progressive (decreasing the supply) [11, 18].

In high blood flow HAIDI cases, a flow-reduction procedure is indicated. Upper arm AVFs have a significantly higher flow than forearm AVFs, due to a larger artery and vein at the time of surgical construction and with higher pressure and blood flow into the access during maturation. Flow reduction in upper arm AVFs to a level comparable to that of a normal radial-cephalic AVF has been shown to result in a significant increase in distal extremity pressures to near normal values [88].

There are two approaches to flow reduction—(1) precision banding or its equivalent, either surgical or endovascular, and (2) the revascularization using distal inflow (RUDI). As in cases with low to normal access blood flow, the treatment goal is to reduce access blood flow while preserving the access for dialysis. In addition to the risk of development of HAIDI, high blood flow rates in the dialysis access also place the patient at risk for serious cardiac problems such as progressive left ventricular hypertrophy and high-output cardiac failure. For this reason, flow reduction can have multiple benefits [88].

Precision Banding

Access banding creates a narrowed zone within the access, preferably close to or at the AV anastomosis. The goal of the procedure is to reduce access flow and thereby obtain an increase in both distal arterial blood pressure and flow [88, 89, 96, 97]. It can be performed successfully only in those patients with high flow-associated HAIDI. Reducing flow in a low blood flow access to the degree necessary to alleviate ischemic symptoms can predictably result in access thrombosis.

Historically, banding was done blindly without using any objective parameter to guide the degree of lumen reduction or change in access blood flow [20, 51, 53]. As a result a high incidence of access thrombosis was observed [98]. More recently techniques for controlling the degree of flow reduction achieved have been used to accomplish what has been referred to as “precision-banding” [6].

Precision-banding refers to banding performed in conjunction with some technique for controlling the degree of lumen reduction or change in access blood flow. To assure success, accurate intra-operative flow measurements to judge the degree of access flow restriction should be considered mandatory [88]. In one report, significant changes in access blood flow were noted with only 0.5 mm variations in lumen diameter achieved with banding [99]. When blood flow has been reduced to levels no lower than 500–600 ml/min for AVFs and 600–700 ml/min for AVGs, post-banding access patency rates have been much better [88, 100].

Precision banding can be performed using either a surgical or an endovascular approach. Because it is a less invasive approach, endovascular banding has become the preferred technique. In cases in which banding fails to relieve the ischemic symptoms, a PAI procedure is recommended [90].

Endovascular banding—Endovascular precision-banding is achieved by using some type of precise sizing dowel placed either intraluminally or extraluminally to assure that a controlled degree of luminal restriction has been achieved. This should be accompanied by ultrasound access blood flow measurement immediately post-banding to assess the effect and adjust the degree of banding using objective blood flow criteria. Endovascular banding can be accomplished using either an intraluminal approach (angioplasty balloon or vascular dilator) [61, 101] or an extraluminal approach (vascular dilator) [99, 101] (Fig. 18.14). Endovascular banding has the advantage of being minimally invasive, only a small skin incision is required, the lumen of the access can be reduced to a defined size, and the procedure can be easily reversed simply by using an angioplasty balloon.

Surgical banding: Surgical banding, better stated as surgical flow reduction, can be performed by a variety of techniques including using a ligature, titanium clip, prosthetic cuff, suture plication of the conduit, adding an interposition graft segment of smaller diameter, or revising the arterial anastomosis to a smaller diameter [20, 51, 53, 88, 95].

Regardless of the technique used, precision banding should be performed in conjunction with intraoperative measurement of vascular access blood flow, digital blood pressure or both [88]. The goal should be to reduce blood flow to

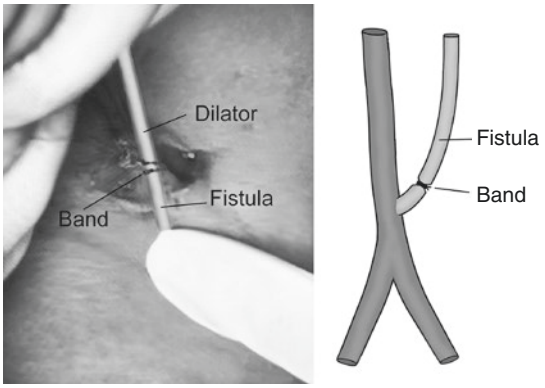

Fig. 18.14 Extraluminal precision banding using a vascular dilator

approximately 500–600 mL/min in AVFs and 600–700 mL/min in AVG's [88, 100]. The goal for digital blood pressure should be equal to or greater than 50 mmHg or a DBI equal to or greater than 0.6 [53]. In addition to these objective measures, it is important to assess improvement in the patient's signs and symptoms of ischemia intraoperatively as soon as the procedure is completed in order to confirm that it has been effective. Ideally, flow reduction surgery should be performed under local with monitored anesthesia care [6]. This permits the necessary assessment of the patient's symptoms.

Revision Using Distal Inflow (RUDI)

The RUDI procedure is accomplished by closing the original arteriovenous anastomosis with the distal brachial artery at the elbow and revising the arterial inflow to the more distal proximal radial or ulnar artery. This can be accomplished either by direct anastomosis of the outflow vein to the new inflow artery or with an interposition graft (saphenous vein or ePTFE) (Fig. 18.15). In the initial report [102], the RUDI procedure was performed 4 patients with resolution of symptoms in all cases; however, access blood flow measurements were not reported. In a study [103] involving 29 RUDI procedures, high access blood flow was the surgical indication in all instances, with HAIDI present in 19 cases. Preoperative mean brachial artery blood flow rates were 2181 mL/min, and postoperative they were 990 mL/min for a mean reduction of 55%. HAIDI symptoms resolved completely in 69% of patients with an additional 31% having partial resolution. At 1-year primary assisted patency was 74%, and cumulative patency was 87%.

The RUDI procedure should be considered as an alternative to banding in high blood flow associated HAIDI. It should be used only if the forearm artery not used for the procedure (distal inflow) is demonstrated to be patent, otherwise it creates a high risk of persisting hand ischemia. The major disadvantages to the RUDI procedure are that it is more invasive than most banding procedures, and the resulting flow reduction is not predictable and cannot be tailored to the individual patient's requirements [104].

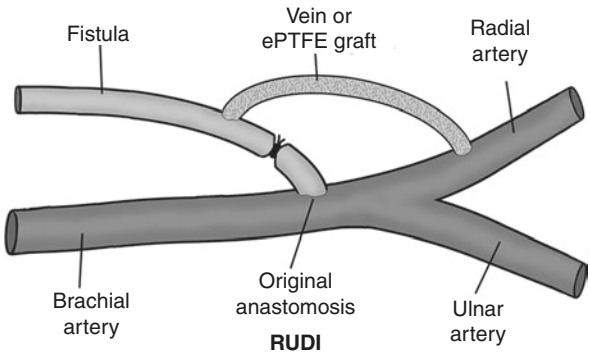

Fig. 18.15 Revision using distal inflow (RUDI)

Last Resort

As previously emphasized, the goal of HAIDI treatment is two-fold, relieving ischemia while preserving the access. It is not always possible to accomplish both. When this is the case, the access must be sacrificed in order to minimize tissue loss and disability. Access ligation is imperative when other corrective procedures are not suitable or have failed. Provision of a means for continuing dialysis is necessary in most cases. Many patients who arrive at this point have a very poor prognosis and a short life expectancy. If dialysis is to continue in these cases, the insertion of a tunnel dialysis catheter would be the most appropriate solution. In cases with a better prognosis, it may be possible to create a new AV access. Before such an event, a thorough evaluation and detailed planning are important to minimize the risk of recurrent hand ischemia. Peritoneal dialysis should also be considered.

Ischemic Monomelic Neuropathy

Ischemic monomelic neuropathy (IMN) is a distinct clinical entity associated with interference of blood flow in a major limb artery resulting in a sudden diversion of nerve blood supply leading to ischemic injury of the neural tissue [105]. Symptom onset is immediate and neurologic symptoms are dominant in the absence of significant clinical ischemia of the hand. Typically, the hand is warm, capillary refill is preserved and a palpable radial or ulnar pulse, or audible Doppler signal is present [106, 107].

Incidence

The true incidence of IMN following hemodialysis access surgery has not been defined. Based upon reports in the literature it is uncommon, and some say it is rare. However, it is likely that because of the broad spectrum of signs and symptoms that may be present, the condition is underdiagnosed and underreported. In addition, in the past some surgeons have not distinguished this complication from HAIDI [78]. Nevertheless, in the literature the incidence of IMN has ranged from 0.3% to 4.4% [108, 109].

IMN is seen primarily in association with arteriovenous accesses that are brachial artery-based [107], although an association with the femoral artery in the case of a lower extremity AVG has been reported [109]. The condition is seen most often in association with the creation of an AVG [3], and there are no reports of IMN precipitated by a distal forearm procedure. Although there are case reports to the contrary [110], the condition occurs almost exclusively in diabetic hemodialysis patients [95, 111], particularly older ones with pre-existing peripheral neuropathy and/or peripheral vascular disease. IMN also occurs more frequently in women than men.

Clinical Features

The syndrome develops quickly, typically within minutes to hours of AVF creation, although, the patient's symptoms, which may be severe, are frequently not recognized for several hours because of sedation or the time required for a regional block to dissipate.

Clinically, IMN presents a spectrum of signs and symptoms ranging from mild to devastating. IMN is a polyneuropathy, and involvement of a single upper limb nerve should bring the diagnosis into serious question [107]. The pathognomic feature of ischemic monomelic neuropathy is the presence of diffuse neurologic dysfunction, usually in the absence of significant ischemic changes in the tissues of the hand and fingers. The symptoms of the neuropathy include pain, paresthesias and numbness in the distribution of all three forearm nerves along with diffuse motor weakness or paralysis. The sensory component, typically a persistent neuropathic burning pain, is generally more prominent than the motor component. The hand is generally warm and often a palpable radial artery pulse or audible radial artery Doppler signal is present [112].

Because the forearm muscles may be relatively spared compared to the intrinsic hand musculature, complete paralysis of the intrinsic hand muscles may be present with less severe weakness of wrist flexion and extension. This may result in a picture that resembles a mono-neuropathy (Fig. 18.16), although a careful neurological examination will confirm that all three nerves are involved. Poor wrist extension (radial nerve), poor function of the intrinsic hand musculature (ulnar nerve) and poor thumb opposition (median nerve) are typically present to some degree.

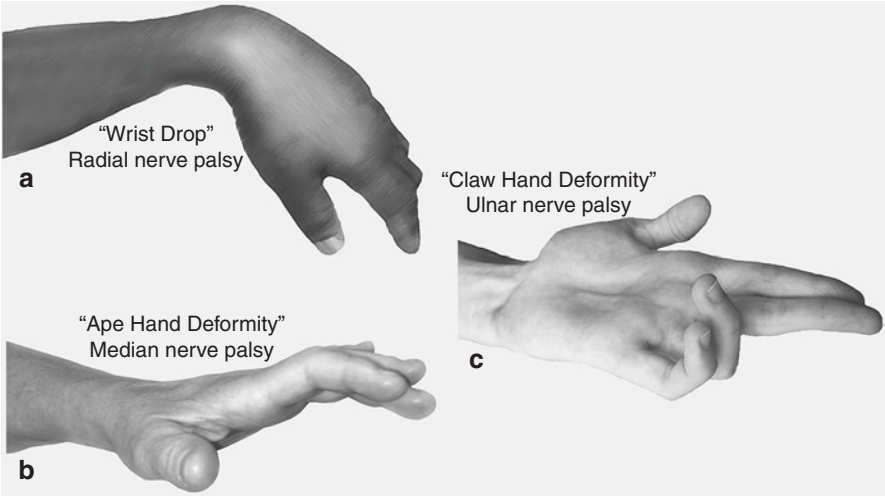

Fig. 18.16 Types of nerve palsy affecting distal upper extremity

Pathogenesis

IMN is a condition which, on its surface, appears to be unique; an ischemic injury occurs that is selective for one tissue (nerve), but spares other tissues (muscle, skin). The pathogenesis of this phenomenon as it relates to IMN is dependent upon two factors—the characteristics of the microcirculation of the nerve and the comparative metabolic needs of the tissues involved (relative rate of oxygen consumption).

Large peripheral nerves have dedicated intraneural blood vessels, the *vasa nervorum*, while the metabolic needs of small nerves are met by diffusion from surrounding tissues [113]. The *vasa nervorum* is composed of nutrient arteries which are variable in their site of origin and have considerable overlap in their distribution. These vessels give rise to an extensive, intercommunicating, anastomotic network organized along the epineurial, perineurial and endoneurial tissue planes of the nerve. This extensive anastomotic network, together with the overlap in the distribution of the nutrient arteries, assures that there are collateral circulatory mechanisms available to provide an uninterrupted perfusion of the nerve in the event that one of the nutrient vessels is interrupted.

Although extensive, the anatomical distribution of this microcirculation does have "watershed zones." These are areas that lie between adjacent nutrient arteries along the length of the nerve where there is very little overlap in the capillary plexuses (Fig. 18.17). The presence of such a zone can result in a proximal band of ischemic nerve injury where blood flow in the nutrient artery is compromised due to temporary major vessel occlusion, with good filling distal and proximal to it, distal perfusion being maintained by collateral arteries. The existence of such an event was shown in postmortem injection studies [114, 115], where an area of non-filling of the intraneural blood vessels in devascularized nerves was demonstrated, with filling of vessels distal to that area.

Based upon these studies it is postulated that this scenario occurs in IMN [116]. IMN reflects ischemic nerve damage occurring as a flash injury when the brachial artery is either occluded during the creation of an access or from a mechanism similar to HAIDI. In other words, it is an unusual variant of HAIDI. The antecubital area has been shown to be a watershed zone for the *vasae nervorum* perfusing the three

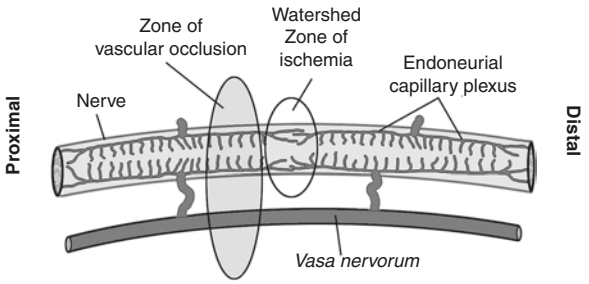

Fig. 18.17
Pathogenesis of IMN

nerves that supply the lower arm [117]. With occlusion of the brachial artery at the time of AVF creation, the area of poorest perfusion (and thus maximal damage) is a band of nutrient vessels to the nerves in this watershed zone. Thus, selective nerve damage occurs. Sensory nerve fibers are more sensitive to ischemic insult than are motor fibers. As a result, sensory symptoms tend to be more prominent than motor.

Diagnosis

IMN is a clinical diagnosis—signs and symptoms of significant nerve deficit occurring immediately after AV access surgery that are disproportionate to any ischemic change observed in other tissues of the affected extremity. In most instances, this clinical picture is all that is required. If the clinical picture is equivocal, electromyography and nerve conduction studies can be used to confirm the diagnosis. These studies typically show axonal loss, low amplitude or absent responses to sensory and motor nerve stimulation, and relatively preserved nerve conduction velocities.

Although prompt recognition is important, diagnosis is often delayed. Initially, delay is due to the time required for sedation or anesthesia to dissipate. Due to soft tissue swelling or a hematoma causing nerve compression, hand or digital numbness and tingling can occur following AV access surgery, confusing the picture [56]. Additionally, the presence of severe diabetic neuropathy may complicate evaluation.

The primary issue in the diagnosis of IMN is a high level of suspicion. Often, IMN is not suspected when a problem arises post operatively because it is an uncommon occurrence. Any patient complaining of neurological symptoms following AV access creation should have an immediate neurologic evaluation to facilitate early diagnosis and management. It is also recommended that all diabetic patients, especially those who are older and have pre-existing peripheral neuropathy and/or peripheral vascular disease be routinely evaluated immediately after access surgery for evidence of IMN. Severe pain in the hand following a brachial artery-based dialysis access procedure should immediately raise the suspicion of IMN.

Differential Diagnosis

The differential diagnosis in these cases includes acute HAIDI, surgical injury and carpal tunnel syndrome. Severe hand pain in the absence of ischemic changes to the tissues of the hand and fingers, along with the usual warm hand, should rule out the possibility of acute HAIDI. Weakness of the hand and digits further suggests IMN. Involvement of a single upper limb nerve in the setting of vascular access surgery should exclude the diagnosis of IMN and prompt a search for local nerve compression secondary to a complication of the surgery [57, 107]. Occasionally, carpal tunnel syndrome can be unmasked or exacerbated by access surgery. This is thought to be secondary to edema and venous hypertension in the area of the flexor retinaculum associated with the procedure [58, 59].

Management

Early recognition of IMN is crucial. Both the NKF-KDOQI Practice Guidelines [118] and the practice guidelines of the Society for Vascular Surgery [119], recommend closure of the AV access as soon as a diagnosis of IMN is made. Early recognition and rapid AV access ligation have been reported to result in some recovery of motor function and decrease in residual sensory symptoms in the affected hand [108, 110, 120]. Unfortunately, this is not the rule, and in most cases the neuropathy is permanent or only partially reversible [78, 121, 122]. This being the case, the recommendation for AV access closure is based more on medical-legal concerns than purely medical ones.

The decision to close a functioning access requires careful deliberation. Because of the medical profile of the patient likely to develop IMN, many cases may have few or no alternative sites for future hemodialysis access construction. In addition, this complication can recur at other sites, given that the underlying risk factors are unchanged. There are reports, mostly anecdotal, of cases of IMN in which the access was not closed [123–125]. In one report involving two patients [123], the AV access was used successfully for dialysis, and rehabilitation therapy resulted in significant improvement in muscle strength early but with persistence of paresthesias. After 11 months of follow-up, the improved hand weakness was stable and the paresthesias had disappeared.

It is not clear which IMN cases will benefit from rehabilitation therapy rather than AV access closure. The signs and symptoms associated with this disorder represent a spectrum ranging from mild to devastating. As is the case with HAIDI, identifying where an individual patient lies within the spectrum might offer an opportunity for differences in approach to therapy. In the reported cases which have responded to rehabilitation therapy, symptoms were not severe [123–125].

Long-term outcome of IMN patients with more severe symptoms has not been well documented. In most instances, the motor deficits result in some degree of permanent hand disability. Long-standing pain resulting from IMN has been reported [121].

Subclavian Steal Syndrome

As described above, the AV access exerts a significant hemodynamic effect on the peripheral vascular bed associated with the feeding artery. In the process of shunting a large volume of blood from the arterial to the venous circulation, the access can "steal" blood from the peripheral vessel bed. This steal phenomenon not only affects the vessels supplying the hand but has also been described involving the subclavian artery. There are two different syndromes which generally occur separately but can occur together: (1) subclavian- vertebral artery steal, and (2) subclavian-coronary artery steal, when the internal thoracic artery has been used for coronary artery revascularization.

Most cases of subclavian steal are asymptomatic [126], but this is not always the case, and symptomatic cases have been reported. For this reason, physicians caring for hemodialysis patients should be aware of this potential problem.

Subclavian-Coronary Artery Steal Syndrome

The need for coronary artery bypass surgery (CABG) in the dialysis patient is not unusual, reflecting the high incidence of cardiovascular disease in this patient population. The use of the internal thoracic artery to create a bypass to the left anterior descending coronary artery for this procedure has been found to have definite advantages [127]. However, given the right circumstances, subclavian-coronary artery steal characterized by a reversal of blood flow in the internal thoracic artery graft, stealing blood from the coronary arteries, can occur.

Subclavian-coronary artery steal can cause symptoms of myocardial ischemia. In these patients, cardiac hypokinesia, dyspnea, ischemic electrocardiogram changes, and angina, especially aggravated during hemodialysis, is typically seen. In addition, myocardial infarction and decreased long-term survival have been reported [128–133]. However, only rarely are cases symptomatic. Cardiac symptoms have been reported to occur in as many as 4.5% of patients following coronary bypass surgery utilizing the internal thoracic artery [134]. Available evidence suggests that in asymptomatic cases, the presence of the subclavian- coronary artery steal phenomenon does not affect long-term survival [135].

Subclavian-Vertebral Artery Steal Syndrome

The clinical manifestations of symptomatic subclavian-vertebral artery steal are neurologic and are due to reversal of blood flow in the vertebral artery, stealing blood from the vertebrobasilar region of the brain [136–139]. These features were illustrated by the first case to be reported [136]. A long-term hemodialysis-dependent patient presented with a 3-month history of transient episodes of ataxia, discoordination, vertigo, presyncope, and a tendency to fall toward his left side, occurring 2 to 3 times per week. Each episode lasted 15 to 20 minutes with spontaneous resolution. The episodes were precipitated by using his left arm, most consistently while taking a warm shower. Blood flow volume in his left arm brachial-cephalic AVF was 5.8 L/min. The subclavian artery was free of stenosis. Retrograde flow was demonstrated in the ipsilateral vertebral artery which reverted to antegrade with manual compression of the AVF. A surgical flow reduction procedure was performed reducing access blood flow to 1.9 L/min. Blood pressure in the subclavian artery measured at the time of surgery was 80/50 mm Hg (mean 68). With AVF occlusion, it was elevated to a level of 163/103 mm Hg (mean 126). Blood flow in the left vertebral artery became antegrade following surgery, and the patient's symptomatic episodes resolved.

Pathogenesis

Two factors have been found to predispose to the development of subclavian steal: (1) subclavian artery stenosis and (2) a high blood flow vascular access. The most common cause for subclavian steal is ipsilateral subclavian artery stenosis developing proximal to the origin of the internal thoracic artery [139–142]. The exact prevalence of this lesion is not known; however, an incidence in the range of 0.2% to 6.8% in patients having a CABG has been reported [132]. With proximal subclavian stenosis, there is a marked pressure drop in the artery distal to the point of narrowing. The fact that there are other arteries not affected by this decreased pressure feeding into the peripheral vascular beds supplied by the arteries proximal to the point of stenosis makes asymptomatic retrograde flow in these vessels possible (Fig. 18.18).

In the case of the vertebral artery, the low peripheral resistance of the cerebral circulation along with the communicating arterial circuits derived from the contralateral vertebral and the carotid arteries facilitates retrograde flow in the affected vessel while providing adequate perfusion to the cerebral tissue, preventing ischemic changes. The vascular bed derived from the coronary arteries provides the same function in the case of subclavian-coronary artery steal. This suggests that the symptomatic cases represent a failure in this compensatory mechanism due to vascular disease.

Increased blood flow in the subclavian artery due to high-flow upper extremity AV access can also induce subclavian steal [128–133]. Normally, blood flow in the vertebral or the internal thoracic artery grafted to the coronary artery is antegrade during both systole and diastole. High blood flow velocity in the subclavian artery associated with an ipsilateral AV access can result in a Venturi effect creating retrograde flow in the connecting artery (Fig. 18.19).

Management

Most cases of subclavian steal are asymptomatic and generally go unrecognized. Therefore, if discovered, treatment is rarely warranted. Symptomatic cases have

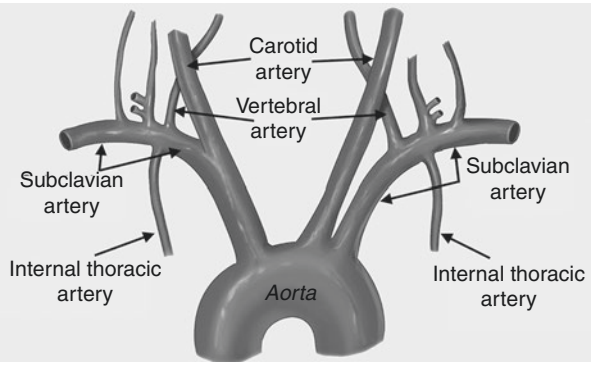

Fig. 18.18 Vessels of aortic arch showing the relationship of vertebral an internal thoracic artery to subclavian artery

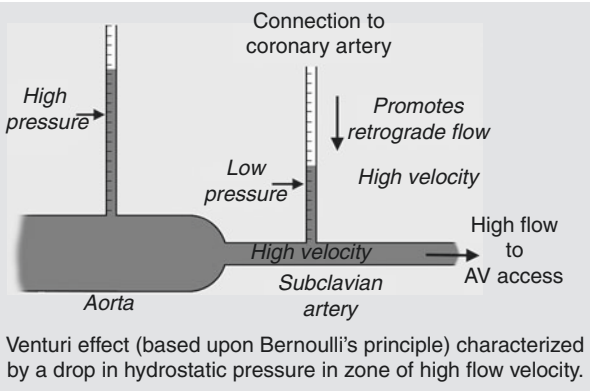

Fig. 18.19 Venturi effect resulting in retrograde flow in vessel connected to subclavian artery

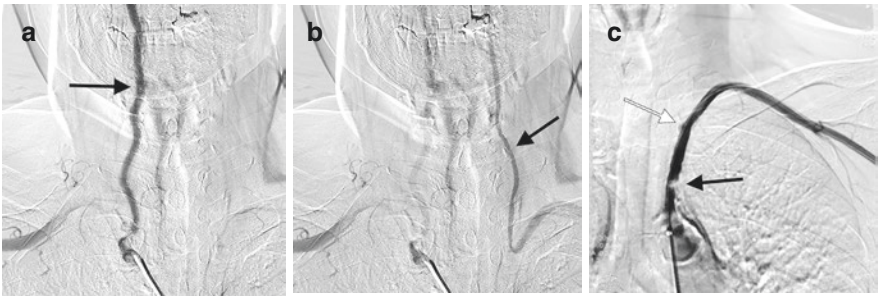

Fig. 18.20 Subclavian-vertebral artery steal syndrome. (**a**) right carotid artery (arrow) at beginning of radiocontrast injection, (**b**) vertebral artery with retrograde blood flow (arrow) late after radiocontrast injection, (**c**) radiocontrast injection of left subclavian artery showing lesion (black arrow) central to origin of vertebral artery (white arrow). (Case courtesy of Prof. Lihong Zhang, First Hospital of Hebei Medical University, Shijiazhuang City, Hebei Province, China)

been successfully managed with interventional treatment. Angioplasty of subclavian stenosis (generally with stent placement) has been shown to be beneficial in those cases where this has been the predisposing factor [132, 142] (Fig. 18.20). In cases associated with a high blood flow AV access, flow reduction has been found to be effective in some reported cases [136]. In other reports, management has consisted of closure of the access [133].

Some investigators have recommended that when a hemodialysis patient is referred for a CABG, the internal thoracic artery contralateral to an upper arm AV access should be used. In addition, if a patient has previously had a CABG, the arm contralateral to the internal thoracic artery used for coronary revascularization should be selected for the creation of an AV access [129]. However, other investigators have felt that this degree of caution is not necessary [135]. In view of the infrequency of symptoms, it is probably enough to be aware that these syndromes related to subclavian steal can occur, recognize them when they present, and treat them appropriately at that time.

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Role of Drug-Eluting Balloons in Dialysis Access Interventions

19

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Introduction

Balloon angioplasty is a well-established treatment option in the maintenance of dialysis accesses. Angioplasty is indicated for stenotic lesions greater than 50% with successful treatment represented by less than 30% residual stenosis. Recently, DCBs, which were first applied to diseased peripheral arteries, have been employed in treating lesions in dialysis accesses with the objective to prolong the time between interventions.

DCBs are more complex than standard angioplasty balloons. The concept of DCBs is to deliver medication to the treated lesion that will help prevent restenosis from endothelial proliferation. The key components of DCBs are therefore the drug and the excipient.

Paclitaxel has emerged as the primary drug on DCBs. Paclitaxel's capacity to prevent endothelial hyperplasia in vessels is derived from the drug's ability to halt endothelial proliferation. Prior studies have shown that it can inhibit smooth muscle and fibroblast proliferation and migration, both believed to play key roles in the development of neointimal hyperplasia [1]. On a cellular level, paclitaxel activates the mitotic checkpoint which causes a treated cell to experience mitotic arrest. In this phase of mitosis, the mitotic checkpoint delays separation of the chromosomes [2]. Due to its ability to inhibit cell growth, the use of paclitaxel in treating certain malignancies (ovarian, breast, lung cancer as well as Kaposi's sarcoma) has been

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well documented. One of paclitaxel's advantages in vascular treatment is its high lipophilic properties which promotes rapid cellular uptake and increased dwell times in treated vessels [3].

The efficacy of paclitaxel in preventing recurrent stenosis and neointimal hyperplasia in peripheral arterial disease has been studied in several large randomized controlled trials. For the first time in 2012, Katsanos et al. demonstrated the benefit of employing DCBs for treating stenotic venous lesions in arteriovenous fistulas and grafts in maintaining primary patency in a small single-center randomized controlled trial [4]. In the randomized study group of 40 patients, the cumulative target lesion primary patency at 6 months was significantly higher after DCB application (70% in DCB group vs. 25% in simple balloon angioplasty (BA) group). Subsequently in 2015, Kitrou et al. published 1-year data that reported Target Lesion Revascularization (TLR) – free survival of 308 days in the DCB group compared to 161 days for the balloon angioplasty (BA) group [5]. Also, the access circuit primary patency was 270 days for DCB group compared to 161 days for the BA group. Since then, several small single-center studies have evaluated DCBs for the treatment of failing dialysis access [6, 7].

It should be noted that in June of 2019, the U.S. Food and Drug Administration (FDA) addressed the issue of potential late mortality after treatment with paclitaxel-coated devices (balloons and stents) in treating peripheral artery disease (PAD) in the femoropopliteal arteries. While this has been suggested by various studies, it should be accepted with caution as additional data to support or refute this claim is needed. Further discussion of this topic is beyond the scope of this chapter in addition to the lack of sufficient evidence at the time of publication.

DCB paclitaxel concentrations range from 2-μg/mm^2 to 3.5-μg/mm^2. It is important to note that while many DCBs exist that are approved for use in Europe, currently only three DCBs have FDA approval. These include Lutonix (BARD, Murray Hill, NJ, USA), IN.PACT DCB platform (Medtronic Vascular, Santa Clara, CA, USA) and Stellarex DCB (Spectranetics, Colorado Springs, CO, USA).

Recently, several new balloons employing sirolimus have been introduced to the market and have been granted breakthrough device designation by the FDA, however, as there is limited data of its efficacy in human trials, discussion of these balloons will be deferred when more data is available.

Excipients, or drug carriers, play key roles in the transfer of the drug to the vessel wall. Excipients act to increase drug solubility, drug transfer and drug uptake. In addition, excipients act to increase sustained paclitaxel reservoirs in the tissue with the goal of prolonged drug effects. A variety of excipients exist, however, the excipients that are currently found on the three FDA approved DCBs are polysorbate/sorbitol (Lutonix), urea (IN.PACT), and polyethylene glycol (Stellarex).

Currently, the three FDA approved DCBs are available in either 0.018″ (Lutonix DCB) and 0.035″ (Lutonix DCB, IN.PACT AV DCB and Stellarex DCB) over-the-wire (OTW) platforms.

Equipment

Lutonix DCB

The Lutonix DCB is an OTW percutaneous transluminal angioplasty (PTA) balloon with paclitaxel coating currently available in the market for both peripheral arterial disease and dialysis access applications [8]. The device was initially approved by the FDA in 2014 for lower extremity peripheral arterial disease (more specifically femoropopliteal arterial disease) and eventually gained FDA premarket approval for use in the treatment of stenotic lesions in native arteriovenous fistulas in 2017.

Indication for Use

The indications for application of the Lutonix DCB initially included PTA of de novo or recurrent stenosis of femoral and popliteal arteries (lesion length up to 150 mm and reference vessel diameter of 4–6 mm) after pre-dilation and vessel preparation. After premarket approval for dialysis work, this was extended to stenotic lesions in the native arteriovenous fistulas with lesion length up to 80 mm and index vessel diameter of 4–12 mm after pre-dilation [9].

Contraindications

The Lutonix DCB is contraindicated for use in the following anatomy and patient types:

- Patients who are unable to tolerate the recommended post-treatment antiplatelet therapy according to the manufacturer's Instructions for Use (IFU) and standard clinical practice [10].
- Women who are breastfeeding, pregnant, or are intending to become pregnant, or men intending to father children over the next 2 years.
- Lesions that do not allow for complete inflation of the balloon or lesions in locations that would not permit the placement of an appropriate delivery system.

Additionally, the safety of the device among pediatric population is unclear.

Device Handling and Application

According to the manufacturer's IFU, a paclitaxel coating is evenly distributed over the Lutonix DCB in a non-polymer form at a concentration of 2 μg/mm^2. The excipients used as the carrier for paclitaxel in this device are polysorbate and sorbitol [10].

Before using the DCB, the target vessel and lesion must be prepared and predilated since the Lutonix DCB is a semi-compliant low-pressure balloon and might not be able to dilate tight strictures. While vessel preparation for femoral and popliteal arterial disease may be complex and require advanced techniques such as atherectomy, arteriovenous fistula lesion preparation includes crossing the lesion using

the physician's wire and catheter of choice and predilating it with a high-pressure balloon with the goal of a residual stenosis less than 30%. The Lutonix DCB can then be applied to the target lesion following successful and non-complicated vessel preparation defined by a lack of dissection or rupture, a lack of residual hemodynamically significant stenosis (less than 30%), and complete effacement of the waist with the high-pressure balloon [11].

Prior to use, the balloon segment of the Lutonix DCB should not be immersed in water or touched with wet gloves or gauze. The manufacturer recommends against removing the metal stylet or protective cover until lesion preparation is achieved and the DCB is ready to be advanced over the wire to reach the target lesion. The balloon should not be inflated outside of the body or prior to reaching the target lesion. Advancing the balloon over the wire and inflating it across the target lesion for drug delivery should be fast, and preferably take less than 30 seconds [10]. If complete balloon expansion exceeds 3 minutes, the manufacturer recommends using a new balloon. The balloon should be inflated appropriately to ensure maximum wall opposition and remain inflated for 2 minutes to ensure adequate drug delivery. After treating a lesion with the Lutonix DCB, a course of at least 4 weeks of dual antiplatelet therapy is recommended.

Prior Studies

In 2016, a single-center retrospective study provided initial safety and effectiveness data for the Lutonix DCB in 39 failing fistulas and grafts [12]. The study reported an overall target lesion primary patency of 72% at 6 months and a median primary patency of 260 days. The study did not show a difference in patency of de novo versus restenotic lesions as well as fistulas versus grafts. Interestingly, in patients who required two sessions of angioplasty using DCBs, statistically significant improved patency was observed after the second treatment (180 versus 270 days, p value = 0.032) [12]. This was followed by a small single-center randomized controlled trial by the same group evaluating the efficacy of the Lutonix DCB in treating symptomatic central venous stenosis in dialysis accesses [13]. Overall, 40 patients with symptomatic central venous stenosis (de novo or recurrent) were randomized 1:1 to either balloon angioplasty or balloon angioplasty plus Lutonix DCB. The study did not show any difference between grafts and fistulas or de novo versus restenotic lesions.

More recently, the initial results of the Lutonix trial was published in the Clinical Journal of the American Society of Nephrology [11]. The study was a multicenter, prospective, randomized clinical trial, which was performed under an investigational device exemption from the FDA. The study included patients with upper extremity matured native arteriovenous fistulas actively being used and created more than 30 days prior to the intervention. Included stenotic lesions could be anywhere from the arteriovenous anastomosis to the axillary and subclavian vein junction with more than 50% stenosis, less than 10 cm length and reference vessel diameter of 4–12 mm. Patients were randomized 1:1 to Lutonix DCB plus angioplasty or angioplasty alone. Patients were followed clinically by telephone call and reintervention to treat stenotic lesions other than the primary lesion was allowed

without terminating the primary patency of the primary lesion. The study did not meet its primary endpoint, which was the primary patency of the target lesion at 6 months (71 ± 4% for Lutonix versus 63 ± 4% for angioplasty alone, p value = 0.06), however, there was a significant difference in primary patency at 210 days (64 ± 4% for Lutonix versus 53 ± 4% for angioplasty alone, p value = 0.03). There was also no difference in the circuit primary patency between groups at 6 months (62 ± 4% for Lutonix versus 58 ± 4% for angioplasty alone, p value = 0.25). The primary safety endpoint of the study which was freedom from localized or systemic major adverse events within 30 days from the index intervention did not show any difference between groups.

In conclusion, the existing data [11–13] shows a signal towards improved target lesion primary patency following treatment with angioplasty and Lutonix DCB versus angioplasty alone both in the arteriovenous fistula circuit and central veins with index vessel diameter of 4–12 mm. Although the Lutonix trial did not show any improved overall primary patency of the circuit at 6 months, further results including the long term follow up from the Lutonix trial out to 24 months and cost-effectiveness analysis would be very helpful in shedding light over the potential benefit of DCBs such as Lutonix for the treatment of failing dialysis accesses.

IN.PACT AV DCB

The IN.PACT arteriovenous paclitaxel-coated percutaneous transluminal angioplasty balloon catheter, hereafter referred to as the IN.PACT AV DCB, is an OTW balloon catheter with a DCB at the distal tip. The IN.PACT AV DCB leverages technology from the IN.PACT Admiral platform, which has been successfully used for treatment of femoropopliteal arterial disease. According to the manufacturer's data, the drug component, referred to as FreePac™ coating, consists of the drug paclitaxel and the excipient urea. The balloon surface has a nominal paclitaxel dose density of 3.5 μg/mm^2.

Indications for Use

The IN.PACT AV DCB is indicated for percutaneous transluminal angioplasty, after appropriate vessel preparation, for the treatment of stenotic and obstructive lesions up to 100 mm in length in native arteriovenous dialysis fistulas with reference vessel diameters of 4 to 12 mm [14].

Contraindications

The IN.PACT AV DCB is contraindicated for use in the following anatomy and patient types:

- Coronary arteries, renal arteries, and supra-aortic/cerebrovascular arteries.
- Patients who cannot receive the recommended antiplatelet and/or anticoagulant therapy post treatment.

- Patients determined to have a lesion that prevents complete inflation of an angioplasty balloon or proper placement of the delivery system.
- Patients with known allergies or sensitivities to paclitaxel.
- Women who are breastfeeding, pregnant, or are intending to become pregnant, or men intending to father children.

It is unknown whether paclitaxel will be excreted in human milk and whether there is a potential for adverse reaction in nursing infants from paclitaxel exposure.

Device Handling and Application

According to the manufacturer's IFU, the IN.PACT AV DCB is manufactured with nominal balloon diameters of 4 to 12 mm, and balloon lengths of 40 to 120 mm. The IN.PACT AV DCB is an 0.035" OTW balloon catheter that requires a 5 Fr to 9 Fr introducer sheath depending on the diameter of the balloon [14].

Before the application of the DCB, the target lesion should be prepared and predilated since the IN.PACT AV DCB itself is a semi-compliant low-pressure balloon and might not be able to dilate tight strictures. The target lesion should be predilated with a high-pressure balloon with the goal of residual stenosis less than 30%.

The catheter lumen should be flushed through the guidewire port with heparinized normal saline, however, care should be taken to not rinse or wipe the balloon. This product is not intended for the expansion or delivery of a stent or graft. The manufacturer advises to not exceed the rated burst pressure (RBP), which is based on the results of in vitro testing, as higher pressures may result in a ruptured balloon with possible intimal damage and/or dissection.

The manufacturer recommends, at a minimum, single antiplatelet therapy (e.g. aspirin, clopidogrel, ticlopidine, or prasugrel) before the procedure and for at least 4 weeks after the procedure [14].

Prior Studies

In 2019, the manufacturer received FDA approval of the IN.PACT AV DCB for the treatment of failing arteriovenous (AV) access in patients with end-stage renal disease (ESRD) undergoing dialysis. FDA approval is based on results from the (currently underway) IN.PACT AV Access study (https://clinicaltrials.gov/ct2/show/NCT03041467). This prospective, global, multicenter, blinded, randomized (1:1), investigational device exemption study enrolled 330 patients and evaluated the safety and effectiveness of the IN.PACT AV DCB at 29 sites in the United States, Japan, and New Zealand. The early data from the trial indicated that patients treated with the IN.PACT AV DCB maintained patency longer and required 56% fewer reinterventions than those treated with standard PTA through 6 months. Through 12 months, the data showed no difference in mortality rates between the IN.PACT AV DCB group and the PTA control group. Additionally, the data demonstrated that superior patency was achieved with the IN.PACT AV DCB versus BA in de novo and restenotic lesions, as well as all studied types of AV access.

In conclusion, the existing data suggests improved target lesion primary patency following treatment with angioplasty and IN.PACT AV DCB versus angioplasty

alone, both in the arteriovenous fistula circuit and central veins with index vessel diameter of 4–12 mm and target lesion up to 100 mm in length. The IN.PACT AV access study early data is promising and further results, including the long term follow up from the trial in 2023 and cost-effectiveness analysis, would be very helpful in shedding more light over the potential benefit of the IN.PACT AV DCB for the treatment of failing dialysis accesses.

Stellarex DCB

Stellarex DCB is a paclitaxel-coated balloon catheter with a coating consisting of a hydrophilic polymer excipient (polyethylene glycol 8000) dispersed with amorphous and crystalline paclitaxel particles at a dose of 2 μg/mm^2 [15]. It is designed for high coating durability and minimal particulate loss, enabling adhesion and transfer of paclitaxel from the balloon to the vessel wall when exposed to aqueous conditions [15]. Two radiopaque markers are present on the DCB to assist in precise positioning. As with the Lutonix DCB and the IN.PACT AV DCB, the Stellarex DCB is a semi-compliant balloon and thus requires similar vessel preparation. The 0.035" OTW DCB comes in a range of 4–6 mm diameters with a balloon length of 40–120 mm.

Indication for Use

Currently, the Stellarex 0.035" OTW DCB platform was approved for the treatment of de novo and restenotic lesions in superficial femoral and popliteal arteries. The Stellarex 0.014" OTW DCB was designed for below-the-knee peripheral artery disease but is only CE marked and is not approved for distribution, sale or use in the USA.

Contraindications

- Patients with known hypersensitivity to paclitaxel or structurally related compounds.
- Patients who cannot receive recommended antiplatelet and/or anticoagulation therapy.
- Women who are breastfeeding, pregnant or are intending to become pregnant or men intending to father children.
- Coronary arteries, renal arteries, and supra-aortic/cerebrovascular arteries
- Patients judged to have a lesion that prevents complete inflation of an angioplasty balloon or proper placement of the delivery system.

Device Handling and Application

The Stellarex 0.035" DCB requires a minimum 6 French introducer sheath. Diagnostic angiography is performed to evaluate the eligibility of a lesion for treatment. Appropriate intravenous anticoagulation is administered before the angioplasty. The lesion is then crossed over and pre-dilatation is performed with a standard PTA balloon of 1 mm less than that of the reference vessel diameter. A

minimum inflation time of 60 seconds is recommended according to the manufacturer's IFU [15].

Both prior to and after the procedure, the manufacturer recommends dual antiplatelet therapy (clopidogrel and acetylsalicylic acid (ASA or aspirin)) to optimize the effects of treatment with the Stellarex DCB. While the optimal duration of antiplatelet therapy is at the discretion of the physician, the manufacturer provides recommendations in the IFU as follows:

Pre-Procedure

- Clopidogrel 75 mg/day for 3 days prior to the angioplasty procedure or 300 mg as a loading dose on the day of the procedure.
- Acetylsalicylic acid (ASA) 81 mg/day to 325 mg/day on the day of the angioplasty procedure or prior at the discretion of the physician.

Post-Procedure

- Clopidogrel: 75 mg/day for a minimum of 30 days following the angioplasty procedure or prolonged use at the discretion of the physician. The recommended dose of ticlopidine is 250 mg twice a day in patients with a clopidogrel allergy.
- Acetylsalicylic acid (ASA): Minimum of 81 mg/day for a minimum of 6 months following the angioplasty procedure.

Prior Studies

The safety and effectiveness of the Stellarex 0.035" DCB in the femoropopliteal arteries is supported with data from the ILLUMENATE Pivotal study and the ILLUMENATE European Randomized Clinical Trial [16, 17]. The Stellarex ILLUMENATE Below-the-Knee Investigational Device Exemption study, designed to determine the safety and efficacy of the Stellarex 0.014" DCB, is currently underway. The results of these studies, which focus on lower extremity peripheral artery disease rather than dialysis accesses, are beyond the scope of this chapter.

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Non-tunneled Hemodialysis Catheter

20

Suh Chien Pang, Chieh Suai Tan, Anil Agarwal, and Steven Wu

Introduction

While tunneled dialysis catheters (TDC) are preferred over non-tunneled dialysis catheters (NTDC), the use of non-tunneled dialysis catheter is indicated when

1. Duration of catheter use is expected to be short, e.g. 1 week
2. Presence of bacteremia or sepsis where placement of a tunneled dialysis catheter is contraindicated
3. Deranged coagulation parameters where placement of a tunneled dialysis catheter is considered high risk for bleeding
4. Failed AV access cannulation and the access is either being rested or waiting for an intervention

Compared to the traditional method using anatomic landmark, insertion of central venous catheter under ultrasound guidance by a trained operator increases the likelihood of successful insertion [1, 2] and reduces the likelihood of inadvertent arterial puncture [3, 4]. Ultrasound imaging allows localization of the target vein and detection of any variant anatomy or intraluminal thrombosis. Hence, integrating

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both knowledge from anatomic landmark techniques and ultrasound guidance is important to achieve successful and safe dialysis catheter placement.

Sites of Insertion

Similar to tunneled dialysis catheter insertion, the preferred site of insertion, in descending order, is the internal jugular (IJ), external jugular (EJ) and femoral vein. In the absence of prior central vein stenosis or pacemaker, the right side approach is preferred over left side due to its more direct anatomy. Due to the high incidence of stenosis associated with placement of subclavian catheters, subclavian line insertions should be avoided unless it is absolutely necessary, where there are no other alternatives. Femoral catheters are associated with a higher incidence of infection, especially in individuals with higher body mass index [5, 6]. The femoral catheter should not be placed on the same side of the transplanted kidney as it can lead to venous obstruction or thrombosis of the transplanted kidney.

For the non-tunneled IJ approach, the puncture site is generally higher (superior) than that commonly used during the placement of a tunneled dialysis catheter (Fig. 20.1). Such an approach has the advantage of providing adequate space for placement of a tunneled dialysis catheter at a later stage. In general, a new puncture site is preferred during tunneled dialysis catheter insertion as conversion of a non-tunneled catheter to a tunneled catheter carries the risk of entry-site contamination and "kinking" of the catheter due to the difference in the angle of approach.

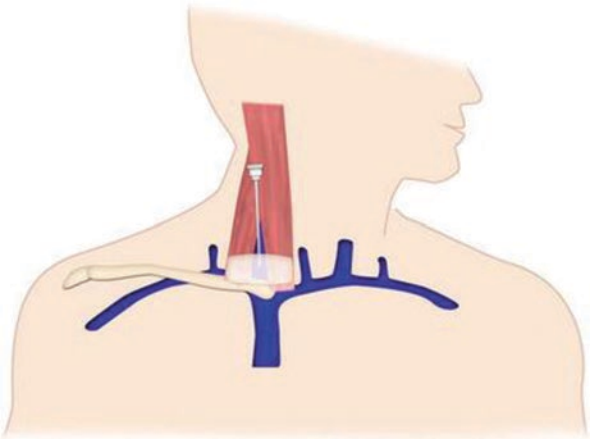

Fig. 20.1 Site of puncture for right internal jugular approach. Identify the two heads of the sternocleidomastoid muscle and place the ultrasound probe just above the level of the clavicle. Identify the internal jugular vein on the ultrasound screen and adjust the probe such that the vein is in the center of the screen. The needle is then inserted at the midpoint on the superior border of the ultrasound probe

Equipment

Prepare the following items on a sterile trolley:

1. A micropuncture introducer set (contains a 21 Gauge needle, a 0.018 in. wire and a 5 Fr sheath)
2. A 0.035 in. hydrophilic wire
3. A set of 8, 10 and 12 Fr dilators
4. A non-cuffed dialysis catheter
5. 1 % Lignocaine (Lidocaine)
6. Normal Saline
7. Syringes: Two 10 cc syringes and one 20 cc syringe
8. A number 11 surgical blade
9. One 2/0 non absorbable suture
10. A needle holder
11. A hemostat
12. ACD solution
13. Ultrasound probe with sterile cover and gel

Steps for Right IJ Non-Tunneled Catheter Insertion

1. The following steps require use of micropuncture set, ultrasound guidance and fluoroscopy. For bedside insertion, usually only ultrasound guidance is available and is considered adequate except in difficult cases. Not all non-tunneled catheter insertion kits include the micropuncture introducer set which needs to be acquired separately. Review the indications for non-tunneled dialysis catheter insertion and plan the site of placement. An informed consent must be obtained from the patient or appropriate authorized person.
2. Scan the bilateral IJ veins to assess their patency and finalize the site of placement. If the vein appears small or collapsed, place the patient in the Trendelenburg position to distend the vein. If the vein appears large with multiple collateral veins in the neck, central vein stenosis may be present. If the vein is noncompressible, a thrombus may be present.
3. Clean and drape the insertion site along with a large area of neck and chest (Fig. 20.2a).
4. Place the ultrasound probe within the sterile sleeve and place it below the apex of the anatomical triangle that is formed between the two heads of the sternocleidomastoid muscle and the clavicle. Position it perpendicular to the IJ vein to obtain a transverse view of the vein. Infiltrate the skin over the insertion site with 1 % lidocaine (Fig. 20.2b).
5. Insert the 21 gauge micropuncture access needle from the superior aspect of the probe into the vein under real-time ultrasound guidance. Avoid puncturing through the muscle as this would result in discomfort whenever the patient turns his head (Fig. 20.2c).

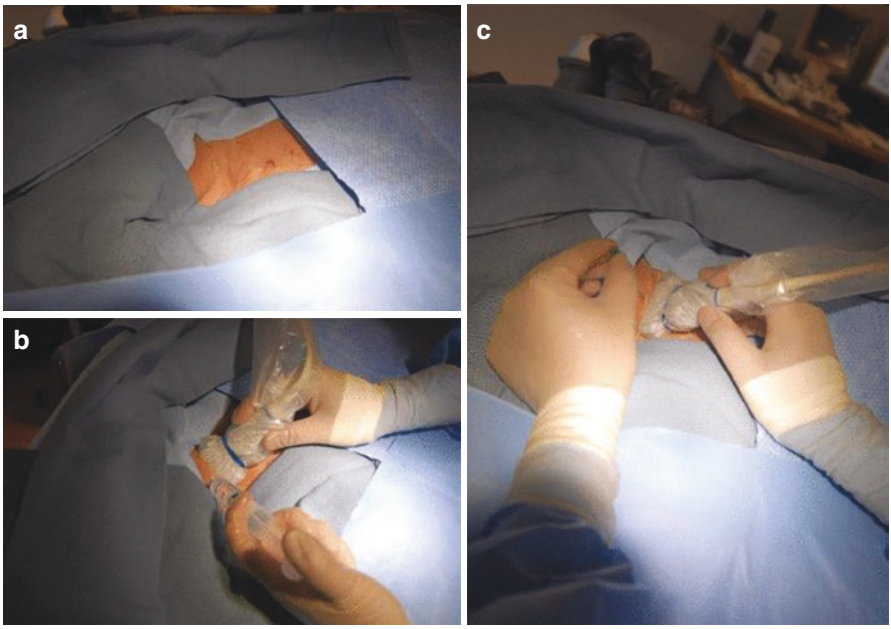

Fig. 20.2 (**a**) Clean and drape the operative site. (**b**) Infiltrate the insertion site with lidocaine. (**c**) Puncture the vein with a micropuncture needle under ultrasound guidance

6. After successful cannulation, insert the 0.018 in. wire into the vein under fluoroscopy. Make a small incision along the wire to enlarge the venotomy site. Exchange the needle for the 5 Fr sheath over the 0.018 in. wire (Fig. 20.3a–c).
7. Exchange the 0.018 in. wire for a 0.035 in. wire and advance the tip into the inferior vena cava (IVC) under fluoroscopy if available. This can be achieved by asking the patient to hold his breath after deep inspiration. Occasionally, a 5 Fr Kumpe catheter is needed to steer the tip of the wire into the IVC. (The micropuncture set is not universally utilized for insertion of non-tunneled catheters and a direct puncture with 18 gauge needle is often utilized followed by advancement of 0.035 in. guidewire).
8. Remove the sheath and serially dilate the venotomy tract using 8, 10 and 12 Fr dilators (Fig. 20.4a). The tip of the 8 Fr dilator is positioned at the distal superior vena cava at full inspiration and the external portion of the dilator is marked at the venotomy site. The full length of the dilator is approximately 20 cm. The distance between the marking at the venotomy site and the tip of the dilator is equal to the intravascular distance from the venotomy site to the distal superior vena cava. This measurement is used to determine the length of the non-tunneled catheter to be placed. In general, for the right IJ, the length of the catheter used is between 15 and 20 cm. For the left IJ placement, the length of the catheter used is between 20 and 24 cm.

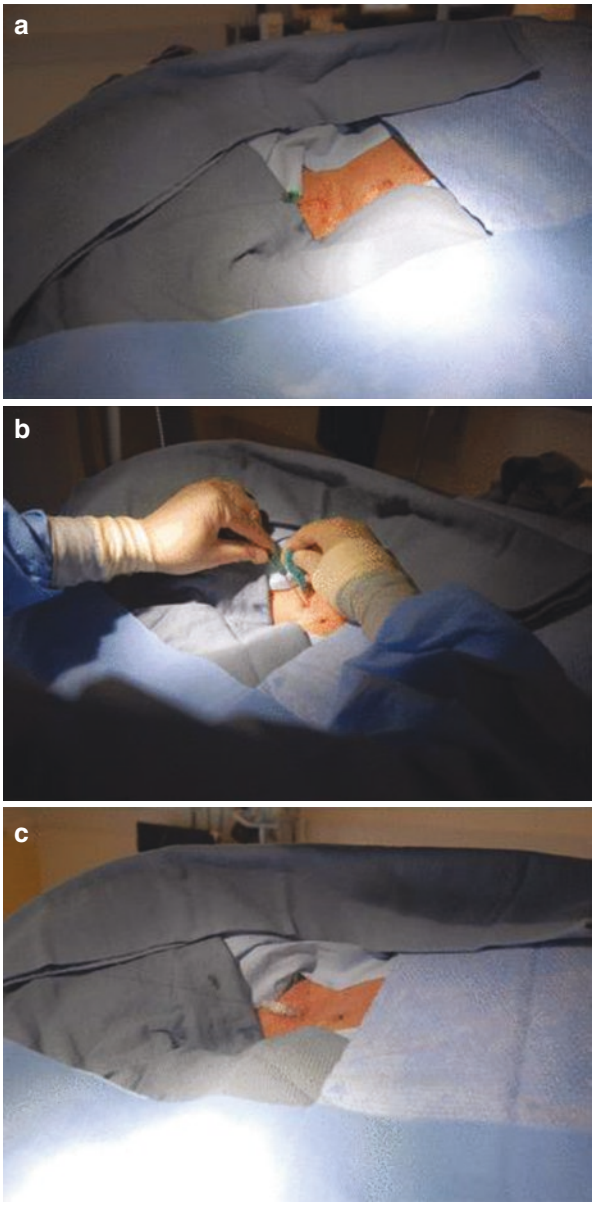

Fig. 20.3 (**a**) Insert the micro-wire into the needle. (**b**) Make a small incision in the skin before exchanging the needle for a sheath. (**c**) Insert the sheath over the wire

9. After final dilatation with the 12 Fr dilator, insert the non-tunneled dialysis catheter over the guidewire.
10. The catheter tip is positioned at the distal superior vena cava under fluoroscopy.
11. Remove the guidewire and test the flow of the catheter using a 20 mL syringe. The syringe should fill up rapidly within 3 seconds without much resistance and

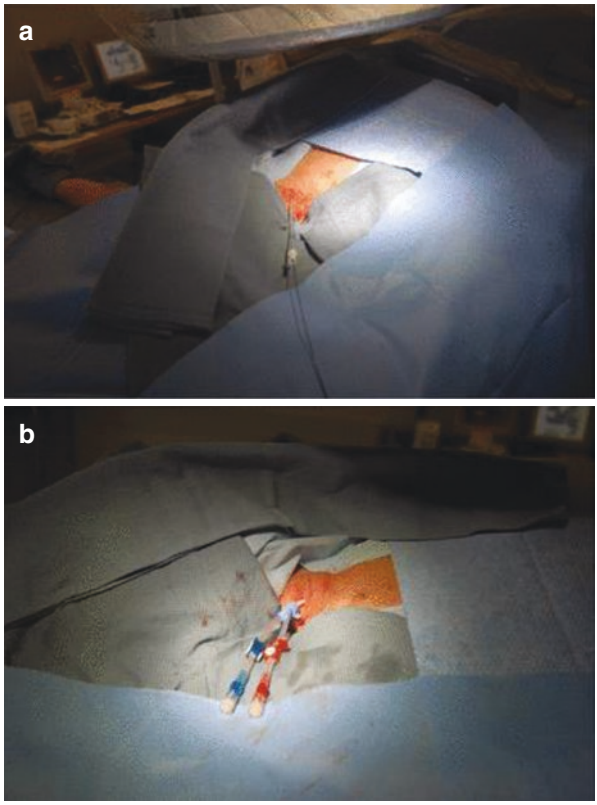

Fig. 20.4 (**a**) Remove the micropuncture sheath and dilate the venotomy tract with dilators. (**b**) Placement of catheter

there should not be any resistance during flushing. The tip position may be rotated to allow best possible flow.

12. Once the flow of the catheter has been optimized, flush the catheter with normal saline. Lock the catheter with anticoagulant to prevent thrombus formation within the catheter.
13. Suture the wings of the catheter to the skin with non-absorbable sutures. Cover the wound and catheter with sterile breathable dressings.
14. The dressing should be changed whenever it is moist or wet. Water impermeable dressings should be used during shower to keep the wound dry (Fig. 20.4b).

Complications of Non-Tunneled Catheter Insertion

Acute Complications

The acute complications for non-tunneled dialysis catheter insertion are similar to those seen with tunneled dialysis catheter insertion. Briefly, these include:

1. Arterial puncture
2. Pneumothorax and hemothorax
3. Air embolism
4. Arrhythmia

The approach and treatment strategies for the complications are described in Chap. 21.

Subacute Complications

1. Infection
   Non-tunneled dialysis catheters should not be left in-situ for more than 1 week as they have a higher risk of infection compared to tunneled dialysis catheters. In the event of infection, the patient should be treated with broad spectrum antibiotics and the non-tunneled dialysis catheter should be removed.
2. Poor flow
   Do not attempt thrombolysis with tPA if the non-tunneled dialysis catheter develops poor flow. The appropriate treatment is to adjust it under fluoroscopy or place a new catheter over a guidewire.

Timing of Removal of Non-Tunneled Dialysis Catheter

The 2019 KDOQI guideline 10.10 [7] recommends limiting non-tunneled dialysis catheter use to 2 weeks as the infection rates increase sharply after 2 weeks [8] and are much higher as compared to infection rates in tunneled dialysis catheter.

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Tunneled Hemodialysis Catheter

21

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Insertion of Tunneled Dialysis Catheter

Introduction

Despite its well-known complications, Central Venous Catheter (CVC) are used in 80% of all incident hemodialysis patients [1]. Insertion of a central venous catheter for hemodialysis is an interventional procedure that involves applying various endovascular techniques. It involves obtaining vascular access under real-time ultrasound guidance, wire manipulations, sheath placements and fluoroscopic localization of the catheter tip.

Sites of Insertion

The preferred site of insertion is the right internal jugular (IJ) vein, as it is the shortest and most direct route to the right atrium. The alternative insertion sites are the

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left IJ, followed by the right external jugular (EJ), left EJ, right femoral and left femoral vein. Subclavian veins should not be used for catheter placement as they are associated with an unacceptably high incidence of stenosis, which would compromise future upper limb AV access placement. The femoral catheter should not be placed on the same side of the transplanted kidney as it can lead to venous obstruction or thrombosis of the transplanted kidney. As the use of CVC is associated with central vein stenosis, the site of insertion should be contralateral to the planned AV-access site [2]. A femoral catheter should be avoided in potential kidney transplant candidate as it may limit the use of the iliac vein during kidney transplantation.

General Features of Tunneled Dialysis Catheter

Tunneled dialysis catheters are commonly used for a longer duration. They are made of a softer materials (e.g. polyurethane/polycarbonate copolymer or silicone) to enhance longevity within the vessel and reduce the risk of vascular damage. The catheters have two lumens of different inner luminal shapes and tip design (step tip, split tip, symmetric tip) with sides holes of varying forms. They come with a polyester cuff in the subcutaneous tract for tissue in-growth to fix the catheter in place and to provide a physical barrier against infection. There were no significant differences in primary assisted patency, infection and thrombosis rate between the various CVC types and designs [3–7].

Equipment

Prepare the following items on a sterile trolley (Fig. 21.1):

1. 21 Gauge micropuncture access needle
2. 0.018 in. micro-wire and 0.035 in. hydrophilic wire
3. A 5 Fr micropuncture sheath
4. A set of 8, 10 and 12 Fr dilators
5. 14–16 French dilator with a peel-away sheath

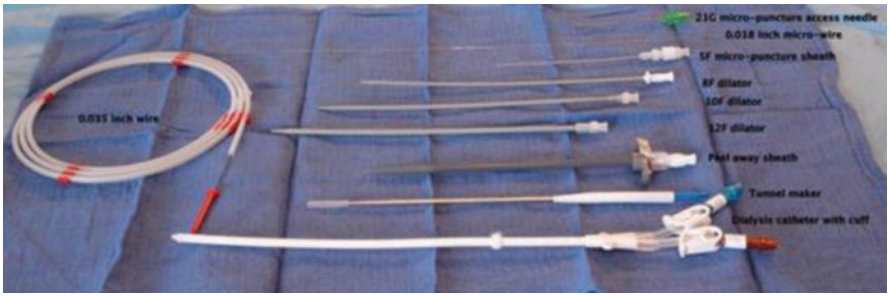

Fig. 21.1 Tools for insertion of tunneled dialysis catheter

6. A tunnel maker
7. Cuffed dialysis catheter
8. 1% Lidocaine solution for injection
9. Normal Saline
10. Syringes
11. A number 11 surgical blade
12. One 2/0 non-absorbable suture
13. One 2/0 absorbable suture
14. Needle holder
15. Hemostat

Steps for a Right Sided IJ Tunneled Catheter Insertion

1. Review the indications for tunneled dialysis catheter insertion and locate the site of placement.
2. Prepare equipment as described.
3. Scan both IJ veins to assess their patency (Fig. 21.2a–d). Place the ultrasound probe just above the clavicle and perpendicular to the IJ vein to obtain a transverse view of the vein and mark the site of placement.
4. Clean and drape the patient.
5. Infiltrate the insertion site with 1% lidocaine.
6. Make a small venotomy incision at the desired insertion site and dilate the incision with an artery forceps.
7. Place the ultrasound probe within the sterile sleeve and place it next to the skin incision. Insert the 21 gauge micropuncture access needle from the lateral aspect of the probe through the venotomy incision into the vein under real-time ultrasound guidance (Fig. 21.3a–c). The flow of blood from the needle hub indicates successful cannulation.
8. After successful cannulation, insert the 0.018 in. micro-wire through the micropuncture needle into the vein. Confirm the position with fluoroscopy (Fig. 21.4a).
9. Exchange the needle for the 5 Fr micropuncture sheath over the 0.018 in. micro-wire.
10. Remove the 0.018 in. micro-wire together with the inner dilator from the micropuncture sheath and insert a 0.035 in. wire into the sheath (Fig. 21.4b).
11. Advance the tip into the inferior vena cava (IVC) under fluoroscopy. This can be achieved by asking the patient to hold his/her breath after deep inspiration (Fig. 21.4c).
12. Occasionally, a 5 Fr Kumpe catheter is needed to steer the tip of the 0.035 in. guidewire into the IVC. Place the Kumpe catheter over the wire, withdraw the tip of the wire into the catheter, change the direction of the Kumpe catheter tip and re-insert the wire into the IVC (Fig. 21.4d).
13. Remove the micropuncture sheath and serially dilate the venotomy tract using the 8, 10 and 12 Fr dilators. Position the tip of the 10 Fr dilator at the proximal right atrium and make a marking on the external portion of the dilator at the

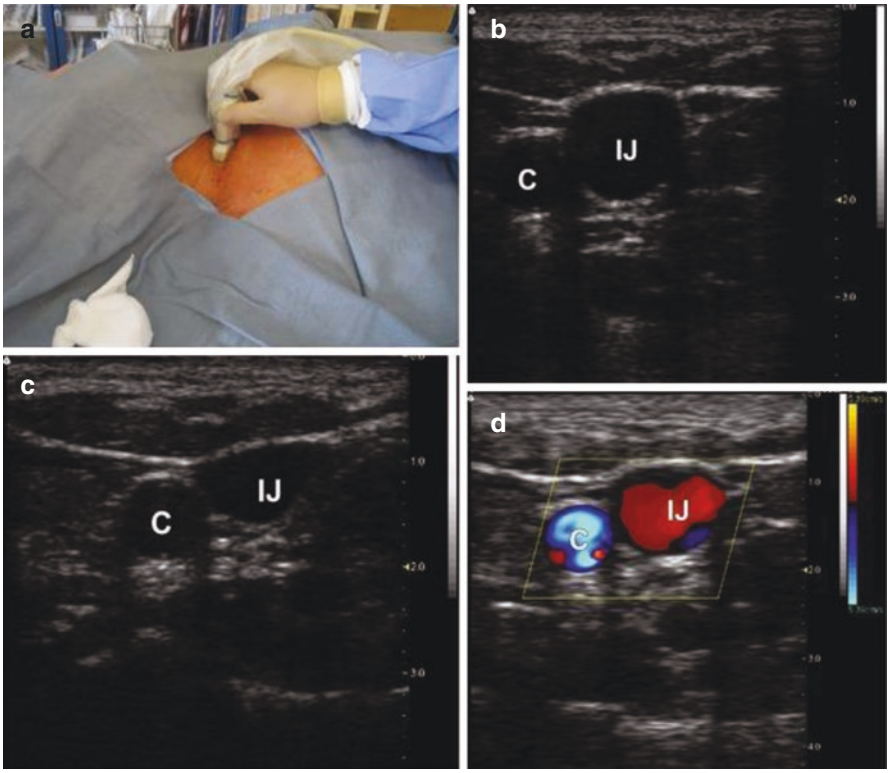

Fig. 21.2 (**a**) Place the ultrasound probe just above the clavicle, between the two heads of the sternocleidomastoid muscle. (**b**) The internal jugular (IJ) vein lies lateral to the carotid artery. (**c**) The internal jugular vein is easily compressible with the ultrasound probe. (**d**) Occasionally, color Doppler may be used to confirm flow within the vessels. C carotid artery; IJ internal jugular vein

venotomy site. The 12 Fr dilator is left in-situ while preparing the catheter for insertion (Fig. 21.5a).

14. The dilators are 20 cm in length and the distance from the marking on the 10 Fr dilator to the tip represents the intravascular distance from the venotomy site to the proximal right atrium (Fig. 21.5b).
15. Catheter lengths are measured from the cuff to the tip. For the right and left IJ approaches, the catheter lengths used are 19 and 23 cm respectively. A 28 cm catheter is usually used for the femoral approach. The intravascular segment of the catheter is estimated using the marking on the 10 Fr dilator. The position of the exit site should be approximately 2 cm from the cuff of the catheter (Fig. 21.5c).

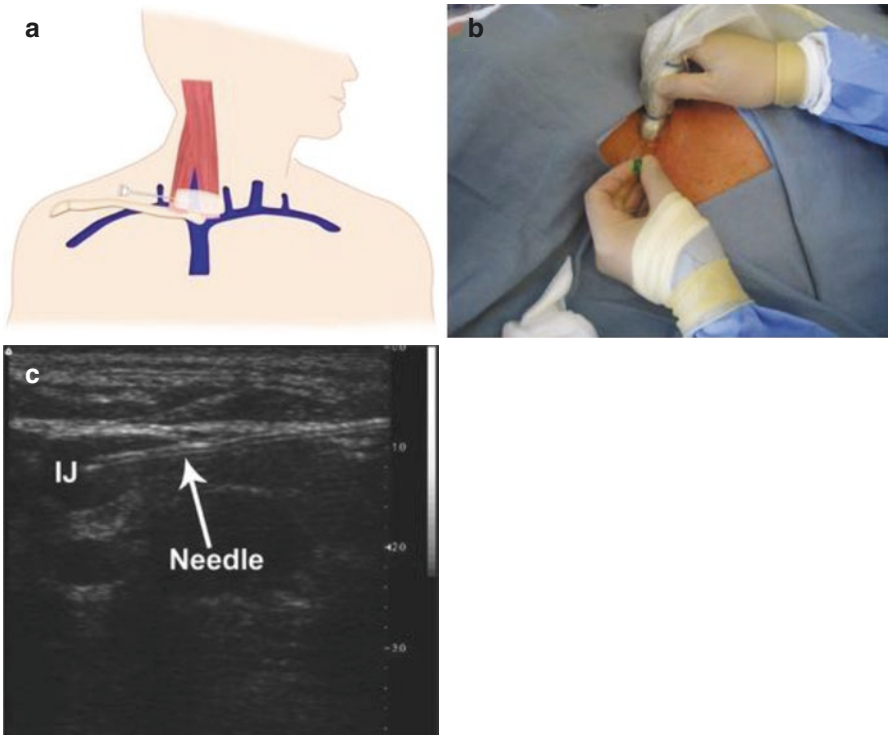

Fig. 21.3 (**a**) Insert the needle at the midpoint on the lateral aspect of the probe under real time ultrasound imaging. (**b**) Advance the needle under ultrasound guidance. The needle should be moving parallel to the superior border of the ultrasound probe to stay within the ultrasound plane. (**c**) The needle should be visualized to enter the IJ vein on real-time ultrasound imaging

16. After infiltrating the skin with lidocaine, make a small incision at the desired exit site (Fig. 21.6a).
17. To create a subcutaneous tunnel, use the tunnel maker. Mount the catheter to the end of the tunnel maker (Fig. 21.6b).
18. Create the tunnel using the tunnel maker and pull the tunnel maker-catheter assembly towards the venotomy site (Fig. 21.6c).
19. The catheter is pulled through the tunnel to the venotomy site (Fig. 21.6d).
20. Remove the 12 Fr dilator and insert a peel-away sheath/dilator set and place it into the superior vena cava under fluoroscopy. Remove the 0.035 in. wire and inner dilator from the peel-away sheath and insert the catheter into the peel-away sheath (Fig. 21.7a).
21. Insert the catheter tip into the peel-away sheath (Fig. 21.7b).

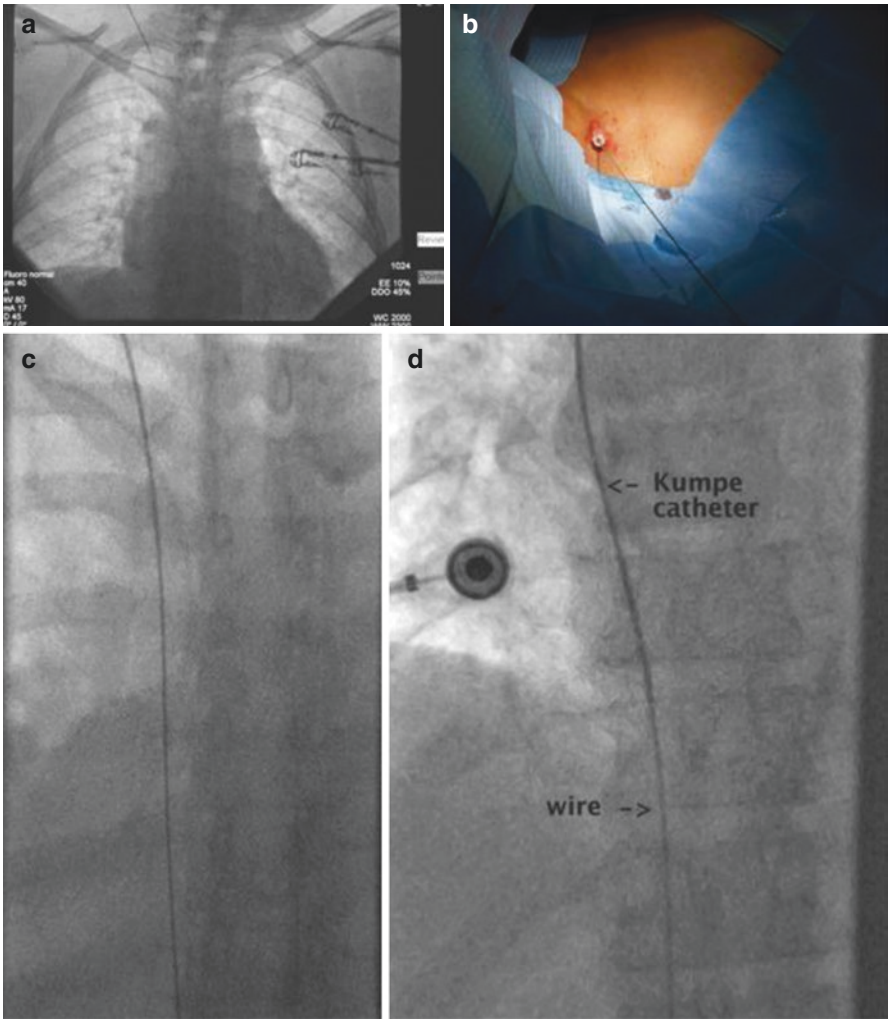

Fig. 21.4 (**a**) Insert the micro-wire under fluoroscopy. (**b**) Exchange for a 0.035 in. wire. (**c**) Ensure that the tip of the wire is in the IVC. (**d**) Use of Kumpe catheter

22. Using a series of "push" (pushing in the catheter with the thumb) and "pull" (pulling apart the peel-away sheath), push the entire length of the catheter into the vein (Fig. 21.7c).
23. Position the tip of the catheter at the right mid-atrium under fluoroscopy, which is approximately 5 cm inferior to the level of the tracheal bifurcation (Fig. 21.7d). Test the flow of the catheter using a 20 ml syringe. The syringe should fill up rapidly within 3 seconds without much resistance and there should not be any resistance during flushing. The tip position may be adjusted to allow best possible flow.

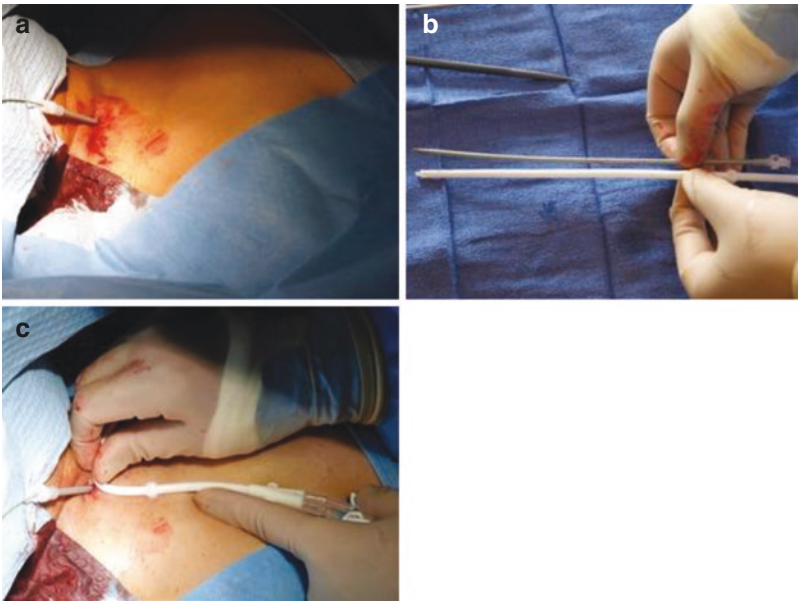

Fig. 21.5 (**a**) Leave the 12 Fr dilator in situ after dilatation. (**b**) Use the 10 Fr dilator to estimate the length of the intravascular portion of the catheter. (**c**) Mark the position of the exit site

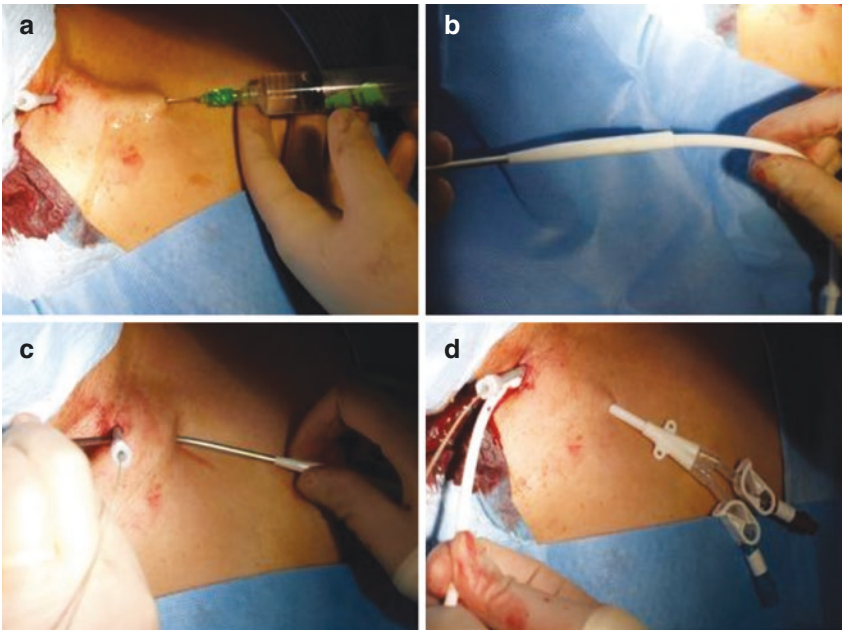

Fig. 21.6 (**a**) Infiltrate the skin with lignocaine. (**b**) Mount the catheter to the end of the tunnel maker. (**c**) Create a subcutaneous tunnel using the tunnel maker. (**d**) The catheter is pulled through the tunnel

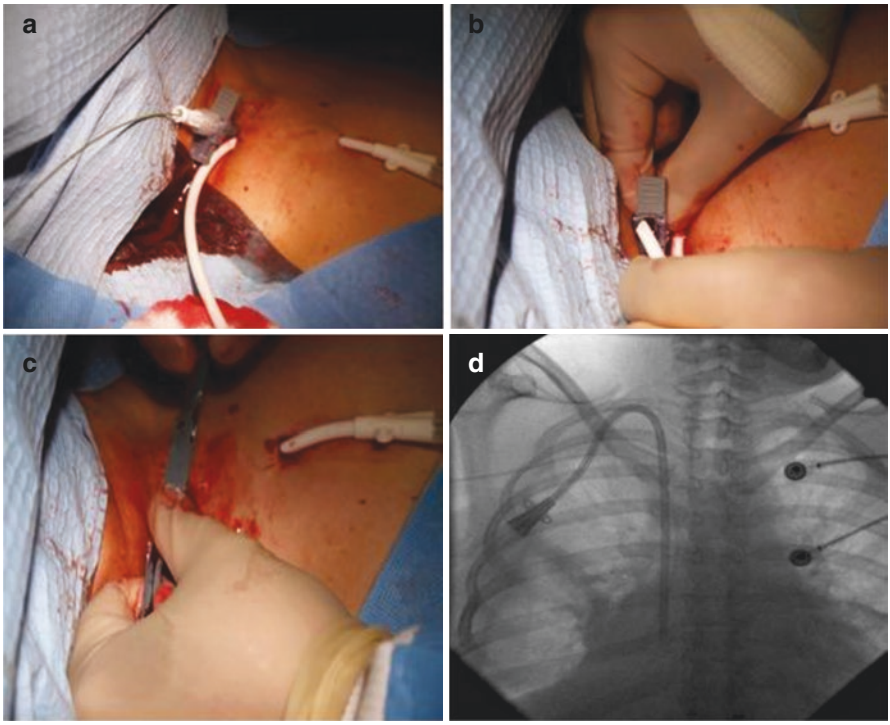

Fig. 21.7 (**a**) Insert the peel-away sheath. (**b**) Insert the catheter into the peel-away sheath. (**c**) Push the catheter into the vein via the peel-away sheath. (**d**) Check the final position of the catheter

24. Once the flow of the catheter has been optimized, flush the catheter with normal saline. Lock the catheter with an anticoagulant to prevent thrombus formation within the catheter. Either heparin (1000–5000 units/ml) or 4% acid citrate dextrose (ACD) solution can be used. The exact amount needed is usually indicated on the hub of the catheter. The risk of heparin-induced thrombocytopenia can be avoided by using ACD as a locking agent instead of heparin.
25. The venotomy site is closed using absorbable interrupted sutures and a non-absorbable purse string suture is applied at the exit site to secure hemostasis (Fig. 21.8a). The catheter is secured to the chest by suturing the wings of the catheter to the skin with non-absorbable sutures.
26. The surgical sites should be covered with sterile breathable dressings to protect the wound. Patients should be given clear instructions on how to care for their catheters after tunneled catheter placement. In particular, the dressings should be changed whenever it is moist or wet. Water impermeable dressings should be used during showers to keep the wound dry (Fig. 21.8b).
27. Heparin free dialysis should be ordered if the catheter is being used immediately after placement.

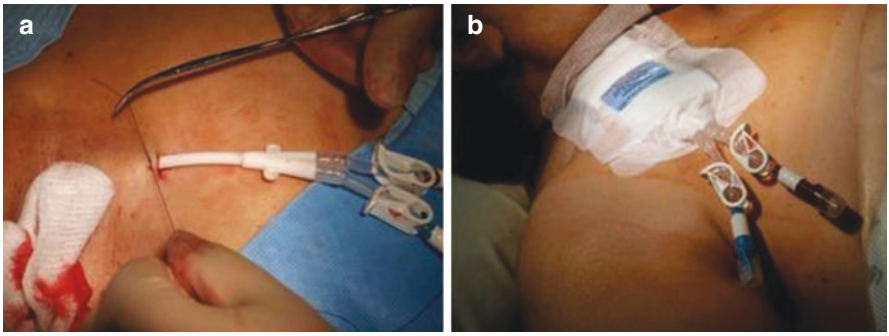

Fig. 21.8 (**a**) Close the exit site with a purse string suture. (**b**) Protect the wound with sterile dressings

28. All non-absorbable sutures can be removed 14 days after insertion. Before suture removal, confirm that the catheter is anchored to the subcutaneous tissue via the catheter cuff by giving it a gentle tug.

Optimal Dialysis Catheter Tip Placement

Optimal catheter tip placement is associated with reduced catheter dysfunction and infection irrespective of left- or right-sided approach [8]. A malpositioned catheter tip can lead to catheter malfunction, right atrial trauma, vascular perforation, venous thrombosis and arrhythmia. Confirmatory imaging should always be performed to ensure that the catheter tip is placed in the right mid atrium.

Challenging Dialysis Catheter Insertion (Table 21.1)

Acute Complications of Tunneled Dialysis Catheter Insertion

Regardless of how "minor" or "simple" the procedure is perceived to be, never underestimate the complications that may arise during the procedure. Obeying the "rules" of endovascular intervention and developing good habits during training can go a long way to decrease procedure related complications. The following are some of the complications that one may encounter during dialysis catheter placement, and the precautions and steps to treat them if they occur.

Table 21.1 Summarises some of the potential clinical challenges that may occur during tunneled catheter insertion and the possible steps that can be taken to overcome the challenges

Clinical challenges	Troubleshooting
Central vein stenosis	Central vein stenosis remains one of the most common vascular access–related complications, with an occurrence rate of up to 40% in prevalent hemodialysis patients [9]. Unexpected severe stenosis of the central veins warrant additional fluoroscopy during catheter insertion or abandonment of the procedure. In addition, previous internal jugular catheter can increase the risk of such abnormalities. However, successful placement of catheter can be performed after angioplasty of stenotic central vein. (Fig. 21.9a–c)
Existing central vein stent	The brachiocephalic vein is a common site for stent placement which may occasionally resulted in jailing of major veins. In this case, insertion of dialysis catheter through the side or within the stent may be performed. (Fig. 21.10a, b)
Left superior vena cava (SVC) persistence	Persistent left SVC is a rare but important congenital vascular anomaly with a reported incidence of 0.3% to 1.3%. Any endovascular procedure of left-sided AV-access may be made difficult and complicated by arrhythmia and coronary sinus thrombosis. Persistent left SVC should be suspected if there is failure of the guidewire or catheter to cross the midline on imaging. A definite diagnosis can be made by angiography (Fig. 21.10c) and when seen, catheter insertion on this side should be avoided.

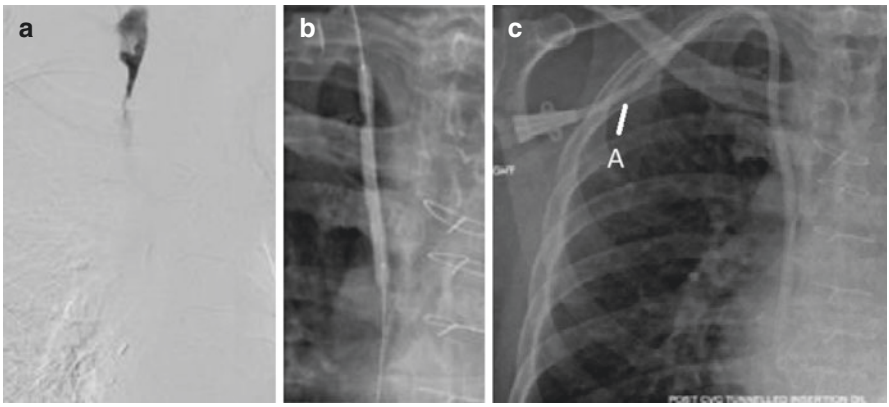

Fig. 21.9 (**a**) Central venogram showed severe stenosis (90%). (**b**) Angioplasty of stenotic zone. (**c**) Tunneled catheter in place

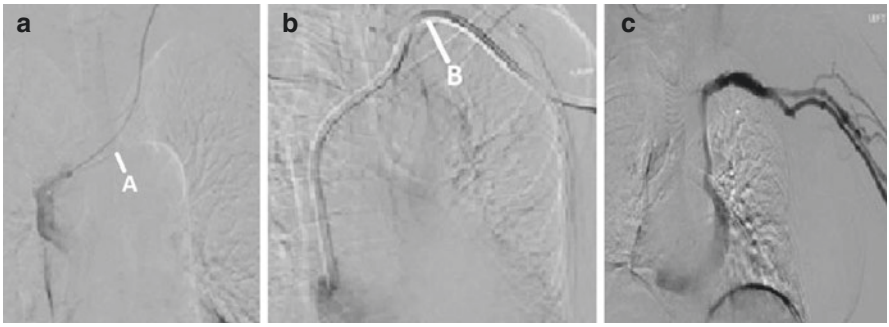

Fig. 21.10 (**a**) Initial angiogram showed patent central vein distal to stent. **A** Stent in brachiocephalic vein (**b**) Fluoroscopic appearance of chest post tunneled catheter placement. (**c**) Venography of the left upper extremity demonstrated a persistent left SVC draining into the coronary sinus

Arterial Puncture

Prevention is always easier than treatment.

1. Always access the vein under real-time ultrasound guidance and be cognizant of the depth and ultrasound plane.
2. Always use the micropuncture set to gain access the vein as cannulation created using the micropuncture needle is small and bleeding can be stopped readily by compression.
3. Always verify the position of the micropuncture wire by fluoroscopy.

In the event of an arterial puncture, treatment is dependent on which stage of the procedure the complication is discovered.

1. If the complication is discovered before dilatation of the venotomy tract, the wires and micropuncture sheath can be safely removed. Direct compression can be applied to arrest the bleeding.
2. If the complication is discovered after dilatation of the venotomy tract, leave the dilator in-situ to tamponade the vessel and call for help. The arterial puncture can be closed either by open surgical repair or using an arterial closure device.

Pneumothorax

In the event of a pneumothorax, chest tube insertion is often necessary to evacuate the air leak.

Hemothorax

In the event of a hemothorax, surgical intervention is often necessary to stop the bleeding and evacuate the blood.

Air Embolism

Preventive measures include:

1. Identify high risk patients. Dehydrated patients are at increased risk of air embolism during line insertion. Their veins may be collapsed or show variation in size with the respiratory cycle on ultrasound. Give fluid boluses and perform the insertion with the patient in the Trendelenburg position to minimize the risk of air embolism.
2. Always occlude the hub of the needle and close the hemostatic valve of the peel-away sheath during the procedure. As an added precaution, pinch the peel-away sheath between your fingers after you have removed the inner dilator.
3. Instruct the patient to hold his/her breath during puncture of the IJ vein and insert the wire though the needle rapidly after successful puncture to avoid this complication.
4. The patient should be instructed to hold his/her breath during exchanges over the wire.

If there is significant air embolism

1. Immediately place the patient in the left lateral decubitus and Trendelenburg position. If cardiopulmonary resuscitation is needed, place the patient in a supine and head down position.
2. Administer 100% oxygen and perform endotracheal intubation if necessary.
3. Attempt removal of air from the circulation by aspirating from the central venous catheter.
4. Fluid resuscitate the patient and consider hyperbaric oxygen treatment.

Cardiac Arrhythmia

To prevent the wire from triggering arrhythmias during the procedure, always pass the guidewire tip into the IVC.

Vessel Injury During Dilatation of the Venotomy Tract

The guidewire might kink or buckle during dilatation of the venotomy tract. Always pull back the wire slightly before pushing the dilators into the vessels.

Subacute Complications of Tunneled Dialysis Catheter

Suboptimal Flow

1. If the tunneled catheter has poor flow within a week of placement, it is often due to suboptimal positioning of the catheter tip, migration of catheter tip or kinking of catheter.
   (a) Check the position of the catheter tip on a chest x ray. In particular, look for any kinks in the catheter (Fig. 21.11a–c)
   (b) Withdraw the catheter if the tip of the catheter is distal to the mid atrium. If the tip of the catheter is proximal to the mid atrium, advancing the catheter carries the risk of contaminating the subcutaneous tunnel tract and infection. In the latter situation, exchanging the catheter over a guide wire is preferred.

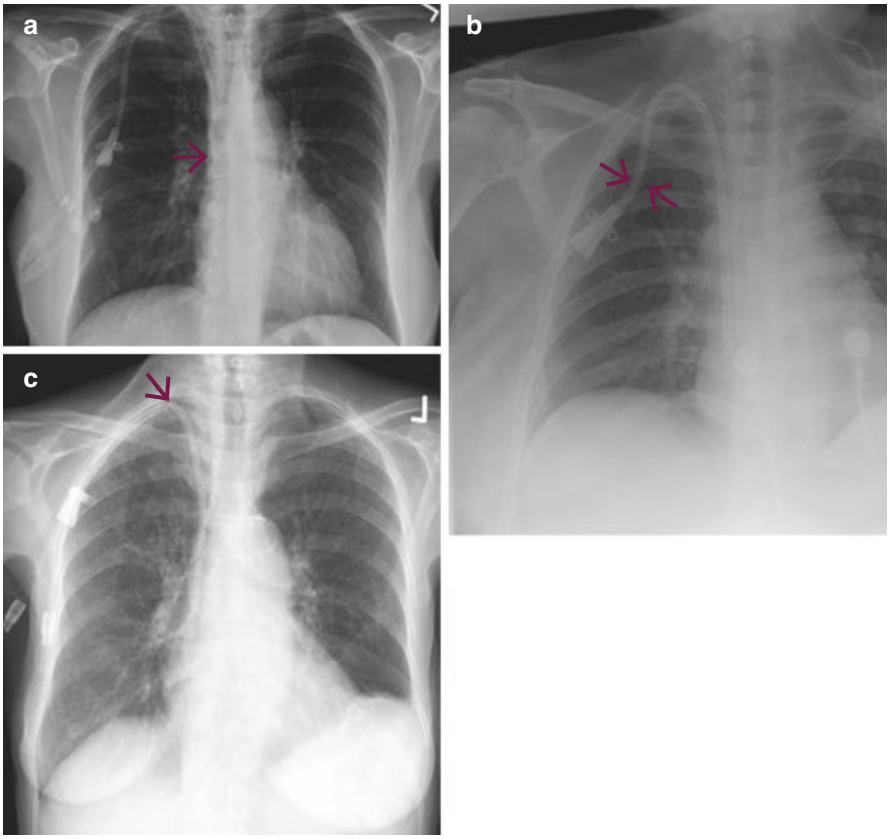

Fig. 21.11 (**a**) Catheter is too short. *Arrow* shows that the of catheter is in the superior vena cava. (**b**) Tip of catheter is in an optimal position but the *arrow* shows that catheter is "kinked" by the purse string suture at the exit site. (**c**) *Arrow* shows that the catheter is "kinked" at the venotomy site

2. If the catheter tip is in the correct position, a trial of a thrombolytic agent may be attempted.
   (a) The procedure should be carried out in a sterile manner. Clean and drape the patient.
   (b) Remove the caps of the catheter ports and aspirate 5 ml of blood from each lumen to remove the locking agent.
   (c) Instill 2 ml of TPA (1 mg/ml) into each lumen and allow it to dwell for half an hour.
   (d) Aspirate both catheter ports and discard the initial 5 ml of blood.
   (e) Test catheter flow with a 20 ml syringe. If the flow remains suboptimal, schedule for catheter exchange over a guide wire.
3. If the catheter develops poor flow more than a month after placement, it is probably secondary to obstruction from fibrin sheath formation around the catheter tip. Fibrin sheath has been demonstrated in 47% of long-term CVCs at the time of removal [10]. A trial of tPA may be attempted. If unsuccessful, exchanging the tunneled catheter over a guidewire with or without disruption of the fibrin sheath is the treatment of choice.
   (a) Check the position of the catheter tip on chest x-ray.
   (b) Aspirate both catheter ports and discard the initial 5 ml of blood which contains the locking agent
   (c) Insert a 0.035 in. angled stiff guide wire through the venous port of the catheter into the inferior vena cava (Fig. 21.12a).
   (d) Free the pre-existing catheter cuff by blunt dissection and withdraw the catheter gently by approximately 3 cm. Gently inject 10–15 ml of contrast material into the arterial port to visualize the fibrin sheath (Fig. 21.12b).
   (e) Remove the pre-existing catheter and insert a 12 or 14 mm angioplasty balloon catheter over the wire via the subcutaneous tunnel tract, and inflate the balloon in the SVC to disrupt the fibrin sheath (Fig. 21.12c).
   (f) Exchange a new tunneled dialysis catheter over the guide wire and place the tip within the proximal SVC. Inject 10–15 ml of contrast via the arterial port to check for residual fibrin sheath (Fig. 21.12d). If fibrin sheath is still present, repeat the angioplasty. If there is no residual fibrin sheath, advance the catheter tip to the desired position in the mid atrium.

Catheter-Related Infection

Patients dialyzing using a CVC are at increased risks of catheter-related infection and have increased morbidity and mortality with a reported incidence of 1.1 to 5.5 episodes per 1000 catheter day [11–13].

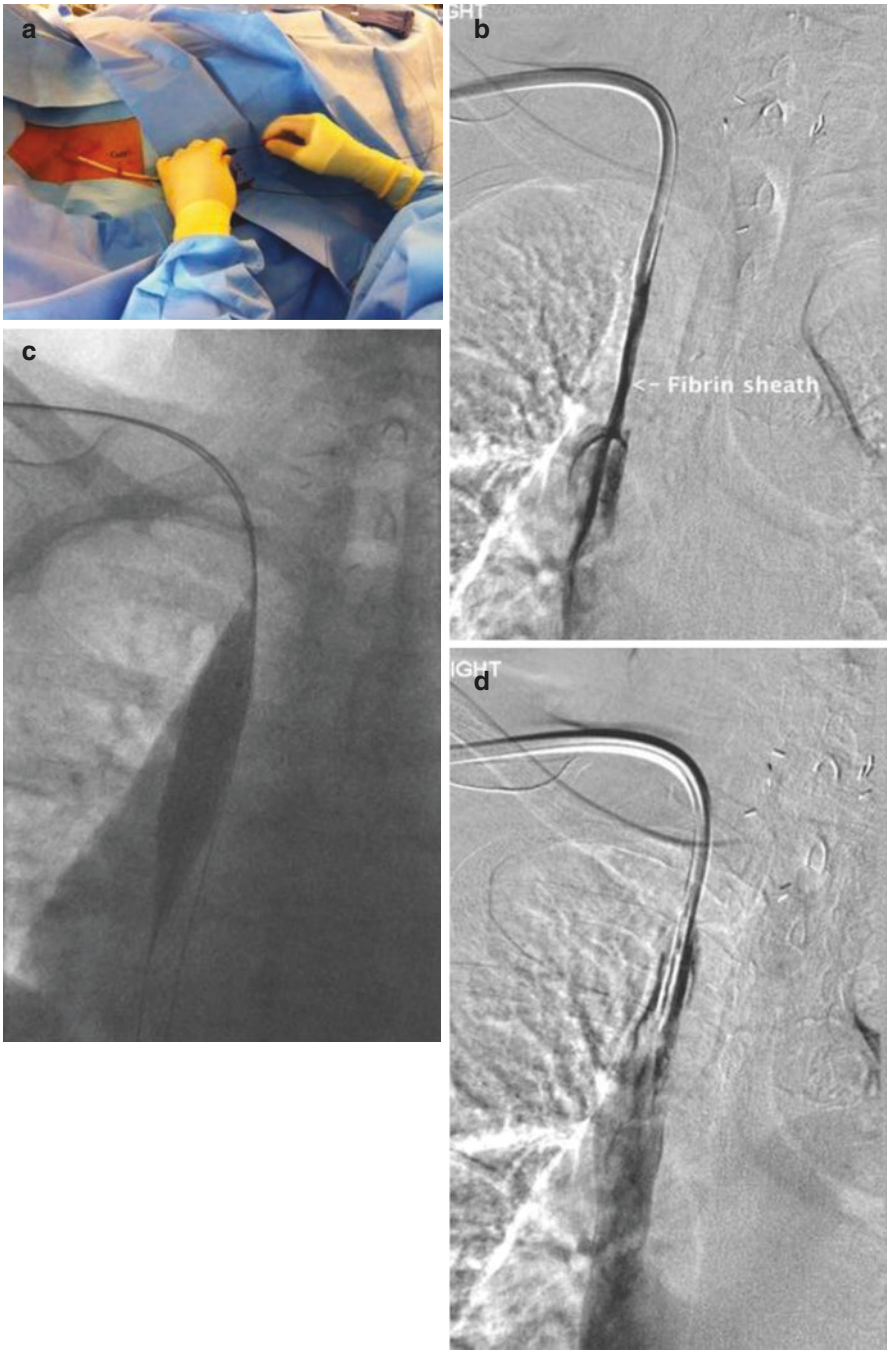

Fig. 21.12 (**a**) Insertion of guidewire into catheter. (**b**) The presence of fibrin sheath is demonstrated. (**c**) Disruption of fibrin sheath with angioplasty balloon. (**d**) Outcomes post angioplasty

Catheter-related Bloodstream Infections (CRBSI)

The approach to catheter-related bloodstream infection (CRBSI) is as shown in Fig. 21.13.

1. All patients with suspected dialysis catheter related systemic bacteremia should be treated with broad spectrum antibiotics until the blood culture results are known. Empiric antibiotics should include coverage for both gram-positive

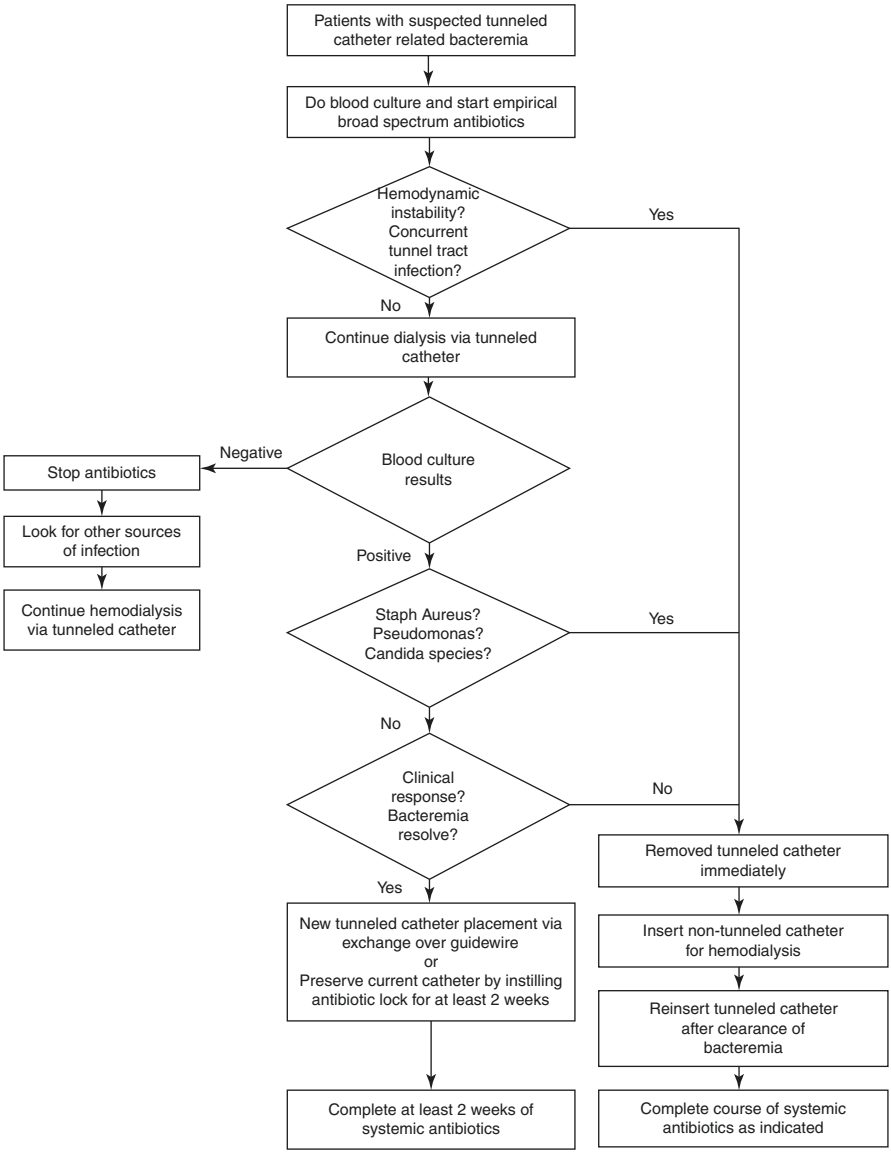

Fig. 21.13 Treatment algorithm for catheter related bacteremia

cocci and gram-negative rods, as gram-negative rods account for a significant proportion of CRBSI following the staphylococcus species. A loading dose of intravenous (IV) vancomycin 20 mg/kg followed by IV vancomycin 500 mg during the last 30 min of each subsequent dialysis session plus IV gentamicin 1 mg/kg or ceftazidime 1 g after each dialysis session is recommended by the Infectious Diseases Society of America. Antibiotic lock may also be used in conjunction with systemic antibiotics. However, there are concerns about developing antibiotic resistant organisms.

(a) If the patient is hemodynamically stable with no signs of systemic infection, the catheter can continue to be used while blood culture results are pending.
    (i) If the patient responds well to treatment and blood cultures grow organisms other than S. Aureus, Pseudomonas species or Candida species, a new catheter may be exchanged over a guidewire to replace the pre-existing catheter or the pre-existing catheter may be preserved with concurrent antibiotic lock therapy for at least 2 weeks.
    (ii) If the patient becomes hemodynamically unstable, has persistent symptoms, bacteremia persists despite IV antibiotics, blood cultures grow S. Aureus, Pseudomonas or Candida species, or metastatic infection develops, the tunneled catheter should be removed and reinserted after clearance of the bacteremia. A temporary catheter is inserted for dialysis access during this period.
    (iii) The total duration of antibiotics is 10–14 days and the patient can be managed as an outpatient if stable.

(b) If the patient is hemodynamically unstable or has a concurrent tunneled tract infection, the tunneled dialysis catheter should be removed immediately and reinserted after clearance of the bacteremia. A temporary dialysis catheter is required for dialysis access during this period.

Tunnel Tract Infection

1. Tunnel tract infection is defined as infection of the portion of the subcutaneous tunnel that extends between the catheter cuff and the venotomy site (Fig. 21.12). Exit site drainage should be collected for Gram staining and culture when present. Broad spectrum antibiotics targeting both gram-positive and gram-negative organisms are required accompanied by either a catheter exchange with a new subcutaneous tunnel or remove and place a new catheter at a new entry site. Treatment duration typically is 10–14 days in the absence of concurrent bacteremia (Fig. 21.14).

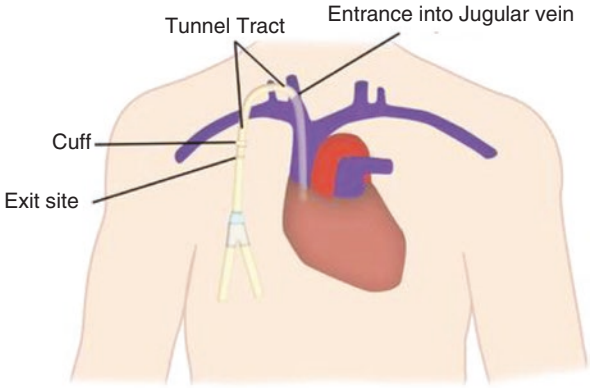

Fig. 21.14 Tunnel tract infection is defined as infection of the subcutaneous tunnel between the cuff and the entrance into the jugular vein. Exit site infection is usually superficial and does not involve the cuff

Exit Site Infection

1. Exit site infection is usually superficial and involves tissues distal to the catheter cuff, however, if it is left untreated, it may progress to become a tunnel tract infection with loss of catheter access. Exit site discharge should be collected for Gram staining and culture when present.
2. Depending on the severity of the infection, oral or systemic antibiotics may be used.
3. If the infection does not improve or progresses despite antibiotic therapy, exchange of the catheter with creation of new tunnel tract and exit site may be attempted.

Removal of Tunneled Dialysis Catheter

Introduction

Although tunneled catheters have lower infection rates compared to non-tunneled dialysis catheters, they should be removed once the patient's vascular access is ready for cannulation. Prolonged catheter usage is associated with the development of central vein stenosis, hence an effort should be made to minimize the duration of catheter use.

Removal of a tunneled dialysis catheter is generally a simple and straightforward procedure that can be performed in an outpatient office. Difficulties may be met if the cuff is located more than 2 cm from the exit site or when the cuff is "stuck down" by profound fibrosis, usually from prolonged catheter use.

Steps for Catheter Removal

1. Examine the patient. The cuff should be palpable approximately 2 cm from the exit site (Fig. 21.15a). The purse string stitch may be present if the catheter was placed recently.

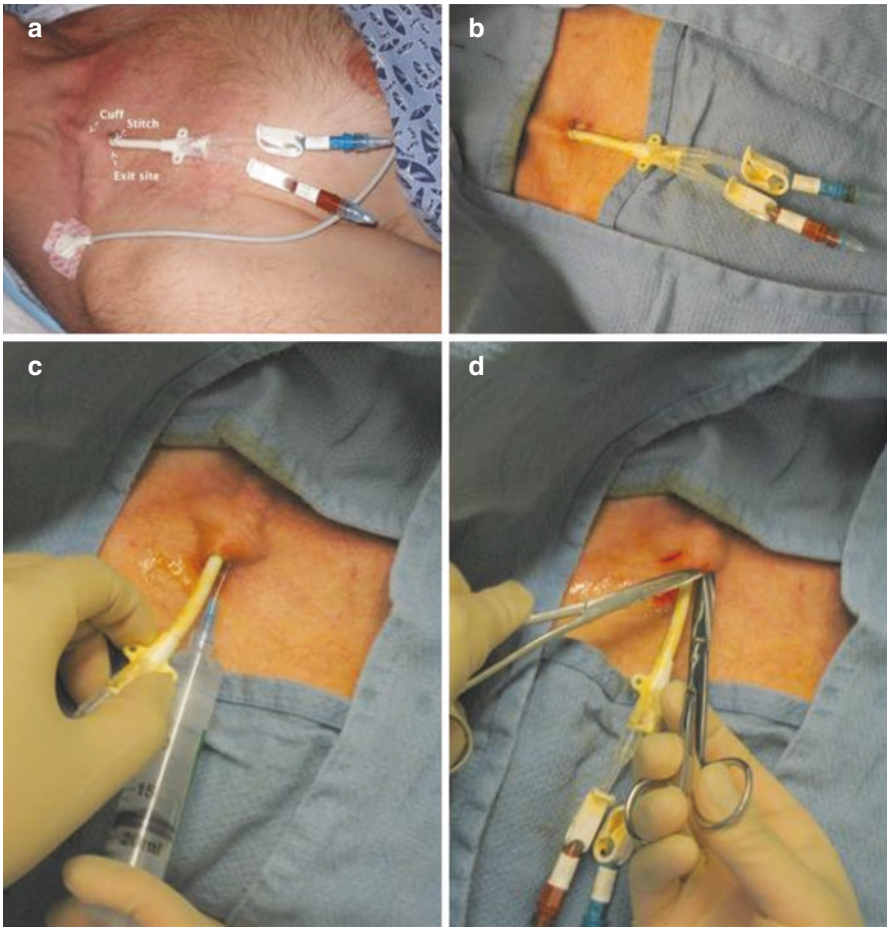

Fig. 21.15 (**a**) Physical examination of the patient. (**b**) Clean and drape the patient. (**c**) Anesthetize the exit site and cuff. (**d**) Remove any stitches that are present

2. Clean and drape the patient (Fig. 21.15b). Patient should be in the Trendelenburg position for tunneled catheter removal.
3. Inject lidocaine at the exit site and around the cuff (Fig. 21.15c). The hydrostatic pressure generated by the injection around the cuff will help to separate it from the surrounding tissue.
4. Remove any stitches that are present (Fig. 21.15d).
5. Using a combination of gentle traction and blunt dissection with a hemostat, separate the cuff from the surrounding tissue (Fig. 21.16a–b).
6. The cuff is the only part of the catheter that is tethered to the body. Once it is free, the catheter can be easily removed (Fig. 21.16c). Compress the internal jugular vein while removing the catheter.

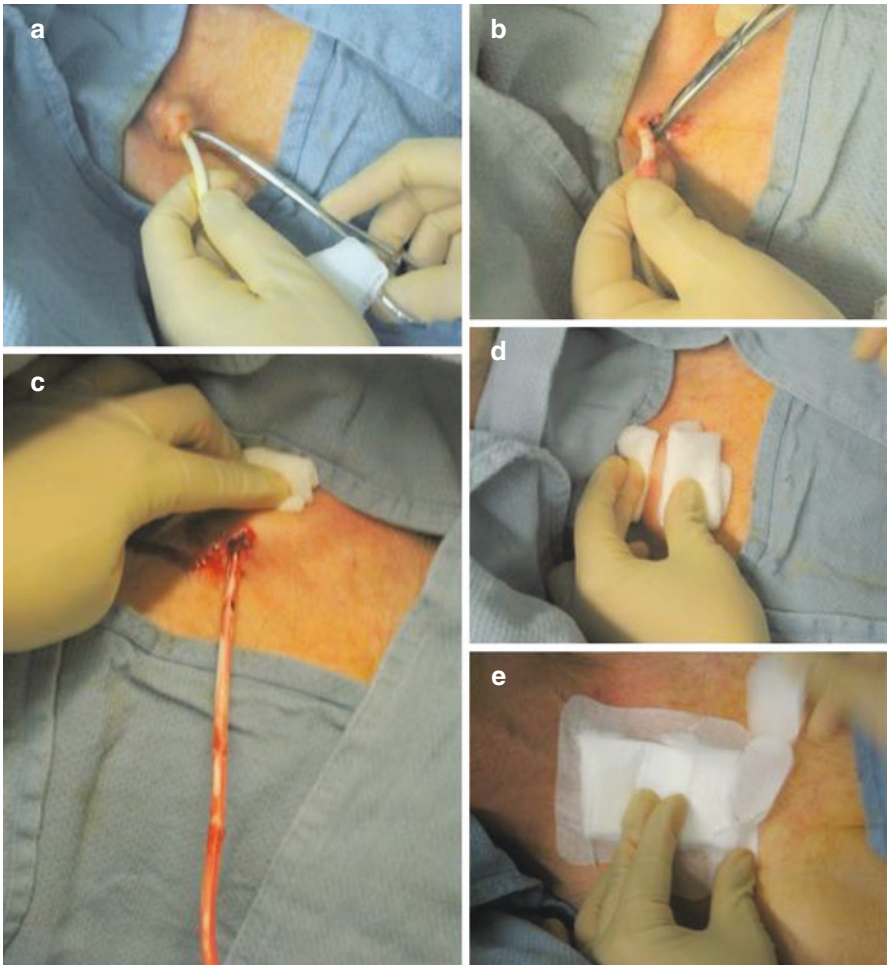

Fig. 21.16 (**a**) Blunt dissection around the cuff using an artery forceps. (**b**) Gentle traction to free the cuff. (**c**) Removal of the catheter. (**d**) Secure hemostasis by direct compression. (**e**) Wound dressing

7. Compress the internal jugular vein at the root of the neck after removal for 3–5 min (Fig. 21.16d).
8. Observe for any bleeding complications after removal.
9. No stitches are usually required. Cover the wound with dressing (Fig. 21.16e).

Table 21.2 Potential problems and troubleshooting

Problem	Troubleshooting
Cuff is placed too far from the exit site. Unable to reach cuff with hemostat	Make a small incision over the cuff and do blunt dissection through the incision to free up the cuff
Cuff became separated from the catheter and was left behind in the subcutaneous tunnel after removal of the catheter	Palpate for the cuff in the subcutaneous tunnel. Make a small incision over the cuff. Using a hemostat and forceps, remove the cuff using blunt dissection techniques

Some of the potential problem and steps for troubleshotting are summarised in Table 21.2.

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Surgical Placement of Hemodialysis Vascular Accesses

22

Shouwen Wang and James F. Markmann

Introduction

The goal of surgical creation of an arteriovenous vascular access is to produce a conduit that can be easily cannulated repeatedly for hemodialysis therapy. Because of their reduced complication rates and improved efficiency, arteriovenous fistulas (AVF) and arteriovenous grafts (AVG) are preferred vascular accesses for hemodialysis over catheters [1].

Given their reduced complication rates and longevity over AVGs, AVFs should be considered first line therapy in all patients needing hemodialysis vascular accesses (Table 22.1) [1]. However, the choice between AVF and AVG for a particular patient may be influenced by many factors. The availability of suitable vasculature is a crucial factor to consider. Other factors to consider are: the patient's general health and life expectancy, whether the patient is on dialysis or close to needing dialysis, obesity, and others [2]. Creating a usable AVF may require weeks or even months, and sometimes endovascular interventions or secondary surgery may be required to promote its maturation. An AVG can be cannulated soon after placement, thus may potentially minimize catheter-depend duration if a patient is already on hemodialysis or eliminate the need for a catheter if a patient needs to be started on dialysis non-urgently. However, an AVG generally requires more maintenance interventions and does not last as long as an AVF.

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Table 22.1 Comparison of AVF and AVG

	AVF	AVG
Indications	Usually should be considered first and preferred for most patients	When AVF not practical, or may be preferred in some patients
Configuration	One anastomosis: Artery to adjacent vein or transposed vein	Two anastomoses: Artery to graft and graft to vein
Blood vessel requirements	Usually artery ≥2 mm and vein ≥2.5 mm in diameters	Require larger vessels for anastomoses
Surgical technical difficulty	Moderate	Higher than AVF
Time required before usable	Weeks to months, may need interventions to facilitate maturation	Days to 2–3 weeks
Timing of placement	May be created in advance of needing dialysis. Need time to mature, lower maintenance requirements	Before needing dialysis. Need short time before usable, but higher complications and maintenance requirements
Early postoperative complications	Low	Much higher than AVF
Incidence of distal ischemia (steal syndrome)	Low	Several fold higher than AVF
Access flow rate	Variable, remain patent at low flow rate, more likely to develop flow >2 L/min and related complications	Moderate, more likely to clot if flow <600 ml/min
Incidence of thrombosis	Low	High
Incidence of aneurysms	Moderate	Pseudoaneurysms, lower than AVF
Potential infection	Low	Several fold higher than AVF
Longevity	Usually long	<AVF, generally less than a few years
Overall preference	Preferred	<AVF, >catheter

Surgical creation of AVF and AVG can be safely performed in an outpatient setting or same-day surgical suite of a hospital. Although general anesthesia or regional nerve block has often been employed for these surgical procedures, local anesthesia plus conscious sedation generally provides sufficient comfort for patient going through these procedures. One advantage of conscious sedation is that nerve injury can be minimized as the patient can still respond to nerve stimuli during surgery. The following sections describe surgical procedures performed under local anesthesia plus conscious sedation.

While similar surgical skills and techniques can be used for creation of AVF and AVG, there are substantial differences in how these procedures are performed. Generally, the placement of an AVG is technically more difficult and time-consuming than an AVF creation. For clarity, the creation of AVF and placement of AVG are illustrated separately in the following sections.

Creation of Autogenous Arteriovenous Fistulas

Native vessels are utilized to create AVFs. Although many blood vessels may be used for AVF creation (Table 22.2), the blood vessels in the upper extremity are most often chosen for their convenient locations and higher success rate. The generally accepted criteria for fistula creation are an artery ≥2 mm and a vein ≥2.5 mm in diameters at the anastomosis sites [3]. Additionally, sufficient diameters of the feeding artery and draining veins along their paths are essential. In younger patients or patients with limited vasculature, smaller vessels may be exploited to create an AVF.

Pneumatic tourniquet has been employed to assist AVF creation. It can reduce procedure time, minimize required dissection, reduce vascular trauma by eliminating vascular clamps and potentially improve the outcomes of surgical procedures of hemodialysis access [4, 5]. It may be utilized in over 90% of the patients for fistula creation based on our experience, but this practice varies widely between surgeons and institutions. The following section describes the creation of a brachiocephalic fistula assisted by pneumatic tourniquet.

Table 22.2 Configurations of autogenous AV fistulas [3]

Arteries	Veins	Comments
Posterior radial branch	Cephalic	Snuffbox, uncommon
Radial	Cephalic	Most common forearm
Radial	Transposed forearm cephalic	May be considered in obese patients.
Brachial/PRA	Transposed forearm cephalic (loop)	Less common
Radial	Transposed forearm basilic	Less common
Ulnar	Transposed forearm basilic	Less common
Brachial or PRA	Transposed forearm basilic (loop)	Less common
Brachial/PRA	Cephalic	Most common
Brachial	Transposed upper cephalic	Uncommon, when cephalic far away from brachial artery
Brachial/PRA	Upper basilic	Common, needs one- or two- stage transposition
Brachial/PRA	Brachial	Uncommon, need two-stage transposition
Brachial/PRA	Median antebrachial or perforating vein	Bidirectional flow, not a first-line choice
Radial or brachial	Translocated saphenous	Rare
Femoral	Transposed saphenous	Uncommon
Femoral	Transposed femoral	Uncommon
Posterior tibial	Great saphenous	Uncommon

PRA proximal radial artery

Equipment for AVF Creation (Fig. 22.1)

1. Surgical optical loops (2.5×)
2. Supplies for sterilization and draping of extremities, gowns, gloves
3. Normal saline for flushing vessels and incision
4. Syringes (10 and 20 ml), needles (25 gauge), and heparin tip
5. 1% Lidocaine solution (without epinephrine)
6. Gauze (4″x4″) and Lap sponges
7. Surgical blades (No. 15 for skin; No. 11 for arteriotomy and venotomy)
8. Scissors (Tenotomy scissors for tissue dissection; suture scissors)
9. Forceps (DeBakey for skin; Gerald for blood vessels and finer tissue)
10. Electrocautery unit, grounding pad and monopolar handswitch pencil
11. Vessel clips and clip appliers (small and medium, Ethicon™)
12. Vacuum suction equipment, tubing and tips (Yankauer and Frazier)
13. Retractors (Weitlaner with dull jaw; Senn)
14. Kelly hemostats, right-angle hemostat, and needle holder
15. Vascular loops (round elastic rubber band for holding vessels)
16. Potts scissors
17. Garrett vessel dilators/probes (1, 1.5, 2, 2.5 mm in diameters)
18. Castro needle holder
19. 3–0 sutures for skin (Vicryl ™—absorbable or Prolene ™—non-absorbable)
20. Sterile strips
21. Bulldog and Glover Bulldog vessel clamps
22. Pneumatic tourniquet system
23. Esmark elastic bandage
24. Non-absorbable vessel sutures with taper-point needles (CV-8 Gore-Tex™ and 7–0 Prolene ™)

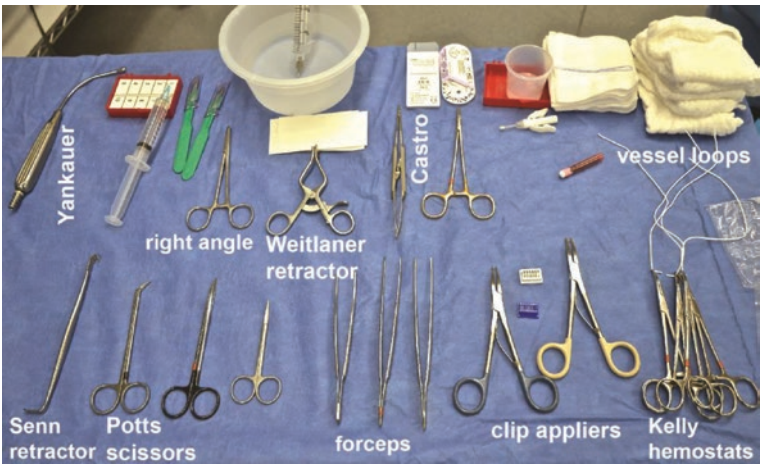

Fig. 22.1 Equipment for AVF creation. Main sterile tools used during AVF surgery

Steps of AVF Creation (Brachiocephalic AVF)

1. Preoperatively, the patient is examined and relevant history is reviewed.
2. Blood vessels on the chosen arm are visualized with ultrasound (Fig. 22.2) and a surgical plan is formulated. Locations of the vessels for anastomosis are marked.
3. The surgical instruments are organized on a sterile table (Fig. 22.1).
4. A pneumatic tourniquet cuff is applied on the upper arm and connected to the control unit system (Fig. 22.3).
5. The arm is prepared and draped to create a sterile surgical field (Fig. 22.4a).
6. Sedation medications (midazolam and fentanyl) are given intravenously. The starting doses of these medications need to be tailored to an individual patient. A dedicated nurse monitors continuous electrocardiogram, pulse oximetry, and intermittent blood pressure.
7. Local anesthetic (1% lidocaine) is infiltrated along the incision site and around the vein that will be dissected.
8. The arm is exsanguinated with Esmark bandage and the pneumatic tourniquet cuff is inflated to preset pressure (Fig. 22.4b). Please refer to a recent review for further discussion of tourniquet use [4].
9. A longitudinal incision is made over the distal brachial artery near the elbow crease. Blunt and sharp dissections are carried out laterally to free 2–3 cm of

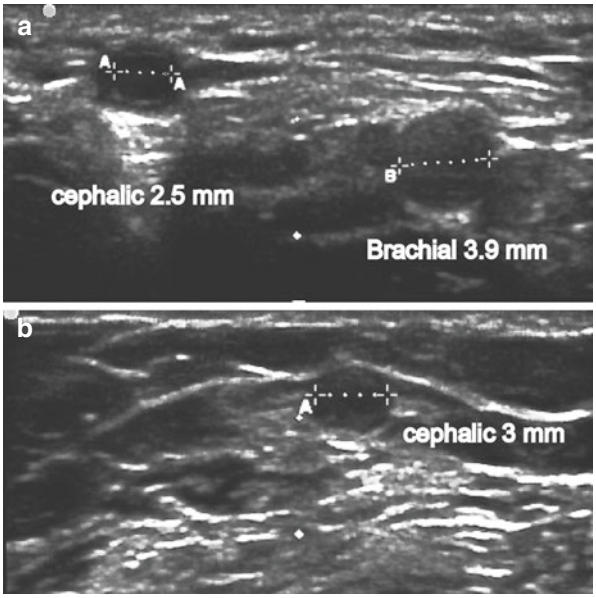

Fig. 22.2 Preoperative evaluation before AVF creation. Preoperative ultrasound images reveal (**a**) cephalic vein (*left*) and adjacent brachial artery (*right*) at the elbow of a right arm with venous tourniquet applied; (**b**) upper arm cephalic vein

Fig. 22.3 Utilizing an automated pneumatic tourniquet system. The system consists of inflatable cuff, connection tubing and pressure control device. The selection of tourniquet cuff (Panel **a**) is based on the size and shape of a patient's limb and the location of surgical site. Notice the contoured conical shape of the two larger cuffs. The standard cuff width is 14 cm for the upper arm (second right). The cuff is connected to the pressure control device via connection tubing (Panel **b**). The cuff is applied on the upper arm over a double-layered stretchable protective sleeve (stockinet) to prevent injury to the underlying skin (Panel **c**) [4]

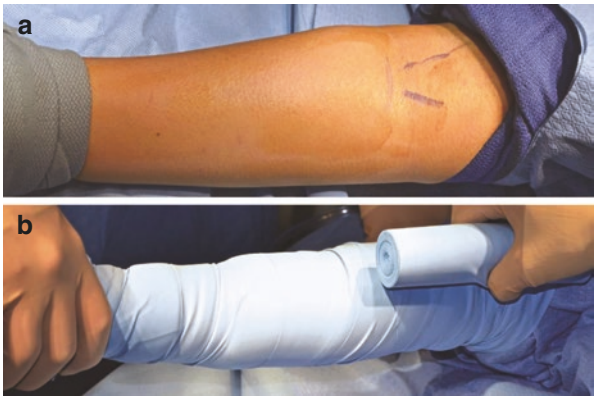

Fig. 22.4 Preparation of the arm for AVF surgery. (**a**) Prepared arm ready for surgery. Notice the markings on the skin to indicate location of cephalic vein and brachial artery based on preoperative ultrasound evaluation. (**b**) The arm is exsanguinated with Esmark elastic bandage and the pneumatic tourniquet is then inflated

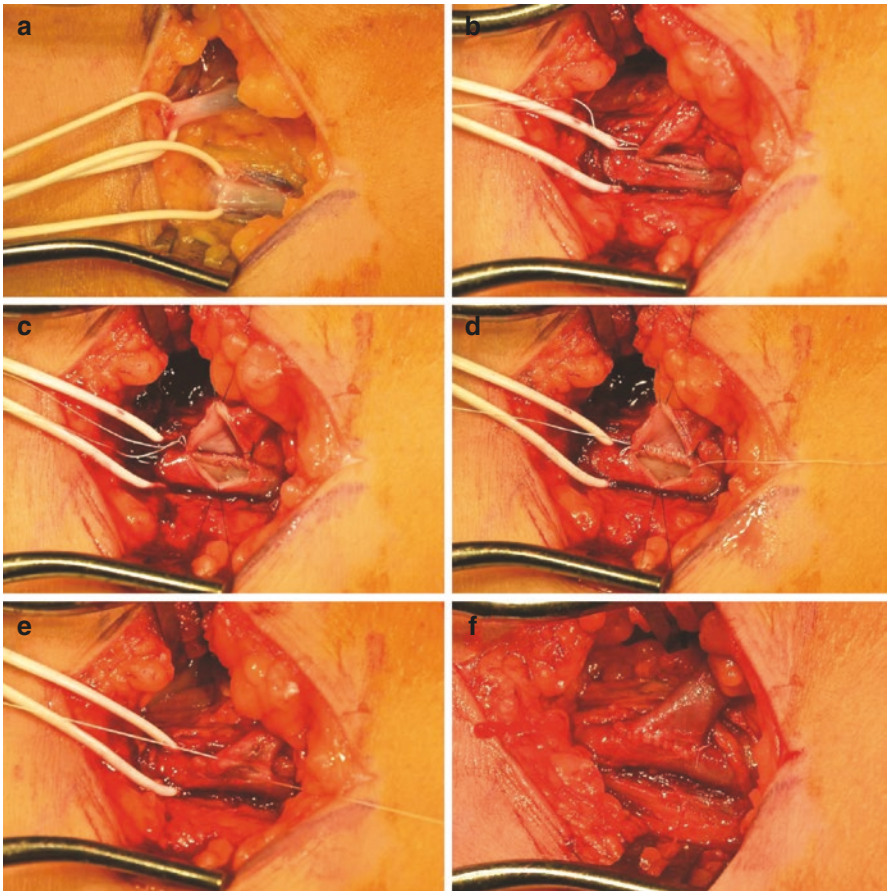

Fig. 22.5 Exposure of vessels and arteriovenous anastomosis. (**a**) The cephalic vein (*upper*) and the brachial artery (*lower*) are isolated. (**b**) The transected cephalic vein is anchored to the toe-corner of the longitudinal arteriotomy on the brachial artery with a CV-8 Gore-Tex™ suture. Care should be taken not to produce torsion on the cephalic vein. (**c**) The heel-corner of the anastomosis is anchored with 7–0 Prolene™ suture. (**d**) The posterior anastomosis is finished with the CV-8 Gore-Tex™ suture in a continuous fashion. (**e**) The CV-8 Gore-Tex™ suture is used to continue suturing the corners of the anastomosis. (**f**) Finished brachial artery to cephalic vein anastomosis with established flow through the anastomosis

the cephalic vein from surrounding tissues. Blunt and sharp dissections are carried out posteriorly through the bicipital aponeurosis. The distal brachial artery is exposed (Fig. 22.5a). Care should be taken to preserve the adjacent nerves and vessels.

10. The distal cephalic vein is clipped or ligated, and the cephalic vein is transected. The cephalic vein is swung toward the brachial artery. There should be no laxity or excessive tension between the end of the cephalic vein and the side of the brachial artery. The cephalic vein may be gently dilated with saline flush.

A 4–6 mm arteriotomy is made on the anterior aspect of the brachial artery using a No. 11 blade followed by Potts scissors (Fig. 22.5b). Care is taken not to damage the posterior wall of the brachial artery.

11. The toe corner of the anastomosis is anchored with a CV-8 Gore-Tex™ or 7–0 Prolene™ suture (Fig. 22.5b). Attention is paid that the cephalic vein is not twisted. Some lateral rotation of the cephalic vein is desirable when it is swung toward the brachial artery in order to avoid twisting [6]. The heel corner of the anastomosis is anchored with a 7–0 Prolene™ suture to assure good alignment between the end of the cephalic vein and arteriotomy (Fig. 22.5c). The anterior aspects of the cephalic vein and brachial artery are pulled open with a 7–0 Prolene™ suture to expose the posterior edges of the cephalic vein and the arteriotomy (Fig. 22.5c). The posterior anastomosis is accomplished with the CV-8 suture in a continuous over-and-over fashion (Fig. 22.5d). The remaining anastomosis is completed using the continuous CV-8 suture. Several passes of the suture are required to turn the corners of the anastomosis (Fig. 22.5e). The suture is tied snugly, avoiding "purse-string effect" on the anastomosis.
12. The pneumatic tourniquet is released. Now blood flow through the fistula is established (Fig. 22.5f). Check the anastomosis for any significant leaks. Mild oozing at the anastomosis often will stop spontaneously. Any significant leakage at the anastomosis may be repaired with interrupted CV-8 suture. Subcutaneous fat may be sutured to the leakage site to facilitate hemostasis. Palpate the fistula for a smooth thrill. Check the proximal cephalic vein to release any tissue compression.
13. Palpate radial pulse if it is palpable preoperatively. Unwrap the hand and check oxygenation on the fingers using an oximeter. Good plethysmography wave and oxygen saturation well above 90% indicate sufficient hand circulation (Fig. 22.6).
14. Check the incision to assure good hemostasis. The incision is closed with 3–0 Vicryl sutures in layers: subcutaneous and subcuticular (Fig. 22.6d). Sterile strips and dressings are applied, or a skin glue such as Dermabond™ is utilized.

Placement of Prosthetic Arteriovenous Grafts

For some patients, AVG placement may be required (such as in a patient with exhausted superficial veins for AVF) or preferred (such as a patient with a life expectancy <2 years and already on hemodialysis). Because of the increased technical difficulty of anastomosing vessels to synthetic grafts and higher blood flow rate required to maintain AVG patency, larger arteries and veins are needed for the anastomoses with AVG. The generally accepted criteria for graft placement are an artery ≥2 mm (radial or ulnar arteries) and a vein ≥4 mm in diameters at the anastomosis sites [3]. Given the increased incidence of steal syndrome when distal brachial artery is used for access inflow, a larger diameter of brachial artery is preferred (≥4 mm, or ≥ 3 mm if 4-mm tapered graft is used). The most commonly used hemodialysis grafts are made of synthetic ePTFE (expanded polytetrafluoroethylene). Biological grafts, such as decellularized bovine heterografts, are also commercially available [7]. These biological grafts are more expensive than synthetic grafts, but some data suggest that they

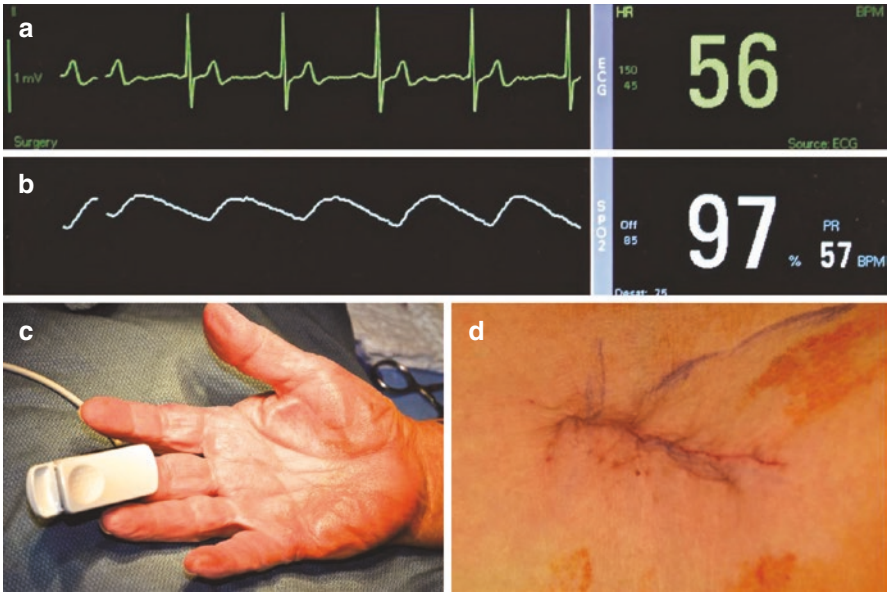

Fig. 22.6 Evaluation of distal circulation and closure of the incision. The distal circulation is evaluated before skin closure. (**a**) Electrocardiographic monitoring. (**b**) Corresponding plethysmography wave and oxygen saturation reading through the probe on a digit of the operated arm (**c**). (**d**) Closed skin incision

Table 22.3 Configurations of prosthetic AV grafts [3]

Arteries	Veins	Comments
Radial	Antecubital (straight)	Less common
Brachial	Antecubital (forearm loop)	Common
Brachial	Axillary, basilic, brachial	Most common
Axillary/ brachial	Axillary, basilic, brachial (upper arm loop)	Less common
Femoral	Femoral (thigh loop)	When arm not suitable
Axillary	Contralateral axillary (chest wall, necklace)	Rare, when extremity access exhausted
Axillary	Ipsilateral axillary (chest loop)	Rare, when extremity access exhausted
Axillary	Ipsilateral internal jugular (chest loop)	Rare, when extremity access exhausted
Axillary	Femoral (body wall)	Rare, when extremity access exhausted

have superior clinical outcomes than synthetic grafts [8]. A graft may be placed at various locations with various configurations (Table 22.3). The following section describes the placement of a 6-mm synthetic AVG in the upper arm in a typical configuration.

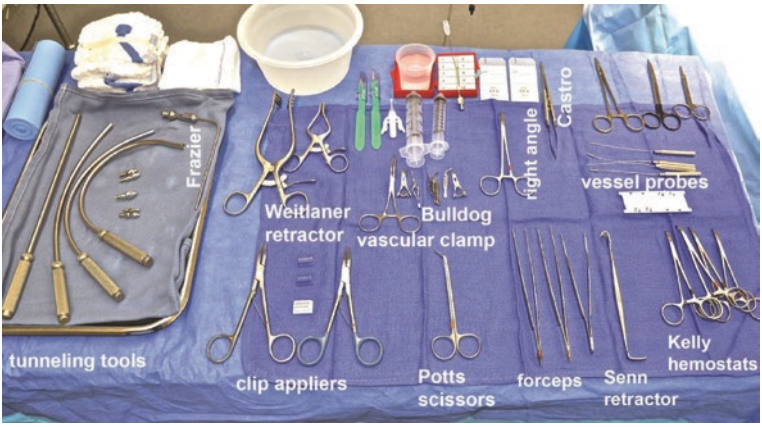

Fig. 22.7 Equipment for AVG placement. Main sterile tools used during AVG surgery

Equipment for AVG Placement (Fig. 22.7)

1. 1–20 of equipment for AVF creation
2. Tunneling tools
3. Graft for hemodialysis (ePTFE—expanded polytetrafluoroethylene)
4. DeBakey vascular clamp
5. Non-absorbable vessel sutures with taper-point needles (CV-6 Gore-Tex™ or 6–0 Prolene ™)

Steps of AVG Placement (Brachiobasilic AVG)

1. Preoperatively, the patient is examined and relevant history is reviewed.
2. Blood vessels on the chosen arm are visualized with ultrasound and surgical plan is formed. Locations of the vessels for anastomosis with the graft are marked (Fig. 22.8).
3. Prophylactic antibiotics (2 g of cefazolin or 1 g of vancomycin) are administered preoperatively.
4. The surgical instruments are organized on a sterile table (Fig. 22.7).
5. The arm is prepared and draped to create a sterile surgical field (Fig. 22.8).
6. Sedation medications (midazolam and fentanyl) are given intravenously tailored to an individual patient. Continuous electrocardiogram, pulse oximetry, and intermittent blood pressure are monitored.
7. Local anesthetic (1% lidocaine) is infiltrated along the incision sites.
8. An oblique incision is made over the proximal basilic vein using a No. 15 blade. Blunt and sharp dissections are carried out to free 2–3 cm of the proximal basilic vein from surrounding tissues. A vessel loop is passed around the vein

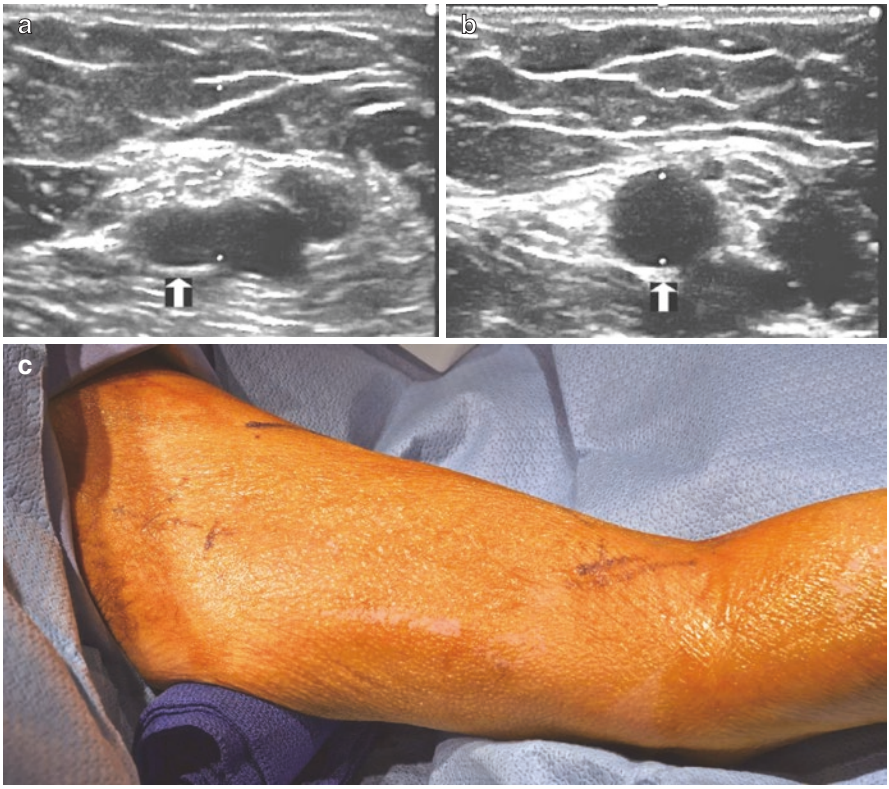

Fig. 22.8 Preparation of the arm for AVG surgery. (**a**) Preoperative ultrasound image of the proximal basilic vein (*arrow*) adjacent to the proximal brachial artery. (**b**) Ultrasound image of the brachial artery (*arrow*) above the elbow crease. (**c**) Prepared arm ready for AVG surgery. Notice the markings on the skin planned based on preoperative ultrasound evaluation

to assist handling. Care should be taken to preserve the adjacent nerves and vessels (Fig. 22.9).

9. A longitudinal incision is made over the distal brachial artery above the elbow crease. Blunt and sharp dissections are carried out to free 2 cm of the distal brachial artery from surrounding tissues (Fig. 22.9).
10. Local anesthetic (0.5% lidocaine) is infiltrated subcutaneously along the intended graft path using a spinal needle.
11. A tunneling tool with proper curvature and a tip similar to the diameter of the graft is passed subcutaneously from the distal incision to the proximal incision. Attention is made that the tunneling tool is easily palpable from the skin so that the graft will be superficial enough for future cannulation (Fig. 22.10a).
12. The graft is attached to the tunneling tool and pulled through the subcutaneous tunnel (Fig. 22.10b). A graft of 6 mm in diameter or 7 mm in diameter with a 4 mm diameter taper toward the arterial end is typically chosen.

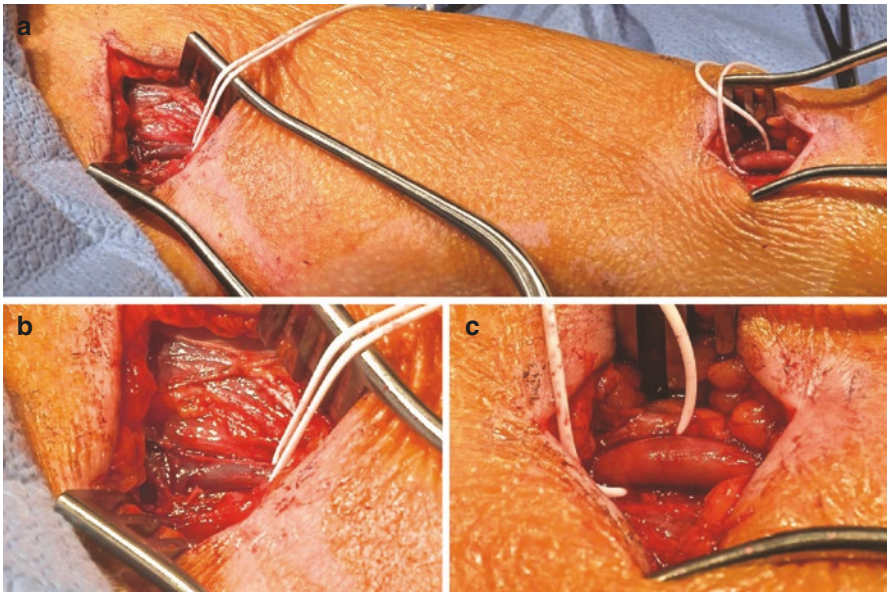

Fig. 22.9 Exposure of the vessels for graft anastomosis. (**a**) Two incisions on the upper arm for exposing the vessels (identified with vessel loops). (**b**) Enlarged view of the exposed proximal basilic vein. (**c**) Enlarged view of the exposed distal brachial artery

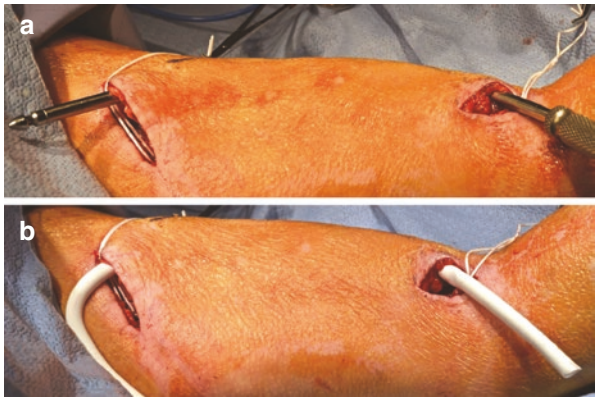

Fig. 22.10 Tunneling of graft. (**a**) A tunneling tool is passed subcutaneously between the incisions. (**b**) The graft is pulled through the tunnel with the tunneling tool

13. The graft is trimmed to the proper length for anastomosis. A somewhat oblique trim of the venous end of the graft (beveled) is desirable to increase the circumference of the anastomosis. The venous anastomosis is typically performed first so that the flow through the graft will be immediately established after finishing the arterial anastomosis and there will be no excessive pressure buildup at the arterial anastomosis.
14. Two Bulldog clamps are applied on the basilic vein proximally and distally to control blood circulation. A longitudinal venotomy is made on the basilic vein

using a No. 11 blade followed by Potts scissors to match the graft end. Care is taken not to damage the posterior wall. The anastomosis is accomplished using an over-and-over continuous CV-6 Gore-Tex™ suture. The suturing is started at the toe or heel of the anastomosis. The use of parachute suture technique will enable accurate placement of sutures. The remaining anastomosis is completed using the continuous suture. The suture is tied snugly, avoiding "purse-string effect" on the anastomosis (Fig. 22.11a, b).

15. The Bulldog clamps are removed. The graft is flushed with saline from the arterial end. A DeBakey vascular clamp is applied on the graft to prevent back bleeding. Mild oozing at the anastomosis often will stop spontaneously. Any significant leakage at the anastomosis may be repaired with interrupted CV-6 suture. Subcutaneous fat may be sutured to the leakage site to facilitate hemostasis.
16. Two Glover Bulldog clamps are applied on the brachial artery proximally and distally to control blood circulation. A longitudinal arteriotomy is made on the brachial artery using a No. 11 blade followed by Potts scissors to extend the arteriotomy to 6 mm in length. Care is taken not to damage the posterior wall. The anastomosis is accomplished using an over-and-over continuous CV-6 Gore-Tex™ suture. The suturing is started with the posterior anastomosis. The use of parachute suture technique will enable accurate placement of sutures. The remaining anastomosis is completed using the continuous suture. The

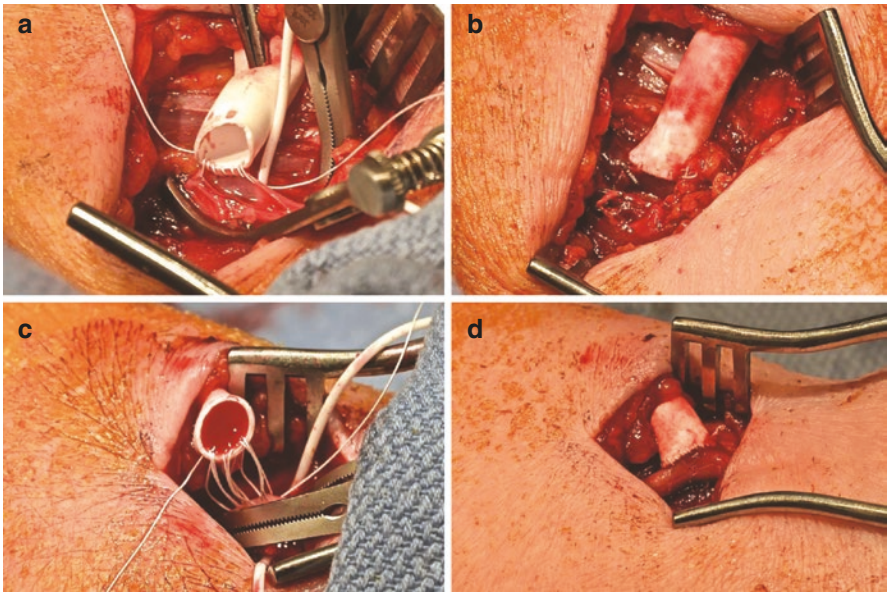

Fig. 22.11 Graft-vessel anastomoses. (**a**) Graft-basilic vein anastomosis—suturing the posterior anastomosis with CV-6 Gore-Tex™ suture. (**b**) Finished graft-basilic vein anastomosis. (**c**) Graft-brachial artery anastomosis—suturing the posterior anastomosis with CV-6 Gore-Tex™ suture using the parachute technique. (**d**) Finished graft-brachial artery anastomosis

suture is tied snugly, avoiding "purse-string effect" on the anastomosis (Fig. 22.11c, d).

17. The DeBakey vascular clamp on the graft is released first, followed by releasing the Bulldog clamps on the brachial artery. Now blood flow through the graft is established. Check the arterial and venous anastomoses for any significant leaks. Palpate the graft for a smooth thrill.
18. Palpate radial pulse if it is palpable preoperatively. Unwrap the hand and check oxygenation on the fingers using oximeter. Good plethysmography wave and oxygen saturation well above 90% indicate sufficient hand circulation (Fig. 22.12).
19. Check the incisions to assure good hemostasis. The incisions are closed with 3–0 Vicryl sutures in layers: subcutaneous and subcuticular (Fig. 22.13). Sterile strips and dressings are applied, or a skin glue such as Dermabond™ is utilized.

Tips and Troubleshooting

Potential issues and complications that may be encountered during and shortly after surgical creation of AVF and AVG are discussed in Table 22.4.

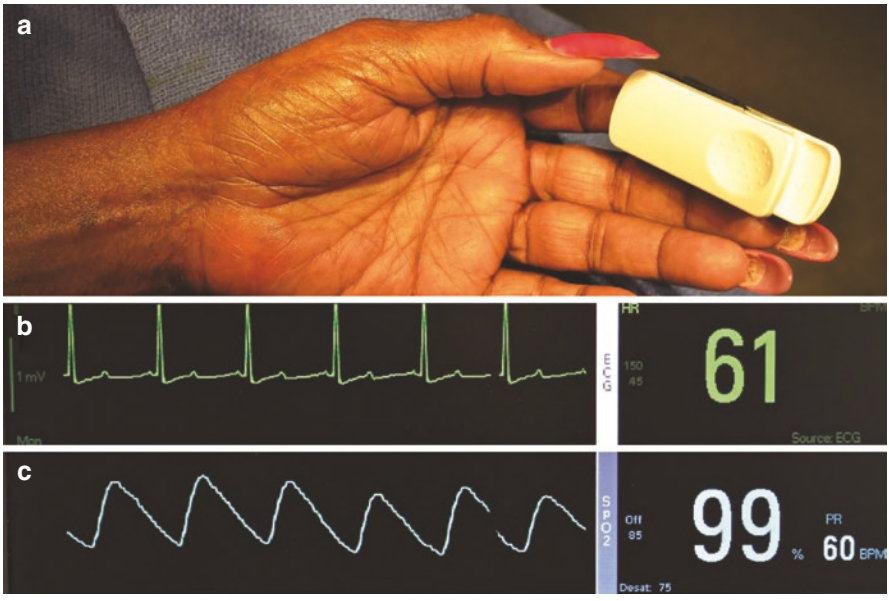

Fig. 22.12 Evaluation of distal circulation. (**a**) A pulse oximetry probe is placed on a finger. (**b**) Electrocardiographic monitoring. (**c**) Corresponding plethysmography wave and oxygen saturation reading

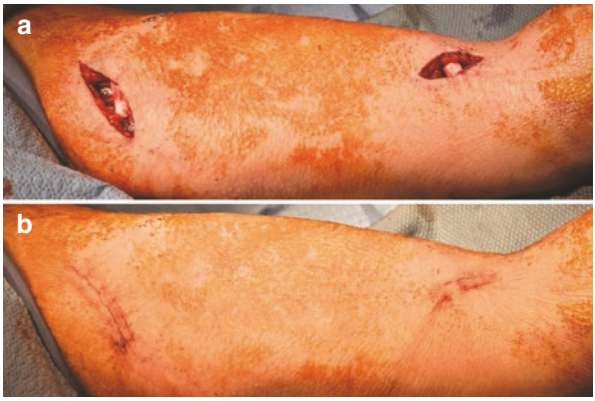

Fig. 22.13 Closure of the incisions. (**a**) Before closure of the incisions. (**b**) After closure of the incisions

Table 22.4 Potential issues and complications

Problem	Troubleshooting
Prophylactic antibiotics	Prophylactic antibiotics are not required for most AVF creation. A single preoperative dose of an antibiotic with gram-positive coverage is standard of care for clean surgical cases. Prophylactic antibiotics are essential given the more extensive nature of the AVG placement and the introduction of prosthetic graft material [9]
Intraoperative heparin	There is no established role of intraoperative intravenous heparin during surgical creation of vascular accesses. However, in patients with history of thrombogenic disorders or early fistula failure, intraoperative heparin may potentially be helpful [9]
Leaks at the anastomosis	Minor oozing at the anastomosis usually stops a few minutes after completing the anastomosis. Significant leaks need to be suture repaired. An interrupted suture or U-shaped suture is typically sufficient. For larger leaks, especially at the corners, a piece of subcutaneous fat may be applied over the leaking hole when additional sutures are applied
Distal ischemia: During surgery	When arteries proximal to the radial artery are used as inflow, the arteriotomy needs to be limited to 4–6 mm in length. Also, the arterial anastomosis is completed with a continuous suture to limit excessive future increase of the anastomosis. These maneuvers decrease the incidence of steal syndrome and future development of excessive access flow [3]. After completion of the arterial anastomosis, it is recommended that hand circulation be evaluated before incision closure. A simple approach is to check the plethysmography and digital pulse oximetry after the supplemental oxygen is turned off. If the oxygen saturation is below 90% and the plethysmography wave is flat, insufficient distal circulation is suggested. The fistula or graft may be compressed to see if the oxygen saturation and wave recovers. A simple technique to enhance the distal flow is to band down the fistula vein or graft with vascular clips (Fig. 22.14). A return of oxygen saturation to >90% and reliable plethysmography wave suggest sufficient distal circulation

Problem	Troubleshooting
Ischemic monomelic neuropathy (IMN)	IMN is a rare early complication of vascular access surgery. The key feature of IMN is the presence of neurological deficits in the absence of circulatory insufficiency. Timely recognition is crucial and the AVF or AVG needs to be ligated immediately [3]
AVF creation without tourniquet	For AVF creations without pneumatic tourniquet, bulldog clamps or vessel loops need to be applied on the vein and artery to control blood circulation before performing the anastomosis
Metal clips for graft-venous anastomosis	Besides sutures, specially designed clips may also be used to complete the graft-venous anastomosis. Limited study suggests that they may be associated with reduced anastomosis stenosis [10]

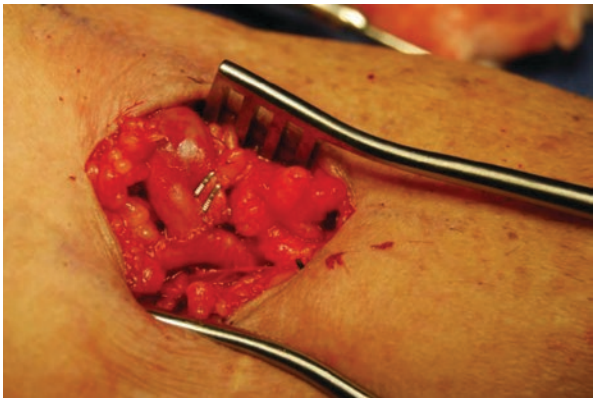

Fig. 22.14 Reduction of fistula vein near the arterial anastomosis with clips

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Endovascular Creation of Arteriovenous Fistulas

23

Christine Chen, Marcin Kolber, Ahmed Kamel Abdel Aal, and Harold Park

Introduction

The Centers for Medicare and Medicaid services (CMS) first started the National Vascular Access Improvement Initiative (NVAII) in 2003 and the Fistula First Breakthrough Initiative (FFBI) resulted in 2005. The initiative was developed to promote arteriovenous fistula (AVF) creation in patients requiring long-term hemodialysis. AVFs have been shown to demonstrate greater patency rates and fewer complications than dialysis grafts. The National Kidney Foundation (NKF) first published the Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines in 1997 addressing the management of vascular access in patients requiring hemodialysis. Subsequent updates in the guidelines have occurred in 2000, 2006 and as recently as 2019. The 2006 updated guidelines, which provide the most recent recommendations on vascular access, recommend the surgical creation of AVFs of the upper extremity occurring first in the distal arm followed by more proximal sites. To this effect, a radiocephalic fistula in the wrist is preferred over an elbow brachiocephalic fistula or, lastly, a transposed brachiobasilic fistula [1].

Traditional, surgically-created AVFs usually fall into one of three configurations: radiocephalic, brachiocephalic, or brachiobasilic. Surgical arteriovenous fistulas (SAVFs) have a reported maturation failure rate of 20–60% [2], and acute thrombosis rate of up to 20% [3]. Additional information provided by the United States Renal Data System (USRDS) in 2018, reported that between primary surgical failures and maturation failures, 39% of AVFs created between June 2014 and May 2016 in the United States were unsuccessful [4]. Forearm radiocephalic AVFs were shown to have higher primary failure rates and shorter durations of patency [3, 5].

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However, upper arm brachiocephalic and brachiobasilic fistulas have a higher rate of arterial steal syndrome and high-output cardiac failure, likely due to use of the brachial artery and associated higher flow [6, 7]. Other configurations have been described, including use of the proximal radial artery, first described in 1977 [8]. Proximal radial artery AVFs (PRA-AVFs) have been shown to have higher primary, assisted primary, and cumulative patency rates compared to radiocephalic fistulas and lower risk of arterial steal syndrome and high-output cardiac failure compared to more proximal upper extremity fistulas [9, 10].

Ellipsys

Equipment/Procedure

The Ellipsys Vascular System (Avenu Medical, Inc., San Juan Capistrano, California) is a device which was developed to create a percutaneous AVF for hemodialysis access using pressure and thermal resistance. This thermal resistance anastomosis device (TRAD) creates a side-to-side anastomosis between the proximal radial artery (PRA) and an adjacent deep communicating vein in the proximal forearm.

By creating the anastomosis through an endovascular approach, the Ellipsys Vascular system offers the benefits of a PRA-AVF with the additional advantage of avoiding surgical scarring which may promote stenosis, and a surgical scar which may limit the cannulation zone. Additionally, use of this device may allow both median superficial veins to remain intact, creating a Y-shaped fistula which may allow more length and options for cannulation sites [11]. It has also been suggested that lower pressure may produce fewer aneurysms and less outflow turbulence with resultant shear stress to the vessel wall [12].

The procedure is typically performed in an outpatient setting, under locoregional anesthesia nerve block or local anesthesia with or without moderate sedation. The entire procedure is performed using ultrasound guidance, without need for radiation. As described by Hull et al. [13], a standard micropuncture needle and wire are used to obtain retrograde access into the median cubital or brachial vein. The access needle is then advanced intravenously under ultrasound guidance to a position where the PRA is adjacent. The needle is then used to puncture the PRA and the wire is advanced distally into the radial artery; confirmed with ultrasound. A 6 Fr Glidesheath Slender sheath (Terumo Medical Corp, Somerset, New Jersey) is then advanced into the artery. Through this, the TRAD is advanced into the sheath in an "open" position with the tip of the device in the PRA and the base in the accessed vein. The sheath is then retracted and traction is applied to the Ellipsys catheter until the tip of the device engages the wall of the PRA. The device is then closed and activated to fuse and cut the anastomosis. Once the anastomosis is created, the device can be removed via the sheath and manual pressure held for hemostasis. This tissue fused anastomosis allows for immediate balloon dilation if needed. In initial trials of the device, immediate balloon dilation was utilized on a case-by-case basis as needed to adjust and redirect flow into the outflow vein used for hemodialysis.

Mallios et al. [11] modified this technique by empirically performing angioplasty to nominal pressure using a 5 mm balloon immediately after TRAD use and found that doing so avoided the need for additional angioplasty to improve access flow.

Indications/Contraindications

Anatomic criteria for the Ellipsys device include a distance between the PRA and deep communicating vein <1.5 mm and vessel diameters of at least 2 mm. The device is contraindicated for use in patients who have a distance between the target artery and vein of >1.5 mm.

Prior Studies

As of 2019, there have been five publications describing experience with the Ellipsys device.

In 2017, Hull et al. [14] published a prospective single-arm 6-week evaluation of the Ellipsys Vascular Access System. Primary efficacy endpoints were successful creation of the fistula and fistula patency by Doppler ultrasound evaluation. Secondary endpoints were brachial artery flow volume > 400 mL/min and/or 3+ two-needle dialysis at the prescribed rate. Safety endpoints were < 50% incidence of minor complications and < 1% incidence of major device-related complications as defined by vascular access reporting standards, as well as electrical shock causing tissue injury and significant non-target embolization. Twenty six patients were enrolled in the study. Technical success (successful creation of the fistula) was 88% (23/26), with failures due to access failure caused by venous bleeding from the initial puncture or vasospasm. A fused anastomosis which remained intact throughout the initial 6-week evaluation period was achieved in 96% (22/23) of patients. At the end of 6 weeks, 77% of patients met the primary clinical endpoint. There were no major device-related anastomosis complications. Patients with access failure remained surgical candidates. Early fistula thrombosis occurred in 3 fistulas, none with arterial thrombosis or emboli. Other minor complications included minor hematoma and tract fistula formation (1 patient) of which the patients still went on to use the AVF successfully.

In 2018, Hull et al. [13] went on to publish a prospective single-arm trial involving 5 sites and 103 patients, comparing the Ellipsys device 90-day performance against meta-analysis of surgical results obtained from the literature. The primary efficacy endpoints were brachial artery flow volume ≥ 500 mL/min and target vein diameter ≥ 4 mm in > 49% of patients at 90 days. The primary safety endpoint was absence of serious device-related complications, including vessel perforation or dissection, procedural electrical shock, and embolization of a previously uninvolved arterial area. The primary, or index, procedure was performed in an office-based outpatient setting as described previously. Secondary maturation procedures included balloon dilation, brachial vein embolization, basilic vein ligation or

embolization, valvulotomy, and surgical transposition. These procedures redirected flow from deep to superficial veins, isolated outflow into a specific target vein, and/or brought matured veins closer to the skin surface. Technical success (defined as successful creation of the fistula) and clinical success (defined as clinically detectable fistula on discharge) were both 95%. Mean procedure time was 23.7 minutes. Second-stage maturation procedures were performed in 99/103 patients at a mean of 35.1 days, including anastomotic balloon dilations, deep brachial vein embolizations, cubital vein occlusions, accessory vein embolizations, and surgical transpositions. An additional 66 procedures in 36 patients were performed to maintain fistula function. The primary endpoints were met by 86% of the patients at 90 days, exceeding the 49% performance goal. Functional fistula patency by Kaplan-Meier analysis was 98.4%, 98.4%, and 92.3% at 90, 180, and 360 days. There were no major device complications or device-related serious adverse events. There was no separation or pseudoaneurysm formation at the anastomosis. Mean time to 2-needle dialysis was 100 days compared to surgical mean time to 2-needle dialysis of 360 days.

Mallios et al. [11] performed a retrospective review of data from a single center's experience with the Ellipsys system. Primary endpoints were technical success, post-operative patency by Doppler ultrasound or angiography, access flow rate, time to first use, and percutaneous AVF-related complications. Percutaneous AVF creation was attempted in 34 patients, with technical success in 97%. Creation of the percutaneous AVF was performed with the Ellipsys device as described above. AVF creation in the first 14 patients was followed by gentle inflation of a 4 or 5 mm balloon at the anastomosis, followed by planned balloon angioplasty of the anastomosis to 6 mm at 1 week after percutaneous AVF creation. In the remaining 20 patients, balloon angioplasty of the anastomosis and proximal outflow vein was performed to 5 mm at nominal pressure at the time of percutaneous AVF creation, without planned follow-up angioplasty. Primary patency (no further intervention after planned 1 week maturation angioplasty, if applicable) was 82%, and primary assisted patency and secondary patency were 94%. Six patients required a second angioplasty for fistula maturation, all in the initial group of 14 with planned 1 week angioplasties. After implementation of immediate 5 mm angioplasty at the time of percutaneous AVF creation, this was no longer needed. All fistulas were used or ready for dialysis by clinical or ultrasound evaluation by 6 weeks. There were no adverse events related to percutaneous AVF creation.

The retrospective review of data performed by Hebibi et al. [12] at a single center included 34 patients. The Ellipsys device was used as described above, without empiric angioplasty. 71% (24/34) patients had successful 2-needle cannulation within 10 days to 6 weeks after fistula creation. 44% required no further intervention. 35% required secondary angioplasty to assist maturation. Two patients had cannulation difficulties which were resolved by converting to surgical fistulas.

Beathard et al. [15] described two-year follow-up after use of the Ellipsys device at five vascular access programs in the United States with a total of 105 patients. In these patients, percutaneous AVF creation was performed as described previously, with immediate balloon dilation of the anastomosis as described by Mallios et al.

There was technical success in 98% with successful two-needle dialysis in all but 3 cases (1 patient did not reach the point of requiring dialysis during the study period and 2 were on peritoneal dialysis with AVFs created as backup). There were 6 instances (5.7%) of late fistula failure of which the causes were unspecified by the authors. Cumulative patency rates were 96.1%, 92.8%, and 91.6% at 6, 12, and 24 months. Additionally, patient satisfaction surveys were sent, with a 39% response rate. The responses demonstrated a high level of satisfaction with the procedure with a lack of pain perceived by the patients in 95% of cases.

WavelinQ

Equipment/Procedure

The WavelinQ (formerly EverlinQ) EndoAVF System (Becton Dickinson, Franklin Lakes, NJ, USA) is a dual-catheter system that is placed in a closely-applied artery and vein in the upper forearm, usually the ulnar artery and vein, and utilizes strong rare-earth magnets on both catheters to align and maintain appropriate positioning. A radiofrequency (RF) electrode on the venous catheter is activated adjacent to a ceramic backstop on the arterial catheter, heating and vaporizing the vessel walls between the two and creating a side-to-side AVF [16].

The first-generation 6 Fr catheter system required a 7 Fr venous sheath and a 6 Fr arterial sheath. The most recent iteration uses dual 4 Fr catheters, which achieved 510(k) clearance from the FDA in February 2019 [17]. Arterial and venous access can be aligned in parallel or anti-parallel orientation, with either or both catheter access sites in the wrist or upper arm.

Prior to the procedure, patients should undergo ultrasound Doppler vascular mapping to identify adequate vessel diameters for fistula creation in the forearm. Specifically, the presence of a perforator vein in the proximal forearm with a diameter of ≥ 2 mm must be identified. In clinical studies, patients were selected based on the criteria of target artery and vein diameters ≥ 2 mm with superficial venous anatomy suitable to create a SAVF based on KDOQI guidelines [1, 2, 18]. An Allen test is adequate to assess for patency and dominance of the radial and ulnar arteries.

The procedure is usually performed under moderate sedation with local anesthesia. Patients receive intravenous heparin (between 1000 and 5000 units, depending on study design/operator). Because the side-to-side anastomosis is fundamentally different from the end-to-side anastomosis seen in SAVFs, it is recommended the anastomosis is created in proximity to the perforating vein that communicates the deep venous system (ulnar or radial vein) to the superficial venous system (cephalic and/or basilic veins) (Fig. 23.1). When the catheters are coapted via magnets, the convex electrode (on the venous catheter) should be aligned with the concave ceramic plate (Fig. 23.2a). The channel is created with a 0.7 second RF pulse (Fig. 23.2b) after which the devices are removed. An angiogram is performed via the arterial access to confirm successful AVF creation (Fig. 23.2c). The more central deep brachial vein is frequently embolized (using coils or a vascular plug) to further

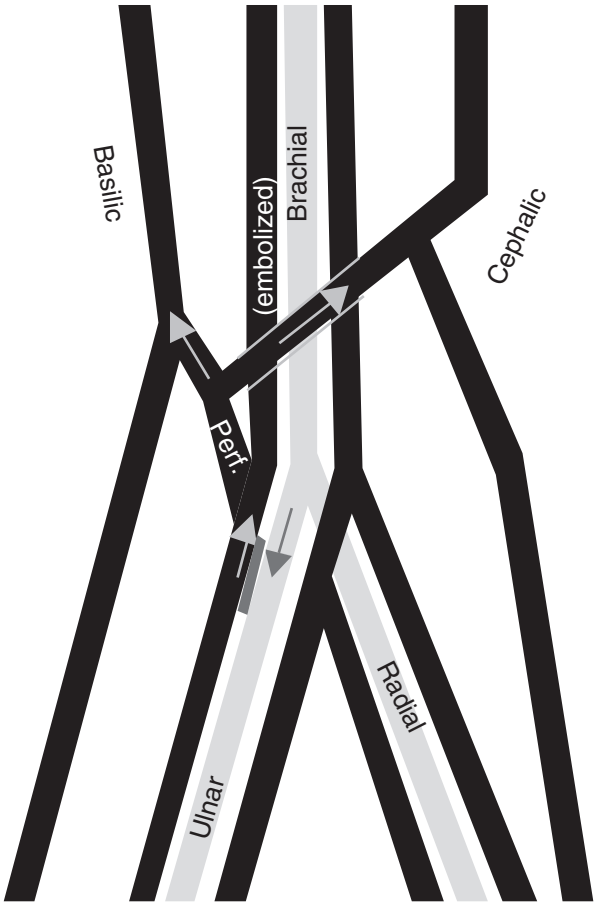

Fig. 23.1 Diagram of the anatomic relationship between created fistula, arteries (light gray), and veins (black). Arrows signify direction of flow. Often, a competing brachial vein is embolized to promote outflow via the perforator, cephalic, and/or basilic vein

encourage flow from the deep venous system through the perforator vein into the target superficial access veins.

Indications/Contraindications

Non-dialysis dependent (chronic kidney disease (CKD) stage 4 or 5) or dialysis dependent patients in need of hemodialysis access, with or without a previously failed fistula, could benefit from endovascular AVF creation [19].

In recent clinical studies, patients were excluded for non-amenable vascular anatomy (patients with central venous narrowing > 50% or other known upper extremity occlusions/stenosis), heart failure, bleeding diathesis or hypercoagulable state, immunosuppression, acute infection, severe congestive heart failure (ejection fraction < 20%) or known contrast dye allergy not amenable to premedication [2, 18].

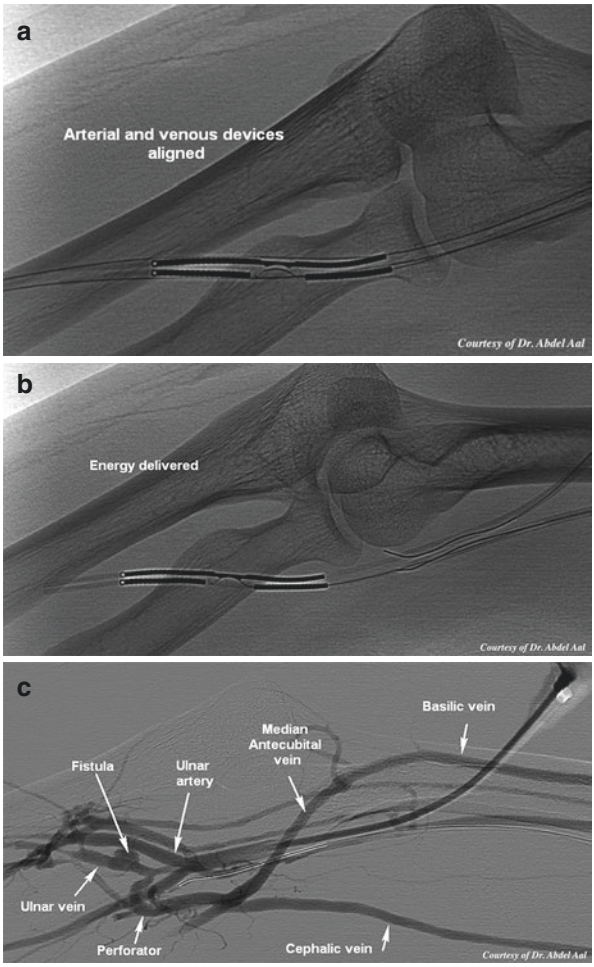

Fig. 23.2 (**a**) Aligned arterial and venous catheters with deployed electrode prior to radiofrequency pulse activation. (**b**) Following pulse activation, the spring-loaded electrode is fully apposed to the ceramic endplate signifying fistula creation. (**c**) Completion angiogram via the brachial artery demonstrates the fistula and outflow vessels

Prior Studies

All studies evaluating the EverlinQ/WavelinQ system are single-arm prospective trials enrolling 60 patients or fewer.

Efficacy of the 6 Fr system was first evaluated by Rajan et al. [18] in a single-arm prospective study of 33 patients, 32 of which experienced technical success without extravasation. All underwent ulnar artery-to-vein anastomosis. Four patients expired during the 6-month follow-up phase for reasons unrelated to the procedure. Of the remaining 28 patients, 24 were undergoing successful hemodialysis at 6 months with a subsequent intervention rate of 0.6 per patient during this time to assist maturation. Most of these interventions were for coil embolization of the competing brachial vein in an earlier cohort of patients that did not have the vein coiled at the index procedure. The remaining interventions were surgical AVF (n = 1) creation,

balloon-assisted maturation (n = 3) and thrombin injection (n = 2). Only one patient experienced late thrombosis at 3.5 months, related to central venous stenosis.

Eight patients with CKD 4/5 were evaluated by Radosa et al. [19] who were deemed unsuitable for a surgical Brescia-Cimino fistula. All fistulas were created in the ulnar artery and vein. The proximal brachial vein was embolized with an Amplatzer Vascular Plug (St. Jude Medical Inc., St Paul, MN, USA). Except for 1 patient lost to follow-up, all were undergoing successful hemodialysis at 6 months. Only 1 patient required interim intervention, a basilic vein superficialization procedure.

The Novel Endovascular Access Trial (NEAT) [20] evaluated 60 patients for ulnar endovascular AVF creation with the 6 Fr system. Technical success was 98%. The single failure was due to the use of a braided vascular sheath that acted as an energy sink that prevented adequate radiofrequency energy delivery to create the anastomosis. Suitable endovascular AVF for hemodialysis was achieved in 52/60 patients (87%) with mean venous access diameter change at 3 months of 1.7 to 5.9 mm (median cubital vein), 2.0 to 5.2 mm (cephalic vein), and 1.8 to 6.0 mm (basilic vein). At 12 months, primary endovascular AVF patency was 69% and cumulative patency was 84%, with 19 patients undergoing 24 interval interventions for transposition (n = 5), tributary vein embolization (n = 5), thrombectomy/thrombolysis (n = 3), balloon angioplasty (n = 2), thrombin pseudoaneurysm injections (n = 2), and surgical arterial repairs (n = 2). Included in these interventions were 3 endovascular AVF ligations and 2 new arteriovenous graft placements. Of the 42 patients that still ultimately required hemodialysis within the 12-months follow-up period, 28 had successful cannulation (67%) with mean time to cannulation of 112 days.

The newer, 4-Fr catheter system was evaluated in the Endovascular Access System Enhancements (EASE) Trial [2] in 32 patients, with most fistulas created in the radial vessels (63%), and the majority of arterial access via the wrist (72%). Technical success was 94% and primary and cumulative patency rates at 6 months were 83 and 87%, respectively. Post-procedure intervention rates were 0.21 per patient-year, though full 6-month follow-up was only achieved in 22/32 patients, of which 5 patients expired from causes unrelated to the procedure. Overall, successful cannulation was achieved in 20/27 patients (74%) at follow-up.

A meta-analysis of the above studies demonstrated a pooled endovascular AVF technical success rate of 99%, 90-day maturation rate of 88%, and 6-month cumulative patency rate of 92% [21]. These results are better than the SAVF maturation rates of 64% to 76% in much larger cohort studies from the United States [22] and the Netherlands [23]. There are however no randomized controlled trials to directly, however, compare the outcomes of endovascular AVFs to SAVFs.

Arnold et al. [24] evaluated a first-year cost analysis of endovascular AVFs versus SAVFs based on repeat intervention data from the NEAT trial [20]. Propensity score analysis matched patients in the trial with similar patients from the USRDS data obtained from 2011 to 2013. First-year event rate for intervention in the new endovascular AVF cohort was 0.74 versus 7.22 for the incident SAVF cohort. The 1-year intervention-free period in the endovascular AVF group was 70% versus 18%

in the matched surgical cohort. This translated into an estimated cost savings of $16,494 per patient-year in patients initiating hemodialysis with endovascular AVFs versus SAVFs.

Overall, initial experiences with the Ellipsys Vascular System and WavelinQ EndoAVF System devices have been positive with high technical and clinical success rates. While initial studies have shown good short-term results, further studies evaluating the long-term patency and financial benefits of percutaneously created AVFs are needed.

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Surgical Management of Deep Fistula Veins

24

Shouwen Wang

Introduction

Deep veins are increasingly used for creations of arteriovenous fistulas. Not uncommon in patients with chronic kidney diseases, the cephalic veins are too small or have been damaged by repeated phlebotomy or cannulations or have been used for arteriovenous access. In these patients, the usually undamaged basilic veins become preferred for arteriovenous fistula creations [1]. However, the basilic veins usually cannot be cannulated directly and require transposition before use. Additionally, the prevalence of obesity is increasing, causing the cephalic veins to be too deep for direct cannulations. These deep veins pose additional challenges for creating functional autogenous arteriovenous fistulas. The goal of surgical management is to render these fistula veins easily cannulatable for hemodialysis therapy.

The basilic vein is located in the medial-posterior aspect of the forearm, making it awkward for cannulation. A transposition to the anterior aspect of the forearm will facilitate its use. The basilic vein is located deep in the medial aspect of the upper arm and is always accompanied by the medial cutaneous nerve; therefore, it should not be cannulated without transposition.

Various surgical techniques have been used to transpose the basilic vein, which is discussed further in the ensuing sections. The author prefers basilic elevation transposition approach for its potential advantages: reduced procedure time and reduced incidence of swing segment stenosis [2].

There has been debate on one-stage versus two-stage basilic vein transposition in the literature. In one-stage transposition, the native vein is transposed and the arteriovenous anastomosis is created during the same surgical session. In two-stage transposition, an arteriovenous anastomosis is created first to allow the vein to grow

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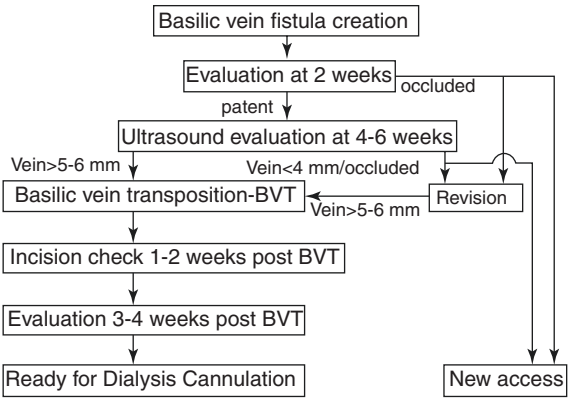

Fig. 24.1 A flow-chart of two-stage basilic vein fistula creation. Similar flow chart may be applied to managing deep cephalic veins

and the vein transposition is performed several weeks later. Most data support that two-stage approach is associated with improved clinical outcomes, even though it takes a longer time for the fistula to be functional. One-stage approach may be considered if the native basilic vein is already 5–6 mm in diameter [3]. The author prefers two-stage approach (Fig. 24.1).

Deep vein transposition can be safely performed in an outpatient setting or same-day surgical suite of a hospital. General anesthesia or regional nerve block has often been employed for these surgical procedures. Additionally, local anesthesia plus conscious sedation generally provides sufficient comfort for patient going through these procedures. One advantage of conscious sedation is that nerve injury can be minimized as the patient can still respond to nerve stimuli during surgery, with a caveat of some patient discomfort when nerves are stimulated. The following section describes basilic vein transposition as a second stage procedure performed under local anesthesia plus conscious sedation.

Equipment for Basilic Vein Elevation Transposition (Fig. 24.2)

1. Syringes (10 and 20 ml), needles (25 gauge)
2. 1% lidocaine solution (without epinephrine, may also use 1% lidocaine +2% prilocaine +8.4% sodium bicarbonate in a 10 + 10+ 2 ml combination)
3. Gauze (4″ × 4″) and Lap sponges
4. Surgical blades (No. 15)
5. Scissors (Tenotomy scissors for tissue dissection; suture scissors)
6. Forceps (DeBakey)
7. Electrocautery unit, grounding pad and monopolar handswitch pencil
8. Vessel clips and clip appliers (small and medium, Ethicon™)
9. Vacuum suction equipment, tubing and tips (Yankauer and Frazier)
10. Retractors (Weitlaner with dull jaw; Senn)
11. Kelly hemostats, right-angle hemostat, and needle holder
12. Vascular loops (round rubber band for holding vessels or nerves)

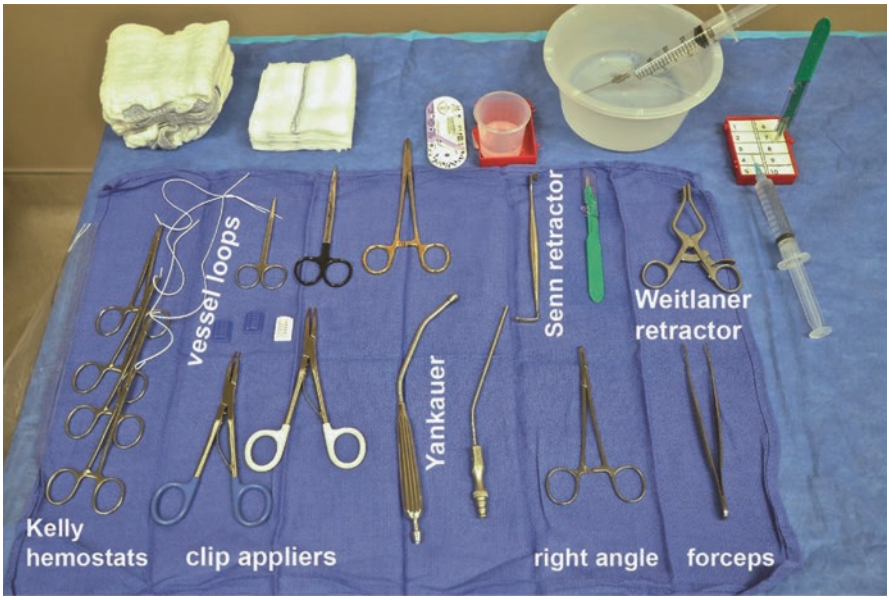

Fig. 24.2 Equipment for basilic vein transposition. Main sterile tools used during vein elevation transposition surgery

13. 2–0 silk or Vicryl™ suture with needle and 3–0 silk tie suture
14. Jackson-Pratt drain set (optional)
15. 3–0 suture for skin (Vicryl™ – absorbable)
16. Sterile strips
17. Coban™ elastic bandage.

Steps of Basilic Vein Elevation Transposition (Second Stage)

1. Preoperatively, the patient is examined and relevant history is reviewed.
2. The basilic vein is visualized with ultrasound and its path is marked (Fig. 24.3a).
3. The surgical instruments are organized on a sterile table (Fig. 24.2).
4. The arm is prepared and draped to create a sterile surgical field.
5. Sedation medications (midazolam and fentanyl) are given intravenously. The starting doses of these medications need to be tailored to an individual patient. A dedicated nurse monitors continuous electrocardiogram, pulse oximetry, and intermittent blood pressure.
6. Local anesthetic (1% lidocaine) is infiltrated along the incision site.
7. A longitudinal incision is made over the basilic vein (Fig. 24.3b).
8. Blunt and sharp dissections are carried out to free the basilic fistula vein from surrounding tissues. The dissection is extended several centimeters proximal to the incision to ensure a smooth transition when the vein is transposed. Care is taken to preserve the accompanying medial cutaneous nerve (Fig. 24.4).

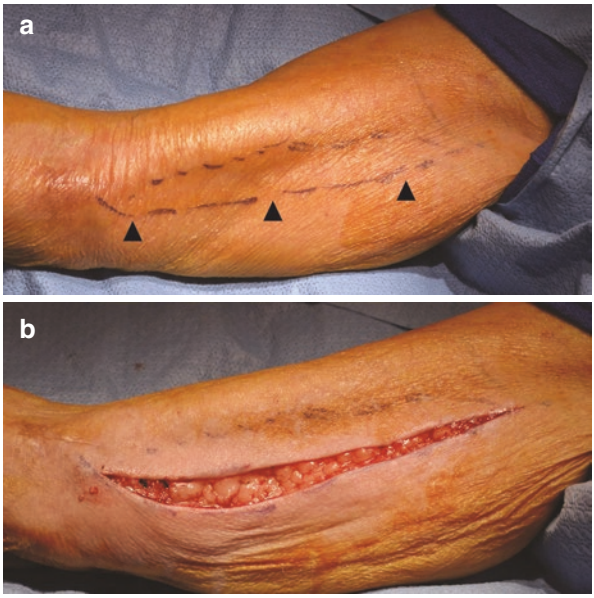

Fig. 24.3 Surgical planning and incision. (**a**) The basilic vein is marked based on preoperative ultrasound (*lower line*) and the planned new location of the basilic vein is indicated (*upper line*). (**b**) An incision is made over the basilic vein after lidocaine infiltration

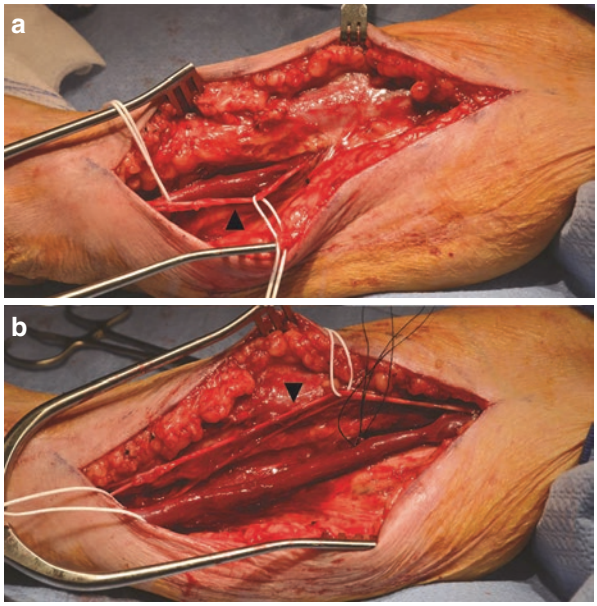

Fig. 24.4 Isolation of the basilic vein. (**a**) Careful dissection is carried out in the lower portion of the upper arm to isolate the basilic vein. (**b**) Further dissection is performed to free up the proximal basilic vein, and side branches are tied off or clipped. The medial cutaneous nerve is separated away from the basilic vein (*arrowheads*)

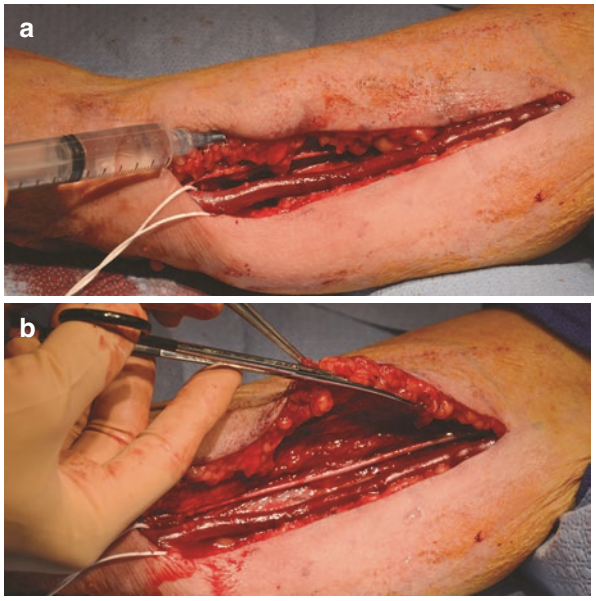

Fig. 24.5 Creation of anterior pocket. (**a**) The subcutaneous tissue is infiltrated with 0.5% lidocaine. (**b**) The subcutaneous tissue is separated with scissors

9. The anterior subcutaneous tissue is infiltrated with 0.5% lidocaine (Fig. 24.5a). The subcutaneous tissue is then dissected with scissors to create a pocket (Fig. 24.5b).
10. The excessive subcutaneous adipose tissue may be carefully trimmed along the intended basilic vein path (Fig. 24.6). Hemostasis is achieved by cauterization.
11. The basilic fistula vein is swung anteriorly to the subcutaneous pocket and fixed in position by suturing the subcutaneous tissue to the lateral soft tissue or fascia of the biceps muscle with sutures. It is preferred to start the fixation distally (Fig. 24.7). Care is taken that the sutures will not cause compression of the basilic fistula vein and the swing segments both distally and proximally have smooth transitions (Fig. 24.8a). Alternatively, similar to the superficialization of cephalic vein described later in this chapter, the soft tissues may be approximated together first with sutures (such as 2–0 Vicryl™), leaving the basilic vein in the superficial tissue plane. The basilic vein is then placed in the subcutaneous pocket and the incision is closed with subcutaneous and subcuticular sutures.
12. The proximal basilic fistula vein is compressed to make sure that the basilic fistula vein is easily palpable from the skin along its transposed path (Fig. 24.8b).
13. A Jackson-Pratt drain is placed along the incision to prevent postoperative fluid accumulation (Fig. 24.9). This is optional and may be considered in patients taking antiplatelet or anticoagulation medications.
14. The subcutaneous layer is closed with intermittent 3–0 Vicryl™ suture (Fig. 24.9). Care is taken not to puncture the transposed basilic fistula vein.

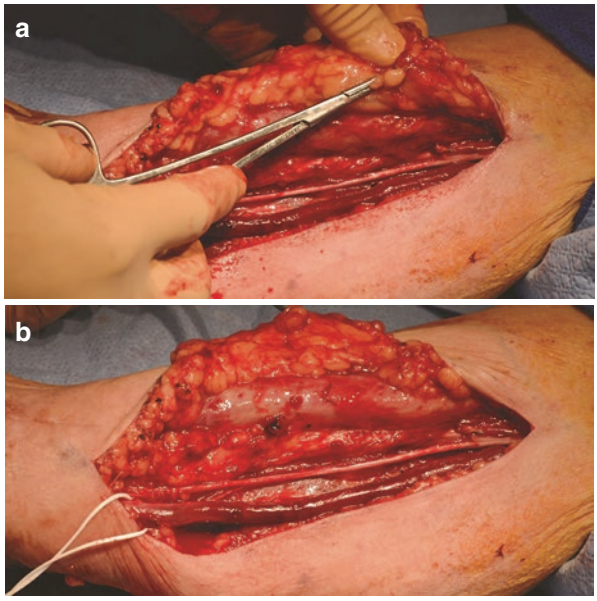

Fig. 24.6 Creation of anterior pocket – continued. (**a**) Hemostasis is achieved by cauterizing small bleeders. Excessive subcutaneous adipose tissue along the new vein path is carefully trimmed if needed. (**b**) The anterior subcutaneous pocket is ready to accommodate the basilic vein

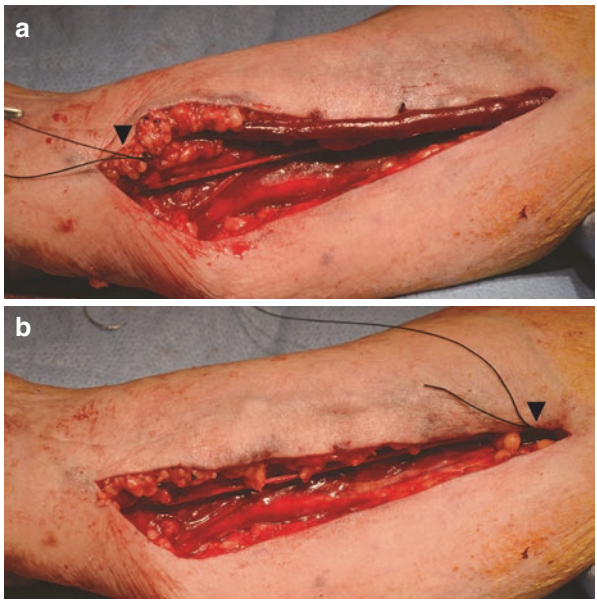

Fig. 24.7 Fixation of the basilic vein. (**a**) The basilic vein is fixated to the anterior pocket by approximating the subcutaneous tissue to the lateral soft tissue or the fascia of the biceps, starting distally. (**b**) several more sutures are applied to secure the basilic vein in the anterior pocket. Care is taken not to compress the basilic vein and not to produce kinks

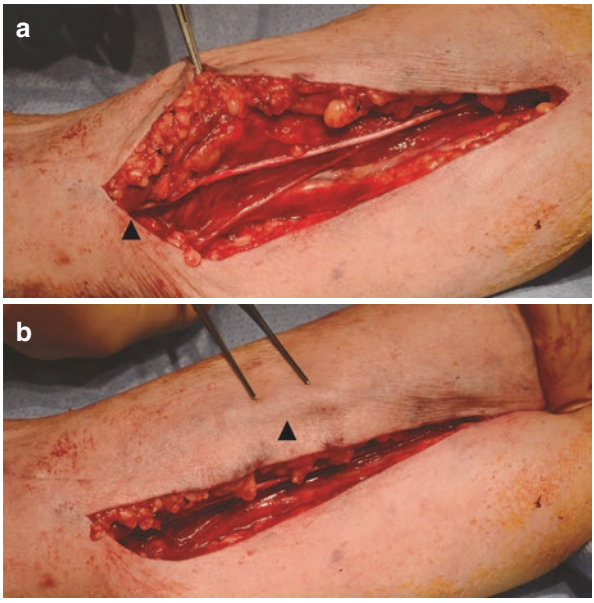

Fig. 24.8 Assessment of the transposed basilic vein. (**a**) The distal and proximal swing segments of the basilic vein are examined to assure smooth transition (*arrowhead*). The fistula thrill should be smooth, soft, and easily palpable along its path. (**b**) The proximal basilic vein is compressed to assess the palpability of the basilic vein along its path. Notice bulging of the basilic vein (*arrowhead*)

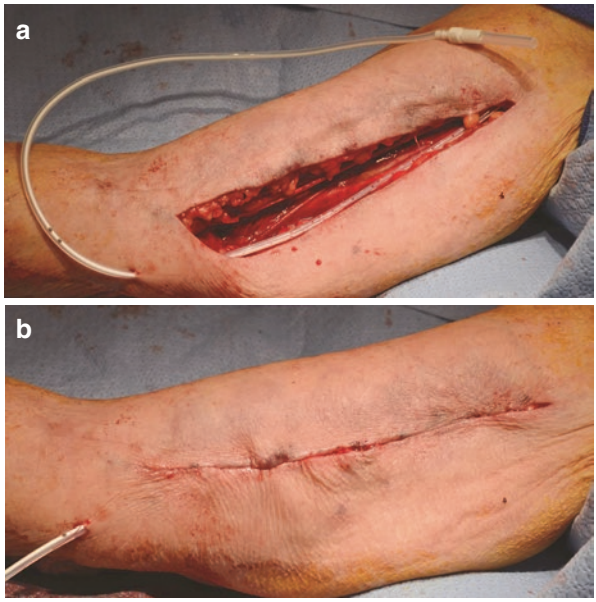

Fig. 24.9 Placement of Jackson-Pratt drain. (**a**) A draining tube is placed to prevent fluid accumulation. This is optional. (**b**) The subcutaneous tissue is approximated with 3–0 Vicryl™ sutures. Care is taken not to puncture the fistula vein during suturing

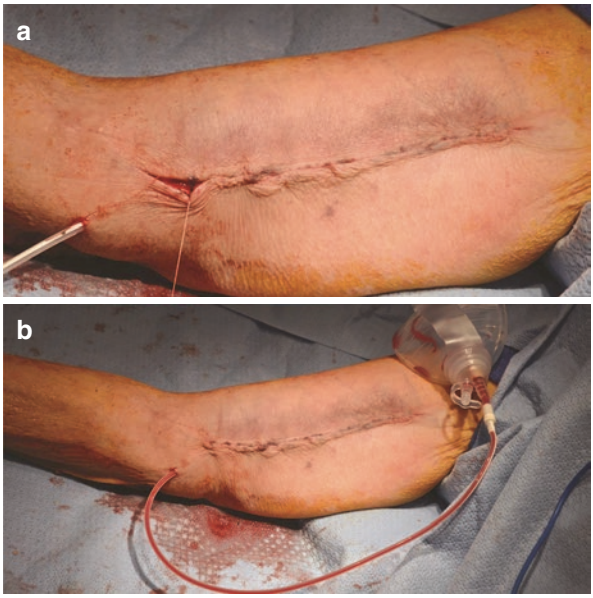

Fig. 24.10 Skin closure. (**a**) The skin is closed with a continuous subcuticular 3–0 Vicryl™ suture. (**b**) The suction bulb of the Jackson-Pratt drain is attached to the draining tube and suction is applied. The draining tube is secured to the skin with sterile strips

15. The skin is closed with a continuous subcuticular 3–0 Vicryl™ suture (Fig. 24.10a). The bulb reservoir is then attached to the Jackson-Pratt draining tube and suction is applied (Fig. 24.10b).
16. Sterile strips and dressing are applied.
17. An elastic bandage may be applied snugly on the arm to minimize minor blood oozing and provide support to the surgery site. Care is taken not to cause significant compression of the basilic fistula vein.
18. The Jackson-Pratt drain is removed in 2 days.

Other Surgical Techniques for Superficialization of Deep Fistula Veins

Transposed basilic vein fistula is one of the preferred autogenous arteriovenous access types [1, 3]. Traditionally, the basilic vein is transposed by tunneling it through a subcutaneous track (Fig. 24.11). While it is effective, a common complication associated with tunnel transposition is proximal swing segment stenosis that often causes dysfunction of a fistula [4]. The development of such lesions may be partially due to twist (axial torsion) and kink of the swing segment produced during the tunneling process. A simple fistula elevation approach was reported to be useful for superficialization of both basilic and cephalic veins [5]. However, this approach leaves the fistula vein directly under the surgical incision, which may be less desirable for repeated cannulations. The basilic anterior elevation transposition as described in the above section does not require transection and tunneling of the basilic vein, as such the twist and kink in the proximal swing segment are

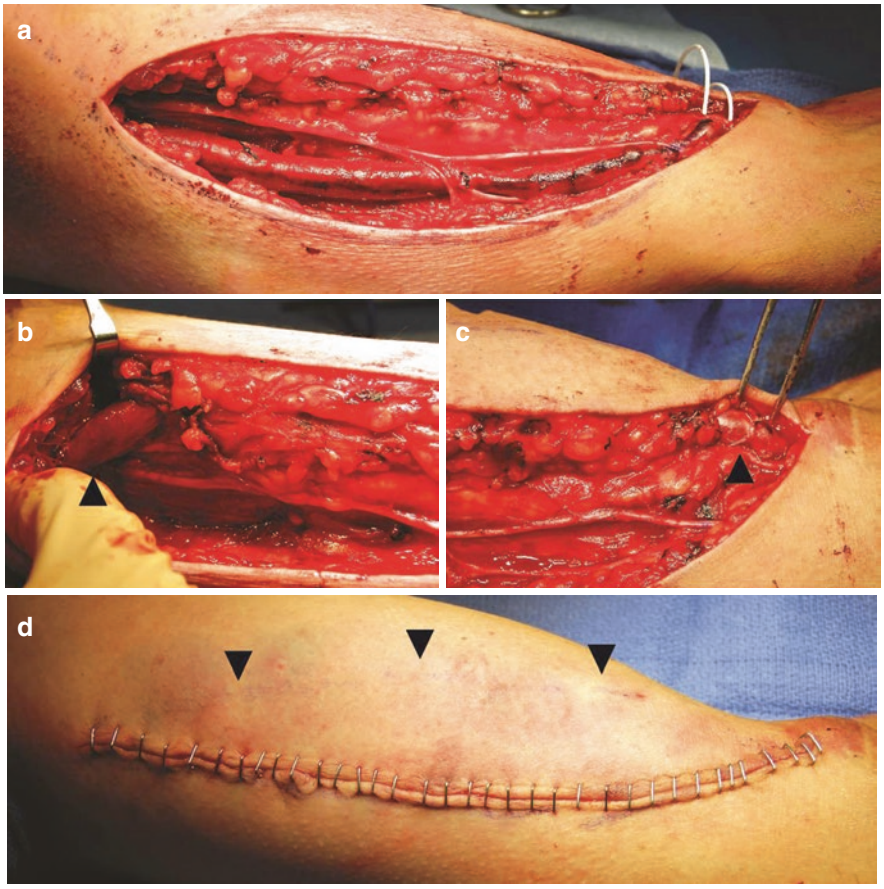

Fig. 24.11 Basilic fistula vein tunnel transposition (second stage). (**a**) The basilic fistula vein is isolated. Notice its relationship with the medial cutaneous nerve. The front surface of the vein is marked with marking pen to aid alignment during tunneling and anastomosis. The basilic fistula vein is then transected distally and tunneled subcutaneously with tunneling tools. (**b**) The proximal swing segment of the basilic fistula vein. (**c**) the distal end-end venous anastomosis after tunneling. (**d**) The incision is closed with sutures and staples. The *arrowheads* point to the location of the transposed basilic fistula vein in its tunnel

minimized. Based on the author's experience, the typical swing segment stenosis is significantly reduced and the procedure time is reduced as compared with tunnel transposition [2]. Furthermore, since the basilic fistula vein is transposed away from the skin incision, the incision scar will not be in the way of cannulation. Given these advantages, the author prefers the elevation transposition approach (Table 24.1).

Surgical approaches for superficializing basilic veins have been employed to superficialize deep cephalic veins: tunnel transposition [3], simple elevation [5], and elevation transposition (Fig. 24.12). Additionally, lipectomy (Fig. 24.13) [6] and liposuction [7] have been utilized to superficialize cephalic veins since there is no

Table 24.1 Surgical approaches for superficialization of basilic fistula vein

Surgical approach	Comments
Tunnel transposition	Commonly used approach
	Technically more challenging
	Longer procedure time
	May use multiple small incisions for vein isolation
	Axial torsion and kink of proximal swing segment may cause local fistula vein stenosis
Elevation	Less commonly used
	Easier procedure
	Incision scar on top of fistula vein, less desirable
Elevation transposition	Easier and shorter procedure than tunnel transposition
	Incision scar away from fistula vein, more desirable than elevation alone
	No axial torsion, minimal kink, therefore potentially reduced future swing segment stenosis
	A preferred approach

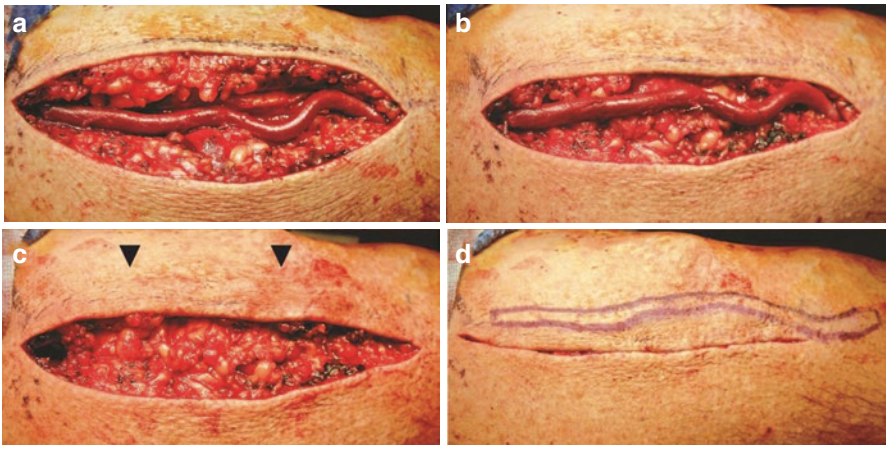

Fig. 24.12 Cephalic fistula vein elevation transposition (second stage). This is a forearm arteriovenous fistula. (**a**) The deep fistula vein is isolated. (**b**) A lateral subcutaneous pocket is created and the subcutaneous tissue is approximated with 2–0 Vicryl™ sutures, leaving the cephalic fistula vein superficial. (**c**) The cephalic fistula vein is fixated to the lateral subcutaneous pocket by suturing the subcutaneous tissues (*arrowheads* indicate the new vein location). (**d**) The skin is approximated with intermittent sutures and subsequently closed with a subcuticular suture. The marking highlights the cephalic fistula vein segment that is easily palpable

significant nerve trunk superficial to the cephalic veins (Table 24.2). Lipectomy is easier to perform. Since the fistula vein is not manipulated, future fistula vein stenosis may potentially be avoided. However, it may leave some disfiguration of the extremity and may not be suitable for very deep fistula veins. Different from the initial report of using multiple transverse incisions [6], the author prefers a single longitudinal incision lateral or medial to the fistula vein that is easier for exposure and leaves no scar on top of the fistula vein. Another issue with lipectomy is

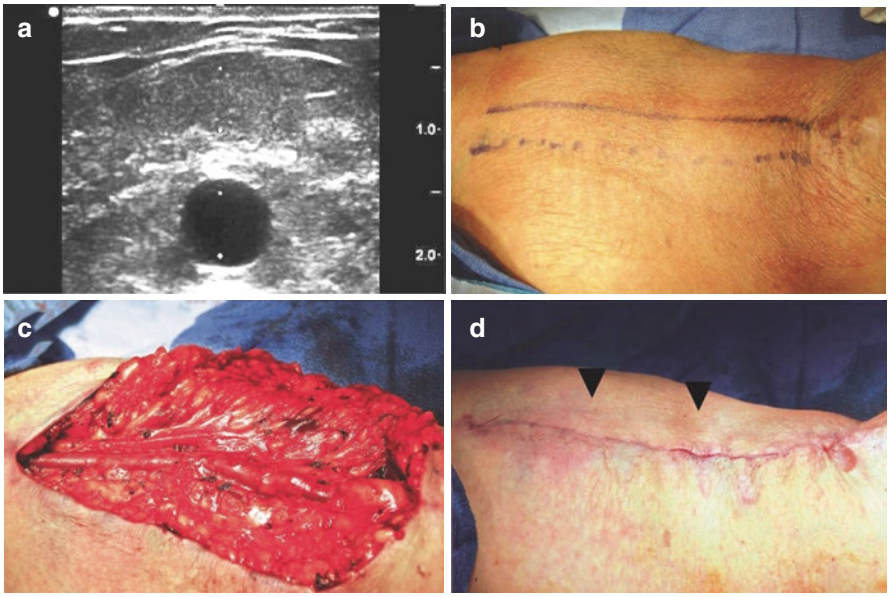

Fig. 24.13 Lipectomy for superficialization of cephalic fistula vein. (**a**) A preoperative ultrasound image shows fistula vein about 13 mm deep. (**b**) An incision is planned (*dotted markings*) medial to the fistula vein (*solid marking*). (**c**) The subcutaneous adipose tissue over the fistula vein is resected carefully. (**d**) The skin is closed. The fistula vein is now just underneath the skin (*arrowheads*). A Jackson-Pratt drain may be used to prevent fluid collection over the fistula vein

Table 24.2 Surgical approaches for superficialization of cephalic fistula veins

Surgical approach	Comments
Tunnel transposition	See corresponding comments in Table 24.1
Elevation	See corresponding comments in Table 24.1
Elevation transposition	See corresponding comments in Table 24.1
	A reliable alternative to other approaches
Lipectomy	Easier procedure, more suitable for forearm fistula
	The fistula vein is not moved, avoiding future lesions
	May cause some disfiguration of the extremity
	Not suitable for very deep fistula veins
	Different from the initial report of using multiple transverse incisions [5], a single longitudinal incision lateral or medial to the fistula vein may be preferred based on the authors' experience
Liposuction	Newer approach, not commonly used
	May be performed through very small incisions
	Ultrasound guidance may be needed for safety
	May leave some disfiguration
	Importantly, more clinical experience is needed to evaluate its safety and effectiveness

potential fluid accumulation on top of the fistula post surgery, which may delay use of the fistula [8]. Liposuction has recently been used for removing adipose tissue over cephalic fistula veins and appears to be a less invasive approach. However, clinical data are very limited and more clinical experience is needed to evaluate its safety and effectiveness.

When other veins in the upper extremity are exhausted, the brachial veins may be utilized for fistula creation. Similar to basilic veins, the brachial veins need transposition. A two-stage approach is preferred since the brachial veins are usually small [9].

Ultrasound guidance may be employed to facilitate cannulation of moderately deep cephalic fistulas [10]. Superficialization of fistula veins may potentially be avoided in selected patients where ultrasound device is routinely available. However, ultrasound is still rarely available in dialysis units. Upper arm basilic fistulas should not be cannulated without transposition except the short segment near the elbow because the medial cutaneous nerve is invariably positioned on top of the proximal basilic vein.

Tips and Troubleshooting

Potential issues and complications that may be encountered during and shortly after surgical management of deep fistula veins are discussed in Table 24.3.

Table 24.3 Potential issues and complications

Problem	Troubleshooting
Prophylactic antibiotics	Prophylactic antibiotics are needed given the more extensive nature of these surgical procedures. A single dose of an antibiotic with gram-positive coverage (such as 2 g of cefazolin or 1 g of vancomycin intravenously) is administered preoperatively
Intraoperative heparin	Since the fistula flow is not interrupted, no intraoperative heparin is needed for elevation transposition
Medial cutaneous nerve	The medial cutaneous nerve is typically located on top of the basilic vein in the upper arm. This nerve needs to be isolated from the basilic vein. The basilic vein can generally be elevated and transposed anteriorly after being separated from the nerve. If necessary, the nerve fiber may be gently separated longitudinally to give way to the basilic vein [2]
Distal ischemia	Occasionally, distal ischemic symptoms may develop after the first stage creation of a basilic arteriovenous fistula. Plethysmography and digital pulse oximetry may be used to confirm the decreased distal circulation. Doppler ultrasound may be used to evaluate the arterial circulation and fistula blood flow. If excessive fistula flow contributes to the distal ischemia, a simple technique to limit fistula flow is to band down the fistula vein close to the arterial anastomosis with external dilator-assisted banding (e-DAB) or clips during second stage vein transposition. E-DAB is achieved by tying a 3–0 silk suture over the vein and a dilator placed external to the vein (typically 10 French = 3.3 mm). The internal diameter of the vein lumen will be similar to the outside diameter of the dilator after removal of the dilator [11, 12]. A return of oxygen saturation to well above 90% and reliable plethysmography wave suggest sufficient distal circulation

Table 24.3 (continued)

Problem	Troubleshooting
Minimizing wound complications	A long incision is generally required to isolate a sufficient length of the basilic vein for transposition. One concern of long incision is potentially increased incidence of incision complications: Infection, delayed healing, or skin necrosis. These complications may be minimized with prophylactic antibiotic use, meticulous surgical techniques during surgery and incision closure, and limited dissection when creating the anterior or lateral pocket. A Jackson-Pratt drain can reduce fluid accumulation and may potentially reduce incision complications in selected patients. Two small incisions may be used for superficialization of moderately deep basilic vein if it can be untangled from the medial cutaneous nerve (Fig. 24.14) and cephalic vein (Fig. 24.15) in selected patients

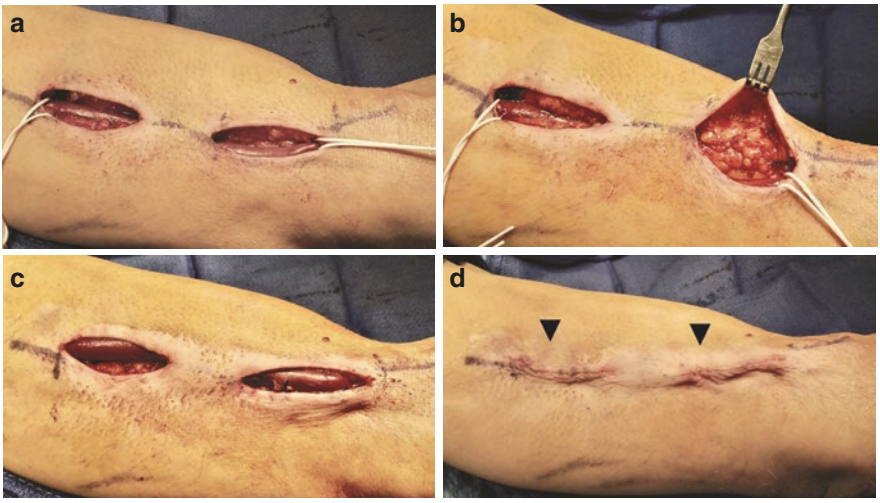

Fig. 24.14 Basilic vein elevation transposition via two small incisions (second stage). This is an upper arm arteriovenous fistula. (**a**) The basilic vein is isolated via two small incisions. (**b**) An anterior subcutaneous pocket is created. (**c**) The subcutaneous tissue is approximated with 2–0 Vicryl™ sutures, leaving the basilic vein superficial. (**d**) The skin is approximated with intermittent subcutaneous and subcuticular sutures. The arrowheads indicate the basilic vein position in the subcutaneous pocket

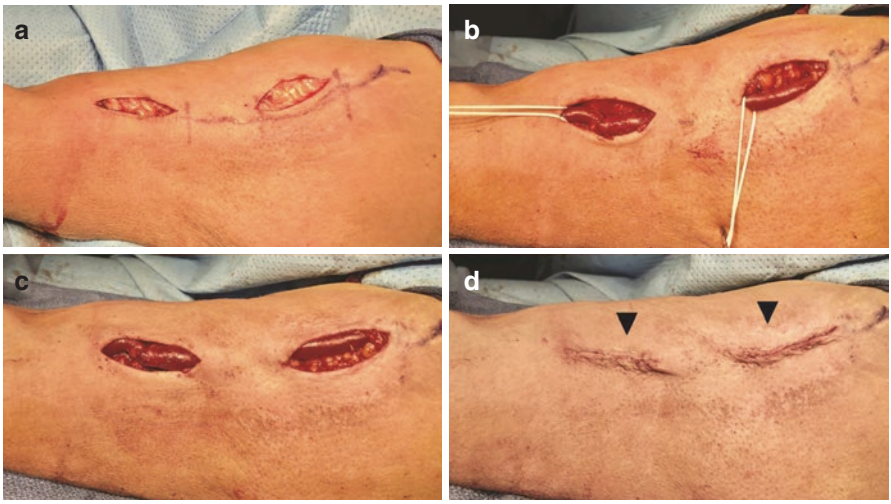

Fig. 24.15 Cephalic vein elevation transposition via two small incisions (second stage). This is an upper arm arteriovenous fistula. (**a**) Two small incisions are made over the cephalic vein. (**b**) The cephalic vein is isolated. (**c**) A medial subcutaneous pocket is created and the subcutaneous tissue is approximated with 2–0 Vicryl™ sutures, leaving the cephalic vein superficial. (**d**) The skin is approximated with intermittent subcutaneous and subcuticular sutures. The arrowheads indicate the cephalic vein position in the subcutaneous pocket

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Preoperative and Postoperative Care for Hemodialysis Vascular Access Surgery

25

Shouwen Wang and Nahel Elias

Introduction

Preoperative evaluation and postoperative care are integral parts of successful hemodialysis access surgery [1]. Clinical information pertinent to vascular access selection and creation are essential to proper planning and operative success. Likewise, organized postoperative care is required in order to minimize surgical complications and achieve a functional dialysis access, as well as avoiding unnecessary futile procedures that would not yield a functional dialysis access.

Preoperative Evaluation

Creating a functioning arteriovenous (AV) access requires careful preoperative evaluation and planning. The scopes of preoperative assessment are: to obtain patient history and to perform physical examination relevant to vascular access selection, to select vascular image studies needed for a patient, and to formulate a patient-specific plan for creating a proper long-term dialysis access or protecting vasculature for future dialysis access creation.

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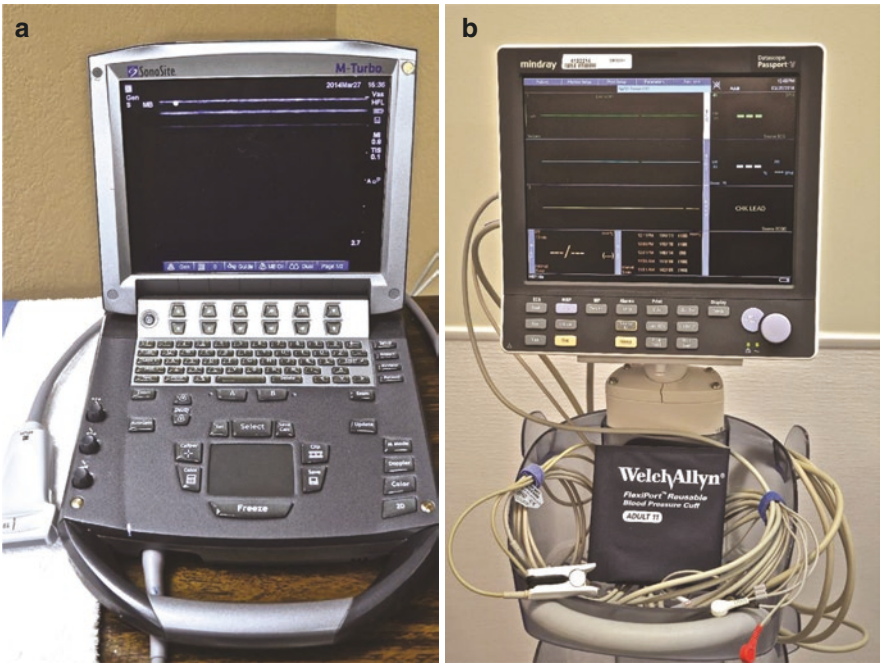

Fig. 25.1 Equipment for preoperative and postoperative evaluation. (**a**) Portable Duplex Doppler ultrasound machine. (**b**) Data monitor, for monitoring electrocardiogram, blood pressure, plethysmography and oximetry

Equipment for Preoperative Evaluation

1. Sphygmomanometer for blood pressure measurements.
2. Duplex Doppler ultrasound machine for vessel mapping and evaluation (Fig. 25.1a).
3. Pulse oximeter for assessing peripheral circulation (Fig. 25.1b).

Components of Preoperative Evaluation

1. **Decide when a patient needs surgical evaluation**. It is recommended that patients with advanced CKD (late stage 4, eGFR<20–25 ml/min) who have elected hemodialysis as their choice of renal replacement therapy, and are candidates for it, be referred to an access surgeon in order to evaluate and plan construction of AV access [1].
2. **Obtain patient history relevant to vascular access**. Certain clinical factors are associated with increased difficulty of establishing a functional AV access (Table 25.1).

Table 25.1 Patient history relevant to vascular access selection

Components	Specific history relevant to vascular access
Demographic	Advanced age, female gender
Co-morbid conditions	Diabetes mellitus, peripheral vascular disease, severe congestive heart failure, stroke, lupus, sickle cell disease, skin diseases, cigarette smoking, intravenous drug abuse
Hypercoagulable states	Recurrent thrombotic events may prompt screening for: Hyperhomocyst einemia, factor V mutations, etc.
Medications	Warfarin, clopidogrel, aspirin, immunosuppressive medications or chemotherapy
Prior dialysis access	History of failed or failing dialysis access
Prior procedures involving related vasculature	Hemodialysis catheters, central venous catheters, PICCs, pacemakers, defibrillators, arterial catheters, cardiac surgery, trauma, breast surgery and axillary lymph note dissection
Social history and overall condition	Social support structure, working profession, overall medical condition and life expectancy

Table 25.2 Physical examination relevant to vascular access selection

Components	Specific findings and relevance to access surgery
Body habitus	Body mass index, shape and fatty tissue of the upper extremities, depth of veins: May limit access selection or dictate the need for vein elevation
Skin	Forearm eczema, extensive solar keratosis, thin and fragile skin: Upper arm access may be more suitable
Neurological	Neuropathy, motor or sensory abnormality: Help to differentiate with ischemic steal syndrome and may influence access planning
Cardiac	Evidence of congestive heart failure (edema, rales): Need to be managed before access surgery
Central vein devices	Location of current or prior hemodialysis catheter or pacemaker or defibrillator: Imaging of the central vein may be required to assess possible stenosis
Arterial examination	Palpation of brachial, radial, and ulnar arteries: Evaluate the health of potential arterial inflow for AV access
	Bilateral arm blood pressure check: Unequal readings suggest arterial disease
	Barbeau's test (more accurate than Allen's test): Assess the adequacy of palmar arch and ulnar artery circulation for forearm access [2]
	Sites of previous arterial catheter or arterial donation for coronary revascularization: Absence of radial artery or potential stenosis
Venous examination	The arm veins inspected with and without a venous pressure tourniquet: Outflow veins need to be uninterrupted and distensible
	The presence of enlarged superficial veins on the chest or arms: Suggesting central vein stenosis or occlusion

3. **Perform physical examination relevant to vascular access**. Physical examination may reveal important clues that may affect access planning (Table 25.2).
4. **Use ultrasound imaging to assess vasculature relevant to dialysis access**. Ultrasound vessel mapping of both veins and arteries of the upper extremity is of critical importance for access planning. An understanding of the anatomy of the

upper extremity vessels is required for documentation and planning [3]. Ultrasound can assess the diameter, depth, patency, continuity, and distensibility of relevant veins. The distensibility of the veins may be assessed with and without a venous pressure tourniquet. The vein diameters with tourniquet applied are recorded and used as criteria for autogenous access creations (Fig. 25.2a). Both diameter and distensibility have been found to independently predict AV access success. Arterial diameters and abnormalities such as high brachial bifurcation can be easily identified by ultrasound examination. Arterial calcification and venous intimal hyperplasia or stenosis may also be easily visible on ultrasound imaging, which may substantially affect access planning [1]. Since the ultrasound images are fragmented, a freehand diagram may be employed to supplement the ultrasound images for documentation (Fig. 25.2b).

5. **Perform contrast venography if clinically indicated**. Contrast venography needs to be considered if history and physical findings suggest a central vein stenosis or occlusion. It may also be used for peripheral vein mapping, particularly when ultrasound imaging is not available. Compared to ultrasonography, venous mapping by venography provides better data on continuity, branching, and central vein patency, while it is limited in assessing distensibility and depth. Limiting the quantity of contrast and hydration are the key strategies for contrast nephropathy prophylaxis in patients with advanced chronic kidney disease. The risk of contrast nephropathy is very low when low dose contrast is used. When adequate ultrasound peripheral vein mapping has been performed for a patient, contrast venography can be limited to visualize the axillary and central veins only in order to minimize contrast quantity. Make sure the arm is fully elevated during contrast injection to enhance central vein visualization (Fig. 25.3).
6. **Formulate patient-specific surgical plans**. Arteriovenous fistulas (AVF) are generally preferred over arteriovenous graft (AVG) given the advantages the AVFs have [1, 4]. Specific recommendations should be followed in order to optimize the AV access options for a patient (Table 25.3). In some clinical scenarios, AVG may be preferred over AVF that is discussed further in a previous chapter. The guiding principle in this decision-making process is to choose the access option that will be best suited to a specific patient. The best access for a patient will provide sufficient access flow to meet the patient's long-term dialysis therapy needs while having the least potential for complications and requiring the least number of interventions.
7. **Protect vasculature for future AV access creation**. Instrumentation to veins and arteries may cause damage to these vessels and dramatically affect their suitability for AV access creation. Specific guidelines or recommendations have been developed regarding phlebotomy, peripheral and central venous catheters [5], cardiovascular implantable electronic devices [6], and transarterial approaches for cardiovascular interventions [2]. Following these clinical precautions in patients with chronic kidney disease will help to preserve related vasculature for future AV access creation and minimize potential complications (Table 25.4).

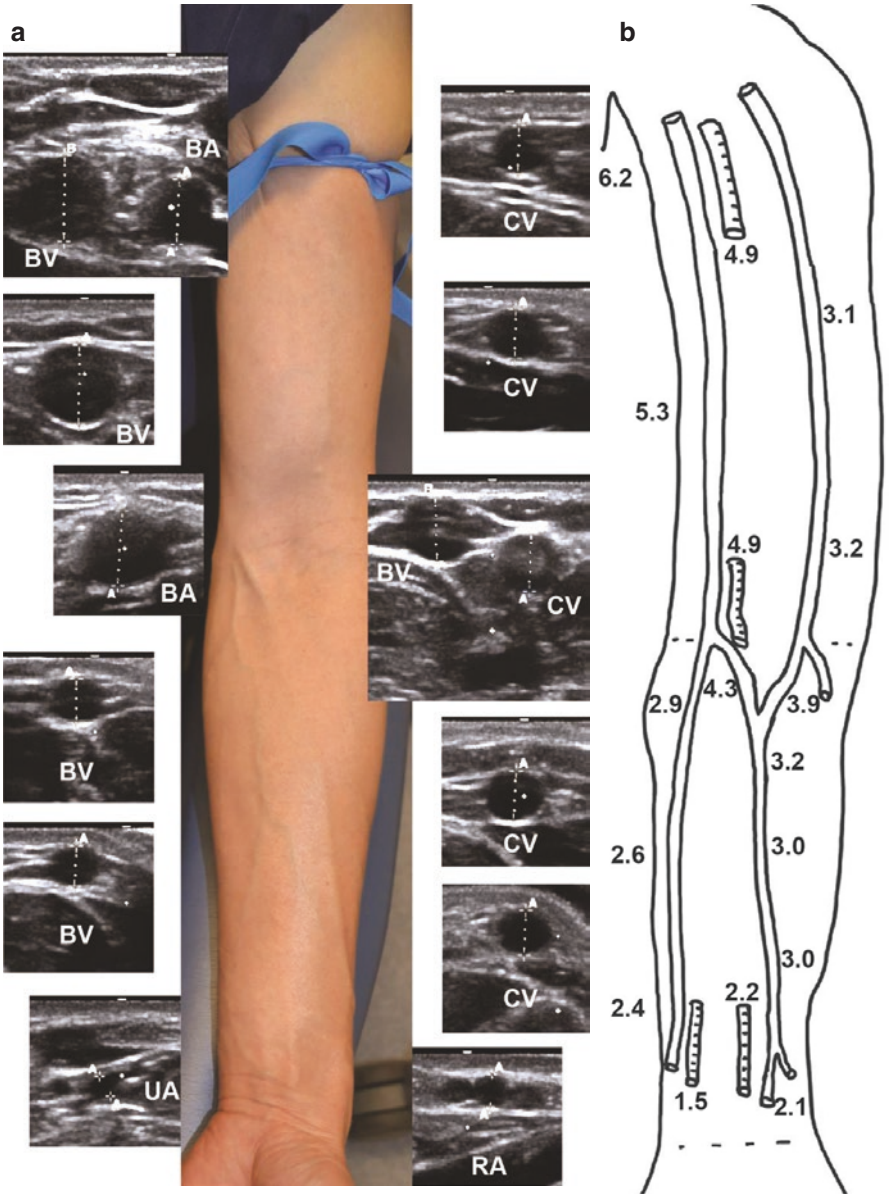

Fig. 25.2 Ultrasound vessel mapping of the upper extremity. (**a**) Photograph of a arm with a venous tourniquet applied and selected ultrasound images showing arm veins and arteries. (**b**) A freehand diagram illustrating the findings of ultrasound vessel mapping. *RA* radial artery, *UA* ulnar artery, *BA* brachial artery, *CV* cephalic vein, *BV* basilic vein. Numbers shown are vessel diameters in millimeters

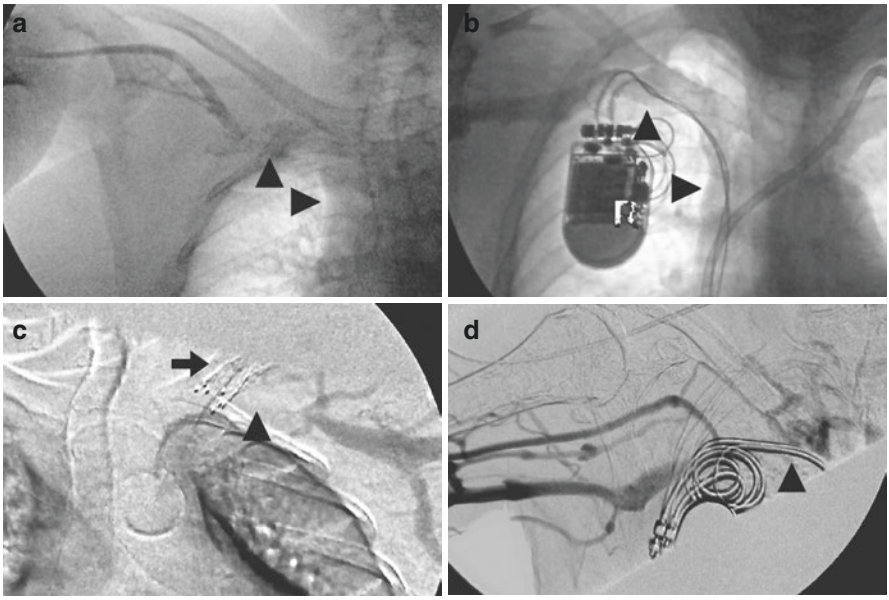

Fig. 25.3 Contrast venography for assessing the patency of central veins. (**a**) Patent central veins (*arrowheads*). Only cephalic vein is highlighted due to needle placement. (**b**) Patent central veins (*arrowheads*) in the presence of a cardiac device. (**c**) Completely occluded subclavian vein (*arrowhead*) due to prior cephalic arch stent (*arrow*). (**d**) Severe subclavian vein stenosis (*arrowhead*) due to cardiac device wires. Notice the presence of collateral veins

Table 25.3 Key operative strategies to optimize AV access creation [1]

• Place arteriovenous accesses distally in the upper extremity in order to preserve proximal sites for future accesses
• Consider autogenous AVFs first before prosthetic AVGs are placed
• Use upper extremity access sites first, give preference to non-dominant arm when access opportunities are equal in both arms
• Select forearm autogenous AVFs first when arterial and venous anatomy is suitable
• Choose upper arm autogenous AVF or forearm prosthetic AVG when forearm veins are exhausted
• Plan conversion of forearm prosthetic AVG to secondary autogenous AVF at any sign of AVG failure. This may be accomplished by using either the proximal matured outflow veins or veins of remote sites when outflow veins not suitable
• Use lower extremity and body wall access sites only when upper extremity access sites have been exhausted

8. **Assess the adequacy of the ulnar artery and palmar arch**. When the distal radial artery is considered for arterial inflow of an AV access, the adequacy of the ulnar artery and palmar arch circulation should be assessed prior to surgery. Allen's test has been traditionally used for this purpose. However, this test relies on subjective observation of circulation recovery after arterial compression.

Table 25.4 Recommendations of vessel preservation for AV access

• Identify chronic kidney disease (CKD) patients who may need hemodialysis therapy in the future. These include advanced CKD patients or patients with a functional kidney transplant
• Both veins and arteries on the upper extremities need to be preserved for future access creation in these patients
• The dorsal veins of the hand are the preferred location for phlebotomy and intravenous access
• The internal jugular veins are the preferred location for central venous accesses
• The external jugular veins are acceptable alternatives for venous access
• The subclavian veins should not be used for central venous accesses
• Placement of a PICC should be avoided
• Cardiovascular implantable electronic devices should be placed contralateral to the side of planned AV access. Alternatively, these devices with epicardial leads need to considered in CKD and dialysis patients to avoid damage to the central veins
• Transradial and transbrachial approaches for cardiovascular interventions need to be avoided in CKD and dialysis patients. If these approaches become necessary, their impact on AV access creation need to be properly assessed
• Policy and procedure for vessel preservation in CKD patients in healthcare settings should be established

Fig. 25.4 Barbeau's test. An oximeter is used to assess the adequacy of ulnar artery and palmar arch circulation. After radial artery compression, if there is flattening of plethysmography wave and reduction of oxygen saturation, inadequate circulation is suggested

Recently, Barbeau's test was developed where transcutaneous pulse oximetry and plethysmography was used to assess the ulnar artery and palmer arch circulation and shown to be more accurate than Allen's test (Fig. 25.4) [2]. Given its advantage, Barbeau's test is preferred when an oximeter is available.

Postoperative Care

Proper postoperative care is a continuation in the effort of creating a functional vascular access. The scope of postoperative care is: to care for incisions, to assess access functionality, to salvage nonfunctional or failed AV access, and to manage access-associated complications.

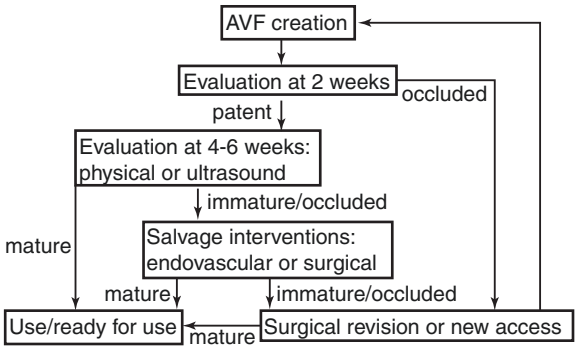

Fig. 25.5 Flow chart of postoperative management after AVF creation. If a fistula fails to mature, various salvage options need to be considered with a goal to optimize the long-term outcome of the access

The postoperative follow-up schedule differs for AVF from AVG. After an AVG placement, the incisions and graft patency are checked in 1–2 weeks. Depending on the type of the graft placed and clinical healing, a graft can be cannulated in 1–2 weeks post placement. Further follow-up is needed if clinical issues with using the AVG arise. After an AVF creation, the incision and AVF patency are examined in 10–14 days. If an AVF fails, the patient will need re-assessment for new AV access, typically with clinical and ultrasound evaluation. If an AVF remains patent, its maturation will be assessed 4–6 weeks post creation. A flow chart of management post AVF creation is presented in Fig. 25.5. A non-mature AVF may need endovascular or surgical interventions to become functional: inflow or outflow modification, collateral vein(s) ligation, transposition to more superficial position in the subcutaneous tissue, or other interventions.

Equipment for Postoperative Care

1. 1–3 of equipment for preoperative evaluation
2. Dressings: gauze (2″ × 2″, 4″ × 4″), Telfa™ non-adherent pad
3. TipStop® or other compression dressing
4. Elastic bandage wrap (Coban™)
5. Suture removal kit
6. Adhesive tapes
7. Sterile wound closure strips
8. Alcohol wipes
9. Staple removal kit, if staples are used
10. Adhesive bandage (e.g. Bandaid™)
11. Topical antibiotic ointment or cream

Components for Postoperative Care

1. **Care for the incisions**. Uneventful incision healing is generally expected after AV access creations. The incidence of incision complications is typically low. Topical antibiotic ointment or cream may be used for mild inflammation. Rarely,

delayed incision healing may need cleaning and re-suturing. Necrotic or infected tissue may need debridement to assure subsequent healing.

2. **Assess the functionality of AV access**. Various methods may be used to assess the functionality of AV accesses, ranging from physical examination to invasive procedures. A prosthetic AVG can be cannulated in 1–2 weeks provided the graft remains patent, and there is no significant tissue edema, peri-graft seroma, or any evidence of infection. The assessment for the functionality of an autogenous AVF is conducted 4–6 weeks after creation and requires considerable experiences and expertise (Table 25.5, Figs. 25.6 and 25.7).
3. **Salvage nonfunctional or failed AV access**. A major challenge of AV access surgery is that relatively high percentage of AV access created either fail or is nonfunctional at initial evaluation. Encouragingly, most of these AV accesses can be saved and used subsequently for hemodialysis therapy. Open surgery, endovascular interventions, or a combination of both may be employed to successfully salvage these AV accesses. The choice between open and endovascular interventions may be influenced by clinical scenario and local expertise. Specific clinical situations and management considerations are presented in Table 25.6 [1].

Table 25.5 Assessing the functionality of autogenous AV access

Components	Definitions, findings, and interpretations
Functionality	A mature AVF requires three components: Adequate diameter to permit safe cannulation, adequate access flow to permit efficient dialysis, and sufficiently superficial to permit accurate and safe cannulation
	The specific requirements for these components may vary geographically (due to variation in dialysis practices and technical skills)
Physical examination	Physical examination is very valuable tool
	A normal AVF has a soft pulse and continuous bruit, collapse with arm elevation, and augments sufficiently with access compression
	May be used to estimate access diameter, its depth from the skin, the presence of accessory veins, the presence of inflow and outflow stenosis and other abnormalities
Duplex ultrasound examination	Ultrasound is helpful when physical evaluation is equivocal or abnormal findings are present
	Ultrasound may measure the three required components of access functionality
	When access diameter is ≥4 mm, access blood flow is ≥500 ml/min and access is superficial, there is 95% likelihood of being usable for dialysis [1]
	Ultrasound may identify anatomical lesions and provide guidance for further interventions: Venous, arterial, anastomosis stenosis; competing veins or large accessory branches; excessive depth from the skin
Contrast angiography	Contrast angiography can identify anatomical lesions affecting maturation and functionality of AV access
	Corrective endovascular or surgical interventions can be performed at the same time: Angioplasty, stenting, obliteration of accessory veins, and surgical revisions

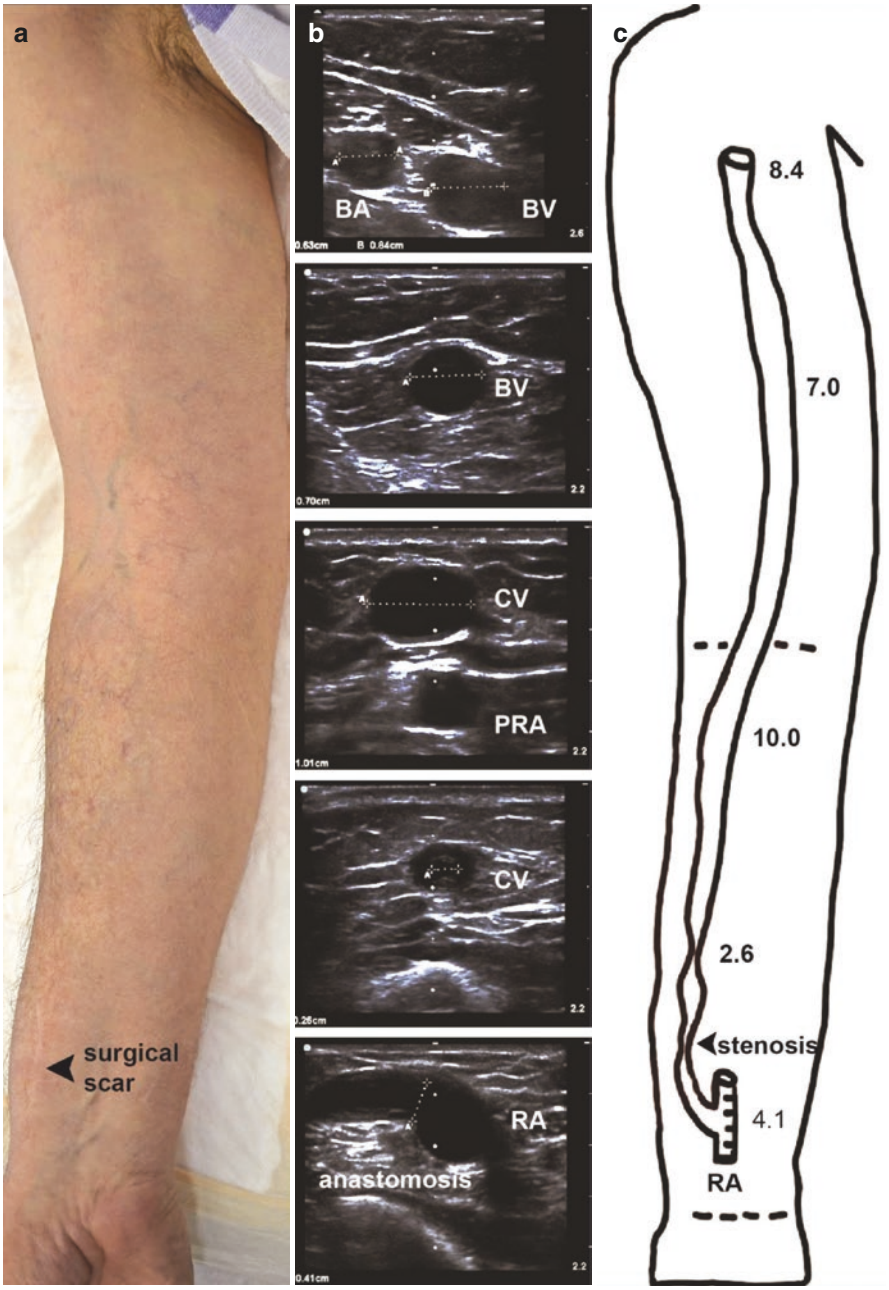

Fig. 25.6 Ultrasound evaluation of an AVF. (**a**) Photograph showing an arm with a forearm AV fistula. (**b**) Selected ultrasound images of the fistula conduit. (**c**) A freehand diagram may be used to illustrate the ultrasound findings as a supplement. Stenotic lesions of the cephalic vein are identified in this study. *RA* radial artery, *PRA* proximal radial artery, *CV* cephalic vein, *BV* basilic vein. Numbers shown are vessel diameters in millimeters

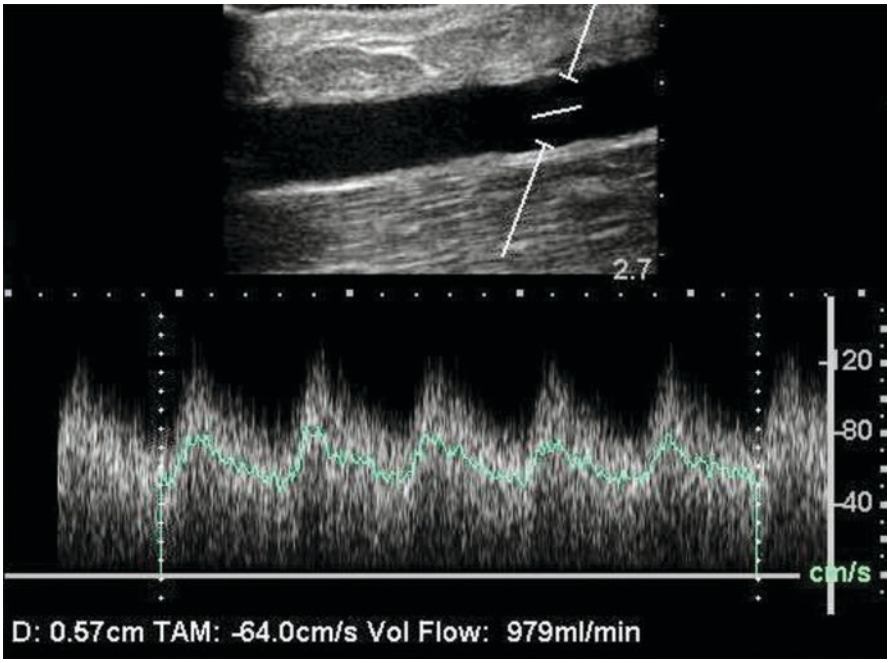

Fig. 25.7 Doppler ultrasound evaluation of AVF flow. Doppler ultrasound may be employed to measure the fistula flow non-invasively. This measurement may supplement the findings of physical examination and ultrasound imaging

4. **Manage AV access-associated complications**. AV access-associated complications may occur in the early postoperative period (<30 days) or subsequently that may require surgical attention. Common early postoperative complications may include incision complications, hematoma or other fluid collections, and distal ischemia. Common late access-associated complications may include venous hypertension, ischemic steal syndrome, aneurysm, bleeding, high-output heart failure, and infection. Brief discussions of these complications and their management are presented in Table 25.7 and 25.8 [7–10].
5. **Use of adjunctive therapy**. According to a recently published practice guideline, there are insufficient data to support the routine use of aspirin, ticlopidine, clopidogrel, ticagrelor, prasugrel, dipyridamole, sulphinpyrazone, warfarin or other oral anticoagulants, fish oil, statins, vonapanitase, glyceryl trinitrate, iontophoretic injection of salvia miltiorrhiza or prednisolone for improving AVF maturation or maintaining the long-term arteriovenous access patency. Hand-and-arm exercise and far infrared therapy may be considered for improving AVF maturation [11].

Table 25.6 Management of nonfunctional or failed AV access

Clinical situations	Management considerations
Access too deep	A deep AV access needs to be managed to avoid difficulty with cannulation and trauma to the patient
	A deep prosthetic graft is typically due to technical error during placement. Consider lipectomy, graft revision or new graft
	A deep AV fistula should be evaluated with ultrasound first to identify anatomical details. Consider elevation, lipectomy, or transposition
Side branches	Large (>2 mm), patent side-branch may affect AVF maturation especially if it has significant flow (the presence of bruit while transiently occluding the main outflow vein proximally is a good indicator)
	A fistula needs to be image studied to rule out stenosis that may cause side branch dilation
	Consider surgical ligation or endovascular coil embolization if no stenosis identified
Insufficient arterial inflow	More often an issue for AVF than AVG
	Arterial anastomosis stenosis most common, followed by subclavian artery stenosis
	Consider angioplasty or surgical revision
Poor venous outflow	May locate anywhere along the access conduit
	Common locations are post-arterial anastomosis segment for AVF and venous anastomosis for AVG
	Consider endovascular intervention or surgical revision
Thrombosis	A major complication of AV accesses
	May occur for various reasons. Stenotic lesions of the artery, anastomosis, or veins often present
	Mainstay of clotted access management is clot elimination and treatment of stenotic lesions
	Endovascular techniques, open surgical management, or combination of both may be employed to restore patency of a clotted access

Table 25.7 Management of AV access-associated complications [7]

Complications	Management considerations
Bleeding	Perioperative or post dialysis cannulation
	Etiology multifactorial, platelet dysfunction, aspirin, clopidogrel, warfarin, venous obstruction
	Consider: Vasopressin, cryoprecipitate, estrogen, restore red cell volume, enhanced dialysis, direct pressure, suture, repair or revision, angioplasty
Infection	May be related to surgery (early, <30 days) or dialysis cannulations (late, > 30 days)
	Bacteria of skin origin are most common
	Much more frequent in AVG than in AVF
	Duplex ultrasound or tagged leukocyte scan may help evaluation
	AVF: May resolve with antibiotics or local surgical intervention
	AVG: Requires partial or total excision of graft

Table 25.7 (continued)

Complications	Management considerations
Fluid collections (noninfectious)	Differentiate from abscess and pseudoaneurysm
	Hematoma may simply be observed or aspirated
	Seroma is related to prosthetic graft. Consider: Aspiration, debridement or graft replacement
	Lymphocele or lymphorrhea may be managed by compression or surgical exploration
Pseudoaneurysm and aneurysm	Pseudoaneurysm occurs mostly in AVG, rarely in AVF. Consider observation, surgical repair or revision, stent-graft placement (avoid if possible)
	Aneurysm occurs in AVF. Only need intervention if there are associated complications [8]
Venous hypertension	Due to outflow obstruction and increased access flow
	Often manifest as edema or access dysfunction
	Catheters and cardiac devices are major culprits
	Side-side AV anastomosis may cause incompetent valve and retrograde venous hypertension. Consider retrograde branch ligation
	Central vein stenosis: Consider angioplasty, stent-graft, and surgical bypass
	Regional stenosis (such as graft venous anastomosis and cephalic arch stenosis): Angioplasty, stent-graft, and surgical bypass or outflow transposition
	Superior vena cava obstruction has worse clinical consequences
Distal ischemia (steal syndrome)	Incidence higher with AVG than with AVF
	Over half develop within 30 days of surgery
	Clinical presentations vary
	Cases with mild symptoms may be observed
	Cases with significant symptoms need clinical and arteriogram evaluation and further management
	Table 25.8 Lists pathologies and management options
High-output heart failure	Rare, essentially a complication of autogenous AVF
	Differentiate from heart failure not related to access
	Present as right heart failure
	AV access flow >3 L/min or > 30% of cardiac output is consistent with diagnosis, but heart failure may occur at lower access flow. Symptoms typically improve after access flow management
	Consider access flow reduction procedures, rarely access ligation
Neuropathy	Peripheral neuropathy is very common in patients with advanced kidney disease or on dialysis
	Diabetic and uremic polyneuropathy are bilateral
	Ischemic monomelic neuropathy is rare and characterized by neural deficit without arterial insufficiency. Need access ligation or revision urgently
	Entrapment neuropathy (median or ulnar nerve) is common in dialysis patients and may be incited or exacerbated by AV access creation. Consider decompression if symptoms are significant
	Electrodiagnostic studies may help differentiation and guide management

Table 25.8 Managing distal ischemia based on underlying pathologies [9, 10]

Clinical pathologies	Management considerations
Excessive access flow	Flow reduction procedures (DAB or balloon assisted banding, RUDI)
	May need reconstruction of inflow segment if this segment is large (>15 mm), plus DAB
Artery stenosis proximal to anastomosis	Angioplasty, possible stent
Artery stenosis distal to anastomosis	Angioplasty, stent if possible
	Flow reduction, PAI
Distal radial artery ischemic steal	Flow reduction
	Occlusion of distal radial artery
Distal ulnar artery ischemic steal	Flow reduction
	Occlusion of distal ulnar artery
Ulnar artery lesion in radiocephalic fistula	Angioplasty, stent
	PAI
Low-normal access flow without localized lesion	PAI or DR
	Access ligation if intervention ineffective
Limb/tissue threatening ischemia	PAI
	Access ligation if intervention ineffective
Ischemic monomelic neuropathy	Differentiate from steal syndrome, access ligation urgently

DAB dilator-assisted Banding, *RUDI* revision using distal inflow, *PAI* proximalization of arterial inflow, *DR* distal revascularization (no interval ligation)

Tips and Troubleshooting

Potential issues and complications that may be encountered before and after surgical creations of AVF and AVG are discussed in Table 25.9.

Table 25.9 Potential issues and complications

Problem	Trouble shooting
Vasoconstriction (small vessels)	Ambient temperature can significantly influence the diameter of the blood vessels. Low temperature causes vasoconstriction of both arteries and veins, while high temperature causes vasodilation. An ambient temperature of 75–80 Fahrenheit mimics typical natural environments and is recommended during ultrasound vessel mapping
Image studies of vessels relevant to vascular access	Ultrasound vessel mapping: Essential, but cannot visualize central vessels
	Contrast venography: Mainly used to assess central veins when central lesion suspected
	Arteriography: May consider when there is significant peripheral vascular disease
	MR angiography: Rarely used
Strategies for minimizing contrast nephropathy	Limit contrast quantity is a key strategy
	Hydration, using isotonic solution (such as normal saline)
	Carbon dioxide gas as an alternative contrast material. Requires separate equipment
Ischemic steal syndrome (post surgery)	Distal ischemia may occur any time after an AV access surgery. The incidence is substantially higher after AVG than after AVF creations
	1. Mild-moderate distal ischemia symptoms occur during the early postoperative period may be minimized with intermittent compression of the AVF or AVG by the patient or family member (<5 min at a time)
	2. For significant distal ischemia symptoms during early postoperative period after AVG placements, the AVG may be intentionally thrombosed or ligated. Distal ischemia symptoms often do not return when the graft flow is re-established later (in 2 weeks)
	3. If possible, the etiology of distal ischemia should be investigated endovascularly. The access can be preserved and distal ischemia managed based on underlying pathophysiology (Table 25.8)
	4. If distal ischemia is due to excessive access flow, various techniques may be utilized to limit the access flow to enhance the distal circulation. Dilator-assisted banding is a simple, economical approach that may be used even without fluoroscopic guidance [9]. Distal circulation may be assessed intra-operatively with pulse oximetry
	5. In patients with significant peripheral vascular disease and the predicted incidence of distal ischemia is high, the proximal brachial artery or axillary artery may be used for anastomosis to minimize distal ischemia
Arm edema post access surgery	Arm edema occurs more frequently after AVG placements than AVF creations. Near peak access flow is produced soon after AVG placements, which is different from the gradual increase of access flow after AVF creations. Arm edema often resolves spontaneously 2–3 weeks after AVG placements, presumably due to adaptation of the vasculature. Persistent edema often suggests draining vein stenosis and needs to be investigated with venography. Arm edema can incite or exacerbate carpal tunnel syndrome, which needs to be differentiated from distal ischemia

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