

## Editors' Choice

## The Society for Vascular Surgery practice guidelines on follow-up after vascular surgery arterial procedures



R. Eugene Zierler, MD,<sup>a</sup> William D. Jordan, MD,<sup>b</sup> Brajesh K. Lal, MD,<sup>c</sup> Firas Mussa, MD,<sup>d</sup> Steven Leers, MD,<sup>e</sup> Joseph Fulton, MD,<sup>f</sup> William Pevec, MD,<sup>g</sup> Andrew Hill, MD,<sup>h</sup> and M. Hassan Murad, MD, MPH,<sup>i</sup> *Seattle, Wash; Atlanta, Ga; Baltimore, Md; Columbia, SC; Pittsburgh, Pa; Poughkeepsie, NY; Sacramento, Calif; Ottawa, Ontario, Canada; and Rochester, Minn*

## ABSTRACT

Although follow-up after open surgical and endovascular procedures is generally regarded as an important part of the care provided by vascular surgeons, there are no detailed or comprehensive guidelines that specify the optimal approaches with regard to testing methods, indications for reintervention, and follow-up intervals. To provide guidance to the vascular surgeon, the Clinical Practice Council of the Society for Vascular Surgery appointed an expert panel and a methodologist to review the current clinical evidence and to develop recommendations for follow-up after vascular surgery procedures. For those procedures for which high-quality evidence was not available, recommendations were based on observational studies, committee consensus, and indirect evidence. Recognizing that there are numerous published reports on the role of duplex ultrasound for surveillance of infrainguinal vein bypass grafts, the Society commissioned a systematic review and meta-analysis on this topic.

The panel classified the strength of each recommendation and the corresponding quality of evidence on the basis of the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) system: recommendations were graded either *strong* or *weak*, and the quality of evidence was graded *high*, *moderate*, or *low*. The resulting recommendations represent a wide variety of open surgical and endovascular procedures involving the extracranial carotid artery, thoracic and abdominal aorta, mesenteric and renal arteries, and lower extremity arterial revascularization. The panel also identified many areas in which there was a lack of high-quality evidence to support their recommendations. This suggests that there are opportunities for further clinical research on testing methods, threshold criteria, and the role of surveillance as well as on the modes of failure and indications for reintervention after vascular surgery procedures. (J Vasc Surg 2018;68:256-84.)

**Keywords:** Surveillance; Duplex imaging; Postoperative follow-up; Clinical guidelines

## SCOPE OF THE PROBLEM

Open surgical and endovascular interventions for the treatment of vascular disease span a wide variety of vessels and techniques. Whereas much is known about

the durability of well-established procedures such as infrainguinal vein bypass grafts, the clinical outcomes of the newer endovascular approaches are less well documented.<sup>1-4</sup> All vascular procedures have modes of failure that must be identified and managed appropriately to provide the best possible long-term results. Follow-up of patients after vascular surgery procedures is generally regarded as the key to detection of recurrent disease and other complications that can lead to morbidity and mortality. The primary goal of follow-up in this setting is to detect significant problems at an early stage when they can be managed most safely and effectively, even before clinical signs and symptoms are evident. However, for most vascular surgery procedures, the optimal methods and frequency for follow-up are not clear. The challenge to the vascular surgeon is to develop a follow-up plan for each patient that will achieve this goal while minimizing costs, risks, and disruption of the patient's lifestyle.

The simplest approach to follow-up is *clinical monitoring* with a periodic vascular history and physical examination. The term *surveillance* describes the routine, planned use of serial objective testing to evaluate the status of a vascular procedure. Surveillance is generally performed in patients with no current evidence of a problem related

From the Department of Surgery, University of Washington, Seattle<sup>a</sup>; the Department of Surgery, Emory University, Atlanta<sup>b</sup>; the Department of Surgery, University of Maryland, Baltimore<sup>c</sup>; the Department of Surgery Palmetto Health/University of South Carolina School of Medicine, Columbia<sup>d</sup>; the Division of Vascular Surgery, University of Pittsburgh Medical Center, Pittsburgh<sup>e</sup>; the Department of Surgery, Westchester Medical Center, Poughkeepsie<sup>f</sup>; the Division of Vascular Surgery, University of California, Davis, Sacramento<sup>g</sup>; the Division of Vascular & Endovascular Surgery, The Ottawa Hospital & University of Ottawa, Ottawa<sup>h</sup>; and the Division of Preventive Medicine, Mayo Clinic, Rochester.<sup>i</sup>

Author conflict of interest: none.

Correspondence: R. Eugene Zierler, MD, Department of Surgery, University of Washington, 1959 NE Pacific St, Box 356410, Seattle, WA 98195-6410 (e-mail: [gzierler@uw.edu](mailto:gzierler@uw.edu)).

Independent peer-review and oversight has been provided by the members of the SVS Document Oversight Committee: Thomas L. Forbes, MD (Chair), Ali AbuRahma, MD, Kwame Amankwah, MD, Neal Barshes, MD, Ruth Bush, MD, Ronald L. Dalman, MD, Hans Henning Eckstein, MD, Anil Hingorani, MD, Eva Rzcudlo, MD, Marc Schermerhorn, MD.

0741-5214

Copyright © 2018 by the Society for Vascular Surgery. Published by Elsevier Inc. <https://doi.org/10.1016/j.jvs.2018.04.018>

**Table I.** Criteria for grading strength of a recommendation and quality of evidence

Strength of recommendation	
1 (Strong)	
	Benefits > Risks
	Risks > Benefits
2 (Weak)	
	Benefits ≈ risks
	Quality of evidence precludes accurate assessment of risks and benefits.
Quality of evidence	
A (High)	
	Additional research is considered very unlikely to change confidence in the estimate of the effect.
B (Moderate)	
	Further research is likely to have an important impact on the estimate of the effect.
C (Low)	
	Further research is very likely to change the estimate of the effect.
Adapted from Guyatt G, Gutterman D, Baumann MH, Addrizzo-Harris D, Hylek EM, Phillips B, et al. Grading strength of recommendations and quality of evidence in clinical guidelines. <i>Chest</i> 2006;129:174-81.	

to the procedure and is based on the assumption that significant abnormalities may not be detected by clinical monitoring alone. *Diagnostic testing* refers to the use of various physiologic or imaging methods in a patient who has signs or symptoms suggestive of a problem with a previous vascular procedure, including an abnormal finding on surveillance evaluation. Such testing may include limb blood pressure measurements, duplex ultrasound (DUS), computed tomography (CT) or magnetic resonance (MR) imaging with and without contrast enhancement, and catheter-directed angiography.

## METHODS AND EVIDENCE

To provide guidance to the vascular surgery community, the Clinical Practice Council of the Society for Vascular Surgery appointed an expert panel of vascular surgeons and a methodologist to develop recommendations for follow-up after vascular surgery procedures. A review of the available clinical evidence was completed to serve as the basis for these recommendations. Because of the extensive literature on the role of DUS for surveillance of infrainguinal vein bypass grafts, a dedicated de novo systematic review and meta-analysis were carried out on that topic by the Evidence-based Practice Center of the Mayo Clinic College of Medicine, Rochester, Minnesota. For those procedures for which high-quality evidence could not be found, recommendations are based on observational studies, committee consensus, and indirect evidence.

The strength of each recommendation and the corresponding quality of evidence were graded separately on

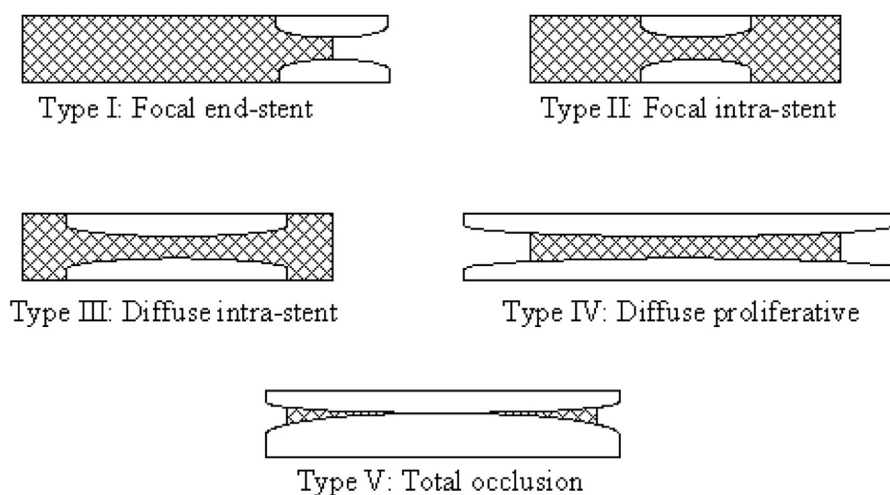
the basis of the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) system (Table I).<sup>5,6</sup> A recommendation was considered *strong* (grade 1) when benefits clearly outweighed risks (or risks outweighed benefits); a *weak* (grade 2) recommendation was made when risks and benefits were closely balanced or low-quality evidence precluded a definitive evaluation of risks and benefits. The quality of evidence was graded *high* (A), *moderate* (B), or *low* (C) according to an estimate of whether additional research would be unlikely, likely, or very likely to change the effect. The quality of evidence from randomized trials and observational studies was initially considered *high* or *low*, respectively; the quality of evidence could then be adjusted up or down on the basis of GRADE domains (eg, consistency, precision). In these guidelines, the panel denoted *strong* and *weak* recommendations by the terms *we recommend* and *we suggest*, respectively. Some strong recommendations for surveillance were made despite low-quality evidence. This was done when the costs and risks of surveillance were considered to be relatively low and the early detection of complications was deemed critical from a patient's perspective.

The panel's recommendations for follow-up after vascular surgery procedures are presented in the following sections. The included procedures involve the extracranial carotid artery, thoracic and abdominal aorta, mesenteric and renal arteries, and lower extremity arterial revascularization. Where appropriate, recommendations for both open surgical and endovascular interventions are included. Considering the large number of arterial procedures that needed to be covered, and recognizing the highly specialized nature of procedures for venous disease, the panel chose not to include recommendations for follow-up after superficial and deep venous interventions. Surveillance after arteriovenous hemodialysis access procedures is also not covered because that topic has been discussed in a separate clinical practice guideline document.<sup>7</sup>

These guidelines for follow-up after vascular surgery arterial procedures emphasize vascular laboratory testing and vascular imaging. Other aspects of follow-up, such as medical management and risk factor modification, are not specifically addressed. It is essential that vascular laboratory testing be performed by qualified personnel using appropriate instrumentation, as demonstrated by individual credentialing and facility accreditation. Whereas the panel has aimed to make specific recommendations that are generalizable and applicable to most patients, it is impossible to account for every clinical eventuality, and surgeons should use their best clinical judgment along with these guidelines in the management of the individual patient.

## EXTRACRANIAL CAROTID ARTERY

Carotid endarterectomy (CEA) is the preferred treatment for symptomatic<sup>8,9</sup> and asymptomatic<sup>10,11</sup> patients with high-grade extracranial carotid stenosis compared



**Fig.** Morphologic patterns of in-stent restenosis (ISR) based on B-mode imaging; type I, focal  $\leq 10$  mm, end-stent lesions; type II, focal  $\leq 10$  mm, intrastent lesions; type III, diffuse  $>10$  mm, intrastent lesions; type IV, diffuse  $>10$  mm, proliferative lesions extending outside the stent; and type V, total occlusion.

with best medical therapy. The large number of CEAs performed worldwide has resulted in a number of post-CEA restenosis cases. It has also been shown that carotid artery stenting (CAS) is technically feasible and safe in high-risk patients requiring carotid intervention.<sup>12-15</sup> With the approval of CAS in the United States for high-risk symptomatic patients with significant carotid stenosis ( $\geq 70\%$ ) and neurologic symptoms (ipsilateral stroke, transient ischemic attack, and amaurosis fugax), it is likely that the number of CAS procedures will continue to increase. This will result in a number of post-CAS in-stent restenosis (ISR) cases.

#### Modes of failure

Two mechanisms can account for restenosis after carotid procedures. Restenosis early ( $<24$  months) after the procedure is generally attributed to neointimal hyperplasia. Restenosis that occurs later after carotid procedures is believed to be caused by progressive atherosclerosis.<sup>16</sup> The patterns of developing neointimal hyperplastic lesions after CAS may reflect the aggressiveness of the hyperplastic response and may also predict the future development of high-grade restenosis ( $\geq 80\%$ ) that may require reintervention. The morphologic patterns of ISR have been studied using B-mode imaging, and specific patterns of post-CAS ISR have been described (Fig).<sup>17</sup> Mapping these patterns with DUS after CAS may assist in determining the frequency with which patients must be observed for future high-grade restenosis.

#### Incidence and treatment of carotid restenosis

The Asymptomatic Carotid Atherosclerosis Study (ACAS) follow-up data demonstrated that carotid restenosis 3 to 18 months after CEA, defined as Doppler-determined diameter reductions of  $\geq 60\%$ , occurred in

at least 7.6% of patients.<sup>18</sup> In a study of patients undergoing CAS during a follow-up period of 1 to 74 months (mean,  $18.8 \pm 10$  months), 22 of 122 patients (18.0%) demonstrated ISR  $\geq 40\%$ .<sup>19</sup> All restenosis patients were asymptomatic on presentation and were diagnosed by DUS during routine follow-up. Only five of these patients demonstrated high-grade ISR ( $\geq 80\%$ ), whereas the remaining fell in the lower ranges. The projected 5-year rate for ISR  $\geq 80\%$  was 6.4%.

In the Carotid Revascularization Endarterectomy vs Stenting Trial (CREST), restenosis or occlusion rates were found to be no different between 1086 patients undergoing CAS and 1105 undergoing CEA at 2 and 4 years after revascularization.<sup>20</sup> Restenosis (defined as a reduction in diameter of at least 70% diagnosed by a peak systolic velocity [PSV] of at least 300 cm/s) or occlusion occurred in 120 patients (58 CAS, 62 CEA). The Kaplan-Meier estimate for the frequency of this composite outcome at 2 years was 6.0% for CAS and 6.3% for CEA (hazard ratio, 0.90; 95% confidence interval, 0.63-1.29;  $P = .58$ ) after adjustment for age, sex, and symptomatic status; the Kaplan-Meier estimate at 4 years was 6.7% for CAS and 6.2% for CEA (hazard ratio, 0.94; 95% confidence interval, 0.66-1.33;  $P = .71$ ).

The Endarterectomy vs Angioplasty in Patients with Symptomatic Severe Carotid Stenosis (EVA-3S) trial also randomized patients to CAS or CEA and had ultrasound follow-up information on 507 patients.<sup>13</sup> Restenosis rates for  $\geq 50\%$  diameter reduction were higher after CAS (12.5%) vs CEA (5%), whereas restenosis rates for  $\geq 70\%$  diameter reduction were modest and equivalent after CAS or CEA (3.3% vs 2.8%, respectively, at 3 years of follow-up). The Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy (SAPPHIRE)

trial<sup>14</sup> and the Stent-Protected Angioplasty vs Carotid Endarterectomy (SPACE) study<sup>15</sup> have reported similarly low restenosis rates ranging from 2.8% during 3 years to 10.5% during 5 years after CAS.

The clinical significance of restenosis after carotid procedures is still debated. Because of the small number of patients who develop significant restenosis after CAS or CEA and the lack of clinical trials observing these patients for extended periods, there is relatively little evidence to guide treatment. The incidence of symptomatic restenosis after CEA is low and ranges from 0% to 8.2%.<sup>20</sup> In patients with asymptomatic restenosis, the risk of stroke or progression to total occlusion is small.<sup>21,22</sup> Based on the low incidence of symptoms in this cohort of patients, it has been proposed that careful clinical follow-up alone may be appropriate for asymptomatic patients.

Others have taken a more aggressive approach toward asymptomatic restenosis and have elected to operate on high-grade ( $\geq 80\%$ ) asymptomatic lesions. In the series of 206 redo CEA procedures reported by O'Hara et al,<sup>23</sup> only 43% had symptoms. Mansour et al<sup>24</sup> operated on 82 restenoses, of which 66% were symptomatic and the remaining had high-grade asymptomatic restenoses of  $\geq 80\%$ . DeGroote et al<sup>25</sup> reported a low complication rate with redo CEA for asymptomatic high-grade ( $>80\%$ ) and symptomatic ( $>50\%$ ) carotid restenosis. The rationale for an operative approach to asymptomatic restenosis is that it is extremely difficult to predict which high-grade lesions will progress to occlusion and subsequent neurologic deterioration.

### Risk factors for carotid restenosis

Predictors for neointimal hyperplasia are the subject of continued investigation. Diabetes is a well-known predictor of early and aggressive intimal hyperplasia and ISR after coronary artery stenting.<sup>26,27</sup> One report observed an increased incidence of ISR in patients with uncontrolled diabetes undergoing CAS.<sup>28</sup> A multivariate analysis of the CREST cohort demonstrated that patients with restenosis (regardless of whether they had undergone CAS or CEA) were more likely to be younger, women, diabetic, and hyperlipidemic.<sup>20</sup> Restenosis was more common in smokers than in nonsmokers who underwent CEA, but no difference was identified in those who underwent CAS. In a separate study, restenotic lesions involving the entire length of the stent (type IV; Fig) and a history of diabetes were independent predictors of high-grade ISR and reintervention (odds ratio, 5.1 and 9.7, respectively).<sup>17</sup>

### Methods and instrumentation for follow-up

DUS is the standard technique used to observe patients treated with CEA or medical therapy alone for carotid artery disease. The advantages of DUS for follow-up of patients undergoing carotid revascularization are well recognized—it is noninvasive, free of complications, and readily available in vascular laboratories. Although the

reported experience with DUS for assessment of primary native carotid artery stenosis is extensive and long-standing,<sup>29</sup> the issue of establishing specific velocity criteria for patients undergoing CAS has been addressed only very recently. Two studies initially reported altered blood flow velocities after carotid stent placement.<sup>30,31</sup> The authors proposed that these variations in velocity measurements adversely affected the accuracy of DUS in CAS patients, and they concluded that ultrasound velocity measurements as an index of stenosis were not consistent after carotid stent placement.

Stenting has been reported to alter the biomechanical properties of the carotid artery such that compliance was reduced.<sup>32</sup> The enhanced stiffness of the stent-arterial wall complex rendered the flow-pressure relationship of the carotid artery closer to that observed in a rigid tube, so that the energy normally applied to dilate the artery resulted in an increased velocity. Subsequent studies have confirmed that DUS is reliable in the diagnosis of carotid restenosis after CAS, provided appropriate adjustments are made in the threshold criteria used to diagnose diameter reduction.<sup>33-38</sup>

CT angiography and MR angiography are alternative methods for determining restenosis after carotid procedures. The attendant radiation, nephrotoxic contrast agents, and expense incurred with these modalities means that they are more frequently used to confirm a suspected restenosis after CEA or CAS, such as when elevated velocities are identified on follow-up DUS. CT and MR angiography are also used to assess the aortic arch and the anatomy of the proximal common carotid artery (CCA) and distal internal carotid artery (ICA), regions that are not well visualized on DUS. Cervical contrast angiography is an invasive but reliable method of confirming restenosis, especially in the event that a therapeutic intervention is being considered.

**Examination technique and protocols.** The technique of DUS after CAS and CEA is similar to that used for the diagnosis of native carotid occlusive disease with additional emphasis on B-mode imaging. Bilateral examination using a high-resolution linear array transducer should be performed in cross-sectional and longitudinal scan planes starting at the proximal CCA, progressing through the treated ICA and into the distal native ICA.

In CAS cases, the stent may traverse the origin of the external carotid artery, but flow through the stent interstices into the external carotid artery is usually maintained. An assessment should be made of stent apposition to the surrounding plaque, expansion of the lumen, and luminal encroachment due to neointimal hyperplasia. The morphology of any hyperplastic lesion can be mapped according to the classification mentioned previously. Velocity waveforms and luminal diameter measurements should be obtained at multiple locations. The minimum number of sampling sites will

**Table II.** Optimal velocity threshold criteria for varying severity of in-stent restenosis (ISR) after carotid artery stenting (CAS)

ISR, % diameter reduction	Velocity criteria
≥20	PSV ≥150 cm/s and ICA/CCA ratio ≥2.15
≥50	PSV ≥220 cm/s and ICA/CCA ratio ≥2.7
≥80	PSV ≥340 cm/s and ICA/CCA ratio ≥4.15

CCA, Common carotid artery; ICA, internal carotid artery; ICA/CCA ratio, PSV in the stented ICA/PSV in the native CCA; PSV, maximum peak systolic velocity.  
A PSV threshold of ≥300 cm/s was used to identify ≥70% stenosis in the Carotid Revascularization Endarterectomy versus Stenting Trial (CREST).<sup>20</sup>  
Adapted from Lal BK, Hobson RW 2nd, Goldstein J, Chakhtoura EY, Duran WN. Carotid artery stenting: is there a need to revise ultrasound velocity criteria? *J Vasc Surg* 2004;39:58-66; and Lal BK, Hobson RW 2nd, Tofighi B, Kapadia I, Cuadra S, Jamil Z. Duplex ultrasound velocity criteria for the stented carotid artery. *J Vasc Surg* 2007;47:63-73.

depend on the location and the length of the stent. In general, samples should be obtained from the proximal, mid, and distal segments of the stent, with additional samples taken distal and proximal to the stent. The maximum PSV within the stent as well as the ratio of this value to the native CCA PSV (ICA/CCA ratio) should be measured.

In CEA cases, the arteriotomy closure sutures may be seen as bright, evenly spaced echoes along the near wall of the CCA and ICA in the B-mode image. If a patch was used, it can create a dilation at the endarterectomy site of varying dimensions. Whereas a vein patch may be indistinguishable in appearance from the wall of the native artery, the dilation and the sutures can help identify its presence. Prosthetic patches may have a typical ultrasound signature. For example, a Dacron patch will appear as a thick, brightly echogenic surface, and polytetrafluoroethylene will typically appear as a bright double line that represents the thickness of the material and the effects of ultrasound penetration.

**Interpretation criteria.** The utility of DUS in the detection of native carotid artery disease is well documented and has led to the use of the PSV, end-diastolic velocity (EDV), and ICA/CCA ratio, either alone or in some combination, to define normal and increasingly stenotic carotid vessels. Doppler ultrasound velocities correlate with angiographic percentage stenosis in the native ICA, and the appropriate threshold velocities signifying different degrees of stenosis have been intensively analyzed and identified.<sup>29,39</sup>

In recent reports,<sup>32,33</sup> DUS velocity measurements were compared with luminal stenosis measured by conventional angiography or CT angiography during follow-up of CAS patients (n = 310 observations). Receiver operating characteristic (ROC) analysis demonstrated the optimal threshold criteria for ISR shown in Table II. These velocity criteria have been subsequently substantiated in

additional studies.<sup>34-38</sup> Emerging data suggest the need for similar revisions to velocity criteria after CEA; however, additional studies need to be performed before firm recommendations can be made.<sup>40,41</sup>

Summary of recommendations
1. After CEA or CAS, we recommend surveillance with DUS at baseline and every 6 months for 2 years and annually thereafter until stable (ie, until no restenosis or ISR is observed in two consecutive annual scans). The first or baseline DUS should occur soon after the procedure, preferably within 3 months, with the goal of establishing a post-treatment baseline. Considering the small risk of delayed restenosis or ISR, some interval of regular surveillance (eg, every 2 years) should be maintained for the life of the patient.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: B (Moderate)
2. For patients undergoing CAS with diabetes, aggressive patterns of ISR (type IV), prior treatment for ISR, prior cervical radiation, or heavy calcification, in addition to the baseline DUS we recommend surveillance with DUS every 6 months until a stable clinical pattern is established and annually thereafter.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: B (Moderate)
3. We recommend that DUS after CAS include at least the following assessments:
A. Doppler measurement of PSV and EDV in the native CCA; in the proximal, mid, and distal stent; and in the distal native ICA. As discussed before, modified threshold velocity criteria should be used to interpret the significance of these velocity measurements after CAS.
B. B-mode imaging should be used to supplement and to enhance the accuracy of velocity criteria to estimate the severity of luminal narrowing.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: C (Low)

## THORACIC AND ABDOMINAL AORTA

### Surveillance after thoracic endovascular aortic repair (TEVAR)

TEVAR has become the first-line treatment for descending thoracic aneurysms. Furthermore, TEVAR has been expanded to other thoracic aortic diseases, such as penetrating ulcers, blunt aortic injuries, and complicated aortic dissections.<sup>42-47</sup> These indications are usually in a population of younger patients with added concerns about long-term durability and efficacy. At present, there is no evidence that TEVAR for descending thoracic aneurysmal disease is superior to open repair for long-term outcomes. This paucity of convincing long-term data and the need for reintervention have led to recommendations for lifelong surveillance. Given that TEVAR is no longer restricted to a cohort of older patients, the consequences of lifelong surveillance deserve

special consideration, especially in younger patients (eg, trauma and dissection).<sup>42,43</sup> The accumulated radiation dose of treatment followed by yearly lifelong CT scans places these patients at an increased risk of radiation-induced cancer.<sup>48</sup>

**Rationale for surveillance.** As for endovascular aneurysm repair (EVAR) in the abdominal aorta, it will be necessary to show that the benefits of surveillance after TEVAR are justified by the risks and cost over time. These benefits may include prevention or treatment of graft-related problems, such as endoleak, collapse, and migration, as well as of aortic abnormalities, such as aneurysmal degeneration of contiguous segments.<sup>49-52</sup> The rationale is that intervention might reduce the risk of aneurysm-related death. However, unlike EVAR, which is mostly done for aneurysmal disease, TEVAR is used in disparate pathologic processes, including aneurysms, dissection, trauma, penetrating ulcers, and intramural hematoma.<sup>53</sup> Thoracic aneurysms and dissections have post-TEVAR complications similar to those of abdominal aortic stent grafts, namely, progressive aortic dilation and endoleaks, and therefore close follow-up is warranted.<sup>54</sup> Conversely, blunt thoracic aortic injuries are typically focal and are associated with TEVAR done in younger patients.<sup>55</sup> Complications have been seen in the short term and include unsuccessful exclusion and endograft infolding. Surveillance regimens in those patients may be relaxed yet not completely stopped as the long-term device behavior is still uncertain.<sup>56</sup>

**Endoleak.** Endoleak occurs in up to 30% of aneurysm patients after TEVAR.<sup>57,58</sup> Type I and type III endoleaks represent the most concerning types and are considered treatment failures because of persistent aneurysmal sac pressurization, and these require immediate attention to prevent rupture and possible death.<sup>59-62</sup> Most type II endoleaks originating from the left subclavian artery require intervention, especially if they are associated with sac enlargement.<sup>63</sup> At 2 years of follow-up, 56% of these required a secondary intervention.<sup>64</sup> Presence of late endoleak was the only predictor of decreased survival in a single-center prospective series, emphasizing the potential hazards of undetected and untreated late TEVAR complications.<sup>65</sup>

**Migration and collapse.** Proximal migration can lead to encroachment on arch vessel ostia,<sup>66</sup> whereas device collapse can lead to type IA endoleak at up to 3 years after TEVAR.<sup>67</sup> Both of these device-related complications can occur in the early and late postoperative periods. Other rare complications of TEVAR include retrograde type A dissection and aortoesophageal fistula, both often requiring open conversion for salvage.<sup>67</sup> Jonker et al<sup>68</sup> reviewed endograft collapse after TEVAR in 60 cases collected from 32 publications. Those cases were performed for trauma in 39 (65%) and acute or chronic type B aortic dissections in 9 (15%). The median time interval between TEVAR and diagnosis of endograft

collapse was 15 days (range, 1 day-79 months). The prognosis is not entirely benign, with a 30-day mortality of 8.3% and freedom from procedure-related death at 3 years after diagnosis of stent graft collapse of 83.1% for asymptomatic patients compared with 72.7% for patients who had symptoms at diagnosis ( $P = .029$ ). Endograft collapse was associated with excessive oversizing in 20% of cases, and a small radius of curvature of the aortic arch was responsible in 48%.

**Aortic remodeling and detection of contiguous aneurysms.** Aortic remodeling and false lumen thrombosis are both predictive of survival after TEVAR-treated chronic type B dissection, and surveillance is important to monitor these end points.<sup>69</sup> A systematic review of 17 studies and >500 patients with dissection showed that reinterventions were observed in up to 60% of patients when no regular follow-up was documented.<sup>51</sup> Most patients required secondary reinterventions for enlargement of the untreated aorta remote from the stent graft repair.<sup>52</sup> Post-TEVAR aneurysmal progression warrants regular follow-up, even in the absence of endoleak.<sup>70</sup>

**Duration of surveillance.** Late complications after TEVAR for either aneurysmal disease or dissection are common and can develop at various time points regardless of the indication or device selection.<sup>63</sup> Based on the currently available evidence, it seems prudent to continue ongoing surveillance for TEVAR, as the duration after which no further complications occur has not been identified for either aneurysmal disease or aortic dissection.<sup>52,71</sup>

**Surveillance modalities.** CT scanning is ideal for follow-up after TEVAR, especially with three-dimensional projections and delayed image acquisition to detect low-flow endoleaks. Despite the cumulative risks of radiation exposure, the benefits of continued surveillance are likely to be justified.<sup>72</sup> Certain predisposing factors have been shown to increase the risks of endoleak, including "bird-beaking" at the proximal seal zone,<sup>73</sup> compromised proximal seal zone due to length or angulation, greater length of aortic coverage and number of components used,<sup>74</sup> and larger diameter at the proximal seal zone and coverage of the left subclavian artery without endovascular or open ligation.<sup>75</sup> Grafts placed along the lesser curve of a large aneurysm have the potential for outward migration with resultant component dislocation and type III endoleak.<sup>76</sup>

### Follow-up after open surgical repair for thoracic aortic aneurysm

Two multicenter trials comparing TEVAR with open surgical repair for the treatment of thoracic aortic aneurysms reported 5-year results.<sup>59,77</sup> In both trials, the follow-up of the open (control) arm was incomplete. Of 94 patients in the Gore TAG (W. L. Gore & Associates, Flagstaff, Ariz) trial, reinterventions directly related to open aneurysm repair occurred in two patients, with

one having a proximal anastomotic collection drained and one having débridement and drain placement for an aorto-esophageal fistula. Follow-up consisted of patient visits, four-view chest radiographs, and CT scans at 1 month, 6 months, and 12 months and then yearly. The aneurysm reintervention rate was 2.1% for the open controls.<sup>59</sup> In the Zenith TX2 (Cook Medical, Bloomington, Ind) trial, 70 patients were enrolled in the open (control) arm. Patients in the control group underwent clinical evaluation before discharge or at 1 month and then at 12 months and yearly thereafter for up to 5 years. Secondary interventions occurred at similar rates between the groups.<sup>77</sup> Thus, the data on reintervention after open thoracic aortic repair are limited to recent clinical trials that compared new devices with the open standard. There are limited clinical data on which to base a decision, but the degenerative process of aortic aneurysms raises the clinical suspicion that surveillance at some interval is warranted because of the potential for late failures.

#### Surveillance after endovascular abdominal aortic aneurysm repair

The overall use of EVAR in comparison to open surgery for treatment of abdominal aortic aneurysms has risen sharply in the past 15 years—from 5.2% in 2000 to 74% in 2012—even though the total number of abdominal aortic aneurysm repairs has remained stable at 45,000 cases per year.<sup>78</sup> Current guidelines recommend the use of serial CT scanning after EVAR to monitor for aneurysm expansion, endoleak, graft migration, and structural failures.<sup>79</sup>

**Existing recommendations and surveillance protocols.** Protocols for EVAR surveillance were established as an extension of the initial Food and Drug Administration-sponsored pivotal trials and consist of four-view plain abdominal radiographs and CT imaging at 1 month, 6 months, and 12 months after initial repair and yearly thereafter.<sup>79,80</sup> More recently, increased concern about the cost and cumulative risk of cancer from radiation exposure has led to increased interest in use of ultrasound as the sole modality for post-EVAR follow-up.<sup>81</sup> Although ultrasound avoids radiation exposure and use of nephrotoxic contrast agents, questions have been raised in the past about the variable sensitivity of ultrasound in identifying endoleaks.<sup>82-85</sup> A meta-analysis of 10 published studies comparing color duplex ultrasound (CDU) with contrast-enhanced CT found a sensitivity and specificity of 69% and 91%, respectively, with greater sensitivity in detecting type I and type III endoleaks than type II endoleaks.<sup>86</sup> Recent studies have suggested that CDU has a high degree of correlation with CT scanning in detection of endoleak and sac size.<sup>87-90</sup> Moreover, contrast-enhanced ultrasound (CEUS) has increased sensitivity, specificity, negative predictive value, and accuracy compared with CDU.<sup>91-93</sup>

Vascular practices have increasingly been using CDU, with or without CEUS, as an alternative to CT imaging, especially in patients with renal insufficiency, young patients, and those with a shrinking aneurysm sac. If CT imaging does not identify an endoleak or sac enlargement at 30 days, the 6-month CT imaging study can be eliminated and follow-up done with CDU, as long as the patient's endograft can be imaged by ultrasound.<sup>94-96</sup> Conversely, if an endoleak or other abnormalities are detected at 30 days and beyond, a more rigorous imaging protocol is implemented, usually with CT imaging at 6 months. Similarly, contrast imaging will be required if a new endoleak is detected or sac enlargement is identified by ultrasound.<sup>97</sup> This need for rigorous surveillance decreases over time as the aneurysm continues to shrink.<sup>98,99</sup> However, there is a risk of late endoleak up to 15% despite normal findings on initial CT imaging.<sup>100</sup> The utility of ultrasound is limited in obese patients or those presenting with substantial bowel gas or a large ventral hernia.

**Examination technique and protocols.** In the absence of endoleak or aneurysm sac enlargement on CT scan in the first year after EVAR, DUS (including CDU) may be a reasonable alternative for surveillance. However, these examinations should be performed by a skilled vascular technologist in an accredited vascular laboratory. The basic technique of DUS testing after EVAR is similar to that used for the diagnosis of abdominal aortic aneurysms.

Fasting patients are scanned using the B-mode (gray-scale) and color flow DUS modalities. Measurements of the aneurysm sac diameter are recorded, and color flow, Doppler spectral waveforms, and ultrasound contrast (for CEUS) can be used to identify endoleaks. For diameter measurements and assessment of arterial anatomy, gray-scale imaging is performed beginning at or above the level of the renal arteries and followed down at least to the common iliac artery bifurcations or beyond the distal limb attachment sites. For assessment of aortic aneurysm sac diameter, electronic caliper measurements are made from outer wall to outer wall. Both anteroposterior and transverse diameter can be measured; however, the aortic walls are usually visualized more clearly in the anteroposterior direction. Special attention is directed to the area of maximum dilation of the aneurysm where both limbs of the endograft are usually visualized. A meticulous evaluation for the presence of pulsatile color flow is performed at the attachment sites proximally and distally as well as at the junctional points of modular grafts and throughout the aneurysm sac. Potential areas of endoleak, such as the inferior mesenteric artery or lumbar arteries, are also inspected.

The utility of DUS scanning as a screening test for the detection of abdominal aortic aneurysms has been well documented in a large population-based trial.<sup>101,102</sup> The cross-sectional dimensions of an abdominal aortic aneurysm on DUS correlate well with CT scanning.<sup>87</sup> Both

CDU and CEUS have been shown to correlate with post-EVAR CT scans for the detection of endoleak or sac enlargement.<sup>92,103</sup>

### Follow-up after open surgical repair for abdominal aortic aneurysm

Surveillance programs have not been well described after open repair of an abdominal aortic aneurysm.<sup>104,105</sup> This is largely due to the low incidence of graft-related complications.<sup>106,107</sup> However, the most common complication after open repair is graft limb occlusion, especially if the limb extends to the common femoral artery.<sup>108</sup> Furthermore, open abdominal aortic aneurysm repair carries a threefold increased risk of incisional hernia compared with surgery performed for arterial occlusive disease.<sup>109</sup> The probability of a para-anastomotic aneurysm is 0.8% at 5 years, 6.2% at 10 years, and 35.8% at 15 years.<sup>110</sup> Future aneurysmal degeneration in contiguous or noncontiguous segments of the aorta is reported in between 0.5% and 10%.<sup>111-114</sup> The Open Versus Endovascular Repair (OVER) Veterans Affairs Cooperative Study Group reported 2-year outcomes of 881 patients randomized to open or endovascular abdominal aortic aneurysm repair, including any aortoiliac procedures at any time during follow-up.<sup>115</sup> The 55 secondary therapeutic procedures in the open repair group included 24 incisional hernia repairs, 7 aortic graft procedures, 4 procedures for wound complications, 4 amputations in three patients, 4 laparotomies for bowel obstruction, 2 laparotomies for hematoma, 2 procedures to relieve claudication, and 8 miscellaneous minor procedures.

Summary of recommendations
1. We recommend contrast-enhanced CT scanning at 1 month and 12 months and then annually after TEVAR for thoracic aortic aneurysm. If the 1-month CT scan detects an abnormality, a repeated CT scan at 6 months should be considered.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: B (Moderate)
2. We recommend contrast-enhanced CT scanning at 1 month, 6 months, and 12 months and then annually after TEVAR for thoracic aortic dissection.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: B (Moderate)
3. We recommend contrast-enhanced CT scanning at 1 month and 12 months and then annually after TEVAR for blunt thoracic aortic injury. If the 1-month CT scan detects an abnormality, a repeated CT scan at 6 months should be considered. Future studies may provide data to support longer surveillance intervals after TEVAR for traumatic injury once a stable clinical pattern is established.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: B (Moderate)
4. We recommend CT scanning with or without contrast enhancement at 5-year intervals after open surgical repair for thoracic aortic disease.

(Continued)

Continued.

Strength of Recommendation: 1 (Strong)
Quality of Evidence: C (Low)
5. We recommend contrast-enhanced CT scanning at 1 month and 12 months after EVAR, with consideration of more frequent imaging if an endoleak or other abnormality of concern is detected at 1 month.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: B (Moderate)
6. We recommend DUS at 12-month intervals as alternative imaging surveillance after EVAR if no endoleak or sac enlargement was detected during the first year.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: B (Moderate)
7. We recommend DUS and non-contrast-enhanced CT scanning as alternative imaging surveillance after EVAR in patients with contraindications to iodinated contrast agents.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: B (Moderate)
8. We recommend total aortic imaging with non-contrast-enhanced CT scanning at 5-year intervals after open surgical repair or EVAR to detect aneurysmal degeneration of other aortic segments.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: C (Low)

### MESENTERIC ARTERIES

Chronic mesenteric arterial insufficiency is manifested by postprandial pain and weight loss, and untreated, it is a potentially fatal condition. Treatment options by open surgical reconstruction include transaortic endarterectomy of the celiac and superior mesenteric arteries, antegrade bypass from the supraceliac aorta to the celiac and superior mesenteric arteries, and retrograde bypass from the infrarenal aorta or an iliac artery to the superior mesenteric and celiac arteries. Endovascular approaches consist of balloon angioplasty, with or without stenting, of the celiac, superior mesenteric, or inferior mesenteric arteries. As stenoses in the mesenteric arteries are typically orificial with a high likelihood of plaque recoil after balloon angioplasty alone, balloon-expandable stents are commonly deployed.

#### Rationale for follow-up

Open surgical repair is more durable than endovascular repair for revascularization of the mesenteric arteries (Tables III and IV).<sup>116</sup> However, patients with chronic mesenteric arterial insufficiency are typically malnourished, frail, and poor candidates for major surgical procedures. Consequently, endovascular intervention has become the more frequent treatment modality for chronic mesenteric arterial insufficiency, trading an increased risk of recurrent stenosis or occlusion for lower periprocedural morbidity and mortality.<sup>116,117</sup> This is a

**Table III. A,** Open mesenteric artery repair, primary patency

Reference	No. <sup>a</sup>	4 years, %	5 years, %
Jimenez, <sup>219</sup> 2002	47		69
Kruger, <sup>220</sup> 2007	39		92
Zerbib, <sup>221</sup> 2008	34	79	

<sup>a</sup>Number of patients or cases.**Table III. B,** Open mesenteric artery repair, secondary patency

Reference	No. <sup>a</sup>	1 year, %	2 years, %	5 years, %	6 years, %
McMillan, <sup>118</sup> 1995	25				89
Jimenez, <sup>219</sup> 2002	47			100	
Atkins, <sup>117</sup> 2007	34	90			
Davies, <sup>222</sup> 2009	17		100		

<sup>a</sup>Number of patients or cases.

reasonable strategy because although primary patency of angioplasty with stenting of the mesenteric arteries is not ideal, assisted and secondary patency rates are good (Table IV).<sup>116</sup> However, for this strategy to be safe and effective, surveillance is required as mesenteric infarction and death may be the consequence of a failed intervention. Clinical follow-up alone is not sensitive to failing or even failed mesenteric arterial reconstructions.<sup>118</sup> Therefore, both open and endovascular mesenteric arterial reconstructions should undergo imaging surveillance.

### Methods and instrumentation for follow-up

**Examination techniques and protocols.** DUS is the most common modality for follow-up of mesenteric arterial interventions. Patients should be fasting overnight before the examination to reduce interference with ultrasound imaging due to bowel gas. Doppler flow waveforms are obtained from the celiac axis and the splenic, common hepatic, superior mesenteric, and inferior mesenteric arteries, recording the PSV and EDV at multiple locations along the arteries. Direction of flow is recorded, particularly in the common hepatic artery, as flow may be reversed in that vessel in the presence of occlusion or stenosis of the celiac axis.

**Interpretation criteria.** There are no well-established DUS criteria to diagnose recurrent stenosis after endovascular or open surgical mesenteric arterial reconstruction.<sup>119</sup> Published experience suggests that the standard criteria used to identify stenosis in the native celiac axis and superior mesenteric artery will overestimate the degree of restenosis after bypass or stenting.<sup>120-122</sup>

AbuRahma et al<sup>120</sup> compared DUS and arteriographic findings in 30 stented celiac and 32 stented superior mesenteric arteries. Based on ROC analysis, they recommended the PSV thresholds for ISR in the celiac axis and

superior mesenteric artery that are shown in Table V. In a similar study, Soult et al<sup>123</sup> reviewed 103 paired DUS scans and arteriograms after mesenteric artery stenting, including 66 superior mesenteric and 37 celiac arteries, and proposed the PSV thresholds listed in Table V. Other authors have advocated various other criteria<sup>124-126</sup> Baker et al<sup>122</sup> advocated obtaining a baseline DUS scan and reintervening if the PSV increased significantly from baseline or was  $\geq 500$  cm/s. There are no published criteria for diagnosis of restenosis after mesenteric artery bypass grafts.

### Summary of recommendations

1. There are no prospective reports documenting the efficacy of a surveillance protocol after mesenteric artery stenting or bypass grafts; however, recurrent mesenteric ischemia is potentially life-threatening. Therefore, after mesenteric artery (celiac, superior mesenteric, and inferior mesenteric) angioplasty with or without stenting or mesenteric artery bypass grafting, we recommend the following:

A. Clinical follow-up and baseline DUS within 1 month of the procedure.

B. Clinical follow-up and DUS at 6 months, 12 months, and then annually thereafter.

Strength of Recommendation: 1 (Strong)

Quality of Evidence: C (Low)

2. We suggest contrast imaging for patients with symptoms of recurrent mesenteric ischemia after mesenteric artery stents or bypass grafts or for the following DUS findings:

A. Celiac axis: PSV  $>370$  cm/s or a substantial increase from the post-treatment baseline PSV (what constitutes a substantial increase has not been defined).

B. Superior mesenteric artery: PSV  $>420$  cm/s or a substantial increase from the post-treatment baseline PSV (what constitutes a substantial increase has not been defined).

C. Inferiormesenteric artery: Substantial increase from the post-treatment baseline PSV (what constitutes a substantial increase has not been defined).

Strength of Recommendation: 2 (Weak)

Quality of Evidence: C (Low)

## RENAL ARTERIES

Renal artery reconstruction is typically considered to preserve renal function or to manage severe hypertension in the presence of significant occlusive disease. Atherosclerosis is the most common etiology, causing  $>90\%$  of renal artery stenoses. Fibromuscular dysplasia is the second most common cause. The results of intervention are more favorable for fibromuscular dysplasia than for renal artery atherosclerosis.<sup>127</sup>

Open surgical reconstruction, either endarterectomy or bypass, has been largely supplanted by percutaneous angioplasty and stenting.<sup>128</sup> The literature reports good anatomic outcomes after renal artery angioplasty and stenting; however, the Angioplasty and Stenting for

**Table IV. A.** Endovascular mesenteric artery repair, primary patency

Reference	Subgroup	No. <sup>a</sup>	1 year, %	2 years, %	3 years, %
Landis, <sup>223</sup> 2005		29	70		
Atkins, <sup>117</sup> 2007		31	58		
Sarac, <sup>224</sup> 2008		65	65		
Fioole, <sup>225</sup> 2010		51	86	60	
Peck, <sup>226</sup> 2010		49			64
Sharafuddin, <sup>227</sup> 2012		26	58	33	
Ahanchi, <sup>124</sup> 2013	Celiac	40	18		
	SMA	92	55		

SMA, Superior mesenteric artery.  
<sup>a</sup>Number of patients or cases.

**Table IV. B.** Endovascular mesenteric artery repair, assisted primary patency

Reference	No. <sup>a</sup>	1 year, %	2 years, %
Landis, <sup>223</sup> 2005	29	88	
Atkins, <sup>117</sup> 2007	31	65	
Sarac, <sup>224</sup> 2008	65	97	
Fioole, <sup>225</sup> 2010	51	88	79
Sharafuddin, <sup>227</sup> 2012	26	80	60

<sup>a</sup>Number of patients or cases.

**Table IV. C.** Endovascular mesenteric artery repair, secondary patency

Reference	No. <sup>a</sup>	1 year, %	2 years, %
Davies, <sup>222</sup> 2009	15		65
Sarac, <sup>224</sup> 2008	65	99	
Tallarita, <sup>125</sup> 2011	157		72

<sup>a</sup>Number of patients or cases.

Renal Artery Lesions (ASTRAL) and Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL) randomized trials failed to demonstrate a clinical benefit of renal artery intervention compared with best medical therapy for patients with moderate stenosis and hypertension.<sup>129,130</sup>

### Rationale for follow-up

Restenosis is not uncommon after renal artery intervention, although reported rates are variable because of different types of interventions, different definitions of restenosis, and different follow-up modalities (Table VI). Nonetheless, there is a substantial rate of restenosis after renal artery angioplasty and stenting. Various techniques have been described to treat renal artery ISR, including simple balloon angioplasty, cutting balloon angioplasty, repeated stenting with a bare-metal stent, repeated stenting with a drug-eluting stent, insertion of a covered stent, and surgical

**Table V.** Optimal velocity threshold criteria for varying severity of in-stent restenosis (ISR) after mesenteric artery stenting

Reference	ISR, % diameter reduction	PSV threshold	
		Celiac axis	Superior mesenteric artery
AbuRahma, <sup>120</sup> 2012	≥50	274 cm/s	325 cm/s
	≥70	363 cm/s	412 cm/s
Soult, <sup>123</sup> 2016	≥70	289 cm/s	445 cm/s

PSV, Peak systolic velocity, maximum.

bypass.<sup>131-138</sup> However, recurrent stenosis after reinterventions is also common, occurring at rates of 20% to 71% after secondary interventions.<sup>131-138</sup> No single technique appears to be superior.<sup>131,132,136-138</sup>

### Methods and instrumentation for follow-up

**Examination techniques and protocols.** CT angiography has been shown to be effective and sensitive but not specific for detecting renal artery ISR.<sup>139,140</sup> After stenting, metallic artifact can affect the ability to visualize the flow lumen compared with the renal artery diameter. In addition, CT angiography requires iodinated vascular contrast material, a serious drawback in patients with renal artery disease.

DUS is the most common modality used for follow-up of renal artery interventions. As for the evaluation of mesenteric artery interventions, patients should be fasting overnight before the examination to limit bowel gas and to improve visualization of the renal arteries. A low-frequency, curved linear or phased array transducer, typically 1 to 5 MHz, is most commonly used. PSVs and EDVs are recorded in the supramesenteric aorta and in the origin, proximal, mid, distal, and hilar renal artery segments. A renal to aortic velocity ratio is calculated as the highest renal artery PSV divided by the supramesenteric aortic PSV. This ratio is valid only if the aortic PSV is in the normal range (typically between 40 and 100 cm/s). The length of each kidney should also be measured.

**Table VI. A.** Rates of restenosis after intervention for renal artery atherosclerosis

Reference	No. <sup>a</sup>	% with restenosis at follow-up, months							
		6	9	12	18	24	36	60	72
Lewis, <sup>228</sup> 1994	18	38							
Dorros, <sup>229</sup> 1995	92	25							
Harjai, <sup>230</sup> 1997	44		25						
White, <sup>231</sup> 1997	80		19						
Rundback, <sup>232</sup> 1998	28			25					
Rocha-Singh, <sup>233</sup> 1999	180			12					
Rodriguez-Lopez, <sup>234</sup> 1999	96						26		
Henry, <sup>235</sup> 1999	259							21	
Van de Ven, <sup>236</sup> 1999	13	25							
Yutan, <sup>237</sup> 2001	86							37	
Ahmadi, <sup>238</sup> 2002	32			6		6			36
Sivamurthy, <sup>239</sup> 2004	183							30	
Nolan, <sup>240</sup> 2005	96			25					
Muller-Hülsbeck, <sup>241</sup> 2005	50	13			25				
Sapoval, <sup>242</sup> 2005	52	14							
Sahin, <sup>243</sup> 2006	15	0		8		31			
Rastan, <sup>244</sup> 2008	55			4					
Rocha-Singh, <sup>245</sup> 2008	117		21						
Klonaris, <sup>246</sup> 2008	14			0			10		
Misra, <sup>247</sup> 2008 (drug-eluting stents)	16			22		32			
Misra, <sup>247</sup> 2008 (bare-metal stents)	9			42		53			
Corriere, <sup>248</sup> 2009	101	50		60					
Davies, <sup>132</sup> 2009	619							19	
Thalhammer, <sup>249</sup> 2010	105	17							
Laird, <sup>250</sup> 2010	188			13					
Jaff, <sup>251</sup> 2012	241		10						
Simone, <sup>133</sup> 2013	216			16			59		

<sup>a</sup>Number of renal arteries.

**Interpretation criteria.** Published experience suggests that PSV is increased after stenting of the renal artery.<sup>141</sup> Thus, the criteria used to diagnose native renal artery stenosis need to be modified in evaluating stented renal arteries. Suggested threshold velocity criteria to diagnose renal artery ISR are listed in [Table VII](#).

#### Summary of recommendations

1. There are no prospective reports documenting the efficacy of a surveillance protocol after renal artery interventions. After renal artery angioplasty with or without stenting or renal artery bypass or endarterectomy, we suggest the following:

A. Clinical follow-up and baseline DUS within 1 month of the procedure.

B. Clinical follow-up and DUS at 6 months and 12 months and then annually thereafter.

Strength of Recommendation: 2 (Weak)

Quality of Evidence: C (Low)

(Continued)

Continued.

2. We suggest contrast-enhanced imaging for loss of renal parenchyma (a decrease in kidney length of >1 cm) or for the following DUS findings:

A. Renal artery: PSV  $\geq$ 280 cm/s or a substantial increase from the post-treatment baseline PSV (what constitutes a substantial increase has not been defined).

B. Renal to aortic velocity ratio of  $\geq$ 4.5.

Strength of Recommendation: 2 (Weak)

Quality of Evidence: B (Moderate)

## OPEN LOWER EXTREMITY ARTERIAL REVASCULARIZATION

Open surgical revascularization of the lower extremities includes many different procedures in a variety of anatomic locations. Anatomic and extra-anatomic bypass can be performed with prosthetic or autogenous materials, and each carries its own morbidity and failure rates. Surveillance protocols include clinical assessment,

**Table VI. B.** Rates of restenosis after intervention for renal artery fibromuscular dysplasia

Reference	No. <sup>a</sup>	% with restenosis at follow-up, years				
		1	2	3	5	9
Birrer, <sup>252</sup> 2002	31	23				
Kim, <sup>253</sup> 2008	16		22			
Ham, <sup>254</sup> 2010 <sup>b</sup>	62	9		20	20	
Ham, <sup>254</sup> 2010 <sup>c</sup>	12	27		51	51	
Mousa, <sup>127</sup> 2012	43	5			29	50

<sup>a</sup>Number of arteries.  
<sup>b</sup>Takayasu arteritis and fibromuscular dysplasia; open reconstructions.  
<sup>c</sup>Takayasu arteritis and fibromuscular dysplasia; endovascular reconstructions.

**Table VII.** Optimal velocity threshold criteria for varying severity of in-stent restenosis (ISR) after renal artery stenting

Reference	No. <sup>a</sup>	ISR, % diameter reduction	Renal artery PSV, cm/s	RAR
Bakker, <sup>255</sup> 1999	9	>50	226	2.7
Chi, <sup>141</sup> 2009	67	>70	395	5.1
Mohabbat, <sup>256</sup> 2009 <sup>b</sup>	518	>60	280	4.5
Fleming, <sup>257</sup> 2010	30	>60	250	
Del Conde, <sup>258</sup> 2013	132	>60	296	4.4

PSV, Peak systolic velocity; RAR, renal to aortic velocity ratio.  
<sup>a</sup>Number of arteries.  
<sup>b</sup>Renal artery stents and stent grafts inserted during endovascular aortic reconstruction.

measurement of the ankle-brachial index (ABI), and DUS of the treated arterial segment or bypass graft.<sup>142,143</sup> The optimal frequency and duration of surveillance depend on the intervention and arterial bed, but surveillance generally begins immediately after surgery and then continues at 3, 6, and 12 months and then every 6 to 12 months thereafter.<sup>144</sup>

Aortoiliac and infrainguinal interventions constitute the two major groups of open lower extremity revascularization procedures. Aortoiliac interventions can be further classified as anatomic and extra-anatomic bypasses, which are usually but not exclusively performed with prosthetic material. Infrainguinal interventions may use autogenous or prosthetic conduits, and there are a number of adjuncts that can be applied to the prosthetic procedures, including vein cuffs and distal arteriovenous fistulas.<sup>145-147</sup>

### Rationale for follow-up

Mechanisms of failure after open lower extremity revascularization range from inadequate inflow or outflow to problems inherent in the conduit used. Anastomoses are particularly prone to intimal hyperplastic lesions, and

vein grafts—especially those using veins other than the great saphenous vein—frequently develop intrinsic problems related to a variety of factors. DUS provides the ability to accurately assess all aspects of an arterial reconstruction over time and is therefore appealing as a means of long-term surveillance. Numerous studies have demonstrated that identifying and repairing graft-threatening lesions prolongs bypass patency,<sup>148-150</sup> but there have been few comprehensive reviews on this topic.

### Methods and instrumentation

Methods for postoperative surveillance include both physiologic testing and direct imaging modalities and have been described primarily with infrainguinal bypass. Approaches for follow-up of aortoiliac reconstruction are less well defined in the literature. The ABI has proved less sensitive than direct imaging with DUS, which has been the historical method most often applied to bypass graft surveillance.<sup>151</sup> When DUS is performed, it includes the native inflow and outflow vessels, proximal and distal anastomoses, and multiple locations along the course of the bypass graft conduit.

### Aortoiliac revascularization

**Aortobifemoral bypass.** Aortobifemoral bypass may be performed for aneurysmal or occlusive disease. Patency rates are excellent and in the range of 88% to 93% at 3 to 5 years.<sup>152,153</sup> High flow rates and large conduits undoubtedly contribute to these high patency rates, and as a result, there are no studies describing long-term surveillance for these bypasses.

**Iliofemoral bypass.** This procedure is performed for unilateral iliac artery occlusive disease as an alternative to femoral-femoral bypass and avoids bilateral groin incisions. In a review of 468 unilateral iliofemoral occlusions treated during a 20-year period, Mellièrè et al<sup>154</sup> used DUS surveillance to follow 144 iliofemoral bypasses and compared them with alternative bypasses and endarterectomy. At 8 years, primary patency was 66% and secondary patency was unchanged. Carsten et al<sup>155</sup> reported 5-year secondary patency of 93.3%, suggesting that iliofemoral bypass is a durable alternative to aortobifemoral and femoral-femoral bypass, again using DUS in addition to ABI and clinical examination. In a retrospective case series, Nazzal et al<sup>156</sup> reported 61.3% primary patency and 80.5% secondary patency at 5 years, although surveillance was not used.

**Femoral-femoral bypass.** Extra-anatomic femoral-femoral bypass can be performed for unilateral iliac artery occlusive disease or at the time of EVAR when only one iliofemoral system is intact and generally results in patency rates lower than those of aortobifemoral bypass. In a prospective case study review, Piotrowski et al<sup>157</sup> demonstrated 5-year primary patency rates of 72%, 56%, and 35% for aortobifemoral, iliofemoral, and femoral-femoral bypasses, respectively, although surveillance was not clearly described. When femoral-femoral bypass was

performed with aortouni-iliac EVAR, Hinchliffe et al<sup>158</sup> reported a 5-year cumulative patency of 83%, suggesting that the durability of this procedure is better in treating aneurysmal disease. Surveillance was done by CT scan in this series. Stone et al<sup>159</sup> used DUS-derived criteria (PSV >300 cm/s and PSV ratio >3.5) to guide revision of femoral-femoral grafts and increased 5-year primary or assisted primary patency from 62% to 88%.

**Axillobifemoral bypass.** Axillobifemoral bypass is an extra-anatomic revascularization performed in two main groups of patients: those unfit for a direct aortic reconstruction, and those with infection of a previous aortic graft to avoid placement of a new graft in the infected field. The length of the bypass, the general health of the patients, and the presence of infection results in a relatively high risk of occlusion and other complications. Although this procedure is relatively common, there is very little literature addressing surveillance. Cumulative patency rates of 85% to 87% at 3 to 4 years were reported in early studies without any mention of surveillance.<sup>160,161</sup>

### Infringuinal revascularization

**Autogenous vein bypass.** Although lower extremity bypass with autogenous vein remains one of the most common procedures performed by vascular surgeons, there are multiple modes of failure and complications. Selection of patients, comorbid conditions, inadequate inflow or outflow, and imperfect conduits are some of the factors contributing to adverse outcomes. Moreover, once it is successfully performed, the bypass itself is at risk for early, midterm, or late failure. Early failure (within 30 days) is generally attributed to technical error. Midterm failure (30 days-24 months) usually results from intimal hyperplastic lesions affecting the conduit and anastomoses. Late failures (after 24 months) frequently result from progression of atherosclerosis involving the inflow or outflow vessels.<sup>162</sup> It is the midterm and late failures that make surveillance appealing as ABI and DUS can dependably identify graft-threatening lesions that can then be treated to avoid progression to occlusion.

A focal increase in PSV can be used to calculate a velocity ratio (Vr), defined as the PSV at the site of a stenosis divided by the PSV in a normal vessel segment proximal to the stenosis. Hemodynamic features of a successful infringuinal bypass graft include an ABI >0.9 or an increase in ABI of at least 0.15 and graft flow velocity >45 cm/s with low-resistance outflow waveforms. Increased risk for graft thrombosis is indicated by a focal increase in PSV of 180 to 300 cm/s and a Vr of 2.0 to 3.5. The highest risk for graft thrombosis is conferred by a focal increase in PSV to >300 cm/s, Vr >3.5, graft flow velocity <45, and drop in ABI >0.15 (Table VIII).<sup>163</sup>

Colledge et al<sup>164</sup> in 1996 performed a systematic review of infringuinal bypasses, including 2680 under DUS

surveillance and 3969 not surveyed. Surveillance identified 493 stenoses in 469 (19%) grafts, of which 98 (26%) developed recurrent stenoses after intervention. The total numbers of deaths and bypass graft occlusions were higher in the nonsurveillance group; however, limb salvage was not different between the two groups. Retrospective case review series suggest that close surveillance enhanced assisted primary patency, especially when risk stratification was used to identify the highest risk bypasses.<sup>1,148</sup> A revision rate of 30.9% resulted from such intensive follow-up.<sup>142,144</sup> Ihlberg et al<sup>165,166</sup> described two randomized controlled trials comparing DUS surveillance with simple clinical follow-up. In both studies, primary patency, assisted primary patency, and secondary patency were no different between the two groups. Of note, very few graft revisions were necessary in either study, so the statistical power of the studies was low. Mofidi et al,<sup>167</sup> in a prospective cohort study, demonstrated that a normal DUS scan at 6 weeks predicted excellent durability without the need for frequent DUS surveillance.

A prospective randomized controlled trial performed by Lundell et al<sup>150</sup> showed a significant benefit with intensive DUS surveillance. Assisted primary and secondary patency rates at 3 years were 78% and 82% in surveyed grafts compared with 53% and 56% in nonsurveyed bypasses. Of note, patency of prosthetic bypasses was unaffected by surveillance. Davies et al<sup>168</sup> reported the results of the Vein Graft Surveillance Randomised Trial (VGST), in which 594 bypasses were randomized to a DUS surveillance or nonsurveillance protocol. The nonsurveillance group had ABI measurement and clinical examination on the same schedule as the surveillance group. There was no difference in primary patency, assisted primary patency, secondary patency, and limb salvage between the groups. However, a significant number of bypasses were subjected to some secondary interventions in both protocols (16% nonsurveyed and 22% surveyed bypasses). In addition, early graft failures may have been missed, and DUS criteria for stenosis were not standardized.

In an attempt to clarify the utility of DUS surveillance for infringuinal autogenous vein bypass grafts, the Evidence-based Practice Center of the Mayo Clinic College of Medicine conducted a systematic review and meta-analysis of the current literature on this topic at the request of the Society for Vascular Surgery, and a detailed description of this review has been published separately.<sup>169</sup> The review summarized 15 studies and noted variation in the DUS surveillance protocols used. Compared with ABI combined with clinical examination, DUS surveillance was not associated with a significant change in primary, assisted primary, or secondary patency or mortality. Surveillance with DUS was associated with a nonsignificant reduction in amputation rate (odds ratio, 0.70; confidence interval, 0.23-2.13).

**Table VIII.** Duplex ultrasound (DUS) velocity and ankle-brachial index (ABI) threshold criteria for stratification of risk for thrombosis of infrainguinal vein grafts

Category	High-velocity criteria (PSV)	Velocity ratio (Vr)	Low-velocity criteria (GFV), cm/s	Change in ABI
Highest risk	>300 cm/s	>3.5	<45	>0.15
High risk	>300 cm/s	>3.5	>45	<0.15
Moderate risk	180-300 cm/s	>2.0	>45	<0.15
Low risk	<180 cm/s	<2.0	>45	<0.15

GFV, Graft flow velocity; PSV, peak systolic velocity; Vr, PSV velocity ratio—PSV at the site of a stenosis divided by the PSV in a normal vessel segment proximal to the stenosis. Adapted from Bandyk DF, Seabrook GR, Moldenhauer P, Lavin J, Edward J, Cato R, et al. Hemodynamics of vein graft stenosis. *J Vasc Surg* 1988;8:688-95.

Whereas certain DUS velocity parameters were strongly correlated with vein graft failure, there was a poor correlation between high-grade stenosis on CT angiography and vein graft failure. A priori established subgroup analyses did not show statistically significant interactions to explain heterogeneity of effect. The systematic review demonstrated that the evidence base supporting routine DUS surveillance of infrainguinal vein grafts remains dependent on low-quality evidence. The review also concluded that considering the opportunity for early intervention offered by DUS and the noninvasive nature and low cost of this approach, DUS can be incorporated in surveillance protocols of lower extremity vein grafts that can be individualized on the basis of the setting and resources (Table IX).

**Prosthetic bypass.** In the randomized controlled trial reported by Lundell et al,<sup>150</sup> surveillance was not effective in predicting failure of prosthetic arterial bypass grafts in the lower extremity. Numerous retrospective case reviews suggest no utility of DUS in identifying such bypasses as “at risk” for failure. However, there is a suggestion that low graft flow velocities may predict failure and support the use of anticoagulation with warfarin.<sup>170-173</sup>

Summary of recommendations
1. We recommend clinical examination and ABI, with or without the addition of DUS, in the early postoperative period to provide a baseline for further follow-up after aortobifemoral bypass. This evaluation should be repeated at 6 and 12 months and then annually as long as there are no new signs or symptoms.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: C (Low)
2. We recommend clinical examination and ABI, with or without the addition of DUS, in the early postoperative period to provide a baseline for further follow-up after iliofemoral bypass. This evaluation should be repeated at 6 and 12 months and then annually as long as there are no new signs or symptoms.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: C (Low)

(Continued)

Continued.

3. We recommend clinical examination and ABI, with or without the addition of DUS, in the early postoperative period to provide a baseline for further follow-up after femoral-femoral bypass. This evaluation should be repeated at 6 and 12 months and then annually as long as there are no new signs or symptoms.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: C (Low)
4. We recommend clinical examination and ABI, with or without the addition of DUS, in the early postoperative period to provide a baseline for further follow-up after axillobifemoral bypass. This evaluation should be repeated at 6 and 12 months and then annually as long as there are no new signs or symptoms.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: C (Low)
5. Based on the high prevalence of abnormalities detected by DUS as well as the relatively low associated cost and risks, we recommend clinical examination, ABI, and DUS for infrainguinal vein graft surveillance. This should include an early postoperative baseline evaluation and follow-up at 3, 6, and 12 months and at least annually thereafter. More frequent surveillance may be considered when uncorrected abnormalities are identified on DUS or when alternative vein conduits (other than great saphenous vein) are used.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: B (Moderate)
6. After prosthetic infrainguinal bypass grafts, we recommend clinical examination and ABI, with or without the addition of DUS, in the early postoperative period to provide a baseline for further follow-up. This evaluation should be repeated at 6 and 12 months and then annually as long as there are no new signs or symptoms.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: B (Moderate)

## ENDOVASCULAR LOWER EXTREMITY ARTERIAL REVASCULARIZATION

With continuing innovation and development of endovascular therapy (EVT), the number of patients with

**Table IX.** Baseline characteristics of participants in the 15 included studies for the systematic review and meta-analysis on duplex ultrasound (DUS) surveillance for infrainguinal vein bypass grafts

Study	Patients, No.	Vein grafts, No.	Mean follow-up, months	Age, mean years	Type of vein graft, No.		Median ABPI	Indication for surgery	Schedule for surveillance	Type of vein used in graft (No.)	Revised vein, %
					First Reoperation	Time					
Buth, <sup>259</sup> 1991	147	155	36	NR	NR	NR	NR	Rest pain, ulcer, and gangrene in 76 patients Intermittent claudication in 39 patients One patient with aneurysmal disease	Every 3 months for the first year, and then every 6 months during the second year	In situ (78)	26.72
Dalsing, <sup>260</sup> 1995	143	112	17.6	NR	NR	NR	NR	NR	1, 3, 6, 9, and 12 months and every 6 months after that	In situ (74) Reversed (20) Nonreversed (2)	NR
Davies, <sup>168</sup> 2005	594	NR	18	70	NR	NR	0.48	Claudication in 182 patients Critical ischemia in 392 patients	1 day, 5 days, then 3, 6, 9, 12, and 18 months	In situ (555)	8
Ferris, <sup>261</sup> 2003	204	224	24	86.5	NR	NR	NR	Ischemic tissue loss (47%), ischemic rest pain (32%), disabling claudication (18%), and popliteal aneurysm (3%)	First year: every 3 months Second and third years: every 6 months Following years: once annually	In situ (175)	30
Golledge, <sup>262</sup> 1996	50	50	12	70	NR	NR	NR	NR	1.5, 3, 6, 9, and 12 months	Reversed (46) In situ (4)	NR
Idu, <sup>263</sup> 1993	187	201	21	NR	NR	NR	NR	Limb threatening (68.44%) Tissue necrosis (28.3%) Rest pain (40.1%)	First year: every 3 months Second year: every 6 months	Reversed (79) In situ (40)	NR
Ihlberg, <sup>166</sup> 1999	344	362	12	73	NR	NR	NR	Intermittent claudication (19%) Rest pain (28.5%) Ischemic ulcer (38.7%) Gangrene (14.2%) Popliteal aneurysm (1.3%)	1, 3, 6, 9, and 12 months	In situ (110)	NR
Ihlberg, <sup>165</sup> 1998	179	185	12	73	33	152	NR	Critical leg ischemia (128)	1, 3, 6, 9, and 12 months	In situ (118) Ex situ (34)	9.8
Laborde, <sup>264</sup> 1992	115	124	16	NR	NR	NR	NR	Ischemic rest pain and nonhealing ulcer (96%)	1, 3, 6, 9, and 12 months, then every 6 months	In situ (124)	30
Lewis, <sup>265</sup> 1998	143	148	1.5	69	NR	NR	NR	NR	NR	Reversed (40) In situ (103)	NR

**Table IX.** Continued.

Study	Patients, No.	Vein grafts, No.	Mean follow-up, months	Age, years, mean	Type of vein graft, No.		Median ABPI	Indication for surgery	Schedule for surveillance	Type of vein used in graft (No.)	Revised vein, %
					First Reoperation	Time					
Lundell, <sup>150</sup> 1995	156	106	36	75	88	18	NR	Intermittent claudication (2%) Rest pain (39.2%) Ischemic ulcer (34.1%) Gangrene (19.4%) Popliteal aneurysm (3%)	1, 3, 6, 9, 12, 15, 18, 21, 24, and 36 months	Reversed (15) In situ (88)	NR
Moody, <sup>266</sup> 1990	63	63	11.2	NR	NR	NR	NR	NR	1.5, 3, 6, 9, and 12 months	NR	NR
Polak, <sup>267</sup> 1990	14	15	2	NR	NR	NR	NR	NR	NR	Reversed (1) In situ (13)	NR
Stierli, <sup>268</sup> 1992	41	43	15	72	NR	NR	NR	NR	NR	In situ (25) Reversed (4) Nonreversed (14)	NR
Visser, <sup>269</sup> 2001	310	293	12	70.1	NR	NR	NR	37.2% of the surgery was due to critical limb ischemia 36.2% of the surgery was due to rest pain 26.6% of the surgery was due to intermittent claudication	3, 6, 9, and 12 months	NR	NR

ABPI, Ankle-brachial pressure index; NR, not reported.  
From Abu Dabrh AM, Mohammed K, Farah W, Haydour Q, Zierler RE, Wang Z, et al. Systematic review and meta-analysis of duplex ultrasound surveillance for infrainguinal vein bypass grafts. *J Vasc Surg* 2017;66:885-91.

peripheral arterial occlusive disease treated by these methods has been steadily increasing and has surpassed those revascularized by open surgical approaches. Techniques such as angioplasty, stenting, and atherectomy continue to expand in popularity.<sup>174-178</sup> Angioplasty includes “plain old balloon angioplasty,” cryoplasty, cutting balloon angioplasty, and medicated balloon angioplasty. Two general types of stents are available, self-expanding and balloon-expandable stents, both of which are available either bare or covered with polytetrafluoroethylene, bioabsorbable, and drug eluting. There are several categories of atherectomy or plaque removal devices, such as orbital, rotational, and laser. Often, multiple techniques or devices are used during a single intervention.

**Rationale for follow-up**

The optimal frequency and method for follow-up after EVT for lower extremity arterial occlusive disease have not been established. A follow-up program should be

founded on patient-based clinical outcomes, hemodynamic status, and anatomic assessment. Mounting evidence suggests that optimizing medical management of patients with peripheral arterial disease will improve bypass graft or EVT patency as well as improve mortality.<sup>179-182</sup> This is particularly important for patients with critical limb ischemia as suboptimal medical therapy in this population can increase the risk of major amputation or death by a factor of 8.<sup>183</sup>

Clinical assessment consists of a focused history, physical examination with evaluation of peripheral pulses, evaluation of existing wounds, inspection of arterial access sites, and ABI measurement. Medical management and lifestyle modifications should be optimized.<sup>184</sup> Patients at higher risk for contrast-induced nephropathy—those with a preintervention serum creatinine concentration >1.5 mg/dL or estimated glomerular filtration rate <60 mL/min/1.73 m<sup>2</sup> and those with diabetes mellitus—should have repeated laboratory testing at the first follow-up visit.<sup>185</sup> Patients who develop acute

kidney injury after angiography are at greater risk for progressive kidney disease.<sup>186</sup>

The most important limitation for all EVT in the lower extremity arteries is restenosis of the treated segment. Restenosis has generally been defined as >50% narrowing of the luminal diameter or >75% reduction of the cross-sectional area. The pathophysiologic mechanism of restenosis is complex and incompletely understood. After angioplasty, stent placement, or atherectomy, the local vessel reacts to the mechanical injury with an inflammatory response. The initial response is platelet activation and thrombus formation. This is then followed by activation of cytokines and growth factors, which leads to migration and proliferation of smooth muscle cells within the intima and media. This results in intimal thickening and increased extracellular matrix that may lead to restenosis because of the increase of intimal mass.<sup>187,188</sup> Simultaneously, arterial remodeling is occurring. The mechanisms of arterial remodeling are relatively unexplored compared with intimal hyperplasia. Neointimal hyperplasia appears to be the main cause of restenosis after stenting, whereas restenosis after angioplasty or atherectomy occurs from a combination of constrictive arterial remodeling along with neointimal hyperplasia.<sup>189-193</sup> Atherosclerotic changes can arise within the neointima, but this is usually a late phenomenon occurring >2 years after the intervention.

For those arteries treated by stenting, an additional concern has been the occurrence of stent fracture. Published reports suggest that the incidence of stent fracture ranges from 2% to 65%.<sup>194,195</sup> The clinical consequences of these fractures have been debated, but the most recent literature has suggested little clinical impact of stent fractures in the absence of restenosis or symptomatic decline.<sup>196,197</sup> Therefore, the role of surveillance specifically targeting the identification of stent fractures is not established.

### Methods and instrumentation

Despite the widespread use of EVT and the general availability of DUS for follow-up, the optimal frequency and techniques for surveillance in this setting are not well established. Some authors have suggested that clinical examination alone is more useful than DUS,<sup>198</sup> whereas others have reported that routine DUS surveillance can assist in preventing failure of EVT.<sup>199</sup> Velocity criteria for detecting restenosis in peripheral arteries after EVT are also not well established. DUS velocity and other noninvasive testing criteria have been extrapolated on the basis of experience with detection of lesions in untreated native arteries or stenoses in autogenous vein bypass grafts; however, these criteria may not be valid after EVT.<sup>33,34</sup>

The natural history of restenosis after EVT is still unclear, and therefore it is difficult to determine whether

reintervention is clinically beneficial. Reintervention is often more expensive than the initial procedure, especially for treating ISR, as costly techniques are typically employed, such as atherectomy, covered stents, and, recently, drug-eluting balloons and stents. In general, patency after treatment of restenosis is shorter than after treatment of the primary lesion, and multiple reinterventions are often undertaken.

A baseline DUS within the first month after EVT is recommended for all patients undergoing lower extremity interventions to establish a post-treatment baseline and to identify those with residual stenoses. Such residual stenoses are frequently missed on completion angiography and are associated with lower patency rates and increased risk of amputation.<sup>200</sup> The results of secondary interventions are also extremely poor in patients with residual stenotic lesions.<sup>201</sup>

### Aortoiliac revascularization

Excellent midterm and long-term results have been reported for EVT of occlusive aortoiliac disease. Studies have reported primary patency rates of 64% to 82% after 5 years.<sup>202-204</sup> In a large retrospective study, Soga et al<sup>205</sup> reported primary patency of aortoiliac stenting in 2096 patients to be 93%, 83%, and 78% at 1 year, 3 years, and 5 years. Reintervention was performed in 133 patients for restenosis. These excellent results are similar to those of other large reported series. There are few data concerning DUS surveillance after aortoiliac EVT. Spijkerboer et al<sup>206</sup> reported 72 iliac artery lesions that were treated with balloon angioplasty and followed by DUS at 1 month, 3 months, and 1 year. They used a Vr >2.5 to indicate a hemodynamically significant stenosis. Having identified restenosis by DUS, they found that the clinical outcomes in those with a significant residual stenosis or restenosis were not significantly different from those with a normal finding on DUS.

Investigators in Melbourne, Australia, determined that maximum PSV was the most consistent measurement for serial follow-up of iliac artery interventions.<sup>207</sup> Decisions for reintervention were based on clinical criteria rather than on DUS findings. Based on life-table curves, a threshold value of PSV >300 cm/s was identified. An increase to PSV >300 cm/s occurred in 55% of limbs that proceeded to reintervention for symptomatic restenosis and in three of six limbs that progressed to occlusion. Primary patency and assisted primary patency were 67% and 95%, respectively, at 4 years. The authors concluded that DUS surveillance could help identify limbs with residual or recurrent stenosis or occlusion after iliac artery EVT; however, their reintervention decisions were not guided by the DUS results. Most iliac artery interventions are done for claudication, and a restenosis or occlusion is unlikely to present with limb-threatening ischemia. Furthermore, treatment of iliac

artery restenosis is associated with very high success rates, even in cases of occlusions.

### Femoropopliteal revascularization

Anatomically, the femoropopliteal segment is the most commonly treated arterial segment, and DUS is commonly used to evaluate this segment before and after EVT. Even so, there are few studies investigating its utility and validity in detecting restenosis after EVT. Investigators at the University of Pittsburgh Medical Center conducted a retrospective review of 330 limbs that underwent stenting of the superficial femoral artery.<sup>208</sup> These patients were seen in follow-up at 1 month, 3 months, 6 months, and every 6 months thereafter. Follow-up included clinical evaluation, ABI measurement, and DUS. Criteria for high-grade restenosis were PSV >300 cm/s and Vr >3.5. Patients who had recurrent symptoms, evidence of recurrent or de novo stenosis on DUS, or significant decrease in ABI (>0.15) underwent angiography and intervention when it was deemed appropriate. Seventy-eight limbs had DUS images and angiography within 30 days of each other. Multiple potential thresholds for PSV and Vr were analyzed for sensitivity, specificity, positive predictive value, and negative predictive value to determine optimal criteria for >50% stenosis and >80% stenosis. The combination of PSV >190 cm/s and Vr >1.5 to identify a >50% stenosis was associated with a sensitivity of 85%, specificity of 95%, positive predictive value of 98%, and negative predictive value of 67%. Combining a PSV >275 cm/s and a Vr >3.5 to identify an >80% stenosis was associated with a sensitivity of 74%, specificity of 94%, positive predictive value of 88%, and negative predictive value of 85%. The authors concluded that these criteria may help determine which patients with superficial femoral artery stents have significant restenosis, thereby guiding secondary interventions to optimize assisted patency rates.

Shrikhande et al<sup>14</sup> reported a similar surveillance protocol with follow-up at 1 month, 3 months, and 6 months and yearly thereafter. They examined all forms of EVT—percutaneous transluminal angioplasty, stenting, and atherectomy—for femoropopliteal or tibial artery occlusive disease. A comparable reintervention strategy was followed; those with recurrent or de novo stenosis by DUS or recurrent symptoms underwent angiography and reintervention when it was considered appropriate. They concluded that a PSV >223 cm/s had both high specificity and sensitivity in the femoropopliteal segment to detect a >70% stenosis. Using a Vr >2.5 also identified a high percentage of patients with a >70% angiographic stenosis. A poor correlation was found between DUS findings and angiographic measurements in the tibial vessels.

Special consideration may be given to follow-up for complex stenting of the femoropopliteal segment as this has the potential for the most difficult reinterventions for restenosis. The 3-year results of the VIABAHN

Endoprosthesis vs Bare Nitinol Stent in the Treatment of Long Lesion ( $\geq 8$  cm) Superficial Femoral Artery Occlusive Disease (VIBRANT) trial, which compared the long-term outcomes of complex superficial femoral artery disease treated with Viabahn stent grafts with those treated with bare-metal nitinol stents, showed similar albeit disappointing primary patency rates of 24% and 26%, respectively.<sup>209</sup> Assisted primary patency was better for bare-metal stents (89% vs 70%), although secondary patency rates were similar (89% vs 80%). More than 30% of patients in both groups underwent at least one reintervention. The authors concluded that a rigorous surveillance program and judicious reintervention resulted in high assisted primary and secondary patency rates and sustained symptomatic improvement. This reintervention rate is similar to that in other studies that employed DUS surveillance protocols.

Whereas multiple series have shown encouraging assisted primary patency rates for EVT, it is unclear whether reinterventions in the absence of symptoms conferred a clinical benefit. Bui et al<sup>5</sup> investigated the natural history of restenosis in the femoropopliteal segment after EVT. Ninety-four limbs were entered into a DUS surveillance protocol after EVT, and the mean follow-up was 22 months. Moderate stenosis was defined by a PSV of 200 to 300 cm/s or a Vr of 2 to 3, and severe stenosis was defined by a PSV >300 cm/s or a Vr >3. In general, reintervention was performed only on the basis of symptoms regardless of DUS findings, which clouds any judgment of the utility of surveillance, although it offers some insight into the natural history of restenosis. Severe stenosis developed in 25 of 94 limbs, of which 13 (52%) presented with recurrent symptoms. Nine severe stenoses were found in asymptomatic limbs with no intervention required at mean follow-up of 10 months. A total of 11 femoropopliteal segments occluded, and the status of the treated arterial segment documented on the preceding DUS examination was normal in one, moderate stenosis in nine, and severe stenosis in one. The authors concluded that in contrast to autogenous vein grafts, severe restenosis after EVT is less likely to thrombose, and the treated segment is more often patent at the time of clinical deterioration. In addition, reintervention based on clinical findings rather than on the DUS criteria established for vein graft reintervention may be more beneficial after EVT.

ISR has been a particularly challenging problem in the femoropopliteal segment. A comparison of the severity of ISR and outcomes of reintervention revealed similar procedural success for occlusions and nonocclusive restenosis (96% vs 100%), although the development of subsequent stenosis and occlusion was higher for those who initially had occlusion.<sup>210</sup> Patients in this study underwent routine DUS surveillance every 3 months for 1 year and every 6 to 12 months thereafter. Reintervention for ISR was performed primarily for

**Table X.** Threshold criteria for grading lower extremity angioplasty site and in-stent restenosis (ISR)

Reference	Stenosis category	PSV	Velocity ratio (Vr)	EDV	Distal artery flow pattern
Tinder, <sup>1</sup> 2008	<50%	<180 cm/s	<2	—	Normal
	>50%, Moderate	180-300 cm/s	2-3.5	>0 cm/s	Monophasic
	>70%, Severe	>300 cm/s	>3.5	>45 cm/s	Damped, monophasic
	Occluded	—	—	No flow detected	Damped, monophasic
Baril, <sup>208</sup> 2009	<50%	<190 cm/s	<1.5		
	>50%	190-275 cm/s	1.5-3.5		
	>80%	>275 cm/s	>3.5		
	Occluded	No flow detected			
Bui, <sup>3</sup> 2012	Normal	<200 cm/s	<2.0		
	Moderate	200-300 cm/s	2.0-3.0		
	Severe	>300 cm/s	>3.0		
Shrikhande, <sup>4</sup> 2011	>70%	>223 cm/s	>2.5		

EDV, End-diastolic velocity; PSV, peak systolic velocity; Vr, PSV velocity ratio—PSV at the site of a stenosis divided by the PSV in a normal vessel segment proximal to the stenosis.

claudication in patients with nonocclusive ISR, whereas 56% of patients with occlusive ISR underwent intervention for critical limb ischemia. No patients initially treated for claudication presented with critical limb ischemia on developing ISR.

This contrasted with the results of Vartanian et al,<sup>211</sup> who examined reinterventions for bare-metal stents and stent grafts in the femoropopliteal segment. The proportion of claudicants presenting with critical limb ischemia or acute limb ischemia after reocclusions was 9% for bare-metal stents and 17% for stent grafts. Yeo et al<sup>212</sup> examined their experience with treatment of femoropopliteal ISR. Indications for intervention were DUS evidence of restenosis with or without associated symptoms. EVT was performed on 22 limbs. Primary patency was 55% and 48% at 6 months and 1 year, respectively. Nine of these 22 interventions were on occlusions, which had an 89% technical success rate.

In patients with claudication, Jones et al<sup>213</sup> evaluated the correlation between DUS-detected restenosis and symptom recurrence after EVT. This was a retrospective series of 88 limbs in 71 patients providing 183 pairs of DUS scans and symptom status to compare. The primary level of disease treated was in the femoropopliteal arteries in 56% and the iliac arteries in 44%. Eighty-two percent of patients underwent angioplasty with adjunctive bare-metal stent placement. Using ROC analysis, a Vr >2.5 was accurate in distinguishing symptomatic from asymptomatic patients after EVT of the femoropopliteal arteries, but there was little correlation between DUS and symptom status after EVT of the iliac arteries. There were 12 limbs that were asymptomatic with a Vr >2.5, of which 5 eventually developed symptoms and required reintervention. Some of the threshold criteria for grading of femoropopliteal angioplasty site restenosis and ISR are summarized in [Table X](#).

### Tibial revascularization

EVT in the tibial arteries is associated with low patency rates and high restenosis rates.<sup>214-216</sup> Contrary to interventions performed in the iliac or femoropopliteal arteries, EVT in the tibial arteries is almost exclusively done for critical limb ischemia. Saqib et al<sup>217</sup> reported on the outcomes and predictors of restenosis for EVT of the tibial arteries. A PSV >300 cm/s and Vr of >3.5 were used as indicators of severe restenosis. Patients with worsening wounds were re-evaluated with angiography regardless of the DUS findings. Tibial artery restenosis or occlusion occurred in 96 limbs (41%). Of these, only 10 limbs (10%) were asymptomatic. Limb loss was significantly higher in patients with restenosis or occlusion compared with those with continued primary patency (27% vs 4%). Of those patients with restenosis, 44% underwent repeated EVT and 21% required open surgical bypass. The need for further interventions in patients with repeated EVT was high (36%). There have been conflicting reports about the utility of DUS after EVT in the tibial arteries, with some authors finding poor correlation to angiographic findings,<sup>4</sup> whereas others have found it to be reliable for surveillance after interventions.<sup>218</sup>

Critical limb ischemia after EVT, especially with tissue loss, does not have a benign course. Restenosis and symptomatic deterioration are common and can occur early after revascularization. Frequent follow-up is essential to assist with limb salvage and to limit cardiovascular morbidity and mortality; however, the utility of a DUS surveillance program with current diagnostic criteria is of questionable value if frequent clinical assessment is performed. The efficacy of repeated tibial artery intervention based on DUS findings in the presence of clinical stability or improvement has not been established. Thus, clinical assessment and ABI may provide better data for clinical decision-making than DUS.

Summary of recommendations
1. We recommend clinical examination, ABI, and DUS within the first month after aortoiliac segment EVT to provide a post-treatment baseline and to evaluate for residual stenosis. Clinical examination and ABI, with or without the addition of DUS, should be performed at 6 and 12 months and then annually as long as there are no new signs or symptoms.
Strength of Recommendation: 1 (Strong)
Quality of Evidence: C (Low)
2. We suggest clinical examination, ABI, and DUS within the first month after femoropopliteal artery EVT to provide a post-treatment baseline and to evaluate for residual stenosis. Continued surveillance at 3 months and then every 6 months is indicated for the following:
A. Patients with interventions using stents because of the potential increased difficulty of treating an occlusive vs stenotic in-stent lesion.
B. Patients undergoing angioplasty or atherectomy for critical limb ischemia because of increased risk of recurrent critical limb ischemia should the intervention fail.
Strength of Recommendation: 2 (Weak)
Quality of Evidence: C (Low)
3. We suggest clinical examination, ABI, and DUS within the first month after tibial artery EVT to provide a post-treatment baseline and to evaluate for residual stenosis. Continued surveillance at 3 months and then every 6 months should be considered. Those patients with a deteriorating clinical vascular examination, return of rest pain, nonhealing wounds, or new tissue loss should undergo repeated DUS.
Strength of Recommendation: 2 (Weak)
Quality of Evidence: C (Low)

## CONCLUSIONS

It is generally accepted that achieving optimal outcomes from open surgical and endovascular procedures depends on periodic follow-up and appropriate reintervention. The options for follow-up range from a simple vascular history and physical examination (often including ABI measurement for procedures involving the lower extremity arteries) to sophisticated imaging methods such as CT or MR angiography and more invasive catheter angiography. Noninvasive vascular laboratory tests, particularly DUS, are ideally suited for this purpose because they are safe and relatively low in cost, and they provide objective anatomic and physiologic information that can be used to assess the durability of a vascular intervention over time. The goal of routine surveillance is to identify intervention sites that are at risk for failure, even in the absence of signs or symptoms. However, this approach is justified only if the consequences of failure are severe and early reintervention can improve the outcome. Additional requirements are

the availability of accurate testing methods with clinically relevant threshold criteria and appropriate follow-up or testing intervals.

Although there are many publications on follow-up and surveillance after a wide range of open surgical and endovascular procedures, there has not been a comprehensive review of this topic that has provided detailed practice guidelines. The expert panel appointed by the Clinical Practice Council of the Society for Vascular Surgery reviewed the relevant literature in an effort to make specific recommendations for follow-up after commonly performed arterial procedures. With the possible exception of DUS for surveillance of infrainguinal vein bypass grafts, the panel found a general lack of high-quality evidence to serve as a basis for these recommendations. Therefore, the majority of recommendations in this paper are based on observational studies, committee consensus, and indirect evidence. Among the total of 24 recommendations made by the panel, 19 are *strong* and 5 are *weak*; however, the quality of evidence supporting these recommendations was graded *moderate* in 11 and *low* in 13, whereas none were graded *high*. Consequently, it is likely that these recommendations will require updating as new evidence emerges, particularly with regard to the frequency and duration of follow-up testing.

This review clearly shows that there is a pressing need for better clinical evidence on all aspects of follow-up after vascular surgery procedures. These include the role of routine surveillance, modes of failure, indications for reintervention, and resulting outcomes. Because the vascular laboratory plays a central role in follow-up, there are abundant opportunities for clinical research involving testing methods, threshold criteria, and surveillance protocols. Until more and better data are available, the recommendations on follow-up from this panel can provide some guidance. However, the variety of currently performed open surgical and endovascular procedures and the ongoing development of new interventional techniques will continue to present challenges to vascular surgeons as they pursue the best outcomes possible for their patients.

## AUTHOR CONTRIBUTIONS

Conception and design: RZ, WJ, MM  
 Analysis and interpretation: RZ, WJ, BL, FM, SL, JF, WP, AH, MM  
 Data collection: Not applicable  
 Writing the article: RZ, WJ, BL, FM, SL, JF, WP, AH  
 Critical revision of the article: RZ, WJ, BL, FM, SL, JF, WP, MM  
 Final approval of the article: RZ, WJ, BL, FM, SL, JF, WP, AH, MM  
 Statistical analysis: Not applicable  
 Obtained funding: Not applicable  
 Overall responsibility: RZ  
 RZ and WJ contributed equally and share first authorship.

## REFERENCES

- Tinder CN, Chavenpun JP, Bandyk DF, Armstrong PA, Back MR, Johnson BL, et al. Efficacy of duplex ultrasound surveillance after infrainguinal vein bypass may be enhanced by identification of characteristics predictive of graft stenosis development. *J Vasc Surg* 2008;48:613-8.
- Shames ML. Duplex surveillance of lower extremity endovascular interventions. *Perspect Vasc Surg Endovasc Ther* 2007;19:370-4; discussion: 375.
- Bui TD, Mills JL, Ihnat DM, Gruessner AC, Goshiima KR, Hughes JD. The natural history of duplex detected stenosis after femoropopliteal endovascular therapy suggests questionable clinical utility of routine duplex surveillance. *J Vasc Surg* 2012;55:346-52.
- Shrikhande GV, Graham AR, Aparajita R, Gallagher KA, Morrissey NJ, McKinsey JF, et al. Determining criteria for predicting stenosis with ultrasound duplex after endovascular intervention in infrainguinal lesions. *Ann Vasc Surg* 2011;25:454-60.
- Guyatt G, Gutterman D, Baumann MH, Addrizzo-Harris D, Hylek EM, Phillips B, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians task force. *Chest* 2006;129:174-81.
- Murad MH, Montori VM, Sidawy AN, Ascher E, Meissner MH, Chaikof EL, et al. Guideline methodology of the Society for Vascular Surgery including experience with the GRADE framework. *J Vasc Surg* 2011;53:1375-80.
- Sidawy AN, Spergel LM, Besarab A, Allon M, Jennings WC, Padberg FT Jr, 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(Suppl):2S-25S.
- North American Symptomatic Carotid Endarterectomy Trial Collaborators, Barnett HJ, Taylor DW, Haynes RB, Sackett DL, Peerless SJ, et al. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med* 1991;325:445-53.
- Barnett HJ, Taylor DW, Eliasziw M, Fox AJ, Ferguson GC, Haynes RB, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. *N Engl J Med* 1998;339:1415-25.
- Hobson RW 2nd, Weiss DC, Fields WS, Goldstone J, Moore WS, Towne JB, et al. Efficacy of carotid endarterectomy for asymptomatic carotid stenosis. The Veterans Affairs Cooperative Study Group. *N Engl J Med* 1993;328:221-7.
- Goldstein MR. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. Endarterectomy for asymptomatic carotid artery stenosis. *JAMA* 1995;274:1505.
- Brott TG, Hobson RW 2nd, Howard G, Roubin GS, Clark WM, Brooks W, et al. Stenting versus endarterectomy for treatment of carotid-artery stenosis. *N Engl J Med* 2010;363:11-23.
- Arquiza C, Trinquart L, Touboul PJ, Long A, Feasson S, Terriat B, et al. Restenosis is more frequent after carotid stenting than after endarterectomy: the EVA-3S study. *Stroke* 2011;42:1015-20.
- Gurm HS, Yadav JS, Fayad P, Katzen BT, Mishkel GJ, Bajwa TK, et al. Long-term results of carotid stenting versus endarterectomy in high-risk patients. *N Engl J Med* 2008;358:1572-9.
- Eckstein HH, Ringleb P, Allenberg JR, Berger J, Fraedrich G, Hacke W, et al. Results of the Stent-Protected Angioplasty versus Carotid Endarterectomy (SPACE) study to treat symptomatic stenoses at 2 years: a multinational, prospective, randomised trial. *Lancet Neurol* 2008;7:893-902.
- Lal BK. Recurrent carotid stenosis after CEA and CAS: diagnosis and management. *Semin Vasc Surg* 2007;20:259-66.
- Lal BK, Kaperonis EA, Cuadra S, Kapadia I, Hobson RW 2nd. Patterns of in-stent restenosis after carotid artery stenting: classification and implications for long-term outcome. *J Vasc Surg* 2007;46:833-40.
- Moore WS, Kempczinski RF, Nelson JJ, Toole JF. Recurrent carotid stenosis: results of the asymptomatic carotid atherosclerosis study. *Stroke* 1998;29:2018-25.
- Lal BK, Hobson RW 2nd, Goldstein J, Geohagan M, Chakhtoura E, Pappas PJ, et al. In-stent recurrent stenosis after carotid artery stenting: life table analysis and clinical relevance. *J Vasc Surg* 2003;38:1162-8.
- Lal BK, Beach KW, Roubin GS, Lutsep HL, Moore WS, Malas MB, et al. Restenosis after carotid artery stenting and endarterectomy: a secondary analysis of CREST, a randomised controlled trial. *Lancet Neurol* 2012;11:755-63.
- Lattimer CR, Burnand KG. Recurrent carotid stenosis after carotid endarterectomy. *Br J Surg* 1997;84:1206-19.
- Healy DA, Zierler RE, Nicholls SC, Clowes AW, Primozich JF, Bergelin RO, et al. Long-term follow-up and clinical outcome of carotid restenosis. *J Vasc Surg* 1989;10:662-8.
- O'Hara PJ, Hertzner NR, Karafa MT, Mascha EJ, Krajewski LP, Beven EG. Reoperation for recurrent carotid stenosis: early results and late outcome in 199 patients. *J Vasc Surg* 2001;34:5-12.
- Mansour MA, Kang SS, Baker WH, Watson WC, Littooy FN, Labropoulos N, et al. Carotid endarterectomy for recurrent stenosis. *J Vasc Surg* 1997;25:877-83.
- DeGroot RD, Lynch TG, Jamil Z, Hobson RW 2nd. Carotid restenosis: long-term noninvasive follow-up after carotid endarterectomy. *Stroke* 1987;18:1031-6.
- Abizaid A, Kornowski R, Mintz GS, Hong MK, Abizaid AS, Mehran R, et al. The influence of diabetes mellitus on acute and late clinical outcomes following coronary stent implantation. *J Am Coll Cardiol* 1998;32:584-9.
- Mehran R, Dangas G, Abizaid AS, Mintz GS, Lansky AJ, Satler LF, et al. Angiographic patterns of in-stent restenosis: classification and implications for long-term outcome. *Circulation* 1999;100:1872-8.
- Willfort-Ehringer A, Ahmadi R, Gessl A, Gschwandtner ME, Haumer A, Lang W, et al. Neointimal proliferation within carotid stents is more pronounced in diabetic patients with initial poor glycaemic state. *Diabetologia* 2004;47:400-6.
- Faught WE, Mattos MA, van Bemmelen PS, Hodgson KJ, Barkmeier LD, Ramsey DE, et al. Color-flow duplex scanning of carotid arteries: new velocity criteria based on receiver operator characteristic analysis for threshold stenoses used in the symptomatic and asymptomatic carotid trials. *J Vasc Surg* 1994;19:818-27.
- Robbin ML, Lockhart ME, Weber TM, Vitek JJ, Smith JK, Yadav J, et al. Carotid artery stents: early and intermediate follow-up with Doppler US. *Radiology* 1997;205:749-56.
- Ringer AJ, German JW, Guterman LR, Hopkins LN. Follow-up of stented carotid arteries by Doppler ultrasound. *Neurosurgery* 2002;51:639-43.
- Lal BK, Hobson RW 2nd, Goldstein J, Chakhtoura EY, Duran WN. Carotid artery stenting: is there a need to revise ultrasound velocity criteria? *J Vasc Surg* 2004;39:58-66.
- Lal BK, Hobson RW 2nd, Tofighi B, Kapadia I, Cuadra S, Jamil Z. Duplex ultrasound velocity criteria for the stented carotid artery. *J Vasc Surg* 2007;47:63-73.
- Stanziale SF, Wholey MH, Boules TN, Selzer F, Makaroun MS. Determining in-stent stenosis of carotid arteries by duplex ultrasound criteria. *J Endovasc Ther* 2005;12:346-53.

35. Chi YW, White CJ, Woods TC, Goldman CK. Ultrasound velocity criteria for carotid in-stent restenosis. *Catheter Cardiovasc Interv* 2007;69:349-54.
36. Chahwan S, Miller MT, Pigott JP, Whalen RC, Jones L, Comerota AJ. Carotid artery velocity characteristics after carotid artery angioplasty and stenting. *J Vasc Surg* 2007;45:523-6.
37. Setacci C, Chisci E, Setacci F, Iacoponi F, de Donato G. Grading carotid intrastent stenosis, a 6 year follow-up study. *Stroke* 2008;39:1189-96.
38. AbuRahma AF, Abu-Halimah S, Bensenhaver J, Dean LS, Keiffer T, Emmett M, et al. Optimal carotid duplex velocity criteria for defining the severity of carotid in-stent restenosis. *J Vasc Surg* 2008;48:589-94.
39. Beach KW, Leotta DF, Zierler RE. Carotid Doppler velocity measurements and anatomic stenosis: correlation is futile. *Vasc Endovasc Surg* 2012;46:466-74.
40. AbuRahma AF, Stone P, Deem S, Dean LS, Keiffer T, Deem E. Proposed duplex velocity criteria for carotid restenosis following carotid endarterectomy with patch closure. *J Vasc Surg* 2009;50:286-91.
41. Benzing T, Wilhoit C, Wright S, McCann PA, Lessner S, Brothers TE. Standard duplex criteria overestimate the degree of stenosis after eversion carotid endarterectomy. *J Vasc Surg* 2015;61:1457-63.
42. Grabenwoger M, Alfonso F, Bachet J, Bonser R, Czerny M, Eggebrecht H, et al. Thoracic endovascular aortic repair (TEVAR) for the treatment of aortic diseases: a position statement from the European Association for Cardio-Thoracic Surgery (EACTS) and the European Society of Cardiology (ESC), in collaboration with the European Association of Percutaneous Cardiovascular Interventions (EAPCI). *Eur J Cardiothorac Surg* 2012;42:17-24.
43. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE Jr, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease: executive summary. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. *Catheter Cardiovasc Interv* 2010;76:E43-86.
44. Nienaber CA, Rousseau H, Eggebrecht H, Kische S, Fattori R, Rehders TC, et al. Randomized comparison of strategies for type B aortic dissection: the INvestigation of STEnt Grafts in Aortic Dissection (INSTEAD) trial. *Circulation* 2009;120:2519-28.
45. Brunkwall J, Lammer J, Verhoeven E, Taylor P. ADSORB: a study on the efficacy of endovascular grafting in uncomplicated acute dissection of the descending aorta. *Eur J Vasc Endovasc Surg* 2012;44:31-6.
46. Patel HJ, Hemmila MR, Williams DM, Diener AC, Deeb GM. Late outcomes following open and endovascular repair of blunt thoracic aortic injury. *J Vasc Surg* 2011;53:615-20.
47. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma multicenter study. *J Trauma* 2008;64:561-70; discussion: 570-1.
48. Zoli S, Trabatttoni P, Dainese L, Annoni A, Saccu C, Fumagalli M, et al. Cumulative radiation exposure during thoracic endovascular aneurysm repair and subsequent follow-up. *Eur J Cardiothorac Surg* 2012;42:254-9; discussion: 259-60.
49. Kasirajan K, Dake MD, Lumsden A, Bavaria J, Makaroun MS. Incidence and outcomes after infolding or collapse of thoracic stent grafts. *J Vasc Surg* 2012;55:652-8; discussion: 658.
50. Hansen CJ, Bui H, Donayre CE, Aziz I, Kim B, Kopchok G, et al. Complications of endovascular repair of high-risk and emergent descending thoracic aortic aneurysms and dissections. *J Vasc Surg* 2004;40:228-34.
51. Thrumurthy SG, Karthikesalingam A, Patterson BO, Holt PJ, Hinchliffe RJ, Loftus IM, et al. A systematic review of mid-term outcomes of thoracic endovascular repair (TEVAR) of chronic type B aortic dissection. *Eur J Vasc Endovasc Surg* 2011;42:632-47.
52. Kang WC, Greenberg RK, Mastracci TM, Eagleton MJ, Hernandez AV, Pujara AC, et al. Endovascular repair of complicated chronic distal aortic dissections: intermediate outcomes and complications. *J Thorac Cardiovasc Surg* 2011;142:1074-83.
53. D'Souza S, Duncan A, Aguila F, Oderich G, Ricotta J, Kalra M, et al. TEVAR for non-aneurysmal thoracic aortic pathology. *Catheter Cardiovasc Interv* 2009;74:783-6.
54. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). *Eur Heart J* 2014;35:2873-926.
55. Khojnezhad A, Azizzadeh A, Donayre CE, Matsumoto A, Velazquez O, White R, et al. Results of a multicenter, prospective trial of thoracic endovascular aortic repair for blunt thoracic aortic injury (RESCUE trial). *J Vasc Surg* 2013;57:899-905.
56. Khojnezhad A, Donayre CE, Azizzadeh A, White R; RESCUE Investigators. One-year results of thoracic endovascular aortic repair for blunt thoracic aortic injury (RESCUE trial). *J Thorac Cardiovasc Surg* 2015;149:155-61.
57. Alsac JM, Khantalini I, Julia P, Achouh P, Farahmand P, Capdevila C, et al. The significance of endoleaks in thoracic endovascular aneurysm repair. *Ann Vasc Surg* 2011;25:345-51.
58. Nienaber CA, Kische S, Ince H. Thoracic aortic stent-graft devices: problems, failure modes, and applicability. *Semin Vasc Surg* 2007;20:81-9.
59. Makaroun MS, Dillavou ED, Wheatley GH, Cambria RP; Gore TAG Investigators. Five-year results of endovascular treatment with the Gore TAG device compared with open repair of thoracic aortic aneurysms. *J Vasc Surg* 2008;47:912-8.
60. Matsumura JS, Cambria RP, Dake MD, Moore RD, Svensson LG, Snyder S, et al. International controlled clinical trial of thoracic endovascular aneurysm repair with the Zenith TX2 endovascular graft: 1-year results. *J Vasc Surg* 2008;47:247-57; discussion: 257.
61. Fairman RM, Criado F, Farber M, Kwolek C, Mehta M, White R, et al. Pivotal results of the Medtronic Vascular Talent Thoracic Stent Graft System: the VALOR trial. *J Vasc Surg* 2008;48:546-54.
62. Grabenwoger M, Fleck T, Ehrlich M, Czerny M, Hutschala D, Schoder M, et al. Secondary surgical interventions after endovascular stent-grafting of the thoracic aorta. *Eur J Cardiothorac Surg* 2004;26:608-13.
63. Czerny M, Funovics M, Sodeck G, Dumfarth J, Schoder M, Juraszek A, et al. Long-term results of thoracic endovascular aortic repair in atherosclerotic aneurysms involving the descending aorta. *J Thorac Cardiovasc Surg* 2010;140(Suppl):S179-84; discussion: S185-90.

64. Leurs LJ, Harris PL, Buth J; EUROSTAR Collaborators. Secondary interventions after elective endovascular repair of degenerative thoracic aortic aneurysms: results of the European collaborators registry (EUROSTAR). *J Vasc Interv Radiol* 2007;18:491-5.
65. Czerny M, Grimm M, Zimpfer D, Rodler S, Gottardi R, Hutschala D, et al. Results after endovascular stent graft placement in atherosclerotic aneurysms involving the descending aorta. *Ann Thorac Surg* 2007;83:450-5.
66. Canaud L, Alric P, Branchereau P, Joyeux F, Hireche K, Berthet JP, et al. Open versus endovascular repair for patients with acute traumatic rupture of the thoracic aorta. *J Thorac Cardiovasc Surg* 2011;142:1032-7.
67. Girdauskas E, Falk V, Kuntze T, Borger MA, Schmidt A, Scheinert D, et al. Secondary surgical procedures after endovascular stent grafting of the thoracic aorta: successful approaches to a challenging clinical problem. *J Thorac Cardiovasc Surg* 2008;136:1289-94.
68. Jonker FH, Schlosser FJ, Geirsson A, Sumpio BE, Moll FL, Muhs BE, et al. Endograft collapse after thoracic aortic repair. *J Endovasc Ther* 2010;17:725-34.
69. Tsai TT, Evangelista A, Nienaber CA, Myrmet T, Meinhardt G, Cooper JV, et al. Partial thrombosis of the false lumen in patients with acute type B aortic dissection. *N Engl J Med* 2007;357:349-59.
70. Trimarchi S, Tolenaar JL, Jonker FH, Murray B, Tsai TT, Eagle KA, et al. Importance of false lumen thrombosis in type B aortic dissection prognosis. *J Thorac Cardiovasc Surg* 2013;145(Suppl):S208-12.
71. Steuer J, Eriksson MO, Nyman R, Bjorck M, Wanhainen A. Early and long-term outcome after thoracic endovascular aortic repair (TEVAR) for acute complicated type B aortic dissection. *Eur J Vasc Endovasc Surg* 2011;41:318-23.
72. Fattori R, Nienaber CA, Rousseau H, Beregi JP, Heijmen R, Grabenwoger M, et al. Results of endovascular repair of the thoracic aorta with the Talent Thoracic stent graft: the Talent Thoracic Retrospective Registry. *J Thorac Cardiovasc Surg* 2006;132:332-9.
73. Ueda T, Fleischmann D, Dake MD, Rubin GD, Sze DY. Incomplete endograft apposition to the aortic arch: bird-beak configuration increases risk of endoleak formation after thoracic endovascular aortic repair. *Radiology* 2010;255:645-52.
74. Czerny M, Funovics M, Sodeck G, Dumfarth J, Schoder M, Juraszek A, et al. Results after thoracic endovascular aortic repair in penetrating atherosclerotic ulcers. *Ann Thorac Surg* 2011;92:562-6; discussion: 566-7.
75. Piffaretti G, Mariscalco C, Lomazzi C, Rivolta N, Riva F, Tozzi M, et al. Predictive factors for endoleaks after thoracic aortic aneurysm endograft repair. *J Thorac Cardiovasc Surg* 2009;138:880-5.
76. Hausegger KA, Oberwalder P, Tiesenhausen K, Tauss J, Stanger O, Schedlbauer P, et al. Intentional left subclavian artery occlusion by thoracic aortic stent-grafts without surgical transposition. *J Endovasc Ther* 2001;8:472-6.
77. Matsumura JS, Melissano G, Cambria RP, Dake MD, Mehta S, Svensson LC, et al. Five-year results of thoracic endovascular aortic repair with the Zenith TX2. *J Vasc Surg* 2014;60:1-10.
78. Dua A, Kuy S, Lee CJ, Upchurch GR Jr, Desai SS. Epidemiology of aortic aneurysm repair in the United States from 2000 to 2010. *J Vasc Surg* 2014;59:1512-7.
79. Chaikof EL, Dalman RL, Eskandari MK, Jackson BM, Lee WA, Mansour MA, et al. The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. *J Vasc Surg* 2018;67:2-77.
80. Hirsch AT, Haskal ZJ, Hertzner NR, Bakal CW, Creager MA, Halperin JL, et al. ACC/AHA 2005 guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): executive summary a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease) endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; and Vascular Disease Foundation. *J Am Coll Cardiol* 2006;47:1239-312.
81. Brenner DJ, Hall EJ. Computed tomography—an increasing source of radiation exposure. *N Engl J Med* 2007;357:2277-84.
82. AbuRahma AF, Welch CA, Mullins BB, Dyer B. Computed tomography versus color duplex ultrasound for surveillance of abdominal aortic stent-grafts. *J Endovasc Ther* 2005;12:568-73.
83. Raman KC, Missig-Carroll N, Richardson T, Muluk SC, Makaroun MS. Color-flow duplex ultrasound scan versus computed tomographic scan in the surveillance of endovascular aneurysm repair. *J Vasc Surg* 2003;38:645-51.
84. AbuRahma AF. Fate of endoleaks detected by CT angiography and missed by color duplex ultrasound in endovascular grafts for abdominal aortic aneurysms. *J Endovasc Ther* 2006;13:490-5.
85. Sun Z. Diagnostic value of color duplex ultrasonography in the follow-up of endovascular repair of abdominal aortic aneurysm. *J Vasc Interv Radiol* 2006;17:759-64.
86. Ashoke R, Brown LC, Rodway A, Choke E, Thompson MM, Greenhalgh RM, et al. Color duplex ultrasonography is insensitive for the detection of endoleak after aortic endografting: a systematic review. *J Endovasc Ther* 2005;12:297-305.
87. Collins JT, Boros MJ, Combs K. Ultrasound surveillance of endovascular aneurysm repair: a safe modality versus computed tomography. *Ann Vasc Surg* 2007;21:671-5.
88. Sandford RM, Bown MJ, Fishwick G, Murphy F, Naylor M, Sensier Y, et al. Duplex ultrasound scanning is reliable in the detection of endoleak following endovascular aneurysm repair. *Eur J Vasc Endovasc Surg* 2006;32:537-41.
89. Manning BJ, O'Neill SM, Haider SN, Colgan MP, Madhavan P, Moore DJ. Duplex ultrasound in aneurysm surveillance following endovascular aneurysm repair: a comparison with computed tomography aortography. *J Vasc Surg* 2009;49:60-5.
90. Bargellini I, Cioni R, Napoli V, Petrucci P, Vignali C, Cicorelli A, et al. Ultrasonographic surveillance with selective CTA after endovascular repair of abdominal aortic aneurysm. *J Endovasc Ther* 2009;16:93-104.
91. Napoli V, Bargellini I, Sardella SG, Petrucci P, Cioni R, Vignali C, et al. Abdominal aortic aneurysm: contrast-enhanced US for missed endoleaks after endoluminal repair. *Radiology* 2004;233:217-25.
92. Henaio EA, Hodge MD, Felkai DD, McCollum CH, Noon GP, Lin PH, et al. Contrast-enhanced duplex surveillance after endovascular abdominal aortic aneurysm repair: improved efficacy using a continuous infusion technique. *J Vasc Surg* 2006;43:259-64; discussion: 264.
93. Iezzi R, Basilico R, Giancristofaro D, Pascali D, Cotroneo AR, Storto ML. Contrast-enhanced ultrasound versus color duplex ultrasound imaging in the follow-up of patients

- after endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 2009;49:552-60.
94. Sternbergh WC 3rd, Greenberg RK, Chuter TA, Tonnessen BH; Zenith Investigators. Redefining post-operative surveillance after endovascular aneurysm repair: recommendations based on 5-year follow-up in the US Zenith multicenter trial. *J Vasc Surg* 2008;48:278-84; discussion: 284-5.
95. Go MR, Barbato JE, Rhee RY, Makaroun MS. What is the clinical utility of a 6-month computed tomography in the follow-up of endovascular aneurysm repair patients? *J Vasc Surg* 2008;47:1181-6; discussion: 1186-7.
96. Chaer RA, Gushchin A, Rhee R, Marone L, Cho JS, Leers S, et al. Duplex ultrasound as the sole long-term surveillance method post-endovascular aneurysm repair: a safe alternative for stable aneurysms. *J Vasc Surg* 2009;49:845-9; discussion: 849-50.
97. Tomlinson J, McNamara J, Matloubieh J, Hart J, Singh MJ, Davies MG, et al. Intermediate follow-up after endovascular aneurysm repair: can we forgo CT scanning in certain patients? *Ann Vasc Surg* 2007;21:663-70.
98. Corriere MA, Feurer ID, Becker SY, Dattilo JB, Passman MA, Guzman RJ, et al. Endoleak following endovascular abdominal aortic aneurysm repair: implications for duration of screening. *Ann Surg* 2004;239:800-5; discussion: 805-7.
99. Gelfand DV, White GH, Wilson SE. Clinical significance of type II endoleak after endovascular repair of abdominal aortic aneurysm. *Ann Vasc Surg* 2006;20:69-74.
100. Greenberg RK, Chuter TA, Cambria RP, Sternbergh WC 3rd, Fearnot NE. Zenith abdominal aortic aneurysm endovascular graft. *J Vasc Surg* 2008;48:1-9.
101. Ashton HA, Buxton MJ, Day NE, Kim LG, Marteau TM, Scott RA, et al. The Multicentre Aneurysm Screening Study (MASS) into the effect of abdominal aortic aneurysm screening on mortality in men: a randomised controlled trial. *Lancet* 2002;360:1531-9.
102. Thompson SG, Brown LC, Sweeting MJ, Bown MJ, Kim LG, Glover MJ, et al. Systematic review and meta-analysis of the growth and rupture rates of small abdominal aortic aneurysms: implications for surveillance intervals and their cost-effectiveness. *Health Technol Assess* 2013;17:1-118.
103. Abbas A, Hansrani V, Sedgwick N, Ghosh J, McCollum CN. 3D contrast-enhanced ultrasound for detecting endoleak following endovascular aneurysm repair (EVAR). *Eur J Vasc Endovasc Surg* 2014;47:487-92.
104. Hallett JW Jr, Marshall DM, Petterson TM, Gray DT, Bower TC, Cherry KJ Jr, et al. Graft-related complications after abdominal aortic aneurysm repair: reassurance from a 36-year population-based experience. *J Vasc Surg* 1997;25:277-84; discussion: 285-6.
105. Conrad MF, Crawford RS, Pedraza JD, Brewster DC, Lamuraglia GM, Corey M, et al. Long-term durability of open abdominal aortic aneurysm repair. *J Vasc Surg* 2007;46:669-75.
106. Biancari F, Ylonen K, Anttila V, Juvonen J, Romsa P, Satta J, et al. Durability of open repair of infrarenal abdominal aortic aneurysm: a 15-year follow-up study. *J Vasc Surg* 2002;35:87-93.
107. Hertzner NR, Mascha EJ, Karafa MT, O'Hara PJ, Krajewski LP, Beven EG. Open infrarenal abdominal aortic aneurysm repair: the Cleveland Clinic experience from 1989 to 1998. *J Vasc Surg* 2002;35:1145-54.
108. Matsumura JS, Pearce WH, Cabellon A, McCarty WJ 3rd, Yao JS. Reoperative aortic surgery. *Cardiovasc Surg* 1999;7:614-21.
109. Takagi H, Sugimoto M, Kato T, Matsuno Y, Umemoto T. Postoperative incision hernia in patients with abdominal aortic aneurysm and aortoiliac occlusive disease: a systematic review. *Eur J Vasc Endovasc Surg* 2007;33:177-81.
110. Szilagyi DE, Smith RF, Elliott JP, Hageman JH, Dall'Olmo CA. Anastomotic aneurysms after vascular reconstruction: problems of incidence, etiology, and treatment. *Surgery* 1975;78:800-16.
111. Plate G, Hollier LA, O'Brien P, Pairolero PC, Cherry KJ, Kazmier FJ. Recurrent aneurysms and late vascular complications following repair of abdominal aortic aneurysms. *Arch Surg* 1985;120:590-4.
112. Crawford ES, Saleh SA, Babb JW 3rd, Glaeser DH, Vaccaro PS, Silvers A. Infrarenal abdominal aortic aneurysm: factors influencing survival after operation performed over a 25-year period. *Ann Surg* 1981;193:699-709.
113. DeBaakey MD, Crawford ES, Cooley DA, Morris GC Jr, Royster TS, Abbott WP. Aneurysm of abdominal aorta analysis of results of graft replacement therapy one to eleven years after operation. *Ann Surg* 1964;160:622-39.
114. Sterpetti AV, Feldhaus RJ, Schultz RD, Blair EA. Identification of abdominal aortic aneurysm patients with different clinical features and clinical outcomes. *Am J Surg* 1988;156:466-9.
115. Lederle FA, Freischlag JA, Kyriakides TC, Padberg FT Jr, Matsumura JS, Kohler TR, et al. Outcomes following endovascular vs open repair of abdominal aortic aneurysm: a randomized trial. *JAMA* 2009;302:1535-42.
116. van Petersen AS, Kolkman JJ, Beuk RJ, Huisman AB, Doelman CJ, Geelkerken RH. Open or percutaneous revascularization for chronic splanchnic syndrome. *J Vasc Surg* 2010;51:1309-16.
117. Atkins MD, Kwolek CJ, LaMuraglia GM, Brewster DC, Chung TK, Cambria RP. Surgical revascularization versus endovascular therapy for chronic mesenteric ischemia: a comparative experience. *J Vasc Surg* 2007;45:1162-71.
118. McMillan WD, McCarthy WJ, Bresticker MR, Pearce WH, Schneider JR, Golan JF, et al. Mesenteric artery bypass: objective patency determination. *J Vasc Surg* 1995;21:729-40; discussion: 740-1.
119. Liem TK, Segall JA, Wei W, Landry GJ, Taylor LM, Moneta GL. Duplex scan characteristics of bypass grafts to mesenteric arteries. *J Vasc Surg* 2007;45:922-7; discussion: 927-8.
120. AbuRahma AF, Mousa AY, Stone PA, Hass SM, Dean LS, Keiffer T. Duplex velocity criteria for native celiac/superior mesenteric artery stenosis vs in-stent stenosis. *J Vasc Surg* 2012;55:730-8.
121. Schoch DM, LeSar CJ, Joels CS, Erdoes LS, Sprouse LR, Fugate MW, et al. Management of chronic mesenteric vascular insufficiency: an endovascular approach. *J Am Coll Surg* 2011;212:668-75; discussion: 675-7.
122. Baker AC, Chew V, Li CS, Lin TC, Dawson DL, Pevac WC, et al. Application of duplex ultrasound imaging in determining in-stent stenosis during surveillance after mesenteric artery revascularization. *J Vasc Surg* 2012;56:1364-71; discussion: 1371.
123. Soult MC, Wuamett JC, Ahanchi SS, Stout CL, Larion S, Panneton JM. Duplex ultrasound velocity criteria for in-stent restenosis of mesenteric arteries. *J Vasc Surg* 2016;64:1366-72.
124. Ahanchi SS, Stout CL, Dahl TJ, Carty RL, Messerschmidt CA, Panneton JM. Comparative analysis of celiac versus mesenteric artery outcomes after angioplasty and stenting. *J Vasc Surg* 2013;57:1062-6.
125. Tallarita T, Oderich GS, Macedo TA, Glociczki P, Misra S, Duncan AA, et al. Reinterventions for stent restenosis in

- patients treated for atherosclerotic mesenteric artery disease. *J Vasc Surg* 2011;54:1422-9.
126. Armstrong PA. Visceral duplex scanning: evaluation before and after artery intervention for chronic mesenteric ischemia. *Perspect Vasc Surg Endovasc Ther* 2007;19:386-92; discussion: 393-4.
  127. Mousa AY, Campbell JE, Stone PA, Broce M, Bates MC, AuRahma AF. Short- and long-term outcomes of percutaneous transluminal angioplasty/stenting of renal fibromuscular dysplasia over a ten-year period. *J Vasc Surg* 2012;55:421-7.
  128. Liang P, Hurks R, Bensley RP, Hamdan A, Wyers M, Chaikof E, et al. The rise and fall of renal artery angioplasty and stenting in the United States, 1988-2009. *J Vasc Surg* 2013;58:1331-8.
  129. ASTRAL Investigators, Wheatley K, Ives N, Gray R, Kalra PA, Moss JG, et al. Revascularization versus medical therapy for renal-artery stenosis. *N Engl J Med* 2009;361:1953-62.
  130. Cooper CJ, Murphy TP, Cutlip DE, Jamerson K, Henrich W, Reid DM, et al. Stenting and medical therapy for atherosclerotic renal-artery stenosis. *N Engl J Med* 2014;370:12-22.
  131. Stone PA, Campbell JE, AbuRahma AF, Hamdan M, Broce M, Nanjundappa A, et al. Ten-year experience with renal artery in-stent stenosis. *J Vasc Surg* 2011;53:1026-31.
  132. Davies MG, Saad WA, Bismuth JX, Peden EK, Naoum JJ, Lumsden AB. Outcomes of endoluminal reintervention for restenosis after percutaneous renal angioplasty and stenting. *J Vasc Surg* 2009;49:946-52.
  133. Simone TA, Brooke BS, Goodney PP, Walsh DB, Stone DH, Powell RJ, et al. Clinical effectiveness of secondary interventions for restenosis after renal artery stenting. *J Vasc Surg* 2013;58:687-94.
  134. Bax L, Mali WP, Van de Ven PJ, Beek FJ, Vos JA, Beutler JJ. Repeated intervention for in-stent restenosis of the renal arteries. *J Vasc Interv Radiol* 2002;13:1219-24.
  135. Otah KE, Alhaddad IA. Intravascular ultrasound-guided cutting balloon angioplasty for renal artery stent restenosis. *Clin Cardiol* 2004;27:581-3.
  136. Zeller T, Rastan A, Schwarzwald U, Mueller C, Schwarz T, Frank U, et al. Treatment of in-stent restenosis following stent-supported renal artery angioplasty. *Catheter Cardiovasc Interv* 2007;70:454-9.
  137. Zeller T, Sixt S, Rastan A, Schwarzwald U, Muller C, Frank U, et al. Treatment of reoccurring in-stent restenosis following reintervention after stent-supported renal artery angioplasty. *Catheter Cardiovasc Interv* 2007;70:296-300.
  138. Kiernan TJ, Yan BP, Eisenberg JD, Ruggiero NJ, Gupta V, Drachman D, et al. Treatment of renal artery in-stent restenosis with sirolimus-eluting stents. *Vasc Med* 2010;15:3-7.
  139. Puchner S, Stadler A, Minar E, Lammer J, Bucek RA. Multi-detector CT angiography in the follow-up of patients treated with renal artery stents: value of different reformation techniques compared with axial source images. *J Endovasc Ther* 2007;14:387-94.
  140. Raza SA, Chughtai AR, Wahba M, Cowling MG, Taube D, Wright AR. Multislice CT angiography in renal artery stent evaluation: prospective comparison with intra-arterial digital subtraction angiography. *Cardiovasc Intervent Radiol* 2004;27:9-15.
  141. Chi YW, White CJ, Thornton S, Milani RV. Ultrasound velocity criteria for renal in-stent restenosis. *J Vasc Surg* 2009;50:119-23.
  142. Olin JW, Allie DE, Belkin M, Bonow RO, Casey DE, Creager MA, et al. ACCF/AHA/ACR/SCAI/SIR/SVM/SVN/SVS 2010 performance measures for adults with peripheral arterial disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Performance Measures, the American College of Radiology, the Society for Cardiac Angiography and Interventions, the Society for Interventional Radiology, the Society for Vascular Medicine, the Society for Vascular Nursing, and the Society for Vascular Surgery (Writing Committee to Develop Clinical Performance Measures for Peripheral Artery Disease). *J Am Coll Cardiol* 2010;56:2147-81.
  143. Rooke TW, Hirsch AT, Misra S, Sidawy AN, Beckman JA, Findeiss L, et al. Management of patients with peripheral artery disease (compilation of 2005 and 2011 ACCF/AHA Guideline Recommendations): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* 2013;61:1555-70.
  144. Hodgkiss-Harlow K, Bandyk DF. Ultrasound assessment during and after carotid and peripheral intervention. In: Pellerito JS, Polak JF, editors. *Introduction to vascular ultrasonography*. 6th ed. Philadelphia: Elsevier Saunders; 2012. p. 307-23.
  145. Miller JH, Foreman RK, Ferguson L, Faris I. Interposition vein cuff for anastomosis of prosthesis to small artery. *Aust N Z J Surg* 1984;54:283-5.
  146. Griffiths GD, Nagy J, Black D, Stonebridge PA. Randomized clinical trial of distal anastomotic interposition vein cuff in infrainguinal polytetrafluoroethylene bypass grafting. *Br J Surg* 2004;91:560-2.
  147. Laurila K, Lepantalo M, Teittinen K, Kantonen I, Forsell C, Vilkkio P, et al. Does an adjuvant AV-fistula improve the patency of a femorocrural PTFE bypass with distal vein cuff in critical leg ischaemia? A prospective randomized multicenter trial. *Eur J Vasc Endovasc Surg* 2004;27:180-5.
  148. Nguyen LL, Conte MS, Menard MT, Gravereaux EC, Chew DK, Donaldson MC, et al. Infrainguinal vein bypass graft revision: factors affecting long-term outcome. *J Vasc Surg* 2004;40:916-23.
  149. Armstrong PA, Bandyk DF, Wilson JS, Shames ML, Johnson BL, Back MR. Optimizing infrainguinal arm vein bypass patency with duplex ultrasound surveillance and endovascular therapy. *J Vasc Surg* 2004;40:724-31.
  150. Lundell A, Lindblad B, Bergqvist D, Hansen F. Femoropopliteal-crural graft patency is improved by an intensive surveillance program: a prospective randomized study. *J Vasc Surg* 1995;21:26-34.
  151. Lane TR, Metcalfe MJ, Narayanan S, Davies AH. Post-operative surveillance after open peripheral arterial surgery. *Eur J Vasc Endovasc Surg* 2011;42:59-77.
  152. Kashyap VS, Pavkov ML, Bena JF, Sarac TP, O'Hara PJ, Lyden SP, et al. The management of severe aortoiliac occlusive disease: endovascular therapy rivals open reconstruction. *J Vasc Surg* 2008;48:1451-7.
  153. Onahara T, Komori K, Kume M, Ishida M, Ohta S, Takeuchi K, et al. Multivariate analysis of long-term results after an axillo-bifemoral and aortobifemoral bypass in patients with aortoiliac occlusive disease. *J Cardiovasc Surg* 2000;41:905-10.
  154. Mellièrre D, Desgranges P, de Wailly GW, Roudot-Thoraval F, Allaire E, Becquemin JP. Extensive unilateral iliofemoral occlusions: durability of four techniques of arterial reconstructions. *Vascular* 2004;12:285-92.
  155. Carsten CG 3rd, Kalbaugh CA, Langan EM 3rd, Cass AL, Cull DL, Snyder BA, et al. Contemporary outcomes of iliofemoral bypass grafting for unilateral aortoiliac occlusive disease: a 10-year experience. *Am Surg* 2008;74:555-9.
  156. Nazzal MM, Hoballah JJ, Jacobovicz C, Mohan CR, Martinasevic M, Ryan SM, et al. A comparative evaluation of femorofemoral crossover bypass and iliofemoral bypass for

- unilateral iliac artery occlusive disease. *Angiology* 1998;49:259-65.
157. Piotrowski JJ, Pearce WH, Jones DN, Whitehill T, Bell R, Patt A, et al. Aortobifemoral bypass: the operation of choice for unilateral iliac occlusion. *J Vasc Surg* 1988;8:211-8.
158. Hinchliffe RJ, Alric P, Wenham PW, Hopkinson BR. Durability of femorofemoral bypass grafting after aortouniiliac endovascular aneurysm repair. *J Vasc Surg* 2003;38:498-503.
159. Stone PA, Armstrong PA, Bandyk DF, Keeling WB, Flaherty SK, Shames ML, et al. Duplex ultrasound criteria for femorofemoral bypass revision. *J Vasc Surg* 2006;44:496-501.
160. Harris EJ Jr, Taylor LM, McConnell DB, Moneta GL, Yeager RA, Porter JM. Clinical results of axillobifemoral bypass using externally supported polytetrafluoroethylene. *J Vasc Surg* 1990;14:416-20.
161. Sharp WJ, Hoballah JJ, Mohan CR, Kresowik TF, Martinasevic M, Chalmers RT, et al. The management of the infected aortic prosthesis: a current decade of experience. *J Vasc Surg* 1994;19:844-50.
162. Whittemore AD, Clowes AW, Couch NP, Mannick JA. Secondary femoropopliteal reconstruction. *Ann Surg* 1981;193:35-42.
163. Bandyk DF, Seabrook GR, Moldenhauer P, Lavin J, Edward J, Cato R, et al. Hemodynamics of vein graft stenosis. *J Vasc Surg* 1988;8:688-95.
164. Golledge J, Beattie DK, Greenhalgh RM, Davies AH. Have the results of infrainguinal bypass improved with the widespread utilization of postoperative surveillance? *Eur J Vasc Endovasc Surg* 1996;11:388-92.
165. Ihlberg L, Luther M, Tierala E, Lepantalo M. The utility of duplex scanning in infrainguinal vein graft surveillance: results from a randomized controlled study. *Eur J Vasc Endovasc Surg* 1998;16:19-27.
166. Ihlberg L, Luther M, Alback A, Kantonen I, Lepantalo M. Does a completely accomplished duplex-based surveillance prevent vein graft failure? *Eur J Vasc Endovasc Surg* 1999;18:395-400.
167. Mofidi R, Kelman J, Berry O, Bennett S, Murie JA, Dawson AR. Significance of the early postoperative duplex result in infrainguinal vein bypass surveillance. *Eur J Vasc Endovasc Surg* 2007;34:327-32.
168. Davies AH, Hawdon AJ, Sydes MR, Thompson SG; VGST Participants. Is duplex surveillance of value after leg vein bypass grafting? Principal results of the Vein Graft Surveillance Randomised Trial (VGST). *Circulation* 2005;112:1985-91.
169. Abu Dabrh AM, Mohammed K, Farah W, Haydour Q, Zierler RE, Wang Z, et al. Systematic review and meta-analysis of duplex ultrasound surveillance for infrainguinal vein bypass grafts. *J Vasc Surg* 2017;66:885-91.
170. Dunlop P, Sayers RD, Naylor AR, Bell PR, London NJ. The effect of a surveillance programme on the patency of synthetic infrainguinal bypass grafts. *Br J Vasc Endovasc Surg* 1996;11:441-5.
171. Calligaro KD, Doerr K, McAfee-Bennett S, Krug R, Raviola CA, Dougherty MJ. Should duplex ultrasonography be performed for the surveillance of femoropopliteal and femorotibial arterial prosthetic bypasses? *Ann Vasc Surg* 2001;15:520-4.
172. Brumberg RS, Back MR, Armstrong PA, Cuthbertson D, Shames ML, Johnson BL, et al. The relative importance of graft surveillance and warfarin therapy in infrainguinal prosthetic bypass failure. *J Vasc Surg* 2007;46:1160-6.
173. Carter A, Murphy MO, Halka AT, Turner NJ, Kirton JP, Murray D, et al. The natural history of stenoses within lower limb arterial bypass grafts using a graft surveillance program. *Ann Vasc Surg* 2007;21:695-703.
174. Kudo T, Chandra FA, Kwun WH, Haas BT, Ahn SS. Changing pattern of surgical revascularization for critical limb ischemia over 12 years: endovascular vs open bypass surgery. *J Vasc Surg* 2006;44:304-13.
175. Egorova NN, Guillerme S, Gelijns A, Morrissey N, Dayal R, McKinsey JF, et al. An analysis of the outcomes of a decade of experience with lower extremity revascularization including limb salvage, lengths of stay, and safety. *J Vasc Surg* 2010;51:878-85.
176. Rowe VL, Lee W, Weaver FA, Etzioni D. Patterns of treatment for peripheral arterial disease in the United States: 1996-2005. *J Vasc Surg* 2009;49:910-7.
177. Sachs T, Pomposelli F, Hamdan A, Wyers M, Schermerhorn M. Trends in the national outcomes and costs for claudication and limb threatening ischemia: angioplasty vs bypass graft. *J Vasc Surg* 2011;54:1021-31.
178. O'Brien-Irr MS, Harris LM, Dosluoglu HH, Dryjski ML. Procedural trends in the treatment of peripheral arterial disease by insurer status in New York State. *J Am Coll Surg* 2012;215:311-21.
179. Collaborative overview of randomised trials of antiplatelet therapy—II: maintenance of vascular graft or arterial patency by antiplatelet therapy. Antiplatelet Trialists' Collaboration. *BMJ* 1994;308:159-68.
180. O'Neil-Callahan K, Katsimaglis G, Tepper MR, Ryan J, Mosby C, Ioannidis JP, et al. Statins decrease perioperative cardiac complications in patients undergoing noncardiac vascular surgery: the Statins for Risk Reduction in Surgery (StaRRS) study. *J Am Coll Cardiol* 2005;45:336-42.
181. Cacoub PP, Abola MT, Baumgartner I, Bhatt DL, Creager MA, Liao CS, et al. Cardiovascular risk factor control and outcomes in peripheral artery disease patients in the Reduction of Atherothrombosis for Continued Health (REACH) Registry. *Atherosclerosis* 2009;204:e86-92.
182. Cacoub PP, Zeymer U, Limbourg T, Baumgartner I, Poldermans D, Rother J. Effects of adherence to guidelines for the control of major cardiovascular risk factors on outcomes in the REduction of Atherothrombosis for Continued Health (REACH) Registry Europe. *Heart* 2011;97:660-7.
183. Chung J, Timaran DA, Modrall JG, Ahn C, Timaran CH, Kirkwood ML, et al. Optimal medical therapy predicts amputation-free survival in chronic critical limb ischemia. *J Vasc Surg* 2013;58:972-80.
184. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG, et al. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). *J Vasc Surg* 2007;45:S5-67.
185. Mehran R, Aymong ED, Nikolsky E, Lasic Z, Iakovou I, Fahy M, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. *J Am Coll Cardiol* 2004;44:1393-9.
186. James MT, Ghali WA, Tonelli M, Faris P, Knudtson ML, Pannu N, et al. Acute kidney injury following coronary angiography is associated with a long-term decline in kidney function. *Kidney Int* 2010;78:803-9.
187. Welt FC, Rogers C. Inflammation and restenosis in the stent era. *Arterioscler Thromb Vasc Biol* 2002;22:1769-76.
188. Nakatani M, Takeyama Y, Shibata M, Yorozyua M, Suzuki H, Koba S, et al. Mechanisms of restenosis after coronary intervention: difference between plain old balloon angioplasty and stenting. *Cardiovasc Pathol* 2003;12:40-8.
189. Pasterkamp G, de Kleijn DP, Borst C. Arterial remodeling in atherosclerosis, restenosis and after alteration of blood flow:

- potential mechanisms and clinical implications. *Cardiovasc Res* 2000;45:843-52.
190. Post MJ, de Smet BJ, van der Helm Y, Borst C, Kuntz RE. Arterial remodeling after balloon angioplasty or stenting in an atherosclerotic experimental model. *Circulation* 1997;96:996-1003.
  191. Herity NA, Ward MR, Lo S, Yeung AC. Review: clinical aspects of vascular remodeling. *J Cardiovasc Electrophysiol* 1999;10:1016-24.
  192. Mintz GS, Popma JJ, Pichard AD, Kent KM, Satler LF, Wong C, et al. Arterial remodeling after coronary angioplasty a serial intravascular ultrasound study. *Circulation* 1996;94:35-43.
  193. Post MJ, Borst C, Kuntz RE. The relative importance of arterial remodeling compared with intimal hyperplasia in lumen renarrowing after balloon angioplasty. A study in the normal rabbit and the hypercholesterolemic Yucatan micropig. *Circulation* 1994;89:2816-21.
  194. Scheinert D, Scheinert S, Sax J, Piorkowski C, Braunlich S, Ulrich M, et al. Prevalence and clinical impact of stent fractures after femoropopliteal stenting. *J Am Coll Cardiol* 2005;45:312-5.
  195. Rits J, Van Herwaarden JA, Jahrome AK, Krievins D, Moll FL. The incidence of arterial stent fractures with exclusion of coronary, aortic, and non-arterial settings. *Eur J Vasc Endovasc Surg* 2008;36:339-45.
  196. Davaine JM, Quérat J, Guyomarch B, Brennan MÁ, Costargent A, Chaillou P, et al. Incidence and the clinical impact of stent fractures after primary stenting for TASC C and D femoropopliteal lesions at 1 year. *Eur J Vasc Endovasc Surg* 2013;46:201-12.
  197. Iida O, Nanto S, Uematsu M, Ikeoka K, Okamoto S, Nagata S. Influence of stent fracture on the long-term patency in the femoro-popliteal artery: experience of 4 years. *JACC Cardiovasc Interv* 2009;2:665-71.
  198. Tielbeek AV, Rietjens E, Buth J, Vroegindewij D, Schol FP. The value of duplex surveillance after endovascular intervention for femoropopliteal obstructive disease. *Eur J Vasc Endovasc Surg* 1996;12:145-50.
  199. Mewissen MW, Kinney EV, Bandyk DF, Reifsnnyder T, Seabrook GR, Lipchik EO, et al. The role of duplex scanning versus angiography in predicting outcome after balloon angioplasty in the femoropopliteal artery. *J Vasc Surg* 1992;15:860-5.
  200. Humphries MD, Pevac WC, Laird JR, Yeo KK, Hedayati N, Dawson DL. Early duplex scanning after infrainguinal endovascular therapy. *J Vasc Surg* 2011;53:353-8.
  201. Robinson WP 3rd, Nguyen LL, Bafford R, Belkin M. Results of second-time angioplasty and stenting for femoropopliteal occlusive disease and factors affecting outcomes. *J Vasc Surg* 2011;53:651-7.
  202. Timaran CH, Prault TL, Stevens SL, Freeman MB, Goldman MH. Iliac artery stenting versus surgical reconstruction for TASC (TransAtlantic Inter-Society Consensus) type B and type C iliac lesions. *J Vasc Surg* 2003;38:272-8.
  203. De Roeck A, Hendriks J, Delrue F, Lauwers P, Van Schil P, De Maeseneer M, et al. Long-term results of primary stenting for long and complex iliac artery occlusions. *Acta Chir Belg* 2006;106:187-92.
  204. Vorwerk D, Guenther RW, Schürmann K, Wendt G, Peters I. Primary stent placement for chronic iliac artery occlusions: follow-up results in 103 patients. *Radiology* 1995;194:745-9.
  205. Soga Y, Iida O, Kawasaki D, Yamauchi Y, Suzuki K, Hirano K, et al. Contemporary outcomes after endovascular treatment for aorto-iliac artery disease. *Circ J* 2012;76:2697-704.
  206. Spijkerboer AM, Nass PC, De Valois JC, Eikelboom BC, Overtom TT, Beek FJ, et al. Iliac artery stenoses after percutaneous transluminal angioplasty: follow-up with duplex ultrasonography. *J Vasc Surg* 1996;23:691-7.
  207. Myers KA, Wood SR, Lee V. Vascular ultrasound surveillance after endovascular intervention for occlusive iliac artery disease. *Cardiovasc Surg* 2001;9:448-54.
  208. Baril DT, Rhee RY, Kim J, Makaroun MS, Chaer RA, Marone LK. Duplex criteria for determination of in-stent stenosis after angioplasty and stenting of the superficial femoral artery. *J Vasc Surg* 2009;49:133-8.
  209. Geraghty PJ, Mewissen MW, Jaff MR, Ansel GM; VIBRANT Investigators. Three-year results of the VIBRANT trial of VIABAHN endoprosthesis versus bare nitinol stent implantation for complex superficial femoral artery occlusive disease. *J Vasc Surg* 2013;58:386-95.
  210. Armstrong EJ, Singh S, Singh GD, Yeo KK, Ludder S, Westin G, et al. Angiographic characteristics of femoropopliteal in-stent restenosis: association with long-term outcomes after endovascular intervention. *Catheter Cardiovasc Interv* 2013;82:1168-74.
  211. Vartanian SM, Johnston PC, Walker JP, Runge SJ, Eichler CM, Reilly LM, et al. Clinical consequence of bare metal stent and stent graft failure in femoropopliteal occlusive disease. *J Vasc Surg* 2013;58:1525-31.
  212. Yeo KK, Malik U, Laird JR. Outcomes following treatment of femoropopliteal in-stent restenosis: a single center experience. *Catheter Cardiovasc Interv* 2011;78:604-8.
  213. Jones DW, Graham A, Connolly PH, Schneider DB, Meltzer AJ. Restenosis and symptom recurrence after endovascular therapy for claudication: does duplex ultrasound correlate with recurrent claudication? *Vascular* 2015;23:47-54.
  214. Smolock CJ, Anaya-Ayala JE, Kaufman Y, Bavare CS, Patel MS, El-Sayed HF, et al. Current efficacy of open and endovascular interventions for advanced superficial femoral artery occlusive disease. *J Vasc Surg* 2013;58:1267-75.
  215. Graziani L, Piaggese A. Indications and clinical outcomes for below knee endovascular therapy: review article. *Catheter Cardiovasc Interv* 2010;75:433-43.
  216. Ferraresi R, Centola M, Ferlini M, Da Ros R, Caravaggi C, Assaloni R, et al. Long-term outcomes after angioplasty of isolated, below-the-knee arteries in diabetic patients with critical limb ischaemia. *Eur J Vasc Endovasc Surg* 2009;37:336-42.
  217. Saqib NU, Deomenick N, Cho JS, Marone L, Leers S, Makaroun MS, et al. Predictors and outcomes of restenosis following tibial artery endovascular interventions for critical limb ischemia. *J Vasc Surg* 2013;57:692-9.
  218. Fernandez N, McEnaney R, Marone LK, Rhee RY, Leers S, Makaroun M, et al. Predictors of failure and success of tibial interventions for critical limb ischemia. *J Vasc Surg* 2010;52:834-42.
  219. Jimenez JG, Huber TS, Ozaki CK, Flynn TC, Berceli SA, Lee WA, et al. Durability of antegrade synthetic aortomesenteric bypass for chronic mesenteric ischemia. *J Vasc Surg* 2002;35:1078-84.
  220. Kruger AJ, Walker PJ, Foster WJ, Jenkins JS, Boyne NS, Jenkins J. Open surgery for atherosclerotic chronic mesenteric ischemia. *J Vasc Surg* 2007;46:941-5.
  221. Zerbib P, Khoury-Helou A, Lebuffe G, Massouille D, Nunes B, Chambon JP. Surgical revascularization for chronic intestinal ischemia. *Minerva Chir* 2008;63:191-8.
  222. Davies RS, Wall ML, Wilverman SH, Simms MH, Vohra RK, Bradbury AW, et al. Surgical versus endovascular reconstruction for chronic mesenteric ischemia: a contemporary UK series. *Vasc Endovascular Surg* 2009;43:157-64.

223. Landis MS, Rajan DK, Simons ME, Hayeems EB, Kachura JR, Sniderman KW. Percutaneous management of chronic mesenteric ischemia: outcomes after intervention. *J Vasc Interv Radiol* 2005;16:1319-25.
224. Sarac TP, Altinel O, Kashyap V, Bena J, Lyden S, Srivastava S, et al. Endovascular treatment of stenotic and occluded visceral arteries for chronic mesenteric ischemia. *J Vasc Surg* 2008;47:485-91.
225. Fioule B, van de Rest HJ, Meijer JR, van Leersum M, van Koevorden S, Moll FL, et al. Percutaneous transluminal angioplasty and stenting as first-choice treatment in patients with chronic mesenteric ischemia. *J Vasc Surg* 2010;51:386-91.
226. Peck MA, Conrad MF, Kwolek CJ, LaMuraglia GM, Paruchuri V, Cambria RP. Intermediate-term outcomes of endovascular treatment for symptomatic chronic mesenteric ischemia. *J Vasc Surg* 2010;51:140-7.e1-2.
227. Sharafuddin MJ, Nicholson RM, Kresowik TF, Amin PB, Hoballah JJ, Sharp WJ. Endovascular recanalization of total occlusions of the mesenteric and celiac arteries. *J Vasc Surg* 2012;55:1674-81.
228. Lewis BE, Leya FS, Johnson SA, Grassman ED, McKierman TL, Mason JR, et al. Improved hemodynamic, angiographic and functional results after renal artery stenting. *J Invasive Cardiol* 1994;6:136-40.
229. Dorros G, Jaff M, Jain A, Dufek C, Mathiak L. Follow-up of primary Palmaz-Schatz stent placement for atherosclerotic renal artery stenosis. *Am J Cardiol* 1995;75:1051-5.
230. Harjai K, Khosla S, Shaw D, Collins T, Jenkins S, White C, et al. Effect of gender on outcomes following renal artery stent placement for renovascular hypertension. *Cathet Cardiovasc Diagn* 1997;42:381-6.
231. White CJ, Ramee SR, Collins TJ, Jenkins JS, Escobar A, Shaw D. Renal artery stent placement: utility in lesions difficult to treat with balloon angioplasty. *J Am Coll Cardiol* 1997;30:1445-50.
232. Rundback JH, Gray RJ, Rozenblit G, Poplasky MR, Babu S, Shaw P, et al. Renal artery stent placement for the management of ischemic nephropathy. *J Vasc Interv Radiol* 1998;9:413-20.
233. Rocha-Singh KJ, Mishkel GJ, Katholi RE, Ligon RA, Armbruster JA, McShane JH, et al. Clinical predictors of improved long-term blood pressure control after successful stenting of hypertensive patients with obstructive renal artery atherosclerosis. *Catheter Cardiovasc Interv* 1999;47:167-72.
234. Rodriguez-Lopez JA, Werner A, Ray LI, Verikokos C, Torruella LJ, Martinez E. Renal artery stenosis treated with stent deployment: indications, technique, and outcome for 108 patients. *J Vasc Surg* 1999;29:617-24.
235. Henry M, Amor M, Henry I, Ethevenot G, Tzvetanov K, Courvoisier A, et al. Stents in the treatment of renal artery stenosis: long-term follow-up. *J Endovasc Surg* 1999;6:42-51.
236. Van de Ven PJ, Kaatee R, Beutler JJ, Beek FJ, Woittiez AJ, Buskens E, et al. Arterial stenting and balloon angioplasty in ostial atherosclerotic renovascular disease: a randomised trial. *Lancet* 1999;353:282-6.
237. Yutan E, Glickerman DJ, Caps MT, Hatsukami T, Harley JD, Kohler TR, et al. Percutaneous transluminal revascularization for renal artery stenosis: Veterans Affairs Puget Sound Health Care System experience. *J Vasc Surg* 2001;34:685-93.
238. Ahmadi R, Schillinger M, Sabeti S, Loewe C, Miekusch W, Haumer M, et al. Renal artery PTA and stent implantation: immediate and late clinical and morphological outcome. *Wien Klin Wochenschr* 2002;114:21-7.
239. Sivamurthy N, Surowiec SM, Culakova E, Rhodes JM, Lee D, Sternbach Y, et al. Divergent outcomes after percutaneous therapy for symptomatic renal artery stenosis. *J Vasc Surg* 2004;39:565-74.
240. Nolan BW, Schermerhorn ML, Rowell E, Powell RJ, Fillingim MF, Rzucidlo EM, et al. Outcomes of renal artery angioplasty and stenting using low-profile systems. *J Vasc Surg* 2005;41:46-52.
241. Muller-Hülsbeck S, Frahm C, Behm C, Schafer PJ, Bolte H, Heller M, et al. Low-profile stent placement with the monorail technique for treatment of renal artery stenosis: midterm results of a prospective trial. *J Vasc Interv Radiol* 2005;16:963-71.
242. Sapoval M, Zahringer M, Pattynama P, Rabbia C, Vignali C, Maleux G, et al. Low-profile stent system for treatment of atherosclerotic renal artery stenosis: the GREAT trial. *J Vasc Interv Radiol* 2005;16:1195-202.
243. Sahin S, Cimşit C, Andaç N, Baltacıoğlu F, Tuğlular S, Akoğlu E. Renal artery stenting in solitary functioning kidneys: technical and clinical results. *Eur J Radiol* 2006;57:131-7.
244. Rastan A, Krankenberg H, Muller-Hulsbeck S, Sixt S, Tubler T, Muller C, et al. Improved renal function and blood pressure control following renal artery angioplasty: the renal artery angioplasty in patients with renal insufficiency and hypertension using a dedicated renal stent device study (PRECISION). *EuroIntervention* 2008;4:208-13.
245. Rocha-Singh K, Jaff MR, Lynne Kelley E; RENAISSANCE Trial Investigators. Renal artery stenting with noninvasive duplex ultrasound follow-up: 3-year results from the RENAISSANCE renal stent trial. *Catheter Cardiovasc Interv* 2008;72:853-62.
246. Klonaris C, Katsargyris A, Alexandrou A, Tsigris C, Giannopoulos A, Bastounis E. Efficacy of protected renal artery primary stenting in the solitary functioning kidney. *J Vasc Surg* 2008;48:1414-22.
247. Misra S, Thatipelli MR, Howe PW, Hunt C, Mathew V, Barsness GW, et al. Preliminary study of the use of drug-eluting stents in atherosclerotic renal artery stenoses 4 mm in diameter or smaller. *J Vasc Interv Radiol* 2008;19:833-9.
248. Corriere MA, Edwards MS, Pearce JD, Andrews JS, Geary RL, Hansen KJ. Restenosis after renal artery angioplasty and stenting: incidence and risk factors. *J Vasc Surg* 2009;50:813-9.
249. Thalhammer C, Ferriani V, Husmann M, Rufibach K, Meier T, Amann-Vesti BR. Predictive value of duplex ultrasound for restenosis after renal artery stenting. *Clin Hemorheol Microcirc* 2010;45:217-24.
250. Laird JR, Rundback J, Zierler RE, Becker GJ, O'Shaughnessy C, Shuck JW, et al. Safety and efficacy of renal artery stenting following suboptimal renal angioplasty for de novo and restenotic ostial lesions: results from a nonrandomized, prospective multicenter registry. *J Vasc Interv Radiol* 2010;21:627-37.
251. Jaff MR, Bates M, Sullivan T, Popma J, Gao X, Zaugg M, et al. Significant reduction in systolic blood pressure following renal artery stenting in patients with uncontrolled hypertension: results from the HERCULES trial. *Catheter Cardiovasc Interv* 2012;80:343-50.
252. Birrer M, Do DD, Mahler F, Triller J, Baumgartner I. Treatment of renal artery fibromuscular dysplasia with balloon angioplasty: a prospective follow-up study. *Eur J Vasc Endovasc Surg* 2002;23:146-52.
253. Kim HJ, Do YS, Shin SW, Park KB, Cho SK, Choe YH, et al. Percutaneous transluminal angioplasty of renal artery fibromuscular dysplasia: mid-term results. *Korean J Radiol* 2008;9:38-44.
254. Ham SW, Kumar SR, Wang BR, Rowe VL, Weaver FA. Late outcomes of endovascular and open revascularization for

- nonatherosclerotic renal artery disease. *Arch Surg* 2010;145:832-9.
255. Bakker J, Beutler JJ, Elgersma OE, de Lange EE, de Kort GA, Beek FJ. Duplex ultrasonography in assessing restenosis of renal artery stents. *Cardiovasc Intervent Radiol* 1999;22:475-80.
256. Mohabbat W, Greenberg RK, Mastracci TM, Cury M, Morales JP, Hernandez AV. Revised duplex criteria and outcomes for renal stents and stent grafts following endovascular repair of juxtarenal and thoracoabdominal aneurysms. *J Vasc Surg* 2009;49:827-37; discussion: 837.
257. Fleming SH, Davis RP, Craven TE, Deonanan JK, Godshall CJ, Hansen KJ. Accuracy of duplex sonography scans after renal artery stenting. *J Vasc Surg* 2010;52:953-7; discussion: 958.
258. Del Conde I, Galin ID, Trost B, Kang J, Lookstein R, Woodward M, et al. Renal artery duplex ultrasound criteria for the detection of significant in-stent restenosis. *Catheter Cardiovasc Interv* 2013;83:612-8.
259. Buth J, Disselhoff B, Sommeling C, Stam L. Color-flow duplex criteria for grading stenosis in infrainguinal vein grafts. *J Vasc Surg* 1991;14:716-26; discussion: 726-8.
260. Dalsing MC, Cikrit DF, Lalka SG, Sawchuk AP, Schulz C. Femorodistal vein grafts: the utility of graft surveillance criteria. *J Vasc Surg* 1995;21:127-34.
261. Ferris BL, Mills JL Sr, Hughes JD, Durrani T, Knox R. Is early postoperative duplex scan surveillance of leg bypass grafts clinically important? *J Vasc Surg* 2003;37:495-500.
262. Colledge J, Wright I, Lane IF. Comparison of clinical follow-up and duplex surveillance of infrainguinal vein bypasses. *Cardiovasc Surg* 1996;4:766-70.
263. Idu MM, Blankenstein JD, de Gier P, Truyen E, Buth J. Impact of a color-flow duplex surveillance program on infrainguinal vein graft patency: a five-year experience. *J Vasc Surg* 1993;17:42-52; discussion: 52-3.
264. Laborde AL, Synn AY, Worsey MJ, Bower TR, Hoballah JJ, Sharp WJ, et al. A prospective comparison of ankle/brachial indices and color duplex imaging in surveillance of the in situ saphenous vein bypass. *J Cardiovasc Surg (Torino)* 1992;33:420-5.
265. Lewis DR, McGrath C, Irvine CD, Jones A, Murphy P, Smith FC, et al. The progression and correction of duplex detected velocity shifts in angiographically normal vein grafts. *Eur J Vasc Endovasc Surg* 1998;15:394-7.
266. Moody P, Gould DA, Harris PL. Vein graft-surveillance improves patency in femoro-popliteal bypass. *Eur J Vasc Surg* 1990;4:117-21.
267. Polak JF, Donaldson MC, Dobkin GR, Mannick JA, O'Leary DH. Early detection of saphenous vein arterial bypass graft stenosis by color-assisted duplex sonography: a prospective study. *AJR Am J Roentgenol* 1990;154:857-61.
268. Stierli P, Aeberhard P, Livers M. The role of colour flow duplex screening in infra-inguinal vein grafts. *Eur J Vasc Surg* 1992;6:293-8.
269. Visser K, Idu MM, Buth J, Engel GL, Hunink MC. Duplex scan surveillance during the first year after infrainguinal autologous vein bypass grafting surgery: costs and clinical outcomes compared with other surveillance programs. *J Vasc Surg* 2001;33:123-30.

Submitted Apr 3, 2018; accepted Apr 11, 2018.