ESC European Society of Cardiology

European Heart Journal - Cardiovascular Imaging (2018) **19**, 1195–1221
doi:10.1093/ehjci/jey103

EACVI POSITION PAPER

Focus on echovascular imaging assessment of arterial disease: complement to the ESC guidelines (PARTIM 1) in collaboration with the Working Group on Aorta and Peripheral Vascular Diseases

Muriel Sprynger[1]*, Fausto Rigo[2], Marie Moonen[1], Victor Aboyans[3], Thor Edvardsen[4], Monica L. de Alcantara[5], Marianne Brodmann[6], Katerina K. Naka[7], Serge Kownator[8], Iana Simova[9], Charalambos Vlachopoulos[10], Jean-Claude Wautrecht[11], and Patrizio Lancellotti[1]

This document was reviewed by members of the 2014–2016 and 2016–2018 EACVI Scientific Documents Committee: Victoria Delgado, Raluca Dulgheru, Kristina H. Haugaa, Frank Flachskampf, Alessia Gimelli, Bernhard Gerber, Nuno Cardim, Bernard Cosyns, Denisa Muraru, Pier Giorgio Masci, and Maurizio Galderisi

[1]Department of Cardiology, University of Liege Hospital, GIGA Cardiovascular Sciences, B35, Avenue de l'Hôpital, 1, 4000 Liege, Belgium; [2]Division of Cardiology, dell'Angelo Hospital Mestre-Venice, 30174 Venezia, Italy; [3]Department of Cardiology, Dupuytren University Hospital, avenue Martin Luther King, 2, 87042 Limoges, France; [4]Department of Cardiology, Oslo University Hospital, Sognsvannsveien, 20, NO-0027 OSLO, Norway; [5]Department of Cardiology, Americas Medical City Hospital, avenue Jorge Curi, 550, 22775-001 Rio de Janeiro, Brasil; [6]Division of Angiology, Department of Internal Medicine, Medical University Graz, Auenbruggerplatz, 27, A-8036 Graz, Austria; [7]2nd Cardiology Department, University of Ioannina Medical School, University Campus, GR45110 Loannina, Greece; [8]Centre Cardiologique et Vasculaire, rue de Longwy, 12, 57100 Thionville, France; [9]Department of Cardiology, Acibadem City Clinic Cardiovascular Center, University Hospital, Okolovrasten pat Str, 127, 1407 Sofia, Bulgaria; [10]First Department of Cardiology, Athens Medical School, Athens, Greece; and [11]Department of Vascular Diseases, Hôpital Erasme, route de Lennik, 808, 1070 Brussels, Belgium

Received 25 June 2018; editorial decision 27 June 2018; accepted 28 June 2018; online publish-ahead-of-print 18 September 2018

The main goal of the present document is to provide a set of practical recommendations for ultrasound imagers who are interested in artery diseases or for physicians who intend to undertake vascular procedures. This is the first part of the work. It is dedicated to general principles of ultrasonography, cervicoencephalic, subclavian, aortoiliac and lower extremity arteries, abdominal aorta, and popliteal aneurysms. It also discusses miscellaneous items such as medial arterial calcinosis, arterial embolism, arteritis, arterial stents and bypasses, false aneurysms, aortic dissection, popliteal entrapment syndrome, and iliac endofibrosis.

Keywords recommendations • ultrasound • Doppler • cervicoencephalic arteries • subclavian artery • abdominal arteries • lower extremity arteries • abdominal aorta aneurysm • popliteal aneurysm • medial arterial calcinosis • arteritis • stent • bypass • false aneurysm • dissection • popliteal entrapment • iliac endofibrosis

Table of contents

* Corresponding author. Tel: +32 (4) 366 7194; Fax: +32 (4) 366 7195. E-mail: msprynger@chuliege.be

Introduction

Currently non-invasive vascular imaging includes Duplex ultrasonography (DUS), computed tomography (CT), magnetic resonance imaging (MRI), and less used techniques such as transcutaneous oxygen tension (TcPO2), laser Doppler, capillaroscopy, positron emission tomography (PET), and nuclear lymphography. Angiography is no longer the gold standard, and is mostly limited to endovascular procedures (i.e. stenting) or very peculiar diagnoses. Compared with other vascular imaging techniques, DUS has many advantages: low cost, safety, availability, haemodynamic data, physiological testing, and is most of the time the first-line imaging technique. The main goal of the present document is to provide a set of practical recommendations for imagers who are involved in cardiovascular (CV) diseases or perform vascular procedures. Non-invasive cardiologists may also be interested in getting more familiar to vascular DUS.

This is the first part of the document. It should be considered as a complement to the ESC-ESVS guidelines.[1]

General principles of vascular ultrasonography

Though cardiologists are very familiar to ultrasonography (US), they must be aware that performing 'good quality' vascular US requires a different skill set from echocardiography, a very strict methodology and a formal time-consuming training. For each location (cervicoencephalic, lower limb, and intracranial arteries), a minimal training of 100 exams per year is required. But cardiologists can learn a lot from 'having a look at vessels' which are a key element of the CV system. This document will focus on the specific aspects of vascular US.

Probes

US probes vary in shape, size, and emission frequency. *Table 1* illustrates the display. For difficult cases (obese patients, swollen limbs, and highly calcified vessels), the 'abdominal' convex low-frequency probe can be used for cervicoencephalic arteries (CEA) and limb vessels examinations and the sectorial 'cardiac' probe for aorta examinations.

Table 1 Transducer frequencies typically used for ultrasound examination

Superficial veins	Linear probe	10–14 MHz
Cervicoencephalic vessels	Linear probe	7.5–10 MHz
Abdomen (adult)	Convex probe	3.5 MHz
Transcranial Doppler/ heart (adult)	Sectorial probe	2–3 MHz

Presets

Emission power, amplification gain, dynamic range, and velocity must be individually pre-programmed for each application.

High-resolution B-mode scanning

High-resolution B-mode scanning (*Figure 1*) is used to display the morphological features of vessels and surrounding structures (e.g. muscles, bones).

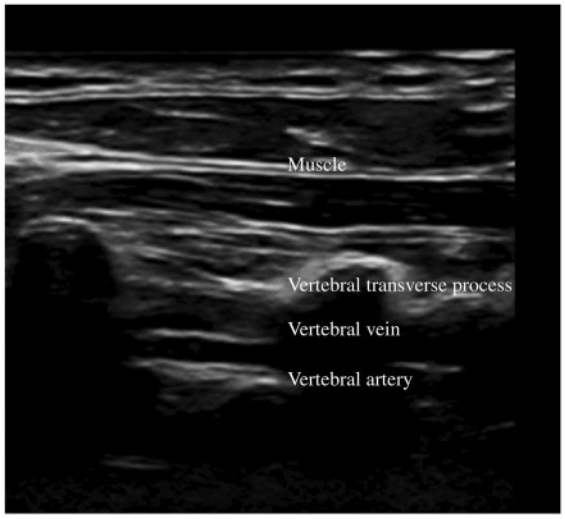

Figure 1 High-resolution B-mode scanning: vertebral vessels in longitudinal view.

DUS

DUS combines pulsed wave (PW) Doppler spectrum and B-mode sonography. The width of the sample must equal half to two-thirds of the vessel lumen. Peak systolic velocity (PSV) and velocity ratio are basic elements in assessing arterial stenosis. In low-resistive arteries (e.g. internal carotid, vertebral, renal arteries), the end diastolic velocity (EDV) is measured (*Figure 2*) and the resistance index (RI) can be calculated: RI = (PSV – EDV)/PSV [In high resistive arteries (e.g. limb arteries), EDV is not taken into account (close to zero).]. RI is useful in specific circumstances (e.g. internal carotid arteries, renal artery stenosis, haemodialysis fistula).

Colour Doppler flow imaging

Colour Doppler flow imaging (*Figure 3*) preserves the advantages of conventional US adding colour-coded blood flow patterns. The colour code depends on the direction of blood flow (see the scale on the corner of the screen) and on the average mean velocity of moving blood cells within the sample volume at a given point in time.

Power Doppler sonography

Power Doppler displays the strength of the Doppler signal in colour, rather than the speed and direction information (*Figure 4*). Its sensitivity for detection of flow is three times higher than conventional colour Doppler, making this technique particularly useful for small vessels and for low-velocity flows, especially for the display of echolucent or anfractuous plaques. Being less angle dependent than colour Doppler, it gives a better display of curving or tortuous vessels. Unlike colour Doppler, it is not altered by aliasing. Its main disadvantage is a lack of information on blood flow direction.

eFlow and B-flow

eFlow (HITACHI-ALOKA) is a blood flow imaging mode having high spatial resolution and high-temporal resolution. In addition, it enhances the processing to discriminate blood flow from tissue (*Figure 5*). The directional eFlow offers blood flow information with high resolution. B-flow (GE) directly images blood reflectors and tissue information simultaneously. eFlow and B-flow give a very precise display of the vessel wall and the plaque.

The angle of insonation

The angle of insonation (angle of incidence, theta or θ angle) is the angle between the incident ultrasound beam and the blood velocity vector (*Figure 6*). The velocity vector must be parallel to the artery axis if there is no stenosis or a moderate one, and parallel to the stenosis flow direction if the stenosis is more severe and oblique. In colour and PW Doppler, θ angle must be <60° (usually between 45 and 60°) in order to avoid overestimation of blood velocity.

Assessment of arterial stenosis

Arterial stenosis is characterized by a high PSV, a high EDV in low-resistive vessels with severe stenosis, a high systolic velocity ratio (SVR, PARTIM I, Carotid Arteries, DUS, Infrainguinal Bypasses, Post-Transluminal Percutaneous Angioplasty/Stenting Surveillance Sections), colour Doppler aliasing, spectral broadening of the Doppler waveform and post-stenotic turbulence. Colour aliasing occurs when the flow velocity exceeds the Nyquist limit. It guides the placement of the spectral Doppler sample volume in case of stenosis. Arterial velocity and SVR are the most reliable tools used to detect and quantify arterial stenosis provided that θ angle is <60°.

Contrast enhanced US

Currently, SonoVue® (sulfur hexafluoride with a phospholipid shell) (Bracco, introduced in 2001) is the only contrast agent used in Europe for contrast enhanced ultrasonography (CEUS) of vascular lesions. Current vascular indications are transcranial Doppler (TCD), display of neovascularization in unstable plaques or leak detection after endovascular aorta repair (EVAR). With US contrast agents the incidence of severe hypersensitivity or anaphylactoid reactions is lower than with current X-ray ones and is comparable to that of MR

contrast agents. US contrast agents are not licensed in pregnancy. Caution should be exercised in patients with severe coronary artery disease (CAD) and pulmonary hypertension. Unstable CAD in the 7 days prior to administration is a relative contraindication.[2] These contraindications derive from unproven causal association between contrast injection and death in severely compromised cardiac patients. The association has not been found in very large cohorts including acute cardiac patients.

Key points

(1) Dedicated probes and pre-sets must be used according to the application.
(2) The blood velocity vector must be aligned with the blood flow direction and the angle of insonation (angle of incidence, theta or θ angle) must be <60°.

CEA examination

Normal examination

DUS is usually the first-line imaging choice for the evaluation of CEA, especially for atherosclerosis. Main indications are listed in *Table 2*. In order to avoid misleading results, DUS should include thorough examination with a standardized protocol. A high frequency (≥7 MHz) linear probe will be used, but for short and/or thick necks, a lower frequency (3–5 MHz) curviline probe ('abdominal probe') may be useful. Positioning of the patient is important in order to ensure operator's comfort and performance. In most cases, the operator is seated at the head or at the side of the patient.

Scanning is performed in both cross sectional and longitudinal views and must include evaluation of the entire course of the accessible portions of the common carotid artery (CCA), the external carotid artery (ECA), the internal carotid artery (ICA), the vertebral artery (VA), and the subclavian artery (SCA) bilaterally. The examination should start with B-mode imaging. In order to screen for anechogenic and/or irregular plaques, imaging must be completed by colour Doppler (Power Doppler, eFlow, or B-flow, if available and necessary). PW Doppler is mandatory too.

Anatomical description is beyond the scope of this document. It should be noted, however, that there are up to 35% of anatomical variants, including branching, level of carotid bifurcation (CBif), length, coiling, and kinking of the arteries.[3]

Key points

Scanning should include cross-sectional and longitudinal views of the entire course of the accessible portions of the CCA, ECA and ICA, VA, and SCA bilaterally, in B-mode and colour-Doppler. PW Doppler is mandatory too.

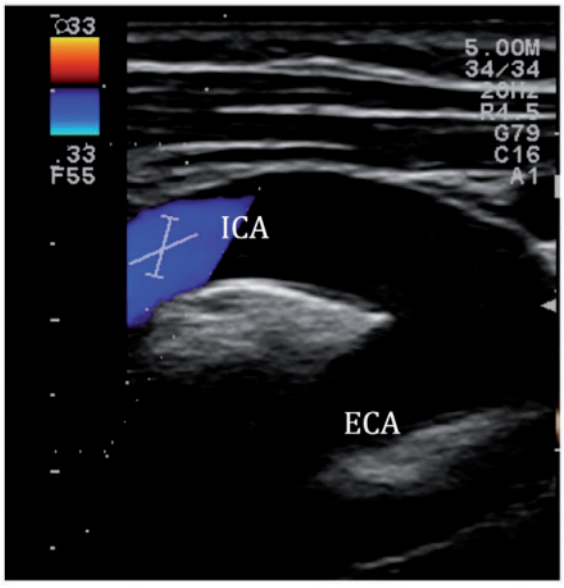

Figure 3 2D echo and colour Doppler flow imaging: carotid bifurcation.

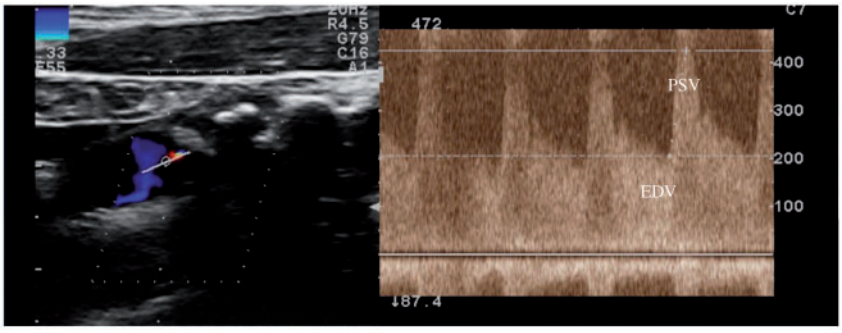

Figure 2 Severe internal carotid artery stenosis with aliasing (arrow on colour Doppler) and high peak systolic and end-diastolic velocities (pulsed wave Doppler).

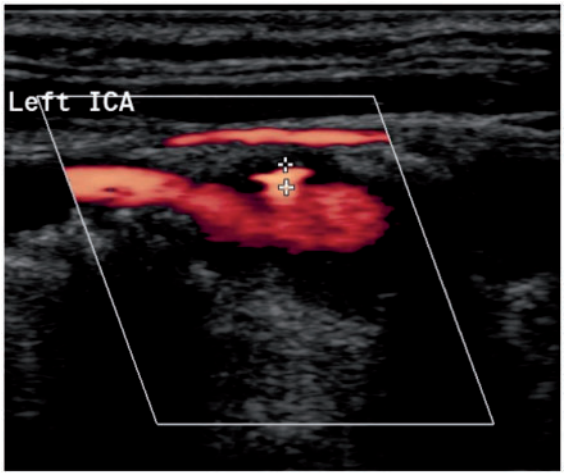

Figure 4 Power Doppler sonography showing an anfractuous plaque in the internal carotid artery.

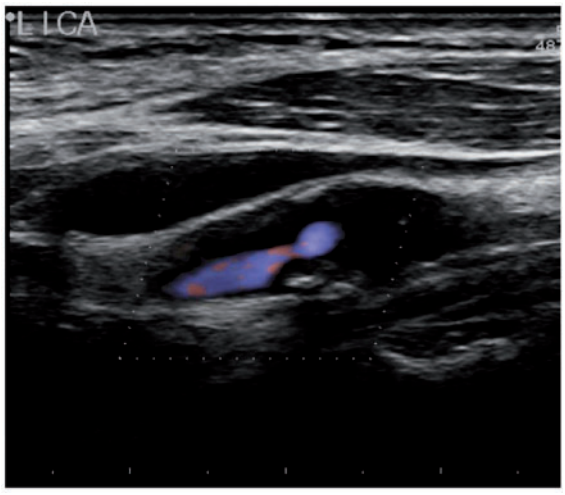

Figure 5 eFlow with better display of of an echolucent plaque.

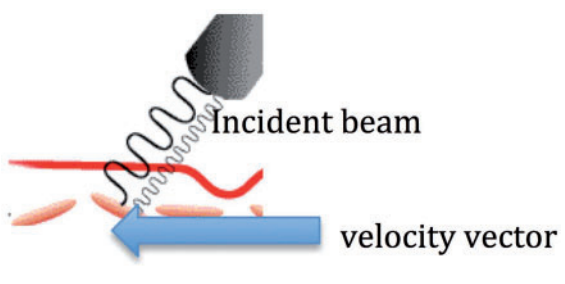

Figure 6 Angle of insonation.

Table 2 Main indications for Duplex ultrasounds of cervicoencephalic arteries

Symptoms	• Stroke/TIA • Visual disturbances/amaurosis fugax • Suspicion of carotid or vertebral dissection/recent Claude–Bernard–Horner • Vertebral insufficiency • Pulsatile tinnitus
Clinical examination	• Cervical bruit • Discrepancy in blood pressure taken in both upper extremities
Surveillance	• >50% carotid stenosis • Carotid endarterectomy or stenting
Cardiovascular risk assessment	• In intermediate risk patients
Screening	• Patients with coronary artery disease and/or lower extremity artery disease (see PARTIM II) • Personal history of neck irradiation
Arteritis	• Giant cell disease, Takayasu disease
Cervical/head trauma	

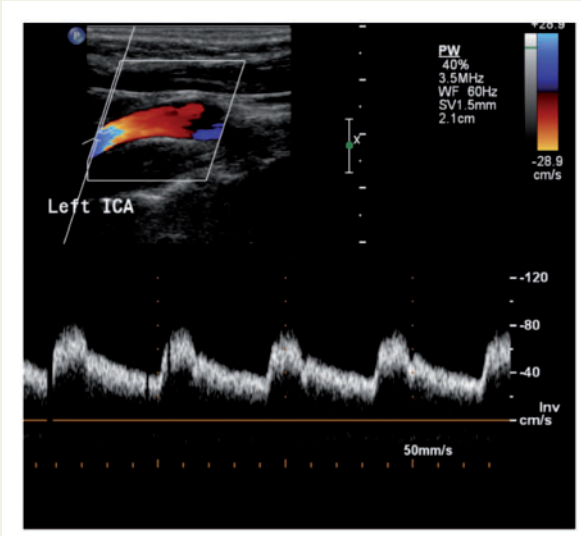

Figure 7 Internal carotid artery (2D echo and pulsed wave Doppler).

Carotid arteries

CCA can be imaged from its origin from the brachiocephalic trunk on the right side and from the aortic arch on the left side, to its ending at the CBif. ICA can be imaged at its origin at the end of the CBif and should be followed as high as possible. (*Figures 3 and 7*) The ECA can be differentiated from the ICA by the presence of branches and a higher RI (*Figure 8*).

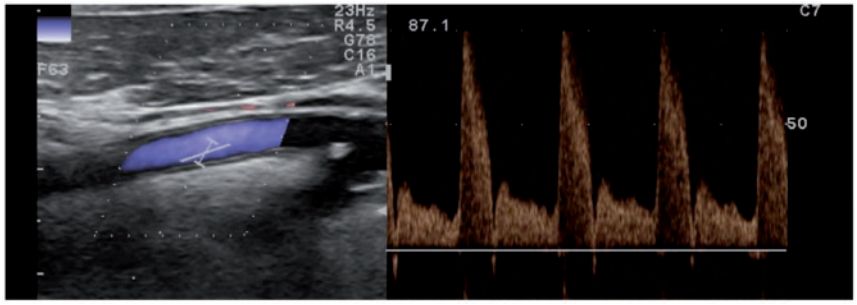

Figure 8 External carotid artery (2D echo and pulsed wave Doppler).

Flow velocities, measured with a θ angle ≤60°, must be recorded in standard locations: proximal CCA (close to the clavicle), mid-CCA, and distal CCA (just before the CBif), ECA, proximal, mid and distal extracranial ICA.

The proximal ICA is a frequent site of stenosis and requires special attention. The distal extracranial ICA should be sampled as high as possible. Often a posterior approach (posterior to the sternocleidomastoidian muscle) is better than an anterior one. PSV and EDV must be measured at each standard ICA site (and at stenotic sites). The carotid ratio is calculated by dividing the maximal $_{ICA}$ PSV by the distal $_{CCA}$ PSV. Normal values are as follows: $_{ICA}$ PSV <125 m/s, $_{ICA}$ EDV <40 m/s, and carotid ratio <2.

In tortuous vessels, correct alignment of the US beam with blood flow direction can be more difficult and lead to overestimated PSV, mimicking stenosis.

Key points

(1) The ECA can be differentiated from the ICA by the presence of branches and a higher RI.
(2) The carotid ratio is calculated by dividing the maximal $_{ICA}$ PSV by the distal $_{CCA}$ PSV.
(3) Normal values are: $_{ICA}$ PSV <125 m/s, $_{ICA}$ EDV <40 m/s, and carotid ratio <2.
(4) Correct alignment of the US beam with blood flow direction is mandatory.

Vertebral arteries

In most patients, VAs are congenitally asymmetrical with unilateral hypoplasia (<2.2 mm) or atresia [without vertebrobasilar (VB) confluence]. VAs are anatomically divided into three extracranial portions (V0 = ostium, V1 between V0 and V2, and V2 in the vertebral transverse processes) and one intracranial portion (V4). The visualization of V0–V1 with US is possible in approximately 65–85% of the cases, but it is the most frequent location of atherosclerosis. If there is any suspicion of VA stenosis, effort must be made to sample as close to the VA origin as possible.

To visualize the VA, the sonographer must first image the mid CCA longitudinally from an anterior approach, then slide the transducer posteriorly until parts of V2 appear between the acoustic shadows of the vertebral transverse processes (*Figure 1*). After obtaining this B-mode image, colour Doppler must be added to assess the direction of flow, and then PW Doppler, in order to evaluate the waveform shape and record PSV. Normal vertebral flow is parallel to CCA flow. Normal PSV values for V2 are approximately 20–60 cm/s and slightly higher at the origin of the VA. RI is higher in the case of V4 atresia and V3 occlusion.

Subclavian arteries

Recording of SCA flow is part of the CEA examination. Normal waveform is triphasic.

Atherosclerosis

Usually atherosclerosis develops gradually. The first structural change in the arterial wall is an increase in the thickness of the intimal and medial layers [intima-media thickness (IMT)], with reduced elasticity and increased arterial stiffness. Later, atherosclerotic plaques form and grow. They can protrude into the lumen, leading to arterial stenosis. At any stage of the chronic process, 'acute' atherosclerotic events can interrupt this development and lead to CV complications. As DUS can usually give accurate structural and haemodynamic information, it is usually considered as the first-line imaging technique. MR and/or CT angiography may be necessary in the case of poor echogenicity (e.g. thick neck, heavy calcifications) or when carotid endarterectomy (EA) is considered. They should be associated when carotid artery stenting (CAS) is considered or in the case of stroke.

Intima-media thickness and plaques

Examination of the carotid wall gives an opportunity to evaluate subclinical alterations in wall structure that might precede CV clinical events.

Intima-media thickness

Carotid IMT has been widely used in trials and in clinical use, but one of its major issues is discrepancy in methodology.[4,5] Currently its systematic use in CV risk assessment is no longer recommended by the ESC guidelines on CV prevention 2016.[6] Anyway, we think that standard measurement criteria need to be briefly described here.[5]

Focus depth (30–40 mm), frame rate (>15–25 Hz), and gain settings should be adjusted in order to optimize edge detection.

Measurement must be taken in diastole. The CCA must be placed in a horizontal position in a longitudinal view with clearly defined lumen-intima and media-adventitia interfaces (perpendicular to the US beam). The posterior approach offers the best resolution. Insonation from multiple angles and zooming are not recommended.[4] The usual site of measurement is the posterior wall of the CCA at least 5 mm below the distal end of the artery, in a segment free of atherosclerotic plaque.

Measuring IMT in the CCA gives better quality images and better intra- and interobserver reproducibility, but as the CCA is less prone to atherosclerosis than the CBif, it leads to underestimation of the real atherosclerotic burden.

IMT should be measured automatically or semi-automatically (150–200 measurements along a 10-mm arterial segment).[6] Measurements should be made in triplicate and IMT values averaged (*Figure 9*). Reproducibility of IMT measurement increases when values from both CCA are combined. Serial studies of IMT to assess progression or regression in individual patients are not recommended.[5,6]

IMT progresses with increasing age and all known CV risk factors (RFs) accelerate this progress. IMT values are significantly higher in men than in women as well as on the left side compared to the right one. Reference values differ from one country to another and defining cut-offs is a controversial topic. For the American Society of Echocardiography Task Force, IMT ≥75th percentile is indicative of increased CV risk, and values from the 25th to the 75th percentile are indicative of unchanged CV risk. There are also more conservative cut-off suggestions: IMT values ≥ age-adjusted 97.5th percentile to be defined as abnormal (and predictive of increased vascular risk).[7]

Key points

(1) Due to the lack of standardization regarding the definition and measurement of IMT, its high variability and low intra-individual reproducibility, the systematic use of carotid IMT in CV risk assessment is no longer recommended by the ESC guidelines on CV prevention 2016.
(2) Serial studies of IMT to assess progression or regression in individual patients are not recommended.

Carotid plaques

Carotid plaques can be present separately or concomitantly with increased IMT. According to the Mannheim Carotid Intima-Media and Plaque Consensus, plaques are focal structures encroaching into the arterial lumen of at least 0.5 mm, or 50% of the adjacent wall thickness, or demonstrating a thickness >1.5 mm as measured from intima-lumen to media-adventitia interfaces.[5]

In order to detect eccentric plaques and measure maximal plaque size, screening requires scanning of the entire cervical carotid tree and visualization in both longitudinal (multiple angle approach) and cross-sectional views at the sites of focal atherosclerosis.[5,6] Location, thickness, and number of plaques should be recorded. Plaques typically develop first in the CBif and the proximal ICA. Colour Doppler with careful adjustment of the velocity scale is mandatory to detect echolucent and/or irregular plaques.[6] Power Doppler, e-flow, and B-flow must be used if available. (*Figures 4 and 10*).

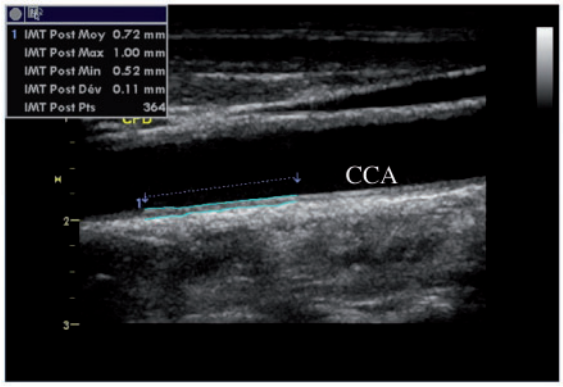

Figure 9 IMT measurement in the common carotid artery.

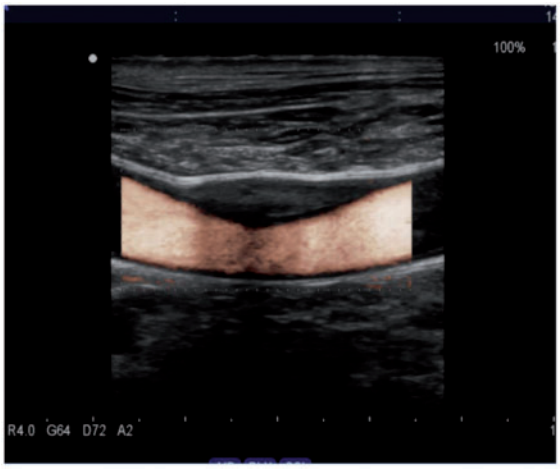

Figure 10 Echolucent plaque in a common carotid artery (longitudinal view).

Presence of plaques further adds to the CV risk assessment because they have a better specificity than IMT alone. According to the ESC guidelines on CV disease 2016, carotid artery plaque assessment may be considered to be a risk modifier in CV risk prediction in some cases.[6] Plaque characterization could have some prognostic implications on stroke. (PARTIM I, Surveillance After Surgery section).

Key points

(1) Plaques are focal structures encroaching into the arterial lumen of at least 0.5 mm or 50% of the surrounding IMT value, or demonstrating a thickness >1.5 mm as measured from intima-lumen to media-adventitia interfaces (Mannheim Consensus).
(2) Plaques have a better specificity for CV risk assessment than IMT alone and may be considered to be a risk modifier in CV risk prediction in some cases.

Stenosis and occlusion of carotid arteries

Due to turbulence and shifts in shear stress flow dividers and branch points of the CEA are more prone to develop stenotic lesions. These parts are mainly the CBif and the origin for the VA.

Carotid artery stenosis/occlusion

ICA stenosis. Apart from highly calcified lesions, stenoses can often be evaluated by direct 2D echo measurements with the use of colour Doppler and if possible, Power Doppler, eFlow, or B-Flow imaging (*Figure 5*). Settings must be optimized and rigorous cross sectional view is mandatory. Longitudinal views may be useful too if the stenosis is not too eccentric.

There are several methods of measurements of carotid stenosis. The most widely used are the North American Symptomatic Carotid Endarterectomy Trial Collaborators' (NASCET)[8] and the European Carotid Surgery Trialists' (ECST)[9] methods (*Figure 11*). As the NASCET method is better related to haemodynamics and angiography, it is proposed as the standard method. On the other hand, the ECST method is better related to plaque burden. The area method (*Figure 12*) can be used too. Interestingly, using the ECST or the area method must be clearly mentioned as they both overestimate the stenosis (*Table 3*).

Compared with CT-scan or MR, the main asset of US is the haemodynamic data. The degree of carotid stenosis can be estimated with $_{ICA}$ PSV, $_{CCA}$ EDV, and carotid ratio ($_{ICA}$ PSV/$_{CCA}$ PSV). There are several tables for estimation of ICA stenosis according to velocities. One of the most widely used was the Society of Radiologists in Ultrasound Consensus (San Francisco 2002).[10] According to the results of carotid stenosis trials (NASCET, ACAS), ICA stenoses were classified as <50%, 50–69%, >70% stenosis, near-occlusions, and occlusions. Grading carotid stenosis using US has been reviewed in 2012 (*Table 4*).[11] In >50% ICA stenosis, PSV is >125 cm/s. EDV mostly increases in >70% stenosis (*Figure 2*). In order to avoid errors due to special haemodynamic conditions [low/high cardiac output (Low/high cardiac outputs, respectively decrease/increase PSV in the CEA.), aortic stenosis (Severe aortic stenosis decreases PSV in the CEA.), contralateral carotid severe stenosis or occlusion (Carotid severe stenosis or occlusion increase the PSV in the contralateral ICA.), collateral flow. . .], 2D echo, $_{ICA}$ PSV, $_{ICA}$ EDV, and carotid ratio must all be integrated. Gender differences in blood velocities across carotid stenosis should also be considered.[12]

Colour Doppler can help detecting severe carotid stenosis by showing aliasing (*Figure 2*).

In the case of severe stenosis (>80%) or occlusion of the ICA, TCD may be useful. The ipsilateral middle cerebral artery (MCA) Doppler waveform is usually altered unless collateralization via the Willis circle is fully functional. Most of the time, PSV are asymmetrical (>30% variation between the right and left MCA flows) and/or the time to peak velocity is >70 ms on the side of the stenosis. The ipsilateral RI may be lowered due to compensatory vasodilation. If TCD is not available, the ophtalmic artery (OphtA) flow must be checked (To perform PW Doppler measurement of OphtA, a 1.5–4.0 MHz linear transducer is positioned on the closed eyelid with minimal pressure. The OphtA is identified as a large caliber vessel adjacent to the optic nerve. The sample volume marker lay approximately 40–50 mm from the eyelid.) (*Figure 13*). Severe ICA stenosis will lower ophtalmic PSV (>30% asymmetry) or reverse the OphtA flow (from the eyeball to the origin of the ophtalmic nerve).

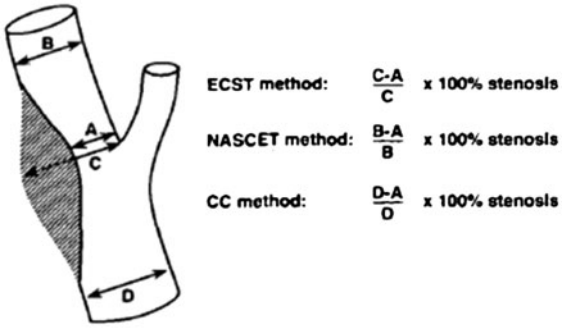

Figure 11 The NASCET/ECST methods of measuring carotid bulb stenosis: *A*, *B*, and *D* are measured with colour Doppler; *C* is the normal lumen diameter before the development of the stenosis.

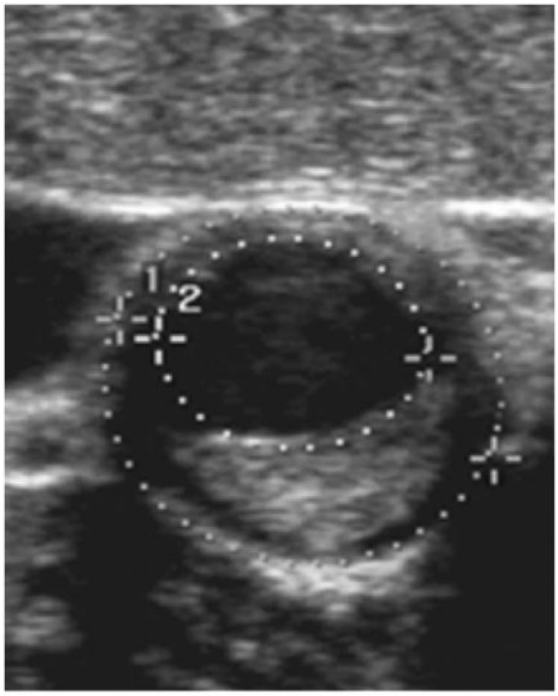

Figure 12 The area method of measuring carotid stenosis.

Table 3 Grading of carotid stenosis according to NASCET and ECST

NASCET (% stenosis)	ECST (% stenosis)
50	75
70	85
80	90

Table 4 Combined criteria for grading internal carotid artery stenosis

% stenosis	10–40%	50%	60%	70%	80%	90%	Occlusion
PSV threshold		125 cm/s		230 cm/s			
PSV average	≤160 cm/s	210 cm/s	240 cm/s	330 cm/s	370 cm/s	Variable	
PSV post-stenotic				≥50 cm/s	<50 cm/s	<30 cm/s	
Collateral flow (circle of Willis/ophtalmic artery)				+ -	+	+	+
EDV in the stenosis			<100 cm/s	>100 cm/s			
Carotid ratio	<2	≥2	≥2	>4	>4		

According to von Reutern *et al.*[11]
EDV, end diastolic velocity; PSV, peak systolic velocity.

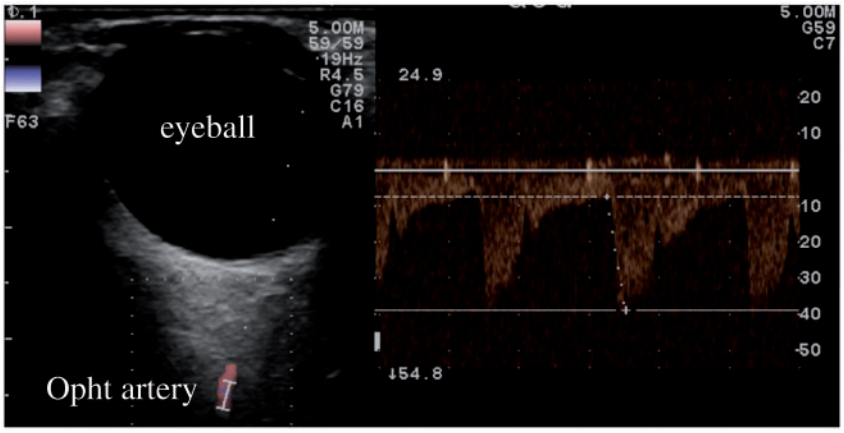

Figure 13 Ophtalmic flow.

Compared with conventional angiography the sensitivity and specificity of DUS for detection or exclusion of ICA stenosis ≥70% range between 85 and 90%.

The morphological characteristics of the stenosis could also have some prognostic and therapeutic implications, but they are subjective and not sufficiently validated. In fact, echolucent stenoses are supposed to be more at risk than iso- or hyperechogenic ones because they usually correspond to lipid deposits, intraplaque necrosis, or haemorrhage. An irregular surface with anfractuosity may correspond to an ulcerated unstable plaque. As blood flow velocities are low in anfractuous lesions, they are better detected by Power Doppler, eFlow, or B-Flow with adapted filters and velocities (*Figure 4*).

Key points

(1) Whenever possible ICA stenosis must be estimated in colour-Doppler (or eFlow, B-Flow, or Power Doppler) and PW Doppler.
(2) The standard measurement method is the NASCET method.
(3) Echolucent and/or anfractuous carotid plaques are supposed to be more at-risk of stroke.

CCA stenosis. CCA stenosis is less common and less well studied than ICA stenosis. It can result from atherosclerosis, inflammatory processes [Takayasu disease, giant cell arteritis (GCA)], or from scarring (e.g. after neck irradiation) and has been linked to stroke and transitory ischaemic attack (TIA) too. The diagnostic criteria for significant CCA stenosis are unclear though doubling of PSV between adjacent segments has been suggested as for other vascular sites. According to a retrospective analysis, comparing DUS with CT, DUS evaluation is highly sensitive, specific, and accurate for detecting lesions in mid and distal CCA. PSV >180 cm/s and EDV >30 cm/s performed as most accurate criteria for detection of ≥50% CCA stenosis.[13]

Carotid artery occlusion. It can be a final step in the gradual process of atherosclerotic plaque evolution but it can also be due to local plaque destabilization with thrombosis or to a large embolus. In the case of carotid artery occlusion, DUS demonstrate the absence of flow and an occlusive thrombus in the lumen (*Figure 14*). Indirect signs of ICA occlusion are a slower and more resistive flow in the ipsilateral CCA (pulsus parvus) (*Figure 15*), a flow inversion in the ipsilateral OphtA, and a damped flow in the ipsilateral MCA. In some cases, an 'internalized flow' (similar to normal ICA flow) in the ipsilateral ECA can mislead the operator.

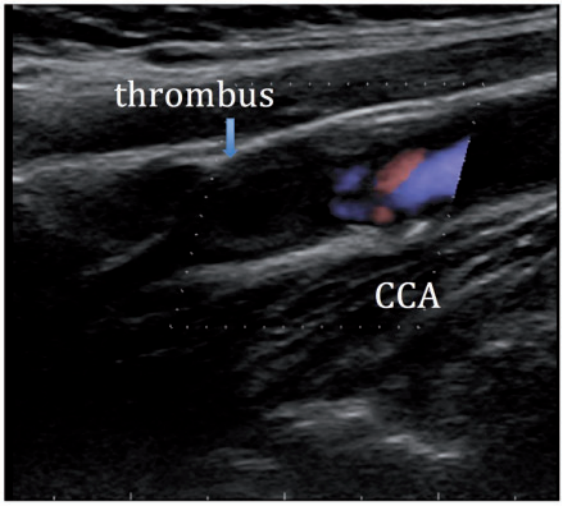

Figure 14 Occlusive thrombus in the CCA.

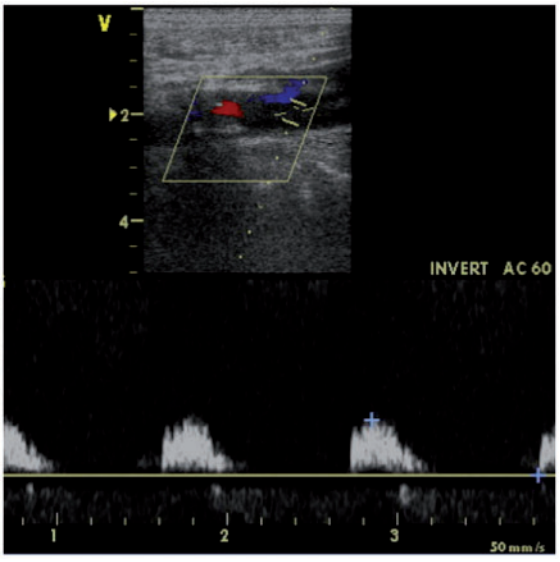

Figure 15 Resistive flow in the common carotid artery due to an occlusion of the ipsilateral internal carotid artery.

Table 5 Gender differences in blood velocities across 65–75% unilateral carotid stenosis

	Men (cm/s)	Women (cm/s)
PSV	231 ± 12	295 ± 17
EDV	78 ± 7	101 ± 8

EDV, end diastolic velocity; PSV, peak systolic velocity.

Pitfalls in assessment of ICA stenosis.[14] Evaluation of CEA can sometimes be challenging and requires awareness of some common pitfalls.

- Gender: $_{ICA}$ PSV is generally higher in women than in men (*Table 5*).
- Carotid stents decrease vessel compliance and increase blood velocity in the vessel (PARTIM I, follow-up after CAS section).
- Tandem lesions: in the case of 2 consecutive lesions, blood velocity may be reduced between the leskions. This should be suspected when there is a discrepancy between B-mode images and PSV. In such a case, the second lesion should be sought for. If it is intracranial, TCD, CT-scan, or MRI need to be performed.
- A long stenosis can lower PSV and lead to underestimation of the severity of the stenosis. 2D echo may help avoid this pitfall.
- Very severe ICA stenosis: $_{ICA}$ PSV increases exponentially with the severity of the ICA stenosis, before decreasing abruptly in very severe stenosis. In that case, high $_{ICA}$ EDV is a sign of severe stenosis. Indirect signs of severe ICA stenosis (TCD, OphtA) can also be sought for, but CT-scan or MR angiography (MRA) may be required to determine the degree of stenosis.
- Near-occlusion (near total occlusion, pseudo-occlusion): If the ICA stenosis is very severe ('hairline lumen'), it can be haemodynamically equivalent to an occlusion with good collaterals. Intra- and post-stenotic blood velocity is then very slow and can be missed by DUS, especially if pulsed repetition frequency (PRF) is set to detect higher flow velocities. This should be suspected with every occlusion detected by US, especially if MCA flows are symmetrical. In order to differentiate near-occlusion from real occlusion, gain should be set to avoid image noise. PRF and filter settings must be lowered to detect very slow flows. Power Doppler, eFlow, B-Flow, and CEUS could also help, but sometimes CT is necessary for the differential diagnosis. The gold standard for distinguishing a pseudo-occlusion from a true occlusion remains conventional arteriography. DUS usually have a better sensitivity than MRI (and sometimes CT-scan) in this indication.
- Aortic stenosis and proximal artery stenosis: if a stenosis is located somewhere between the aortic valve and the proximal part of the CCA, PSV is decreased in the CCA and the ICA ('pulsus parvus and tardus' phenomenon). This can be observed bilaterally (aortic valve stenosis) or unilaterally (stenosis of the brachiocephalic trunk or the proximal CCA). In the case of severe stenosis or occlusion of the right brachiocephalic trunk, blood velocities are lowered in the right CCA, ICA, and ECA as well as in the right SCA with a variable degree of subclavian steal syndrome.
- In case of severe calcification, flow velocities can be difficult to assess. The operator can choose to measure velocities before and after the calcification or try to view the vessel from another approach. The 'abdominal probe' is often very useful. Sometimes different imaging modalities are required (MRI).
- Contralateral carotid occlusion or severe stenosis: flow velocities are usually higher because of increased collateral flow.
- Anatomic variants: some anatomic variants may affect the quality of duplex carotid scanning. A tortuous carotid artery (coiling or kinking) can lead to overestimation of PSV. That is why perfect alignment with the flow direction is mandatory. A high CBif or a short neck can impede the examination of the ICA and tilting the probe posteriorly may be helpful. Another anatomic variant is the persisting trigeminal artery (remnant of the foetal cerebral circulation that bridges the carotid and basilar arterial territories) which

reduces flow resistance in the ICA with high EDV and low RI (normal range 0.7–0.9).

Vertebral arteries

Non-invasive imaging of the VA is less well studied than that of carotid arteries although atheromatous changes develop with a similar frequency in both anatomic locations. Though a stenosis of the VA can be clinically suspected, it is often diagnosed incidentally, whereas imaging CEA for other causes.

Vertebral stenosis is often located at the origin of the artery and may be detected by a delayed rise time in the post-stenotic segment (indirect sign) or by direct insonation of the origin of the artery and detection of focal velocities >100 cm/s. VA occlusion is more difficult to diagnose and differentiate from congenital aplasia or hypoplasia. 2D echo can help. Distal obstructions of the VA (V3) can be detected by a demonstration of a high resistive flow—pulsus parvus in V2.[15] TCD can confirm the cervical vertebral occlusion by recording a reverse flow in the ipsilateral intracranial VA (V4).

In many cases, US detection of an ostial VA stenosis is difficult. Dedicated probes may help but CTA and contrast-enhanced MRA have higher sensitivity and specificity. Catheter-based contrast angiography remains the gold standard of VA imaging for revascularization candidates,[3] but indications are rare.

SCA stenosis may alter vertebral flow by causing subclavian steal syndrome. This pathology is described in PARTIM II.

Key points

(1) VA stenosis is often located at the origin of the artery.
(2) VA stenosis may be detected by a delayed rise time in the post-stenotic segment or by focal velocities >100 cm/s.
(3) VA occlusion is more difficult to diagnose and differentiate from congenital aplasia or hypoplasia.
(4) V3 obstructions can be detected by a high resistive flow in V2 .and a reverse flow in the ipsilateral V4.

Surveillance of asymptomatic carotid stenosis on best medical treatment

According to the 2017 ESC-ESVS guidelines,[1] carotid EA should be considered in patients with an asymptomatic 60–99% ICA stenosis with average surgical risk (<3%) and life expectancy >5 years (IIaB) if an increased risk of late ipsilateral stroke is suspected. This recommendation is validated by data suggesting that the risk of stroke with 'BMT alone'[16] is lower than what was reported in previous studies like ACAS or ACST. Targeting the at-risk patient who will benefit from carotid surgery or stenting and considering 'BMT alone' for the majority have become burning issues. For patients under BMT alone, surveillance of the carotid stenosis and evaluation of stroke risk have become increasingly important.

Unlike the severity of an asymptomatic carotid stenosis, its progression (>20%) is a risk predictor of stroke and TIA, and of a composite endpoint of myocardial infarction, percutaneous coronary or peripheral intervention, coronary or vascular surgery, amputation, stroke, and all-cause mortality. A first 6-month, DUS will identify a progression of the stenosis, leading to an adaptation of the therapeutic strategy. In the case of non-progression, surveillance can be scheduled every 12–18 months.

According to Geroulakos *et al.*[17] (*Table 6*), plaque characterization can also be predictive of stenosis progression and clinical events: hypoechoic, heterogeneous, and irregular plaques are risk markers of CV events, especially stroke and TIA. As hypoechoic plaques are at higher risk of progression, Geroulakos' Type 1, 2, and 3 carotid plaques should be followed more closely. Other features associated with increased risk of stroke are: contralateral TIA/stroke, spontaneous embolization on TCD monitoring (HITS), impaired cerebral reserve, large plaques, intraplaque haemorrhage or lipid-rich necrotic core (MRA).

Key points

(1) EA should be considered in patients with an asymptomatic 60–99% ICA stenosis with average surgical risk (<3%) and life expectancy >5 years, if an increased risk of late ipsilateral stroke is suspected (IIaB, 2017 ESC-ESVS guidelines).
(2) Features associated with increased risk of stroke are: contralateral TIA/stroke, stenosis progression (>20%), HITS, impaired cerebral reserve, large and/or echolucent plaques, increased juxta-luminal black (hypoechogenic) area, intraplaque haemorrhage, or lipid-rich necrotic core (MRA).
(3) A first 6-month, DUS will identify a progression of the stenosis. In the case of non-progression, surveillance can be scheduled every 12–18 months.
(4) Hypoechoic plaques (Geroulakos' Type 1, 2, and 3) should be followed more closely.

Surveillance after carotid surgery

Up to about 2 years after surgery, restenosis is generally attributed to neointimal hyperplasia with accumulation of smooth muscle cells and fibrous tissue. The risk of stroke is usually low. Later, restenosis is generally caused by recurrent atherosclerosis with increased macrophage infiltration, calcification, and lipid core resembling primary plaques with higher risk of stroke or TIA. An isoechoic, concentric plaque is in favour of neointimal hyperplasia, whereas atherosclerotic lesions are more heterogeneous and eccentric, mimicking a primary plaque.

Table 6 Characterization of the carotid plaque

Type 1	**Predominantly echolucent with a thin echogenic cap**
Type 2	Intermediate echolucent lesions with small areas of echogenicity
Type 3	Intermediate echogenic lesions with small areas of echolucency (<25%)
Type 4	Uniformly echogenic lesions (equivalent to homogenous)
Type 5	Cannot be classified (acoustic shadowing artefact)

According to Geroulakos *et al.*[17]

Table 7 Peak systolic velocity, end diastolic velocity, and carotid ratio cut-offs for restenosis after carotid endarterectomy with patch

	PSV (cm/s)	EDV (cm/s)	Carotid ratio
>30%	155	41	1.64
>50%	213	60	2.25
>70%	274	80	3.35

According to AbuRahma *et al.*[16]
EDV, end diastolic velocity; PSV, peak systolic velocity.

The PSV velocity cut-off for >60% restenosis is between 150 cm/s and 220 cm/s. For EA with patch closing, restenosis cut-offs have been proposed by AbuRahma *et al.*[18] (*Table 7*) It is noteworthy that the mean PSV of a normal ICA distal to carotid EA patching is higher than normal carotid arteries.

Protocols of surveillance in the different trials and guidelines recommend DUS at 1, 3, 6, 12 months, and then annually. However, considering the low risk of recurrent stenosis or occlusion after EA, a periprocedural control followed by a yearly surveillance should be sufficient in most cases. In at-risk population (women, diabetics, smokers) and in patients with an ipsilateral restenosis ≥50%, contralateral disease progression is more frequent and surveillance may be appropriate.

Key points

(1) Restenosis after carotid EA: upto about 2 years after surgery, it is generally attributed to neointimal hyperplasia (low risk of stroke). Later, restenosis is generally caused by recurrent atherosclerosis (higher risk of stroke).
(2) The PSV cut-off for >60% restenosis is between 150 and 220 cm/s.
(3) Protocol of DUS surveillance after carotid EA: 1, 3, 6, 12 months, and then annually or at least, a periprocedural control followed by a yearly surveillance except in 'at-risk population'.

Follow-up after carotid stenting

According to several studies (EVA-3S,[19] CREST, ICSS. . .), CAS and carotid EA are associated with low and similar long-term (up to 10 years) risks of recurrent ipsilateral stroke beyond the procedural period.

Different reports indicate that CAS may cause a reduction of compliance resulting in an increase of blood velocity despite any restenosis. Therefore, velocity cut-offs are higher for CAS. Furthermore, DUS velocities could be higher when using closed cell stents as compared with open cell stents. Different thresholds of PSV have been proposed for high-grade in-stent restenosis. Based on the current available literature, a PSV value of 300–350 cm/s and a carotid ratio >4–4.5 could be used as relatively good and sensitive predictors of high-grade in-stent restenosis. *Table 8* proposes cut-offs for in stent re-stenosis.[20]

Table 8 In-stent restenosis cut-offs

% stenosis (NASCET)	PSV (cm/s)	EDV (cm/s)	Carotid ratio
≥30%	>150	>42	1.5–2.5
≥50%	>220	>88	2.7–3.4
≥80%	>320	>119	4.1–4.5

According to AbuRahma *et al.*[20]
EDV, end diastolic velocity; PSV, peak systolic velocity.

Immediate post-stenting DUS examination provides a baseline value for future surveillance comparisons. According to the literature, DUS can be recommended at 1, 6, 12 months, and then annually.

Key points

(1) A PSV value of 300–350 cm/s and a carotid ratio >4–4.5 could be used as relatively good and sensitive predictors of high grade in-stent restenosis.
(2) After carotid stenting, DUS can be recommended at 1, 6, 12 months, and then annually.

Arteritis

Giant cell disease

GCA disease can affect supraaortic arteries in two ways:

The 'temporal arteritis' or 'Horton's disease' mainly involving temporal arteries. It can coexist with GCA of the CEA, but is usually limited to temporal arteries.

The second type which is mainly located in the subclavian, axillary and brachial arteries with a consecutive involvement of the CCAs and ECAs. Temporal arteries are usually not affected by arteritis in that circumstance (PARTIM II).

DUS is the first-line imaging technique for GCA. PET-CT and MRA can be associated in 'extra-temporal' involvement.

The examination of temporal arteries should start at the origin of the common superficial temporal artery and continue as far as possible to the distal area of the frontal and parietal ramus. Both sides need to be examined. Flow velocities may be measured, but except for stenosis, they provide no additional information for the diagnosis. In temporal arteritis, there is a typical hypoechoic dark area around the perfused lumen ('halo sign') due to oedema of the artery wall, and sometimes there is a consequent stenosis of the temporal arteries (*Figure 16*) in the acute phase.[21] The halo disappears within 2–3 weeks after starting steroid therapy. Scanning has always to be displayed both in longitudinal and cross-sectional views. The use of modern scanners with high resolution is essential (longitudinal linear 12 or 14 MHz transducers). The colour intensity must be adapted in order to avoid overpainting that could mask the hypoechoic halo. Lesions detected by US usually show the typical pattern of skip lesions (consecutive short lesions separated by normal unaffected portions due to segmentary arteritis). US can also detect occluded vessels. In this case, the artery can be found by grey scale US with no

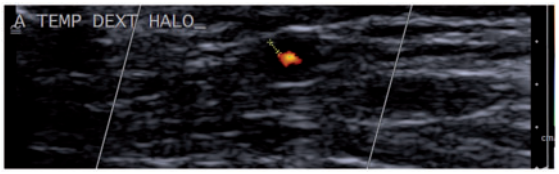

Figure 16 Giant cell arteritis: colour Doppler image of an inflamed temporal artery. The black region surrounding the red zone in the image shows the characteristic « halo sign ».

colour Doppler signal in the obliterated lumen. The sensitivity of temporal artery DUS is 87% with regard to the clinical diagnosis, and the specificity is about 96%. Pre-operative temporal DUS can guide the biopsy. In the case of positive DUS, biopsy might not be necessary.

Key points

(1) GCA can affect supraaortic arteries in two ways: 'temporal arteritis', also named 'Horton's disease', mainly limited to temporal arteries, and arteritis mainly located in the subclavian, axillary, and brachial arteries with a consecutive involvement of the CCAs and ECAs, and sometimes of the aorta.
(2) In GCA, DUS show a typical skip lesion with a hypoechoic dark area around the perfused lumen ('halo sign'). This sign disappears within 2–3 weeks after starting steroids.

Takayasu disease

Though patients with Takayasu disease are notably younger, GCA and Takayasu have close similarities in terms of thickening of the vessel wall and response to steroids with some overlap in the pattern of involved arteries.[22] However, Takayasu most commonly affects the left post-vertebral SCA, CCAs, and renal arteries (branches of the aorta) and never involves the temporal arteries. Both entities may involve the aorta (PARTIM II). In Takayasu elastic fibres are destructed (elastophagia), sometimes leading to aneurysm and fibrosis can lead to stenosis and sometimes thrombosis.

Imaging usually associates DUS, CTA, MRA, and PET-CT.

The typical Takayasu lesion identified by DUS is a long, smooth, homogeneous concentric thickening of the arterial wall, usually more hyperechogenic than in GCA ('Macaroni' sign). In contrast, an atherosclerotic plaque is shown to be non-homogeneous, often calcified and associated with an irregular vessel wall (*Figure 17*).

DUS may be more sensitive than any other diagnostic procedure, through its ability to detect the intima-media thickening (resolution of 0.1–0.2 mm in CCA) associated with early pre-stenotic lesions in the CCAs and SCAs ('prepulseless' disease). The high resolution of DUS raises the possibility that it may also help monitoring disease activity and response to treatment.[22] Carotid contrast echo allows

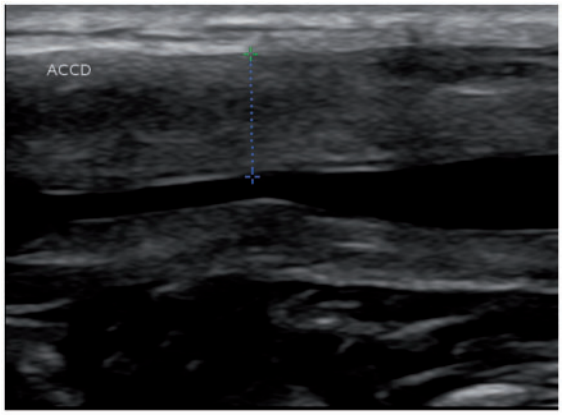

Figure 17 Ultrasound image of a right common carotid artery with typical multilayer thickening of vessel wall in Takayasu disease.

dynamic assessment of carotid wall neovascularization, which is a potential marker of disease activity.[23] Sadly, though widely available, the use of high-resolution DUS is relatively underinvestigated in Takayasu disease.

Key points

(1) Takayasu disease most commonly affects the left post-vertebral SCA, CCAs, and renal arteries, and never involves the temporal arteries. It may involve the aorta.
(2) Takayasu disease onset is usually at an age of 40 years.
(3) DUS show a long, smooth, homogeneous concentric thickening of the arterial wall, usually more hyperechogenic than in GCA ('Macaroni' sign).
(4) Takayasu disease may lead to arterial stenosis, aneurysm and sometimes thrombosis.

Carotid/VA dissection

CEA dissection is a major cause of ischaemic stroke in young and middle-aged adults (mean age 45 years).[24] It can occur after major and minor head and neck trauma, but other RF have been proposed such as hypertension and fibromuscular dysplasia (FMD) (PARTIM I, FMD section). Though genetic factors probably act as parts of a multifactorial predisposition, a monogenic connective tissue disease (vascular Ehlers–Danlos syndrome, Loeys–Dietz syndrome) is rarely found.

DUS is a widely available and quick non-invasive diagnostic tool for dissection diagnosis, but it requires much experience and expert training. In ICA dissection, a mural haematoma is located in the arterial wall and causes a stenosis primarily located in the post-bulbar part of the artery. This portion of the ICA may be studied with a curvilinear probe,[25] but in many cases only indirect DUS signs are present

(increased or decreased pulsatility or bilateral blood flow velocity difference of >50%). VA dissection causes a stenosis or occlusion in approximately 80% of all cases but the sensitivity of DUS is even slightly lower in the vertebral circulation compared to the anterior circulation. This implies that both positive and negative US findings should be confirmed by MRI (or CT). Whenever possible, TCD should be added in order to evaluate the haemodynamic repercussions of carotid/vertebral dissection.

Recurrent dissections are two-fold more frequent than previously thought, and follow-up (FUp) studies can preferably be done by DUS.[25] DUS are also useful for assessing re-opening of the artery or regression of the stenosis during the FUp.

Key points

(1) Immediate imaging of the cervical arteries should be performed in all young or middle-aged adult patients with ischaemic stroke, TIA, or transient monocular blindness.
(2) MRI is the first line imaging method, but US performed by an experienced sonographer and completed by TCD can in most cases give a fast and reliable diagnosis.

Fibromuscular dysplasia

FMD is a non-atherosclerotic, non-inflammatory vascular disease. It is usually found in the renal (60–75%) and extracranial cerebrovascular circulation (25–30%), but it has also been described in almost every medium-sized or large artery of the body. It results in luminal narrowing with or without associated aneurysm or dissection of the media.[26]

FMD of CEA, including the VA, is often bilateral. The ICA is typically involved at the level of C1–C2 (mid to distal cervical ICA). FMD of the CEA and SCA is more multifocal and bilateral than isolated RA FMD. Patients with cerebrovascular FMD have a higher incidence of intracranial aneurysms (especially intracranial ICA and MCA) recommending the realization of cerebral MRI or CT-scan in those patients.[26]

DUS can visualize velocity shifts and turbulences indicative of arterial stenosis in the mid- to distal-cervical ICA and the VA. A « string of beads » pattern may be identified too, although it is less common and requires good US training. Although it may not be specific to FMD, severe tortuosity (S curve) of ICA and/or VA in a less than 70 year old patient should also alert the sonographer. Because of the distal location of the FMD, DUS examination of the entire ICA and VA is mandatory, but currently, CT-scan is acknowledged as better than MRA and DUS. Because of the nature and morphology of the FMD lesions, DUS standard cut-offs used for grading of atherosclerotic carotid stenoses do not apply. Unfortunately we do not have validated data yet.[27]

DUS can be an excellent modality for surveillance of carotid artery FMD, but fortunately the disease is usually not progressive. DUS surveillance can be performed every 6 of 12 months initially and then annually.[27]

Key points

(1) Because of the distal location of the carotid FMD, DUS examination of the entire ICA is mandatory, and DUS probably has a lower sensitivity than angiography and CT-scan.
(2) DUS standard criteria used for atherosclerotic carotid stenoses do not apply in FMD.
(3) Due to a higher incidence of intracranial aneurysms (ICA and MCA) in FMD, cerebral MRI, or CT-scan are recommended in patients with such dysplasia.
(4) DUS diagnosis of vertebral FMD is challenging.
(5) For complete evaluation of cervical artery dissection, CTA and MRA are the preferred modalities.

Radiation-induced carotid atherosclerosis

Increased IMT and higher grade of abnormality in the bulb wall were observed at doses higher than 35 Gy.[28] The relative risk of stroke has been reported to be higher in patients treated for head and neck cancer compared with patients treated by radiation for breast cancer patients or non-Hodgkin's lymphoma. The risk of radiation-induced carotid atherosclerosis increases with smoking and with the time interval from radiotherapy. Routine US screening should be proposed to patients who had received radiotherapy more than 5 years previously.[29]

The disease is limited to (or more severe in) the irradiated area.

The diagnostic and grading of radiation-induced carotid atherosclerosis using DUS is based on the same principles as atherosclerotic carotid artery stenosis grading and plaques morphologic description.

Aortoiliac and lower extremity arteries

Normal examination

Scanning technique

DUS is the mainstay in non-invasive lower extremity artery disease (LEAD) imaging. The complexity and the accuracy of the examination are influenced by the clinical context (*Table 9*). General principles of US are of course applied for LEAD (PARTIM I, General Principles of Vascular Ultrasonography section). A 5–10 MHz linear array transducer is used for limb examination. A low-frequency curvilinear probe is preferred for aortoiliac screening or for enlarged or swollen limbs. The patient is supine on a dedicated examination table with the trunk slightly inclined at 30°, to allow relaxation of the muscles of the abdominal wall. The arms are positioned along the body and the legs should adopt a slight external rotation with discrete knee flexion.

The examination usually begins with the recording of the ankle artery flow and systolic blood pressure (SBP). The ankle brachial index (ABI) is a simple method to confirm a suspected LEAD, and is also indicative of the severity of the disease. The technical aspects of ABI measurement are described in PARTIM I, Ankle-Brachial

Table 9 Levels of lower extremity artery ultrasonography

	Level/accuracy of the examination	Specific indications
Focused exploration	Limited to the lesion site	-Screening for LEAD in high risk asymptomatic patients (ABI) -Screening for AAA-Early systematic follow-up after surgery -Searching for complication of endovascular procedures (pseudoaneurysm, arteriovenous fistula, haematoma. . .)
Standard examination	Definition and description of the location of affected site(s)	-Symptomatic LEAD (intermittent claudication, critical limb ischaemia, abnormal peripheral pulse, arterial bruit. . .)
Comprehensive examination	Detailed and accurate description (ultrasonic arteriography)	-Preoperative examination -Guiding endovascular procedures

AAA, abdominal aortic aneurysm; ABI, ankle brachial index; LEAD, lower extremity artery disease.

Index section. The cut-off values of ABI values have been recently adapted.[30]

The limb exploration is then continued segment by segment. It is strongly recommended to fully evaluate one leg at a time rather than compare both sides segment by segment. Iliac vessels are generally assessed indirectly by the analysis of the Doppler waveform in the common femoral artery (CFA). Direct examination of the iliac axis at the end of the examination is of course recommended, mainly if an iliac stenosis is suspected, but the patient morphotype or presence of abdominal gas do not always allow it. Calf arteries can be evaluated in detail but the examination is usually limited to the recording of the ankle arteries flow. This is the order in which we advise to proceed: ankle/pedal arteries, CFA, origin of the deep femoral arteries (DFA), superficial femoral arteries (SFA), popliteal arteries (PopA) on both legs with the linear probe, and then abdominal aorta (AA), external (EIA), internal (IIA), and common iliac arteries (CIAs) with the curvilinear probe. In a normal examination, Doppler waveform is triphasic from iliac to ankle arteries (*Figure 18*).

During image acquisition it is convenient to use a schematic representation of the aortoiliac and LEA in order to annotate it and progressively reproduce an angiography-like description.

Key points

(1) ABI = ankle artery SBP/brachial artery SBP.
(2) Cut-offs: 0.9 ≤ ABI ≤ 1.4

CFA

The CFA runs from the inguinal ligament to its division into SFA and DFA, 3–6 cm distally. The CFA is detected in 2D echo and colour Doppler is then added. It is located externally to the common femoral vein. A normal Doppler signal is triphasic. An abnormal morphology with increased rise time and/or damped signal suggests a significant iliac disease (indirect sign).

The femoral bifurcation requires a detailed examination, as it is a privileged site for atherosclerosis. The DFA runs postero-laterally to supply the major thigh muscles. It should be scanned along its proximal centimetres, as it is a major collateral pathway in patients with significant SFA disease.

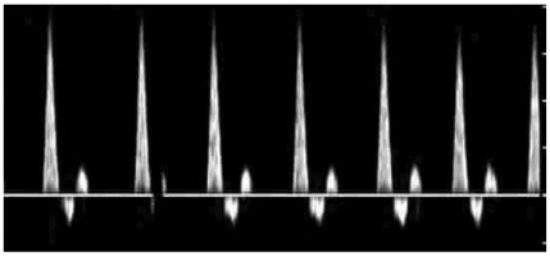

Figure 18 Triphasic flow.

SFA

The SFA runs downwards along the antero-medial edge of the thigh, anterior to the SFV. In the lower third of the thigh, it passes through the adductor canal, becoming posterior to the femur. The examination begins at the femoral bifurcation and is carried along the length of the thigh using colour Doppler. Doppler velocity waveform is sequentially recorded, as many times as necessary to check all points of possible disease but at least in the upper, middle and lower thigh. Specific attention is paid to the adductor canal where the SFA can be the site of short stenosis or occlusion. In some cases, the use of a lower-frequency curvilinear transducer ('abdominal probe') may be helpful, as at this particular location the vessel is deep located.

Popliteal artery

In the popliteal fossa, the popliteal artery (PopA) is anterior to the vein. Below the knee, it divides into the anterior tibial artery (ATA) and the tibioperoneal trunk. The height of this division is variable.

Calf arteries

The posterior tibial artery (PTA) is easily accessible behind the medial malleolus, where its position is constant, and then followed along the leg. The peroneal artery runs deeply, lying closer of the posterior aspect the tibia and the interosseous membrane. The ATA is identified through an antero-lateral approach. The detailed description of the calf arteries may require the use of US contrast agents but most of

the time they are simply evaluated by comparing Doppler velocity waveform between the PopA and the ankle vessels.

Foot arteries

In foot blood flow is supplied by the ATA through the dorsalis pedis artery (dorsal part of the foot) and by the PTA, dividing in medial and lateral plantar arteries (plantar part). Diabetic arteriopathy typically involves the tibioperoneal vessels, with sparing of the pedal vessels (assisting pedal bypass).

Abdominal aorta

Optimally, the patient should be fasting for about 12 h (at least 6 h) prior to the examination to limit interference by bowel gas, but it is rarely so. The examination begins with the probe placed in a subcostal position. The AA is located anterior to the vertebral bodies (L1–L4), slightly left of midline. The AA is then progressively scanned transversally and longitudinally from the diaphragm to the aortoiliac bifurcation. The AA should be measured in a cross-sectional view with the US beam perpendicular to the AA axis. The anteroposterior and transverse diameters should be equal. If it is not possible to obtain a circular section of the AA (in case of dilatation and/or sinuosity), one can calculate the mean diameter of the ellipse or try to measure the AA diameter in a good longitudinal view, provided the diameter is perpendicular to the AA axis. Diameters are measured from adventitia to adventitia. Alternative methods are described in PARTIM I, AAA section. The anteroposterior diameter measurement is more accurate than the transverse one because perpendicularity of US confers a more precise display. In case of abundant bowel gas, the patient is asked to turn on the left side and the transverse and longitudinal diameters are then obtained in the coronal plane.

As the transducer is slowly moved caudally, the abdominal branches of the AA are progressively identified: the celiac trunk dividing into common hepatic and splenic arteries, the superior mesenteric artery (1 cm distally to the celiac trunk), the right renal artery emerging from the anterolateral portion of the aorta (generally seen more easily than the left one), the left renal artery caudally and posteriorly and finally, the inferior mesenteric artery.

Spectral Doppler analysis of intestinal arteries will demonstrate different resistance waveform profiles, depending on the delay relative to the last meal for 'intestinal arteries' (low-resistance profile following a meal and normal resistance profile in fasting patients). The normal renal artery spectral Doppler always demonstrates a low-resistance waveform.

Key points

(1) The anteroposterior and transverse diameters of the AA are preferably obtained in a cross sectional view and should be equal. Diameters are measured from adventitia to adventitia. The anteroposterior diameter measurement is more accurate than the transverse one.
(2) If the AA is dilated or tortuous, one can calculate the mean diameter of the ellipse or try to measure the AA diameter (perpendicular to its axis) in a good longitudinal view, provided the diameter is perpendicular to the AA axis.

Iliac axis

As previously mentioned, the iliac vessels can firstly be indirectly evaluated from the CFA. A triphasic Doppler signal in the CFA of an asymptomatic patient excludes an ipsilateral severe stenosis on the iliac axis. If the anatomy is favourable, it is recommended to combine waveform recording in the CFA with a direct examination. If necessary, the patient is asked to turn on one side and then on the other. The image acquisition sequence includes the examination of the aortic bifurcation, the CIA, the iliac bifurcations, the EIAs, and iliofemoral junctions. Whenever it is possible, proximal IIAs should be examined. As for lower limbs, it is recommended to evaluate one side at a time.

Exercise testing

Eliciting symptoms through exercise may diagnose LEAD in patients with typical exercise leg symptoms and normal (or near normal) ABI by unmasking a lesion non-critical at rest that becomes significant on exercise. Many exercise protocols exist. It is usually more convenient to measure ABI with continuous wave (CW) Doppler. Patients with symptomatic LEAD will demonstrate a fall in the ABI, directly correlated to the severity of the occlusive disease (PARTIM I, Post-Exercise ABI section).

Key points

(1) Non-critical lesions (especially iliac stenoses) can show a normal or near-normal ABI at rest.
(2) Exercise-testing can unmask these lesions.

Atherosclerosis

LEAD is mainly due to atherosclerosis. As a generalized process, atherosclerosis typically involves both legs, though often not to the same extent. Iliac stenoses/occlusions are located in the CIA, IIA, and/or EIA. Most common sites of femoral artery atherosclerotic lesions are the femoral bifurcation and the adductor canal. The DFA mainly supplies thigh muscles. Though it is rarely affected by atherosclerosis distal to its origin, stenosis at its origin is more common in patients with atherosclerosis of the femoral bifurcation and is clinically relevant in case of femoropopliteal (FP) occlusion, as DFA is the principal SFA collateral. Occlusion of both the femoral and popliteal arteries is also common.

DUS

Combined with ankle- or toe-brachial index, the major asset of DUS is the haemodynamic estimation of the stenosis, which better correlates with its ischaemic effects and patient's symptoms, in contrast to angiography that only provides morphological information.[31] Apart from acute critical ischaemia, DUS provides all the information necessary for first-line management decisions in the majority of patients. It can also differentiate atherosclerotic from non-atherosclerotic vascular diseases, and it is particularly useful for the FUp after surgical and endovascular procedures.

Despite its great advantages, DUS is operator-dependent and requires adequate qualification and training. Uniform training and interaction with the surgical team is a requirement for large vascular laboratories.

Performance of DUS may also be limited by obesity, bowel gas, edema, surgical incisions, scars, ulcers, joint contracture, and arterial calcification producing acoustic shadowing (calcified plaques, medial arterial calcification).

Plaque/calcification

Atherosclerotic lesions appear on 2D imaging as irregularities of the endothelial surface, intima-media thickening and plaques. They may cause vessel stenosis. Atherosclerotic calcifications must be differentiated from medial arterial calcinosis (MAC), which is not atherosclerotic (PARTIM I, MAC section).

Images are best obtained in the longitudinal axis of the vessels, while transverse views may occasionally be used to define anatomic relationships. Evaluation of plaque morphology is not a formal part of DUS examination of LEAD at present.

Iliofemoral arteries are the most common location of atherosclerotic plaques especially in men and should probably be looked for as a CV risk marker in intermediate risk people.[32]

Stenosis/occlusion

Technical modalities are described in PARTIM I, Normal examination section. A preliminary survey enables identification of stenosis (turbulent flow and aliasing in high-grade stenosis), occlusions and origins of collaterals. Abnormal findings need to be confirmed and quantified (Grading of Stenosis section—PARTIM I) in the longitudinal plane by PW Doppler. In addition to colour-Doppler, Power Doppler imaging, eFlow, or B-Flow can help.

PW Doppler waveform and PSV must be recorded at all the standard locations (CFA, proximal, middle and distal SFA, origin of the CFA, PopA) and at any area of colour or grayscale abnormality (lumen narrowing, plaque and/or flow disturbances such as aliasing, turbulent jet, increased velocity), as well as in the arterial segment 2–4 cm proximal (upstream) to any stenosis. PW Doppler spectra may indirectly suggest the absence or presence of a tight stenosis upstream according to the triphasic or monophasic waveform, respectively. In difficult cases a hyperaemia or stress test (treadmill or else) is necessary to rule out significant aorto-iliac stenoses even with normal or almost normal CFA waveforms.

Key points

(1) The morphology of the waveform is important.
(2) In difficult cases a hyperaemia or stress test is necessary to rule out significant aorto-iliac stenoses even with normal or almost normal CFA waveforms.

Grading of stenosis

In lower limbs, the stenosis severity is assessed by its haemodynamic consequences (Doppler) and not by its morphologic appearance. A ≥50% reduction in diameter is expected to increase PSV, proportionally to the degree of stenosis.

Modifications of PSV, SVR (SVR = intra-stenotic/pre-stenotic PSV) (direct criteria) and flow distal to the stenosis (indirect criteria) are shown in *Table 10* and discussed in detail below. In normal individuals, PSV and arterial diameters decrease towards the periphery, while the normal triphasic flow profile is preserved.[30] As many other factors may also affect PSV (e.g. SBP, reduced vessel wall elasticity, sympathetic tone, presence of an upstream stenosis, collaterals), it is difficult to define a PSV threshold for haemodynamically significant stenosis and local SVR is preferred. A SVR ≥2 is consistent with a ≥50% stenosis and SVR ≥3 with a ≥70% stenosis. Distal to a medium/high-grade stenosis or an occlusion, the normal triphasic flow profile is altered with a post-stenotic decrease in PSV and increase in EDV due to vasodilatation. A delayed systolic upstroke is also seen. Severe below-knee lesions cause a significant discrepancy between ankle/pedal and popliteal waveforms. It is noteworthy that normal triphasic pattern may also change to monophasic waveform in case of normally occurring peripheral dilatation, caused by factors such as increased muscle activity on exercise, fever, and local infection.

The haemodynamic effects of a stenosis also depend on a complex interaction of different factors: presence of collaterals (see Effect of Collaterals section), length or morphology of the stenosis, multiple stenoses.[30] Proximal to a high-grade stenosis, a pattern of high-resistance flows immediately upstream the stenosis is reflected by an increase in the reverse flow component (Pre-stenotic pulsatility may also be reduced before sites of origins of collaterals.).

The extent and severity of LEAD must be characterized. Morphologic findings, direct and indirect criteria that help identify and quantify a stenosis (or an occlusion) should be included in the report.[33] Multilevel disease is very common in LEAD and should be expected in patients with critical limb ischaemia. In these cases, flow pattern should be investigated at short-length intervals and great care should be taken in the assessment of lesions severity.

Key points

(1) Morphologic findings, direct and indirect criteria that help identify and quantify a stenosis (or an occlusion) should be included in the report.
(2) A local SVR ≥2 is consistent with a ≥50% stenosis and SVR ≥3 corresponds to a ≥70% stenosis.
(3) Multilevel disease is very common in LEAD and should be expected in patients with critical limb ischaemia.

Effect of collaterals

Collateral arteries are often detected proximal and distal to a severe stenosis or occlusion, as they tend to reconstitute the main arterial trunk. Several pitfalls in DUS interpretation are related to the presence of collaterals:

- Flow from collaterals coursing parallel to the occluded artery may be misinterpreted as flow within the occluded segment.
- If collateralization is poor, slow post-occlusive flow may lead to over-estimation of the occluded segment.
- If collateralization is poor, the Doppler waveform distal to the occlusion segment is damped with a large diastolic component, resulting from peripheral dilatation in response to chronic

Table 10 Grading the severity of stenotic lesions in lower extremity arteries

Severity of stenosis	Intra-stenotic waveform	SVR	Colour flow and waveform just distal to the stenosis	Waveform far distal to the stenosis	Waveform proximal to the stenosis
No stenosis	Triphasic waveform	<1.3	No or minimal broadening Markedly pulsatile flow Steep systolic upslope	Unchanged	Unchanged
Mild or low grade stenosis (20–49%)	Triphasic waveform	<2	Waveform is usually triphasic Minimal spectral broadening	Same as pre-stenotic	Normal
Moderate or intermediate stenosis (50–75%)	Monophasic waveform with loss of reverse flow component PI[a] slightly reduced	2–3.9	Waveform monophasic Eddy currents Slight turbulence Spectral broadening with partial filling-in of the clear area under the systolic peak	Monophasic waveform PI[a] reduced	Normal
High-grade stenosis (>75%)	Monophasic with loss of the reverse and diastolic forward flow components) PI[a] reduced (increase in PSV and EDV)	≥4	Monophasic flow Considerable turbulence Marked broadening (completely filled-in systolic window)	Damped monophasic waveform with reduced PSV and lengthened systolic acceleration time (>100 ms) PI[a] reduced	Amplitude normal or reduced with increasing stenosis severity (compared to other side) PI[a] increased immediately proximal to a high-grade stenosis PI[a] may be reduced before sites of collaterals origin
Occlusion (100%)	No flow signal detectable Length of occlusion may be estimated by the scan distance between exit and re-entry collateral arteries	–	Monophasic Markedly damped waveform Very reduced flow downstream	Very flat and reduced systolic peak Monophasic	Amplitude very low 'thump' pattern immediately proximal to the occlusion: increased PI[a], small complex with low PSV and large negative component PI[a] reduced before sites of collaterals' origin

Modified from Schäberle.[31]
EDV, end diastolic velocity; PI, pulsatility index; PSV, peak systolic velocity; SVR, systolic velocity ratio; V, velocity.
[a]PI = PSV-EDV/mean velocity.

peripheral ischaemia. The poorer the collateral situation, the more monophasic the waveform and the lower the PSV become.

- Accelerated PSV in a collateral may be misinterpreted as a stenosis due to the smaller diameter of the collateral.
- If collateralization is good, PSV in the stenotic segment may be lower than expected (The flow volume in the immediate prestenotic segment decreases with increasing flow through the collaterals.).
- If collateralization is good, blood flow distal to an occluded segment can approach normal flow.
- In the case of CFA/EIA occlusion, flow may be reversed in the DFA and supply ipsilateral CFA and/or SFA. In the case of CIA occlusion, the same pattern can be seen in the IIA.

Doppler ultrasound protocols

Complete DUS scanning of the entire arterial network is labour- and time-consuming, especially if infrapopliteal arteries are to be assessed. In these cases and especially when a bypass is considered, another imaging technique is usually required. However, even in this situation, DUS can be an important aid in determining the most appropriate site of anastomosis by identification of the least calcified portion of the vessel. Consequently the protocol can differ according to clinical questions as well as specific anatomic considerations. A short DUS or a detailed artery mapping or a focused, limited examination may be appropriate.

Short Doppler ultrasound protocol

A short protocol is used when all level waveforms are clearly triphasic, with satisfactory ABI, in an asymptomatic patient. ABI and angle-corrected spectral Doppler waveforms with velocity measurements should be obtained bilaterally at a minimum, from the following sites in the LEA: CFA, proximal SFA, mid SFA, distal SFA, PopA. If clinically appropriate, imaging of the iliac, deep

femoral, tibio-peroneal, and dorsalis pedis arteries can also be performed.

Short protocols may also vary according to the treatment plan, being different for limb salvage, claudication, or emergency situations. For example, patients with Stage II LEAD, most frequently do not require a full evaluation of the calf arteries below the tibiofibular trunk as revascularization in this territory is not indicated at this stage. In Stages III and IV LEAD, candidates for surgical revascularization above the PopA may not require pre-operative digital substraction angiography or DUS mapping of the infrapopliteal arteries provided that a short protocol demonstrates a patent, non-stenotic popliteal segment and the primary surgical approach is not expected to be affected by occlusions distal to the PopA or trifurcation.[31]

Long Doppler ultrasound protocol

A long DUS protocol, initially designed to mimic digital substraction angiography, includes ABI and imaging of the aorta, iliac arteries, CFA, SFA, DFA, PopA, calf, and pedal arteries. Such DUS protocols are not often performed because they are time-consuming. However, detailed arterial mapping may be particularly important in patients with renal insufficiency to avoid angiographic contrast exposure.

A long DUS protocol may also be needed before an infrainguinal bypass in order to select arterial segments for placement of proximal and distal anastomoses. In these cases and in order to reduce scanning time, a shorter protocol may stop at the most distal location of a stenosis or occlusion that needs to be bypassed.

Ankle-brachial index

Methodology: ankle-brachial index = ankle SBP/arm SBP

In normal patients, ankle-brachial index (ABI) is >1, since ankle SBP is higher than arm SBP (because of the progressive pulse amplification phenomenon when moving away from the heart.). In patients with LEAD, the ABI tends to decrease as atherosclerosis spares upper limbs arteries. An ABI <0.90 has 75% sensitivity and 86% specificity to diagnose LEAD.[34]

The ABI can be used for two different (but not exclusive) purposes:[1]

- First-line diagnostic tool when LEAD is suspected.
- Prognostic marker for CV events and mortality (e.g. to stratify the risk of an asymptomatic individual with an intermediate CV risk score).[35]

The major asset of the ABI is its high availability at low expense. It requires a hand-held CW Doppler device or a DUS probe, and a pressure cuff. The technical aspects for a standardized measurement of the ABI are extensively presented in a recent consensus document.[35] The patient should rest for 5–10 min prior to the measurement and keep lying still on his back. The pressure cuff is placed just above the ankle (the direct application of the Doppler probe or the cuff on wounded zones should be avoided). When the arterial Doppler signal is detected, the cuff is inflated until the signal disappears, and then slowly deflated. The Doppler signal reappearance determines the SBP. This manoeuvre should be repeated bilaterally at the sites of brachial arteries, PTA and ATA or DPA. If a SBP difference >10 mmHg is found between both arms, a white coat effect must be suspected and brachial SBPs must be checked. If the inter-arm SBP difference persists, the arm with the higher one will be considered as the reference. Despite many attempts to validate alternative methods to measure ABI, mostly using oscillometric devices, Doppler remains nowadays the standard reference {The oscillometric method [automated blood pressure (BP) cuffs] presents several drawbacks, especially as it may overestimate pressure in case of low ankle pressures, leading to missing some LEAD cases.}.[36]

The ABI of each leg should be calculated by dividing the higher of the PTA or DPA SBP by the higher of the arms SBP. If assessing LEAD, the ABI of each lower extremity should be taken into account separately. Conversely when ABI is used to determine the CV risk, the lower ABI between the two legs is retained as patient's ABI. The reproducibility of the ABI is acceptable when performed by a trained physician/technician.

When LEAD is clinically suspected, an ABI >0.90 does not definitely rule out the diagnosis, and further investigations, including post-exercise ABI and/or US is necessary.

Post-exercise ABI

The ABI is first measured at rest, and then after exercise test (usually treadmill test using Strandness protocol, at 3 km/h speed and 10% slope), which is stopped when the patient reaches its maximal walking distance. A post-exercise ankle SBP decrease >30 mmHg or a post-exercise ABI decrease >20% are diagnostic for LEAD.[35]

Surveillance

In non-revascularized patients with LEAD, ABI decreases slowly over time as the disease progresses. Because of reproducibility issues, the ABI change at an individual level is significant only if it exceeds ±0.15. In trained but non-revascularized patients, ABI change is poorly correlated with symptoms evolution or functional performance. After revascularization, ABI alone is insufficient to estimate the perfusion amelioration and the risk of secondary thrombosis (An ABI increase of 0.10 and 0.15 in the revascularized limb predicts no residual stenosis >50% with sensitivities of 79% and 67% and specificities of 92% and 100%, respectively.[37]).

MAC

Occasionally ankle SBP and ABI are excessively high (ABI >1.40) or even not measurable because of arterial incompressibility despite cuff pressures as high as 250 mmHg (higher pressures are uncomfortable and useless. They should be avoided.). These situations are related to MAC (MAC is distinct from atherosclerosis as it does not occlude the arterial lumen, unless it is associated with atherosclerosis, which occurs in 40-50% of cases. Isolated MAC appears to have favourable limb and general outcomes if not associated with occlusive LEAD.[38]) (PARTIM I, MAC section), mostly observed in the very elderly, or in the case of long lasting diabetes or renal failure with haemodialysis. In this case, the toe-brachial index (TBI) is useful: TBI = toe SBP/humeral SBP (*Table 11*). Toe SBP can be measured with photoplethysmography, LASER-Doppler or capillaroscopy. TBI normal values are ≥0.7.

Key points

- ABI = lower extremity higher SBP/upper arm higher SBP
- Normal value: 0.9 ≤ ABI ≤1.4 (on a lying patient)
- When LEAD is clinically suspected and ABI is >0.90, post-exercise ABI and/or US may be necessary. With the treadmill test using Strandness protocol (3 km/h speed and 10% slope), a post-exercise ankle BP decrease >30 mmHg or a post-exercise ABI decrease >20% are diagnostic for LEAD.
- After revascularization, ABI alone is insufficient to estimate the perfusion amelioration and the risk of secondary thrombosis
- ABI is also a prognostic marker for CV events and mortality. The lower ABI between the two legs is retained as patient's ABI
- FUp: The ABI change at an individual level is significant only if it exceeds ±0.15.
- Occasionally ABI is >1.40 or even not measurable because of arterial incompressibility (MAC). TBI can assess distal flow in those cases.

Table 11 Ankle-brachial index, toe systolic blood pressure, and toe-brachial index

	Normal	Abnormal
ABI	1–1.4	≤ 0.9 (0.91–0.99: borderline) >1.4: non-compressible arteries – unreliable
Toe SBP	120 ± 20 mmHg	
TBI	>0.7	

ABI, ankle brachial index; SBP, systolic blood pressure; TBI, toe-brachial index.

Aneurysms

Aneurysms are the most frequent abdominal and lower extremity disease after atherosclerosis. US assessment is pivotal for their detection and FUp. Patients with aortic or peripheral aneurysms are at high risk for presenting aneurysms in other localizations, either simultaneously or over time, so that lifelong DUS assessment of other main localizations is warranted.

Abdominal aortic aneurysm

The abdominal portion is the most frequent localization of aortic aneurysms, mostly infra-renal. The most commonly accepted definition of abdominal aortic aneurysm (AAA) is a diameter >30 mm.[39] Some authors propose a diameter exceeding 1.5 time the 'expected normal' infra-renal aortic diameter, which can be estimated if the adjacent portion is normal with parallel borders. This is suggested to compensate for variations due to gender, as AA is on average 2 mm smaller in women than in males. The 2014 ESC guidelines on the diagnosis and management of aortic diseases suggest indexing the aortic diameter to body surface in outlier body size.[39] At least, when AAA diameter nears the thresholds for intervention, gender or body surface should be taken into account.

The ESC guidelines recommend population screening of AAA by US in men >65 years. It may also be considered in women >65 years with a history of smoking. Targeted screening for AAA should also be considered in first-degree siblings of a patient with AAA.[39] Some countries have developed population AAA screening programmes, but this is still lacking in many countries. The ESC guidelines recommend opportunistic US screening for AAA at the end of any transthoracic echocardiography (TTE) performed in the population at risk as defined above.[39] DUS is the cornerstone method for the screening and diagnosis of AAA and healthcare workers can be trained to measure the diameter of the AA, even using portable machines. The AA can be reliably imaged in >95% of subjects and AAA diagnosed with excellent sensitivity and specificity.[40] Because of the increased risk of tandem lesions, patients with AAA should have thoracic aorta imaging.[41]

AA scanning is described in PARTIM I, Abdominal Aorta section. The most consensual measurement is the outer-to-outer anteroposterior diameter, as the evidence for the optimal threshold for intervention is based on the external diameter.[41] An alternative method measuring inner-to-inner (internal) diameter is proposed, but this diameter is on average 4 mm smaller that the external one, with similar reproducibility. The best reproducibility is found with the leading edge-to-leading edge method. However, the diameters obtained by the two latter alternative methods are more difficult to compare with those obtained other imaging methods (CT, MRI). The transversal diameter estimation by US should be considered with caution, as the lateral edges of the aorta are less well delineated because non-perpendicular to the US beam, leading to poorer measurement reproducibility. When the largest diameter of the aneurysm is transversal, it should be attempted to be measured by a lateral incidence (the probe at the patient's flank), or using another imaging method for confrontation. In some cases, the aorta is very tortuous and the actual long axis of the vessel is difficult to determine. In this case, a longitudinal view of the aorta can be proposed to determine the sagittal diameter. Yet, in any report after AA echography, the exact diameter (AP or any else), and the measurement method (external diameters or alternative methods) should be clearly reported. This is of outmost importance for the sake of comparison of the results to other imaging methods, or when the aorta is repeatedly measured over time to assess AAA growth.

Besides its diameter, AAA localization should be described, especially whether the RA are involved, and if not, the diameter of the proximal neck and the distance between the RA and the proximal neck. The presence of mural thrombus (*Figure 19*) should be described. Similarly, the distal extension (whether it prolongs to aneurysmal iliac arteries) of the AAA should be mentioned too. All these data are important for the suitability for proposing EVAR when intervention is indicated. A full assessment of the peripheral arteries distal to the AAA is recommended.[39] But usually CT or MRI are mandatory before intervention.

The ESC guidelines advocate surveillance rather than intervention for AAA of 30–54 mm (the maximal threshold can be 50 mm in

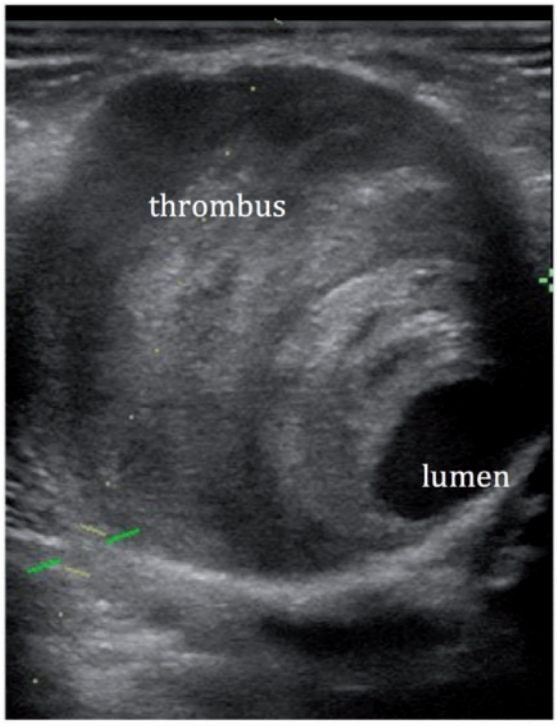

Figure 19 Aortic abdominal aneurysm with mural thrombus.

women, in a case by case discussion).[39] They propose necessary intervals between repeated US imaging, according to the initial aortic diameter. Beyond the diameter, a rapid growth (>10 mm/year or >5 mm in 6 months) is considered at high risk of rupture and intervention is then indicated.

US may diagnose ruptured AAA, but its performance should not delay further imaging (CT) and referral to the operative room.

After open repair, DUS can be useful to assess the proximal and distal anastomosis and the patency of vessels involved during the surgical procedure, although the performance is lower during the postoperative days because of intra-abdominal gas interpositions and surgical wound. After the first year, long-term US surveillance of the prosthetic graft is recommended at loose intervals.

Long-term DUS surveillance can even be more useful after EVAR, especially in the absence of any complication (sac enlargement or endoleaks) identified by CT during the first year, if the abdominal imaging is satisfactory, with or without contrast media, to detect long-term complications. In case of satisfying image in the hands of experts, it can supplant CT beyond the first year of surveillance, but the latter remains the gold standard if any doubt persists. In a meta-analysis, colour-DUS presented 96% sensitivity and 85% specificity to detect any type of endoleaks and 99% sensitivity and 100% specificity to detect Type-1 or -3 endoleaks.[42] Contrast media improve sensibility. Most endoleaks missed by DUS and contrast-enhanced US are Type 2 endoleaks with no need for reintervention.

Key points

(1) Definition of AAA: diameter >30 mm or diameter exceeding 1.5 time the 'expected normal' infra-renal aortic diameter.
(2) US screening for AAA: men >65 years, women >65 years with a history of smoking and first-relatives of patients with aortic aneurysms (ESC). Opportunistic screening of AAA is recommended at the end of any TTE performed in the population at risk (ESC guidelines).
(3) Patients with AAA should have thoracic aorta imaging.
(4) The most consensual measurement is the outer-to-outer anteroposterior diameter.
(5) If the aorta is very tortuous, a longitudinal view of the aorta can be proposed to determine the sagittal diameter. Yet, the measurement method should be clearly reported.
(6) The ESC guidelines advocate surveillance rather than intervention for AAA of 30–54 mm (the maximal threshold can be 50 mm in women, in a case by case discussion). A rapid growth (>10 mm/year or >5 mm in 6 months) is also considered at high risk of rupture.
(7) After open repair, long-term DUS surveillance is recommended at loose intervals.
(8) After EVAR, especially in the absence of sac enlargement or endoleaks identified by CT during the first year and in case of satisfying image in the hands of experts, DUS can supplant CT beyond the first year of surveillance. CT-scan remains the gold standard if any doubt persists.

Popliteal artery aneurysm

Popliteal artery aneurysm (PAA) is generally defined by an increased diameter >15 mm, but the best acknowledged definition is a loss of parietal parallelism with a diameter exceeding 1.5 time the 'expected normal' diameter. PAA are bilateral in 50% of cases and associated with AAA in 40% of cases. So once a PAA is detected, AAA should be systematically screened for.[43] In contrast to AAA, the main complication of PAA is not rupture but multiple repeated distal embolization (*Figure 20*) leading to progressive rarefaction of the distal arterial network prior to the clinical ischaemic presentation. These patients are at high-risk of amputation.

DUS imaging is the method of choice to assess PAA and its complications. A thorough assessment is necessary, to confirm the diagnosis and provide the maximal adventitia-to-adventitia diameter (either lateral or AP) perpendicular to the vessels axis, after having eliminated the other major differential diagnoses: large PopA (arteriomegaly), adventitial cyst, synovial cyst, post-stenotic arterial dilation related to an entrapped PopA, or a false aneurysm at the anastomotic site of a bypass. It should also detect mural thrombosis (colour Doppler or contrast media may be useful), and assess the distal arterial permeability. During the same US imaging sequence, the full arterial vasculature in both limbs must be assessed, and the venous repercussions (thrombosis, compression with venous flow slowing) should be systematically evaluated.

A detailed description of all these parameters is essential when surveillance (rather than intervention) is considered. When PAA

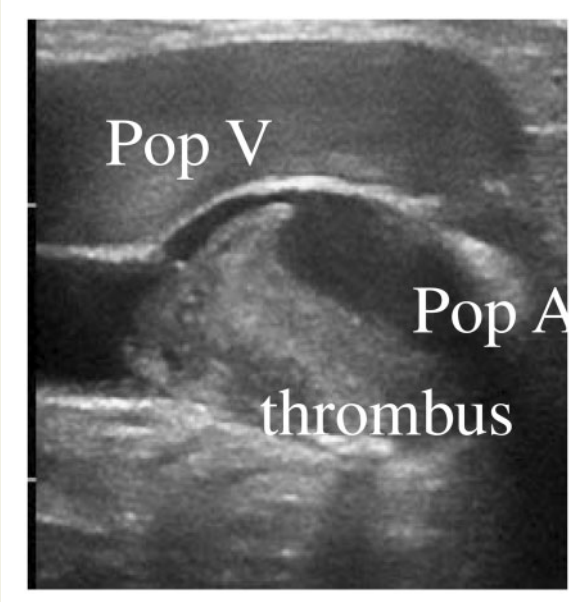

Figure 20 Longitudinal view of a popliteal aneurysm with a large and mobile thrombus (high risk of distal embolism).

diameter is <20 mm without thrombus, watchful waiting can be proposed, with US reassessment every 6 months in the absence of any symptom. PAA >30 mm and/or symptomatic PAA should be operated (endovascular or mostly open repair). Surgical repair can be proposed for PAA between 20 and 30 mm if the operative risk is low, the runoff is acceptable, if there is an adequate venous conduit or a distorsion of the proximal conduit >45° (higher embolic risk). After endovascular or surgical repair, regular US assessment of the operated site (and other arteries) is necessary.

Key points

(1) DUS is the method of choice to assess PAA and its complications.
(2) If PAA diameter is <20 mm without thrombus, watchful waiting can be proposed, with US reassessment every 6 months in the absence of any symptom.
(3) PAA >30 mm should be operated.
(4) Surgical repair can be proposed for PAA between 20 and 30 mm if the operative risk is low.
(5) After repair or reperfusion, regular US assessment of the operated site (and other arteries) is necessary.

Miscellaneous

MAC

Medial arterial calcification (MAC) can occur in otherwise normal young patients, but it is common in elderly patients as well as in patients suffering from diabetes mellitus, severe renal impairment, or hypervitaminosis D. Unlike atherosclerotic calcification, which typically forms in the intima, MAC is located in the medial layers of the artery and does not obstruct the arterial lumen (*Figure 21*). It is frequently encountered in aorta and in lower limb arteries (SFA, PopA, and infrageniculate arteries). It can cause incompressibility of ankle arteries with overestimated (>1.4) or unmeasurable ABI (PARTIM I, MAC section).

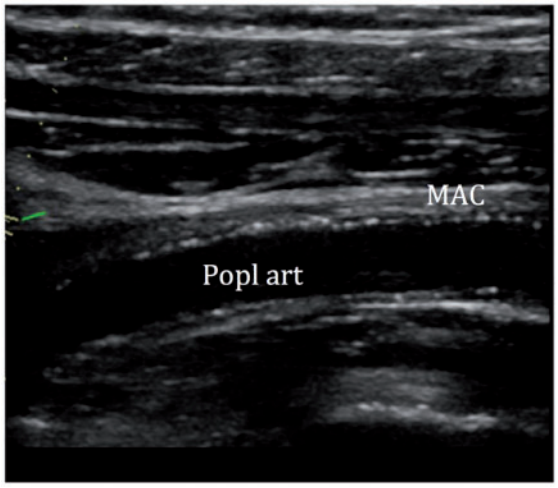

Figure 21 Medial arterial calcinosis: hyperechogenic spots.

Key points

(1) Medial arterial calcification is common in elderly patients as well as in patients suffering from diabetes mellitus, severe renal impairment or hypervitaminosis D.
(2) It can cause incompressibility of ankle arteries with overestimated (>1.4) or unmeasurable ABI.

Arterial embolism

Emboli most frequently lodge at aortoiliac or femoral bifurcations or at popliteal trifurcation and abruptly stop blood flow. In case of acute limb ischaemia, DUS can be performed rapidly and assess the level of occlusion. Anyway imaging should not delay revascularization.

Arteritis

GCA and Takayasu disease are described in PARTIM I, Arteritis section. They may affect the aorta and GCA sometimes extends to LEAs causing intermittent claudication.

Buerger's disease

Thromboangiitis obliterans (TAO) or Buerger's disease is a non-atherosclerotic, segmental, inflammatory disease that most commonly affects the small and medium-sized arteries and veins of the upper and lower extremities. US investigation is used to check for a lack of

atherosclerotic lesions and can identify the distal sites of symptomatic arterial occlusion and other sites of lesions. Measurement of toe SBP, mostly with LASER-Doppler, and measurement of transcutaneous oxygen pressure (TcPO2) can be used to confirm the severity of ischaemia.

A typical pattern which some consider pathognomonic for the disease is the occurrence of 'corkscrew collaterals' that occur typically in the calf and forearm region as a consequence of occlusion of the native vessels. These corkscrew collaterals can be detected by US.

Key points

(1) TAO or Buerger's disease is a non-atherosclerotic, segmental, inflammatory disease that most commonly affects the small and medium-sized arteries and veins of the upper and lower extremities.
(2) Measurement of toe SBP, mostly with LASER-Doppler, and TcPO2 can confirm the severity of ischaemia.
(3) Corkscrew collaterals can be detected by US.

Surveillance after bypass grafting

DUS looks for early (<6 months) (early complications sometimes infection) and late (>6 months) post-surgical complications. The aim of this surveillance is a more effective prevention of graft thrombosis.

A comprehensive description of the procedure and previous DUS examinations should be available to the sonographer before beginning the exam. Unfortunately it is not always so! DUS includes ABI measurement and complete arterial mapping of the lower limb. According to the clinical context, remote sites (aortoiliac arteries, contralateral limb arteries) will be examined too. Colour Doppler findings will guide PW Doppler interrogation.

Aortobifemoral bypass

They may show stenosis, kinking, or pseudoaneurysm. FUp programmes are still debated.

Infrainguinal graft

For infrainguinal vein grafts, the entire length of the graft should be imaged and PSV (with optimal angle of insonation) recorded in the proximal native artery, at the proximal anastomosis, throughout the graft segment, at the distal anastomosis and in the distal native vessel. Stenosis, false-aneurysm, mural thrombus, kinking must be looked for in longitudinal and cross-sectional views especially in the proximal anastomosis graft segment. Aliasing in colour Doppler imaging can help identify stenoses. PSV must be recorded in 3–4 graft sites without stenosis and averaged to calculate the graft flow velocity (GFV). Compression of superficial venous grafts by the probe must be avoided. The SVR (SVR = $PSV_{max}/PSV_{proximal}$ to the stenosis) must be calculated at sites of stenosis.

FUp programme is still controversial but DUS can be proposed 3 weeks, 6 and 12 months post-operatively, and then annually. The surveillance should be individualized to the patient, type of bypass, and duplex scan findings.

Elements of a surveillance programme after infrainguinal vein bypass grafting are shown in *Table 12*. According to Bandyk *et al.*,[45] GFV <40–45 cm/s and absent diastolic forward flow need prompt correction because they are associated with early graft occlusion. High-grade stenoses (>70%) must be corrected too. Early moderate or intermediate stenoses should be monitored and treated in case of progression. Most stenoses develop within the first post-operative year. They are often located within the mid-portion and at anastomotic sites of the venous graft.

Unlike vein grafts, prosthetic grafts fail more frequently because of impaired native vessel outflow or poor arterial inflow. FUp programmes are even less standardized. DUS is advised in the immediate post-operative period and at regular intervals for at least 2 years, taking the interval history (new symptoms), vascular examination, ABI at rest and DUS modifications into account.[46] High-grade stenoses (>70%) are defined as PSV >250 cm/s, EDV >20 cm/s, SVR across the stenosis >3–3.5.

Key points

(1) FUp after bypass grafting looks for stenosis, false-aneurysms, local recurrence of the disease or development of new arterial lesions at remote sites.
(2) Autogenous vein grafts usually fail within their mid-portion and at anastomotic sites. FUp programme is still controversial, but DUS can be proposed 3 weeks, 6 and 12 months post-operatively, and then annually. The surveillance should be individualized to the patient, type of bypass, and duplex scan findings.
(3) For prosthetic grafts, FUp programmes are even less standardized. DUS is advised in the immediate post-operative period and at regular intervals for at least 2 years, taking the interval history (new symptoms), vascular examination, ABI at rest and DUS modifications into account.
(4) Aortobifemoral bypasses may show stenosis, kinking or false aneurysm.
(5) For infrainguinal bypasses, PSV must be recorded in 3–4 non-stenosed graft sites and averaged to calculate the GFV. SVR (SVR = $PSV_{max}/PSV_{proximal}$ to the stenosis) is calculated at sites of stenosis.
(6) GFV <40 cm/s and absent diastolic forward flow need prompt correction.
(7) >70% stenoses must be corrected.
(8) Intermediate and moderate stenoses should be monitored and treated in case of progression

Post-transluminal percutaneous angioplasty/stenting surveillance

Early adverse events are rare except haematomas. They include acute thrombosis, arterial dissection, distal embolism, cholesterol embolism, and local complications of endovascular procedures (false aneurysm, arteriovenous fistula). DUS is currently performed after revascularization (1 and 6 months), often annually thereafter, and at recurrence of symptoms. ABI measurement is mandatory but not sufficient. After iliac stenting, native iliac arteries Doppler cut-offs are used. After FP stenting, PSV should be <180 cm/s and SVR <2.5. SVR

Table 12 Elements of a surveillance programme of infrainguinal vein grafts

Risk of occlusion	Stenosis (diameter)	PSV (cm/s)	Velocity ratio (stenotic PSV/prestenotic PSV)	GFV (cm/s)	ABI reduction
Very high risk	>70%	>300	>3.5	<45 (or staccato)	>0.15
High risk	>70%	>300	>3.5	>45	<0.15
Intermediate risk	50–70%	180–300	>2	>45	<0.15
Low risk	<50% or none	<180	<2	>45	<0.15

Modified from Bandyk.[44]

>2 is commonly considered as predictive of >50% stenosis[47] and requires repeated DUS 2–3 months later. To determine ≥80%, combining PSV ≥275 cm/s and SVR ≥3.5 is highly specific and predictive.[48]

Key points

(1) DUS is currently performed 1 and 6 months after endovascular revascularization, often annually thereafter, and at recurrence of symptoms. ABI measurement is mandatory but not sufficient.
(2) Iliac stenting, native iliac arteries Doppler cut-offs are used.
(3) FP stenting, PSV should be <180 cm/s and SVR <2.5. SVR >2 is commonly considered as predictive of >50% stenosis and requires repeated DUS 2–3 months later.

Complications of endovascular procedures

Arteriovenous fistula

DUS allow easy diagnosis showing a continuous turbulent flow from the artery into the adjacent vein. Closure by US-guided compression has been reported, but surgical repair or endovascular treatment with covered stent is usually required.

Femoral false aneurysm

Blood flows through the injured arterial wall and dissects into surrounding tissues to form a haematoma. If a communication between this haematoma and the femoral artery remains patent, a back and forth blood flow may persist creating a femoral false aneurysm (FFA). DUS is the method of choice for diagnosis. Contrast-enhanced CT will only (rarely) be used if US findings are equivocal. Typical US findings include a collection adjacent to the femoral artery (sac) communicating with it through a small channel (neck). A high velocity bi-directional jet through the neck and a swirling flow in the sac are typically seen on colour flow imaging (*Figure 22*).

Some small FFA (<2–3 cm) may spontaneously clot over time, but spontaneous resolution is unpredictable which has led to treat more actively most of them. Simple compression with the US probe may abolish flow through the 'neck' and cause thrombosis of the aneurysm 'sac'. Local analgesia is required because the procedure is painful and involves successive 10 min compressions that are repeated until the sac is thrombosed (up to 60 min). Success rates of 63–88% have been reported.[49]

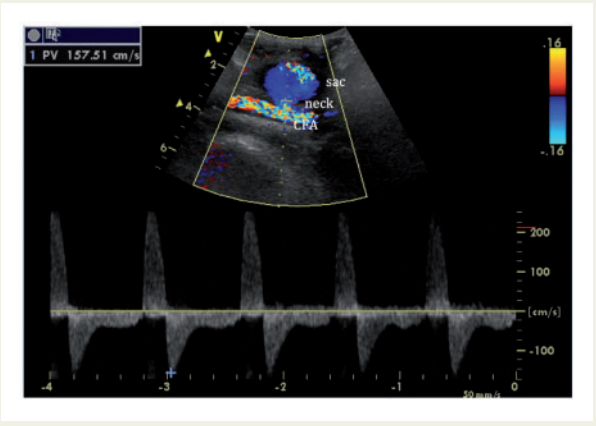

Figure 22 Typical bidirectional flow recorded in the neck of a femoral false aneurysm. CFA, common femoral artery.

Currently US-guided thrombin injection may be recommended for treatment of FFA although studies are relatively small and performed in single-centers.[50]

This technique has a primary success rate ≥95%.[51] Under US control (7 MHz linear array transducer, B-mode, and/or colour Doppler imaging) and aseptic condition, the tip of a 22-gauge spinal needle connected to a 1 mL syringe filled with thrombin (preferably human thrombin 1000 U/mL) is introduced in the sac, as far as possible from the neck. After having checked the position of the needle by aspirating red blood, the thrombin is slowly injected until the thrombus fills the sac. Thrombosis frequently occurs instantly or within a few seconds and the colour flow disturbances disappear both in the sac and the neck. Most FFA will be occluded with 500–1000 U of thrombin but higher doses up to 6000 U were reported. In rare cases, thrombin injection can be immediately complicated by thrombosis of the native artery and venous thrombosis. Delayed reperfusion of the FFA may also occur in up to 6% of cases. Thrombin injection is not recommended for upper arm false aneurysms. Another minimally invasive technique using US-guided para-aneurysmal saline injection has been described.[52,53] It is faster than US-guided compression, and less likely to cause vasovagal reactions. An 18-gauge needle connected to a plastic syringe filled with 0.9% saline solution is introduced and the tip is positioned within 2–5 mm from the 'neck'. Then the saline solution is slowly injected until the resultant tissue swelling completely obliterates the FFA neck. After saline injection, manual compression is

applied for a 5-min period. A randomized study has compared US-guided compression and para-aneurysmal saline injection showing that both techniques are effective with a primary final success rate of 75% and 87.5%, respectively. FFA may also result from previous vascular surgery or trauma. They need a specific treatment that is not addressed in this article.

Key points

(1) DUS is the method of choice for the diagnosis of complications after endovascular procedures (haematoma, FFA, and arteriovenous fistula).
(2) FFA is a collection adjacent to the femoral artery communicating with it through a small channel. A high-velocity bi-directional jet through the neck and a swirling flow in the sac are typically seen on colour flow imaging.
(3) US-guided compression with thrombin injection may be considered as the current 'gold standard' for treatment of FFA. US-guided simple compression with the probe may cause thrombosis of the aneurysm, but it is time-consuming and local analgesia is required.
(4) Thrombin injection is not recommended for upper arm false aneurysms.

Acute aortic dissection

Acute aortic dissections are usually divided according to the Stanford classification in Type A and B. Type A starts in the ascending aorta and Type B in the descending aorta. Another classification, de Bakey's, describes Type I dissection when it involves both ascending and descending aorta, Type II dissection when it includes the ascending aorta and the arch and Type III if it spares the ascending aorta and the arch.

CT, MRI, and transoesophageal echocardiography (TOE) have very high sensitivity and specificity for the diagnosis of acute aortic dissection. The choice of the imaging method is often dependent on local availability, expertise and clinical context. Bedside TOE may be of great interest in the very unstable patient as well as in the post-operative intensive care.

Diagnostic imaging techniques are well described in the 2014 ESC guidelines.[39]

Popliteal entrapment syndrome

It is a rare syndrome caused by congenital abnormal relationship between the PopA and the muscle structures (mostly gastrocnemius or popliteus muscle) in the popliteal fossa. According to Wright classification, there are six distinct anatomical presentations of this syndrome.[54] The most frequent symptom is intermittent claudication, unilateral or sometimes bilateral, in a young athletic patient without CV risk. Intermittent claudication is due to dynamic compression of the PopA during exercise. In long-standing cases, patients may even present with total thrombotic occlusion or aneurysmal degeneration of the PopA and embolism.[55] In 2D echo, the PopA is deviated from its normal course and the PopA and vein are separated by a fibromuscular structure that keeps them apart. Except for patients with popliteal occlusion or stenosis, baseline Doppler examination is normal. Therefore, baseline DUS must be completed by a positional stress test showing popliteal stenosis (increased blood velocity) and disappearance of the pedal pulses with active dynamic compression (plantar flexion against strong resistance).[56] DUS can diagnose popliteal entrapment syndrome and guide CT or MRI but there is a significant false-positive rate. Imaging must correlate to symptoms. Examinations should be performed during the maximal training period.

Key points

(1) It is a rare syndrome caused by congenital abnormal relationship between the PopA and the muscle structures.
(2) The most frequent symptom is intermittent claudication, unilateral or sometimes bilateral, in a young athletic patient without CV risk. Baseline Doppler examination is normal. It must be completed by a positional stress test, performed during the maximal training period.
(3) In long-standing cases, patients may present with total thrombotic occlusion or aneurysmal degeneration of the PopA and embolism.
(4) DUS can diagnose popliteal entrapment syndrome and guide CT or MRI, but there is a significant false-positive rate.

Endofibrosis of the EIA

It is a rare disease exclusively affecting young (usually 20-–25-year-old) endurance athletes (road cycling, marathon, triathlon runners). Endofibrosis can be located in the CFA and CIA but the most common location is the EIA. Kinking (tight bend or curl of the vessel with stenosis) or progressive stenosis of the iliac artery may be associated and reduce blood flow to the lower limb and adversely affect performance. Kinking is sometimes due to extreme vessel length. The disease can be bilateral. DUS is usually normal at rest. In 2D examination (especially during provocative manoeuvres), arterial kinking is a positive diagnostic finding.[57] The predictive value of DUS is low even in trained hands and baseline Doppler must be completed by an exertional test mimicking real life conditions (e.g. running or cycling) and reaching symptoms threshold. As for PopA entrapment testing must be planned during maximal training period. Bilateral ABI are compared at rest and immediately after maximal exercise. Baseline SBP are usually normal. Immediate post-exercise Doppler shows a marked decrease on the involved side. Following maximal exercise, ABI is <0.5 in 85% of patients and is used as a key argument for diagnosis.[58] For other authors, an ABI <0.66 at 1 min after heavy-load exercise is considered to be the optimal cut-off point and a difference of 0.18 between legs is a useful additional diagnostic criterion.[59] Post-stress iliac stenosis also needs to be recorded by Doppler (high-velocity flow) and is usually associated to a systolic murmur. In some cases, 2D echo can visualize homogeneous circumferential wall thickening of the EIA. Baseline CT angiography may look almost normal and should be completed by special manoeuvre (hyperflexion of the thigh).

Provocative exercise tests and DUS can confirm flow limitation before detailed assessment of abnormal anatomy with MRI and digital substraction angiography. MRI with hip flexion has been shown to be the best method to show kinking in the CIA.

These multiple imaging modalities are necessary to identify those most likely to benefit from surgery and to select a patient-adapted surgery technique.[60]

Key points

(1) It is a rare disease exclusively affecting young endurance athletes.
(2) It can be located in the CFA and CIA, but the mostly in the EIA.
(3) DUS is usually normal at rest. It must be completed by an exertional test mimicking real life conditions (e.g. running or cycling) and reaching symptoms threshold.
(4) Arterial kinking during provocative manoeuvres is a positive diagnostic finding.
(5) Testing must be planned during maximal training.
(6) Baseline CT angiography may look almost normal and should be completed by hip flexion.
(7) Multiple imaging modalities are necessary to identify those most likely to benefit from surgery and to select a patient-adapted surgery technique.

Radiation-induced lower limb arteritis

It is a rare complication of radiotherapy with wall thickening of large vessels. It may lead to stenosis and progress to occlusion of the vessel, thrombus formation, ulceration, and distal embolization, several years after radiotherapy. It is often difficult to distinguish radiation arteritis from mere atherosclerosis especially in smoking patients, but localization and confinement to an area previously irradiated, with lack of similar lesions elsewhere, favours radiation as the cause.[61] The dose of radiation reported in the literature to be associated with this unique clinical entity is 20–80 Gy60, and 39.5–80 Gy is specifically mentioned for the iliac and femoral arteries.

Conflict of interest: none declared.

References

1. Aboyans V, Ricco JB, Bartelink ML, Björck M, Brodmann M, Cohnert T *et al.* 2017 ESC guidelines on the diagnosis and treatment of peripheral arterial diseases, in collaboration with the European Society for Vascular Surgery (ESVS): document covering atherosclerotic disease of extracranial carotid and vertebral, mesenteric, renal, upper and lower extremity arteries. Endorsed by: the European Stroke Organization (ESO) the task force for the diagnosis and treatment of peripheral arterial diseases of the European Society of Cardiology (ESC) and of the European Society for Vascular Surgery (ESVS). *Eur Heart J* 2018;**39**:763–816.
2. Piscaglia F, Nolsoe C, Dietrich CF, Cosgrove DO, Gilja OH, Bachmann Nielsen M *et al.* The EFSUMB guidelines and recommendations on the clinical practice of contrast enhanced ultrasound (CEUS): update 2011 on non-hepatic applications. *Ultraschall Med* 2011;**33**:33–59.
3. Brott TG, Halperin JL, Abbara S, Bacharach JM, Barr JD, Bush RL *et al.* 2011 ASA/ACCF/AHA/AANN/AANS/ACR/ASNR/CNS/SAIP/SCAI/SIR/SNIS/SVM/SVS guideline on the management of patients with extracranial carotid and vertebral artery disease. *Stroke* 2011;**42**:e464–540.
4. Dogan S, Duivenvoorden R, Grobbee DE, Kastelein JJ, Shear CL, Evans GW *et al.* Ultrasound protocols to measure carotid intima-media thickness in trials; comparison of reproducibility, rate of progression, and effect of intervention in subjects with familial hypercholesterolemia and subjects with mixed dyslipidemia. *Ann Med* 2010;**42**:447–64.
5. Touboul PJ, Hennerici MG, Meairs S, Adams H, Amarenco P, Bornstein N *et al.* Mannheim carotid intima-media thickness and plaque consensus (2004-2006-2011). An update on behalf of the advisory board of the 3rd, 4th and 5th watching the risk symposia, at the 13th, 15th and 20th European Stroke Conferences, Mannheim, Germany, 2004, Brussels, Belgium, 2006, and Hamburg, Germany, 2011. *Cerebrovasc Dis* 2012;**34**:290–6.
6. Piepoli MF, Hoes AW, Agewall S, Albus C, Brotons C, Catapano AL *et al.* European guidelines on cardiovascular disease prevention in clinical practice: the Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 190 societies and by invited experts) developed with the special contribution of the European Association for Cardiovascular Prevention and Rehabilitation (EACPR). *Eur Heart J* 2016;**37**:2315–81.
7. Tosetto A, Prati P, Baracchini C, Manara R, Rodeghiero F. Age-adjusted reference limits for carotid intima-media thickness as better indicator of vascular risk: population-based estimates from the VITA project. *Thromb Haemost* 2005;**3**:1224–30.
8. North American Symptomatic Carotid Endarterectomy Trial Collaborators, Barnett HJM, Taylor DW, Haynes RB, Sackett DL, Peerless SJ, Ferguson GG *et al.* Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med* 1991;**325**:445–53.
9. MRC European Carotid Surgery Trial: interim results for symptomatic patients with severe (70-99%) or with mild (0-29%) carotid stenosis. European Carotid Surgery Trialists' Collaborative Group. *Lancet* 1991;**337**:1235–43.
10. Grant EG, Benson CB, Moneta GL, Alexandrov AV, Baker JD, Bluth EI *et al.* Carotid artery stenosis: gray-scale and Doppler US diagnosis–Society of Radiologists in Ultrasound Consensus Conference. *Radiology* 2003;**229**:340–6.
11. von Reutern G-M, Goertler M-W, Bornstein NM, Sette MD, Evans DH, Goertler M-W *et al.* Grading carotid stenosis using ultrasonic methods. *Stroke* 2012;**43**:916–21.
12. Comerota AJ, Salles-Cunha SX, Daoud Y, Jones L, Beebe HG. Gender differences in blood velocities across carotid stenoses. *J Vasc Surg* 2004;**40**:939–44.
13. Slovut DP, Romero JM, Hannon KM, Dick J, Jaff MR. Detection of common carotid artery stenosis using duplex ultrasonography: a validation study with computed tomographic angiography. *J Vasc Surg* 2010;**51**:65–70.
14. Romero JM, Lev MH, Chan ST, Connelly MM, Curiel RC, Jackson AE *et al.* US of neurovascular occlusive disease: interpretive pearls and pitfalls. *Radiographics* 2002;**22**:1165–76.
15. Buckenham TM, Wright IA. Ultrasound of the extracranial vertebral artery. *Br J Radiol* 2004;**77**:15–20.
16. Marquardt L, Geraghty OC, Mehta Z, Rothwell PM. Low risk of ipsilateral stroke in patients with asymptomatic carotid stenosis on best medical treatment: a prospective, population-based study. *Stroke* 2010;**41**:e11–7.
17. Geroulakos G, Ramaswami G, Nicolaides A, James K, Labropoulos N, Belcaro G *et al.* Characterization of symptomatic and asymptomatic carotid plaques using high-resolution real-time ultrasonography. *Br J Surg* 1993;**80**:1274–7.
18. 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.
19. Mas JL, Arquizan C, Calvet D, Viguier A, Albucher JF, Piquet P *et al.*; on behalf of the EVA-3S Investigators. Long-term follow-up study of endarterectomy versus angioplasty in patients with symptomatic severe carotid stenosis. *Stroke* 2014;**45**:2750–6.
20. 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.
21. Ball EL, Walsh SR, Tang TY, Gohil R, Clarke JM. Role of ultrasonography in the diagnosis of temporal arteritis. *Br J Surg* 2010;**97**:1765–71.
22. Schmidt WA. Imaging in vasculitis. *Best Pract Res Clin Rheumatol* 2013;**27**:107–18.
23. Schinkel AF, van den Oord SC, van der Steen AF, van Laar JA, Sijbrands EJ. Utility of contrast-enhanced ultrasound for the assessment of the carotid artery wall in patients with Takayasu or giant cell arteritis. *Eur Heart J Cardiovasc Imaging* 2014;**15**:541–6.
24. Debette S, Leys D. Cervical-artery dissections: predisposing factors, diagnosis, and outcome. *Lancet Neurol* 2009;**8**:668–78.
25. Dittrich R, Ritter MA, Ringelstein EB. Ultrasound in spontaneous cervical artery dissection. *Perspect Med* 2012;**1**:250–4.
26. Slovut DP, Olin JW. Fibromuscular dysplasia. *N Engl J Med* 2004;**350**:1862–71.
27. Olin J, Gornik H, Bacharach JM, Biller J, Fine L, Gray B *et al.*; on behalf of the American Heart Association Council on Peripheral Vascular Disease, Council on Clinical Cardiology, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Disease in the Young, Council on Cardiovascular Radiology and Intervention, Council on Epidemiology and Prevention, Council on Functional Genomics and Translational Biology, Council for High Blood Pressure Research, Council on the Kidney in Cardiovascular Disease, and Stroke Council. Fibromuscular dysplasia: state of the science and critical unanswered questions—a scientific statement from the AHA. *Circulation* 2014;**129**:1048–78.

28. Martin JD, Buckley AR, Graeb D, Walman B, Salvian A, Hay JH. Carotid artery stenosis in asymptomatic patients who have received unilateral head-and-neck irradiation. *Int J Radiat Oncol Biol Phys* 2005;**63**:1197–205.
29. Cheng SW, Wu LL, Ting AC, Lau H, Lam LK, Wei WI. Irradiation-induced extracranial carotid stenosis in patients with head and neck malignancies. *Am J Surg* 1999;**178**:323–8.
30. 2011 WRITING GROUP MEMBERS; 2005 WRITING COMMITTEE MEMBERS; ACCF/AHA TASK FORCE MEMBERS. 2011 ACCF/AHA Focused Update of the Guideline for the Management of patients with peripheral artery disease (Updating the 2005 Guideline): a report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. *Circulation* 2011;**124**:2020–45.
31. Schäberle W. *Ultrasonography in Vascular Diagnosis. A Therapy-Oriented Textbook and Atlas*, 2nd ed. Berlin Heidelberg: Springer; 2011.
32. Fernandez-Friera L, Penalvo JL, Fernandez-Ortiz A, Ibanez B, Lopez-Melgar B, Laclaustra M *et al*. Prevalence, vascular distribution, and multiterritorial extent of subclinical atherosclerosis in a middle-aged cohort: the PESA (Progression of Early Subclinical Atherosclerosis) Study. *Circulation* 2015;**131**:2104–13.
33. AbuRahma AF, Bergan J. *Noninvasive Vascular Diagnosis. A Practical Guide to Therapy*, 2nd ed. London: Springer; 2007.
34. Xu D, Zou L, Xing Y, Hou L, Wei Y, Zhang J *et al*. Diagnostic value of ankle-brachial index in peripheral arterial disease: a meta-analysis. *Can J Cardiol* 2013; **29**:492–8.
35. Aboyans V, Criqui MH, Abraham P, Allison MA, Creager MA, Diehm C *et al*. Measurement and interpretation of the ankle-brachial index: a scientific statement from the American Heart Association. *Circulation* 2012;**126**:2890–909.
36. Korno M, Eldrup N, Sillesen H. Comparison of ankle-brachial index measured by an automated oscillometric apparatus with that by standard Doppler technique in vascular patients. *Eur J Vasc Endovasc Surg* 2009;**38**:610.
37. Decrinis M, Doder S, Stark G, Pilger E. A prospective evaluation of sensitivity and specificity of the ankle/brachial index in the follow-up of superficial femoral artery occlusions treated by angioplasty. *Clin Investig* 1994;**72**:592–7.
38. Aboyans V, Lacroix P, Tran MH, Salamagne C, Galinat S, Archambeaud F *et al*. The prognosis of diabetic patients with high ankle-brachial index depends on the coexistence of occlusive peripheral artery disease. *J Vasc Surg* 2011;**53**:984–91.
39. 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.
40. Lindholt JS, Vammen S, Juul S, Henneberg EW, Fasting H. The validity of ultrasonographic scanning as screening method for abdominal aortic aneurysm. *Eur J Vasc Endovasc Surg* 1999;**17**:472–5.
41. Thompson SG, Ashton HA, Gao L, Scott RA. Screening men for abdominal aortic aneurysm: 10 year mortality and cost effectiveness results from the randomised Multicentre Aneurysm Screening Study. *BMJ* 2009;**338**:b2307.
42. Karthikesalingam A, Al-Jundi W, Jackson D, Boyle JR, Beard JD, Holt PJ *et al*. Systematic review and meta-analysis of duplex ultrasonography, contrast-enhanced ultrasonography or computed tomography for surveillance after endovascular aneurysm repair. *Br J Surg* 2012;**99**:1514–23.
43. Thompson MM, Bell PR. ABC of arterial and venous disease. Arterial aneurysms. *BMJ* 2000;**320**:1193–6.
44. Bandyk DF. Surveillance after lower extremity arterial bypass. *Perspect Vasc surg Endovasc Ther* 2007;**19**:376–83.
45. Bandyk DF, Cato RF, Towne JB. A low flow velocity predicts failure of femoropopliteal and femorotibial bypass grafts. *Surgery* 1985;**98**:799–809.
46. Hirsch AT, Haskal ZJ, Hertzer NR, Bakal CW, Creager MA, Halperin JL *et al*. ACC/AHA 2005 practice guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): 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. *Circulation* 2006;**113**:e463–654.
47. Bandyk DF, Chauvapun JP. Duplex ultrasound surveillance can be worthwhile after arterial intervention. *Perspect Vasc Surg Endovasc Ther* 2007;**19**: 354–9.
48. 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.
49. Morgan R, Belli AM. Current treatment methods for postcatheterization pseudoaneurysms. *J Vasc Interv Radiol* 2003;**14**:697–710.
50. Tisi PV, Callam MJ. Treatment for femoral pseudoaneurysms. *Cochrane Database Syst Rev* 2013;**11**:CD004981.
51. Mohler ER, 3rd, Mitchell ME, Carpenter JP, Strandness DE Jr, Jaff MR, Beckman JA *et al*. Therapeutic thrombin injection of pseudoaneurysms: a multicenter experience. *Vasc Med* 2001;**6**:241–4.
52. Gehling G, Ludwig J, Schmidt A, Daniel WG, Werner D. Percutaneous occlusion of femoral artery pseudoaneurysm by para-aneurysmal saline injection. *Catheter Cardiovasc Interv* 2003;**58**:500–4.
53. ElMahdy MF, Kassem HH, Ewis EB, Mahdy SG. Comparison between ultrasound-guided compression and para-aneurysmal saline injection in the treatment of postcatheterization femoral artery pseudoaneurysms. *Am J Cardiol* 2014;**113**: 871–6.
54. Wright LB, Matchett WJ, Cruz CP, James CA, Culp WC, Eidt JF *et al*. Popliteal artery disease: diagnosis and treatment. *Radiographics* 2004;**24**:467–79.
55. Tercan F, Oğuzkurt L, Kizilkiliç O, Yeniocak A, Gülcan O. Popliteal artery entrapment syndrome. *Diagn Interv Radiol* 2005;**11**:222–4.
56. Gourgiotis S, Aggelakas J, Salemis N, Elias C, Georgiou C. Diagnosis and surgical approach of popliteal artery entrapment syndrome: a retrospective study. *Vasc Health Risk Management* 2008;**4**:83–8.
57. Schep G, Bender MH, van de Tempel G, Wijn PF, de Vries WR, Eikelboom BC. Detection and treatment of claudication due to functional iliac obstruction in top endurance athletes: a prospective study. *Lancet* 2002;**359**:466–73.
58. Abraham P, Saumet JL, Chevalier JM. External iliac artery endofibrosis in athletes. *Sports Med* 1997;**24**:221–6.
59. Flors L, Leiva-Salinas C, Bozlar U, Norton PT, Cherry KJ, Housseini AM *et al*. Imaging evaluation of flow limitations in the iliac arteries in endurance athletes: diagnosis and treatment follow-up. *AJR Am J Roentgenol* 2011;**197**: W948–55.
60. Peach G, Schep G, Palfreeman R, Beard JD, Thompson MM, Hinchliffe RJ. Endofibrosis and kinking of the iliac arteries in athletes: a systematic review. *Eur J Vasc Endovasc Surg* 2012;**43**:208–17.
61. Chuang VP. Radiation-induced arteritis. *Semin Roentgenol* 1994;**29**:64–9.