Eur J Vasc Endovasc Surg (2018) 55, 757—818

Editor's Choice — Vascular Access: 2018 Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS)☆

Jürg Schmidli [a,*], Matthias K. Widmer [a], Carlo Basile [a], Gianmarco de Donato [a], Maurizio Gallieni [a], Christopher P. Gibbons [a], Patrick Haage [a], George Hamilton [a], Ulf Hedin [a], Lars Kamper [a], Miltos K. Lazarides [a], Ben Lindsey [a], Gaspar Mestres [a], Marisa Pegoraro [a], Joy Roy [a], Carlo Setacci [a], David Shemesh [a], Jan H.M. Tordoir [a], Magda van Loon [a],

ESVS Guidelines Committee [b], Philippe Kolh, Gert J. de Borst, Nabil Chakfe, Sebastian Debus, Rob Hinchliffe, Stavros Kakkos, Igor Koncar, Jes Lindholt, Ross Naylor, Melina Vega de Ceniga, Frank Vermassen, Fabio Verzini,

ESVS Guidelines Reviewers [c], Markus Mohaupt, Jean-Baptiste Ricco, Ramon Roca-Tey

Keywords: Guideline, Arteriovenous access, Vascular access, Arteriovenous fistula, Arteriovenous graft, Renal insufficiency, Haemodialysis, Surveillance, Complications, ESRD

TABLE OF CONTENTS

☆ The ESVS guidelines for Vascular Access are endorsed by the Vascular Access Society (VAS).

[a] Writing Group: Jürg Schmidli* (Bern, Switzerland), Matthias K. Widmer (Bern, Switzerland), Carlo Basile (Bari, Italy), Gianmarco de Donato (Siena, Italy), Maurizio Gallieni (Milan, Italy), Christopher P. Gibbons (Banbury, UK), Patrick Haage (Witten, Germany), George Hamilton (London, UK), Ulf Hedin (Stockholm, Sweden), Lars Kamper (Witten, Germany), Miltos K. Lazarides (Alexandroupoli, Greece), Ben Lindsey (London, UK), Gaspar Mestres (Barcelona, Spain), Marisa Pegoraro (Milan, Italy), Joy Roy (Stockholm, Sweden), Carlo Setacci (Siena, Italy), David Shemesh (Jerusalem, Israel), Jan H.M. Tordoir (Maastricht, The Netherlands), Magda van Loon (Maastricht, The Netherlands).

[b] ESVS Guidelines Committee: Philippe Kolh (Liege, Belgium), chair), Gert J. de Borst (Utrecht, Netherlands, co-chair and guideline coordinator), Nabil Chakfe (Strasbourg, France), Sebastian Debus (Hamburg, Germany), Rob Hinchliffe (Bristol, UK), Stavros Kakkos (Patras, Greece), Igor Koncar (Belgrade, Serbia), Jes Lindholt (Odense, Denmark), Ross Naylor (Leicester, UK), Melina Vega de Ceniga (Galdakao, Spain), Frank Vermassen (Ghent, Belgium), Fabio Verzini (Perugia, Italy).

[c] ESVS Guidelines Reviewers: Markus Mohaupt (Bern, Switzerland), Jean-Baptiste Ricco (Strasbourg, France), Ramon Roca-Tey (Barcelona, Spain).

* Corresponding author. Bern University Hospital, University of Bern, Freiburgstrasse, 3010 Bern, Switzerland. Tel: +41 31 632 2602; Fax: +41 31 632 2919.
E-mail address: juerg.schmidli@insel.ch (Jürg Schmidli).

https://doi.org/10.1016/j.ejvs.2018.02.001

ABBREVIATIONS

ABBREVIATION AND TERM (SYNONYM)

ABI	Ankle brachial index
ACE	Angiotensin converting enzyme
AVF	Arteriovenous fistula (Synonym: Autogenous or native fistula)
AVG	Arteriovenous graft (Synonym: Prosthetic graft)
BBAVF	Brachiobasilic AVF
BCAVF	Brachiocephalic AVF
BVT	Basilic vein transposition
CE-MRA	Contrast enhanced magnetic resonance angiography
CHF	Congestive heart failure
CKD	Chronic kidney disease
CO	Cardiac output
CO_2	Carbon dioxide
CPR	Cardiopulmonary recirculation
CTA	Computed tomography angiography
CVD	Cardiovascular disease
CVC	Central venous catheter
CVOD	Central venous occlusive disease
DBI	Digital brachial index
DOPPS	Dialysis outcomes and practice patterns study
DEB	Drug eluting balloon
DRIL	Distal revascularisation and interval ligation
DSA	Digital subtraction angiography
DUS	Duplex ultrasonography
ePTFE	expanded polytetrafluoroethylene
EJVES	European Journal of Vascular and Endovascular Surgery
ESC	European Society of Cardiology
ESRD	End stage renal disease
ESVS	European Society for Vascular Surgery
GFR	Glomerular filtration rate
GSV	Great saphenous vein
HD	Haemodialysis
HD catheter	Catheter of any kind used for haemodialysis
HeRO®	Haemodialysis Reliable Outflow device
HIV	Human immunodeficiency virus
IMN	Ischaemic monomelic neuropathy
IVC	Inferior vena cava
KDOQI	Kidney diseases outcome quality initiative
Kt/V	Dialysis rate
LEAVG	Lower extremity AVG
LEAD	Lower extremity atherosclerotic disease
LMWH	Low molecular weight heparin
MAP	Mean arterial pressure
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
MRSA	Methicillin resistant *Staphylococcus aureus*
NCE-MRA	Non-contrast enhanced magnetic resonance angiography

NIH	Neointimal hyperplasia (Synonym: Myointimal hyperplasia)
NKF-KDOQI	National Kidney Foundation for Kidney disease outcome quality initiative
NPWT	Negative pressure wound therapy
NSF	Nephrogenic systemic fibrosis
ntCVC	Non tunnelled central venous catheter (Synonym: indwelling catheter without cuff)
PAVA	Proximalisation of the arteriovenous anastomosis
PD	Peritoneal dialysis
PICC	Peripherally inserted central venous catheter
PNV	Pre-nephrology visit
PTA	Percutaneous transluminal angioplasty (Synonym: balloon angioplasty)
Qa	Access blood flow
Qb	Blood pump flow delivered to the dialyser
RCAVF	Radiocephalic AVF (Synonoym: Brescia-Cimino fistula)
RCT	Randomised controlled trial
RRT	Renal replacement therapy
RUDI	Revision using distal inflow
SFA	Superficial femoral artery
FV	Femoral vein (formerly superficial femoral vein)
FVT	Femoral vein transposition
Stent graft	Former covered stent
tcCVC	Tunnelled cuffed central venous catheter (Synonym: indwelling catheter with cuff)
UDT	Ultrasound dilution technique
URR	Urea reduction ratio
VA	Vascular access
VAILI	Vascular access induced limb ischaemia
VAS	Vascular Access Society
VP	Venous pressure
VP/MAP	Venous pressure adjusted for the mean arterial pressure
WC	Writing Committee

1. METHODOLOGY AND GRADING OF RECOMMENDATIONS

1.1. Purpose

The European Society for Vascular Surgery (ESVS), in line with its mission, appointed the Vascular Access (VA) Writing Committee (WC) to write the current clinical practice guidelines document for surgeons and physicians who are involved in the care of patients with haemodialysis (HD) and VA. The goal of these Guidelines is to summarise and evaluate all the currently available evidence to assist physicians in selecting the best management strategies for all patients needing VA or for pathologies derived from a VA. However, each physician must make the ultimate decision regarding the particular care of an individual patient.[1,2]

Patients with VA for HD are complex and also subject to significant clinical practice variability, although a valid evidence base is available to guide recommendations. The significant technical and medical advances in VA have enabled guidelines to be proposed with greater supporting evidence than before. Potential increases in healthcare costs and risks due to industry and public driven use of novel treatment options make the current guidelines increasingly important.[3–6]

Many clinical situations involving patients with HD and VA have not been studied by randomised clinical trials. Nevertheless, patient care must be delivered and clinical decisions made in these situations. Therefore, this document should also provide guidance when extensive level A evidence is unavailable and in these situations recommendations are determined on the basis of the best currently available evidence.

By providing information on the relevance and validity of the quality of evidence, the reader will be able to gather the most important and evidence based information relevant to the individual patient.

This document is intended to be a guide, rather than a set of rules, allowing flexibility for specific patients' circumstances. The current clinical practice guidelines document provides recommendations for the clinical care of patients with HD and VA including pre-operative, peri-operative and post-operative care and long-term maintenance.

1.2. Methodology

The VA WC was formed by members of the ESVS and Vascular Access Society (VAS) from different European countries, various academic and private hospitals, and includes vascular surgeons, nephrologists, radiologists and clinical nurses in order to maximise the applicability of the final guideline document. The WC met in September 2012 for the first time to discuss the purpose, contents, methodology and timeline of the following recommendations.

The VA WC has performed a systematic literature search in the MEDLINE, EMBASE and COCHRANE Library databases for each of the different topics that are discussed and reviewed in this guidelines document. The latest literature search was performed by August 31st 2017. With regard to the evidence gathered, the following eligibility criteria have been applied:

- Only peer reviewed published literature has been considered
- Published abstracts or congress proceedings have been excluded
- Randomised clinical trials (RCT) as well as meta-analyses and systematic reviews were searched with priority
- Non-RCTs, non-controlled trials and well conducted observational studies (cohort and case control studies) were also included

- Previous guidelines, position papers and published consensus documents have also been included as part of the review process when new evidence was absent
- Minimising the use of reports of a single medical device or from pharmaceutical companies reduced the risk of bias across studies. A grading system based on the European Society of Cardiology (ESC) guidelines methodology was adopted.[7] The level of evidence classification provides information about the study characteristics supporting the recommendation and expert consensus, according to the categories shown in Table 1.

Table 1. Levels of evidence.[7]

Level of Evidence A	*Data derived from multiple randomised clinical trials or meta-analyses.*
Level of Evidence B	*Data derived from a single randomised clinical trial or large non-randomised studies*
Level of Evidence C	*Consensus of opinion of the experts and/or small studies, retrospective studies, registries.*

The recommendation grade indicates the strength of a recommendation. Definitions of the classes of recommendation are shown in Table 2.

Table 2. Grades of strength of recommendations according to the ESC grading system.[7]

Classes of Recommendations	Definition
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, effective.
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure.
Class IIa	*Weight of evidence/opinion is in favour of usefulness/efficacy.*
Class IIb	*Usefulness/efficacy is less well established by evidence/opinion.*
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.

For each recommendation, two members of the WC assessed the strength of a recommendation and the quality of supporting evidence independently. A full master copy of the manuscript with all recommendations was electronically circulated and approved by all WC members. Recommendations that required consensus were discussed and voted upon at meetings and by email among all members of the WC. This system permitted strong recommendations supported by low or very low quality evidence from downgraded RCTs or observational studies only when a general consensus among the WC members and reviewers was achieved. Meta-analyses are quoted in the recommendations according to the following rule: if the recommendation was either of high or low quality the meta-analysis was quoted and the individual studies were not explored. If it was a "grey area" and mixed opinions on the included meta-analysis studies were present, the original data were examined to clearly present the "mixed" findings within several studies. Two members of the WC have prepared each part of the guidelines document. An internal review process was performed before the manuscript was sent to the ESVS Guidelines Committee and selected invited independent external reviewers. External reviewers made critical suggestions, comments and corrections on all preliminary versions of these guidelines. In addition, each member participated in the consensus process concerning conflicting recommendations. The final document has been approved by the ESVS Guidelines Committee and submitted to the European Journal of Vascular and Endovascular Surgery (EJVES). Further updated guidelines documents on VA will be provided periodically by the ESVS when new evidence and/or new clinical practice arise in this field, which could occur every three years.

To optimise the implementation of the current document, the length of the guidelines has been kept as short as possible to facilitate access to guideline information. Conflicts of interest from each WC member were collected prior to the writing process. These conflicts were assessed and accepted by each member of the WC and are reported in this document. In addition, the WC agreed that all intellectual work should be expressed without any interference beyond the honesty and professionalism of all its members during the writing process.

1.3. Definitions

1.3.1. Definition of vascular access. Patients with acute renal failure or end stage renal disease require renal replacement therapy, which includes peritoneal dialysis (PD), haemodialysis (HD) or kidney transplantation (Fig. 1). A VA is essential for patients on HD and can be accomplished with central venous catheters (CVC), but also with arterialisation of a vein or by interposition of a graft between an artery and a vein for the insertion of HD needles. The blood flow available for HD should reach at least 300 ml/min and preferably 500 ml/min depending on the VA modality to allow a sufficient HD.

1.3.2. Other definitions. Arteriovenous fistulas (AVFs) and arteriovenous grafts (AVGs) are established terms to characterise a special kind of VA in patients on HD. An AVF is defined as an autogenous anastomosis between an artery and a vein and an AVG is defined as a VA using a prosthetic graft.

At the beginning of this millennium interventional radiologists and vascular surgeons attempted to clarify the terminology dealing with HD access.[8–10] Some of these definitions have been revised and[11] further refinements made; there is still ongoing discussion amongst VA specialists. Nevertheless outlined below are the definitions that are believed to be currently accepted by the majority of clinicians in the field.

Incidence is the proportion of a given population developing a new condition or experiencing an event within a specified period of time. This could be for example, the number of patients experiencing an event (e.g. patients undergoing VA creation) divided by the number of a given

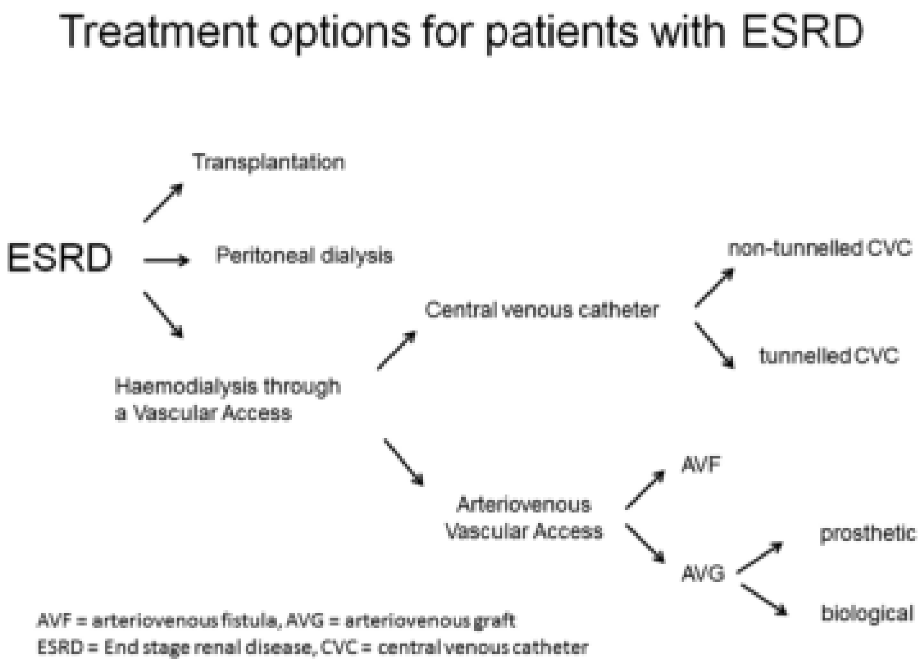


Figure 1. Flowchart of renal replacement treatment options.

population (e.g. the number of patients undergoing HD). For a disease, incidence can be expressed as the number of patients per million population per year.

Prevalence is the total number of cases of a disease within a given population; it includes both new and continuing patients with a certain disease and is expressed as number of patients per million population. Prevalence is a function of incidence (new cases) and outcomes (death or cure).

Point prevalence in %: Number of patients using a specific type of VA at a given point of time multiplied by 100 and divided by the number of patients with a VA at this time.

Period prevalence in %: The mean number of patients using a specific VA over a given time (one year) multiplied by 100 and divided by all the patients using a VA during the same time period.

Hospitalisation days/1000 access days: The numerator is the total number of days of hospitalisation for the study population. The denominator is calculated as the number of days from VA creation or the start date of a study period to permanent (unsalvageable) VA failure, the end of study period, death of the patient, transfer from the dialysis unit or a change in renal replacement modality (PD or transplantation). The calculated rate is the total number of hospitalisation days/total number of VA days multiplied by 1000 to express the number of hospitalisation days per 1000 VA days.

Access abandonment: The day on which a VA is deemed to be permanently unusable or not suitable for cannulation.

Primary VA: Creation of a functioning VA for the first time.

Secondary VA: Ordinary VA creation with AVF or AVG at any location after a failed primary VA (tertiary VA excluded).

Tertiary VA: VA using great saphenous vein (GSV) or femoral vein (FV) translocated to the arm or leg. Unusual VA procedures such as upper or lower limb arterio-arterial loops are included in this category.

Transposition: Relocation of an autogenous vein to a new (more superficial) position in the soft tissues of the same anatomical area (e.g. an upper arm AVF with transposition of the basilic vein).

Translocation: The prepared vein is completely disconnected and inserted in a new anatomical area to create an AVF.

Superficialisation: The index vein is transposed in the subcutaneous tissue and positioned closer to the skin.

Kaplan-Meier life table analysis: A statistical method for calculating time dependent clinical outcomes can be documented such as VA patencies, or infection free survival rates.

Primary patency: The interval between VA creation and the first re-intervention (intervention free VA survival) for VA dysfunction or thrombosis, the time of measurement of patency or the time of its abandonment.

Assisted primary patency: The interval between VA creation and the first occlusion (thrombosis free VA survival) or measurement of patency including operative/endovascular interventions to maintain the VA.

Primary functional patency: The interval between the first use (first cannulation) of a newly created VA and the first re-intervention to rescue the VA or to its abandonment.

Secondary patency: The interval between VA creation and the abandonment of this VA (i.e. thrombosis) after one or more interventions or the time of measurement of patency including achievement of a censored event (death, change of HD modality, loss of follow-up).

Maturation and functionality of VA: Changes that occur in the VA after its creation (increase in VA flow and AVF diameter, wall structure changes, AVG tissue to graft incorporation) making it suitable over time for cannulation.

Mature VA: A VA that is expected to be suitable for HD access and considered appropriate for cannulation with two needles and expected to deliver sufficient blood flow throughout the HD. Therefore it is a pre-cannulation definition.

Functional VA: A VA is functional when it has been cannulated successfully with two needles, over a period of at least 6 HD sessions during a 30 day period, and delivered the prescribed blood flow throughout the HD and achieved adequate HD (usually at least 300 ml/ min). Therefore, it is a post-cannulation definition.

Monitoring: Examination and evaluation of the VA by means of physical examination to detect physical signs that suggest the presence of VA dysfunction.

Surveillance: Periodic evaluation of a VA using haemodynamic tests. This may trigger further diagnostic evaluation.

VA induced (limb) ischaemia: Extremity malperfusion after VA creation. It can be classified in four stages:

stage 1: slight coldness, numbness, pale skin, no pain
stage 2: loss of sensation, pain during HD or exercise
stage 3: rest pain
stage 4: tissue loss affecting the distal parts of the limb, usually the digits

This definition is more appropriate than 'steal' which describes the physiological phenomenon of (even retrograde) blood flow recruitment towards the AVF/AVG.

Recirculation: The return of dialysed blood to the systemic circulation without full equilibration (NKF-DOQI definition).

Kt/V: A parameter to quantify the adequacy of the HD: K = Dialyser clearance of urea, t = effective time of HD V = volume of urea distribution, approximately equal to the patient's body water (60% of the body mass).

Early VA failure: A VA that has occluded within 24 hours of creation.

Early dialysis suitability failure: A VA that cannot be used by the third month following creation despite radiological or surgical intervention.

Late dialysis suitability failure: A VA that is not usable after more than 6 months despite radiological or surgical intervention.

Cannulation failure: Failure is defined as the inability to place and secure two dialysis needles.

Non-tunnelled CVC (ntCVC): An uncuffed catheter providing temporary VA for HD.

Tunnelled cuffed CVC (tcCVC): A subcutaneously tunnelled dual lumen catheter with a cuff that can be used for VA if HD is expected to last for more than two weeks.

Catheter related bacteraemia:

Proven: Bacteraemia with at least one positive percutaneous peripheral vein blood culture and where either the same pathogen was cultured from the catheter tip or a blood culture drawn from a catheter that has a >3 fold greater bacterial colony count than those drawn from a peripheral vein.

Probable: Bacteraemia with positive blood cultures obtained from a catheter and/or peripheral vein in a patient where there is no clinical evidence of an alternative source of an infection.

Catheter exit site infection:

Proven: The presence of a purulent discharge or erythema, induration/and or tenderness at the catheter exit site with a positive bacteriological culture of the serous discharge.

Probable: The clinical signs of infection with negative cultures from the discharge or blood without signs of irritation from gauze, stitches or the cleansing agent.

Catheter tunnel infection:

Proven: The presence of purulent discharge from the tunnel or erythema, induration and/or tenderness over the catheter tunnel with a positive culture.

Probable: Clinical signs of infection around the catheter site with negative cultures from the discharge or blood.

Primary catheter site patency: Interval between catheter insertion and the first intervention to restore the catheter's function.

Secondary catheter site patency: Interval between catheter insertion and exchange or removal of the catheter for any reason.

Continuous catheter site: The time period from initial catheter insertion to catheter site abandonment for any reason including the time period after continuous catheter exchanges in the same target vessel. The time period and number of exchanges are documented e.g. 12 months [3 catheters].

Catheter dysfunction: This is the first occurrence of either a peak flow of 200 ml/minute or less for 30 minutes during HD, a mean blood flow of 250 ml/minute or less during two consecutive dialyses or the inability to initiate HD resulting from an inadequate blood flow, despite attempts to restore patency.

2. EPIDEMIOLOGY OF CHRONIC KIDNEY DISEASE (CKD) STAGE 5

2.1. Epidemiology of chronic kidney disease

Chronic kidney disease (CKD) is a worldwide public health problem. CKD is classified into five stages (Table 3), but renal insufficiency is restricted to stages 3–5, with a glomerular filtration rate (GFR) below 60 ml/min per 1.73 m^2 for 3 months or more irrespective of the cause.[12]

Table 3. Classification of chronic kidney disease based on glomerular filtration rate (GFR).[8–11]

Stage	Description	GFR mL/min/1.73 m^2
Stage 1	Kidney damage with normal or elevated GFR	90+
Stage 2	Kidney damage with mildly decreased GFR	60–89
Stage 3	Moderately decreased GFR	30–59
Stage 4	Severely decreased GFR	15–29
Stage 5	End stage renal disease (ESRD)	<15 or on dialysis

The true incidence and prevalence of CKD within a community are difficult to ascertain as early to moderate CKD is usually asymptomatic. Most studies point to a prevalence of

CKD of around 10%, albuminuria of around 7%, and GFR below 60 ml/min per 1.73 m^2 of around 3%.[13–15]

CKD stage 5 (ESRD) is characterised by GFR below 15 ml/min per 1.73 m^2 and includes two phases: the first one is treated conservatively without dialysis; when the second phase follows, the initiation of renal replacement therapy (RRT) in the form of dialysis or transplantation is required to sustain life.

The incidence of CKD stage 5 refers to the number of patients with ESRD beginning RRT, thus failing to take into account patients not treated by RRT and underestimating the overall true incidence of ESRD. In the dialysis population, prevalence is a function of the incidence (new cases) and outcome (transplantation or death) rates of ESRD.

2.1.1. Epidemiology of end stage renal disease

2.1.1.1. Incidence. The number of patients per year starting RRT has shown an exponential rise.[16] Such a large number of CKD patients requiring dialysis may have three main causes: patient selection, competitive risks and a true increase in CKD incidence:

1. *Selection of patients for RRT:* the steep increase in the incidence of older patients suggests that those very old and/or those affected by particularly severe comorbidities were not given access to dialysis in the first decades of RRT, compared with the more recent years.

2. *Competitive risks*: a study suggested that the number of deaths where CKD is the underlying cause of death increased by 82% between 1990 (27th in the global death rank) and 2010 (18th in the global death rank).[17] A high risk of death exists even in patients in the early stages of CKD, with many individuals in stages 3 and 4 dying before starting RRT.[18,19] In fact, a reduced GFR is considered one of the most important risk factors for coronary heart disease.[20] Substantial improvements in the treatment of cardiac diseases and in survival have occurred in recent decades and this has allowed many patients to survive in the more advanced CKD stages and to require RRT.

3. *The true increase in CKD incidence*: it may also be possible that the increased incidence of ESRD reflects increases in the underlying prevalence of CKD. The Framingham Heart Study has shown that the incidence of type 2 diabetes has doubled from the 1970s to the 1990s.[21] Furthermore, potentially nephrotoxic drugs, such as non-steroidal anti-inflammatory drugs, antibiotics and chemotherapy agents are used more commonly. Finally, reduced mortality from cardiovascular diseases and cancer may be associated with an increase in the number of patients reaching ESRD.

2.1.1.2. Prevalence. Data related to the prevalence of CKD stage 5 are lacking, except for those of registries of ESRD patients treated by dialysis or transplantation. In the USA, of the 547,982 prevalent ESRD patients in 2008, 70 percent were being treated by dialysis while 30 percent had a functioning kidney transplant. In 2008 alone, 112,476 patients entered the US ESRD program. Adjusted rates for incident and prevalent ESRD are 351 and 1,699 cases per million population, respectively. Diabetes and hypertension account for 44% and 27.9% of all causes of incident ESRD, respectively.[22]

The prevalence of a disease increases if the patient survival increases with a constant incidence rate or if the incidence rate increases with a constant survival rate. Thus the rising prevalence of treated ESRD can be attributed either to the increase in the number of patients who start RRT each year and/or to the increased survival of patients with ESRD. Since the incidence rates of treated ESRD have flattened in recent years, longer lifespans of prevalent ESRD patients may partially explain the steady growth of this population.[22] Continuing global efforts should be made in the prevention and treatment of acute and especially chronic conditions potentially leading to ESRD, in particular diabetes and hypertension.

2.2. Demographics of end stage renal disease

The global epidemiology of ESRD is heterogeneous and influenced by several factors. Consequently, the incidence and prevalence of ESRD are markedly different from country to country (Table 4). Disparities in the incidence and prevalence of ESRD within and between developed countries reflect racial and ethnic diversities as well as their impact on the prevalence of diabetes and hypertension in respective countries and communities. The incidence is higher among African and Native Americans and aboriginal people of Australia and New Zealand.[12,22–26] Diabetes as a cause of ESRD is particularly frequent in these populations. Disparities with developing countries are likely to reflect availability of and access to RRT in low and middle income economies rather than a lower incidence of CKD. Diabetes as the primary cause of CKD affects a particularly high percentage of incident patients in the USA.

The elderly are a substantial and growing fraction of the RRT population worldwide, reaching 25–30% in most ESRD registries.[22,24] In the United States, the proportion of patients >65 years of age starting dialysis has increased by nearly 10% annually, representing an overall increase of 57% between

Table 4. Global incidence and prevalence of RRT (per million population) in different parts of the world in 2002 and 2006.

	Incidence		Prevalence	
	2002	2006	2002	2006
UNITED STATES	333	360	1,446	1,626
Caucasians	255	279	1,060	1,194
African Americans	982	1,010	4,467	5,004
Native Americans	514	489	2,569	2,691
Asians	344	388	1,571	1,831
Hispanics	481	481	1,991	1,991
AUSTRALIA	94	115	658	778
Aboriginals, Torres Strait islanders	393	441	1,904	2,070
EUROPE	129	129	770	770
United Kingdom	101	113	626	725
France	123	140	898	957
Germany	174	140	918	957
Italy	142	133	864	1,010
Spain	126	132	950	991
JAPAN	262	275	1,726	1,956

Source: References[22,24,26,30]

1996 and 2003.[22] In Canada, from 1990 until 2001, the incident dialysis rate among patients aged 75 and older increased 74%.[25] Researchers have speculated that more liberal acceptance of the very elderly (≥80 years) into dialysis programs has contributed to the increase in patients with ESRD.[27,28]

CKD is expected to be a major 21st century medical challenge. In developing nations, the growing prevalence of CKD has severe implications on health and economic output.[29] The rapid rise of common risk factors such as diabetes, hypertension and obesity, especially among the poor, will result in even greater and more profound burdens that developing nations are not equipped to handle.[29]

2.3. Epidemiology of vascular access for dialysis

Large differences in VA exist between Europe, Canada, and the United States, even after adjustment for patient characteristics.[31] VA care is characterised by similar issues, but with a different magnitude. Obesity, type 2 diabetes, and peripheral vascular disease, independent predictors of CVC use, are growing problems globally, which could lead to more difficulties in native AVF creation and survival.

Nevertheless, in the USA following the establishment of the Fistula First Initiative, AVF use among prevalent HD patients increased steadily from 34.1% in December 2003 to 60.6% in April 2012.[32] In incident patients, VA statistics at the start of chronic HD in 2009 were: AVF in use 14.3%; AVG in use 3.2%; CVC in use 81.8%; AVF maturing 15.8%; AVG maturing 1.9%. Figures were similar in 2014.[33]

International data from DOPPS (dialysis outcomes and practice patterns study) has shown large variations in VA practice[34] and greater mortality risks have been seen for HD patients dialysing with a catheter, while patients with an usable AVF have the lowest risk.[35] International trends in VA practices have been observed within the DOPPS from 1996 to 2007.[34] Between 2005 and 2007, a native AVF was used by 67—91% of prevalent patients in Japan, Italy, Germany, France, Spain, the UK, Australia and New Zealand, and 50—59% in Belgium, Sweden and Canada. From 1996 to 2007, AVF use rose from 24% to 47% in the USA but declined in Italy, Germany and Spain. Across three phases of data collection, patients were consistently less likely to use an AVF versus other VA types if female, of greater age, having greater body mass index, diabetes, and peripheral vascular disease. In addition, countries with a greater prevalence of diabetes in HD patients had a significantly lower percentage of patients using an AVF. Despite poorer outcomes for CVCs, catheter use rose 1.5—3 fold among prevalent patients in many countries from 1996 to 2007, even among non-diabetic patients 18—70 years old. Furthermore, 58—73% of incident patients used a CVC for the initiation of dialysis in five countries despite 60—79% of patients having been seen by a nephrologist more than 4 months prior to ESRD. The median time from referral to VA creation varied from 5—6 days in Italy, Japan and Germany to 40—43 days in the UK and Canada. Surgery waiting time, along with time from VA creation to first cannulation, significantly affected the possibility of starting HD with a permanent VA.[34]

Patient preference for a CVC varied across countries, ranging from 1% of HD patients in Japan and 18% in the United States, to 42%—44% in Belgium and Canada.[36] Preference for a CVC was positively associated with age, female sex, and former or current catheter use. The observed considerable variation in patient preference for VA suggests that patient preference may be influenced by socio-cultural factors and thus could be modifiable.

The use of CVCs carries a significant risk of serious complications. Lately, in non-renal patients the peripherally inserted central venous catheter (PICC) has gained in popularity due to presumed advantages over other CVCs. However, the use of PICC lines is not indicated in CKD patients because of subsequent adverse VA outcomes, i.e. a lower likelihood (15%—19%) of having a functioning fistula or graft.[37]

Early referral of ESRD patients to the nephrologist is strongly recommended. This approach may minimise the use of catheters and reduce catheter related morbidity and the need for hospitalisation.[38] Early referral to the nephrologist is also required for interventions to delay progression of renal damage and to correct hypertension, anaemia and the metabolic effects of renal failure, discussion of renal replacement treatment options, including living related transplantation and peritoneal dialysis, and psychological preparation for dialysis.[39] When haemodialysis is the choice, time from referral to surgery for VA creation should be as short as possible.[34]

3. CLINICAL DECISION MAKING

3.1. Choice of type of vascular access

Successful HD treatment is only possible with a well functioning VA. The ideal VA should allow cannulation using two needles, deliver a minimum blood flow of at least 300 ml/min through the artificial kidney, is resistant to infection and thrombosis and should have minimum adverse events. The first option for the construction of a VA is the creation of an autogenous AVF. Secondary and tertiary options are prosthetic AVG and CVCs. The reason for creating autogenous AVFs is that observational studies show a lower incidence of postoperative complications and fewer endovascular and surgical revisions for AVF failure in comparison to AVGs.[40—42] In addition, the use of CVCs results in a significantly higher morbidity and mortality rate. The risk of hospitalisation for VA related reasons and particularly for infection is highest for patients on HD with a catheter at initiation and throughout follow-up.[43] The principle of venous preservation dictates that the most distal AVF possible should usually be performed.[44]

The strategy is to start HD in incident patients with a distal autogenous AVF preferably in the non-dominant upper extremity. In cases of a failed distal VA a more proximally located AVF can be performed.

3.2. Timing of referral for vascular access surgery

Timely patient referral for VA creation is of importance for the outcome of the VA. Early referral results in more well functioning autogenous AVFs,[45] while late referral results in a greater chance of AVF non-maturation and the need for a CVC for HD.[46–48] Moreover timely referral slows eGFR decline.[49] Also, HD initiation with a CVC and a long AVF maturation time, results in poorer long-term AVF patency rates. The same factors that predict worse primary AVF survival are also associated with greater risk of final failure. The presence of cardiovascular disease, use of catheters at HD initiation, and early cannulation are independent predictors of final failure. A short time to cannulation is associated with the greatest risk of final failure.[45] (Figs. 2 and 3) Frequent (every 3 months) pre-nephrology visits (PNV) are related to improved patient survival during the first year after initiation of HD, indicating the possible survival benefit with increased attention to PNV, particularly for elderly and diabetic patients.[50,51] From the DOPPS data, significant differences between European countries in referral type and time of VA creation have been reported. Planning of VA surgery varies between <5 days (Italy) to >42 days (UK) after referral to the VA surgeon.[34]

The knowledge and experience of the VA surgeon is of importance in creating predominantly AVFs and has a major impact on the outcome of surgery.[52,53] However, there remain large regional differences between hospitals, concerning the number of autogenous AVFs created and the probability of successful maturation.[54]

wrist RCAVF depends on the outcome of physical examination (inspection and palpation of distal veins and arteries) and additional ultrasound examination. A minimum internal vessel diameter for both radial artery and cephalic vein of 2.0 mm using a proximal tourniquet is considered to be adequate for successful fistula creation and maturation. For brachiocephalic (BCAVF) and brachiobasilic (BBAVF) AVFs a minimum arterial and venous diameter of 3 mm is sufficient.

Major disadvantages are the risk of early thrombosis and non-maturation and, ultimately, access failure. A meta-analysis showed a 17% mean early failure rate.[55] However, recent studies have shown higher failure rates of up to 46%, with one year patencies from 52% to 83% (Table 5). An elderly dialysis population with concurrent comorbidities and poor upper extremity vessels is the reason for these high early failure rates.[56]

When a wrist RCAVF is not possible or has failed, a more proximally located AVF in the forearm, antecubital region or upper arm may be performed. These accesses are called mid-forearm, brachial/radial-deep perforating vein,[57] brachial-median cubital vein, BCAVF and BBAVF. Brachial artery based AVFs deliver a high access flow which favours high HD flows, but may result in reduced distal arterial perfusion and cardiac overload.[58] These types of AVFs show good one year patencies (Tables 6 and 7) with a low incidence of thrombosis (0.2 events per patient/year) and infection (2%).

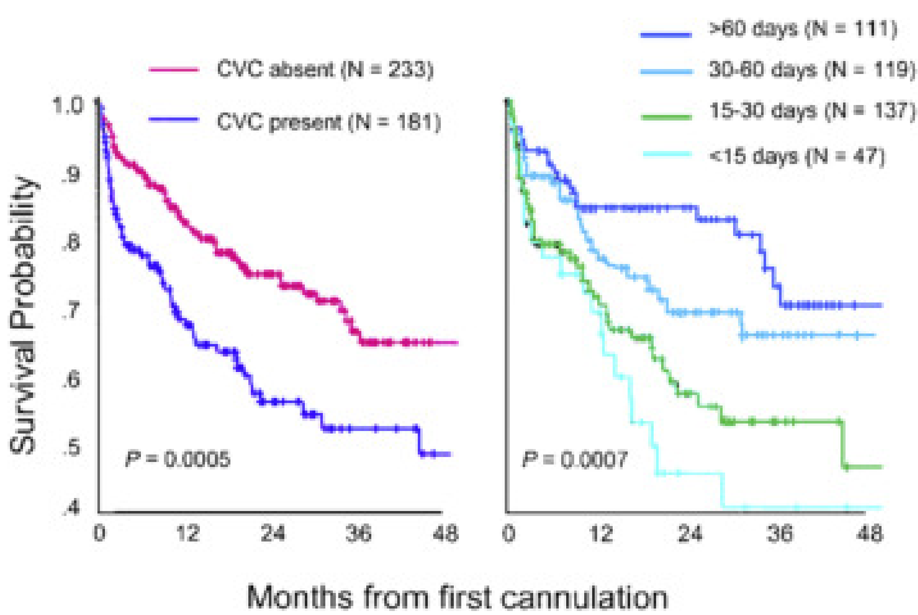


Figure 2. Kaplan-Meier curves of time to AVF failure (primary patency from first cannulation) by use of catheters (CVC) at the initiation of HD (left) and by the time to maturation in days (right). Reproduced with permission from Ravani et al.[45]

3.3. Selection of vascular access modality

3.3.1. Primary option for vascular access — autogenous arteriovenous fistula. The radiocephalic AVF (RCAVF) at the level of the wrist is the first choice for VA creation. When successfully matured, the RCAVF can function for years with a minimum of complications, revisions and hospital admissions. The RCAVF is preferentially created in the non-dominant arm, but the dominant extremity may be chosen if the vessels in the non-dominant arm are unsuitable. The indication to perform a

If direct arteriovenous anastomoses are impossible, vein transposition/translocation can be performed, with redirection of a suitable vein to an available artery (forearm radial/ulnar-basilic AVF) or GSV harvesting from the leg and subsequent implantation between an arm artery and vein (see Chapter 8).

A basilic vein transposition (BVT) in the upper arm is a good choice when RCAVFs or BCAVFs have failed or are not feasible. BBAVFs can be performed in either one or two stage operations.

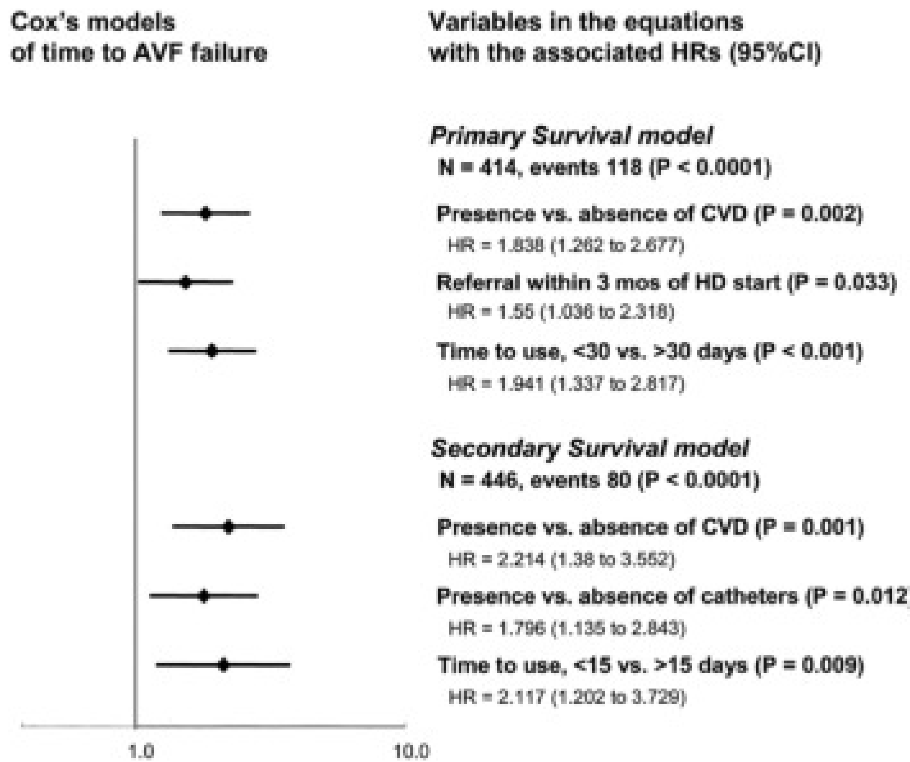


Figure 3. Risk factors associated with primary and secondary access failure. Hazard ratios plotted using a logarithmic scale. Reproduced with permission from Ravani et al.[45]

Table 5. Early failure and one year secondary patency rate of the radiocephalic AVF.

Reference	No. RCAVF	Early failure (%)	Secondary patency (%)
Silva et al.[59]	108	26	83
Golledge et al.[60]	107	18	69
Wolowczyk et al.[61]	208	20	65
Gibson et al.[62]	130	23	56
Allon et al.[63]	139	46	42
Dixon et al.[64]	205	30	53
Ravani et al.[65]	197	5	71
Rooijens et al.[66]	86	41	52
Biuckians et al.[67]	80	37	63
Huijbregts et al.[56]	649	30	70

3.3.1.1. Patient variables and outcome of vascular access. Various studies have shown the important influence of patient variables on choice and outcome of VA. Age and diabetes mellitus negatively influence fistula maturation and increase the risk of AVF failure.[80]

Table 6. Early failure (within one month of access creation) and one year secondary patency rate of brachiocephalic AVF (including brachiocephalic/perforating vein AVF).

Reference	No. BCAVF	Early failure (%)	Secondary patency (%)
Murphy et al.[68]	208	16	75
Zeebregts et al.[69]	100	11	79
Lok et al.[70]	186	9	78
Woo et al.[71]	71	12	66
Koksoy et al.[72]	50	8	87
Palmes et al.[73]	55	9	89
Ayez et al.[74]	87	8	83

Table 7. Early failure (within one month of access creation) and one year secondary patency rate of brachiobasilic AVF.

Reference	No. BBAVF	Early failure (%)	Secondary patency (%)
Murphy et al.[68]	74	3	75
Segal et al.[75]	99	23	64
Wolford et al.[76]	100	20	47
Arroyo et al.[77]	65	8	88
Keuter et al.[78]	52	2	89
Koksoy et al.[72]	50	8	88
Field et al.[79]	140	19	69
Ayez et al.[74]	86	6	73

A systematic review of the literature showed a tendency towards an increased risk of deep vein thrombosis and a decreased risk of catheter occlusion with a PICC.[81] An anatomical region at high risk of thrombosis is the antecubital fossa. Elbow veins represent a valuable source for the creation of a VA for HD, especially in obese patients, elderly patients, diabetics and patients affected by peripheral artery disease.[82] Such veins should be preserved (see Recommendation 14, Chapter 5).[48]

Women usually have smaller vessels than men, which may result in poorer maturation and lower long-term patency. Some studies show that females need more VA revisions and the creation of more AVGs,[62,83–88] while others, including a meta-analysis, could not demonstrate any significant differences in vessel diameters and the probability of maturation between men and women.[55,89]

Diabetes mellitus and arteriosclerosis are the most important causes of renal failure and HD treatment and can have a negative influence on successful use of the VA.[85]

Other variables that influence fistula use are: lower extremity atherosclerotic disease (LEAD), race and obesity.[90]

Patients using calcium channel blockers, aspirin and ACE inhibitors, enjoy better AVF and AVG patency.[91]

3.3.2. Secondary options for vascular access. When there are no options for creating an autogenous AVF, an AVG VA with the implantation of synthetic (expanded polytetrafluoroethylene [ePTFE]; polyurethane; nanograft = electrospun ePTFE graft) or biological material (ovine graft/Omniflow®) can be created. ePTFE is frequently used as an AVG with reasonable short-term patency but long-term patency is hampered by thrombotic occlusions, due to stenoses caused by progressive neointimal proliferation. One and two year primary patency varies between 40–50% and 20–30%, respectively. The secondary patency varies from 70 to 90% (at one year) and 50 to 70% at two years. Multiple interventions to prevent and treat thrombosis are required to achieve these outcomes.[92–96]

Elderly patients may benefit from the use of AVGs, because of the high primary autogenous AVF failure rate in these patients.[97] An important consideration for AVG use (in particular "early stick grafts") might be the avoidance of CVCs with their inherent high risk of infection, in particular when (sub)acute HD treatment is necessary and AVF creation/maturation is problematic.

3.3.3. Lower extremity vascular access. The indications for lower extremity VA are bilateral central venous occlusive disease (CVOD) or inability to create access in the upper extremity. Primary options are autogenous GSV[98] and FV transpositions,[99] and prosthetic graft implantation. Thigh VAs have acceptable patency rates but the handicap of an increased risk of ischaemia and infection.[100]

In a meta-analysis the results of femoral vein transpositions and AVGs are described. The one year primary and secondary patency was 83% and 48% and 93% and 69%, for FV transpositions and AVGs respectively. VA loss due to infection was primarily seen in AVGs (18% vs. 1.6%; $p < .05$). Ischaemia occurs more with lower extremity AVFs than AVGs (21% vs. 7.1%, $p < .05$).[101] In another study the outcome of 70 FV accesses was published with good results but with an 18% incidence of critical ischaemia, for which revision surgery was indicated.[102]

3.3.4. Indications for a permanent catheter for vascular access. Temporary CVCs are frequently used for acute HD or as bridging VA during fistula maturation and complications. Permanent tcCVCs may be indicated in patients with severe VA induced ischaemia, cardiac failure or limited life expectancy. Patients with PD peritonitis or waiting for a planned living related renal transplant can also be dialysed through a CVC for a limited period.

The primary location for a CVC is the right internal jugular vein followed by the left jugular, femoral and subclavian veins as alternative insertion locations. Femoral and subclavian vein CVCs should only be used for short periods, because of the risk of infection and CVOD.

HD via a CVC has increased in the USA, Canada and Europe, with a significantly greater morbidity and mortality risk due to infectious complications in comparison with the use of AVFs and AVGs (Fig. 4).[103,104]

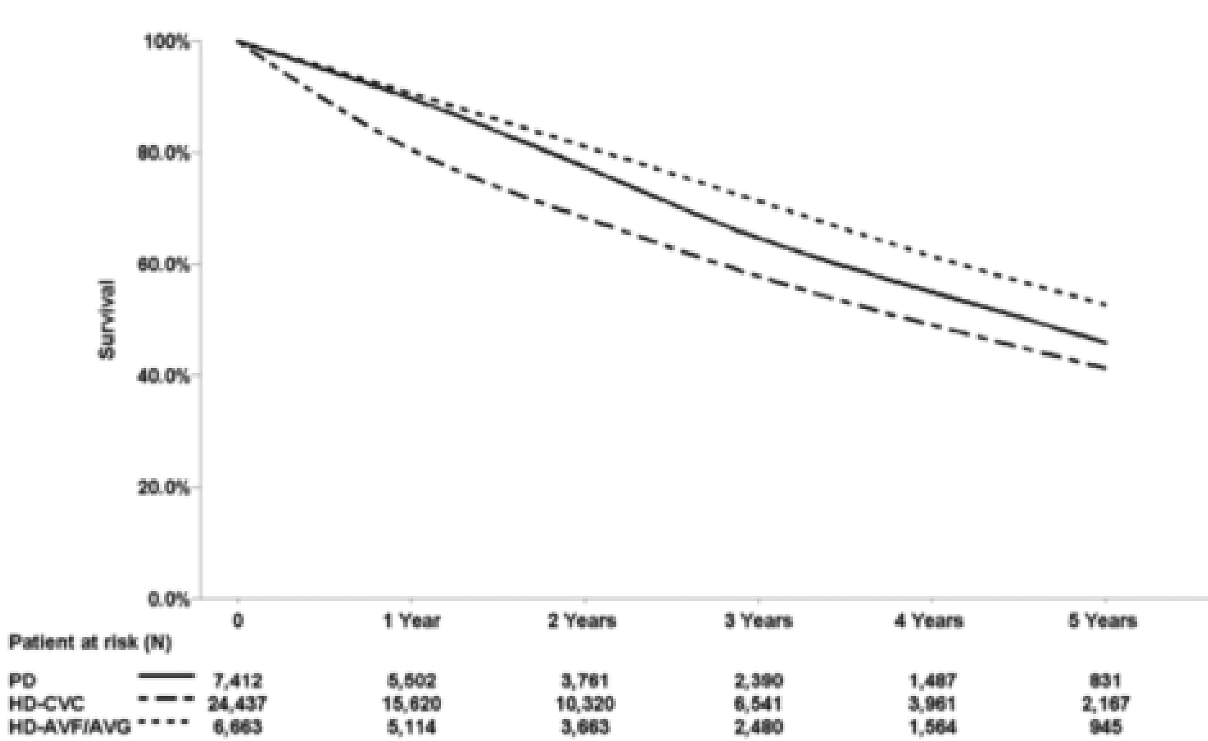


Figure 4. Survival (%) of patients with peritoneal dialysis (PD) versus central venous catheters (HD-CVC) and arteriovenous fistulas/grafts (HD-AVF/AVG), adjusted on the basis of a stratified Cox proportional Hazards model stratified by HD-CVC, PD, and HD-AVF/AVG and adjusted for age, race, gender, era of dialysis initiation, end stage renal disease comorbidity index, primary renal diagnosis, serum albumin, eGFR, province of treatment, and late referral. Reproduced with permission from Perl et al.[104]

The reason for the increased CVC use is the inability to create functioning AVFs because of poor vessel quality in the elderly, comorbid population.

Recommendation 1	Class	Level	Refs.
Referral of chronic kidney disease patients to the nephrologist and/or surgeon for preparing vascular access is recommended when they reach stage 4 of chronic kidney disease (glomerular filtration rate < 30 ml/min/1.73 m^2), especially in cases of rapidly progressing nephropathy.	I	C	48,105
Recommendation 2			
A permanent vascular access should be created 3—6 months before the expected start of haemodialysis treatment.	I	B	45,47,48,50,105
Recommendation 3			
An autogenous arteriovenous fistula is recommended as the primary option for vascular access.	I	A	40,43
Recommendation 4			
The radiocephalic arteriovenous fistula is recommended as the preferred vascular access.	I	B	40,58
Recommendation 5			
When vessel suitability is adequate, the non-dominant extremity should be considered as the preferred location for vascular access.	IIa	C	
Recommendation 6			
A lower extremity vascular access should be considered only when upper extremity access is impossible.	IIa	C	99,101,102
Recommendation 7			
Tunnelled cuffed central venous catheters as a long standing haemodialysis modality should be considered when the creation of arteriovenous fistulas or grafts is impossible or in patients with limited life expectancy.	IIa	B	103,104

4. PRE-OPERATIVE IMAGING

4.1. Pre-operative assessment

Besides a detailed pre-operative history and physical examination, non-invasive ultrasound imaging plays an important role in VA selection. Pre-operative duplex ultrasound (DUS) enhances the success of creation and the outcome of autogenous AVFs.[106] In a randomised trial, a primary failure rate of 25% without pre-operative DUS was observed in comparison with a failure rate of 6% with DUS.[107] Ultrasound venous mapping allows a precise evaluation of the depth of vascular structures[108] and detects VA sites that may be missed by clinical examination alone. Similar results were shown in a meta-analysis.[109]

DUS assessment can measure arterial diameters and flow as well as reveal stenotic segments especially where physical tests (poor radial pulse, unsuitable forearm veins) suggest impaired arterial inflow.[110]

In addition, DUS identifies patients with inadequate vessels in specific VA locations. In a study of 211 consecutive patients DUS found that 50% of them had inadequate arterial inflow for distal RCAVF creation.[111]

DUS provides helpful information before AVF construction such as internal vessel diameters and internal venous lesions.[112] Currently, a minimum pre-operative internal diameter of 2.0 mm for both arteries and veins is recommended before RCAVF creation and a minimum of 4.0 mm for the outflow vein in the elbow for AVG implantation.[48] Furthermore DUS provides important information for the planning of potential future AVF superficialisation.

Digital subtraction angiography (DSA) is helpful in only a small group of selected patients with significant peripheral vascular disease and suspected proximal arterial stenosis. The pre-operative endovascular approach allows identification and treatment in one procedure. However, the risk of potential contrast induced nephropathy must be carefully considered if iodinated contrast is used.[113]

CE-MRA enables accurate pre-operative detection of upper extremity arterial and venous stenosis and occlusions.[114,115] However, contrast enhanced magnetic resonance angiography (CE-MRA) is not recommended, since use of gadolinium is associated with the potential risk of a nephrogenic systemic fibrosis, especially in patients with severely impaired renal function.[116,117] Promising preliminary results for the pre-operative visualisation of arterial and venous vascular structures with non-contrast enhanced MRA (NCE-MRA) are available.[118]

In patients with a history of previous CVCs additional pre-operative imaging of the central veins should be performed, e.g. venography or intravascular ultrasound.[48,119]

Recommendation 8	Class	Level	Refs
Pre-operative ultrasonography of bilateral upper extremity arteries and veins is recommended in all patients when planning the creation of a vascular access.	I	A	106,107,109

4.2. Imaging methods for vascular access surveillance

4.2.1. Duplex ultrasound. DUS as a non-invasive tool is the first line imaging method in patients with suspected VA dysfunction.[48,120,121] However, the diagnostic quality of DUS depends strongly on the experience of the examiner[122–124] and provides no angiographic map for the guidance of further therapy.[125] DUS locates and quantifies stenoses, allows flow measurements and detects thrombotic occlusions[126–130] but evaluation of the central veins may be limited.[121]

DUS is a cost-effective technique for the evaluation of VA maturation, surveillance and complications.[131–133] If CVOD cannot be reliably excluded by DUS, additional imaging methods (e.g. DSA) will be necessary. Surveillance by DUS is reported to prolong AVG patency.[134] Only a few studies are available on DUS as a tool for ultrasound guided percutaneous transluminal angioplasty (PTA) of failing or non-maturing VA, which may be particularly indicated in patients with iodine contrast allergy or with residual kidney function.[135,136]

Although VA infection is primarily diagnosed clinically[137] DUS can supplement information on the extent of infected perivascular tissue and associated thrombosis.

4.2.2. Computed tomography angiography. Multislice computed tomography requires the use of iodinated contrast and radiation and should therefore only be used if no equivalent technique is available. However, compared with DSA computed tomography angiography (CTA) is a less invasive technique that provides important information for further treatment (surgery or PTA) and is less expensive than purely diagnostic DSA.[138] CTA is a reproducible and reliable technique for the detection of $\geq$50% stenosis or occlusion in dysfunctional AVFs[139] and demonstrates excellent correlation in stenosis detection compared with DSA.[140] CTA allows the evaluation of the vascular tree in failing VA before treatment,[140] especially if supplemented by 3D image reconstructions.[141]

For forearm AVFs, CTA provides a good VA visualisation with moderate sensitivity and high specificity for the detection of flow limiting stenoses.[142] For the detection of CVOD the sensitivity of CTA is dependent on the applied examination protocols. In suspected CVOD, CTA should be considered only when DUS or DSA are inconclusive, e.g. for the evaluation of the central veins and visualisation of the vascular tree.[143]

Recommendation 9	Class	Level	Refs
Duplex ultrasound is recommended as the first line imaging modality in suspected vascular access dysfunction.	I	B	120,123

Recommendation 10	Class	Level	Refs.
Computed tomographic angiography may be considered in patients with inconclusive ultrasonographic or angiographic results concerning the degree of central venous stenosis.	IIb	C	140–143

4.2.3. Magnetic resonance angiography (MRA). Gadolinium may cause nephrogenic systemic fibrosis (NSF) in patients with advanced impairment of renal function under HD. Therefore CE-MRA should be used only after carefully weighing the risks and benefits of alternative imaging studies.[116]

Even in the era before NSF had been recognised, CE-MRA had not replaced DUS or DSA for pre-operative evaluation, but was believed to be appropriate in selected cases.[114,144] It allows non-invasive examination of the arterial and venous system.[145,146] Due to the rare use of MR guided VA interventions, CE-MRA is currently used as a purely non-invasive diagnostic tool and potential treatment must be performed by additional percutaneous intervention or surgery.[147]

In comparison with DSA used to evaluate complex AVFs, fewer complications and side effects were observed by the use of CE-MRA.[148]

In another CE-MRA study, a sensitivity of 100% and a specificity of 94% were observed for the arterial and venous systems.[149] In addition, CE-MRA showed high sensitivity, specificity and positive and negative predictive values in the detection of stenosed vessel segments of dysfunctional AVFs and AVGs.[145] NCE-MRA is an evolving technology that has been proposed to avoid the risk of NSF. Pre-operative mapping and post-operative evaluation of the VA have shown promising results in the prediction of failure.[118,150] To date there are no data for the NCE-MRA evaluation of VA dysfunction.

Recommendation 11	Class	Level	Ref.
Contrast enhanced magnetic resonance angiography is not recommended in patients with end stage renal disease, because of the potential risk of gadolinium associated nephrogenic systemic fibrosis.	III	C	117

4.2.4. Digital subtraction angiography. In patients with VA dysfunction pure diagnostic DSA without subsequent intervention is not advised.[121] In selected cases, DSA may be used in pre-operative vein mapping, e.g. when central stenosis or occlusion is suspected or for the surveillance of CVOD, since venography is superior to DUS in the detection of CVOD.[126] In addition, DSA offers the opportunity to identify and treat central lesions during the same procedure.[151] During endovascular treatment and after surgery, DSA is performed to detect inflow, intra-access and outflow stenoses as well as residual stenoses or remaining clots[152] and to reveal CVOD.[151]

Recommendation 12	Class	Level	Ref.
In vascular access dysfunction digital subtraction angiography should be performed only when subsequent intervention is anticipated.	I	C	

Iodinated contrast agents can cause further deterioration of residual renal function. Nevertheless, DSA with diluted iodinated contrast can be performed relatively safely even in patients with end stage kidney disease.[113] However, CO_2 angiography is an effective alternative, without the risk of further impairment of renal function. CO_2 angiography has a sensitivity of 97% and a specificity of 85% in the evaluation of upper limb and central vein stenosis in comparison with conventional venography.[153] Due to the acceptable results of CO_2 angiography and the potential risk of NSF, gadolinium enhanced DSA[154] is no longer indicated. Figure 5 shows a proposed decision making algorithm for imaging.

5. CREATION OF VASCULAR ACCESS

5.1. Technical aspects

5.1.1. Venous preservation. It is essential to preserve the forearm veins in patients who are at risk of CKD as they may require HD in the future.[44,155] Patients and their carers should be instructed to avoid intravenous cannulae and, where possible, venepuncture in the cephalic, basilic or antecubital veins of either arm. If an intravenous cannula is unavoidable, it should preferably be inserted into a vein on the dorsum of the hand to avoid thrombophlebitis of the forearm and upper arm veins**.** The number of available veins for further VA is also maximised by a policy of performing an AVF at the most distal site available.

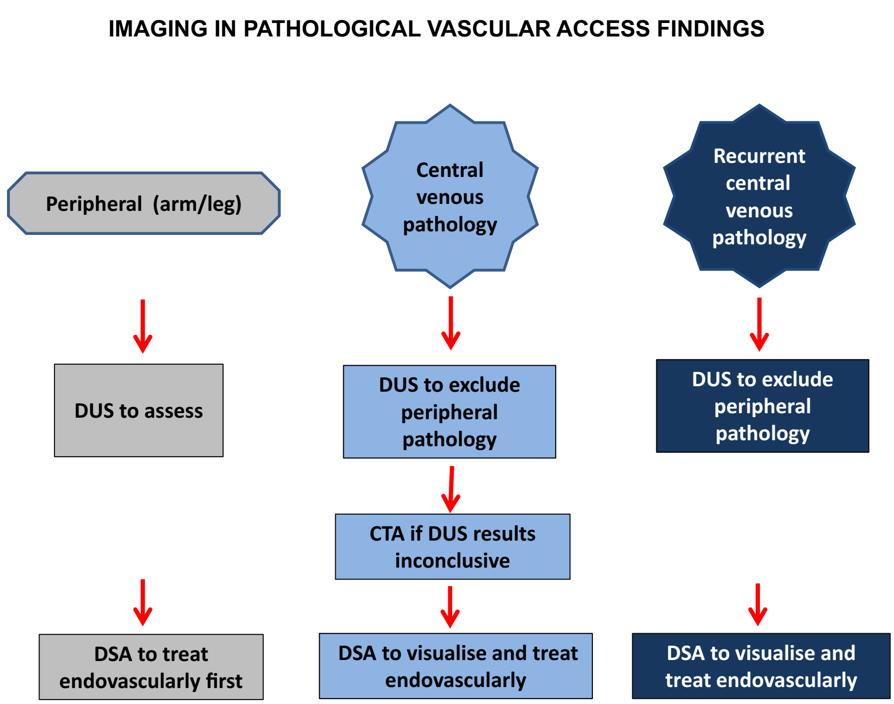


Figure 5. Imaging algorithm in pathological vascular access findings.

Recommendation 13	Class	Level	Ref.
In patients undergoing or likely to require haemodialysis, intravenous cannulae and venipuncture of the cephalic, basilic and the antecubital veins may be harmful and should not be performed.	III	C	39

5.1.2. Arm exercises. Arm exercises have been shown to improve arterial and venous diameters and resting blood flow in the upper limb in comparison with the opposite rested arm in patients with renal failure.[156] Whilst this is likely to be beneficial, it is not yet known whether pre-operative arm exercise improves AVF patency or maturation (although post-operative exercise and a tourniquet has been shown to increase maturation[157] as discussed in Chapter 6).

5.1.3. Pre-operative or peri-operative hydration. VA thrombosis is known to occur during or after hypotension. Rehydration with plasma expanders during VA creation improved primary AVF patency in a randomised study of patients with borderline vessels.[158]

Recommendation 14	Class	Level	Ref.
Adequate pre-operative hydration should be considered for vascular access creation.	IIa	B	158

Recommendation 15	Class	Level	Refs.
Broad spectrum antibiotics should be given prior to insertion of an arteriovenous graft including prophylaxis for *Staphylococcus aureus*.	I	A	159—161
Recommendation 16			
In carriers or in units with a high incidence of methicillin resistant *Staphylococcus aureus* the administration of a parenteral glycopeptide is recommended.	I	B	159—161

5.1.4. Prophylactic antibiotics. There is little evidence concerning the use of prophylactic antibiotics and the creation of VA. However, several randomised trials have shown that pre-operative broad spectrum antibiotic administration reduces the incidence of wound or graft infection by approximately 70% in other vascular surgical procedures.[159] In a small randomised trial cefamandole significantly reduced infection after AVG insertion.[160] Another randomised trial showed that a single 750 mg pre-operative dose of intravenous vancomycin significantly reduced the rate of infection in AVGs from 6% to 1%.[161]

Whilst the incidence of wound infection is greater in the lower limb than the arm, a broad spectrum antibiotic with activity against staphylococci, such as a cephalosporin, amoxycillin/clavulanic acid or a glycopeptide, is recommended pre-operatively for all VA operations to cover any other focus of infection in the patient, especially in diabetics or if a prosthetic graft is to be used. When the local prevalence of methicillin resistant *Staphylococcus aureus* (MRSA) is significant or the patient is a known MRSA carrier, a parenteral glycopeptide such as vancomycin or teicoplanin should be considered. In known carriers of other multi-resistant organisms such as extended spectrum beta lactamase producing organisms an appropriate antibiotic, such as a carbapenem, should be considered according to the bacterial sensitivities.

5.1.5. Pre-operative antiplatelet agents. Evidence concerning the use of antiplatelet agents is incomplete. As discussed more fully in Section 6.2.2., three meta-analyses have favoured antiplatelet agents to reduce VA thrombosis, but the few existing trials have differed in both the drugs and the mode of administration and whether they were given to patients with AVFs or grafts. Moreover, in most trials the antiplatelet agents were only administered post-operatively.[162—164] Amongst the 19 trials cited in the most recent meta-analysis[164] there were only three trials in which antiplatelets were consistently administered before surgery: In one trial aspirin caused a significant reduction in peri-operative fistula thrombosis[165] and in a second trial, clopidogrel was associated with a significant reduction in primary failure of AVFs although maturation was unaffected.[166] However, in a third trial a 35% reduction in primary fistula failure with ticlopidine administration failed to reach significance.[167] Despite the heterogeneity of these trials, it would seem advisable to give aspirin or another antiplatelet agent pre-operatively and to continue it post-operatively in an attempt to reduce VA thrombosis.

5.1.6. Pre-operative physical examination. Prior to surgery the upper limb pulses and superficial veins should be examined clinically by an experienced clinician with and without a venous tourniquet in a warm room in order to ensure maximum vasodilatation. The patient should also be examined for signs of venous hypertension in the limb such as prominent and tortuous collateral veins around the shoulder and upper limb oedema. The site of any CVC or pacemaker should be noted.[168] The chosen site for the fistula should be marked with a permanent marking pen.

Recommendation 17	Class	Level	Ref.
Patients should be examined prior to surgery with a tourniquet in a warm room and the proposed site of an arteriovenous fistula should be marked pre-operatively.	I	C	168

5.1.7. Anaesthesia. The majority of AVFs and many AVGs in the forearm or in the antecubital fossa can easily be performed under local anaesthesia using lidocaine or bupivacaine. Regional anaesthesia such as axillary or brachial block takes more time and usually requires the services of an experienced anaesthetist but has the advantage of causing significant vasodilatation,[1] which some surgeons find helpful and increases the proportion of distal AVFs in their hands.[4] In one randomised trial, stellate ganglion block significantly increased fistula flow, increased early patency and reduced maturation time.[175] In addition, there is evidence from one randomised trial suggesting that regional anaesthesia results in better AVF patency at 3 months than local anaesthesia.[176] More extensive VA procedures such as basilic vein transposition, brachio-axillary grafts or lower limb VA usually require either regional blockade or general anaesthesia.

Recommendation 18	Class	Level	Refs.
Regional anaesthesia should be considered in preference to local anaesthesia for vascular access surgery because of a possible improvement in access patency rate.	IIa	B	169—174, 176

5.1.8. Peri-operative anticoagulation. Peri-operative anticoagulation with systemic heparin is widely used in vascular surgery to prevent intravascular thrombosis during vessel clamping. In two randomised trials, systemic heparinisation (5000 IU intravenously) did not affect subsequent AVF patency but increased the incidence of post-operative haemorrhage.[177,178] In contrast, a third randomised trial found systemic heparin improved early patency without increasing complications.[179] Following a recent meta-analysis of these three trials it was concluded that systemic heparin had no effect on patency but significantly increased post-operative haemorrhage and therefore should be avoided.[180] Nevertheless, units employing tourniquets report no increase in bleeding with systemic heparinisation.[181] Local instillation of heparinised saline or Ringer's solution into the vessels or AVG after clamping is common practice in most units.

5.1.9. Arteriovenous fistula configuration. For AVFs an end to side (vein to artery) anastomosis is preferred over a side to side configuration as it allows easier approximation of the vein and artery and avoids the risk of distal venous hypertension without affecting patency.[182] For RCAVFs an end to end anastomosis has been advised by some to prevent steal syndrome[183] but the incidence of steal in distal AVFs is very low and in the rare occasions where it does occur it can easily be treated by ligation of the distal radial artery under local anaesthetic provided that the ulnar artery is patent.[184,185] Moreover, the radial artery usually remains patent after thrombosis of the VA and provides the blood supply to the hand.

5.1.10. Surgical techniques. It is generally agreed that an AVF should be performed at the most distal site possible, provided the vessels are adequate, in order to preserve as many vessels as possible (see Chapter 3).[44,186] Whilst proximal AVFs have been shown to have a lower initial failure rate and better patency than distal AVFs, as would be expected from larger vessels,[187,188] they have a greater risk of VA induced limb ischemia (VAILI),[189] may be more difficult to cannulate and are less comfortable for the patient.

Whereas excellent results have been obtained for AVF creation using smaller vessels in both adults and children using microsurgery and a tourniquet,[181,190] Duplex studies have suggested that AVF patency is poor if the internal arterial and venous diameters are less than 2 mm when standard vascular surgical techniques are used.[180,191,192]

Whilst the non-dominant arm is usually preferred, if a pacemaker or CVC is present the contralateral side is preferred because of the risk of venous hypertension and possible reduced fistula patency.[193] However, when contralateral VA is impossible, central venous imaging is advised to confirm free venous flow prior to surgery. Lower limb VA is the last option as it has a greater infection risk,[101] is less convenient and less comfortable for the patient.

Recommendation 19	Class	Level	Refs.
In adults when the inner radial arterial diameter is less than 2.0 mm and/or the cephalic venous diameter is less than 2.0 mm by ultrasound measurement an alternative site for access should be considered.	IIa	B	177,191,192,194
Recommendation 20			
If there is an indwelling central venous catheter or pacemaker the vascular access should be created in the opposite arm because of the risk of central venous stenosis and reduced access patency.	I	C	193

The first choice for a VA is either a snuffbox or RCAVF at the wrist, which have similar patency in selected patients.[61,195] A RCAVF may be created at any level in the forearm if the wrist vessels are inadequate or thrombosed but, if this is not possible, a BCAVF would usually be the next choice. In a meta-analysis of fistula patency RCAVFs had poorer patency in the elderly suggesting that a BCAVF might be preferred in such patients[97] but subsequent large series have failed to show any patency difference[196,197] and excellent results have been reported for RCAVFs in the elderly in several units.[61,198] Thus, which VA should be performed in the elderly will be determined by patient characteristics and physician or surgeon preference.

Several configurations of BCAVF are possible[44] using the cephalic vein, the confluence of the cephalic and basilic veins or the deep perforating vein but there is no evidence that one configuration has better patency. The "extension" procedure, which replaces the anastomosis to the brachial artery with one to the radial artery 2 cm from its origin, is technically more demanding but may carry a lower risk of steal.[199] There is a 12% incidence of a high brachial bifurcation so that the ulnar and radial arteries are both present in the cubital fossa.[200] The larger of the two arteries should be used for the anastomosis but, nevertheless, the overall patency may be less than that of standard BCAVFs.[201]

BBAVFs, respectively. The number of re-interventions was significantly higher in patients with AVGs (1.32 versus 0.54 per patient/year).[205] Whether a BBAVF should be performed in one stage or two stages is not settled despite improved patency of the two stage procedure in one study[209] and fewer complications in another.[210] A meta-analysis of one randomised and 7 observational studies failed to show any difference in patency rates between one and two stage procedures.[211] However, with small basilic vein diameters two stage operations might improve maturation. Nevertheless, any advantage of the two stages must be balanced against the 6 week delay between operations as well as the extra cost and inconvenience for the patient. In a non-randomised study, BVT using endoscopic basilic vein harvest has been described and reported to reduce hospital stay without compromising patency.[212] When the basilic vein is inadequate, the brachial veins or venae commitantes can be used but the patency was poorer in some studies.[213–216] Satisfactory results with transposed saphenous or FV in the arm have been described in small series but there are no studies directly comparing them with AVGs.[217,218]

When autogenous options in the arms have been exhausted, AVGs in various configurations such as forearm loops and brachio-axillary grafts increase the possibilities in the upper limb.

Recommendation 21	Class	Level	Refs.
When the upper arm cephalic vein is unavailable, a basilic vein transposition arteriovenous fistula should be considered in preference to an arteriovenous graft because of its improved patency and the reduced risk of infection.	IIa	A	78,204

An ulnar-basilic AVF is also an option although the patency is poorer than for RCAVFs.[202] Various transposition AVFs are also possible in the forearm (eg. ulnar-cephalic or radio-basilic).[203]

When the veins of both forearms are exhausted, a BVT is usually preferred to a forearm loop graft or a brachio-axillary graft because of its better patency[78,204,205] and lower infection rate.[206–208] A meta-analysis of 1509 patients clearly showed the preference for creating BBAVFs instead of AVGs. Pooled secondary patencies were 67% vs. 88% for AVGs and

Lower limb VA is reserved for patients with no remaining options in the arms as it is less comfortable for the patient and has a greater risk of VAILI and infection[101,219] (see Chapter 3) FV transpositions (FVT) are preferred over AVGs in the thigh because of better primary patency and lower infection rates (see Chapter 3).[101,102,220] However, ischaemia was much more frequent for FVTs[101] but it was eliminated in a small series by avoiding them in patients with reduced ankle brachial index (ABI < 0.85) and by tapering the vein at the anastomosis to reduce its diameter to 4.5—5 mm.[99] There is little evidence on the use of GSV thigh loops and, whilst these have been generally regarded as having poor patency,[101] a recent series of 56 saphenous vein transpositions in the thigh reported an acceptable primary patency of 44% at 59 months.[98] When prosthetic VA is necessary in the thigh, there appears to be no significant difference in infection rates or patency between mid and upper thigh AV loops.[101]

Recommendation 22	Class	Level	Refs.
When lower limb vascular access is necessary a femoral vein transposition should be considered in preference to an arteriovenous graft.	IIa	B	101,220

5.1.11. Choice of graft. Both synthetic and biological grafts are available and have been used for VA. In general, synthetic grafts have been preferred because of lower cost and concerns about long-term degeneration in biological grafts, although the latter have a greater resistance to infection and may be preferred in contaminated fields.[221]

ePTFE grafts are the most widely used. There is some evidence from randomised studies that primary patency is better for grafts with an expansion at the venous end[222,223] but heparin bonded grafts failed to show a significant patency advantage up to 1 year in a randomised trial despite a reduced early thrombosis rate[224] and a significantly improved 1 year primary patency in another non-randomised study.[225] One randomised study has also shown reduced thrombosis with a vein cuff at the venous end of a ePTFE graft although the improvement in primary patency failed to reach statistical significance.[226] There is no evidence that patency is affected by carbon coating, or by external or internal support although the latter may prevent kinking. Most surgeons use 6 mm grafts although there is no evidence to support this over other diameters. Stepped or tapered grafts have no proven advantage despite expectations that they might reduce VAILI whilst preserving patency. Most prosthetic grafts can be used after 1—2 weeks although newer multilayer ePTFE grafts are self-sealing and can be safely needled within 1—2 days,[227,228] which can avoid the use of CVCs in some patients.[229] A polyurethane graft may also be used within 1—2 days of insertion and has been reported to have similar patency to BVT and ePTFE[230,231] but had an increased risk of infection compared with ePTFE in one non-randomised study.[219] A removable plastic sheath prevents stretching during tunnelling, thereby reducing perigraft seroma caused by "sweating" and improving patency in one non-randomised study.[232] A biosynthetic graft consisting of a collagen-polyester composite gave acceptable results in one small observational study[233] but had significantly poorer primary patency than BBAVFs in another small randomised study.[204]

Because there are no comprehensive randomised studies comparing several grafts, no definite recommendations can be made concerning which graft should be used routinely but a self-sealing graft would be advisable for patients who have difficult central venous access and who require early HD.

Combining a standard ePTFE graft at the arterial end with a CVC inserted percutaneously (Haemodialysis Reliable Outflow device = "HeRO®" graft) may be a useful alternative to a central venous line in patients with inadequate upper limb veins[234,235] although whether it is preferable to a lower limb VA is uncertain (see Chapter 8).

Biological grafts such as bovine carotid artery or bovine mesenteric vein, which have been rendered immunologically inert, have been used extensively in some units[221] and have compared well with prosthetic grafts in one small randomised study[236] and a further non-randomised study,[237] but their relatively high costs and fears of long-term aneurysm formation and rupture have limited their use. Tissue engineered grafts have been used in a small number of patients but it is too early to determine whether these have any advantages over other grafts.[238]

Recommendation 23	Class	Level	Refs.
When an arteriovenous fistula cannot be created, a biological graft should be considered in preference to a synthetic graft in the presence of infection.	IIa	C	221,236,237
Recommendation 24			
The implantation of a self-sealing arteriovenous graft is recommended for patients who have difficult central venous access and who require early cannulation for haemodialysis.	I	C	229

5.1.12. Sutures or nitinol anastomotic clips. Most surgeons use non absorbable sutures such as polypropylene or ePTFE but there is some evidence from non-randomised studies that the use of non-penetrating nitinol vascular clips may improve the subsequent patency of AVFs[239,240] although this was not confirmed by one small randomised study.[241] Clips are not suitable for use in calcified vessels.

5.1.13. Other challenges: Patient and vessel characteristics. Vessel calcification may limit VA options, particularly in diabetic patients, but an AV anastomosis can be performed to arteries with mild "eggshell" calcification either using firm bulldog clamps or a tourniquet. Severe calcification makes performing the anastomosis difficult and the associated vessel rigidity may compromise maturation. Calcification and increased arterial wall thickness have been shown to significantly increase the primary failure rate of forearm AVFs[112] and calcification may also be a marker of poor prognosis.[242]

Obese patients present difficulties in visualising the veins so that pre-operative DUS scanning is invaluable. When the vein is located deeper than 0.6 cm from the skin surface it may be difficult to cannulate which is a possible cause of reduced patency[243] and either elevation or transposition either as a primary or secondary procedure may facilitate cannulation with patency rates similar to those of non-obese patients.[244–247] Liposuction over a guard has also been used successfully to elevate the vein draining an AVF to facilitate needling.[248,249] An implantable titanium venous window needle guide may be another alternative to aid cannulation in obese patients and has been reported to be useful and durable with low infection rates in a non-randomised study.[250]

5.2. Peri-operative assessment

Whatever form of AVF or AVG is created at the end of the operation there should be a palpable thrill or, at least an audible bruit overlying the anastomosis or over the vein close to the anastomosis. The absence of a bruit has been found to be a good predictor of early AVF thrombosis and whilst DUS measurements of end diastolic velocity were a slightly better predictor the difference in specificity and sensitivity was marginal.[251] If a thrill fails to appear after releasing the clamps on the vessels, application of a vasodilator such as papaverine may aid vasodilatation but if this is unsuccessful the anastomosis should be carefully checked for defects and an embolectomy catheter or a bougie passed. The presence of a strong pulse in the vein draining a fistula without a thrill or bruit usually indicates a downstream venous stenosis or occlusion. Intra-operative or 1 day post-operative blood flow measurements can also identify AVFs at high risk of failure[192,251–254] but are relatively imprecise and probably have little use in day to day practice. Before leaving the operating room, the hand should be assessed for ischaemia including capillary return and, in the case of proximal AVFs, the radial pulse recorded.

Recommendation 25	Class	Level	Ref.
If after creation of a vascular access, there is no thrill or a bruit in the region of the anastomosis, further investigations should be considered.	IIa	C	251

5.3. Peri-operative complications

AVGs or AVFs should be evaluated soon after their creation and then routinely examined during their lifespan either by means of physical examination to detect physical signs that suggest the presence of dysfunction (monitoring) or by periodic evaluation using tests involving special instrumentation (e.g. DUS surveillance). VA thrombosis, including early thrombosis within the first 30 days of creation, is the most frequent complication leading to failure of either autogenous or prosthetic VA procedures.

5.3.1. Haemorrhage. Haemodialysed patients have an increased bleeding tendency with abnormal bleeding times despite normal coagulation studies and platelet counts.[255] Scheduling VA procedures on the day between dialysis sessions decreases exposure to the heparin used to prevent clotting in the HD circuit.

Early post-operative haemorrhage may need rapid intervention to achieve haemostasis while preserving VA function. Direct digital compression is required followed by surgical revision if the bleeding persists. Clinically significant haematomas remaining after the bleeding has stopped may require evacuation to reduce the risk of infection or skin necrosis.

Recommendation 26	Class	Level	Ref.
In order to decrease the exposure of patients to the heparin used during dialysis, scheduling elective access procedures on a day between haemodialysis sessions should be considered.	IIa	C	255

5.3.2. Post-operative infection. VA site infection is an important cause of morbidity and mortality in patients on HD. The reported incidence of infections affecting the VA sites ranges from 0.5 to 5% per year for autogenous AVFs to 4–20% for prosthetic AVGs.[255] Peri-operative infections (within 30 days of creation) have a low incidence (0.8%) and account for only 6% of all VA site infections.[256] They result from contamination during the operation and present as abscesses and wound infections. Autogenous AVF infections are usually localised and in the absence of abscess, pseudoaneurysm or haemorrhage may respond to appropriate antibiotics.[255] Whilst there is no published evidence on the duration of antibiotic therapy, 6 weeks treatment has been recommended by analogy to the treatment of endocarditis.[257,258]

In contrast to late infections, early peri-operative synthetic graft infections involve the entire graft and total graft excision is required.[255,256,259,260] When necessary, brachial artery ligation should be performed and is in most cases well tolerated.[261]

Patients who exhibit systemic signs of infection, bleeding, pseudoaneurysm or involvement of the anastomosis should have their grafts completely removed or their AVF ligated.[261]

Recommendation 27	Class	Level	Ref.
In patients with early peri-operative (<30 days) autogenous arteriovenous fistula infection and absence of haemorrhage or pseudoaneurysm, appropriate antibiotic therapy is recommended.	I	C	255
Recommendation 28			
Early peri-operative (<30 days) arteriovenous graft infection with systemic sepsis, purulent discharge, perigraft abscess or haemorrhage should be treated by total graft removal.	I	C	
Recommendation 29			
For early autogenous arteriovenous fistula infection in the presence of systemic signs, bleeding and involvement of the anastomosis, fistula ligation should be performed.	I	C	255

5.3.3. Non-infected fluid collections. Seromas are occasional complications of prosthetic AVGs but are rare in AVFs. They may result from "sweating" through an ePTFE graft, which can be minimised by the avoidance of stretching.[232] The major concern regarding a seroma is whether it represents a low grade infection. Needle aspiration may be helpful diagnostically and may be curative. If a seroma persists, the VA must be abandoned in favour of a new graft. Other seromas may resorb spontaneously but surgical drainage with excision of the cavity wall or even graft replacement may be necessary.[255]

Lymphatic collections usually resolve spontaneously with or without the aid of repeated aspiration.[255] Persistent lymphorrhoea through a sinus carries a risk of infection. Negative pressure wound therapy (NPWT) dressing devices have been used for open wounds.[262] However, it is probably unwise to directly apply them over vascular anastomoses or the vein draining an AVF as this might result in major haemorrhage from anastomotic disruption or erosion of the vessel.

5.3.4. Early onset of vascular access induced limb ischaemia (See Chapter 7). A wide spectrum of ischaemic symptoms may complicate VA creation. Four stages with similarities to Fontaine's classification for lower limb ischaemia in peripheral arterial disease have been described (see Definitions).[263] Clinically significant limb threatening ischaemia with rest pain (stage 3) or tissue loss (stage 4) occurs in 4–9% of proximal (brachial artery) VA procedures.[255] Usually, the diagnosis of ischaemia can be made easily by the absence of a radial pulse, pallor or slow return of peripheral circulation after compression, or by digital pressures of <50 mm Hg and a digital brachial index (DBI) of <0.6.[263] These changes are reversed by compression of the fistula.

Although more than 80% of steal related limb threatening ischaemia is caused by discordant vascular resistance, 20% results from a proximal inflow stenosis. A DSA may be helpful before embarking on surgical correction in equivocal cases.[264] In half of patients with steal, limb threatening VA induced limb ischaemia develops within a month of VA creation, often appearing immediately after surgery.[265] Patients should be closely observed during the first 24 post-operative hours following proximal VA creation with close observation probably unnecessary beyond that. Monitoring for steal is not recommended beyond the first post-operative month in patients with AVGs, while lifelong monitoring should be performed in proximal AVFs as these may present a delayed onset of steal symptoms after maturation and late vein dilatation.[266]

Early onset VAILI should be treated by immediate surgical correction of the steal. Ligation is the simplest solution, which requires abandonment of the VA site but is advisable for severe symptoms of early onset and should be performed urgently to prevent tissue loss and permanent neurological damage.[255] Some authors have suggested the distal revascularisation and interval ligation procedure (DRIL) but this may not be as successful in early onset steal as for late onset steal.[267]

Ischaemic monomelic neuropathy (IMN) may also rarely occur in the absence of steal, probably as a result of transient ischaemia during surgery. It is characterised by pain with sensory and/or motor deficit of all three major nerves in the affected limb, out of proportion to any residual ischaemia. It can be confirmed by nerve conduction studies. It requires prompt ligation of the VA to prevent continued pain and may progress to a useless clawed hand. Treatment in the chronic phase is often unsatisfactory and relies on analgesics, antidepressants and anticonvulsants.[255]

results but should be delayed for at least 7 days after the VA creation to allow tissue incorporation to prevent puncture site bleeding.[268] In another series of 23 early graft thromboses, poor outcomes were reported following percutaneous de-clotting.[272] During surgical thrombectomy, intraoperative angiography and either PTA or surgical revision of any underlying stenosis should be performed.[268]

Recommendation 30	Class	Level	Refs.
For early limb threatening vascular access induced ischaemia and for all cases of early ischaemic monomelic neuropathy in the absence of steal, the access should be ligated urgently.	I	C	255,267

Recommendation 31	Class	Level	Refs.
For vascular access salvage after early thrombosis, thrombectomy and revision (if needed) should be performed as soon as possible.	I	C	269—271
Recommendation 32			
Thrombolysis should not be used for early vascular access thrombosis within 7 days of creation.	III	C	268

5.3.5. Early thrombosis. The most frequent complication in all VA types is early thrombosis which is defined as thrombosis occurring within 30 days of VA creation.[9] If the VA is to be preserved, treatment within 7 days is advisable. The longer the intervention is delayed the more likely the thrombus is to propagate and become fixed to the vessel wall, making thrombectomy more difficult and less durable because of damage to the endothelium. The thrombus can be removed either surgically using a Fogarty balloon catheter or by endovascular means using pharmacological or mechanical thrombolysis, or a combination of these. Thrombectomy alone is insufficient unless the responsible factor is transient, such as an episode of hypotension, and treatment of any underlying stenosis is required.

Early VA thrombosis is usually attributed to technical errors during surgery but in a series of 20 early AVG thromboses only one patient was found to have technical problems and most grafts thrombosed because of hypotension, hypercoagulable state or previously undetected lesions in the proximal draining vein or central veins.[268] A meta-analysis in 2002 showed that surgical thrombectomy of AVGs gave better results than endovascular thrombectomy up to one year.[269] However, another meta-analysis failed to show any difference between the two modalities for AVGs.[270] In the absence of any randomised trials, there is insufficient evidence regarding thrombectomy of AVFs to draw any definite conclusions although in a systematic review a possible advantage in favour of surgical thrombectomy for long-term patency was suggested.[271]

Endovascular treatment of early post-operative thrombosed grafts by thrombolysis and treatment of any underlying stenosis with PTA/stent has been shown to give good

5.4. Post-operative care

It is wise to keep the patient and the extremity bearing the newly formed AVF warm to promote vasodilatation although there is no evidence to support this. The application of transdermal glyceryl trinitrate to RCAVFs during the immediate post-operative period caused significant vasodilatation and increased blood flow in a small RCT[273] but a larger RCT failed to show any significant improvement in patency at 6 weeks.[274] Patients should be instructed to check the function of their new AVF by palpating the thrill or, in its absence, by auscultation of the bruit. They should be advised to report urgently to the VA nurse or medical team if the thrill or bruit disappears and must have easy access to urgent medical help in the event of bleeding or signs of infection.

5.5. Training of surgeons to perform vascular access

Increasing AVF creation rates over AVGs is an indisputable priority. Training of VA surgeons seems to be the key predictor of whether priority is given to the placement of AVFs rather than AVGs. Surgeons who had performed more AVFs and fewer AVGs during training subsequently created more AVFs and fewer AVGs during their specialist practice.[53] Greater emphasis on VA surgery during training was also associated with higher odds of a patient receiving an AVF versus AVG. Surgeons who had created at least 25 AVFs during training had significantly lower rates of AVF failure than those placed by surgeons who had created fewer than 25 with a relative risk of 0.66.[53,275]

There is conflicting evidence on whether the grade of the operating surgeon affects VA outcomes. Two retrospective studies have shown that well supervised trainees do as well as specialists [276,277] whilst another retrospective study concluded that trainees produced poorer outcomes.[278] The operating surgeon seems to be a significant determinant of AVF outcome,[52] but in a prospective non-randomised study unsupervised vascular trainees performed AVFs equally effectively as consultants[276,279] so that AVFs can provide good training opportunities without detriment to patient care.

Recommendation 33	Class	Level	Refs.
Establishing vascular access training programs is recommended in order to supervise adequate numbers (>25) of autogenous fistulas for each trainee.	I	C	52–54, 275–279

6. SURVEILLANCE OF VASCULAR ACCESS

6.1. Access maturation and care

6.1.1. Concept. When a fistula is created, a continuous flow from the artery to the vein initiates a cascade of changes, altering wall structure, shear stress, and rapidly increasing flow during first 24 hours, achieving most of the increase in flow and vein diameter within 8 weeks of VA creation.[132,186] AVFs are usually not readily usable after creation, but these changes lead the fistula to become suitable for cannulation over time, a process known as maturation.[257]

A fistula is considered mature when it is thought to be appropriate for cannulation with minimal complications, and to deliver the prescribed blood flow throughout the HD procedure. It is established by physical examination of the VA and/or imaging (DUS) by experienced staff before VA cannulation and predicts successful use and flow delivery during HD. It should happen preferably 4–6 weeks after AVF or 2–4 weeks after standard AVG creation.[132,257,280,281]

Cannulation should be considered only in mature VAs because of the risk of puncture complications, VA failure or insufficient HD quality. When a VA is cannulated successfully with two needles over a period of at least 6 HD sessions during a 30 day period, and delivering the prescribed blood flow throughout the HD procedure (at least 350 ml/min),[282] the VA is finally considered adequate for HD (functional and successfully used).

6.1.2. Maturation of arteriovenous fistula

6.1.2.1. Physical examination and other diagnostic methods. Maturation can be established by physical examination of both the venous conduit and its flow. It is usually assessed by the presence of an adequate venous diameter with or without a proximal tourniquet in place (to permit safe landmark recognition and cannulation), a soft easily compressible vein, a continuous audible bruit (an audible low pitched continuous systolic and diastolic bruit), a palpable thrill near the anastomosis extending along the vein for a varying distance, with an adequate length and superficial enough to be punctured with two needles.[186,283] Experienced staff have demonstrated an excellent ability to predict eventual poor fistula functionality.[132,257] Causes of poor functionality include any factors that may cause difficulty in cannulation and flow delivery (thrombosis, arterial or venous stenosis, small diameter or deeply located veins, presence of accessory veins).

Post-operative ultrasound examination between the first 6–8 weeks and 2–4 months[132] after fistula creation is helpful in confirming maturation. In general, a draining vein diameter less than 4 mm and fistula flow of less than 500 ml/min indicates a fistula that is unlikely to mature.[132,280,281] Some groups recommend the rule of 6's to define maturation (at least 6 mm vein diameter and 600 ml/min flow, and less than 6 mm vein depth),[257] which is probably quite conservative.

6.1.2.2. Time to maturation. A VA can be used for cannulation when it is considered mature. However, the optimal delay between creation and use of a VA, whether autogenous or prosthetic, is not unanimously agreed. Premature needling may predispose to VA failure (because of thrombosis or extrinsic compression by haematoma following damage to the thin wall of the freshly arterialised vein), and longer maturation time (>30 days) appears to be associated with lower risk of AVF failure.[284–286] However, early cannulation can reduce the need for a temporary catheter and its complications. Furthermore, significant differences between groups and countries have been observed: AVFs were first cannulated <1 month after creation in 74% of Japanese, 50% of European and only 2% of US facilities.[287] Early cannulations were not associated with increased risk of VA failure, probably also related to the smaller needles and lower flows used in Japanese facilities.

This waiting time is feasible only when there is no impending need for the commencement of HD, which is frequently not the case. Thus, clinicians may be able to select appropriate patients for early fistula cannulation depending on maturation criteria and the time since fistula creation, but also based on the need or the risk of complications of other HD methods.

If AVF maturation has not occurred by 6 weeks, causes of poor functionality should be considered and additional investigations should be performed in order to achieve prompt diagnosis and treatment.[132,280,281,288]

Secondary interventions in previously matured AVFs (i.e. realocation of the anastomosis at a proximal site, thrombectomies or endovascular procedures), or proximal AVFs in patients with previous distal matured AVFs, may need no maturation period if the veins are already mature.

Recommendation 34	Class	Level	Refs.
Arteriovenous fistulas should be considered for cannulation 4–6 weeks after creation, and standard arteriovenous grafts after 2–4 weeks.	IIa	B	284,286,289
Recommendation 35			
If an arteriovenous fistula fails to mature by 6 weeks, additional investigations (like duplex ultrasound) should be considered in order to achieve prompt diagnosis and treatment.	IIa	C	280,290
Recommendation 36			
Arteriovenous fistula cannulation before 2 weeks should generally not be done.	III	C	284
Recommendation 37			
Arteriovenous fistula cannulation between 2 and 4 weeks after creation may be considered in selected patients under close supervision.	IIb	B	284

6.1.3. Time to cannulation of the arteriovenous graft. Because of its stiffer wall, an AVG usually has a weaker thrill over the entire graft than an AVF.[257] In AVGs, maturation is based on the time needed for tissue to graft incorporation and for tissue swelling to decrease after graft implantation, rather than flow increase over time (because the flow is high from the day of surgery with minimal changes over time).[291] It is usually defined as 2–4 weeks (followed by 62% of USA and 61% of European facilities).[287] There was no significant difference in the risk of graft failure between those cannulated early and those cannulated later.[287,289] If maturation takes more time, causes of non-maturation that are unlikely to improve over time should be studied (e.g. excessively deep tunnelling or graft thrombosis).

Some grafts allow for early cannulation within 24–72 hours without major complications (either polyurethane grafts, or multilayer ePTFE grafts allowing self-sealing), avoiding catheters in patients that need early HD and that do not have suitable veins for a fistula. However, this type of graft confers no additional benefit other than early cannulation.[227,230,292]

6.1.4. Access care. After VA surgery, patients should receive information about wound healing, warning signs (infection, symptoms of VAILI, bleeding and other post-operative complications), avoiding fistula compression or injuries, and encouraging an exercise program.[157,293,294]

Patients should be instructed to check the function of their new AVF (self examination), by palpating the thrill. They should be advised to report urgently to the VA nurse or medical team if the thrill disappears and must have easy access to urgent medical help in the event of signs of infection or persistent bleeding in spite of manual compression.[186,257,294]

In patients undergoing HD, experienced staff should examine the fistula during each HD session (before fluid removal).[257] Patients in pre-dialysis therapy should be taught how to perform self examination, and at a minimum they must have physical examination by experienced staff 4–6 weeks post-operatively.[295]

6.1.5. Assessment and treatment of maturation failure. Non-maturation rates differ between groups, ranging from just under 10% in BCAVFs to up to 33%, or even more, in RCAVFs[26]; women, older patients, distal placements and accesses with smaller diameter artery and vein are risk factors for failure to mature.[281,296,297] Additional investigations such as DUS or DSA are indicated if physical examination by experienced staff determines maturation failure 6 weeks after AVF creation or poor prognostic signs (faint or absent thrill, complete access collapse proximally, discontinuous bruit, high pitch continuous systolic audible bruit, pulsatile AVF, small diameter or poorly defined vein, excessive depth, large accessory/collateral veins).[132,288,298]

Non-matured AVFs frequently have one or more potentially remediable problems, and up to 80% can be salvaged after surgical or endovascular correction,[299,300] although thereafter cumulative survival rates are decreased and require more secondary interventions to maintain patency.[301] The most common causes of non-maturation are venous, arterial or anastomotic stenosis, competing veins or large patent branches, and excessive depth from the skin.[63] Depending on the cause, open or endovascular repair can be performed, although in general no significant differences have been found between the two modalities.[300] (see Chapter 7: Clinical Outcomes).

Problem specific salvage procedures increase the proportion of AVFs that are mature and usable for HD,[298] and if a fistula fails to mature the patient should immediately be referred back to the surgeon or the interventionist for prompt evaluation and intervention.[257,302]

6.2. Measures to improve maturation

In addition to prolonged observation after VA creation, pre and intra-operative treatments, or additional post-operative surgical or endovascular procedures (i.e. side branch ligation, superficialisation, treatment of stenotic lesions and others), other post-operative treatments can improve fistula maturation and long-term patency.

6.2.1. Exercise. After AVF creation, vein diameters immediately increase following arm exercise.[293] Compared with non-exercise, hand-arm exercise programs cause significant outflow vein dilatation and increased VA flow. In two randomised clinical trials structured hand exercise programs significantly increased clinical maturation after AVF creation, mainly in distal AVFs.[157,294] Therefore patients should be encouraged to follow a hand-arm exercise program after AVF creation.

Recommendation 38	Class	Level	Refs.
Structured post-operative hand exercise training should be considered, to increase arteriovenous fistula maturation.	IIa	B	157,294

Recommendation 39	Class	Level	Refs.
Long-term anti-thrombotic therapy should not be used to prolong vascular access patency in haemodialysis patients.	III	C	91,303, 310,311

6.2.2. Antiplatelets and anticoagulation. Some systematic reviews and meta-analyses showed that after creation of a VA, antiplatelets can reduce AVF thrombosis (but not AVG thrombosis) by 44% (RR .56, 95%CI .40-.78). However, they do not increase suitability or maturation for HD (RR .62, 95% CI .33-1.16), and they have been unable to demonstrate an improvement in loss of primary unassisted patency, or the need for re-intervention to attain patency or assist maturation.[162−164] Another systematic review and meta-analysis, in spite of low evidence quality due to small and heterogeneous series with short follow-up, showed no beneficial effect for any antiplatelet treatment to increase the patency of AVF or AVG (except ticlopidine, which has been taken off the market in some countries).[303] In another randomised clinical trial aspirin treatment demonstrated no reduction in fistula thrombosis 12 months after AVF creation (RR 1.05).[304] In spite of the heterogeneous studies that support these conclusions, and the weak evidence in some of these topics, there is not enough evidence to firmly recommend antiplatelet treatment to reduce AVF thrombosis or improve maturation. A preventive role of antiplatelet therapy decreasing cardiovascular mortality in ESRD patients had been proposed.[305] Although antiplatelet treatment has been related to a decrease in myocardial infarction (RR 0.87), all cause mortality, cardiovascular mortality and stroke remain similar, and it was related to an increase in major and minor bleeding (RR 1.33 and 1.49).[163,164,306−308] Thus, the real benefit of antiplatelet treatment in improving cardiovascular mortality, specifically in ESRD patients who do not have clinically evident occlusive cardiovascular disease, is doubtful.

Dual therapy (aspirin plus clopidogrel) significantly increased the risk of bleeding, suggesting that this combination may be hazardous.[309]

An anticoagulation strategy using low molecular weight heparin (LMWH) and oral anticoagulants has not been extensively evaluated in HD patients. There is only one randomised study using low dose warfarin for the prevention of AVG failure which found no benefit,[310] while in DOPPS such treatment was associated with worse AVG patency rates.[91] Additionally, in a systematic review increased bleeding events were associated with warfarin use compared with placebo in patients with AVFs or AVGs.[162] Regarding LMWH thrombo prophylaxis, there is only one comparative study with historical controls in a paediatric population reporting a decrease in early fistula failure in the treatment group.[311]

6.2.3. Other treatment options. Calcium channel blockers and angiotensin converting enzyme inhibitors have been associated with improved primary graft and secondary fistula patency respectively in a single observational study, but more conclusive data are lacking.[91]

There are insufficient data available to adequately assess the efficacy of omega-3 fatty acids (fish oil) in improving VA function or maturation.[312,313] In a randomised controlled trial among patients with new VA grafts, daily fish oil ingestion did not decrease the proportion of occluding grafts within 12 months.[313] In a RCT with 567 enrolled patients fish oil did not reduce AVF thrombosis, abandonment or cannulation failure.[304]

Statins have pleiotropic beneficial actions besides lipid lowering but non-randomised studies and nationwide cohort analysis report contradictory results regarding their effects on VA patency rates.[314,315]

As previously described, most recommendations are based on clinical experience, but interventions that clearly improve VA maturation and suitability for HD are needed.[164]

6.3. Cannulation

The maintenance of the VA not only depends on the quality of the blood vessels and the surgical technique used, but also on the way in which the VA is cannulated. After creation of the initial VA, preferably an autogenous AVF, the correct needling technique has a favourable influence on fistula lifespan.[316] Nurses play a pivotal role in the care of VA: they see the patient during every HD session, perform cannulation and assess VA function.[48] VA cannulation is a basic but essential part of HD treatment and requires skill from the nurse, or patient if self-cannulating. A chronic HD patient needs at least 312 needle insertions per year (6 $\times$ 52). It is reasonable to assume that complications caused by cannulation, such as haematoma, infection and pseudoaneurysm formation can have great consequences in terms of suboptimal HD, the need for extra needle insertions, patient discomfort, interventions and even loss of the VA.

Frequent VA complications, particularly with AVGs, have led to the development of VA monitoring protocols[317] whose goals are to identify VA stenosis and enable intervention prior to thrombosis, thereby maximising VA longevity and minimising morbidity.[318–322]

6.3.1. Access care before cannulation

6.3.1.1. Skin preparation. Proper preparation of the access sites using strict aseptic technique can minimize contamination and/or access infection and should be used for all cannulation procedures.[323–325] VA related infections are a leading cause of morbidity and mortality in HD patients. AVGs and CVCs are associated with an increased risk of infection when compared with AVFs.[326] Studies have suggested that the buttonhole cannulation technique is associated with an increased risk of VA related infections.[327–330]

It has been shown that HD patients are more frequently nasal and skin carriers of *Staphylococcus aureus* than the general population.[324] For this reason, meticulous skin preparation prior to any cannulation is of critical importance.

To minimise infections, facilities should have a procedural policy for patient VA preparation.[331] HD nurses should clean the skin with a facility approved antimicrobial preparation. There are several such cleansing solutions available for VA disinfection each one requiring a different length of application and time to be effective.[331] The HD staff should wear clean gloves for cannulation.[323,325] Circular cleansing is generally preferred over the east-west technique although there is no hard evidence to support this at present.

6.3.1.2. Anaesthesia. Pain related to cannulation is a significant concern for some patients. Anaesthetics available for needle insertions include: topical creams such as those containing both lidocaine 2.5% and prilocaine 2.5%, intradermal lidocaine injection, and coolant sprays which cause reduced pain sensation by rapid skin cooling on evaporation.

It has been shown that the depth of anaesthesia with topical anaesthetic creams depends on the contact time: In order to reach a maximal depth of 3 mm, the topical anaesthetic cream has to remain on the skin for 60 minutes and to reach a depth of 5 mm the cream has to be on the skin for 120 minutes.[332] Side effects are rare but include redness/rashes or whitening at the site of the application.

6.3.1.3. Pre-cannulation examination. VA stenosis is the most common cause of VA dysfunction. Monitoring by physical examination to detect the physical signs of dysfunction, before any cannulation, is of utmost importance. Monitoring should consist of a full physical examination of the VA prior to every HD session including inspection, palpation and auscultation.[318–322,331] Inspection may reveal swelling, signs of infection (redness, discharge, oedema), aneurysms, haematoma of the hand and stenosis. Palpation should reveal a characteristic thrill. A change in the strength of the pulse over a short segment may indicate a stenosis, while a pulsatile AVF indicates the presence of a downstream or distal stenosis. Post-stenotic collapse of the vein on elevation of the arm can demonstrate the haemodynamic relevance of a stenosis. The VA should have a bruit on auscultation, which will be high pitched over a stenosis.[48]

Monitoring should also include a review of regular routine laboratory tests, including HD adequacy (urea reduction ratio or Kt/V), and difficulties in cannulation or achieving haemostasis after needle withdrawal, documented recirculation, and other clinical clues.[333] Observed changes over time should be documented and further investigated by means of vascular imaging techniques like DUS, DSA or MRA. Physical examination for the detection of stenosis has a positive predictive value of 70%–80% in AVGs and a specificity of 93% in AVFs.[288,318–321,334]

6.3.2. Cannulation techniques

6.3.2.1. Needle selection. It is important to choose the appropriate needle according to the desired blood pump speed and the available VA flow rate in the VA in order to optimise HD efficiency.

Needle selection is especially critical for the initial cannulation. One method used to select the appropriate needle size is a visual and tactile examination. This examination allows the person performing the cannulation to determine which needle gauge would be most appropriate, based on the size of the vessels of the fistula. If the needle is larger than the diameter of the vein with the tourniquet applied, it may cause damage with cannulation. The needle size should be equal to or smaller than that of the vein (without tourniquet). It is also important to match needle gauge to the blood flow rate. For initial cannulation attempts the smallest needle available, usually a 17 G, is typically used. If the arterial pressure falls below 200–250 mmHg, and the venous pressure is higher than 250 mmHg, the needle size should be increased (i.e., a smaller gauge number should be used). The arterial needle should always have a back eye (an oval hole/opening at the back site of the needle) to maximise the flow from the VA and reduce the need for rotation and flipping of the needle.[335]

6.3.2.2. Ultrasound assisted cannulation. Cannulation related complications are especially common in patients with a new VA, which may result in the use of CVC or single needle HD, especially in autogenous AVFs.[336,337]

DUS guided cannulation of AVFs might improve the cannulation rate of more difficult AVFs, potentially reducing the time required to commence HD and the number of local cannulation complications, but randomised controlled trials of DUS guided cannulation versus unassisted cannulation are needed.[338,339] Ongoing education and training of the HD staff towards theoretical knowledge and cannulation skills, especially for cannulation of new AVFs is essential.[336,340]

After creation of an AVG most patients experience significant tissue swelling as a result of tunnelling so that palpation of the graft is difficult for the cannulating nurse and painful for the patient. Therefore, grafts should generally not be cannulated for at least 2 weeks after placement and only after the swelling has subsided and palpation along the course of the graft can be performed. Early cannulation grafts should, if possible, be left for at least 24 hours after placement and until after the swelling has subsided so that palpation of the course of the AVG can be performed.[335,341]

There are three methods for cannulation of the VA; the rope ladder technique (rotation of cannulation sites), the area technique and the buttonhole technique (constant site cannulation) (Fig. 6).

6.3.2.3. Rope ladder technique. The rope ladder technique uses the entire length of the cannulation segment for cannulation: every HD session, two new puncture sites are created, with approximately 5 cm between the tips of the arterial and venous needles, and at least 3 cm from the anastomosis, avoiding the previous sites. The rope ladder technique results in moderate vessel dilatation over a long vein segment.[342]

The venous needle is placed in the direction of the blood flow (antegrade). Arterial needle placement can be antegrade or retrograde (against the direction of the blood flow). The direction of the arterial needle will not influence the risk of recirculation as long as the VA blood flow is greater than the blood pump flow.[343—346] Bevel position and flipping of needles is a controversial issue. Both bevel up and bevel down cannulation are acceptable until further studies can demonstrate the risk/benefits of either technique.[316,331,347]

Based on assessment of the VA, the dialysis nurse chooses the unique angle of insertion for the HD needle. Generally, the angle of insertion for an AVF is 25 degrees, and for an AVG 45 degrees.[257] Cannulation of an AVG is different from an AVF; grafts are tougher than autogenous vessels. Cannulation related complications are more often seen in autogenous AVFs than in AVGs.[336] The few publications concerning VA handling and the outcome of specific cannulation techniques advise the rope ladder technique for the cannulation of AVGs, to avoid AVG disintegration and the formation of pseudoaneurysms.[335,337,341]

6.3.2.4. Area technique. With the area cannulation technique there will be repeated cannulation in the same area of the VA. This leads to aneurysmal dilatation of the puncture areas with subsequent stenoses in adjacent regions.[342] Also the overlying skin becomes thinner, which leads to longer bleeding times after the needles are removed. This technique is less widely used, and is no longer recommended.[341]

6.3.2.5. Buttonhole technique. Another cannulation technique is the buttonhole (constant site) technique.[348] The buttonhole technique is not used for AVGs.

The buttonhole technique requires different skills of the dialysis nurse than the rope ladder technique as the AVF needs to be repeatedly cannulated at exactly the same site, using the same insertion angle and the same depth of penetration every time.[348,349] After approximately 6—10 sessions a tissue tunnel track is formed with sharp needles, enabling the subsequent use of blunt needles for cannulation. Ideally, a single nurse should cannulate the fistula until an established track is created to reduce the risk of track malformation. The cannulation sites should be selected carefully in an area without aneurysms and with a minimum of 5 cm between the tips of the needles. After a good puncture route is established, the fistula can be punctured with dull edged needles, to prevent damaging the tissue tunnel and the formation of faulty tracks.[350] Following transition to blunt needles, a single cannulator is no longer required. Subsequent cannulators should only use blunt needles and must follow the direction and angle of the developed track.[351,352]

Observational studies have shown several benefits of buttonhole cannulation with reduced complication rates: lower infiltration rates resulting in a reduced incidence of haematoma formation,[327,351,353] fewer aneurysms,[327,351,353] improved haemostasis times[353,354] and less pain during cannulation.[349,355] Various studies have also reported that the

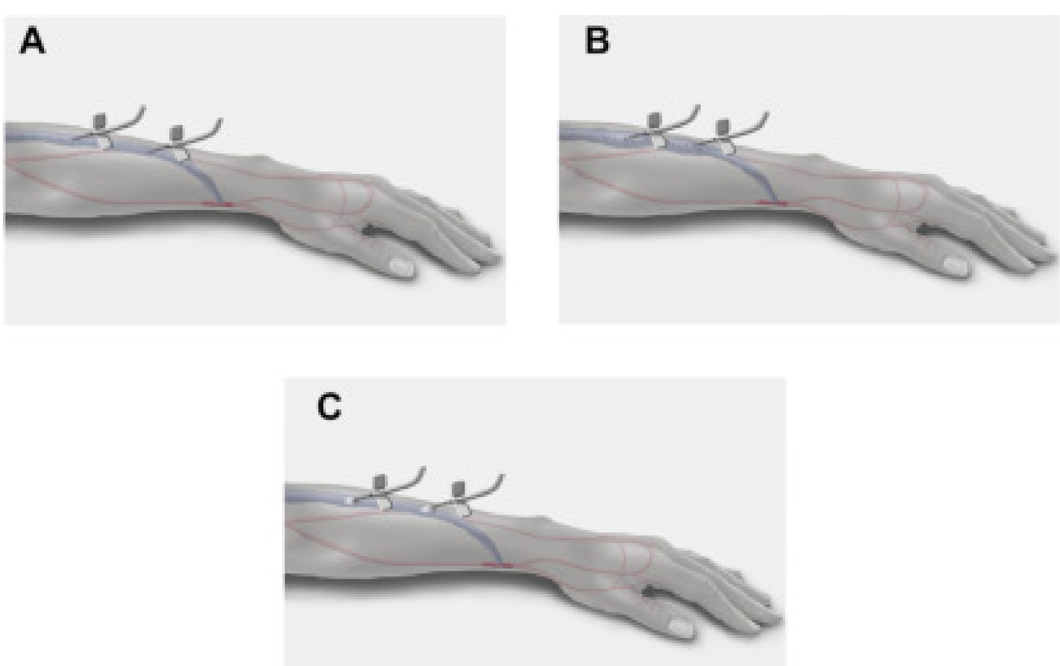


Figure 6. VA cannulation techniques: A: rope ladder technique, B: area technique, C: buttonhole technique.

buttonhole technique contributes to cannulation ease for self-cannulating patients,[351,356,357] which extends the life expectancy of the AVF.[358–360] RCTs regarding the potential benefits of the buttonhole technique have also demonstrated a reduced incidence of aneurysms[361] and fewer haematomas,[361,362] but did not find difference in pain.[361–363]

Studies have reported an increased risk of infection in patients cannulated by the buttonhole technique.[327–330,351,352] These infections ranged from minor skin infections at the VA site to bacteraemia sepsis. Inappropriate application of the disinfection protocol with incomplete scab removal by nursing staff or self-cannulating patients was highlighted as a probable cause of increased infection rates.[327,330] Staff re-education regarding cleansing technique and scab removal resulted in a reduction of infection rates.[329,364]

Correct needle placement with approximately 2 mm of the needle exposed, can prevent the development of large scab formation in buttonhole sites.[349] The best demonstrated practice, touch cannulation technique,[365] decreases the ability of staff members to manipulate needles, resulting in better cannulation success. Antimicrobial prophylaxis has been studied in patients using the buttonhole technique with favourable results.[364,366]

Currently, the available literature does not recommend the routine use of the buttonhole method in all AVFs. However, the buttonhole cannulation technique may be especially appropriate for patients with a short cannulation segment.[367]

Several studies have highlighted the importance of staff experience on VA outcomes. The DOPPS data found that every 20% increase in the number of experienced staff nurses (nurses who had worked in HD >3 years) was associated with an 11% reduction in AVF failure (RR $=$ .89; $p < .005$) and 8% reduction in AVG failure (RR $=$ .92; $p < .001$). Careful consideration of individual AVF and patient characteristics, patient preference and the primary cannulator is required when choosing the most appropriate cannulation method. Cannulator inexperience may result in VA complications regardless of the technique adopted.[368] Therefore, successful VA cannulation requires a high level of awareness and skills of the dialysis nurse, frequent monitoring, and a continued evaluation and education of the needling technique.[327]

6.3.3. Access care after needle withdrawal. To protect the VA from damage and to facilitate proper haemostasis, the technique of needle removal is as *important* as that of cannulation. The needle should be removed at approximately the same angle as it was inserted. After the needle is removed, gentle direct pressure should be applied to the needle exit sites of both the skin and graft or vessel wall, using a two digit technique over a haemostatic dressing.[335] Pressure to the puncture site should not be applied until the needle has been completely removed, to prevent damage of the VA.[331] In general, AVGs require a longer time to achieve haemostasis than AVFs. Whilst compressing, it is important to ensure a flow can be felt in the VA.[335]

The use of clamps to assist haemostasis should be discouraged. When clamps are used, they should only be applied to a mature VA with adequate flow which is monitored closely, and should be used only if flow can still be palpated in the AVF or AVG while the clamp is in place. A dressing should be applied to the cannulation sites using any number of options (with or without a haemostatic agent), but should not encircle the limb to avoid constriction of blood flow to the VA. Prior to the patient leaving the unit, the quality of the bruit and thrill should be assessed and documented.

Difficulties in cannulation or achieving haemostasis after needle withdrawal can be a sign of venous outflow stenosis in a patient with normal bleeding times. If prolonged haemostasis is ongoing, the anticoagulation should be reassessed, dynamic venous pressure readings should be reviewed, and VA flow studies performed to rule out stenosis as a cause.

Recommendation 40	**Class**	**Level**	**Ref.**
Strict aseptic technique is recommended for all vascular access cannulations.	I	C	
Recommendation 41			
Physical examination of the vascular access prior to any cannulation is recommended.	I	C	318–322, 331
Recommendation 42			
In patients with a short cannulation segment the use of the buttonhole technique should be considered over other techniques.	IIa	C	289,342,348, 367,369
Recommendation 43			
The rope ladder technique should be used for cannulation of arteriovenous grafts.	I	C	316,335,341

6.4. Access monitoring and surveillance

6.4.1. Concept. VA function and patency are essential for optimal management of HD patients. Low VA flow and loss of patency limit HD delivery, extend treatment times, and may result in under-dialysis that leads to increased morbidity and mortality.[370] In long-term VAs, especially AVGs, thrombosis is the leading cause of loss of VA patency and increases healthcare expenditure.[371,372] VA related complications account for 15%—20% of hospitalisations among patients undergoing HD.[370,373,374]

The basic concept for VA monitoring and surveillance is that stenoses develop over variable intervals in the great majority of VAs and, if detected and corrected, underdialysis can be minimised or avoided (dialysis dose protection) and the rate of thrombosis can be reduced. Whether prospective monitoring and surveillance can prolong VA survival is currently unproven. A number of monitoring and surveillance methods are available: sequential VA flow, sequential dynamic or static pressures, recirculation measurements, and physical examination.[375]

A multidisciplinary team should be formed at each HD centre[376,377] with a VA team coordinator working proactively to ensure the patient is receiving an adequate HD dose by maintaining VA function and patency.[376,378]

Recommendation 44	Class	Level	Refs.
Routine physical examination is recommended for vascular access surveillance and monitoring.	I	B	318,319,321, 382—384

6.4.2. Monitoring. Monitoring is the examination and evaluation of the VA to diagnose VA dysfunction using physical examination, usually within the HD unit, in order to detect the presence of dysfunction and correctable lesions before VA loss.

6.4.2.1. Physical examination. Physical examination can be used as a monitoring tool to exclude low flow associated with impending fistula and graft failures. There are 3 components to the VA examination: inspection, palpation, and auscultation.[379,380]

A simple inspection can reveal the presence of swelling, ischaemic fingers, fingertip wounds like paronychia, aneurysms, and rich collateral veins. The detection and referral of patients with a non-healing crust over the puncture site can save lives. A strong pulse and weak thrill in the vein central to the anastomosis indicates a draining vein stenosis. A fistula that does not at least partially collapse with arm elevation is likely to have an outflow stenosis. Strictures can be palpated and the intensity and character of the bruits can suggest the location of stenoses.

In AVGs, the direction of flow is easily detected using a simple compression manoeuvre on the middle segment of the graft, the pulsating part indicates the arterial side and the non-pulsating the venous side, thus avoiding inadvertent recirculation by reverse needle insertion. A local intensification of bruit over the graft or the venous anastomosis compared with the adjacent segment suggests a stricture or stenosis.[380,381]

Monitoring by physical examination is cost-effective and a proven method to detect VA abnormalities.[318,319,321,382,383] Unfortunately, nephrologists and HD staff generally have limited knowledge of VA anatomy and function, and regular physical examination of VAs is not generally carried out in HD units. This trend should be reversed by emphasising proper VA training and clinical assessment in HD units.[295,380,384] Clinical monitoring appears to provide equivalent benefit in terms of VA survival in comparison with surveillance programs when coupled with pre-emptive corrective intervention.[385,386]

6.4.3. Surveillance. Surveillance is the periodic examination and evaluation of the VA by using diagnostic tests that may involve special instrumentation to diagnose VA dysfunction. It can be done periodically during or outside HD sessions, to diagnose VA dysfunction, or when monitoring indicates VA dysfunction. The aim of surveillance is the detection of correctable lesions that may necessitate pre-emptive intervention to prevent VA loss. Some diagnostic imaging modalities can also be used to locate the cause of the VA dysfunction.

6.4.3.1. Surveillance during haemodialysis.

6.4.3.1.1. Flow measurement methods. VA blood flow can be measured indirectly by using indicator dilution techniques, or directly by using either DUS or MRA.[155]

6.4.3.1.1.1. Indirect flow measurement. The ultrasound dilution technique (UDT) is the most well validated method for indirectly measuring VA blood flow (Qa).[387—392] In this technique, an indicator (saline) is infused distally into the VA after line reversal. Ultrasonic sensors measure changes in the protein concentration producing dilution curves used for the calculation of Qa. Several factors have been identified that directly influence the accuracy of the measurements.[390,393] Firstly, thorough mixing of the indicator is required. Secondly, as a result of cardiopulmonary recirculation (CPR), the second pass of the indicator will produce errors if it is incorporated into the measurement. CPR increases as VA blood flow increases ($CPR = Qa/CO$) and if incorporated, will cause an underestimation of the true Qa value. Thirdly, the reversal of the blood lines that is required to perform the measurement will also influence the VA blood flow result.

Fourthly, blood pump flow delivered to the dialyser (Qb) must be measured accurately as readings from the blood pump have been shown to overestimate delivered Qb by 10%—20%.[394]

6.4.3.1.1.2. Direct flow measurement. DUS measures blood flow velocity and in order to determine blood flow, cross sectional area needs to be measured. The estimated flow can be inaccurate due to operator dependent determination of the blood velocity, and may be subject to error in estimation of the cross sectional area and the Doppler angle.[394—398] Advances in technology have made newly designed instruments more accurate and reproducible in measuring flow.[291,399] The most popular method of flow measurement is calculation of the flow in the proximal brachial artery and subtracting the flow in the contralateral brachial artery, which is usually between 40 and 150 ml per minute.[400] This technique is supported by most DUS machines using automated multiplication of the time averaged mean velocity in the cross sectional area. VA flow can also be measured by MRA. However, apart from the danger of nephrogenic systemic fibrosis/fibrosing dermopathy, and as this technique is expensive and cannot be performed during HD, it is impractical as a screening tool.

6.4.3.1.2. Access flow and pressure surveillance. AVGs are notorious for recurrent thrombosis due to venous stenosis, necessitating frequent intervention. Dynamic and static dialysis venous pressure (VP) measurements combined with pre-emptive PTA yielded large reductions in thrombosis rates and replacement of VAs.[401,402] These reports led the NKF-KDOQI guidelines to recommend that AVGs and AVFs undergo routine surveillance for stenosis with pre-emptive correction.[403]

The rationale for surveillance is based on the hypothesis that progressive stenosis is detected before thrombosis and VA loss, and a corrective procedure such as PTA can maintain patency of the VA. Non-randomised or observational studies are biased towards finding a treatment benefit.[404] For example, the influence of Qa on the relative risk of thrombosis was used to justify surveillance.[405–407] Although a low Qa is associated with an increased risk of thrombosis, this association does not have adequate accuracy in predicting thrombosis. In contrast, Qa and VP surveillances were found to be inaccurate predictors of graft thrombosis and instead of preventing thrombosis yielded many unnecessary intervention procedures.[408–412] Moreover, PTA induces a mechanical trauma, accompanying neointimal hyperplasia (NIH), risk of stenosis and impaired VA survival.[413] Surveillance guidelines should consider differences in risk of thrombosis. For example, newly constructed grafts have a higher risk of thrombosis than established grafts.[412]

Qa and VP surveillance might improve outcomes if measurements are taken more frequently neutralising haemodynamic variation. Using trend analysis to guide referral decisions rather than relying on a single measurement could be more efficient.

Thus, the screening test should take into account the risks associated with each patient, such as graft age or previous thrombosis, and should not be based solely upon a single Qa measurement.

A systematic review and meta-analysis of available randomised controlled trials evaluated Qa or DUS in AVFs and AVGs.[385] Flow surveillance of AVFs was associated with a significantly reduced relative risk of thrombosis, but no significant improvement in AVF survival. By contrast, there was no evidence that AVG surveillance by flow or DUS reduced thrombosis or improved AVG survival.[385]

Another systematic review and meta-analysis found that serial surveillance of asymptomatic VA for detection and treatment of stenosis may reduce the risk of thrombosis and prolong VA survival more than normal clinical monitoring but these improvements were not statistically significant.[386]

The low yield of VA surveillance led researchers to suggest that the current surveillance paradigm might be false and that perhaps there should be a search for a new paradigm.[414]

Modified recommendations were suggested for using Qa and VP measurements in VA maintenance emphasising the importance of physical examination and clinical assessment.[415] Qa or VP measurements should be correlated with physical and clinical examination but are not appropriate as the sole basis for intervention referrals. AVF Qa $<$ 500 ml/min and AVG Qa $<$ 600 ml/min are associated with stenosis, but should be confirmed and correlated with clinical findings when making an intervention referral. The decrease in Qa should be $>$33% since smaller decreases might be caused by haemodynamic variation.[416] Trend analysis is essential to using static venous pressure adjusted for the mean arterial pressure (VP/MAP) to detect a significant stenosis. The traditional threshold should not be the only basis for an intervention referral.[417]

6.4.3.1.3. Dialysis efficiency measurements.

6.4.3.1.3.1. Recirculation. VA recirculation results from the admixture of dialysed blood with arterial VA blood without equilibration with the systemic arterial circulation of dialysed and non-dialysed blood. AVF recirculation has two components, VA recirculation that may occur when the blood pump flow is greater than VA flow and cardiopulmonary recirculation that results from the return of dialysed blood without full equilibration with all systemic venous return such as in patients with cardiac disease.

Even with ideal sample timing and proper cannulation, laboratory variability in urea based measurement methods will produce variability in calculated recirculation.[418,419] Therefore, individual recirculation values less than 10% using urea based methods may be clinically unimportant. Values greater than 10% using urea based recirculation measurement methods, require investigation.

Recirculation rate and VA function are closely correlated and it can be assumed that improvements in recirculation rate and HD efficiency are parallel. Thus the use of recirculation rates in evaluation of the indications for and effects of PTA might be expected to contribute to an objective assessment method. The immediate recirculation rate is determined by using the haematocrit dilution technique.[388,420] The total rate per HD session is reflected by the urea clearance gap. The correlation between Kt/V and immediate recirculation rate is not clear and it may be more appropriate to assess recirculation rate and HD efficiency of the total recirculation.[421]

6.4.3.1.3.2. Urea reduction ratio and dialysis rate. Kt/V has been suggested as an objective evaluation method for AVF.[422–424] However, it is associated with multiple factors in addition to urea clearance, including the length of HD and blood flow volume (Qb) which can affect Kt/V values. It is necessary to include the recirculation rate as a factor in functional evaluation of an AVF.[419]

Unexplained decreases in delivered dialysis dose, measured by using Kt/V or urea reduction ratio (URR), are frequently associated with venous outflow stenoses.[425]

However, many other factors influence Kt/V and URR, making them less sensitive and less specific for detecting VA dysfunction. Inadequate delivery of dialysis dose is more likely to occur with an AVF than an AVG.

Failure to detect VA dysfunction has consequences for morbidity and mortality,[370,372,426] with significant increases in hospitalisations, hospital days and inpatient expenditure.[426]

Thus the diagnosis of inefficient HD by decreased Kt/V or increased recirculation is very important when accompanied with stenosis. Correction of the stenosis will repair dialysis dose delivery impairment and may improve patient morbidity and mortality.[426,427]

Recommendation 45	Class	Level	Refs.
It is recommended that vascular access surveillance is performed by flow measurement of arteriovenous grafts monthly and arteriovenous fistulas every 3 months.	I	B	405,428,429
Recommendation 46			
When arteriovenous fistula blood flow measurements during dialysis indicate the presence of a vascular access stenosis based on a Qa <500 ml/min, angiographic assessment of the access should be considered.	IIa	B	427,430
Recommendation 47			
Venous pressure adjusted for the mean arterial pressure >.50 (or derived static venous pressure adjusted for the mean arterial pressure >.55) is not a reliable indicator of stenosis and intervention based on this finding is not recommended.	III	C	417
Recommendation 48			
When haemodialysis efficiency is impaired, investigation and correction of an underlying vascular access stenosis should be considered.	IIa	B	370,425,426

Recommendation 49	Class	Level	Refs.
Surveillance of arteriovenous fistulas with duplex ultrasound at regular intervals and pre-emptive balloon angioplasty should be considered to reduce the risk of arteriovenous fistula thrombosis.	IIa	A	385
Recommendation 50			
Surveillance of arteriovenous grafts with duplex ultrasound at regular intervals and pre-emptive balloon angioplasty is not recommended to prevent thrombosis or improve arteriovenous graft functionality.	III	A	385,386

6.4.3.2. Surveillance outside dialysis sessions. Surveillance outside HD sessions can be performed using DUS, MRI, CTA or DSA.

6.4.3.2.1. Ultrasound. DUS is the main imaging modality for VA surveillance. DUS can enhance the understanding of the physiology and pathology of every VA. DUS has been described in Chapter 4.

6.4.3.2.2. Angiography. DSA is the gold standard for the evaluation of VA patency. DSA can be and is used in some centres as a primary surveillance method when clinical monitoring findings indicate VA dysfunction or after DUS examination.

6.4.3.2.3. Magnetic resonance angiography. CE-MRA has been introduced for the evaluation of failing AVFs and AVGs. But it is not recommended in CKD patients due to gadolinium induced NSF.[431,432]

NCE-MRA is an evolving technology that has been proposed to replace CE-MRA while avoiding the risk of NSF. The technology and algorithms are constantly improving but the instruments are as yet expensive and cannot be used widely.[118,150]

6.5. Nursing organisation

6.5.1. Introduction. In the last decades, it has been recognised that nurses play a pivotal role in VA management[433,434] and surveillance.[435] Within Europe, organisation between HD centres varies from country to country.[436,437]

The increasing age and comorbidities of HD patients have resulted in more complex VA,[438] demanding higher levels of expertise in VA management. The coordination of clinical care pathways increasingly relies on nurses[439] from the early stages of planning[48,440,441] to cannulation and HD itself.[341,442] Moreover the expansion of home HD[443–445] has increased the need for patient education and communication skills and remote clinical surveillance.[446,447]

6.5.2. Nursing organisation. Nurses comprise the largest group of healthcare workers and the way in which they organise their work has considerable effects on patient satisfaction and clinical outcomes. There is a consensus that involvement of nurses in clinical management generates clear benefits.[448–452]

6.5.2.1. Nursing models. Nurses professionally involved in HD care planning and audit improve their experience and accountability which increases self-esteem and maintains enthusiasm.[453] Case Management,[454] Primary Nursing[455–458] or similarly structured working models,[459,460] applied to the HD setting, have proved to have positive impact on clinical outcomes as well as management performance.[461–463]

6.5.2.2. Clinical governance. Clinical Governance is defined as a framework through which healthcare organisations are accountable for continuous quality improvement by creating an environment in which excellence in clinical care will flourish. Implementing this concept to VA management should enhance the quality of care, decrease clinical risks and improve clinical outcomes in HD patients.[464–467]

For this reason, many countries have invested in the specialist VA nurses role.[325,468,469]

6.5.2.3. Vascular access nurse.

VA nurse areas of competence:

- Developing and implementing protocols for staff support and patient education
- VA monitoring program implementation
- VA data collection and audit
- Infection and adverse outcome monitoring
- Quality control of VA care
- Central line insertion (after specific training)

6.5.2.3.1. Basic role of vascular access nurse. The first step for a VA management strategy within the HD care team is the appointment of a VA nurse. The VA nurse should be skilled in VA needling and patient care. She/he should be willing to attend VA continuing education activities and should be willing to organise education programs for nurses within the HD service. She/he should be involved in data collection on fistula/graft rate, adverse events, CVC type, VA infection rate and staff turn-over, starting as soon as the VA nurse is appointed and kept thereafter as a continuing quality control audit program. The VA nurse should have a well defined job description, which allows some autonomy, whilst carefully defining the role and relationships with other team members.

6.5.2.3.2. Vascular access nurse coordinator and manager. These represent possible developments of the basic VA nurse role.[473] A VA nurse coordinator is responsible for building up and coordinating the VA nurse team work, nursing activities and pathways of care, patient preparation and education in all settings relating to VA implementation, communication with the VA surgeon, and/or VA interventionalist follow up after surgery, organisation of the first treatment/cannulation. Other activities include organising audits and defining protocols for CVC and AVF management. She/he should have a central role in the multidisciplinary care team. This role requires a full time post in large HD units. The VA coordinator should be a highly skilled and educated nurse, able to support HD nurses in any difficult cannulation or to help with CVC management queries.

A multidisciplinary approach to VA including a VA nurse coordinator reduces re-hospitalisation and complications such as VA thrombosis.[376] This results in extending VA life and reduces the rate of CVC use.[376,378]

Large HD services appoint VA nurse managers. Their activities focus on administration, team management for data collection and evaluation, and political decision making.

Recommendation 51	Class	Level	Refs.
The appointment of one or more vascular access nurses should be considered to improve patient care and clinical outcomes in each haemodialysis service.	IIa	C	378,473

The role and responsibilities of the VA nurse vary from unit to unit. The responsibilities of the VA nurse range from the pre-dialysis and outpatient service to communication with the VA surgeon, coordination of the surgery list and patient and staff education with specific emphasis on cannulation. The VA nurse role can be stratified into three levels, referred to as a VA nurse, VA nurse coordinator[86,470,471] or VA nurse manager.[472]

In larger units the VA nurses work in teams where each member has different responsibilities and roles within the team.

In order to provide examples of VA nurse implementation, the following roles could be introduced in a progressive manner:

6.5.2.4. Future developments. The progression of nursing VA competence enhances the need to organise specific post-graduate VA nursing education, which could be a specific module within a nephrology nurse post-basic education course or VA masters course,[474] in conjunction with universities, industries, professional and patients' associations.

7. LATE VASCULAR ACCESS COMPLICATIONS

7.1. True and false access aneurysms

Generalised vessel enlargement is a normal finding in autogenous VA due to flow induced vascular remodelling.

Aneurysms are localised dilatations, whereas true VA aneurysms involve all layers of the vessel wall and false aneurysms have a wall defect.[475] AVF aneurysms are frequently caused or accompanied by pre-aneurysm or post-aneurysm stenosis.[476,477] A haemodynamically significant stenosis will lead to pulsation of the distal vein and reduced or missing thrill proximally and lead to aneurysmal dilatation.[478–481] Segmental aneurysms without a stenosis may be due to repeated needling in the same area. Large aneurysms can be complicated by wall-adherent thrombi producing local signs of aseptic thrombophlebitis, which can mimic cellulitis secondary to bacterial super-infection of a thrombus. Rapidly growing aneurysms lead to necrosis of the overlying skin and the risk of spontaneous rupture and bleeding. In contrast to AVFs, AVGs do not dilate but false aneurysms may develop after graft destruction from repeated needling or at the anastomosis.[482]

VA aneurysms have been reported in up to 17% of AVFs and false aneurysms in 7% of AVGs.[358] VA aneurysms are easily detected on clinical inspection but DUS allows detection of associated stenoses and wall-adherent thrombi. VA aneurysms with a thin overlying skin, skin erosion or bleeding should be evaluated and treated urgently[476] but aneurysm diameter per se does not correlate with complications.[477]

Cannulation should be avoided in the affected area, especially when this has a thin (often shiny) overlying skin prone to infection, which is a sign of impending perforation. In cases of aneurysm and stenosis progression, surgery with partial resection of the wall of the aneurysm (aneurysmorraphy) and insertion of the resected material as patch along the concomitant stenosis is common.[476,477] Stepwise resection of the aneurysm wall and resizing over a Hegar's probe helps to form a suitable conduit for future cannulation. Other procedures include ligation of the aneurysmal section and bypass or graft interposition. Venous anastomotic aneurysms with a post-stenotic lesion are treated by resection of both lesions and graft interposition to the vein distally. AVG pseudoaneurysms are treated by resection and interposition or bypass. The presence of infection requires exclusion of the aneurysmal section and in most cases, complete resection of the graft (see Section 7:2). In all cases where surgery can provide optimal inner diameter while preserving cannulation sites, PTA should be the second choice. Very little literature exists on the results of surgical treatment of aneurysms. In a small series of 44 VA patients aneurysms or pseudoaneurysms developed in 26 AVFs and 16 AVGs.[477] Primary patency for AVFs was 57% at 12 months and 32% after 48 months.[477] AVFs also fared better than AVGs. In another series of 33 patients the aneurysm was reinforced by an exoprosthesis after aneurysmorraphy which resulted in a 1 year primary patency rate of 93%.[483]

Different types of stent grafts have been used in endovascular treatment of VA aneurysms and remain an option in selected cases.[478,484–490]

Recommendation 52	Class	Level	Refs.
Surgical revision of vascular access aneurysms is recommended if cannulation sites and access diameter can be preserved.	I	C	477,481
Recommendation 53			
Surgical revision of pseudoaneurysms in arteriovenous grafts is recommended when the aneurysm: - limits the availability of cannulation sites or - is associated with pain, poor scar formation, spontaneous bleeding and rapid expansion.	I	C	477,491
Recommendation 54			
Stent graft exclusion of vascular access aneurysms may be considered in selected patients.	IIb	C	479,491,492
Recommendation 55			
Access cannulation through a pseudoaneurysm is not recommended.	III	C	482,493
Recommendation 56			
Outflow stenosis should be ruled out in symptomatic vascular access aneurysms and treated when present.	I	C	479,481,492

7.2. Infection

VA infection is the major type of infection in HD patients and the second most frequent cause of death in these patients, only exceeded by cardiovascular disease.[476,494,495] Uremia, diabetes, multiple comorbidities, CVCs and repeated cannulation of the VA are important risk factors.[260] Infections occur most commonly in association with CVCs, followed by AVGs and rarely in AVFs.[260] Diagnosis is clinical with local signs such as redness, warmth, tenderness, swelling and purulent discharge or skin erosion or ulceration. However, occult infections do occur with fever as the only symptom. DUS may be used to look for peri-graft fluids and radiolabelled leucocyte scans are both sensitive and specific. Non-used VAs may pose an infectious risk which is often not apparent clinically.[137,260,496]

Infections are caused predominantly by gram positive cocci (*Staphylococcus aureus* 50–90%, *S. epidermis, Streptococcus viridans, and Streptococcus faecalis).*[495,497,498] Gram negative organisms are found in about 33% of infections.[495,497–499] Total excision is suggested for grafts infected with *S. aureus*, while *S. epidermidis* is less virulent and subtotal or partial excision can be planned.[260] In two studies MRSA infection was associated with higher mortality compared with methicillin susceptible strains of *S. aureus* in HD patients.[500,501] However, no causal relationship between MRSA and VA infections has been established.[495] AVG infections have been shown to be more common in HIV positive patients (30%) compared with HIV negative (7%) patients. However, no significant increase in VA related infections have been observed in HIV positive patients with AVFs and irrespective of CD4+ counts.[502,503] Due to their immune incompetence, AVGs should therefore be avoided in HIV patients.

Late infections are more frequent (50%) and associated with routine HD.[256,260]

effective way to eradicate infection but usually necessitates placement of a CVC and is associated with a significant amount of tissue destruction when removing established infected grafts.[260,504,505] Subtotal excision refers to removal of the graft leaving only a small stump on the arterial side to be closed. This approach avoids extensive dissection of the artery and the risk of nerve damage. If the infection is localised to a segment of the graft and ultrasound shows no perigraft fluid along the rest of the graft, partial excision of the graft can be performed[488] and temporary CVCs avoided.

Infected graft outcomes are best following total graft removal (1.6% recurrence rate), less good with subtotal excision (19%) and least good (29%) after partial excision.[260,506–510] The literature diverges on the efficacy of conservative treatment (only antibiotics) and the reason may be that some of the patients do not have a definite infection but simply a reaction to the prosthetic material that spontaneously resolves and is erroneously interpreted as an infection. Lately, reports of conservative treatment of infected AVGs with antibiotics, aggressive debridement and NPWT dressing have emerged but the experience is far too scarce to justify any recommendations.[511]

Recommendation 57	**Class**	**Level**	**Refs.**
All vascular access late infections should be treated with antibiotics to cover both gram positive and gram negative organisms.	I	C	497,498
Recommendation 58			
In late vascular graft infection total arteriovenous graft excision is recommended in patients with sepsis, clinical signs of infection, and peri-graft fluid around the whole graft.	I	C	260
Recommendation 59			
Partial excision of an arteriovenous prosthetic graft may be considered in selected cases when sections of the graft are well incorporated and appear to be uninfected.	IIb	C	507,508

In AVFs, rare infections at the AV anastomosis require immediate surgery with resection of the infected tissue. More often, infections in AVFs occur at cannulation sites, especially in buttonhole cannulation with inadequate aseptic technique. Treatment consists of avoiding cannulation at that site. In all cases of AVF infection, antibiotic therapy is begun empirically with broad spectrum antibiotics and then narrowed down based on culture results. Infection of primary AVFs should be treated for a total of 6 weeks, analogous to subacute bacterial endocarditis, however, proper evidence is lacking.[476]

AVG infection is associated with risk of sepsis and suture line disruption with life threatening bleeding.[260] In general, extensive perigraft effusion requires complete graft removal while in some late infections unaffected well incorporated graft segments can be saved.[260,504] Late AVG infection may be caused by transient bacteraemia from a distant site such as infection in the oral cavity. Antibiotic treatment alone is rarely sufficient and may instead require a combination of antibiotics and graft excision. Total graft excision is the most

7.3. Stenosis and recurrent stenosis

Stenosis can occur at any level from the arterial inflow to the venous outflow, often in the juxta-anastomotic areas or even within the graft.[512] Pre-emptive treatment of all stenoses has not been shown to be of benefit.[334,513–517] Therefore only stenosis that have a haemodynamic effect ($\geq$70% decrease in lumen area) and are associated with decreased flow, elevated venous pressures, or an abnormal physical examination (reduced thrill or pulsatile flow) should be treated. The main benefit of pre-emptive treatment of haemodynamically significant stenoses is decreased thrombosis, avoidance of sub-optimal HD and CVCs, and not necessarily prolonged life of the VA.[513,515,516,518]

7.3.1. Inflow arterial stenosis. Stenoses in the subclavian, brachial, radial or ulnar artery are more frequent in the elderly, in diabetics and in hypertension. In addition, stenoses often develop at the arteriovenous anastomosis of AVFs or the arterial anastomosis of AVGs. A prospective multicentre study has demonstrated that about 30% of referrals for stenosis intervention were due either to stenosis in the native artery or at the anastomotic site.[518] In another study 12.5% of dysfunctional AVFs and AVGs were due to inflow stenosis and in 77% endovascular treatment was successful.[519]

PTA is a safe and effective technique with a low rate of re-intervention.[518] For elastic recoil, rapidly recurrent stenosis, or residual stenosis >30% after PTA, the implantation of a stent is recommended.[476] Open options for treatment of stenoses in the native arteries include bypass grafting and endarterectomy but are seldom performed. No randomised studies have been performed between open and endovascular surgery.

7.3.2. Juxta-anastomotic stenosis. For haemodynamic reasons, stenosis often develops in the juxta-anastomotic area around either the arteriovenous anastomosis of AVFs or the arterial anastomosis of AVGs and the first few centimeters (2—5 cm) into the vein/graft.[512]

Traditionally open surgery with creation of a new proximal anastomosis or graft interposition of a short ePTFE graft, has been the preferred method in forearm AVFs,[520] although PTA can be an alternative.[521,522] It has been demonstrated that PTA can be used as the primary approach for juxta-anastomotic stenosis.[523] However, the recurrent stenosis rate is higher than after surgery, and in those patients where early recurrence occurs, surgical revision is indicated. If surgical revision is expected to shorten the usable length of the AVF for cannulation PTA is justified as the primary tool.

7.3.3. Venous outflow stenosis. Reduced VA flow, prolonged bleeding times and elevated venous pressure suggest the presence of a venous outflow stenosis often where the peripheral vein enters the deeper system. PTA is the first treatment option in the outflow veins (cephalic/basilic), especially when the lesion is short (<2 cm). For long segment stenoses (>2 cm), treatment is controversial, including PTA or surgery either by bypass grafting or vein transposition. Grafts should be reserved for patients with exhausted peripheral veins whilst fistula preserving procedures such as PTA or patch angioplasty should be favoured over graft extensions to central venous segments.

Venous outflow stenoses may be resistant to PTA and require high pressure balloons or cutting balloons.[524] Stents or open surgery should be considered if repeated PTA fails. Clinical trials comparing stenting with PTA did not show statistically significant differences in patency.[525—527] Stents used in previous RCTs may have been inferior to more recently used devices especially when nitinol stents were used.[528—530] The use of stent grafts to treat VA stenosis has recently gained consensus since they may decrease the incidence of restenosis by interposing an inert layer to separate the thrombogenic vascular wall from the blood flow and impede the migration of smooth muscle cells.[531] Stent grafts mimic open surgical revision of a graft, preventing elastic recoil and avoiding trans-stent growth of neointimal tissue. A multicentre RCT showed better patency rates for stent grafts vs. simple PTA for the treatment of AVG anastomotic stenosis with a sustained, greater than 2 fold advantage over PTA in the treatment area for primary patency and overall VA patency.[532] Similar favourable results for stent grafts were found in another RCT when treating in-stent restenosis in patients with AVFs and AVGs.[533]

Concerns remain about costs, and on the real value in preventing graft thrombosis.[534] Thus the use of stent grafts to treat AVG venous anastomosis stenosis is reserved for complicated cases. The consensus is that for stenting the venous anastomosis and venous stenoses, stent grafts may be superior to bare stents.

7.3.4. Cephalic arch stenosis. The cephalic vein forms part of the outflow for RCAVF and is the sole outflow for BCAVF. The cephalic arch is prone to the development of haemodynamically significant stenosis[512,528] related to its perpendicular junction with the deeper veins. Stenosis in this region is common and is usually treated by PTA.[512] The cephalic arch is the most frequent location for stenosis of upper arm dysfunctional AVFs, comprising 30%—55% of all upper arm VA stenosis sites.[535] It responds poorly to PTA, with a 6 month primary patency rate of 42%,[476] which is below the 50% unassisted patency rate recommended for intervention for VA stenosis. In a small RCT, stent grafts were shown to be superior to PTA in treating cephalic arch stenosis.[536] When the result of PTA is poor or if associated with vein rupture, or if there was early restenosis (<3 months), stent grafts can be used.[535,537] Because restenosis after stenting in the cephalic arch is an issue, stent grafts have been suggested as an alternative in early recurrent cephalic arch stenosis after PTA.[535,537,538]

A randomised clinical study on the outcome of 25 consecutive patients with recurrent cephalic arch stenosis has shown the following: DSA at 3 months demonstrated[538] restenosis rates of 70% in the bare stent group and 18% in the stent graft group. Life table analysis at 3 and 6 months showed that primary patency was 82% in the stent graft group and 39% in the bare stent group. One year primary patency was 32% in the stent graft group and 0% in the bare stent group. It was concluded that the use of stent grafts for recurrent cephalic arch stenosis significantly improved short-term restenosis rates and long-term patency compared with the use of bare stents. The major drawback of stent grafts in the cephalic arch is possible occlusion of the axillary or subclavian vein that may prevent further VA in the ipsilateral arm, but the rate of this complication is unknown. Therefore, until long-term results are published the use of stent grafts can only be recommended when it is considered unavoidable by an endovascular specialist. The role of drug eluting balloons (DEB) is currently being examined and may offer an alternative to stents in VA.[539,540] A small RCT showed that DEB angioplasty may be a cost-effective option that significantly improves patency after angioplasty of venous stenoses of failing VA.[541] Since the outflow anastomosis can be considered as an experimental model for NIH, future research direction may clarify whether DEBs may offer an alternative to stents in VA.

As an alternative to endovascular therapy, open surgical revision for cephalic arch stenosis has been described and involves diverting the blood flow to other patent veins for example the axillary vein with a primary patency of 60% at 1 year.[542—544] However, such procedures might jeopardise the creation of a future basilic vein fistula. Furthermore, it has been shown that previous endovascular treatment of the cephalic arch decreases the patency of open surgical revision.[545]

for thrombosed AVFs were identified.[271] To date, no randomised studies comparing the 2 alternatives have been published. In forearm AVFs, thrombectomy plus simple reanastomosis of the vein to the artery proximally had a better 1 year secondary patency rate of 70—90%, compared with 44—89% after endovascular therapy.[271]

Thrombolysis or thrombectomy alone are not sufficient to restore long-term patency, since a flow limiting stenosis is present in more than 85% of the cases.[548] Identification and treatment of these underlying lesions are crucial to optimise the long-term result. The combination of thrombolysis with PTA allows a good immediate result ranging from 88 to 99% success, but re-occlusion is frequent.

Recommendation 60	Class	Level	Ref.
Balloon angioplasty is recommended as primary treatment for inflow arterial stenosis of any type of vascular access.	I	C	518,519
Recommendation 61			
Surgical proximal relocation of the vascular access anastomosis should be considered in juxta-anastomotic stenosis in the forearm.	IIa	C	520
Recommendation 62			
Balloon angioplasty is recommended for the treatment of venous outflow stenosis.	I	C	527
Recommendation 63			
Endovascular treatment with stent grafts should be considered for the treatment of cephalic arch stenosis.	IIa	B	538

7.4. Thrombosis

Thrombosis often presents as the final complication after a period of VA dysfunction and is mainly due to progressive stenosis in the VA or in the outflow. Beside other factors, hypotension is a known adverse factor in fistula survival demonstrated in a study with 463 patients[546] and may cause thrombosis at any time. Treatment needs to be started as soon as possible to prevent organisation of the thrombus and endothelial damage in the vein. Early thrombus removal allows immediate use without the need for a CVC.

7.4.1. Treatment of arteriovenous fistula thrombosis. In AVFs, thrombosis usually begins at a stenosis or puncture site and propagates as far as a downstream side branch that is patent. For example in RCAVFs, patent side branches drain the cephalic vein even when the anastomosis is thrombosed. However, in a transposed basilic vein fistula where side branches are ligated during the transposition procedure, the entire vein is thrombosed. Early thrombus removal is more urgent in AVFs compared with AVGs because endothelial damage and phlebitis may preclude further use of the VA. Furthermore thrombus organisation is more pronounced in native vessels.[547] The duration and site of AVF thrombosis as well as the type of VA are important determinants of treatment outcome. Originally the management was exclusively surgical thrombectomy. Later, in the 1980s percutaneous management was proposed with thrombolysis first, in combination with mechanical thrombectomy later. A review of comparative studies of percutaneous thrombectomy vs. surgical thrombectomy for treatment of AVF thrombosis reveals conflicting results and no definitive preference. In a systematic literature review in 2009, 36 studies on endovascular and surgical intervention

Endovascular techniques include pharmacological thrombolysis (urokinase or tissue plasminogen activator), pharmaco-mechanical thrombectomy (lytic agent combined with mechanical thrombus maceration), mechanical thrombectomy (thrombo-suction, hydrodynamic catheter or catheter with a rotational tool) or a combination of these.[549—551] Pharmacological thrombolysis can result in adequate thrombus resolution but it is time consuming and associated with a higher risk of bleeding and incidence of pulmonary embolisation in comparison with surgery. Mechanical thrombectomy devices significantly reduce procedure time. Independently of the type of device used for pharmaco-mechanical or mechanical thrombectomy, the technical success rates are better in AVGs compared with AVFs (99% vs. 93%), although early re-thrombosis is more common in AVG.[552] A direct comparison between three different mechanical devices for endovascular recanalisation of VA thrombosis revealed that the result of PTA in the treatment of underlying stenoses was the only factor predictive of graft patency.[553]

7.4.2. Treatment of arteriovenous graft thrombosis. Unlike AVF thrombosis, treatment of AVG thrombosis is not as urgent but should be managed without jeopardising VA function for the next HD session. Early de-clotting allows for immediate use of the VA without the need for a CVC. Old thrombi (>5 days) are often fixed to the vessel wall beyond the venous anastomosis, making surgical extraction more difficult than interventional treatment. Surgical thrombectomy is performed with a thrombectomy catheter purposely designed for use in grafts. Intra-operative DSA can visualise the central venous outflow as well as the graft in order to exclude residual thrombi and identify and treat the cause of thrombosis which should be an integral part of any surgical or interventional thrombus removal procedure.

A meta-analysis[269] and a systematic review in 2009[271] concluded that surgical thrombectomy and endovascular therapy had comparable results, in particular for thrombosed prosthetic grafts. A randomised study did not show any significant difference between surgical thrombectomy and endovascular treatment.[554] Additionally in a meta-analysis including six randomised trials surgical and endovascular therapy in AVGs were compared. It was concluded that endovascular therapy had similar results in terms of primary and primary assisted patency at 1 year compared with surgical thrombectomy.[270]

axillary vein segments or subclavian segments beneath the clavicle.[557,558,567–569] New self-expandable dedicated venous stents may be more promising.[570]

In view of the reported superiority of stent grafts compared with bare stents for recurrent cephalic arch stenosis these have been used for cases of symptomatic CVOD,[538,571,572] however, the possible disadvantage of covering major venous confluences must be considered.[573,574]

Despite a significant morbidity, surgical revision should be considered in patients with CVOD and failed endovascular attempts.[575–578] Various procedures include bypassing the central occlusion (axillary or brachial vein to jugular vein; axillary vein to saphenous or iliac veins), intrathoracic central venous reconstructions, extra-anatomical venous reconstruction, and non-venous VA (axillary or brachial artery to right atrium bypass). Hybrid procedures combining surgical bypass with endovascular recanalisation with stent

Recommendation 64	Class	Level	Refs.
Surgery or endovascular methods should be considered for treatment of late thrombosis of vascular accesses depending on the centre's expertise.	IIa	B	270,271
Recommendation 65			
Treatment of vascular access thrombosis should include peri-operative diagnosis and treatment of any associated stenosis.	I	C	548,552,553

grafts may also be an option. In addition, high flow AVFs with CVOD may also be treated by flow reducing procedures such as fistula vein banding.[579,580]

7.5. Central venous occlusive disease

CVOD is a common finding with an incidence of 2–40%.[151,514,555–557] It may be asymptomatic but can cause upper extremity, facial or breast swelling, increased venous outflow resistance, post-cannulation bleeding, AVF aneurysms, and may lead to VA loss, and preclude future VA creation in the ipsilateral limb.[151,555] These lesions are associated with prior CVC use, increased blood flow and extrinsic compression (see 7.5.1.).[151,557,558] Twelve to thirteen percent of patients with VA have symptomatic CVOD that may require some form of intervention and 25–50% of all subclavian CVCs are associated with subsequent CVOD, whereas lower rates have been reported for jugular vein catheters.[556,559,560] Clinical suspicion of the diagnosis should be confirmed by either fistulography or CTA. DUS is generally less useful since visualisation of central venous outflow may be difficult but can be of help using defined criteria.[561,562]

There is no ideal treatment for this problem. Withholding treatment in patients with no or minor symptoms can even show significantly better short and long-term central vein patency than treatment of symptomatic cases without detrimental effects on overall dialysis circulation.[563] Since surgery requires sufficient expertise and is associated with increased morbidity, PTA with its low morbidity and good short-term patency has become the accepted treatment for symptomatic CVOD.[476] Poor long-term patency rates after PTA are due to elastic recoil[564] or recurrent NIH and repeated interventions are often necessary.[565,566] According to most studies bare metal stents have not demonstrated an advantage in long-term patency over PTA and are not recommended in mobile

7.5.1. Haemodialysis associated venous thoracic outlet syndrome.

About 10% of central stenoses occur without previous CVC placement.[581] Extrinsic compression of the subclavian vein at the costoclavicular junction is a less common cause of venous hypertension or upper extremity swelling in the VA patient, but should be kept in mind, when no CVC has been used. The aetiology may be compression of the subclavian vein between the clavicle, first rib and costoclavicular ligament causing thickening of the vein wall, stenosis and thrombosis.[582] Lesions may be asymptomatic until placement of a VA, which leads to increased blood flow, arm swelling and/or cannulation problems.

The diagnosis is made by dynamic phlebography with abduction or elevation of the arm. DUS may detect subclavian vein compression before VA placement but the vein segment behind the clavicle is difficult to visualise.[561]

Stenoses with this aetiology respond poorly to PTA and stents invariably fail.[583] The treatment of choice is surgical decompression of the thoracic inlet by first rib resection and venolysis.[584] Residual stenosis may require PTA after decompression. Stent placement should be avoided. The largest series of patients treated this way, consisted of 12 patients, 8 of whom achieved patency beyond 8 months.[584] Occlusion of the subclavian vein usually requires other treatment strategies such as jugular vein turndown,[585] extra-anatomical bypass from the axillary vein to the internal jugular vein[586] or decompression followed by subclavian interposition graft.[584]

Recommendation 66	Class	Level	Refs.
After creation of a vascular access, evaluation of persistent arm oedema by fistulography or computed tomographic angiography is recommended to evaluate ipsilateral central venous outflow.	I	C	561,562
Recommendation 67			
Balloon angioplasty as primary treatment of symptomatic central venous outflow disease is recommended, with repeat interventions if indicated.	I	C	557,558,567, 568
Recommendation 68			
The use of stent grafts may be considered for the treatment of central vein stenosis.	IIb	C	569,571,572
Recommendation 69			
Stenting or repeat balloon angioplasty should be considered if there is significant elastic recoil of the central vein after balloon angioplasty or if the stenosis recurs within 3 months.	IIa	C	565,566

7.6. Vascular access induced limb ischaemia and high flow vascular access

VA induced limb ischaemia, often referred to as hand ischaemia or 'steal' after primary VA, occurs in 5—10% of cases when the brachial artery is used for inflow but in less than 1% of RCAVFs. Increase in age and diabetes in the HD population has raised the incidence of symptomatic peripheral ischaemia of the hand.[587] Other causes of VA associated complications in the forearm, hand and fingers such as carpal tunnel syndrome, venous hypertension and IMN should be considered when clinical symptoms of ischaemia are less pronounced.[588] Regular monitoring after VA placement is mandatory and high risk patients such as the elderly and diabetic patients should be evaluated carefully. Clinical examination should include pulse examination, auscultation for supraclavicular bruits indicating proximal arterial stenosis, bilateral blood pressure measurements, and evaluation of the hand circulation with and without temporary VA occlusion by digital compression.[587] The diagnosis can be confirmed by DUS evaluation of distal arm and hand arteries, finger blood pressure measurement or finger pulse oximetry, preferably with and without temporary VA occlusion. Surgical or endovascular procedures are performed on the basis of anatomical information provided by DSA or CTA.[587,588] The VA surgeon should readily evaluate patients with symptoms of VA induced ischaemia. Non-healing ulcers and emerging digital necrosis should lead to prompt intervention and if limb viability is threatened, VA ligation may be the only option. In cases with milder ischaemia, symptoms during exercise or HD or rest pain, the cause of ischaemia should be diagnosed and therapy aimed at reducing distal ischaemia with maintenance of VA function. Flow reducing arterial stenoses proximal to the anastomosis should be treated by PTA.[589] High flow induced steal with VA induced ischaemia requires reduction of outflow diameter to create a significant stenosis (80%) either through banding[590] or by a surgical revision to decrease anastomosis diameter or through the creation of a novel AV anastomosis in the forearm arteries as opposed to the brachial artery (revision using distal inflow; RUDI) (Fig. 7a,b).[591—594] The procedures should include intra-operative flow monitoring to ensure adequate flow reduction.[595] In RCAVFs with high flow, ligation of the proximal (or distal) limb of the artery, depending on the cause of the elevated flow may be successful (Fig. 7c).[184,185,592,596] VA induced ischaemia with normal or near normal VA flow and significant distal vascular disease represent the majority of cases.[263] Several reports support the use of a DRIL procedure.[594,597—601] More specifically, the AV anastomosis is bridged by a venous bypass and the artery ligated distal to the AV anastomosis (Fig. 7d).[602] The proximal bypass anastomosis should be placed at least 10 cm above the VA anastomosis to ensure adequate deviation of sufficient flow to the distal extremity. In RCAVFs with ischaemia, ligation of the distal limb of the radial artery may be an alternative (Fig. 7e). Intra-operative flow monitoring or DUS may be advisable to verify the increase in peripheral arterial perfusion.[595,598] Alternatively, improved distal perfusion may also be obtained by a more proximal AV anastomosis (PAVA) although only few studies have shown the effectiveness of this technique (Fig. 7f).[602,603] HD patients with VA flow $>$ 1500 ml/min should be monitored regularly by flow measurements, echocardiography and for clinical signs of congestive heart failure (CHF). Patients with progression of symptoms, progressive increase in VA flow or objective signs of heart failure should be considered for the surgical procedures described above.

Recommendation 70	Class	Level	Refs.
In patients with symptomatic vascular access induced extremity ischaemia with arterial inflow stenosis balloon angioplasty should be considered.	IIa	C	587,589

Recommendation 71	Class	Level	Refs.
Symptomatic access induced extremity ischaemia in patients with high flow access should be treated by surgical procedures aimed to reduce access flow.	I	C	591,592
Recommendation 72			
Distal revascularisation and interval ligation should be considered in patients with vascular access induced limb ischaemia and upper arm access without high flow.	IIa	C	597–602

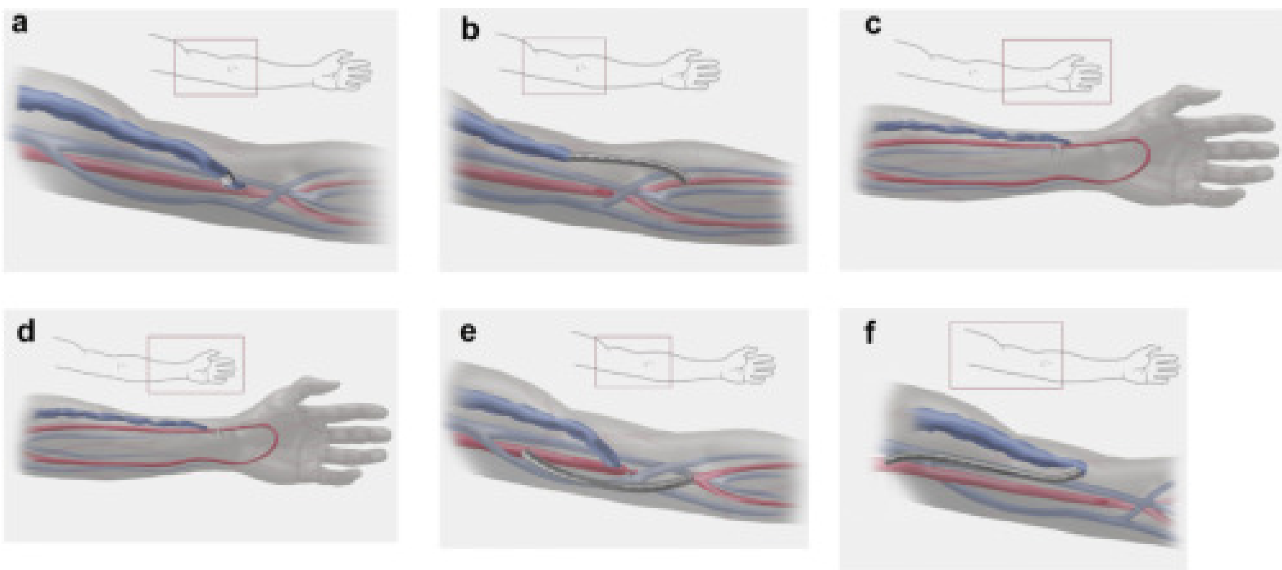


Figure 7. a. Flow reduction by banding of the vein close to the anastomosis. b. Flow reduction by creating inflow from a distal artery with smaller diameter (RUDI procedure). c. Flow reduction of a radiocephalic arteriovenous fistula by proximal ligation of the radial artery. d. Improvement of distal perfusion in a radiocephalic arteriovenous fistula by ligation of the distal radial artery. e. Improvement of distal perfusion by distal revascularisation interval ligation (DRIL) with a vein bypass and ligation of the brachial artery distally the arteriovenous anastomosis. f. Improvement of distal perfusion by creating a more proximal inflow of the arteriovenous fistula (Proximalisation of the arteriovenous anastomosis; PAVA).

7.7. Neuropathy

Distal nerve function can be acutely impaired after VA placement in the upper extremity using the brachial artery as inflow site. The most serious condition, IMN, is caused by axonal ischaemia in peripheral nerves that can lead to severe and non-reversible limb dysfunction.[588,604–606] Other causes are aggravation of pre-existing uraemic or diabetic neuropathy or nerve compression due to post-operative soft tissue oedema.[607] Prevalence and incidence numbers are unknown and case reports prevail. True ischaemic neuropathy can affect either nerve although the radial nerve seems most susceptible.[608] The underlying aetiology appears to be reduced collateral flow in vessels to major nerves in the antecubital fossa, most often after brachiocephalic AVFs, with subsequent ischaemic axonal or reversible demyelinating injuries.[605,609] Diagnosis of acute ischaemic neuropathy after VA creation is difficult. It should be suspected in patients with diabetes and pre-existing neuropathy, distal arterial disease and after creation of upper arm VA. The patient generally presents with immediate post-operative sensory or motor loss in the distribution of one or all of the three major peripheral nerves including motor function compromise causing wrist drop, sensory compromise with paresthesia and numbness or striking pain. Isolated nerve compromise should be suspected to be due to soft tissue nerve compression. The peripheral circulation is usually satisfactory with a warm hand and even with distal pulses. The condition may mimic true VA induced ischaemia, post-operative oedema or carpal tunnel syndrome.[588] It should be treated by immediate VA closure to prevent further neurological deficit.[605,610,611] Despite adequate actions, persistent neurological deficit and extremity malfunction is common.

Recommendation 73	Class	Level	Refs.
Post-operative monitoring for signs of ischaemic neuropathy is recommended in patients with diabetes or pre-existing neuropathy undergoing an upper arm vascular access procedure.	I	C	606,611

Recommendation 74	Class	Level	Refs.
Acute ischaemic neuropathy should be treated by immediate vascular access ligation to prevent further neurological deficit.	I	C	605,610,611

7.8. Non-used vascular access

There is neither consensus nor clinical evidence in favour of routine ligation of a functioning VA following successful kidney transplantation.[612] Reports indicate that most VAs remain patent after kidney transplantation.[613,614] VA closure may be indicated in high risk patients with pre-existing CHF, refractory CHF after transplantation, high flow VAs, other VA complications, and for cosmetic reasons.[612] VA ligation has been shown to improve cardiac function in kidney transplant recipients,[613] but, there are few studies reporting follow-up of cardiac function in transplant patients[612,614,615] and improvement of several physiological parameters have been observed both in patients with a patent VA as well as after VA closure.[614] Non-used AVGs may become infected over time, a possibility which must be considered in all patients with prior synthetic implants. In a series of 20 patients with non-used AVGs who presented with fever or sepsis positive blood cultures were present in 15 of 20 patients and all were positive on indium scans and had pus around the grafts.[137] Interestingly, in the same study 15 of 21 asymptomatic patients with abandoned, thrombosed ePTFE grafts had positive indium scans. Subsequent removal of the AVG in these patients revealed purulence surrounding the graft in 13 of 15 patients. Another study reported that of all graft infections at their centre, 23% were in thrombosed grafts.[256]

Recommendation 75	Class	Level	Ref.
Routine closure of a functioning vascular access after successful kidney transplantation is not recommended.	III	C	612
Recommendation 76			
Vascular access closure should be considered in patients with refractory heart failure after transplantation.	IIa	C	612–615

Recommendation 77	Class	Level	Ref.
When standard upper limb vascular access sites have been exhausted, complex access procedures should be considered according to the availability of suitable vessels.	IIa	C	

8. COMPLEX OR TERTIARY HAEMODIALYSIS VASCULAR ACCESS

There is a subgroup of challenging patients who will require complex tertiary VA. The expectations, age and comorbidities of the HD population are rising as well as the number of years for which people are being sustained on HD. There is also a group of younger patients who become increasingly sensitised with each failed transplant and thus more difficult to re-transplant from both an anatomical as well as an immunological perspective. The factors that result in repeated renal allograft failure are also those that challenge the VA surgeon. These factors include hypotension, thrombophilia and absence of vein in continuity with the right atrium. Achieving tertiary VA often requires the VA surgeon to be inventive, using their understanding of the general principles of fistula formation as well as vascular anatomy to create a VA that may be a unique "one off".

8.1. Tertiary vascular access

8.1.1. Suggested classification of types of tertiary vascular access surgery. The most appropriate tertiary VA procedure for an individual patient depends on the available vessels and the experience of the surgeon. These may be divided into three groups of increasing risk and complexity, which should therefore generally be considered in sequence:

Group one — upper limb, chest wall and translocated autogenous vein from the lower limb (see Chapter 5).

Group two — lower limb.

Group three — VA spanning the diaphragm, and other unusual VA procedures including upper and lower limb arterio-arterial loops.

8.1.1.1. Group one — upper limb, chest wall and translocated autogenous vein from the lower limb. Upper limb VA is preferred because of the increased morbidity when the lower limb is used. When a functioning upper limb VA is jeopardised by central venous stenosis or thrombosis venoplasty or recanalisation, stenting of a stenosed or occluded outflow vein should be attempted to treat arm swelling and preserve the VA. This includes sharp needle recanalisation of the outflow vein if experienced radiological support is available.[616] If recanalisation using endovascular techniques fails then the next option could be a bypass using a prosthetic conduit onto the ipsilateral axillary/subclavian[617] or jugular vein[618] via an infraclavicular or low neck incision respectively

(Fig. 8a). This chest wall surgery uses exposures identical to the ipsilateral axillary artery — vein loop[618,619] and ipsilateral axillary artery — jugular vein loop (Fig. 8b) as well as the crossover bypass necklace procedures[620,621] (Fig. 8c). Another option for patients with functioning upper limb VA compromised by major central stenoses is a prosthetic bypass from the axillary vein to the saphenous, femoral or iliac vein. The surgical exposures spanning the diaphragm are described in Section 8.1.1.3.[576,622] In another series of eight such cases,[577] the upper limb VA continues to be needled in the arm and the long chest wall conduit serves purely to decompress the arm.

In a report of 49 patients, with only one post-operative death and the remainder all continuing to use their upper limb VA, it was concluded that prosthetic veno-venous bypass is a robust solution for patients with occluded central veins.[623]

8.1.1.1.1. Great saphenous vein and femoral vein translocation. The GSV translocated to the upper arm is commonly believed to have high complication and poor maturation rates although acceptable results were reported in a recent small series.[220] Using FV as a conduit in the upper arm has good patency but suffers from a very high complication rate, specifically steal resulting from the calibre mismatch.[624]

8.1.1.1.2. Access to the right atrium. A recent innovation designed to minimally invasively access the right atrium is the HeRO® device. This provides a subcutaneous coupler somewhere in the axilla/upper arm or neck which is at the end of a 5 mm nitinol reinforced silicone catheter that traverses any central venous lesions before entering the right atrium. The coupler can then be attached to a 6 mm ePTFE graft which in turn may act as the

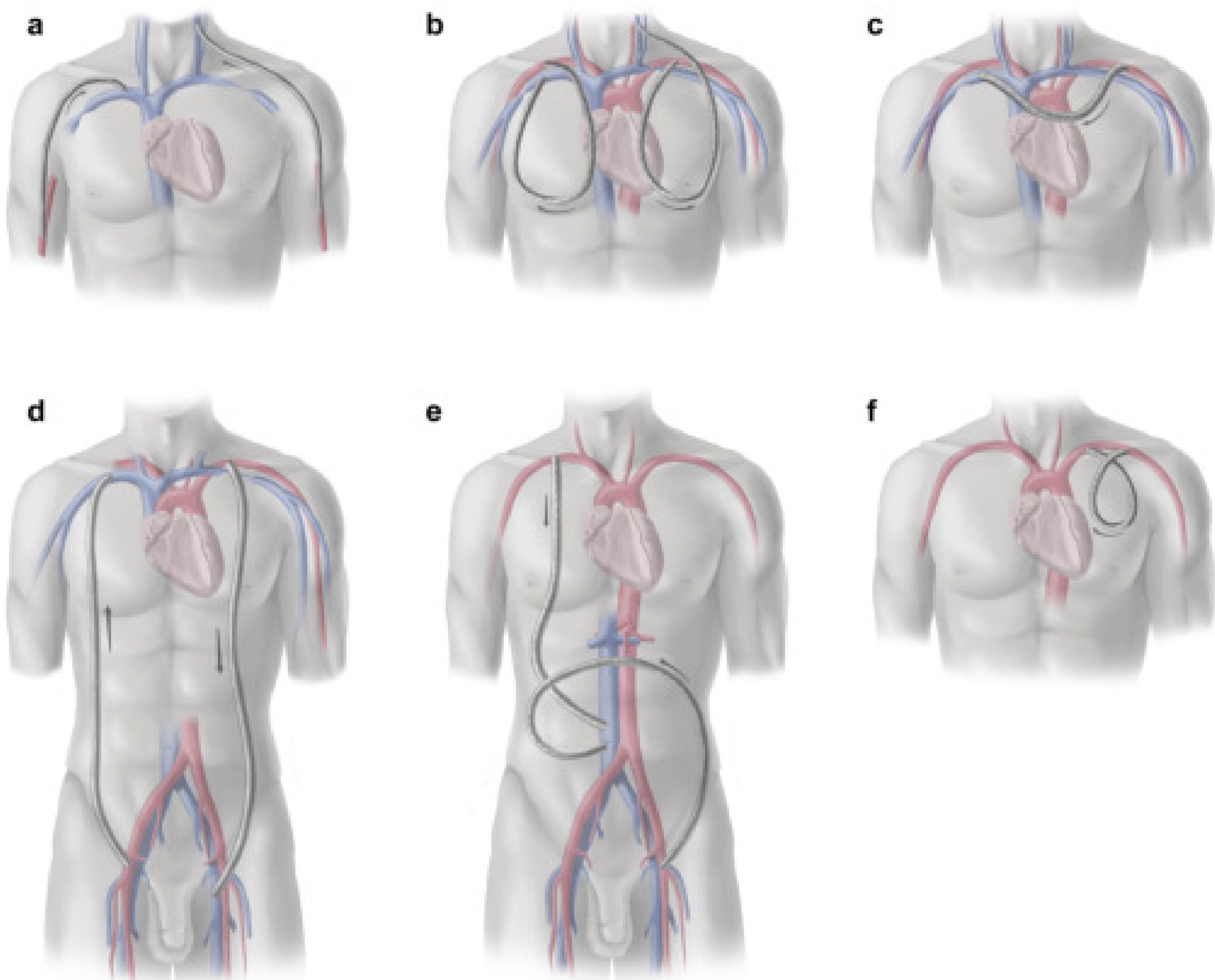


Figure 8. a. Right: VA prosthetic graft from the brachial artery to the ipsilateral subclavian vein via an infraclavicular incision. Left: VA prosthetic graft from the brachial artery to the ipsilateral internal jugular vein via a low neck incision. b. Right: VA prosthetic graft (loop configuration) from the ipsilateral axillary artery to the axillary vein. Left: VA prosthetic graft (loop configuration) from the ipsilateral axillary artery to the jugular vein. c. VA prosthetic graft (crossover configuration) from the axillary artery to the axillary or subclavian vein (necklace). d. Right: VA prosthetic graft from the common femoral artery to the ipsilateral axillary or subclavian vein. Left: VA prosthetic graft from the axillary artery to the ipsilateral femoral vein. e. Right: VA prosthetic graft from the axillary artery to the infrarenal vena cava. Left: VA prosthetic graft from the common femoral artery to the infrarenal vena cava. f. VA prosthetic graft (loop configuration) from the axillary artery to the ipsilateral subclavian artery.

AVG once anastomosed to an inflow or may simply be joined to an existing autogenous AVF in order to salvage or maintain it.

Published experience includes two multicentre studies of 164 and 409 cases respectively,[235,625] and a number of trials comparing the device with other tertiary VA procedures[626] and tcCVCs.[627] The 12 month primary and secondary patency rates were reported as 11% and 32% respectively.[628] A further series reported figures of 9.1% and 45.5%.[629] The HeRO device has also been used successfully to treat VA induced arm oedema.[630] In one study the average number of previous VA attempts prior to placement of a HeRO® catheter was as few as two, and in addition to poor patency rates there was a high complication rate with a particularly high incidence of steal syndrome (24%, all women).[628]

8.1.1.2. Group two — lower limb. Lower limb VA is associated with VAILI[101] and infection[219] reinforcing the importance of reconsidering suitability for peritoneal dialysis. This group comprises AVF formation using either the great saphenous vein, FV or AVG. Imaging of the lower limb arteries and veins including the ilio-caval veins is important when planning any lower limb VA as well as taking a full vascular history and measuring the ABI to avoid operating on a patient with peripheral arterial occlusive disease. Some authors have described lower limb VA being created preferentially as a result of patient choice. Reasons given include the facilitation of two-handed self cannulation, having both hands free during HD.[631,632] The increased risk of sepsis and limb threatening ischaemia does not support this practice.

8.1.1.2.1. Great saphenous vein. Once significant lower limb vascular disease has been excluded a few patients may be suitable for an autogenous posterior tibial to greater saphenous lower extremity AVF at the ankle although data are limited.[633]

Data for the saphenous vein thigh loop, which was first described in 1969 and where the GSV is anastomosed to the superficial femoral artery are also poor.[40,634,635] In a review 48 patients were reported with 56 saphenofemoral AVFs.[98] A loop configuration was avoided by anastomosing the GSV to the mid/lower SFA. The cumulative (i.e. secondary) patency was 65—70% at one year with 5 patients developing pseudoaneurysms.[636] In a small series of 8 patients with saphenous thigh loops the fistulas had poor flow and the complication rate was high with five haematomas, one thrombosis and two fatal haemorrhages.[636] These data suggest that the GSV performs poorly as an AVF in the lower limb.

The main choice to be made is between FVT and a lower extremity AVG (LEAVG) bridging the femoral vessels either at the groin or thigh level (see Chapter 5). Any prosthetic material placed into the groin carries a significant risk of infection with rates of between 18%[637] and 37.5%.[219] In a study 22 on LEAVGs in 21 patients were compared with 60 HeRO® devices in 59 patients.[626] This was an observational study with more obese patients receiving the HeRO® device. There were almost twice as many interventions required per annum to maintain HeRO® patency than lower extremity graft patency (2.21 vs. 1.17) with no differences in infection rate or mortality at 6 months. Obesity, however, was considered an indication for FVT[99] which suggests that a future study is warranted to compare the infective complications of FVT with the HeRO® device.

8.1.1.3. Group three — access spanning the diaphragm, other unusual access and prosthetic upper or lower limb arterio-arterial loops. This small group of patients is subjected to a very disparate and unusual range of operations for which no good evidence base exists. They will by definition be end stage VA patients.

8.1.1.3.1. Axillo-iliac, axillo-femoral and axillo-popliteal. Long grafts are described tunnelled subcutaneously from the axillary artery to the femoral or iliac vein or from the femoral artery to axillary/subclavian vein (Fig. 8d). When deciding which pelvic vessels to use, good quality paired arteries and veins should be preserved to retain technical feasibility for renal transplantation. Bypasses from the axillary artery to the IVC are described[623] and the authors have personal experience of creating a left iliac artery to IVC access (Fig. 8e). An axillary artery to popliteal vein prosthetic fistula is an example of a unique and rare VA tailored to a specific patient's available vessels.[620,638]

8.1.1.3.2. Arterio-arterial chest wall and lower limb loops. These fistulas warrant consideration for patients without easily accessible venous drainage to the right atrium, for patients with LEAD who would be at risk of steal and also because there is no increase in cardiac demand.

In a series of 34 prosthetic axillo-axillary loops placed in 32 patients (Fig 7f),[639] 11 patients were obese, as defined by a body mass index of >30 kg/m^2. The secondary patency rate was 59% at 1 year (median, 18 months) with a one year patient survival of 69%. Infection occurred in 15% of patients. The one year mortality of 30% demonstrates that this end stage VA group is highly morbid. In another report of 36 loop grafts placed in 34 patients of whom 5 had femoral arterio-arterial leg loops, follow-up was much longer.[640] Primary and secondary patency at one year was 73% and 96% and at 3 years 54% and 87%, respectively. Occlusion of the lower limb arterio-arterial shunt required immediate thrombectomy for limb salvage, whereas thrombosis of the upper limb VA did not result in limb threatening ischaemia.

There are a number of anecdotal VA cases that represent case reports, extreme examples of which include the femoro-renal AVG and AVGs sutured to the right atrium via a thoracotomy[641] or sternotomy. These types of procedure are final attempts to gain VA in patients who would otherwise perish. A high peri-operative mortality of this major surgery is therefore both expected and experienced.

8.2. Complex central venous catheters

Conventional tunnelled catheters are discussed in Chapter 3 (3.3.4). Despite the clear evidence that tcCVCs should be avoided by achieving a timely autogenous VA, there remain a significant number of patients who require placement of complex high risk salvage lines such as trans-lumbar, trans-hepatic[630,642] lines and lines through the parenchyma of a failed renal allograft or the native kidney to access the IVC.[643] The morbidity and mortality of complex line insertions and their short-term benefit would suggest that they should only be used after all other options, including complex grafts and PD have been ruled out. In this context, PD catheters which can be safely placed under local anaesthesia,[644–646] may still be possible after previous abdominal surgery[647] and can be used immediately for low volume exchange.[648]

Recommendation 78	Class	Level	Ref.
Individuals should not undergo the insertion of a high risk complex haemodialysis line without serious consideration of either the placement of a peritoneal dialysis catheter or a tertiary vascular access.	III	C	630,642,643, 645,647

9. GAPS IN THE EVIDENCE

Robust evidence is still needed in many aspects of the management of VA. Adequate trials are lacking. As a consequence most recommendations have been rated with a level of evidence B or C.

Future research directions could include:

- Trials on durability of prosthetic grafts and CVCs should be started.
- Trial on AV access versus CVC in the elderly. A trial has been launched on this subject in 2016.[649]
- A registry on post-VA creation ischaemic neuropathy would be of great value.
- Patient specific choices for VA should be investigated. Do patients with limited life expectancy benefit from an AVG more than an AVF?
- RCTs evaluating HD techniques should be undertaken: high vs. low flow dialysis, intensive HD, short and frequent.
- Should age trigger the dialysis access modality?
- Cannulation haemodynamics and damages to the VA through needling.
- Studies on best treatment for central venous obstruction disease.
- Organisation of early patient referral and of pre-dialysis care are major subjects for research. A policy of venous preservation should be taught and implemented.
- Trials on long-term follow-up and cost/benefit analysis for current available treatment techniques.
- Studies on tissue engineered grafts.
- Studies on new anastomotic technologies which need further investigation to assess their efficacy (laser, endovascular AVF construction, external vein support).[650–652]
- Studies on the effect of VA on the glomerular filtration rate in CKD stage 4–5 patients.
- Studies on the pathophysiology of intimal proliferation, haemodynamic, anatomical and flow considerations.

DISCLOSURES

The Authors disclose the following declaration of interest:

Jürg Schmidli	has royalties from Lemaitre
Matthias K. Widmer	has travel grants from Getinge
Carlo Basile	None
Gianmarco de Donato	None
Maurizio Gallieni	is Member of advisory board for Medtronic
Christopher P. Gibbons	None
Patrick Haage	None
George Hamilton	is Consultant for Evexar Ltd, UK
Ulf Hedin	has received consulting fees for lectures from W.L. Gore and Associates
Lars Kamper	None
Miltos K. Lazarides	None
Ben Lindsey	None
Gaspar Mestres	receives speaker's fees from W.L. Gore and Associates
Marisa Pegoraro	None
Joy Roy	None
Carlo Setacci	None
David Shemesh	has Consulting fees from W.L. Gore and Associates and Bard Peripheral Vascular
Jan H.M. Tordoir	None
Magda van Loon	None

ACKNOWLEDGEMENTS

Thomas R. Wyss, MD

For his contribution in preparing and editing this manuscript

APPENDIX A. SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.ejvs.2018.02.001.

REFERENCES

1 Field MJ, Lohr KN. *Clinical practice guidelines: directions for a new program. Committee to advise the Public-Health Service on clinical practice guidelines*. Institute of Medicine; 1990.

2 Field MJ, Lohr KN. *Guidelines for clinical practice: from development to use*. Institute of Medicine; 1992.

3 Dubois RW, Dean BB. Evolution of clinical practice guidelines: evidence supporting expanded use of medicines. *Dis Manag* 2006;**9**:210—23.

4 Sood R, Sood A, Ghosh AK. Non-evidence-based variables affecting physicians' test-ordering tendencies: a systematic review. *Neth J Med* 2007;**65**:167—77.

5 Manchanda P, Honka E. The effects and role of direct-to-physician marketing in the pharmaceutical industry: an integrative review. *Yale J Health Policy Law Ethics* 2005;**5**:785—822.

6 Win HK, Caldera AE, Maresh K, Lopez J, Rihal CS, Parikh MA, et al. Clinical outcomes and stent thrombosis following off-label use of drug-eluting stents. *JAMA* 2007;**297**:2001—9.

7 ESC Recommendations for Guidelines Production [20.09.2016]. www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Guidelines-development/Writing-ESC-Guidelines].

8 Gray RJ, Sacks D, Martin LG, Trerotola SO. Reporting standards for percutaneous interventions in dialysis access. Technology Assessment Committee. *J Vasc Intervent Radiol JVIR* 1999;**10**:1405—15.

9 Sidawy AN, Gray R, Besarab A, Henry M, Ascher E, Silva Jr M, et al. Recommended standards for reports dealing with arteriovenous hemodialysis accesses. *J Vasc Surg* 2002;**35**:603—10.

10 *NKF KDOQI Guidelines*. 2006. Update 2006. Retrieved May 30, 2014, from, http://wwwkidneyorg/professionals/KDOQI/guideline_upHD_PD_VA.

11 Lee T, Mokrzycki M, Moist L, Maya I, Vazquez M, Lok CE. Standardized definitions for hemodialysis vascular access. *Semin Dial* 2011;**24**:515—24.

12 K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Am J Kidney Dis* 2002;**39**:S1—266.

13 Coresh J, Astor BC, Greene T, Eknoyan G, Levey AS. Prevalence of chronic kidney disease and decreased kidney function in the adult US population: Third National Health and Nutrition Examination Survey. *Am J Kidney Dis* 2003;**41**:1—12.

14 Levey AS, de Jong PE, Coresh J, El Nahas M, Astor BC, Matsushita K, et al. The definition, classification, and prognosis of chronic kidney disease: a KDIGO Controversies Conference report. *Kidney Int* 2011;**80**:17—28.

15 Wetzels JF, Kiemeney LA, Swinkels DW, Willems HL, den Heijer M. Age- and gender-specific reference values of estimated GFR in Caucasians: the Nijmegen Biomedical Study. *Kidney Int* 2007;**72**:632—7.

16 Port FK. End-stage renal disease: magnitude of the problem, prognosis of future trends and possible solutions. *Kidney Int Suppl* 1995;**50**:S3—6.

17 Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012;**380**:2095—128.

18 Keith DS, Nichols GA, Gullion CM, Brown JB, Smith DH. Longitudinal follow-up and outcomes among a population with chronic kidney disease in a large managed care organization. *Arch Intern Med* 2004;**164**:659—63.

19 Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. *N Engl J Med* 2004;**351**:1296—305.

20 Sarnak MJ, Levey AS, Schoolwerth AC, Coresh J, Culleton B, Hamm LL, et al. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American Heart Association Councils on Kidney in Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. *Hypertension* 2003;**42**:1050—65.

21 Fox CS, Pencina MJ, Meigs JB, Vasan RS, Levitzky YS, D'Agostino Sr RB. Trends in the incidence of type 2 diabetes mellitus from the 1970s to the 1990s: the Framingham Heart Study. *Circulation* 2006;**113**:2914—8.

22 United States Renal Data System. *USRDS 2010 annual data report: Atlas of chronic kidney disease and end-stage renal disease in the United States*. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Disease; 2010.

23 Bottalico D, Port FK, Schena FP. Outcomes in dialysis: a global assessment. In: Hoerl WH, Koch KM, Lindsay RM, Ronco C, Winchester J, editors. *Replacement of renal function by dialysis*. 5th ed. Dordrecht: Kluwer Academic Publishers; 2004. p. 1411—53.

24 ERA-EDTA. *Registry: ERA-EDTA registry 2005 annual report*. Amsterdam, The Netherlands: Academic Medical Center, Department of Medical Informatics; 2007.

25 Canadian Organ Replacement Register Annual Report 2011. *Treatment of end-stage organ failure in Canada, 2000 to 2009*. Ottawa, Ont: Canadian Institute for Health Information; 2011.

26 Polkinghorne KR, Dent H, Gulyani A, Hurst K, McDonald SP. *Haemodialysis in ANZDATA registry report 2011*. Adelaide, South Australia: Australia and New Zealand Dialysis and Transplant Registry; 2011. p. 1—41.

27 Muntner P, Coresh J, Powe NR, Klag MJ. The contribution of increased diabetes prevalence and improved myocardial infarction and stroke survival to the increase in treated end-stage renal disease. *J Am Soc Nephrol* 2003;**14**:1568—77.

28 Port FK. The end-stage renal disease program: trends over the past 18 years. *Am J Kidney Dis* 1992;**20**:3—7.

29 Nugent RA, Fathima SF, Feigl AB, Chyung D. The burden of chronic kidney disease on developing nations: a 21st century challenge in global health. *Nephron Clin Pract* 2011;**118**:c269—77.

30 Castledine C, van Schalkwyk D, Feest T. UK renal registry 13th annual report (December 2010): chapter 7: the relationship between the type of vascular access used and survival in UK RRT patients in 2006. *Nephron Clin Pract* 2011;**119**:c135—40.

31 Gallieni M, Saxena R, Davidson I. Dialysis access in Europe and North America: are we on the same path? *Semin Intervent Radiol* 2009;**26**:96—105.

32 Arteriovenous fistula first website [20.09.2016]. Available from: www.fistulafirst.org.

33 United States renal data system annual data report; end-stage renal disease in the United States; chapter 4: vascular access 2015 [20.09.2016]. Available from: http://www.usrds.org/2015/view.
34 Ethier J, Mendelssohn DC, Elder SJ, Hasegawa T, Akizawa T, Akiba T, et al. Vascular access use and outcomes: an international perspective from the Dialysis Outcomes and Practice Patterns Study. *Nephrol Dial Transplant* 2008;**23**:3219—26.
35 Ravani P, Palmer SC, Oliver MJ, Quinn RR, MacRae JM, Tai DJ, et al. Associations between hemodialysis access type and clinical outcomes: a systematic review. *J Am Soc Nephrol* 2013;**24**:465—73.
36 Fissell RB, Fuller DS, Morgenstern H, Gillespie BW, Mendelssohn DC, Rayner HC, et al. Hemodialysis patient preference for type of vascular access: variation and predictors across countries in the DOPPS. *J Vasc Access* 2013;**14**:264—72.
37 McGill RL, Ruthazer R, Meyer KB, Miskulin DC, Weiner DE. Peripherally inserted central catheters and hemodialysis outcomes. *Clin J Am Soc Nephrol CJASN* 2016;**11**:1434—40.
38 Davidson I, Gallieni M, Saxena R, Dolmatch B. A patient centered decision making dialysis access algorithm. *J Vasc Access* 2007;**8**:59—68.
39 Jungers P, Massy ZA, Nguyen-Khoa T, Choukroun G, Robino C, Fakhouri F, et al. Longer duration of predialysis nephrological care is associated with improved long-term survival of dialysis patients. *Nephrol Dial Transplant* 2001;**16**:2357—64.
40 Murad MH, Elamin MB, Sidawy AN, Malaga G, Rizvi AZ, Flynn DN, et al. Autogenous versus prosthetic vascular access for hemodialysis: a systematic review and meta-analysis. *J Vasc Surg* 2008;**48**:34S—47S.
41 Almasri J, Alsawas M, Mainou M, Mustafa RA, Wang Z, Woo K, et al. Outcomes of vascular access for hemodialysis: A systematic review and meta-analysis. *J Vasc Surg* 2016;**64**:236—43.
42 Al-Jaishi AA, Liu AR, Lok CE, Zhang JC, Moist LM. Complications of the arteriovenous fistula: a systematic review. *J Am Soc Nephrol* 2017;**28**:1839—50.
43 Ng LJ, Chen F, Pisoni RL, Krishnan M, Mapes D, Keen M, et al. Hospitalization risks related to vascular access type among incident US hemodialysis patients. *Nephrol Dial Transplant* 2011;**26**:3659—66.
44 Gibbons CP. Primary vascular access. *Eur J Vascular Endovasc Surg* 2006;**31**:523—9.
45 Ravani P, Brunori G, Mandolfo S, Cancarini G, Imbasciati E, Marcelli D, et al. Cardiovascular comorbidity and late referral impact arteriovenous fistula survival: a prospective multicenter study. *J Am Soc Nephrol* 2004;**15**:204—9.
46 Avorn J, Winkelmayer WC, Bohn RL, Levin R, Glynn RJ, Levy E, et al. Delayed nephrologist referral and inadequate vascular access in patients with advanced chronic kidney failure. *J Clin Epidemiol* 2002;**55**:711—6.
47 Roubicek C, Brunet P, Huiart L, Thirion X, Leonetti F, Dussol B, et al. Timing of nephrology referral: influence on mortality and morbidity. *Am J Kidney Dis* 2000;**36**:35—41.
48 Tordoir J, Canaud B, Haage P, Konner K, Basci A, Fouque D, et al. EBPG on vascular access. *Nephrol Dial Transplant* 2007;**22**:ii88—117.
49 Sumida K, Molnar MZ, Potukuchi PK, Thomas F, Lu JL, Ravel VA, et al. Association between vascular access creation and deceleration of estimated glomerular filtration rate decline in late-stage chronic kidney disease patients transitioning to end-stage renal disease. *Nephrol Dial Transplant* 2017;**32**:1330—7.
50 Hasegawa T, Bragg-Gresham JL, Yamazaki S, Fukuhara S, Akizawa T, Kleophas W, et al. Greater first-year survival on hemodialysis in facilities in which patients are provided earlier and more frequent pre-nephrology visits. *Clin J Am Soc Nephrol CJASN* 2009;**4**:595—602.
51 de Jager DJ, Voormolen N, Krediet RT, Dekker FW, Boeschoten EW, Grootendorst DC. Association between time of referral and survival in the first year of dialysis in diabetics and the elderly. *Nephrol Dial Transplant* 2011;**26**:652—8.
52 Prischl FC, Kirchgatterer A, Brandstatter E, Wallner M, Baldinger C, Roithinger FX, et al. Parameters of prognostic relevance to the patency of vascular access in hemodialysis patients. *J Am Soc Nephrol* 1995;**6**:1613—8.
53 Saran R, Elder SJ, Goodkin DA, Akiba T, Ethier J, Rayner HC, et al. Enhanced training in vascular access creation predicts arteriovenous fistula placement and patency in hemodialysis patients: results from the Dialysis Outcomes and Practice Patterns Study. *Ann Surg* 2008;**247**:885—91.
54 Huijbregts HJ, Bots ML, Moll FL, Blankestijn PJ. Hospital specific aspects predominantly determine primary failure of hemodialysis arteriovenous fistulas. *J Vasc Surg* 2007;**45**:962—7.
55 Rooijens PP, Tordoir JH, Stijnen T, Burgmans JP, Smet de AA, Yo TI. Radiocephalic wrist arteriovenous fistula for hemodialysis: meta-analysis indicates a high primary failure rate. *Eur J Vasc Endovasc Surg* 2004;**28**:583—9.
56 Huijbregts HJ, Bots ML, Wittens CH, Schrama YC, Moll FL, Blankestijn PJ. Hemodialysis arteriovenous fistula patency revisited: results of a prospective, multicenter initiative. *Clin J Am Soc Nephrol CJASN* 2008;**3**:714—9.
57 Gracz KC, Ing TS, Soung LS, Armbruster KF, Seim SK, Merkel FK. Proximal forearm fistula for maintenance hemodialysis. *Kidney Int* 1977;**11**:71—5.
58 van Hoek F, Scheltinga MR, Kouwenberg I, Moret KE, Beerenhout CH, Tordoir JH. Steal in hemodialysis patients depends on type of vascular access. *Eur J Vasc Endovasc Surg* 2006;**32**:710—7.
59 Silva Jr MB, Hobson 2nd RW, Pappas PJ, Jamil Z, Araki CT, Goldberg MC, et al. A strategy for increasing use of autogenous hemodialysis access procedures: impact of preoperative noninvasive evaluation. *J Vasc Surg* 1998;**27**:302—7. discussion 7—8.
60 Golledge J, Smith CJ, Emery J, Farrington K, Thompson HH. Outcome of primary radiocephalic fistula for haemodialysis. *Br J Surg* 1999;**86**:211—6.
61 Wolowczyk L, Williams AJ, Donovan KL, Gibbons CP. The snuffbox arteriovenous fistula for vascular access. *Eur J Vasc Endovasc Surg* 2000;**19**:70—6.
62 Gibson KD, Gillen DL, Caps MT, Kohler TR, Sherrard DJ, Stehman-Breen CO. Vascular access survival and incidence of revisions: a comparison of prosthetic grafts, simple autogenous fistulas, and venous transposition fistulas from the United States Renal Data System Dialysis Morbidity and Mortality Study. *J Vasc Surg* 2001;**34**:694—700.
63 Allon M, Lockhart ME, Lilly RZ, Gallichio MH, Young CJ, Barker J, et al. Effect of preoperative sonographic mapping on vascular access outcomes in hemodialysis patients. *Kidney Int* 2001;**60**:2013—20.
64 Dixon BS, Novak L, Fangman J. Hemodialysis vascular access survival: upper-arm native arteriovenous fistula. *Am J Kidney Dis* 2002;**39**:92—101.
65 Ravani P, Marcelli D, Malberti F. Vascular access surgery managed by renal physicians: the choice of native arteriovenous fistulas for hemodialysis. *Am J Kidney Dis* 2002;**40**:1264—76.

66 Rooijens PP, Burgmans JP, Yo TI, Hop WC, de Smet AA, van den Dorpel MA, et al. Autogenous radial-cephalic or prosthetic brachial-antecubital forearm loop AVF in patients with compromised vessels? A randomized, multicenter study of the patency of primary hemodialysis access. *J Vasc Surg* 2005;**42**:481—6. discussions 7.

67 Biuckians A, Scott EC, Meier GH, Panneton JM, Glickman MH. The natural history of autologous fistulas as first-time dialysis access in the KDOQI era. *J Vasc Surg* 2008;**47**:415—21. discussion 20—21.

68 Murphy GJ, Saunders R, Metcalfe M, Nicholson ML. Elbow fistulas using autogeneous vein: patency rates and results of revision. *Postgrad Med J* 2002;**78**:483—6.

69 Zeebregts CJ, Tielliu IF, Hulsebos RG, de Bruin C, Verhoeven EL, Huisman RM, et al. Determinants of failure of brachiocephalic elbow fistulas for haemodialysis. *Eur J Vasc Endovasc Surg* 2005;**30**:209—14.

70 Lok CE, Oliver MJ, Su J, Bhola C, Hannigan N, Jassal SV. Arteriovenous fistula outcomes in the era of the elderly dialysis population. *Kidney Int* 2005;**67**:2462—9.

71 Woo K, Farber A, Doros G, Killeen K, Kohanzadeh S. Evaluation of the efficacy of the transposed upper arm arteriovenous fistula: a single institutional review of 190 basilic and cephalic vein transposition procedures. *J Vasc Surg* 2007;**46**:94—9. discussion 100.

72 Koksoy C, Demirci RK, Balci D, Solak T, Kose SK. Brachiobasilic versus brachiocephalic arteriovenous fistula: a prospective randomized study. *J Vasc Surg* 2009;**49**:171—7. e5.

73 Palmes D, Kebschull L, Schaefer RM, Pelster F, Konner K. Perforating vein fistula is superior to forearm fistula in elderly haemodialysis patients with diabetes and arterial hypertension. *Nephrol Dial Transplant* 2011;**26**:3309—14.

74 Ayez N, van Houten VA, de Smet AA, van Well AM, Akkersdijk GP, van de Ven PJ, et al. The basilic vein and the cephalic vein perform equally in upper arm arteriovenous fistulae. *Eur J Vasc Endovasc Surg* 2012;**44**:227—31.

75 Segal JH, Kayler LK, Henke P, Merion RM, Leavey S, Campbell Jr DA. Vascular access outcomes using the transposed basilic vein arteriovenous fistula. *Am J Kidney Dis* 2003;**42**:151—7.

76 Wolford HY, Hsu J, Rhodes JM, Shortell CK, Davies MG, Bakhru A, et al. Outcome after autogenous brachial-basilic upper arm transpositions in the post-National Kidney Foundation Dialysis Outcomes Quality Initiative era. *J Vasc Surg* 2005;**42**:951—6.

77 Arroyo MR, Sideman MJ, Spergel L, Jennings WC. Primary and staged transposition arteriovenous fistulas. *J Vasc Surg* 2008;**47**:1279—83.

78 Keuter XH, De Smet AA, Kessels AG, van der Sande FM, Welten RJ, Tordoir JH. A randomized multicenter study of the outcome of brachial-basilic arteriovenous fistula and prosthetic brachial-antecubital forearm loop as vascular access for hemodialysis. *J Vasc Surg* 2008;**47**:395—401.

79 Field M, Van Dellen D, Mak D, Winter H, Hamsho A, Mellor S, et al. The brachiobasilic arteriovenous fistula: effect of patient variables. *J Vasc Access* 2011;**12**:325—30.

80 Lin SL, Huang CH, Chen HS, Hsu WA, Yen CJ, Yen TS. Effects of age and diabetes on blood flow rate and primary outcome of newly created hemodialysis arteriovenous fistulas. *Am J Nephrol* 1998;**18**:96—100.

81 Johansson E, Hammarskjold F, Lundberg D, Arnlind MH. Advantages and disadvantages of peripherally inserted central venous catheters (PICC) compared to other central venous lines: a systematic review of the literature. *Acta Oncol* 2013;**52**:886—92.

82 Lomonte C, Basile C. On the phenomenology of the perforating vein of the elbow. *Semin Dial* 2009;**22**:300—3.

83 Enzler MA, Rajmon T, Lachat M, Largiader F. Long-term function of vascular access for hemodialysis. *Clin Transplant* 1996;**10**:511—5.

84 Fisher CM, Neale ML. Outcome of surgery for vascular access in patients commencing haemodialysis. *Eur J Vasc Endovasc Surg* 2003;**25**:342—9.

85 Hirth RA, Turenne MN, Woods JD, Young EW, Port FK, Pauly MV, et al. Predictors of type of vascular access in hemodialysis patients. *JAMA* 1996;**276**:1303—8.

86 Kalman PG, Pope M, Bhola C, Richardson R, Sniderman KW. A practical approach to vascular access for hemodialysis and predictors of success. *J Vasc Surg* 1999;**30**:727—33.

87 Polkinghorne KR, McDonald SP, Marshall MR, Atkins RC, Kerr PG. Vascular access practice patterns in the New Zealand hemodialysis population. *Am J Kidney Dis* 2004;**43**:696—704.

88 Rodriguez JA, Lopez J, Cleries M, Vela E. Vascular access for haemodialysis—an epidemiological study of the Catalan Renal Registry. *Nephrol Dial Transplant* 1999;**14**:1651—7.

89 Caplin N, Sedlacek M, Teodorescu V, Falk A, Uribarri J. Venous access: women are equal. *Am J Kidney Dis* 2003;**41**:429—32.

90 Allon M, Ornt DB, Schwab SJ, Rasmussen C, Delmez JA, Greene T, et al. Factors associated with the prevalence of arteriovenous fistulas in hemodialysis patients in the HEMO study. Hemodialysis (HEMO) Study Group. *Kidney Int* 2000;**58**:2178—85.

91 Saran R, Dykstra DM, Wolfe RA, Gillespie B, Held PJ, Young EW. Association between vascular access failure and the use of specific drugs: the Dialysis Outcomes and Practice Patterns Study (DOPPS). *Am J Kidney Dis* 2002;**40**:1255—63.

92 Garcia-Pajares R, Polo JR, Flores A, Gonzalez-Tabares E, Solis JV. Upper arm polytetrafluoroethylene grafts for dialysis access. Analysis of two different graft sizes: 6 mm and 6—8 mm. *Vasc Endovasc Surg* 2003;**37**:335—43.

93 Lenz BJ, Veldenz HC, Dennis JW, Khansarinia S, Atteberry LR. A three-year follow-up on standard versus thin wall ePTFE grafts for hemodialysis. *J Vasc Surg* 1998;**28**:464—70. discussion 70.

94 Tordoir JH, Hofstra L, Leunissen KM, Kitslaar PJ. Early experience with stretch polytetrafluoroethylene grafts for haemodialysis access surgery: results of a prospective randomised study. *Eur J Vasc Endovasc Surg* 1995;**9**:305—9.

95 Barron PT, Wellington JL, Lorimer JW, Cole CW, Moher D. A comparison between expanded polytetrafluoroethylene and plasma tetrafluoroethylene grafts for hemodialysis access. *Can J Surg J Can Chir* 1993;**36**:184—6.

96 Kaufman JL, Garb JL, Berman JA, Rhee SW, Norris MA, Friedmann P. A prospective comparison of two expanded polytetrafluoroethylene grafts for linear forearm hemodialysis access: does the manufacturer matter? *J Am Coll Surg* 1997;**185**:74—9.

97 Lazarides MK, Georgiadis GS, Antoniou GA, Staramos DN. A meta-analysis of dialysis access outcome in elderly patients. *J Vasc Surg* 2007;**45**:420—6.

98 Correa JA, de Abreu LC, Pires AC, Breda JR, Yamazaki YR, Fioretti AC, et al. Saphenofemoral arteriovenous fistula as hemodialysis access. *BMC Surg* 2010;**10**:28.

99 Gradman WS, Laub J, Cohen W. Femoral vein transposition for arteriovenous hemodialysis access: improved patient selection

and intraoperative measures reduce postoperative ischemia. *J Vasc Surg* 2005;**41**:279–84.
100 Geenen IL, Nyilas L, Stephen MS, Makeham V, White GH, Verran DJ. Prosthetic lower extremity hemodialysis access grafts have satisfactory patency despite a high incidence of infection. *J Vasc Surg* 2010;**52**:1546–50.
101 Antoniou GA, Lazarides MK, Georgiadis GS, Sfyroeras GS, Nikolopoulos ES, Giannoukas AD. Lower-extremity arteriovenous access for haemodialysis: a systematic review. *Eur J Vasc Endovasc Surg* 2009;**38**:365–72.
102 Bourquelot P, Rawa M, Van Laere O, Franco G. Long-term results of femoral vein transposition for autogenous arteriovenous hemodialysis access. *J Vasc Surg* 2012;**56**:440–5.
103 Polkinghorne KR, McDonald SP, Atkins RC, Kerr PG. Vascular access and all-cause mortality: a propensity score analysis. *J Am Soc Nephrol* 2004;**15**:477–86.
104 Perl J, Wald R, McFarlane P, Bargman JM, Vonesh E, Na Y, et al. Hemodialysis vascular access modifies the association between dialysis modality and survival. *J Am Soc Nephrol* 2011;**22**:1113–21.
105 Ortega T, Ortega F, Diaz-Corte C, Rebollo P, Ma Baltar J, Alvarez-Grande J. The timely construction of arteriovenous fistulae: a key to reducing morbidity and mortality and to improving cost management. *Nephrol Dial Transplant* 2005;**20**:598–603.
106 Ferring M, Claridge M, Smith SA, Wilmink T. Routine preoperative vascular ultrasound improves patency and use of arteriovenous fistulas for hemodialysis: a randomized trial. *Clin J Am Soc Nephrol CJASN* 2010;**5**:2236–44.
107 Mihmanli I, Besirli K, Kurugoglu S, Atakir K, Haider S, Ogut G, et al. Cephalic vein and hemodialysis fistula: surgeon's observation versus color Doppler ultrasonographic findings. *J Ultrasound Med* 2001;**20**:217–22.
108 Vassalotti JA, Falk A, Cohl ED, Uribarri J, Teodorescu V. Obese and non-obese hemodialysis patients have a similar prevalence of functioning arteriovenous fistula using pre-operative vein mapping. *Clin Nephrol* 2002;**58**:211–4.
109 Georgiadis GS, Charalampidis DG, Argyriou C, Georgakarakos EI, Lazarides MK. The necessity for routine preoperative ultrasound mapping before arteriovenous fistula creation: a meta-analysis. *Eur J Vasc Endovasc Surg* 2015;**49**:600–5.
110 Brown PW. Preoperative radiological assessment for vascular access. *Eur J Vasc Endovasc Surg* 2006;**31**:64–9.
111 Goldstein LJ, Gupta S. Use of the radial artery for hemodialysis access. *Arch Surg* 2003;**138**:1130–4.
112 Malovrh M. Native arteriovenous fistula: preoperative evaluation. *Am J Kidney Dis* 2002;**39**:1218–25.
113 Asif A, Cherla G, Merrill D, Cipleu CD, Tawakol JB, Epstein DL, et al. Venous mapping using venography and the risk of radiocontrast-induced nephropathy. *Semin Dial* 2005;**18**:239–42.
114 Laissy JP, Fernandez P, Karila-Cohen P, Delmas V, Dupuy E, Chillon S, et al. Upper limb vein anatomy before hemodialysis fistula creation: cross-sectional anatomy using MR venography. *Eur Radiol* 2003;**13**:256–61.
115 Planken NR, Tordoir JH, Duijm LE, van den Bosch HC, van der Sande FM, Kooman JP, et al. Magnetic resonance angiographic assessment of upper extremity vessels prior to vascular access surgery: feasibility and accuracy. *Eur Radiol* 2008;**18**:158–67.
116 Fraum TJ, Ludwig DR, Bashir MR, Fowler KJ. Gadolinium-based contrast agents: A comprehensive risk assessment. *J Magn Reson Imaging* 2017;**46**:338–53.
117 Grobner T. Gadolinium—a specific trigger for the development of nephrogenic fibrosing dermopathy and nephrogenic systemic fibrosis? *Nephrol Dial Transplant* 2006;**21**:1104–8.
118 Bode AS, Planken RN, Merkx MA, van der Sande FM, Geerts L, Tordoir JH, et al. Feasibility of non-contrast-enhanced magnetic resonance angiography for imaging upper extremity vasculature prior to vascular access creation. *Eur J Vasc Endovasc Surg* 2012;**43**:88–94.
119 de Graaf R, van Laanen J, Peppelenbosch N, van Loon M, Tordoir J. The value of intravascular ultrasound in the treatment of central venous obstructions in hemodialysis patients. *J Vasc Access* 2016;**17**:S12–5.
120 Doelman C, Duijm LE, Liem YS, Froger CL, Tielbeek AV, Donkers-van Rossum AB, et al. Stenosis detection in failing hemodialysis access fistulas and grafts: comparison of color Doppler ultrasonography, contrast-enhanced magnetic resonance angiography, and digital subtraction angiography. *J Vasc Surg* 2005;**42**:739–46.
121 Tattersall J, Martin-Malo A, Pedrini L, Basci A, Canaud B, Fouque D, et al. EBPG guideline on dialysis strategies. *Nephrol Dial Transplant* 2007;**22**:ii5–21.
122 Bay WH, Henry ML, Lazarus JM, Lew NL, Ling J, Lowrie EG. Predicting hemodialysis access failure with color flow Doppler ultrasound. *Am J Nephrol* 1998;**18**:296–304.
123 Schwarz C, Mitterbauer C, Boczula M, Maca T, Funovics M, Heinze G, et al. Flow monitoring: performance characteristics of ultrasound dilution versus color Doppler ultrasound compared with fistulography. *Am J Kidney Dis* 2003;**42**:539–45.
124 Wiese P, Nonnast-Daniel B. Colour Doppler ultrasound in dialysis access. *Nephrol Dial Transplant* 2004;**19**:1956–63.
125 Dumars MC, Thompson WE, Bluth EI, Lindberg JS, Yoselevitz M, Merritt CR. Management of suspected hemodialysis graft dysfunction: usefulness of diagnostic US. *Radiology* 2002;**222**:103–7.
126 Gadallah MF, Paulson WD, Vickers B, Work J. Accuracy of Doppler ultrasound in diagnosing anatomic stenosis of hemodialysis arteriovenous access as compared with fistulography. *Am J Kidney Dis* 1998;**32**:273–7.
127 Older RA, Gizienski TA, Wilkowski MJ, Angle JF, Cote DA. Hemodialysis access stenosis: early detection with color Doppler US. *Radiology* 1998;**207**:161–4.
128 Shackleton CR, Taylor DC, Buckley AR, Rowley VA, Cooperberg PL, Fry PD. Predicting failure in polytetrafluoroethylene vascular access grafts for hemodialysis: a pilot study. *Can J Surg J Can Chir* 1987;**30**:442–4.
129 Tordoir JH, de Bruin HG, Hoeneveld H, Eikelboom BC, Kitslaar PJ. Duplex ultrasound scanning in the assessment of arteriovenous fistulas created for hemodialysis access: comparison with digital subtraction angiography. *J Vasc Surg* 1989;**10**:122–8.
130 Tordoir JH, Hoeneveld H, Eikelboom BC, Kitslaar PJ. The correlation between clinical and duplex ultrasound parameters and the development of complications in arterio-venous fistulae for haemodialysis. *Eur J Vasc Surg* 1990;**4**:179–84.
131 Lumsden AB, MacDonald MJ, Kikeri D, Cotsonis GA, Harker LA, Martin LG. Cost efficacy of duplex surveillance and prophylactic angioplasty of arteriovenous ePTFE grafts. *Ann Vasc Surg* 1998;**12**:138–42.
132 Robbin ML, Chamberlain NE, Lockhart ME, Gallichio MH, Young CJ, Deierhoi MH, et al. Hemodialysis arteriovenous fistula maturity: US evaluation. *Radiology* 2002;**225**:59–64.
133 Thalhammer C, Aschwanden M, Staub D, Dickenmann M, Jaeger KA. Duplex sonography of hemodialysis access. *Ultraschall Med* 2007;**28**:450–65. quiz 66–71.

134 Malik J, Slavikova M, Svobodova J, Tuka V. Regular ultrasonographic screening significantly prolongs patency of PTFE grafts. *Kidney Int* 2005;**67**:1554—8.

135 Ascher E, Hingorani A, Marks N. Duplex-guided balloon angioplasty of failing or nonmaturing arterio-venous fistulae for hemodialysis: a new office-based procedure. *J Vasc Surg* 2009;**50**:594—9.

136 Gorin DR, Perrino L, Potter DM, Ali TZ. Ultrasound-guided angioplasty of autogenous arteriovenous fistulas in the office setting. *J Vasc Surg* 2012;**55**:1701—5.

137 Nassar GM, Ayus JC. Infectious complications of the hemodialysis access. *Kidney Int* 2001;**60**:1—13.

138 Ye C, Mao Z, Rong S, Zhang Y, Mei C, Li H, et al. Multislice computed tomographic angiography in evaluating dysfunction of the vascular access in hemodialysis patients. *Nephron Clin Pract* 2006;**104**:c94—100.

139 Heye S, Maleux G, Claes K, Kuypers D, Oyen R. Stenosis detection in native hemodialysis fistulas with MDCT angiography. *AJR Am J Roentgenol* 2009;**192**:1079—84.

140 Wasinrat J, Siriapisith T, Thamtorawat S, Tongdee T. 64-slice MDCT angiography of upper extremity in assessment of native hemodialysis access. *Vasc Endovasc Surg* 2011;**45**:69—77.

141 Dimopoulou A, Raland H, Wikstrom B, Magnusson A. MDCT angiography with 3D image reconstructions in the evaluation of failing arteriovenous fistulas and grafts in hemodialysis patients. *Acta Radiol* 2011;**52**:935—42.

142 Rooijens PP, Serafino GP, Vroegindeweij D, Dammers R, Yo TI, De Smet AA, et al. Multi-slice computed tomographic angiography for stenosis detection in forearm hemodialysis arteriovenous fistulas. *J Vasc Access* 2008;**9**:278—84.

143 Karadeli E, Tarhan NC, Ulu EM, Tutar NU, Basaran O, Coskun M, et al. Evaluation of failing hemodialysis fistulas with multidetector CT angiography: comparison of different 3D planes. *Eur J Radiol* 2009;**69**:184—92.

144 Paksoy Y, Gormus N, Tercan MA. Three-dimensional contrast-enhanced magnetic resonance angiography (3-D CE-MRA) in the evaluation of hemodialysis access complications, and the condition of central veins in patients who are candidates for hemodialysis access. *J Nephrol* 2004;**17**:57—65.

145 Froger CL, Duijm LE, Liem YS, Tielbeek AV, Donkers-van Rossum AB, Douwes-Draaijer P, et al. Stenosis detection with MR angiography and digital subtraction angiography in dysfunctional hemodialysis access fistulas and grafts. *Radiology* 2005;**234**:284—91.

146 Han KM, Duijm LE, Thelissen GR, Cuypers PW, Douwes-Draaijer P, Tielbeek AV, et al. Failing hemodialysis access grafts: evaluation of complete vascular tree with 3D contrast-enhanced MR angiography with high spatial resolution: initial results in 10 patients. *Radiology* 2003;**227**:601—5.

147 Bakker CJ, Peeters JM, Bartels LW, Elgersma OE, Zijlstra JJ, Blankestijn PJ, et al. Magnetic resonance techniques in hemodialysis access management. *J Vasc Access* 2003;**4**:125—39.

148 Menegazzo D, Laissy JP, Durrbach A, Debray MP, Messin B, Delmas V, et al. Hemodialysis access fistula creation: preoperative assessment with MR venography and comparison with conventional venography. *Radiology* 1998;**209**:723—8.

149 Planken RN, Tordoir JH, Dammers R, de Haan MW, Oei TK, van der Sande FM, et al. Stenosis detection in forearm hemodialysis arteriovenous fistulae by multiphase contrast-enhanced magnetic resonance angiography: preliminary experience. *J Magn Reson Imaging* 2003;**17**:54—64.

150 Bode A, Caroli A, Huberts W, Planken N, Antiga L, Bosboom M, et al. Clinical study protocol for the ARCH project — computational modeling for improvement of outcome after vascular access creation. *J Vasc Access* 2011;**12**:369—76.

151 Mansour M, Kamper L, Altenburg A, Haage P. Radiological central vein treatment in vascular access. *J Vasc Access* 2008;**9**:85—101.

152 Haage P, Vorwerk D, Piroth W, Schuermann K, Guenther RW. Treatment of hemodialysis-related central venous stenosis or occlusion: results of primary Wallstent placement and follow-up in 50 patients. *Radiology* 1999;**212**:175—80.

153 Heye S, Maleux G, Marchal GJ. Upper-extremity venography: CO_2 versus iodinated contrast material. *Radiology* 2006;**241**:291—7.

154 Le Blanche AF, Tassart M, Deux JF, Rossert J, Bigot JM, Boudghene F. Gadolinium-enhanced digital subtraction angiography of hemodialysis fistulas: a diagnostic and therapeutic approach. *AJR Am J Roentgenol* 2002;**179**:1023—8.

155 NKF KDOQI clinical practice guidelines and clinical practice Recommendations for 2006 updates: hemodialysis adequacy, peritoneal dialysis adequacy and vascular access. *Am J Kidney Dis* 2006;**48**:S1—322.

156 Kumar S, Seward J, Wilcox A, Torella F. Influence of muscle training on resting blood flow and forearm vessel diameter in patients with chronic renal failure. *Br J Surg* 2010;**97**:835—8.

157 Salimi F, Majd Nassiri G, Moradi M, Keshavarzian A, Farajzadegan Z, Saleki M, et al. Assessment of effects of upper extremity exercise with arm tourniquet on maturity of arteriovenous fistula in hemodialysis patients. *J Vasc Access* 2013;**14**:239—44.

158 Malovrh M. Expansion of blood volume increases the primary patency rate of arteriovenous fistulas for hemodialysis in patients with critical arterial quality. *Ther Apher Dial* 2009;**13**:345—9.

159 Stewart AH, Eyers PS, Earnshaw JJ. Prevention of infection in peripheral arterial reconstruction: a systematic review and meta-analysis. *J Vasc Surg* 2007;**46**:148—55.

160 Bennion RS, Hiatt JR, Williams RA, Wilson SE. A randomized, prospective study of perioperative antimicrobial prophylaxis for vascular access surgery. *J Cardiovasc Surg* 1985;**26**:270—4.

161 Zibari GB, Gadallah MF, Landreneau M, McMillan R, Bridges RM, Costley K, et al. Preoperative vancomycin prophylaxis decreases incidence of postoperative hemodialysis vascular access infections. *Am J Kidney Dis* 1997;**30**:343—8.

162 Osborn G, Escofet X, Da Silva A. Medical adjuvant treatment to increase patency of arteriovenous fistulae and grafts. *Cochrane Database Syst Rev* 2008;**4**:CD002786.

163 Coleman CI, Tuttle LA, Teevan C, Baker WL, White CM, Reinhart KM. Antiplatelet agents for the prevention of arteriovenous fistula and graft thrombosis: a meta analysis. *Int J Clin Pract* 2010;**64**:1239—44.

164 Palmer SC, Di Micco L, Razavian M, Craig JC, Ravani P, Perkovic V, et al. Antiplatelet therapy to prevent hemodialysis vascular access failure: systematic review and meta-analysis. *Am J Kidney Dis* 2013;**61**:112—22.

165 Andrassy K, Malluche H, Bornefeld H, Comberg M, Ritz E, Jesdinsky H, et al. Prevention of p.o. clotting of av. cimino fistulae with acetylsalicyl acid. Results of a prospective double blind study. *Klin Wochenschr* 1974;**52**:348—9.

166 Ghorbani A, Aalamshah M, Shahbazian H, Ehsanpour A, Aref A. Randomized controlled trial of clopidogrel to prevent primary arteriovenous fistula failure in hemodialysis patients. *Indian J Nephrol* 2009;**19**:57—61.

167 Grontoft KC, Larsson R, Mulec H, Weiss LG, Dickinson JP. Effects of ticlopidine in AV-fistula surgery in uremia. Fistula Study Group. *Scand J Urol Nephrol* 1998;**32**:276—83.

168 Lomonte C, Basile C. Preoperative assessment and planning of haemodialysis vascular access. *Clin Kidney J* 2015;**8**:278—81.

169 Lo Monte AI, Damiano G, Mularo A, Palumbo VD, Alessi R, Gioviale MC, et al. Comparison between local and regional anesthesia in arteriovenous fistula creation. *J Vasc Access* 2011;**12**:331—5.

170 Malinzak EB, Gan TJ. Regional anesthesia for vascular access surgery. *Anesth Analg* 2009;**109**:976—80.

171 Sahin L, Gul R, Mizrak A, Deniz H, Sahin M, Koruk S, et al. Ultrasound-guided infraclavicular brachial plexus block enhances postoperative blood flow in arteriovenous fistulas. *J Vasc Surg* 2011;**54**:749—53.

172 Reynolds TS, Kim KM, Dukkipati R, Nguyen TH, Julka I, Kakazu C, et al. Pre-operative regional block anesthesia enhances operative strategy for arteriovenous fistula creation. *J Vasc Access* 2011;**12**:336—40.

173 Laskowski IA, Muhs B, Rockman CR, Adelman MA, Ranson M, Cayne NS, et al. Regional nerve block allows for optimization of planning in the creation of arteriovenous access for hemodialysis by improving superficial venous dilatation. *Ann Vasc Surg* 2007;**21**:730—3.

174 Hingorani AP, Ascher E, Gupta P, Alam S, Marks N, Schutzer RW, et al. Regional anesthesia: preferred technique for venodilatation in the creation of upper extremity arteriovenous fistulae. *Vascular* 2006;**14**:23—6.

175 Yildirim V, Doganci S, Yanarates O, Saglam M, Kuralay E, Cosar A, et al. Does preemptive stellate ganglion blockage increase the patency of radiocephalic arteriovenous fistula? *Scand Cardiovasc J* 2006;**40**:380—4.

176 Aitken E, Jackson A, Kearns R, Steven M, Kinsella J, Clancy M, et al. Effect of regional versus local anaesthesia on outcome after arteriovenous fistula creation: a randomised controlled trial. *Lancet* 2016;**388**:1067—74 (10049).

177 D'Ayala M, Smith RM, Martone C, Briggs W, Deitch JS, Wise L. The effect of systemic anticoagulation in patients undergoing angioaccess surgery. *Ann Vasc Surg* 2008;**22**:11—5.

178 Bhomi KK, Shrestha S, Bhattachan CL. Role of systemic anticoagulation in patients undergoing vascular access surgery. *Nepal Med Coll J* 2008;**10**:222—4.

179 Ravari H, Kazemzade GH, Sarookhani A, Khashayar P. Effect of heparin on the patency of arteriovenous fistula. *Acta Med Iran* 2008;**46**:379—82.

180 Smith GE, Gohil R, Chetter IC. Factors affecting the patency of arteriovenous fistulas for dialysis access. *J Vasc Surg* 2012;**55**:849—55.

181 Bourquelot P. Vascular access in children: the importance of microsurgery for creation of autologous arteriovenous fistulae. *Eur J Vasc Endovasc Surg* 2006;**32**:696—700.

182 Wedgwood KR, Wiggins PA, Guillou PJ. A prospective study of end-to-side vs. side-to-side arteriovenous fistulas for haemodialysis. *Br J Surg* 1984;**71**:640—2.

183 Gelabert HA, Freischlag JA. Haemodialysis access. In: Rutherford RB, editor. *Vascular surgery*. 5th ed. Philadelphia: Saunders; 2000. p. 1466—77.

184 Chemla E, Raynaud A, Carreres T, Sapoval M, Beyssen B, Bourquelot P, et al. Preoperative assessment of the efficacy of distal radial artery ligation in treatment of steal syndrome complicating access for hemodialysis. *Ann Vasc Surg* 1999;**13**:618—21.

185 Miller GA, Khariton K, Kardos SV, Koh E, Goel N, Khariton A. Flow interruption of the distal radial artery: treatment for finger ischemia in a matured radiocephalic AVF. *J Vasc Access* 2008;**9**:58—63.

186 Sidawy AN, Spergel LM, Besarab A, Allon M, Jennings WC, Padberg Jr FT, et al. The Society for Vascular Surgery: clinical practice guidelines for the surgical placement and maintenance of arteriovenous hemodialysis access. *J Vasc Surg* 2008;**48**:2S—25S.

187 Ernandez T, Saudan P, Berney T, Merminod T, Bednarkiewicz M, Martin PY. Risk factors for early failure of native arteriovenous fistulas. *Nephron Clin Pract* 2005;**101**:c39—44.

188 Nguyen TH, Bui TD, Gordon IL, Wilson SE. Functional patency of autogenous AV fistulas for hemodialysis. *J Vasc Access* 2007;**8**:275—80.

189 Scali ST, Huber TS. Treatment strategies for access-related hand ischemia. *Semin Vasc Surg* 2011;**24**:128—36.

190 Pirozzi N, Apponi F, Napoletano AM, Luciani R, Pirozzi V, Pugliese F. Microsurgery and preventive haemostasis for autogenous radial-cephalic direct wrist access in adult patients with radial artery internal diameter below 1.6 mm. *Nephrol Dial Transplant* 2010;**25**:520—5.

191 Kordzadeh A, Chung J, Panayiotopoulos YP. Cephalic vein and radial artery diameter in formation of radiocephalic arteriovenous fistula: a systematic review. *J Vasc Access* 2015;**16**:506—11.

192 Wong V, Ward R, Taylor J, Selvakumar S, How TV, Bakran A. Factors associated with early failure of arteriovenous fistulae for haemodialysis access. *Eur J Vasc Endovasc Surg* 1996;**12**:207—13.

193 Yoo DW, Yoon M, Jun HJ. Successful access rate and risk factor of vascular access surgery in arm for dialysis. *Vasc Spec Int* 2014;**30**:33—7.

194 White G, Wilson S. Planning and patient assessment for vascular access surgery. In: Wilson S, editor. *Vascular access principles and practice*. 4th ed. St. Louis: Mosby; 2002. p. 7—13.

195 Twine CP, Haidermota M, Woolgar JD, Gibbons CP, Davies CG. A scoring system (DISTAL) for predicting failure of snuffbox arteriovenous fistulas. *Eur J Vasc Endovasc Surg* 2012;**44**:88—91.

196 Weale AR, Bevis P, Neary WD, Boyes S, Morgan JD, Lear PA, et al. Radiocephalic and brachiocephalic arteriovenous fistula outcomes in the elderly. *J Vasc Surg* 2008;**47**:144—50.

197 Jennings WC, Landis L, Taubman KE, Parker DE. Creating functional autogenous vascular access in older patients. *J Vasc Surg* 2011;**53**:713—9. discussion 9.

198 Brunori G, Verzelletti F, Zubani R, Movilli E, Gaggiotti M, Cancarini G, et al. Which vascular access for chronic hemodialysis in uremic elderly patients? *J Vasc Access* 2000;**1**:134—8.

199 Ehsan O, Bhattacharya D, Darwish A, Al-khaffaf H. 'Extension technique': a modified technique for brachio-cephalic fistula to prevent dialysis access-associated steal syndrome. *Eur J Vasc Endovasc Surg* 2005;**29**:324—7.

200 Kian K, Shapiro JA, Salman L, Khan RA, Merrill D, Garcia L, et al. High brachial artery bifurcation: clinical considerations and practical implications for an arteriovenous access. *Semin Dial* 2012;**25**:244—7.

201 Lioupis C, Mistry H, Junghans C, Haughey N, Freedman B, Tyrrell M, et al. High brachial artery bifurcation is associated with failure of brachio-cephalic autologous arteriovenous fistulae. *J Vasc Access* 2010;**11**:132—7.

202 Bourquelot P, Van-Laere O, Baaklini G, Turmel-Rodrigues L, Franco G, Gaudric J, et al. Placement of wrist ulnar-basilic autogenous arteriovenous access for hemodialysis in adults and children using microsurgery. *J Vasc Surg* 2011;**53**:1298—302.

203 Tordoir JH, Keuter X, Planken N, de Haan MW, van der Sande FM. Autogenous options in secondary and tertiary access for haemodialysis. *Eur J Vasc Endovasc Surg* 2006;**31**:661—6.

204 Morosetti M, Cipriani S, Dominijanni S, Pisani G, Frattarelli D, Bruno F. Basilic vein transposition versus biosynthetic prosthesis as vascular access for hemodialysis. *J Vasc Surg* 2011;**54**:1713—9.

205 Lazarides MK, Georgiadis GS, Papasideris CP, Trellopoulos G, Tzilalis VD. Transposed brachial-basilic arteriovenous fistulas versus prosthetic upper limb grafts: a meta-analysis. *Eur J Vasc Endovasc Surg* 2008;**36**:597—601.

206 Weale AR, Bevis P, Neary WD, Lear PA, Mitchell DC. A comparison between transposed brachiobasilic arteriovenous fistulas and prosthetic brachioaxillary access grafts for vascular access for hemodialysis. *J Vasc Surg* 2007;**46**:997—1004.

207 Sala Almonacil V, Plaza Martínez A, Zaragozá García J, Martínez Parreño C, Al-Raies Bolaños B, Gómez Palonés F, et al. Comparison between autogenous brachial-basilic upper arm transposition fistulas and prosthetic brachial-axillary vascular accesses for hemodialysis. *J Cardiovasc Surg* 2011;**52**:725—30.

208 Woo K, Doros G, Ng T, Farber A. Comparison of the efficacy of upper arm transposed arteriovenous fistulae and upper arm prosthetic grafts. *J Vasc Surg* 2009;**50**:1405—11. e1—2.

209 Gonzalez E, Kashuk JL, Moore EE, Linas S, Sauaia A. Two-stage brachial-basilic transposition fistula provides superior patency rates for dialysis access in a safety-net population. *Surgery* 2010;**148**:687—93. discussion 93—94.

210 Kakkos SK, Haddad GK, Weaver MR, Haddad RK, Scully MM. Basilic vein transposition: what is the optimal technique? *Eur J Vasc Endovasc Surg* 2010;**39**:612—9.

211 Cooper J, Power AH, DeRose G, Forbes TL, Dubois L. Similar failure and patency rates when comparing one- and two-stage basilic vein transposition. *J Vasc Surg* 2015;**61**:809—16.

212 Paul EM, Sideman MJ, Rhoden DH, Jennings WC. Endoscopic basilic vein transposition for hemodialysis access. *J Vasc Surg* 2010;**51**:1451—6.

213 Jennings WC, Sideman MJ, Taubman KE, Broughan TA. Brachial vein transposition arteriovenous fistulas for hemodialysis access. *J Vasc Surg* 2009;**50**:1121—5. discussion 5—6.

214 Lioupis C, Mistry H, Chandak P, Tyrrell M, Valenti D. Autogenous brachial-brachial fistula for vein access. Haemodynamic factors predicting outcome and 1 year clinical data. *Eur J Vasc Endovasc Surg* 2009;**38**:770—6.

215 Morale W, Patane D, Incardona C, Seminara G, Messina M, Malfa P, et al. Venae comitantes as a potential vascular resource to create native arteriovenous fistulae. *J Vasc Access* 2011;**12**:211—4.

216 Torina PJ, Westheimer EF, Schanzer HR. Brachial vein transposition arteriovenous fistula: is it an acceptable option for chronic dialysis vascular access? *J Vasc Access* 2008;**9**:39—44.

217 Rueda CA, Nehler MR, Kimball TA, Dimond KR, Whitehill TA, Peyton BD. Arteriovenous fistula construction using femoral vein in the thigh and upper extremity: single-center experience. *Ann Vasc Surg* 2008;**22**:806—14.

218 Smith GE, Carradice D, Samuel N, Gohil R, Chetter IC. Great saphenous vein transposition to the forearm for dialysis vascular access; an under used autologous option? *J Vasc Access* 2011;**12**:354—7.

219 Maya ID, Weatherspoon J, Young CJ, Barker J, Allon M. Increased risk of infection associated with polyurethane dialysis grafts. *Semin Dial* 2007;**20**:616—20.

220 Hazinedaroglu SM, Tuzuner A, Ayli D, Demirer S, Duman N, Yerdel MA. Femoral vein transposition versus femoral loop grafts for hemodialysis: a prospective evaluation. *Transplant Proc* 2004;**36**:65—7.

221 Berardinelli L. Grafts and graft materials as vascular substitutes for haemodialysis access construction. *Eur J Vasc Endovasc Surg* 2006;**32**:203—11.

222 Sorom AJ, Hughes CB, McCarthy JT, Jenson BM, Prieto M, Panneton JM, et al. Prospective, randomized evaluation of a cuffed expanded polytetrafluoroethylene graft for hemodialysis vascular access. *Surgery* 2002;**132**:135—40.

223 Ko PJ, Liu YH, Hung YN, Hsieh HC. Patency rates of cuffed and noncuffed extended polytetrafluoroethylene grafts in dialysis access: a prospective, randomized study. *World J Surg* 2009;**33**:846—51.

224 Shemesh D, Goldin I, Hijazi J, Zaghal I, Berelowitz D, Verstandig A, et al. A prospective randomized study of heparin-bonded graft (Propaten) versus standard graft in prosthetic arteriovenous access. *J Vasc Surg* 2015;**62**:115—22.

225 Davidson I, Hackerman C, Kapadia A, Minhajuddib A. Heparin bonded hemodialysis e-PTFE grafts result in 20% clot free survival benefit. *J Vasc Access* 2009;**10**:153—6.

226 Lemson MS, Tordoir JH, van Det RJ, Welten RJ, Burger H, Estourgie RJ, et al. Effects of a venous cuff at the venous anastomosis of polytetrafluoroethylene grafts for hemodialysis vascular access. *J Vasc Surg* 2000;**32**:1155—63.

227 Schild AF, Schuman ES, Noicely K, Kaufman J, Gillaspie E, Fuller J, et al. Early cannulation prosthetic graft (Flixene) for arteriovenous access. *J Vasc Access* 2011;**12**:248—52.

228 Lioupis C, Mistry H, Rix T, Chandak P, Tyrrell M, Valenti D. Comparison among transposed brachiobasilic, brachiobrachial arteriovenous fistulas and Flixene vascular graft. *J Vasc Access* 2011;**12**:36—44.

229 Chemla ES, Nelson S, Morsy M. Early cannulation grafts in straight axillo-axillary angioaccesses avoid central catheter insertions. *Semin Dial* 2011;**24**:456—9.

230 Kakkos SK, Andrzejewski T, Haddad JA, Haddad GK, Reddy DJ, Nypaver TJ, et al. Equivalent secondary patency rates of upper extremity vectra vascular access grafts and transposed brachial-basilic fistulas with aggressive access surveillance and endovascular treatment. *J Vasc Surg* 2008;**47**:407—14.

231 Kakkos S, Topalidis D, Haddad R, Haddad GK, Shepard AD. Long-term complication and patency rates of Vectra and IMPRA Carboflo vascular access grafts with aggressive monitoring, surveillance and endovascular management. *Vascular* 2011;**19**:21—8.

232 Schild AF, Baltodano NM, Alfieri K, Livingstone J, Raines JK. New graft for low friction tunneling in vascular access surgery. *J Vasc Access* 2004;**5**:19—24.

233 Palumbo R, Niscola P, Calabria S, Fierimonte S, Bevilacqua M, Scaramucci L, et al. Long-term favorable results by arteriovenous graft with Omniflow II prosthesis for hemodialysis. *Nephron Clin Pract* 2009;**113**:c76—80.

234 Glickman MH. HeRO vascular access device. *Semin Vasc Surg* 2011;**24**:108—12.

235 Gage SM, Katzman HE, Ross JR, Hohmann SE, Sharpe CA, Butterly DW, et al. Multi-center experience of 164 consecutive Hemodialysis Reliable Outflow [HeRO] graft implants for

hemodialysis treatment. *Eur J Vasc Endovasc Surg* 2012;**44**:93—9.
236 Kennealey PT, Elias N, Hertl M, Ko DS, Saidi RF, Markmann JF, et al. A prospective, randomized comparison of bovine carotid artery and expanded polytetrafluoroethylene for permanent hemodialysis vascular access. *J Vasc Surg* 2011;**53**:1640—8.
237 Tahami VB, Hakki H, Reber PU, Widmer MK, Kniemeyer HW. Polytetrafluoroethylene and bovine mesenterial vein grafts for hemodialysis access: a comparative study. *J Vasc Access* 2007;**8**:17—20.
238 McAllister TN, Maruszewski M, Garrido SA, Wystrychowski W, Dusserre N, Marini A, et al. Effectiveness of haemodialysis access with an autologous tissue-engineered vascular graft: a multicentre cohort study. *Lancet* 2009;**373**:1440—6.
239 Shenoy S, Miller A, Petersen F, Kirsch WM, Konkin T, Kim P, et al. A multicenter study of permanent hemodialysis access patency: beneficial effect of clipped vascular anastomotic technique. *J Vasc Surg* 2003;**38**:229—35.
240 Lin PH, Bush RL, Nelson JC, Lam R, Paladugu R, Chen C, et al. A prospective evaluation of interrupted nitinol surgical clips in arteriovenous fistula for hemodialysis. *Am J Surg* 2003;**186**:625—30.
241 Schild AF, Raines J. Preliminary prospective randomized experience with vascular clips in the creation of arteriovenous fistulae for hemodialysis. *Am J Surg* 1999;**178**:33—7.
242 Schlieper G, Kruger T, Djuric Z, Damjanovic T, Markovic N, Schurgers LJ, et al. Vascular access calcification predicts mortality in hemodialysis patients. *Kidney Int* 2008;**74**:1582—7.
243 Feezor RJ. Approach to permanent hemodialysis access in obese patients. *Semin Vasc Surg* 2011;**24**:96—101.
244 Bourquelot P, Tawakol JB, Gaudric J, Natario A, Franco G, Turmel-Rodrigues L, et al. Lipectomy as a new approach to secondary procedure superficialization of direct autogenous forearm radial-cephalic arteriovenous accesses for hemodialysis. *J Vasc Surg* 2009;**50**:369—74. 74 e1.
245 Tordoir JH, van Loon MM, Peppelenbosch N, Bode AS, Poeze M, van der Sande FM. Surgical techniques to improve cannulation of hemodialysis vascular access. *Eur J Vasc Endovasc Surg* 2010;**39**:333—9.
246 Stoikes N, Nezakatgoo N, Fischer P, Bahr M, Magnotti L. Salvage of inaccessible arteriovenous fistulas in obese patients: a review of 132 brachiocephalic fistulas. *Am Surg* 2009;**75**:705—9. discussion 9.
247 Barnard KJ, Taubman KE, Jennings WC. Accessible autogenous vascular access for hemodialysis in obese individuals using lipectomy. *Am J Surg* 2010;**200**:798—802. discussion.
248 Causey MW, Quan R, Hamawy A, Singh N. Superficialization of arteriovenous fistulae employing minimally invasive liposuction. *J Vasc Surg* 2010;**52**:1397—400.
249 Ochoa DA, Mitchell RE, Jennings WC. Liposuction over a shielded arteriovenous fistula for hemodialysis access maturation. *J Vasc Access* 2010;**11**:69—71.
250 Galt S, Crawford M, Blebea J, Ladenheim E, Browne B. The efficacy and durability of the Venous Window Needle Guide implanted on uncannulatable arteriovenous fistulas. *J Vasc Surg* 2016;**64**:708—14.
251 Mestres G, Fontsere N, Garcia-Madrid C, Campelos P, Maduell F, Riambau V. Intra-operative factors predicting 1-month arteriovenous fistula thrombosis. *J Vasc Access* 2012;**13**:193—7.
252 Saucy F, Haesler E, Haller C, Deglise S, Teta D, Corpataux JM. Is intra-operative blood flow predictive for early failure of radiocephalic arteriovenous fistula? *Nephrol Dial Transplant* 2010;**25**:862—7.
253 Berman SS, Mendoza B, Westerband A, Quick RC. Predicting arteriovenous fistula maturation with intraoperative blood flow measurements. *J Vasc Access* 2008;**9**:241—7.
254 Won T, Jang JW, Lee S, Han JJ, Park YS, Ahn JH. Effects of intraoperative blood flow on the early patency of radiocephalic fistulas. *Ann Vasc Surg* 2000;**14**:468—72.
255 Padberg Jr FT, Calligaro KD, Sidawy AN. Complications of arteriovenous hemodialysis access: recognition and management. *J Vasc Surg* 2008;**48**:55S—80S.
256 Schild AF, Simon S, Prieto J, Raines J. Single-center review of infections associated with 1,574 consecutive vascular access procedures. *Vasc Endovasc Surg* 2003;**37**:27—31.
257 Clinical practice guidelines for vascular access. *Am J Kidney Dis* 2006;**48**:S176—247.
258 Sarfati M, Berman S. Complications of haemodialysis access fisulae and grafts. In: Berman S, editor. *Vascular access in clinical practice*. New York: Marcel Dekker; 2002. p. 207—37.
259 Taylor B, Sigley RD, May KJ. Fate of infected and eroded hemodialysis grafts and autogenous fistulas. *Am J Surg* 1993;**165**:632—6.
260 Ryan SV, Calligaro KD, Dougherty MJ. Management of hemodialysis access infections. *Semin Vasc Surg* 2004;**17**:40—4.
261 Padberg Jr FT, Lee BC, Curl GR. Hemoaccess site infection. *Surg Gynecol Obstet* 1992;**174**:103—8.
262 Scollay JM, Skipworth RJ, Severn A, Nagy J, Howd A, Griffiths GD. Vascular access using the superficial femoral vein. *J Vasc Access* 2010;**11**:312—5.
263 Tordoir JH, Dammers R, van der Sande FM. Upper extremity ischemia and hemodialysis vascular access. *Eur J Vasc Endovasc Surg* 2004;**27**:1—5.
264 Wixon CL, Miles JL. Hemodynamic basis for the diagnosis and treatment of angioaccess induced steal syndrome. *Advances Vasc Surg* 2000;**8**:147—59.
265 Schanzer H, Skladany M, Haimov M. Treatment of angioaccess-induced ischemia by revascularization. *J Vasc Surg* 1992;**16**:861—4. discussion 4—6.
266 Lazarides MK, Staramos DN, Kopadis G, Maltezos C, Tzilalis VD, Georgiadis GS. Onset of arterial 'steal' following proximal angioaccess: immediate and delayed types. *Nephrol Dial Transplant* 2003;**18**:2387—90.
267 Aimaq R, Katz SG. Using distal revascularization with interval ligation as the primary treatment of hand ischemia after dialysis access creation. *J Vasc Surg* 2013;**57**:1073—8. discussion 8.
268 Shemesh D, Goldin I, Berelowitz D, Zaghal I, Olsha O. Thrombolysis for early failure of prosthetic arteriovenous access. *J Vasc Surg* 2008;**47**:585—90.
269 Green LD, Lee DS, Kucey DS. A metaanalysis comparing surgical thrombectomy, mechanical thrombectomy, and pharmacomechanical thrombolysis for thrombosed dialysis grafts. *J Vasc Surg* 2002;**36**:939—45.
270 Kuhan G, Antoniou GA, Nikam M, Mitra S, Farquharson F, Brittenden J, et al. A meta-analysis of randomized trials comparing surgery versus endovascular therapy for thrombosed arteriovenous fistulas and grafts in hemodialysis. *Cardiovasc Intervent Radiol* 2013;**36**:699—705.
271 Tordoir JH, Bode AS, Peppelenbosch N, van der Sande FM, de Haan MW. Surgical or endovascular repair of thrombosed dialysis vascular access: is there any evidence? *J Vasc Surg* 2009;**50**:953—6.

272 Yurkovic A, Cohen RD, Mantell MP, Kobrin S, Soulen MC, Chittams J, et al. Outcomes of thrombectomy procedures performed in hemodialysis grafts with early failure. *J Vasc Intervent Radiol JVIR* 2011;**22**:317—24.

273 Akin EB, Topcu O, Ozcan H, Ersoz S, Aytac S, Anadol E. Hemodynamic effect of transdermal glyceryl trinitrate on newly constructed arteriovenous fistula. *World J Surg* 2002;**26**:1256—9.

274 Field M, McGrogan D, Marie Y, Joinson M, Andujar C, Dutton M, et al. Randomized clinical trial of the use of glyceryl trinitrate patches to aid arteriovenous fistula maturation. *Br J Surg* 2016;**103**:1269—75.

275 Goodkin DA, Pisoni RL, Locatelli F, Port FK, Saran R. Hemodialysis vascular access training and practices are key to improved access outcomes. *Am J Kidney Dis* 2010;**56**:1032—42.

276 Gundevia Z, Whalley H, Ferring M, Claridge M, Smith S, Wilmink T. Effect of operating surgeon on outcome of arteriovenous fistula formation. *Eur J Vasc Endovasc Surg* 2008;**35**:614—8.

277 Weale AR, Barwell J, Chant H, Lear PA, Mitchell DC. The impact of training on outcomes in primary vascular access surgery. *Ann R Coll Surg Engl* 2004;**86**:275—80.

278 Fassiadis N, Morsy M, Siva M, Marsh JE, Makanjuola AD, Chemla ES. Does the surgeon's experience impact on radiocephalic fistula patency rates? *Semin Dial* 2007;**20**:455—7.

279 Wilmink TF, Ferring M. Training in vascular access surgery. In: Tordoir J, editor. *Vascular Access*. Edizioni Minerva Medica; 2009. p. 133—40.

280 Ives CL, Akoh JA, George J, Vaughan-Huxley E, Lawson H. Preoperative vessel mapping and early post-operative surveillance duplex scanning of arteriovenous fistulae. *J Vasc Access* 2009;**10**:37—42.

281 Jemcov TK. Morphologic and functional vessels characteristics assessed by ultrasonography for prediction of radiocephalic fistula maturation. *J Vasc Access* 2013;**14**:356—63.

282 Miller PE, Tolwani A, Luscy CP, Deierhoi MH, Bailey R, Redden DT, et al. Predictors of adequacy of arteriovenous fistulas in hemodialysis patients. *Kidney Int* 1999;**56**:275—80.

283 Tordoir JH, Rooyens P, Dammers R, van der Sande FM, de Haan M, Yo TI. Prospective evaluation of failure modes in autogenous radiocephalic wrist access for haemodialysis. *Nephrol Dial Transplant* 2003;**18**:378—83.

284 Rayner HC, Pisoni RL, Gillespie BW, Goodkin DA, Akiba T, Akizawa T, et al. Creation, cannulation and survival of arteriovenous fistulae: data from the Dialysis Outcomes and Practice Patterns Study. *Kidney Int* 2003;**63**:323—30.

285 Brunori G, Ravani P, Mandolfo S, Imbasciati E, Malberti F, Cancarini G. Fistula maturation: doesn't time matter at all? *Nephrol Dial Transplant* 2005;**20**:684—7.

286 Culp K, Flanigan M, Taylor L, Rothstein M. Vascular access thrombosis in new hemodialysis patients. *Am J Kidney Dis* 1995;**26**:341—6.

287 Saran R, Dykstra DM, Pisoni RL, Akiba T, Akizawa T, Canaud B, et al. Timing of first cannulation and vascular access failure in haemodialysis: an analysis of practice patterns at dialysis facilities in the DOPPS. *Nephrol Dial Transplant* 2004;**19**:2334—40.

288 Malik J, Slavikova M, Malikova H, Maskova J. Many clinically silent access stenoses can be identified by ultrasonography. *J Nephrol* 2002;**15**:661—5.

289 van Loon MM, Kessels AG, Van der Sande FM, Tordoir JH. Cannulation and vascular access-related complications in hemodialysis: factors determining successful cannulation. *Hemodial Int* 2009;**13**:498—504.

290 Saran R, Pisoni RL, Young EW. Timing of first cannulation of arteriovenous fistula: are we waiting too long? *Nephrol Dial Transplant* 2005;**20**:688—90.

291 Shemesh D, Goldin I, Berelowitz D, Zaghal I, Zigelman C, Olsha O. Blood flow volume changes in the maturing arteriovenous access for hemodialysis. *Ultrasound Med Biol* 2007;**33**:727—33.

292 Glickman MH, Stokes GK, Ross JR, Schuman ED, Sternbergh 3rd WC, Lindberg JS, et al. Multicenter evaluation of a polytetrafluoroethylene vascular access graft as compared with the expanded polytetrafluoroethylene vascular access graft in hemodialysis applications. *J Vasc Surg* 2001;**34**:465—72. discussion 72—73.

293 Oder TF, Teodorescu V, Uribarri J. Effect of exercise on the diameter of arteriovenous fistulae in hemodialysis patients. *ASAIO J* 2003;**49**:554—5.

294 Fontsere N, Mestres G, Yugueros X, Lopez T, Yuguero A, Bermudez P, et al. Effect of a postoperative exercise program on arteriovenous fistula maturation: A randomized controlled trial. *Hemodial Int* 2016;**20**:306—14.

295 Beathard GA. An algorithm for the physical examination of early fistula failure. *Semin Dial* 2005;**18**:331—5.

296 Malovrh M. Non-matured arteriovenous fistulae for haemodialysis: diagnosis, endovascular and surgical treatment. *Bosn J Basic Med Sci* 2010;**10**:S13—7.

297 Patel ST, Hughes J, Mills Sr JL. Failure of arteriovenous fistula maturation: an unintended consequence of exceeding dialysis outcome quality Initiative guidelines for hemodialysis access. *J Vasc Surg* 2003;**38**:439—45. discussion 45.

298 Singh P, Robbin ML, Lockhart ME, Allon M. Clinically immature arteriovenous hemodialysis fistulas: effect of US on salvage. *Radiology* 2008;**246**:299—305.

299 Nassar GM, Nguyen B, Rhee E, Achkar K. Endovascular treatment of the "failing to mature" arteriovenous fistula. *Clin J Am Soc Nephrol CJASN* 2006;**1**:275—80.

300 McLafferty RB, Pryor 3rd RW, Johnson CM, Ramsey DE, Hodgson KJ. Outcome of a comprehensive follow-up program to enhance maturation of autogenous arteriovenous hemodialysis access. *J Vasc Surg* 2007;**45**:981—5.

301 Lee T, Ullah A, Allon M, Succop P, El-Khatib M, Munda R, et al. Decreased cumulative access survival in arteriovenous fistulas requiring interventions to promote maturation. *Clin J Am Soc Nephrol CJASN* 2011;**6**:575—81.

302 Sands JJ. Vascular access monitoring improves outcomes. *Blood Purif* 2005;**23**:45—9.

303 Tanner NC, Da Silva A. Medical adjuvant treatment to increase patency of arteriovenous fistulae and grafts. *Cochrane Database Syst Rev* 2015;**7**:CD002786.

304 Irish AB, Viecelli AK, Hawley CM, Hooi LS, Pascoe EM, Paul-Brent PA, et al. Effect of fish oil supplementation and aspirin use on arteriovenous fistula failure in patients requiring hemodialysis: A randomized clinical trial. *JAMA Intern Med* 2017;**177**:184—93.

305 Paraskevas KI, Mikhailidis DP, Roussas N, Giannoukas AD. Effect of antiplatelet agents, statins, and other drugs on vascular access patency rates. *Angiology* 2012;**63**:5—8.

306 Chan K, Lazarus JM, Thadhani R, Hakim RM. Anticoagulant and antiplatelet usage associated with mortality among hemodialysis patients. *J Am Soc Nephrol* 2009;**20**:872—81.

307 Ethier J, Bragg-Gresham JL, Piera L, Akizawa T, Asano Y, Mason N, et al. Aspirin prescription and outcomes in hemodialysis patients: the dialysis outcomes and practice patterns study. (DOPPS). *Am J Kidney Dis* 2007;**50**:602—11.

308 Palmer SC, Di Micco L, Razavian M, Craig JC, Perkovic V, Pellegrini F, et al. Antiplatelet agents for chronic kidney disease [review]. *Cochrane Database Syst Rev* 2013:CD008834.
309 Kaufman JS, O'Connor TZ, Zhang JH, Cronin RE, Fiore LD, Ganz MB, et al. Randomized controlled trial of clopidogrel plus aspirin to prevent hemodialysis access graft thrombosis. *J Am Soc Nephrol* 2003;**14**:2313—21.
310 Crowther MA, Clase CM, Margetts PJ, Julian J, Lambert K, Sneath D, et al. Low-intensity warfarin is ineffective for the prevention of PTFE graft failure in patients on hemodialysis: a randomized controlled trial. *J Am Soc Nephrol* 2002;**13**:2331—7.
311 Sharathkumar A, Hirschl R, Pipe S, Crandell C, Adams B, Lin JJ. Primary thromboprophylaxis with heparins for arteriovenous fistula failure in pediatric patients. *J Vasc Access* 2007;**8**:235—44.
312 Bowden RG, Wilson RL, Gentile M, Ounpraseuth S, Moore P, Leutholtz BC. Effects of omega-3 fatty acid supplementation on vascular access thrombosis in polytetrafluorethylene grafts. *J Ren Nutr* 2007;**17**:126—31.
313 Lok CE, Moist L, Hemmelgarn BR, Tonelli M, Vazquez MA, Dorval M, et al. Effect of fish oil supplementation on graft patency and cardiovascular events among patients with new synthetic arteriovenous hemodialysis grafts: a randomized controlled trial. *JAMA* 2012;**307**:1809—16.
314 Chang HH, Chang YK, Lu CW, Huang CT, Chien CT, Hung KY, et al. Statins Improve long term patency of arteriovenous fistula for hemodialysis. *Sci Rep* 2016;**6**:22197.
315 Pisoni R, Barker-Finkel J, Allo M. Statin therapy is not associated with improved vascular access outcomes. *Clin J Am Soc Nephrol CJASN* 2010;**5**:1447—50.
316 Parisotto MT, Schoder VU, Miriunis C, Grassmann AH, Scatizzi LP, Kaufmann P, et al. Cannulation technique influences arteriovenous fistula and graft survival. *Kidney Int* 2014;**86**:790—7.
317 Sands JJ. Vascular access 2007. *Minerva Urol Nefrol* 2007;**59**:237—49.
318 Asif A, Leon C, Orozco-Vargas LC, Krishnamurthy G, Choi KL, Mercado C, et al. Accuracy of physical examination in the detection of arteriovenous fistula stenosis. *Clin J Am Soc Nephrol CJASN* 2007;**2**:1191—4.
319 Campos RP, Chula DC, Perreto S, Riella MC, do Nascimento MM. Accuracy of physical examination and intraaccess pressure in the detection of stenosis in hemodialysis arteriovenous fistula. *Semin Dial* 2008;**21**:269—73.
320 Coentrao L, Faria B, Pestana M. Physical examination of dysfunctional arteriovenous fistulae by non-interventionalists: a skill worth teaching. *Nephrol Dial Transplant* 2012;**27**:1993—6.
321 Leon C, Orozco-Vargas LC, Krishnamurthy G, Choi KL, Mercado C, Merrill D, et al. Accuracy of physical examination in the detection of arteriovenous graft stenosis. *Semin Dial* 2008;**21**:85—8.
322 Salman L, Beathard G. Interventional nephrology: Physical examination as a tool for surveillance for the hemodialysis arteriovenous access. *Clin J Am Soc Nephrol CJASN* 2013;**8**:1220—7.
323 Tokars JI, Arduino MJ, Alter MJ. Infection control in hemodialysis units. *Infect Dis Clin North Am* 2001;**15**:797—812. viii.
324 Kaplowitz LG, Comstock JA, Landwehr DM, Dalton HP, Mayhall CG. Prospective study of microbial colonization of the nose and skin and infection of the vascular access site in hemodialysis patients. *J Clin Microbiol* 1988;**26**:1257—62.
325 Higgins M, Evans DS. Nurses' knowledge and practice of vascular access infection control in haemodialysis patients in the Republic of Ireland. *J Ren Care* 2008;**34**:48—53.
326 Dhingra RK, Young EW, Hulbert-Shearon TE, Leavey SF, Port FK. Type of vascular access and mortality in U.S. hemodialysis patients. *Kidney Int* 2001;**60**:1443—51.
327 van Loon MM, Goovaerts T, Kessels AG, van der Sande FM, Tordoir JH. Buttonhole needling of haemodialysis arteriovenous fistulae results in less complications and interventions compared to the rope-ladder technique. *Nephrol Dial Transplant* 2010;**25**:225—30.
328 MacRae JM, Ahmed SB, Atkar R, Hemmelgarn BR. A randomized trial comparing buttonhole with rope ladder needling in conventional hemodialysis patients. *Clin J Am Soc Nephrol CJASN* 2012;**7**:1632—8.
329 Labriola L, Crott R, Desmet C, Andre G, Jadoul M. Infectious complications following conversion to buttonhole cannulation of native arteriovenous fistulas: a quality improvement report. *Am J Kidney Dis* 2011;**57**:442—8.
330 Doss S, Schiller B, Moran J. Buttonhole cannulation—an unexpected outcome. *Nephrol Nurs J* 2008;**35**:417—9.
331 Ball LK. Improving arteriovenous fistula cannulation skills. *Nephrol Nurs J* 2005;**32**:611—7. quiz 8.
332 Kundu S, Achar S. Principles of office anesthesia: part II. Topical anesthesia. *Am Fam Physician* 2002;**66**:99—102.
333 Kumbar L, Karim J, Besarab A. Surveillance and monitoring of dialysis access. *Int J Nephrol* 2012;**2012**:649735.
334 Robbin ML, Oser RF, Lee JY, Heudebert GR, Mennemeyer ST, Allon M. Randomized comparison of ultrasound surveillance and clinical monitoring on arteriovenous graft outcomes. *Kidney Int* 2006;**69**:730—5.
335 Brouwer DJ. Cannulation camp: basic needle cannulation training for dialysis staff. *Dial Transplant* 1995;**24**:1—7.
336 van Loon MM, Kessels AG, van der Sande FM, Tordoir JH. Cannulation practice patterns in haemodialysis vascular access: predictors for unsuccessful cannulation. *J Ren Care* 2009;**35**:82—9.
337 Lee T, Barker J, Allon M. Needle infiltration of arteriovenous fistulae in hemodialysis: risk factors and consequences. *Am J Kidney Dis* 2006;**47**:1020—6.
338 Schoch M, Smith V. Advanced vascular access workshop for dialysis nurses: a three-year review. *Ren Soc Australas J* 2012;**8**:89—93.
339 van Hooland S, Donck J, Ameye F, Aerden D. Duplex ultrasonography and haemodialysis vascular access: a practical review. *Int J Nephrol Urol* 2010;**2**:283—93.
340 Marticorena RM, Mills L, Sutherland K, McBride N, Kumar L, Bachynski JC, et al. Development of competencies for the use of bedside ultrasound for assessment and cannulation of hemodialysis vascular access. *CANNT J = J ACITN* 2015;**25**:28—32.
341 McCann M, Einarsdottir H, Van Waeleghem JP, Murphy F, Sedgwick J. Vascular access management II: AVF/AVG cannulation techniques and complications. *J Ren Care* 2009;**35**:90—8.
342 Krönung G. Plastic deformation of Cimino fistula by repeated puncture. *Dial Transplant* 1984;**13**:635—8.
343 Brouwer D. Needle placement is paramount to achieving effective dialysis and preserving vascular accesses. *Nephrol Nurs J* 2005;**32**:225—7.
344 Hartigan MF. Vascular access and nephrology nursing practice: existing views and rationales for change. *Adv Ren Replace Ther* 1994;**1**:155—62.
345 English DJ. Retrograde arterial needle placement improves dialysis adequacy. *Nephrol Nurs J* 2005;**32**:224.

346 Ozmen S, Kadiroglu AK, Ozmen CA, Danis R, Sit D, Akin D, et al. Does the direction of arterial needle in AV fistula cannulation affect dialysis adequacy? *Clin Nephrol* 2008;**70**:229—32.
347 Unnikrishnan S, Huynh TN, Brott BC, Ito Y, Cheng CH, Shih AM, et al. Turbulent flow evaluation of the venous needle during hemodialysis. *J Biomech Eng* 2005;**127**:1141—6.
348 Twardowski Z, Kubara H. Different sites versus constant sites of needle insertion into arteriovenous fistulas for treatment by repeated dialysis. *Dial Transplant* 1979;**8**:978.
349 Ball LK. Buttonhole technique. *Nephrol Nurs J* 2012;**39**:151. author reply -2.
350 Ball LK. The buttonhole technique: strategies to reduce infections. *Nephrol Nurs J* 2010;**37**:473—7. quiz 8.
351 Verhallen AM, Kooistra MP, van Jaarsveld BC. Cannulating in haemodialysis: rope-ladder or buttonhole technique? *Nephrol Dial Transplant* 2007;**22**:2601—4.
352 Ludlow V. Buttonhole cannulation in hemodialysis: improved outcomes and increased expense—is it worth it? *CANNT J = J ACITN* 2010;**20**:29—37.
353 Ball LK, Treat L, Riffle V, Scherting D, Swift L. A multi-center perspective of the buttonhole technique in the Pacific Northwest. *Nephrol Nurs J* 2007;**34**:234—41.
354 Kim MK, Kim HS. Clinical effects of buttonhole cannulation method on hemodialysis patients. *Hemodial Int* 2013;**17**:294—9.
355 Castro MC, Silva Cde F, Souza JM, Assis MC, Aoki MV, Xagoraris M, et al. Arteriovenous fistula cannulation by buttonhole technique using dull needle. *J Bras Nefrol* 2010;**32**:281—5.
356 Peterson P. Fistula cannulation: the buttonhole technique. *Nephrol Nurs J* 2002;**29**:195.
357 Lewis C. Let's empower patients with the choice of self-cannulation! *Nephrol Nurs J* 2005;**32**:225.
358 Huber TS, Carter JW, Carter RL, Seeger JM. Patency of autogenous and polytetrafluoroethylene upper extremity arteriovenous hemodialysis accesses: a systematic review. *J Vasc Surg* 2003;**38**:1005—11.
359 Perera GB, Mueller MP, Kubaska SM, Wilson SE, Lawrence PF, Fujitani RM. Superiority of autogenous arteriovenous hemodialysis access: maintenance of function with fewer secondary interventions. *Ann Vasc Surg* 2004;**18**:66—73.
360 Pisoni RL, Young EW, Dykstra DM, Greenwood RN, Hecking E, Gillespie B, et al. Vascular access use in Europe and the United States: results from the DOPPS. *Kidney Int* 2002;**61**:305—16.
361 Struthers J, Allan A, Peel RK, Lambie SH. Buttonhole needling of ateriovenous fistulae: a randomized controlled trial. *ASAIO J* 2010;**56**:319—22.
362 MacRae JM, Ahmed SB, Hemmelgarn BR. Arteriovenous fistula survival and needling technique: long-term results from a randomized buttonhole trial. *Am J Kidney Dis* 2014;**63**:636—42.
363 Chow J, Rayment G, San Miguel S, Gilbert M. A randomised controlled trial of buttonhole cannulation for the prevention of fistula access complications. *J Ren Care* 2011;**37**:85—93.
364 Birchenough E, Moore C, Stevens K, Stewart S. Buttonhole cannulation in adult patients on hemodialysis: an increased risk of infection? *Nephrol Nurs J* 2010;**37**:491—8. 555; quiz 499.
365 Mott S, Prowant BF. The "touch cannulation" technique for hemodialysis. *Nephrol Nurs J* 2008;**35**:65—6.
366 Nesrallah GE, Cuerden M, Wong JH, Pierratos A. *Staphylococcus aureus* bacteremia and buttonhole cannulation: long-term safety and efficacy of mupirocin prophylaxis. *Clin J Am Soc Nephrol CJASN* 2010;**5**:1047—53.
367 Wong B, Muneer M, Wiebe N, Storie D, Shurraw S, Pannu N, et al. Buttonhole versus rope-ladder cannulation of arteriovenous fistulas for hemodialysis: a systematic review. *Am J Kidney Dis* 2014;**64**:918—36.
368 Evans LM. Buttonhole cannulation for haemodialysis: a nursing review. *Ren Soc Australas J* 2012;**8**:146—51.
369 Ball LK. The buttonhole technique for arteriovenous fistula cannulation. *Nephrol Nurs J* 2006;**33**:299—304.
370 Hakim RM, Breyer J, Ismail N, Schulman G. Effects of dose of dialysis on morbidity and mortality. *Am J Kidney Dis* 1994;**23**:661—9.
371 Feldman HI, Kobrin S, Wasserstein A. Hemodialysis vascular access morbidity. *J Am Soc Nephrol* 1996;**7**:523—35.
372 Rocco MV, Bleyer AJ, Burkart JM. Utilization of inpatient and outpatient resources for the management of hemodialysis access complications. *Am J Kidney Dis* 1996;**28**:250—6.
373 Feldman HI, Held PJ, Hutchinson JT, Stoiber E, Hartigan MF, Berlin JA. Hemodialysis vascular access morbidity in the United States. *Kidney Int* 1993;**43**:1091—6.
374 Bay WH, Van Cleef S, Owens M. The hemodialysis access: preferences and concerns of patients, dialysis nurses and technicians, and physicians. *Am J Nephrol* 1998;**18**:379—83.
375 Besarab A. Advances in end-stage renal diseases 2000. Access monitoring methods. *Blood Purif* 2000;**18**:255—9.
376 Allon M, Bailey R, Ballard R, Deierhoi MH, Hamrick K, Oser R, et al. A multidisciplinary approach to hemodialysis access: prospective evaluation. *Kidney Int* 1998;**53**:473—9.
377 Cull DL, Taylor SM, Russell HE, Langan EM, Snyder BA, Sullivan TM. The impact of a community-wide vascular access program on the management of graft thromboses in a dialysis population of 495 patients. *Am J Surg* 1999;**178**:113—6.
378 Dwyer A, Shelton P, Brier M, Aronoff G. A vascular access coordinator improves the prevalent fistula rate. *Semin Dial* 2012;**25**:239—43.
379 Beathard GA. The treatment of vascular access graft dysfunction: a nephrologist's view and experience. *Adv Ren Replace Ther* 1994;**1**:131—47.
380 Beathard GA. Physical examination: The forgotten tool. In: Gray RJ, Sands JJ, editors. *Dialysis access: a multidisciplinary approach*. Philadelphia, PA: Lippincott Williams & Wilkins; 2002. p. 111—8.
381 Trerotola SO, Scheel Jr PJ, Powe NR, Prescott C, Feeley N, He J, et al. Screening for dialysis access graft malfunction: comparison of physical examination with US. *J Vasc Intervent Radiol JVIR* 1996;**7**:15—20.
382 Schuman E, Ronfeld A, Barclay C, Heinl P. Comparison of clinical assessment with ultrasound flow for hemodialysis access surveillance. *Arch Surg* 2007;**142**:1129—33.
383 Leon C, Asif A. Physical examination of arteriovenous fistulae by a renal fellow: does it compare favorably to an experienced interventionalist? *Semin Dial* 2008;**21**:557—60.
384 Beathard GA. Physical examination of the dialysis vascular access. *Semin Dial* 1998;**11**:231—6.
385 Tonelli M, James M, Wiebe N, Jindal K, Hemmelgarn B. Ultrasound monitoring to detect access stenosis in hemodialysis patients: a systematic review. *Am J Kidney Dis* 2008;**51**:630—40.
386 Casey ET, Murad MH, Rizvi AZ, Sidawy AN, McGrath MM, Elamin MB, et al. Surveillance of arteriovenous hemodialysis access: a systematic review and meta-analysis. *J Vasc Surg* 2008;**48**:48S—54S.
387 Krivitski NM. Novel method to measure access flow during hemodialysis by ultrasound velocity dilution technique. *ASAIO J* 1995;**41**:M741—5.

388 Krivitski NM. Theory and validation of access flow measurement by dilution technique during hemodialysis. *Kidney Int* 1995;**48**:244—50.
389 Depner TA, Krivitski NM. Clinical measurement of blood flow in hemodialysis access fistulae and grafts by ultrasound dilution. *ASAIO J* 1995;**41**:M745—9.
390 Krivitski NM, MacGibbon D, Gleed RD, Dobson A. Accuracy of dilution techniques for access flow measurement during hemodialysis. *Am J Kidney Dis* 1998;**31**:502—8.
391 Yeun JY, Depner TA. Role of access flow measurement. In: Gray RJ, Sands JJ, editors. *Dialysis access: a multidisciplinary approach*. Philadelphia: Lippincott Williams and Wilkins; 2002. p. 119—32.
392 K/DOQI clinical practice guidelines for vascular access: update 2000. *Am J Kidney Dis* 2001;**37**:S137—81.
393 Krivitski N, Schneditz D. Arteriovenous vascular access flow measurement: accuracy and clinical implications. *Contrib Nephrol* 2004;**142**:269—84.
394 Sands J, Glidden D, Jacavage W, Jones B. Difference between delivered and prescribed blood flow in hemodialysis. *ASAIO J* 1996;**42**:M717—9.
395 Bos C, Smits JH, Zijistra JJ, Blankestijn PJ, Bakker CJ, Viergever MA. Underestimation of access flow by ultrasound dilution flow measurements. *Phys Med Biol* 2002;**47**:481—9.
396 Gill RW. Measurement of blood flow by ultrasound: accuracy and sources of error. *Ultrasound Med Biol* 1985;**11**:625—41.
397 Oates CP, Williams ED, McHugh MI. The use of a Diasonics DRF400 duplex ultrasound scanner to measure volume flow in arterio-venous fistulae in patients undergoing haemodialysis: an analysis of measurement uncertainties. *Ultrasound Med Biol* 1990;**16**:571—9.
398 Winkler AJ, Wu J, Case T, Ricci MA. An experimental study of the accuracy of volume flow measurements using commercial ultrasound systems. *J Vasc Technol* 1995;**19**:175—80.
399 Zierler BK, Kirkman TR, Kraiss LW, Reiss WG, Horn JR, Bauer LA, et al. Accuracy of duplex scanning for measurement of arterial volume flow. *J Vasc Surg* 1992;**16**:520—6.
400 Malovrh M. Non-invasive evaluation of vessels by duplex sonography prior to construction of arteriovenous fistulas for haemodialysis. *Nephrol Dial Transplant* 1998;**13**:125—9.
401 Besarab A, Sullivan KL, Ross RP, Moritz MJ. Utility of intra-access pressure monitoring in detecting and correcting venous outlet stenoses prior to thrombosis. *Kidney Int* 1995;**47**:1364—73.
402 Schwab SJ, Raymond JR, Saeed M, Newman GE, Dennis PA, Bollinger RR. Prevention of hemodialysis fistula thrombosis. Early detection of venous stenoses. *Kidney Int* 1989;**36**:707—11.
403 NKF DOQI clinical practice guidelines for vascular access. National Kidney Foundation-Dialysis Outcomes Quality Initiative. *Am J Kidney Dis* 1997;**30**:S150—91.
404 Lok CE, Moist LM. Challenges for randomized controlled trials in nephrology: illustrations in vascular access science and care. *J Nephrol* 2007;**20**:632—45.
405 May RE, Himmelfarb J, Yenicesu M, Knights S, Ikizler TA, Schulman G, et al. Predictive measures of vascular access thrombosis: a prospective study. *Kidney Int* 1997;**52**:1656—62.
406 Neyra NR, Ikizler TA, May RE, Himmelfarb J, Schulman G, Shyr Y, et al. Change in access blood flow over time predicts vascular access thrombosis. *Kidney Int* 1998;**54**:1714—9.
407 Paulson WD, Ram SJ, Work J. Use of vascular access blood flow to evaluate vascular access. *Am J Kidney Dis* 2001;**38**:916.
408 Paulson WD, Ram SJ, Birk CG, Work J. Does blood flow accurately predict thrombosis or failure of hemodialysis synthetic grafts? A meta-analysis. *Am J Kidney Dis* 1999;**34**:478—85.
409 Dember LM, Holmberg EF, Kaufman JS. Value of static venous pressure for predicting arteriovenous graft thrombosis. *Kidney Int* 2002;**61**:1899—904.
410 McDougal G, Agarwal R. Clinical performance characteristics of hemodialysis graft monitoring. *Kidney Int* 2001;**60**:762—6.
411 Paulson WD, Ram SJ, Birk CG, Zapczynski M, Martin SR, Work J. Accuracy of decrease in blood flow in predicting hemodialysis graft thrombosis. *Am J Kidney Dis* 2000;**35**:1089—95.
412 Ram SJ, Nassar R, Work J, Abreo K, Dossabhoy NR, Paulson WD. Risk of hemodialysis graft thrombosis: analysis of monthly flow surveillance. *Am J Kidney Dis* 2008;**52**:930—8.
413 Chang CJ, Ko PJ, Hsu LA, Ko YS, Ko YL, Chen CF, et al. Highly increased cell proliferation activity in the restenotic hemodialysis vascular access after percutaneous transluminal angioplasty: implication in prevention of restenosis. *Am J Kidney Dis* 2004;**43**:74—84.
414 Paulson W, Moist L, Lok C. Vascular access surveillance: case study of a false paradigm. *Semin Dial* 2013;**26**:281—6.
415 Paulson WD, Moist L, Lok CE. Vascular access surveillance: an ongoing controversy. *Kidney Int* 2012;**81**:132—42.
416 Ram SJ, Nassar R, Sharaf R, Magnasco A, Jones SA, Paulson WD. Thresholds for significant decrease in hemodialysis access blood flow. *Semin Dial* 2005;**18**:558—64.
417 White JJ, Jones SA, Ram SJ, Schwab SJ, Paulson WD. Mathematical model demonstrates influence of luminal diameters on venous pressure surveillance. *Clin J Am Soc Nephrol CJASN* 2007;**2**:681—7.
418 Besarab A, Sherman R. The relationship of recirculation to access blood flow. *Am J Kidney Dis* 1997;**29**:223—9.
419 Hester RL, Curry E, Bower J. The determination of hemodialysis blood recirculation using blood urea nitrogen measurements. *Am J Kidney Dis* 1992;**20**:598—602.
420 Lindsay RM, Bradfield E, Rothera C, Kianfar C, Malek P, Blake PG. A comparison of methods for the measurement of hemodialysis access recirculation and access blood flow rate. *ASAIO J* 1998;**44**:62—7.
421 Ugawa T, Sakurama K, Yorifuji T, Takaoka M, Fujiwara Y, Kabashima N, et al. Evaluating the need for and effect of percutaneous transluminal angioplasty on arteriovenous fistulas by using total recirculation rate per dialysis session ("clearance gap"). *Acta Med Okayama* 2012;**66**:443—7.
422 Acchiardo SR, Hatten KW, Ruvinsky MJ, Dyson B, Fuller J, Moore LW. Inadequate dialysis increases gross mortality rate. *ASAIO J* 1992;**38**:M282—5.
423 Collins AJ, Ma JZ, Umen A, Keshaviah P. Urea index and other predictors of hemodialysis patient survival. *Am J Kidney Dis* 1994;**23**:272—82.
424 Parker 3rd TF, Husni L, Huang W, Lew N, Lowrie EG. Survival of hemodialysis patients in the United States is improved with a greater quantity of dialysis. *Am J Kidney Dis* 1994;**23**:670—80.
425 Windus DW, Audrain J, Vanderson R, Jendrisak MD, Picus D, Delmez JA. Optimization of high-efficiency hemodialysis by detection and correction of fistula dysfunction. *Kidney Int* 1990;**38**:337—41.
426 Sehgal AR, Dor A, Tsai AC. Morbidity and cost implications of inadequate hemodialysis. *Am J Kidney Dis* 2001;**37**:1223—31.
427 Tessitore N, Mansueto G, Bedogna V, Lipari G, Poli A, Gammaro L, et al. A prospective controlled trial on effect of percutaneous transluminal angioplasty on functioning arteriovenous fistulae survival. *J Am Soc Nephrol* 2003;**14**:1623—7.

428 McCarley P, Wingard RL, Shyr Y, Pettus W, Hakim RM, Ikizler TA. Vascular access blood flow monitoring reduces access morbidity and costs. *Kidney Int* 2001;**60**:1164—72.
429 Smits JH, van der Linden J, Hagen EC, Modderkolk-Cammeraat EC, Feith GW, Koomans HA, et al. Graft surveillance: venous pressure, access flow, or the combination? *Kidney Int* 2001;**59**:1551—8.
430 Tonelli M, Jhangri GS, Hirsch DJ, Marryatt J, Mossop P, Wile C, et al. Best threshold for diagnosis of stenosis or thrombosis within six months of access flow measurement in arteriovenous fistulae. *J Am Soc Nephrol* 2003;**14**:3264—9.
431 Wertman R, Altun E, Martin DR, Mitchell DG, Leyendecker JR, O'Malley RB, et al. Risk of nephrogenic systemic fibrosis: evaluation of gadolinium chelate contrast agents at four American universities. *Radiology* 2008;**248**:799—806.
432 Chopra T, Kandukurti K, Shah S, Ahmed R, Panesar M. Understanding nephrogenic systemic fibrosis. *Int J Nephrol* 2012;**2012**:912189.
433 Maples DC. Nurses' impact on the choice and longevity of vascular access. *Nephrol Nurs J* 2005;**32**:670—4.
434 Joseph AM. The impact of nursing on patient and organizational outcomes. *Nurs Econ* 2007;**25**:30—4. 3.
435 Van Waeleghem JP, Elseviers MM, Lindley EJ. Management of vascular access in Europe. Part 1-A study of center based policies. *Nephrol News Issues* 2000;**14**:30—4.
436 Elseviers M, De Vos JY, Harrington M, Zampieron A, Ormandy P, Kafkia T. Comparison of renal care practice in Europe: centre and patient characteristics. *EDTNA ERCA J* 2006;**32**:8—13.
437 Zampieron A, Elseviers M, Ormandy P, Vlaminck H, De Vos JY, Kafkia T, et al. Development of indicators to measure European variation of nursing activities. *EDTNA ERCA J* 2006;**32**:14—9.
438 Saran R, Pisoni RL, Weitzel WF. Epidemiology of vascular access for hemodialysis and related practice patterns. *Contrib Nephrol* 2004;**142**:14—28.
439 Rutherford MM. The how, what, and why of valuation and nursing. *Nurs Econ* 2008;**26**:347—51. 83; quiz 52.
440 Elizabeth JL, Hanna L, Walker D, Milo E, Koupatsiaris T, De Vos JY, et al. Pre-dialysis education and patient choice. *J Ren Care* 2006;**32**:214—20.
441 Da Silva-Gane M, Goovaerts T, Elseviers MM, Lindley EJ. Information and counselling for patients approaching end-stage renal failure in selected centres across Europe. *EDTNA ERCA J* 2002;**28**:49—55.
442 McCann M, Einarsdottir H, Van Waeleghem JP, Murphy F, Sedgewick J. Vascular access management III: central venous catheters. *J Ren Care* 2010;**36**:25—33.
443 Li PK, Cheung WL, Lui SL, Blagg C, Cass A, Hooi LS, et al. Increasing home-based dialysis therapies to tackle dialysis burden around the world: a position statement on dialysis economics from the 2nd Congress of the International Society for Hemodialysis. *Hemodial Int* 2011;**15**:10—4.
444 Achinger SG, Ikizler TA, Bian A, Shintani A, Ayus JC. Long-term effects of daily hemodialysis on vascular access outcomes: a prospective controlled study. *Hemodial Int* 2013;**17**:208—15.
445 Sadala ML, Miranda MG, Lorencon M, de Campos Pereira EP. Nurse-patient communication while performing home dialysis: the patients' perceptions. *J Ren Care* 2010;**36**:34—40.
446 Hawley CM, Jeffries J, Nearhos J, Van Eps C. Complications of home hemodialysis. *Hemodial Int* 2008;**12**:S21—5.
447 Van Waeleghem JP, Chamney M, Lindley EJ, Pancirova J. Venous needle dislodgement: how to minimise the risks. *J Ren Care* 2008;**34**:163—8.
448 Chien CC, Chou HK, Hung ST. A conceptual model of nurses' goal orientation, service behavior, and service performance. *Nurs Econ* 2008;**26**:374—83.
449 Gardner JK, Thomas-Hawkins C, Fogg L, Latham CE. The relationships between nurses' perceptions of the hemodialysis unit work environment and nurse turnover, patient satisfaction, and hospitalizations. *Nephrol Nurs J* 2007;**34**:271—81. quiz 82.
450 Way M, MacNeil M. Organizational characteristics and their effect on health. *Nurs Econ* 2006;**24**:67—76. 55.
451 Shaver KH, Lacey LM. Job and career satisfaction among staff nurses: effects of job setting and environment. *J Nurs Adm* 2003;**33**:166—72.
452 Wieck KL, Dols J, Landrum P. Retention priorities for the intergenerational nurse workforce. *Nurs Forum* 2010;**45**:7—17.
453 Bowers L. The significance of primary nursing. *J Adv Nurs* 1989;**14**:13—9.
454 Steele DJ, Hamilton E, Arnaout MA. A case management model to improve hemodialysis outpatient outcomes. *Hemodial Int* 2007;**11**:247—51.
455 Harwood L, Ridley J, Lawrence-Murphy JA, Spence-Laschinger HK, White S, Bevan J, et al. Nurses' perceptions of the impact of a renal nursing professional practice model on nursing outcomes, characteristics of practice environments and empowerment—Part I. *CANNT J = J ACITN* 2007;**17**:22—9.
456 Harwood L, Ridley J, Lawrence-Murphy JA, White S, Spence-Laschinger HK, Bevan J, et al. Nurses' perceptions of the impact of a renal nursing professional practice model on nursing outcomes, characteristics of practice environments and empowerment—Part II. *CANNT J = J ACITN* 2007;**17**:35—43.
457 Neyhart CD, McCoy L, Rodegast B, Gilet CA, Roberts C, Downes K. A new nursing model for the care of patients with chronic kidney disease: the UNC Kidney Center Nephrology Nursing Initiative. *Nephrol Nurs J* 2010;**37**:121—30. quiz 31.
458 Dobson S, Tranter S. Organizing the work: choosing the most effective way to deliver nusing care in a hospital hemodialysis unit. *Ren Soc Australas J* 2008;**4**:59—63.
459 Lewis CK. The clinical nurse specialist's role as coach in a clinical practice development model. *J Vasc Nurs* 1996;**14**:48—52.
460 Langstaff D, Gray B. Flexible roles: a new model in nursing practice. *Br J Nurs* 1997;**6**:635—8.
461 Dingemanse SE, Eliens AM, Tol E. Primary nursing in Diatel: investigation of the functioning of primary nursing. *EDTNA ERCA J* 1997;**23**:30—3.
462 Castro Palma EE, Salamanca Catoni MI, Mantuliz Arechabala MC, Henriquez Brantes LX. Primary nursing para el Cuidado de Usuarios en Hemodiálisis Crónica. *Hisp Health Care Int* 2006;**4**:203—10.
463 Flett A. Introducing primary nursing to a satellite dialysis setting in Singapore. *EDTNA ERCA J* 1997;**23**:41—3.
464 Bajardi P, Bergia R, Bardone L. Risk management in nephrology. *G Ital Nefrol* 2009;**26**:534—43.
465 Holley JL. A descriptive report of errors and adverse events in chronic hemodialysis units. *Nephrol News Issues* 2006;**20**:57—8. 60-1, 3 passim.
466 McClellan WM, Goldman RS. Continuous quality improvement in dialysis units: basic tools. *Adv Ren Replace Ther* 2001;**8**:95—103.
467 Bonfant G, Belfanti P, Paternoster G, Gabrielli D, Gaiter AM, Manes M, et al. Clinical risk analysis with failure mode and effect analysis (FMEA) model in a dialysis unit. *J Nephrol* 2010;**23**:111—8.

468 Thomas A. Revisiting quality standards in hemodialysis vascular access: where is the bar? *CANNT J = J ACITN* 2005;**15**:30—3. 6-41; quiz 34—35.
469 Wilson B, Harwood L, Oudshoorn A, Thompson B. The culture of vascular access cannulation among nurses in a chronic hemodialysis unit. *CANNT J = J ACITN* 2010;**20**:35—42.
470 Carlton D. The vascular access coordinator role: an interview with Donna Carlton by Betsy King. *Nephrol Nurs J* 2005;**32**: 688—90.
471 King B, Miller D. Hemodialysis special interest group networking session: improving vascular access cannulation skills. *Nephrol Nurs J* 2004;**31**. 688, 90.
472 Sinclair P, Schoch M, Black K, Woods M. Proof of concept: Developing a peer reviewed, evidence-based, interactive e-learning programme. *J Ren Care* 2011;**37**:108—13.
473 Dinwiddie LC. Investing in the lifeline: the value of a vascular access coordinator. *Nephrol News Issues* 2003;**17**. 49, 52—53.
474 Thomas N, Küntzle W, McCann M, editors. *The European core curriculum for a post-basic course in nephrology nursing*. 2nd ed. Luzern: EDTNA/ERCA; 2004.
475 Kumbar L. Complications of arteriovenous fistulae: beyond venous stenosis. *Adv Chronic Kidney Dis* 2012;**19**:195—201.
476 Clinical practice guidelines for hemodialysis adequacy, update 2006. *Am J Kidney Dis* 2006;**48**:S2—90.
477 Georgiadis GS, Lazarides MK, Panagoutsos SA, Kantartzi KM, Lambidis CD, Staramos DN, et al. Surgical revision of complicated false and true vascular access-related aneurysms. *J Vasc Surg* 2008;**47**:1284—91.
478 Allaria PM, Costantini E, Lucatello A, Gandini E, Caligara F, Giangrande A. Aneurysm of arteriovenous fistula in uremic patients: is endograft a viable therapeutic approach? *J Vasc Access* 2002;**3**:85—8.
479 Shah AS, Valdes J, Charlton-Ouw KM, Chen Z, Coogan SM, Amer HM, et al. Endovascular treatment of hemodialysis access pseudoaneurysms. *J Vasc Surg* 2012;**55**:1058—62.
480 Silas AM, Bettmann MA. Utility of covered stents for revision of aging failing synthetic hemodialysis grafts: a report of three cases. *Cardiovasc Intervent Radiol* 2003;**26**:550—3.
481 Spergel LM, Ravani P, Roy-Chaudhury P, Asif A, Besarab A. Surgical salvage of the autogenous arteriovenous fistula (AVF). *J Nephrol* 2007;**20**:388—98.
482 Delorme JM, Guidoin R, Canizales S, Charara J, How T, Marois Y, et al. Vascular access for hemodialysis: pathologic features of surgically excised ePTFE grafts. *Ann Vasc Surg* 1992;**6**:517—24.
483 Berard X, Brizzi V, Mayeux S, Sassoust G, Biscay D, Ducasse E, et al. Salvage treatment for venous aneurysm complicating vascular access arteriovenous fistula: use of an exoprosthesis to reinforce the vein after aneurysmorrhaphy. *Eur J Vasc Endovasc Surg* 2010;**40**:100—6.
484 Barshes NR, Annambhotla S, Bechara C, Kougias P, Huynh TT, Dardik A, et al. Endovascular repair of hemodialysis graft-related pseudoaneurysm: an alternative treatment strategy in salvaging failing dialysis access. *Vasc Endovasc Surg* 2008;**42**:228—34.
485 Hausegger KA, Tiessenhausen K, Klimpfinger M, Raith J, Hauser H, Tauss J. Aneurysms of hemodialysis access grafts: treatment with covered stents: a report of three cases. *Cardiovasc Intervent Radiol* 1998;**21**:334—7.
486 Moszkowicz A, Behrens G, Gueyikian S, Patel NH, Ferral H. Occlusion of a rapidly expanding hemodialysis graft pseudoaneurysm with placement of a stent graft. *Semin Intervent Radiol* 2007;**24**:34—7.
487 Najibi S, Bush RL, Terramani TT, Chaikof EL, Gunnoud AB, Lumsden AB, et al. Covered stent exclusion of dialysis access pseudoaneurysms. *J Surg Res* 2002;**106**:15—9.
488 Ryan JM, Dumbleton SA, Doherty J, Smith TP. Technical innovation. Using a covered stent (wallgraft) to treat pseudoaneurysms of dialysis grafts and fistulas. *AJR Am J Roentgenol* 2003;**180**:1067—71.
489 Sapoval MR, Turmel-Rodrigues LA, Raynaud AC, Bourquelot P, Rodrigue H, Gaux JC. Cragg covered stents in hemodialysis access: initial and midterm results. *J Vasc Intervent Radiol JVIR* 1996;**7**:335—42.
490 Vesely TM. Use of stent grafts to repair hemodialysis graft-related pseudoaneurysms. *J Vasc Intervent Radiol JVIR* 2005;**16**:1301—7.
491 Shemesh D, Goldin I, Zaghal I, Berelowitz D, Verstandig AG, Olsha O. Stent graft treatment for hemodialysis access aneurysms. *J Vasc Surg* 2011;**54**:1088—94.
492 Pandolfe LR, Malamis AP, Pierce K, Borge MA. Treatment of hemodialysis graft pseudoaneurysms with stent grafts: institutional experience and review of the literature. *Semin Intervent Radiol* 2009;**26**:89—95.
493 Charara J, Guidoin R, Gill F, Guzman R. Morphologic assessment of ePTFE graft wall damage following hemodialysis needle punctures. *J Appl Biomater* 1990;**1**:279—87.
494 Gulati S, Sahu KM, Avula S, Sharma RK, Ayyagiri A, Pandey CM. Role of vascular access as a risk factor for infections in hemodialysis. *Ren Fail* 2003;**25**:967—73.
495 Li PK, Chow KM. Infectious complications in dialysis—epidemiology and outcomes. *Nat Rev Nephrol* 2012;**8**:77—88.
496 Ayus JC, Sheikh-Hamad D. Silent infection in clotted hemodialysis access grafts. *J Am Soc Nephrol* 1998;**9**:1314—7.
497 Kessler M, Hoen B, Mayeux D, Hestin D, Fontenaille C. Bacteremia in patients on chronic hemodialysis. A multicenter prospective survey. *Nephron* 1993;**64**:95—100.
498 Lentino JR, Baddour LM, Wray M, Wong ES, Yu VL. *Staphylococcus aureus* and other bacteremias in hemodialysis patients: antibiotic therapy and surgical removal of access site. *Infection* 2000;**28**:355—60.
499 Anderson JE, Chang AS, Anstadt MP. Polytetrafluoroethylene hemoaccess site infections. *ASAIO J* 2000;**46**:S18—21.
500 Reed SD, Friedman JY, Engemann JJ, Griffiths RI, Anstrom KJ, Kaye KS, et al. Costs and outcomes among hemodialysis-dependent patients with methicillin-resistant or methicillin-susceptible *Staphylococcus aureus* bacteremia. *Infect Contr Hosp Epidemiol* 2005;**26**:175—83.
501 Selvey LA, Whitby M, Johnson B. Nosocomial methicillin-resistant *Staphylococcus aureus* bacteremia: is it any worse than nosocomial methicillin-sensitive *Staphylococcus aureus* bacteremia? *Infect Contr Hosp Epidemiol* 2000;**21**:645—8.
502 Fokou M, Teyang A, Ashuntantang G, Kaze F, Eyenga VC, Chichom Mefire A, et al. Complications of arteriovenous fistula for hemodialysis: an 8-year study. *Ann Vasc Surg* 2012;**26**: 680—4.
503 Curi MA, Pappas PJ, Silva Jr MB, Patel S, Padberg Jr FT, Jamil Z, et al. Hemodialysis access: influence of the human immunodeficiency virus on patency and infection rates. *J Vasc Surg* 1999;**29**:608—16.
504 Calligaro KD, Veith FJ, Gupta SK, Ascer E, Dietzek AM, Franco CD, et al. A modified method for management of prosthetic graft infections involving an anastomosis to the common femoral artery. *J Vasc Surg* 1990;**11**:485—92.
505 Schanzer A, Ciaranello AL, Schanzer H. Brachial artery ligation with total graft excision is a safe and effective approach to

prosthetic arteriovenous graft infections. *J Vasc Surg* 2008;**48**: 655—8.
506 Deneuville M. Infection of PTFE grafts used to create arteriovenous fistulas for hemodialysis access. *Ann Vasc Surg* 2000;**14**:473—9.
507 Schutte WP, Helmer SD, Salazar L, Smith JL. Surgical treatment of infected prosthetic dialysis arteriovenous grafts: total versus partial graft excision. *Am J Surg* 2007;**193**:385—8. discussion 8.
508 Schwab DP, Taylor SM, Cull DL, Langan 3rd EM, Snyder BA, Sullivan TM, et al. Isolated arteriovenous dialysis access graft segment infection: the results of segmental bypass and partial graft excision. *Ann Vasc Surg* 2000;**14**:63—6.
509 Tabbara MR, O'Hara PJ, Hertzer NR, Krajewski LP, Beven EG. Surgical management of infected PTFE hemodialysis grafts: analysis of a 15-year experience. *Ann Vasc Surg* 1995;**9**:378—84.
510 Walz P, Ladowski JS. Partial excision of infected fistula results in increased patency at the cost of increased risk of recurrent infection. *Ann Vasc Surg* 2005;**19**:84—9.
511 Vallet C, Saucy F, Haller C, Meier P, Rafoul W, Corpataux JM. Vacuum-assisted conservative treatment for the management and salvage of exposed prosthetic hemodialysis access. *Eur J Vasc Endovasc Surg* 2004;**28**:397—9.
512 Turmel-Rodrigues L, Pengloan J, Baudin S, Testou D, Abaza M, Dahdah G, et al. Treatment of stenosis and thrombosis in haemodialysis fistulas and grafts by interventional radiology. *Nephrol Dial Transplant* 2000;**15**:2029—36.
513 Dember LM, Holmberg EF, Kaufman JS. Randomized controlled trial of prophylactic repair of hemodialysis arteriovenous graft stenosis. *Kidney Int* 2004;**66**:390—8.
514 Lumsden AB, MacDonald MJ, Isiklar H, Martin LG, Kikeri D, Harker LA, et al. Central venous stenosis in the hemodialysis patient: incidence and efficacy of endovascular treatment. *Cardiovasc Surg* 1997;**5**:504—9.
515 Moist LM, Churchill DN, House AA, Millward SF, Elliott JE, Kribs SW, et al. Regular monitoring of access flow compared with monitoring of venous pressure fails to improve graft survival. *J Am Soc Nephrol* 2003;**14**:2645—53.
516 Work J. Role of access surveillance and preemptive intervention. *Semin Vasc Surg* 2011;**24**:137—42.
517 Ravani P, Quinn RR, Oliver MJ, Karsanji DJ, James MT, MacRae JM, et al. Pre-emptive correction for haemodialysis arteriovenous access stenosis. *Cochrane Database Syst Rev* 2016;**1**:CD010709.
518 Asif A, Gadalean FN, Merrill D, Cherla G, Cipleu CD, Epstein DL, et al. Inflow stenosis in arteriovenous fistulas and grafts: a multicenter, prospective study. *Kidney Int* 2005;**67**: 1986—92.
519 Duijm LE, Liem YS, van der Rijt RH, Nobrega FJ, van den Bosch HC, Douwes-Draaijer P, et al. Inflow stenoses in dysfunctional hemodialysis access fistulae and grafts. *Am J Kidney Dis* 2006;**48**:98—105.
520 Jimenez-Almonacid P, Gruss-Vergara E, Jimenez-Toscano M, Lasala M, Rueda JA, Portoles J, et al. Surgical treatment of juxta-anastomotic stenosis in radiocephalic fistula. A new proximal radiocephalic anastomosis. *Nefrologia* 2012;**32**:517—22.
521 Mortamais J, Papillard M, Girouin N, Boutier R, Cougnaud L, Martin X, et al. Endovascular treatment of juxta-anastomotic venous stenoses of forearm radiocephalic fistulas: long-term results and prognostic factors. *J Vasc Intervent Radiol JVIR* 2013;**24**:558—64. quiz 65.
522 Tessitore N, Mansueto G, Lipari G, Bedogna V, Tardivo S, Baggio E, et al. Endovascular versus surgical preemptive repair of forearm arteriovenous fistula juxta-anastomotic stenosis: analysis of data collected prospectively from 1999 to 2004. *Clin J Am Soc Nephrol CJASN* 2006;**1**:448—54.
523 Napoli M, Prudenzano R, Russo F, Antonaci AL, Aprile M, Buongiorno E. Juxta-anastomotic stenosis of native arteriovenous fistulas: surgical treatment versus percutaneous transluminal angioplasty. *J Vasc Access* 2010;**11**:346—51.
524 Peregrin JH, Rocek M. Results of a peripheral cutting balloon prospective multicenter European registry in hemodialysis vascular access. *Cardiovasc Intervent Radiol* 2007;**30**:212—5.
525 Beathard GA. Gianturco self-expanding stent in the treatment of stenosis in dialysis access grafts. *Kidney Int* 1993;**43**:872—7 (8479123).
526 Quinn SF, Schuman ES, Demlow TA, Standage BA, Ragsdale JW, Green GS, et al. Percutaneous transluminal angioplasty versus endovascular stent placement in the treatment of venous stenoses in patients undergoing hemodialysis: intermediate results. *J Vasc Intervent Radiol JVIR* 1995;**6**:851—5.
527 Hoffer EK, Sultan S, Herskowitz MM, Daniels ID, Sclafani SJ. Prospective randomized trial of a metallic intravascular stent in hemodialysis graft maintenance. *J Vasc Intervent Radiol JVIR* 1997;**8**:965—73.
528 Clark TWI, Hirsch DA, Jindal KJ, Veugelers PJ, LeBlanc J. Outcome and prognostic factors of restenosis after percutaneous treatment of native hemodialysis fistulas. *J Vasc Intervent Radiol JVIR* 2002;**13**:51—9 (11788695).
529 Vogel PM, Parise C. SMART stent for salvage of hemodialysis access grafts. *J Vasc Intervent Radiol JVIR* 2004;**15**:1051—60.
530 Vogel PM, Parise C. Comparison of SMART stent placement for arteriovenous graft salvage versus successful graft PTA. *J Vasc Intervent Radiol JVIR* 2005;**16**:1619—26.
531 Dolmatch B, Dong YH, Heeter Z. Evaluation of three polytetrafluoroethylene stent-grafts in a model of neointimal hyperplasia. *J Vasc Intervent Radiol JVIR* 2007;**18**:527—34.
532 Haskal ZJ, Saad TF, Hoggard JG, Cooper RI, Lipkowitz GS, Gerges A, et al. Prospective, randomized, concurrently-controlled study of a stent graft versus balloon angioplasty for treatment of arteriovenous access graft stenosis: 2-year results of the RENOVA Study. *J Vasc Intervent Radiol JVIR* 2016;**27**:1105—14. e3.
533 Falk A, Maya ID, Yevzlin AS. RESCUE Investigators. A prospective, randomized study of an expanded polytetrafluoroethylene stent graft versus balloon angioplasty for in-stent restenosis in arteriovenous grafts and fistulae: two-year results of the RESCUE Study. *J Vasc Interv Radiol* 2016;**27**:1465—76.
534 Salman L, Asif A. Stent graft for nephrologists: concerns and consensus. *Clin J Am Soc Nephrol CJASN* 2010;**5**:1347—52.
535 Rajan DK, Clark TWI, Patel NK, Stavropoulos SW, Simons ME. Prevalence and treatment of cephalic arch stenosis in dysfunctional autogenous hemodialysis fistulas. *J Vasc Intervent Radiol JVIR* 2003;**14**:567—73 (12761309).
536 Rajan DK, Falk A. A randomized prospective study comparing outcomes of angioplasty versus VIABAHN stent-graft placement for cephalic arch stenosis in dysfunctional hemodialysis accesses. *J Vasc Intervent Radiol JVIR* 2015;**26**:1355—61.
537 Roy-Chaudhury P, Arend L, Zhang J, Krishnamoorthy M, Wang Y, Banerjee R, et al. Neointimal hyperplasia in early arteriovenous fistula failure. *Am J Kidney Dis* 2007;**50**:782—90.
538 Shemesh D, Goldin I, Zaghal I, Berlowitz D, Raveh D, Olsha O. Angioplasty with stent graft versus bare stent for recurrent cephalic arch stenosis in autogenous arteriovenous access for hemodialysis: a prospective randomized clinical trial. *J Vasc Surg* 2008;**48**:1524—31. e1—2.

539 Patanè D, Giuffrida S, Morale W, L'Anfusa G, Pulliatti D, Bisceglie P, et al. Drug-eluting balloon for the treatment of failing hemodialytic radiocephalic arteriovenous fistulas: our experience in the treatment of juxta-anastomotic stenoses. *J Vasc Access* 2014;**15**:338–43.
540 Portugaller RH, Kalmar PI, Deutschmann H. The eternal tale of dialysis access vessels and restenosis: are drug-eluting balloons the solution? *J Vasc Access* 2014;**15**:439–47.
541 Kitrou PM, Katsanos K, Spiliopoulos S, Karnabatidis D, Siablis D. Drug-eluting versus plain balloon angioplasty for the treatment of failing dialysis access: final results and cost-effectiveness analysis from a prospective randomized controlled trial (NCT01174472). *Eur J Radiol* 2015;**84**:418–23.
542 Chen JC, Kamal DM, Jastrzebski J, Taylor DC. Venovenostomy for outflow venous obstruction in patients with upper extremity autogenous hemodialysis arteriovenous access. *Ann Vasc Surg* 2005;**19**:629–35.
543 Kian K, Asif A. Cephalic arch stenosis. *Semin Dial* 2008;**21**: 78–82.
544 Kian K, Unger SW, Mishler R, Schon D, Lenz O, Asif A. Role of surgical intervention for cephalic arch stenosis in the "fistula first" era. *Semin Dial* 2008;**21**:93–6.
545 Wang S, Almehmi A, Asif A. Surgical management of cephalic arch occlusive lesions: are there predictors for outcomes? *Semin Dial* 2013;**26**:E33–41.
546 Puskar D, Pasini J, Savic I, Bedalov G, Sonicki Z. Survival of primary arteriovenous fistula in 463 patients on chronic hemodialysis. *Croat Med J* 2002;**43**:306–11.
547 Haage P, Vorwerk D, Wildberger JE, Piroth W, Schurmann K, Gunther RW. Percutaneous treatment of thrombosed primary arteriovenous hemodialysis access fistulae. *Kidney Int* 2000;**57**:1169–75.
548 Turmel-Rodrigues L, Pengloan J, Rodrigue H, Brillet G, Lataste A, Pierre D, et al. Treatment of failed native arteriovenous fistulae for hemodialysis by interventional radiology. *Kidney Int* 2000;**57**:1124–40.
549 Andriani M, Drago G, Bernardi AM, Da Porto A, De Luca M, Riegler P, et al. Recombinant tissue plasminogen activator (rt-PA) as first-line therapy for declotting of haemodialysis access. *Nephrol Dial Transplant* 1995;**10**:1714–9.
550 Trerotola SO, Vesely TM, Lund GB, Soulen MC, Ehrman KO, Cardella JF. Treatment of thrombosed hemodialysis access grafts: arrow-trerotola percutaneous thrombolytic device versus pulse-spray thrombolysis. Arrow-trerotola percutaneous thrombolytic device clinical trial. *Radiology* 1998;**206**:403–14.
551 Vorwerk D, Schurmann K, Muller-Leisse C, Adam G, Bucker A, Sohn M, et al. Hydrodynamic thrombectomy of haemodialysis grafts and fistulae: results of 51 procedures. *Nephrol Dial Transplant* 1996;**11**:1058–64.
552 Crikis S, Lee D, Brooks M, Power DA, Ierino FL, Levidiotis V. Predictors of early dialysis vascular-access failure after thrombolysis. *Am J Nephrol* 2008;**28**:181–9.
553 Smits HF, Smits JH, Wust AF, Buskens E, Blankestijn PJ. Percutaneous thrombolysis of thrombosed haemodialysis access grafts: comparison of three mechanical devices. *Nephrol Dial Transplant* 2002;**17**:467–73.
554 Uflacker R, Rajagopalan PR, Selby JB, Hannegan C. Thrombosed dialysis access grafts: randomized comparison of the Amplatz thrombectomy device and surgical thromboembolectomy. *Eur Radiol* 2004;**14**:2009–14.
555 Agarwal AK, Patel BM, Haddad NJ. Central vein stenosis: a nephrologist's perspective. *Semin Dial* 2007;**20**:53–62 (17244123).
556 Hernandez D, Diaz F, Rufino M, Lorenzo V, Perez T, Rodriguez A, et al. Subclavian vascular access stenosis in dialysis patients: natural history and risk factors. *J Am Soc Nephrol* 1998;**9**:1507–10.
557 Kundu S. Review of central venous disease in hemodialysis patients. *J Vasc Intervent Radiol JVIR* 2010;**21**:963–8.
558 Bakken AM, Protack CD, Saad WE, Lee DE, Waldman DL, Davies MG. Long-term outcomes of primary angioplasty and primary stenting of central venous stenosis in hemodialysis patients. *J Vasc Surg* 2007;**45**:776–83 (17398386).
559 Bozof R, Kats M, Barker J, Allon M. Time to symptomatic vascular stenosis at different locations in patients with arteriovenous grafts. *Semin Dial* 2008;**21**:285–8 (18397203).
560 Criado E, Marston WA, Jaques PF, Mauro MA, Keagy BA. Proximal venous outflow obstruction in patients with upper extremity arteriovenous dialysis access. *Ann Vasc Surg* 1994;**8**:530–5 (7865390).
561 Labropoulos N, Borge M, Pierce K, Pappas PJ. Criteria for defining significant central vein stenosis with duplex ultrasound. *J Vasc Surg* 2007;**46**:101–7 (17540535).
562 Mickley V. Central vein obstruction in vascular access. *Eur J Vasc Endovasc Surg* 2006;**32**:439–44 (16765068).
563 Renaud CJ, Francois M, Nony A, Fodil-Cherif M, Turmel-Rodrigues L. Comparative outcomes of treated symptomatic versus non-treated asymptomatic high-grade central vein stenoses in the outflow of predominantly dialysis fistulas. *Nephrol Dial Transplant* 2012;**27**:1631–8.
564 Davidson CJ, Newman GE, Sheikh KH, Kisslo K, Stack RS, Schwab SJ. Mechanisms of angioplasty in hemodialysis fistula stenoses evaluated by intravascular ultrasound. *Kidney Int* 1991;**40**:91–5 (1833583).
565 Ronald J, Davis B, Guevara CJ, Pabon-Ramos WM, Smith TP, Kim CY. Treatment of central venous in-stent restenosis with repeat stent deployment in hemodialysis patients. *J Vasc Access* 2017;**18**:214–9.
566 Maya ID, Saddekni S, Allon M. Treatment of refractory central vein stenosis in hemodialysis patients with stents. *Semin Dial* 2007;**20**:78–82.
567 Kim YC, Won JY, Choi SY, Ko H-K, Lee K-H, Lee DY, et al. Percutaneous treatment of central venous stenosis in hemodialysis patients: long-term outcomes. *Cardiovasc Intervent Radiol* 2009;**32**:271–8 (19194745).
568 Ozyer U, Harman A, Yildirim E, Aytekin C, Karakayali F, Boyvat F. Long-term results of angioplasty and stent placement for treatment of central venous obstruction in 126 hemodialysis patients: a 10-year single-center experience. *AJR Am J Roentgenol* 2009;**193**:1672–9 (19933663).
569 Quaretti P, Galli F, Moramarco LP, Corti R, Leati G, Fiorina I, et al. Stent grafts provided superior primary patency for central venous stenosis treatment in comparison with angioplasty and bare metal stent: a retrospective single center study on 70 hemodialysis patients. *Vasc Endovasc Surg* 2016;**50**:221–30.
570 de Graaf R, van Laanen J, Sailer A, Tordoir J. Long segment recanalization and dedicated central venous stenting in an ultimate attempt to restore vascular access central vein outflow. *J Vasc Access* 2014;**15**:S109–13.
571 Anaya-Ayala JE, Smolock CJ, Colvard BD, Naoum JJ, Bismuth J, Lumsden AB, et al. Efficacy of covered stent placement for central venous occlusive disease in hemodialysis patients. *J Vasc Surg* 2011;**54**:754–9.
572 Kundu S, Modabber M, You JM, Tam P, Nagai G, Ting R. Use of PTFE stent grafts for hemodialysis-related central venous

occlusions: intermediate-term results. *Cardiovasc Intervent Radiol* 2011;**34**:949–57 (21069331).
573 Turmel-Rodrigues L, Bourquelot P, Raynaud A, Sapoval M. Primary stent placement in hemodialysis-related central venous stenoses: the dangers of a potential "radiologic dictatorship". *Radiology* 2000;**217**:600–2.
574 Verstandig AG, Berelowitz D, Zaghal I, Goldin I, Olsha O, Shamieh B, et al. Stent grafts for central venous occlusive disease in patients with ipsilateral hemodialysis access. *J Vasc Intervent Radiol JVIR* 2013;**24**:1280–7. quiz 8.
575 Anaya-Ayala JE, Bellows PH, Ismail N, Cheema ZF, Naoum JJ, Bismuth J, et al. Surgical management of hemodialysis-related central venous occlusive disease: a treatment algorithm. *Ann Vasc Surg* 2011;**25**:108–19 (21172586).
576 Hamish M, Shalhoub J, Rodd CD, Davies AH. Axillo-iliac conduit for haemodialysis vascular access. *Eur J Vasc Endovasc Surg* 2006;**31**:530–4 (16427332).
577 Kavallieratos N, Kokkinos A, Kalocheretis P. Axillary to saphenous vein bypass for treatment of central venous obstruction in patients receiving dialysis. *J Vasc Surg* 2004;**40**:640–3.
578 Chandler NM, Mistry BM, Garvin PJ. Surgical bypass for subclavian vein occlusion in hemodialysis patients. *J Am Coll Surg* 2002;**194**:416–21.
579 Suliman A, Greenberg JI, Angle N. Surgical bypass of symptomatic central venous obstruction for arteriovenous fistula salvage in hemodialysis patients. *Ann Vasc Surg* 2008;**22**:203–9.
580 Jennings WC, Miller GA, Coburn MZ, Howard CA, Lawless MA. Vascular access flow reduction for arteriovenous fistula salvage in symptomatic patients with central venous occlusion. *J Vasc Access* 2012;**13**:157–62 (21983828).
581 Oguzkurt L, Tercan F, Yildirim S, Torun D. Central venous stenosis in haemodialysis patients without a previous history of catheter placement. *Eur J Radiol* 2005;**55**:237–42.
582 Thompson JF, Winterborn RJ, Bays S, White H, Kinsella DC, Watkinson AF. Venous thoracic outlet compression and the Paget-Schroetter syndrome: a review and recommendations for management. *Cardiovasc Intervent Radiol* 2011;**34**:903–10.
583 Urschel Jr HC, Patel AN. Paget-Schroetter syndrome therapy: failure of intravenous stents. *Ann Thorac Surg* 2003;**75**:1693–6. discussion 6.
584 Glass C, Dugan M, Gillespie D, Doyle A, Illig K. Costoclavicular venous decompression in patients with threatened arteriovenous hemodialysis access. *Ann Vasc Surg* 2011;**25**:640–5.
585 Gertler JP. Decompression of the occluded subclavian vein in the patient with ipsilateral threatened access by transposition of the internal jugular vein. *ASAIO J* 1995;**41**:896–8.
586 Bachleda P, Utikal P, Kalinova L, Drac P, Zadrazil J, Koecher M, et al. Operating management of central venous hypertension complicating upper extremity dialysis access. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub* 2008;**152**:155–8.
587 Malik J, Tuka V, Kasalova Z, Chytilova E, Slavikova M, Clagett P, et al. Understanding the dialysis access steal syndrome. A review of the etiologies, diagnosis, prevention and treatment strategies. *J Vasc Access* 2008;**9**:155–66.
588 Miles AM. Vascular steal syndrome and ischaemic monomelic neuropathy: two variants of upper limb ischaemia after haemodialysis vascular access surgery. *Nephrol Dial Transplant* 1999;**14**:297–300 (10069179).
589 Guerra A, Raynaud A, Beyssen B, Pagny JY, Sapoval M, Angel C. Arterial percutaneous angioplasty in upper limbs with vascular access devices for haemodialysis. *Nephrol Dial Transplant* 2002;**17**:843–51.
590 Suding PN, Wilson SE. Strategies for management of ischemic steal syndrome. *Semin Vasc Surg* 2007;**20**:184–8.
591 Callaghan CJ, Mallik M, Sivaprakasam R, Iype S, Pettigrew GJ. Treatment of dialysis access-associated steal syndrome with the "revision using distal inflow" technique. *J Vasc Access* 2011;**12**:52–6.
592 Mickley V. Steal syndrome–strategies to preserve vascular access and extremity. *Nephrol Dial Transplant* 2008;**23**:19–24.
593 Sessa C, Pecher M, Maurizi-Balzan J, Pichot O, Tonti F, Farah I, et al. Critical hand ischemia after angioaccess surgery: diagnosis and treatment. *Ann Vasc Surg* 2000;**14**:583–93.
594 Loh TM, Bennett ME, Peden EK. Revision using distal inflow is a safe and effective treatment for ischemic steal syndrome and pathologic high flow after access creation. *J Vasc Surg* 2016;**63**:441–4.
595 Chemla ES, Tang VC, Eyman SA. Intraoperative flow measurements are helpful in the treatment of high-inflow steal syndrome on a predialysis patient with a brachiocephalic fistula: a case report. *Ann Vasc Surg* 2007;**21**:645–7.
596 Schanzer H, Schwartz M, Harrington E, Haimov M. Treatment of ischemia due to "steal" by arteriovenous fistula with distal artery ligation and revascularization. *J Vasc Surg* 1988;**7**:770–3.
597 Knox RC, Berman SS, Hughes JD, Gentile AT, Mills JL. Distal revascularization-interval ligation: a durable and effective treatment for ischemic steal syndrome after hemodialysis access. *J Vasc Surg* 2002;**36**:250–5. discussion 6.
598 Anaya-Ayala JE, Pettigrew CD, Ismail N, Diez-De Sollano AL, Syed FA, Ahmed FG, et al. Management of dialysis access-associated "steal" syndrome with DRIL procedure: challenges and clinical outcomes. *J Vasc Access* 2012;**13**:299–304.
599 Gupta N, Yuo TH, Konig G, Dillavou E, Leers SA, Chaer RA, et al. Treatment strategies of arterial steal after arteriovenous access. *J Vasc Surg* 2011;**54**:162–7.
600 Field M, Blackwell J, Jaipersad A, Wall M, Silva MA, Morgan RH, et al. Distal revascularisation with interval ligation (DRIL): an experience. *Ann R Coll Surg Engl* 2009;**91**:394–8.
601 Misskey J, Yang C, MacDonald S, Baxter K, Hsiang Y. A comparison of revision using distal inflow and distal revascularization-interval ligation for the management of severe access-related hand ischemia. *J Vasc Surg* 2016;**63**:1574–81.
602 Al Shakarchi J, Stolba J, Houston JG, Inston N. Surgical techniques for haemodialysis access-induced distal ischaemia. *J Vasc Access* 2016;**17**:40–6.
603 Zanow J, Kruger U, Scholz H. Proximalization of the arterial inflow: a new technique to treat access-related ischemia. *J Vasc Surg* 2006;**43**:1216–21. discussion 21.
604 Hye RJ, Wolf YG. Ischemic monomelic neuropathy: an underrecognized complication of hemodialysis access. *Ann Vasc Surg* 1994;**8**:578–82 (7865397).
605 Thermann F, Kornhuber M. Ischemic monomelic neuropathy: a rare but important complication after hemodialysis access placement–a review. *J Vasc Access* 2011;**12**:113–9 (21360465).
606 Wodicka R, Isaacs J. Ischemic monomelic neuropathy. *J Hand Surg Am* 2010;**35**:842–3 (19942360).
607 Goldstein LJ, Helfend LK, Kordestani RK. Postoperative edema after vascular access causing nerve compression secondary to the presence of a perineuronal lipoma: case report. *Neurosurgery* 2002;**50**:412–3 (11844280).
608 Talebi M, Salari B, Ghannadan H, Kakaei F, Azar SA. Nerve conduction changes following arteriovenous fistula construction in hemodialysis patients. *Int Urol Nephrol* 2011;**43**:849–53 (20419395).

609 Brennan AM, McNamara B, Plant WD, O'Halloran DJ. An atypical case of acute ischaemic monomelic neuropathy post vascular access surgery in a patient with Type 1 diabetes mellitus. *Diabet Med* 2005;**22**:813—4 (15910638).
610 Miles AM. Upper limb ischemia after vascular access surgery: differential diagnosis and management. *Semin Dial* 2000;**13**:312—5.
611 Thermann F, Brauckhoff M, Kornhuber M. Dialysis shunt-associated ischaemic monomelic neuropathy: neurological recovery preserving the dialysis access. *Nephrol Dial Transplant* 2006;**21**:3334—6 (16854852).
612 Soleimani MJ, Shahrokh H, Shadpour P, Shirani M, Arasteh S. Impact of dialysis access fistula on cardiac function after kidney transplantation. *Iran J Kidney Dis* 2012;**6**:198—202.
613 Unger P, Wissing KM, de Pauw L, Neubauer J, van de Borne P. Reduction of left ventricular diameter and mass after surgical arteriovenous fistula closure in renal transplant recipients. *Transplantation* 2002;**74**:73—9.
614 van Duijnhoven EC, Cheriex EC, Tordoir JH, Kooman JP, van Hooff JP. Effect of closure of the arteriovenous fistula on left ventricular dimensions in renal transplant patients. *Nephrol Dial Transplant* 2001;**16**:368—72.
615 Cridlig J, Selton-Suty C, Alla F, Chodek A, Pruna A, Kessler M, et al. Cardiac impact of the arteriovenous fistula after kidney transplantation: a case-controlled, match-paired study. *Transpl Int* 2008;**21**:948—54.
616 Miller GA, Koh E, Khariton A, Preddie DC, Hwang WW, Savransky Y, et al. Sharp needle recanalization for salvaging hemodialysis accesses with chronically occluded peripheral outflow. *J Vasc Access* 2012;**13**:22—8.
617 Teruya TH, Schaeffer D, Abou-Zamzam AM, Bianchi C. Arteriovenous graft with outflow in the proximal axillary vein. *Ann Vasc Surg* 2009;**23**:95—8.
618 Hazinedaroglu S, Karakayali F, Tuzuner A, Ayli D, Demirer S, Duman N, et al. Exotic arteriovenous fistulas for hemodialysis. *Transplant Proc* 2004;**36**:59—64.
619 Ono K, Muto Y, Yano K, Yukizane T. Anterior chest wall axillary artery to contralateral axillary vein graft for vascular access in hemodialysis. *Artif Organs* 1995;**19**:1233—6.
620 Chemla ES, Morsy M, Anderson L, Makanjuola D. Complex bypasses and fistulas for difficult hemodialysis access: a prospective, single-center experience. *Semin Dial* 2006;**19**:246—50.
621 McCann RL. Axillary grafts for difficult hemodialysis access. *J Vasc Surg* 1996;**24**:457—61. discussion 61—62.
622 Rueckmann I, Berry C, Ouriel K, Hoffart N. The synthetic axillofemoral graft for hemodialysis access. *ANNA J* 1991;**18**:567—71.
623 Jakimowicz T, Galazka Z, Grochowiecki T, Nazarewski S, Szmidt J. Vascular access for haemodialysis in patients with central vein thrombosis. *Eur J Vasc Endovasc Surg* 2011;**42**:842—9.
624 Huber TS, Hirneise CM, Lee WA, Flynn TC, Seeger JM. Outcome after autogenous brachial-axillary translocated superficial femoropopliteal vein hemodialysis access. *J Vasc Surg* 2004;**40**:311—8.
625 Al Shakarchi J, Houston JG, Jones RG, Inston N. A review on the Hemodialysis Reliable Outflow (HeRO) graft for haemodialysis vascular access. *Eur J Vasc Endovasc Surg* 2015;**50**:108—13.
626 Steerman SN, Wagner J, Higgins JA, Kim C, Mirza A, Pavela J, et al. Outcomes comparison of HeRO and lower extremity arteriovenous grafts in patients with long-standing renal failure. *J Vasc Surg* 2013;**57**:776—83. discussion 82—83.
627 Katzman HE, McLafferty RB, Ross JR, Glickman MH, Peden EK, Lawson JH. Initial experience and outcome of a new hemodialysis access device for catheter-dependent patients. *J Vasc Surg* 2009;**50**:600—7. 7 e1.
628 Kokkosis AA, Abramowitz SD, Schwitzer J, Schanzer H, Teodorescu VJ. Experience of HeRO dialysis graft placement in a challenging population. *Vasc Endovasc Surg* 2013;**47**:278—80.
629 Gage SM, Ahluwalia HS, Lawson JH. Salvaging vascular access and treatment of severe limb edema: case reports on the novel use of the hemodialysis reliable outflow vascular access device. *Ann Vasc Surg* 2011;**25**:e1—5.
630 Smith TP, Ryan JM, Reddan DN. Transhepatic catheter access for hemodialysis. *Radiology* 2004;**232**:246—51.
631 Khadra MH, Dwyer AJ, Thompson JF. Advantages of polytetrafluoroethylene arteriovenous loops in the thigh for hemodialysis access. *Am J Surg* 1997;**173**:280—3.
632 Bhandari S, Wilkinson A, Sellars L. Saphenous vein forearm grafts and gortex thigh grafts as alternative forms of vascular access. *Clin Nephrol* 1995;**44**:325—8.
633 Flora HS, Chaloner EJ, Day C, Barker SG. The ankle arteriovenous fistula: an approach to gaining vascular access for renal haemodialysis. *Eur J Vasc Endovasc Surg* 2001;**22**:376—8.
634 Pierre-Paul D, Williams S, Lee T, Gahtan V. Saphenous vein loop to femoral artery arteriovenous fistula: a practical alternative. *Ann Vasc Surg* 2004;**18**:223—7.
635 Ryan JJ, Perkins JD. Saphenous to popliteal arteriovenous fistulas as a suitable alternative in chronic hemodialysis. *Surg Gynecol Obstet* 1989;**168**:550—1.
636 Lynggaard F, Nordling J, Iversen Hansen R. Clinical experience with the saphena loop arteriovenous fistula on the thigh. *Int Urol Nephrol* 1981;**13**:287—90.
637 Taylor SM, Eaves GL, Weatherford DA, McAlhany Jr JC, Russell HE, Langan 3rd EM. Results and complications of arteriovenous access dialysis grafts in the lower extremity: a five year review. *Am Surg* 1996;**62**:188—91.
638 Calder FR, Chemla ES, Anderson L, Chang RW. The axillary artery-popliteal vein extended polytetrafluoroethylene graft: a new technique for the complicated dialysis access patient. *Nephrol Dial Transplant* 2004;**19**:998—1000.
639 Kendall Jr TW, Cull DL, Carsten 3rd CG, Kalbaugh CA, Cass AL, Taylor SM. The role of the prosthetic axilloaxillary loop access as a tertiary arteriovenous access procedure. *J Vasc Surg* 2008;**48**:389—93.
640 Zanow J, Kruger U, Petzold M, Petzold K, Miller H, Scholz H. Arterioarterial prosthetic loop: a new approach for hemodialysis access. *J Vasc Surg* 2005;**41**:1007—12.
641 Khan AR, Blackwell LM, Stafford SJ, Thompson AD, Romero RJ, Goodier CD, et al. Femororenal arteriovenous graft: a viable option for hemodialysis access. *Ann Vasc Surg* 2008;**22**:136—9.
642 Herscu G, Woo K, Weaver FA, Rowe VL. Use of unconventional dialysis access in patients with no viable alternative. *Ann Vasc Surg* 2013;**27**:332—6.
643 Murthy R, Arbabzadeh M, Lund G, Richard 3rd H, Levitin A, Stainken B. Percutaneous transrenal hemodialysis catheter insertion. *J Vasc Intervent Radiol JVIR* 2002;**13**:1043—6.
644 Henderson S, Brown E, Levy J. Safety and efficacy of percutaneous insertion of peritoneal dialysis catheters under sedation and local anaesthetic. *Nephrol Dial Transplant* 2009;**24**:3499—504.
645 Medani S, Shantier M, Hussein W, Wall C, Mellotte G. A comparative analysis of percutaneous and open surgical techniques for peritoneal catheter placement. *Perit Dial Int* 2012;**32**:628—35.
646 Chow KM, Szeto CC, Leung CB, Kwan BC, Pang WF, Li PK. Tenckhoff catheter insertion by nephrologists: open dissection technique. *Perit Dial Int* 2010;**30**:524—7.

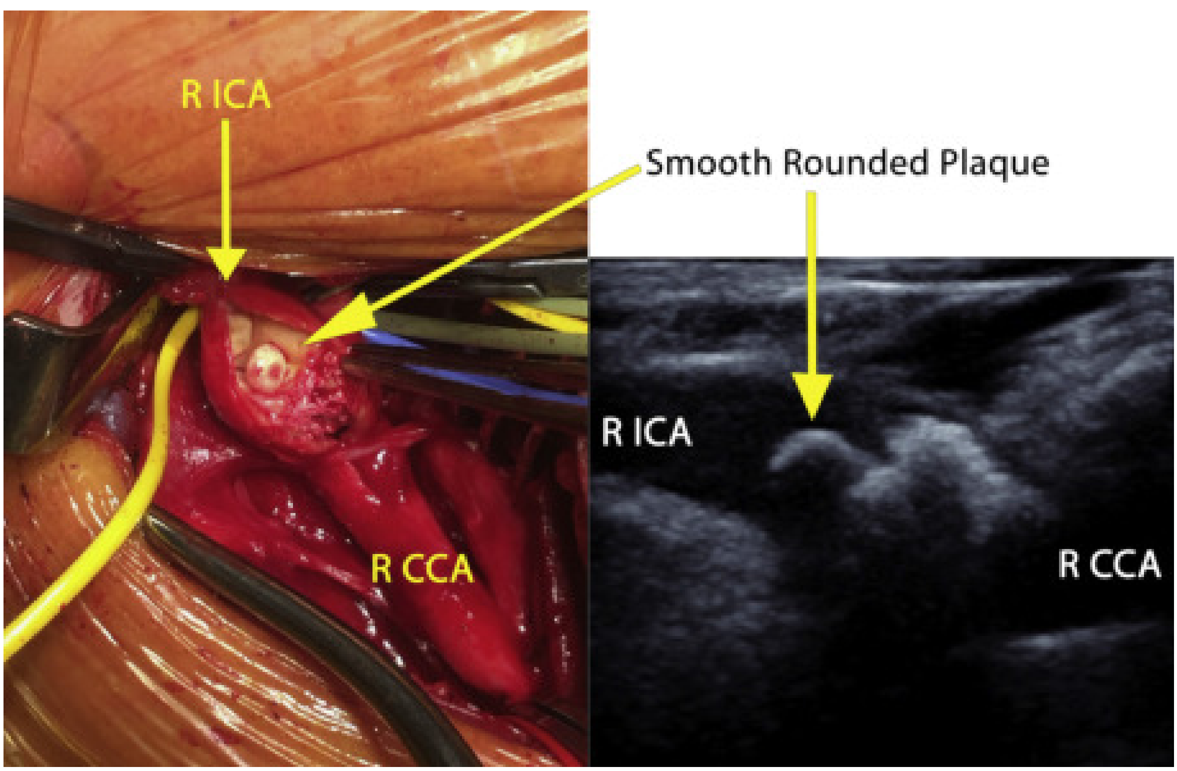
647 Chuengsaman P, Panomrerngsak A, Sriudom K. Does previous abdominal operation affect peritoneal dialysis complications and outcomes? *J Med Assoc Thai* 2011;**94**:S64—70.
648 Jo YI, Shin SK, Lee JH, Song JO, Park JH. Immediate initiation of CAPD following percutaneous catheter placement without break-in procedure. *Perit Dial Int* 2007;**27**:179—83.
649 Quinn R, Ravani P, Investigators AH. ACCESS HD pilot: a randomised feasibility trial comparing catheters with fistulas in elderly patients starting haemodialysis. *BMJ Open* 2016;**6**: e013081.
650 Chemla E, Velazquez CC, D'Abate F, Ramachandran V, Maytham G. Arteriovenous fistula construction with the VasQ external support device: a pilot study. *J Vasc Access* 2016;**17**: 243—8.
651 Nikam M, Chemla ES, Evans J, Summers A, Brenchley P, Tavakoli A, et al. Prospective controlled pilot study of arteriovenous fistula placement using the novel Optiflow device. *J Vasc Surg* 2015;**61**:1020—5.
652 Rajan DK, Lok CE. Promises for the future: minimally invasive fistula creation. *J Vasc Access* 2015;**16**:S40—1.

Eur J Vasc Endovasc Surg (2018) 55, 818

COUP D'OEIL

An Unusual Polypoid Symptomatic Carotid Plaque

Stephen C. Crockett *, **Matthew Metcalfe**
Lister Hospital, Stevenage, United Kingdom



A 71 year old man presented with six episodes of right amaurosis fugax over the preceding month. He underwent an uncomplicated carotid endarterectomy under local anaesthesia. The image shows the rounded polyp on ultrasound and intra-operatively the plaque projecting into the lumen, distal to the 70—79% stenosis at the right internal carotid artery origin. Histology was in keeping with an atheromatous plaque. Around 20% of carotid plaques are smooth, but unusually this was almost spherical. Current evidence suggests major plaque irregularities infer a greater risk of neurological events, in which case this polypoid lesion is rare in both its morphology and its presentation.

* Corresponding author.
E-mail address: stephencrockett@doctors.org.uk (Stephen C. Crockett).

https://doi.org/10.1016/j.ejvs.2018.01.029