*Chapter 25*

# *Ultrasound Evaluation of Cerebrovascular Obstructive Diseases*

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## Content



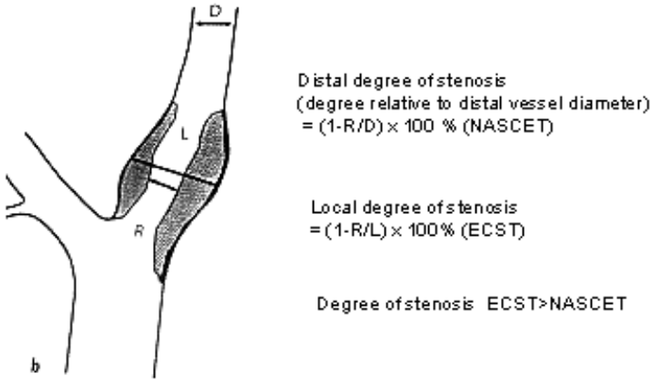




## Carotid arteries

The publication of the results of the European Carotid Surgery Trial (ECST) [1] and the North American Symptomatic Carotid Endarterectomy Trial (NASCET) [2] has highlighted the existence of a close link between the degree of carotid stenosis and the risk of stroke. This highlights the importance of the accurate assessment of the degree of internal carotid artery diameter reduction. The precise characterisation of stenosis severity has been driven by more recent reports, which have indicated that, in patients who have suffered a transient ischaemic attack (TIA) and who have "congruent" carotid disease, the number of strokes prevented by expedite carotid endarterectomy (CEA) or carotid artery stenting (CAS) (within 14 days of onset of symptoms) is significantly greater in patients who have 70–99% stenoses compared with patients who have 50–69% stenoses (3,4). The NASCET and ECST trials did not report similar outcomes [3,4] probably mainly due to the adoption of two different methods for measuring stenosis severity [5]. For example, 70% stenosis calculated according to the NASCET criteria corresponds to an 83% stenosis according to the ECST criteria (Figure 1).

*Figure 1 Grading of internal carotid artery stenosis - NASCET vs ECST criteria*




| | | | | | | |
|---|---|---|---|---|---|---|
| Distal (%) D | 50 | 60 | 37 | 70 | 75 | 35 |
| Local (%) 40 | 70 | 75 | 30 | 82 | 35 | 30 |

## Introduction

The use of different criteria has led to considerable confusion regarding which stenosis should be used in clinical practice. However, the European criteria may be more suited to prognostic and clinical requirements because plaque dimensions can be used to anticipate the occurrence of embolic events more accurately than the stenosis-dependent decrease in cerebral perfusion. The results achieved by the American and European trials have prompted an extensive worldwide effort to identify reliable and universally reproducible criteria to calculate internal carotid artery stenosis by using unenhanced duplex ultrasonography. The method relies mainly on velocity criteria and it has been used to provide national and international society consensus documents [6–8]. To date, standard duplex ultrasound criteria for the grading of internal carotid artery stenosis does not exist. At present, the degree of stenosis in symptomatic patients is usually calculated according to the Moneta's (NASCET) criteria, whereby an internal carotid artery: common carotid artery peak systolic velocity (ICA/CCA PSV) ratio of 4 indicates a 70% stenosis [9].

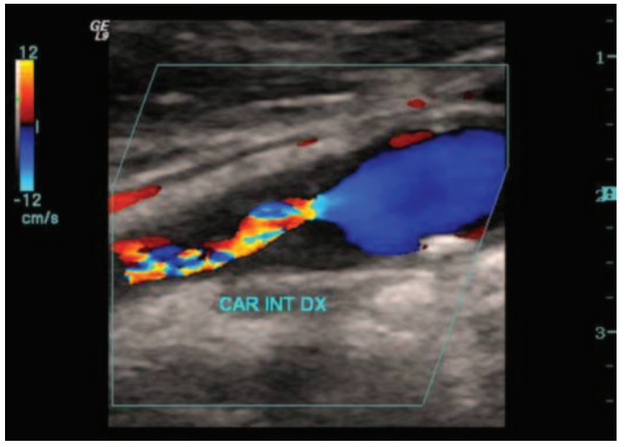
## Indications to ultrasound examination of the carotid arteries

The sonographic screening of severe carotid disease and consecutive preventive strategies, such as carotid endarterectomy, can significantly reduce the incidence of stroke in at risk patients [1,2,7]. However, taking into account the cost, examination time and capacities, ultrasound screening is not appropriate for the general population. A valuable approach involves examining preselected patients using the association between carotid disease and disease predictors, such as neck bruits, peripheral arterial disease and history of stroke or TIA. In most studies risk factors for atherosclerosis such as advanced age, arterial hypertension, diabetes, hyperlipidemia, smoking and adiposity can not be used independently for prognosis of carotid disease in cases where there is substantial of peripheral arterial disease, which is a marker of the presence of a generalized atherosclerotic disease [10].

## Technical and methodological considerations

The ultrasound machines presently available provide an integrated approach to the screening of cerebrovascular obstructive diseases, for both the extra- and intracranial vessels. Ultrasound techniques can accurately depict arterial wall morphology by two-dimensional examination and they can simultaneously define cerebrovascular haemodynamics by Duplex. Standard protocols should be used to ensure the reproducibility of the information collected. The examination of extracranial vessels requires greyscale examination, and the colour coded and/or power imaging should be performed with high frequency (>7.5 MHz) linear transducers. However, under particular conditions such as short or "hostile" necks, to investigate the very proximal or very distal carotid segments, for the examination of the vertebral arteries or in the presence of calcified arteries the use of lower frequency transducers (3–5Mhz) is recommended. The insonation angle should be ≤60%; the sample volume should be placed at the site of greater stenosis, which could be in the centre of the highest colour velocity jet (Figure 2).

*Figure 2 Two-dimensional and colour flow representation of a stenosis of the internal carotid artery. The maximum lumen reduction and the colour velocity jet can be easily recognised and they can be used to place the sample volume of the spectral Doppler for velocity measurements.*



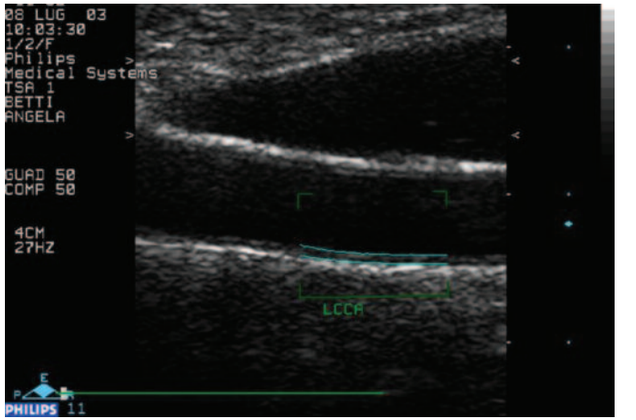





## Arterial wall characterization: defining the thickness of the intima-media complex

Ultrasound techniques allow one to define the binary echographic pattern, which corresponds to the sum of the intimal (endothelial) and medial layers of the carotid arterial wall. The intima-media complex (IMT) measurement is the only imaging parameter for cardiovascular risk assessment presently included by the AHA. Several studies have provided evidence of its value as an independent predictive factor of cardiovascular disease [11, 12]. Several different protocols have been proposed, although there is not a universal standard protocol owing to side differences and because measurements can vary along the proximal or distal segments of the same artery, and are operator dependent. It has been recommended that the IMT measurement in the distal and far wall of the common carotid artery should be performed 1cm from the carotid bulb [13]. All measurements should be performed only if the thickness of the arterial walls is homogeneous and devoid of focal atherosclerotic disease. The normal IMT value in the general population is ≤0.9mm and values can vary according to age, gender and race [14]. The evaluation can be done freehand and based of the average value of at least three contiguous measurements. A partially automatic estimate can also be performed through dedicated software, which can provide a measurement derived from the average of 10 measurements in a 1cm segment. The use of these systems allows a less operator-dependent evaluation (Figure 3).

*Figure 3 Partially automatic IMT measurement: a computer assisted "edge" tracking software allows an operator independent estimate of the average thickness of the IMT complex along a 1cm segment of the far wall of the distal common carotid artery.*




## The atherosclerotic plaque

The atherosclerotic plaque can be detected by 2D ultrasound examination as a ≥1.5mm focal thickening of the arterial wall, which encroaches the vessel lumen or a focal thickening exceeding 50% of the thickness of the closest unaffected arterial segment [13]. Morphological plaque characterisation can be performed subjectively, but the accuracy and reproducibility is very operator dependent. In addition, some studies have found poor correlation or no correlation at all between sonographical and histological findings [15–18]. However, it has been reported [19] that the as-

sociation of ultrasound plaque features with symptoms, when assessed retrospectively, is generally more reliable than the association between histology and ultrasound findings. Some more reproducible techniques have recently been introduced i.e. videodensitometry and radiofrequency analysis. In the ICAROS study, for example, a greyscale median analysis of plaques measured by computer-assisted techniques has proved to be effective in the diagnosis of echo-lucent vulnerable plaques in patients undergoing carotid stenting [20]. Computerised texture analysis of ultrasound images by intravascular ultrasound techniques of symptomatic carotid plaques can identify those that are associated with brain infarction [21]. Unfortunately these methods are not widely available within ultrasound instruments in the majority of the vascular laboratories. Excellent results have been obtained with visual plaque assessment with adequate attention to imaging details [22]. De Bray et al [23] suggested satisfactory and fairly reproducible results in describing plaque echogenicity can be achieved using the echo levels of the following structures as reference values: a hypoechoic plaque corresponds to the echogenicity of blood flow, an intermediate echogenicity to corresponds to the sternocleidomastoid muscle, and an hyperechoic plaque corresponds to that of bony structures. The "eye-ball" morphological characterisation by ultrasound techniques allows one to distinguish between different plaques according to echogenicity, structure homogeneity (homogeneous vs etereogenous), surface (regular, irregular or ulcerated plaques). Adhering to these criteria, five different classes of atherosclerotic plaques have been proposed by Gray-Weale et al [24] and by Geroulakos et al [25]. Table 1 shows the risk level associated with each plaque category.

2D examination of the plaque surface shows it can be very irregular with scallops; finding of an excavation of 2mm is considered consistent with an ulcerated plaque.

To avoid misinterpretations owing to serial plaques, the presence of an ulcer should always by confirmed in both the longitudinal and transverse scanning planes.

*Table 1 Carotid plaque classification according to Gray-Weale and the relative grade of risk.*

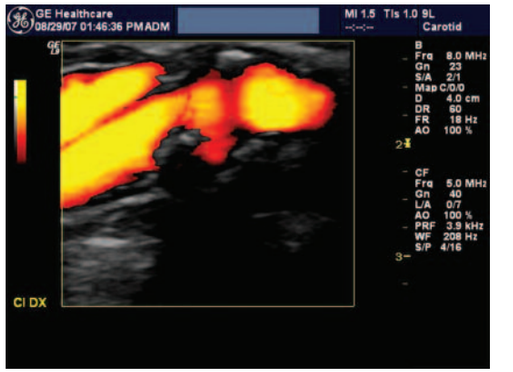
| CAROTID PLAQUE TYPES | RISK OF SYMPTOMS |
|---|---|
| Type 1: Uniformly hypo-echogenic (echolucent) | High |
| Type 2: Predominantly hypo-echogenic (echolucent) ( >50% of plaque structure) | High |
| Type 3: predominantly echogenic (>50% of plaque structure) | Lower |
| Type 4: Uniformly echogenic | Lowest |
| Type 5 : Unclassified due to calcification or poor visualization | Unknown |

The use of power-angio and/or colour flow imaging can help to achieve a better vessel/lumen separation because of the sharper definition of the plaque surface boundaries (Figure 4).



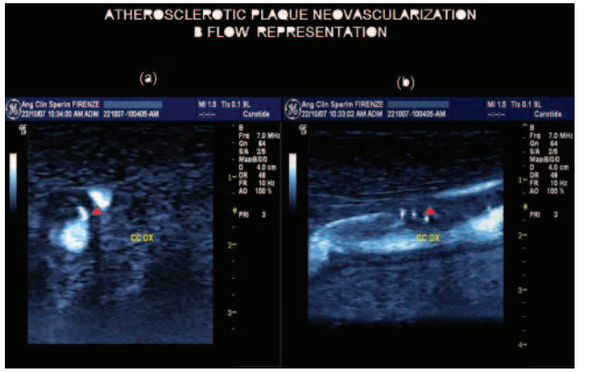


*Figure 4 Power-angio imaging sharply depicts a deep surface irregularity most probably due to an ulcerated lesion.*



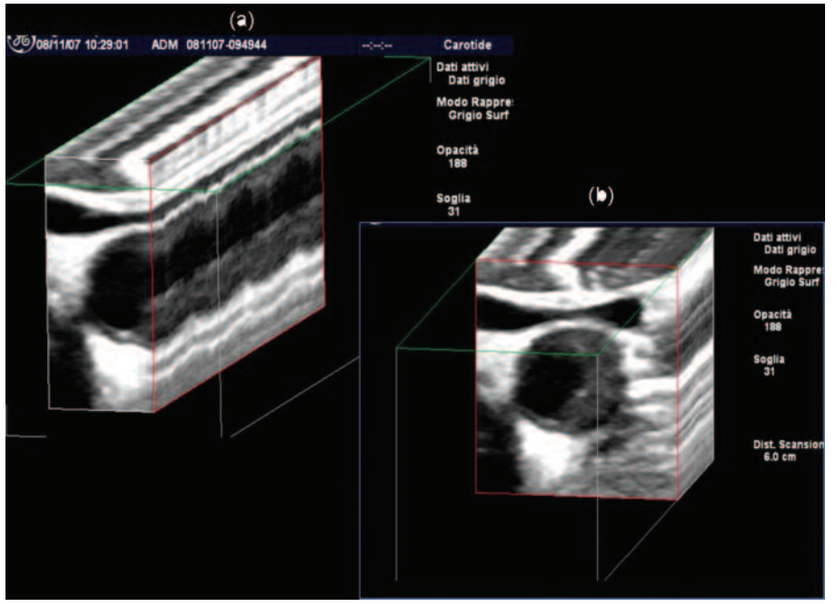

Additional parameters of plaque instability have been described, particularly atherosclerotic plaque neovascularization. It has been demonstrated that the plaque neovessels can be effectively detected using ultrasound techniques (CFI and power angio). Preliminary reports in small study groups have found a positive correlation between sonographic and histological findings [26]. In addition, the presence and number of neovessels were correlated with embolic activity, and the histological findings suggested inflammation [27]. Neovascularisation is considered to be a reliable index of plaque activity, therefore, subsequent studies have been designed to detect and quantify neoangiogenesis. Significant progress in this field was made after the introduction of "B flow" techniques (Figure 5) and above all by the adoption of echo-contrast agents [28]. A recent retrospective study on 147 stroke patients who underwent contrast-enhanced carotid ultrasound has reported that the presence and degree of adventitial vasa vasorum and plaque neovascularisation were directly associated with cardiovascular deaths and cardiovascular events [28]. These results seem very promising and may provide a non-invasive adjunctive "window" to identify vulnerable plaques, but the method still needs to be standardised and the data need to be confirmed with further investigations.

*Figure 5 Transverse (a) and longitudinal (b) views of the carotid bulb at B flow imaging. Red arrows indicate multiple sites of neovascularization.*



A further development in defining the atherosclerotic burden of the entire carotid artery is three-dimensional ultrasound evaluation (Figure 6). Despite the potential benefit from its application, the technique has not yet been introduced in the majority of ultrasound machines.

*Figure 6 3D imaging of the common carotid artery. The longitudinal and cross sectional views can accurately define the global atherosclerotic burden of the vessel.*




## Grading the carotid stenosis

Atherosclerotic plaque can be associated with vessel lumen reduction, which causes a significant haemodynamic effect. Estimation of the degree of stenosis by direct measurement of the minimum viable lumen diameter or through planimetric evaluation is accepted as accurate for all non-haemodynamic (<50% degree) stenosis. However, in the presence of haemodynamic (≥50%) stenosis, Doppler velocity estimates are more accurate and are considered the method of choice for the quantification of the stenosis. A meta-analysis of 41 studies of 4876 carotids in 2541 patients has demonstrated that Doppler velocity measurements and neuroradiological measurements gave similar results for carotid stenosis, ranging from 70–99% reductions in diameter [29]. Ultrasound characterisation of the degree of carotid stenosis is therefore feasible, effective and accurate, and is required to select the most appropriate therapeutic strategy.

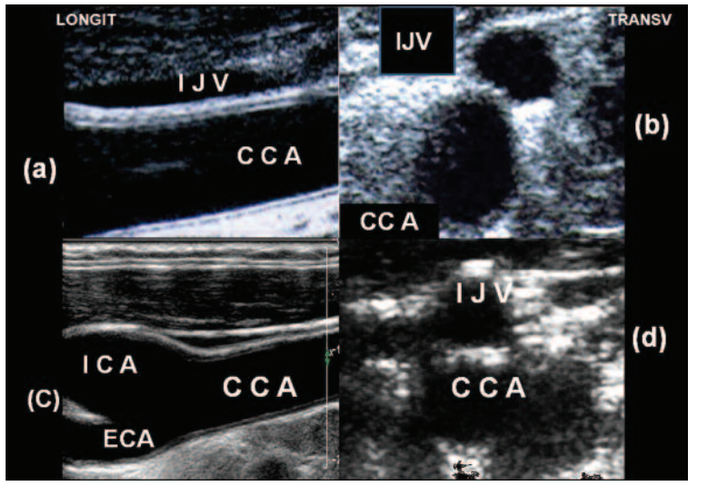
## Methods

Colour-coded imaging and conventional (spectral) Doppler allow the haemodynamic evaluation of the carotid and vertebral arteries. 2D ultrasound must be routinely performed on both transverse and longitudinal planes by applying the transducer posteriorly or anteriorly along the sternocleidomastoideal muscle (Figure 7).



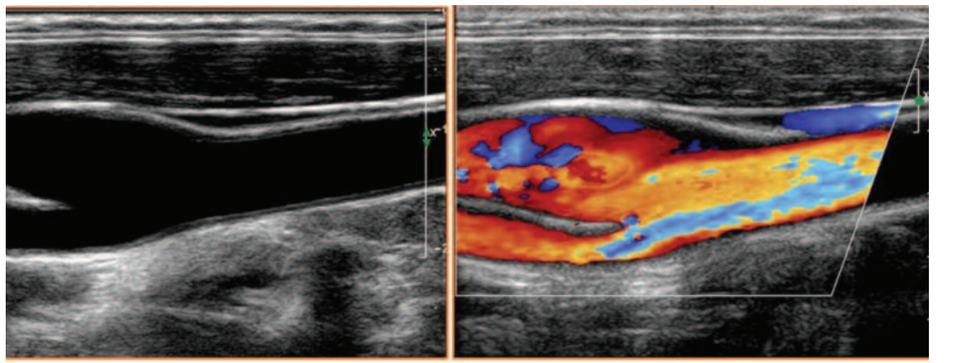


*Figure 7 2D longitudinal and transverse views of the middle part of the common carotid artery (a,b) and carotid bifurcation (c,d).*



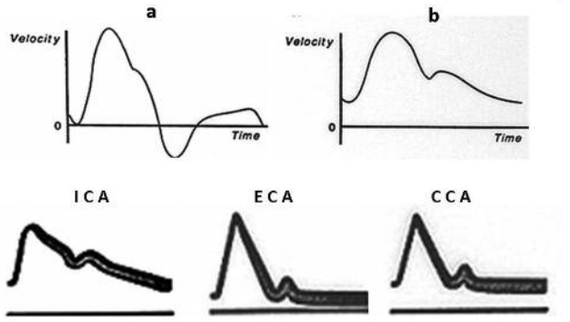

The 2D echo, combined with colour flow or power-angio imaging, provides a roadmap for accurate spectral Doppler haemodynamic testing of specific sites of interest on the longitudinal sections (Figure 8).

*Figure 8 Characteristic 2D (a) and colour flow appearance (b) of the common carotid artery up to its bifurcation on the longitudinal plane.*



Spectral Doppler examination can than be applied to the different vascular segments on the longitudinal plane. Each artery exhibits a distinctive flow-velocity profile: the vessels supplying the brain (internal carotid artery and vertebral arteries) show a monophasic, continuous low-resistance profile; the external carotid arteries are more pulsatile high resistance vessels; their profile is usually polyphasic with an early diastolic notch or an early reverse wave; the common carotid artery profile is usually intermediate because it reflects a composite (high and low resistance) outflow pattern (Figure 9).

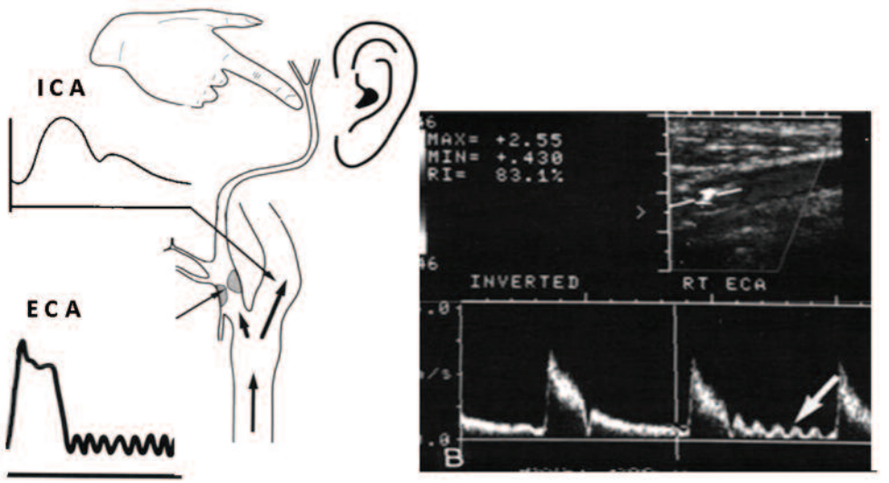
*Figure 9 Schematic representation of a high resistance profile as the one observed in muscular arteries (a) and a low resistance arterial profile (b). The lower panels show the typical velocity profiles of the internal, external and common carotid arteries.*




Importantly, tapping on the superficial temporal artery in front of the ear causes notches in the external carotid artery Doppler spectrum, but it does not affect the internal carotid artery spectrum (Figure 10).

Because temporal tapping is reliable in a small minority of patients [30], ECA should also always be identified to adhere to additional criteria, such as smaller size and proximal branching.

*Figure 10 Typical saw-tooth appearance of the velocity profile of the external carotid artery during the so called "tapping" manoeuvre. No signal modulation can be detected in the internal carotid artery during oscillating temporal compression.*

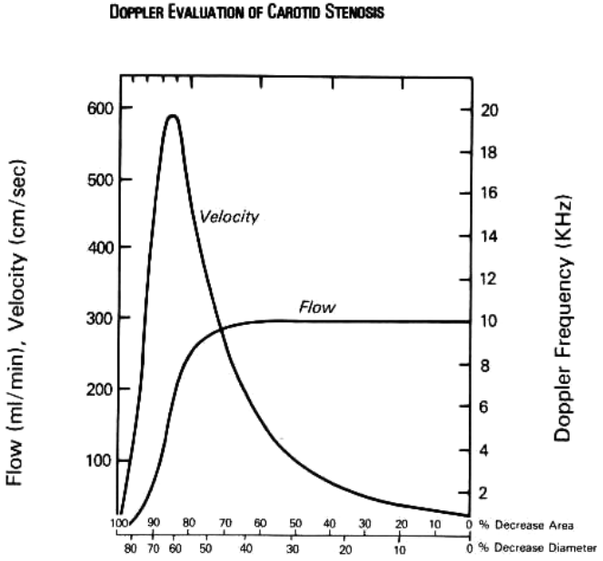



The main criterion for the diagnosis of carotid stenosis is the demonstration of a focal acceleration of flow at the level of the lumen reduction. Colour-flow imaging can detect a focal area of turbulent flow that exhibits a colour mosaic-like appearance at the site of maximum acceleration of flow within the stenosis (Figure 2). In association with the 2D echo ultrasound, CFI is a convenient tool for optimal positioning of sample volume spectral Doppler analysis but it does not provide a reliable index of stenosis severity. The first demonstration of a direct and positive relationship between focal flow velocity acceleration and the degree of diameter reduction of the internal carotid artery was originally shown by Spencer [31]) (Figure 11).





*Figure 11 Flow velocity and volume flow rate in the carotid artery at increasing values of diameter reductions. According to the continuity law, intrastenotic flow acceleration does not occur until there is greater than 50% diameter reduction or a 75% reduction in cross-sectional area.*




The increase in velocity is proportional to the severity of the stenosis and it is highest in 80–99% stenosis. In conditions near to occlusion (>99% stenosis), the high friction and the elevated resistance to flow, which occurs at this degree of diameter reduction leads to a drop in flow velocity close to zero. To diagnose an haemodynamic stenosis, the peak systolic velocity should exceed a critical cut-off value after positioning the sample volume at the site of maximum lumen reduction, which should coincide with the highest focal velocity acceleration jet at colour flow imaging (Figure 2). A haemodynamically significant (>50%) stenosis is assumed at peak systolic flow velocities of >125cm/s and intermediate to high-grade stenosis (70–79%) at 180–250cm/s. The sensitivity and specificity of these velocity criteria are 90–96% and 86–93%, respectively [32–34]. A parallel angiographic evaluation has shown a good correlation between mean PSV and percentage of stenosis when measured arteriographically [35]. A low (<50%) stenosis can be diagnosed when PSV is less than 125cm/s. In the presence of low-grade stenosis, disease severity can be quantified more accurately than velocity criteria through the direct measurement of diameter reduction on 2D greyscale examination. This can be done by comparing the diameter of the vessel lumen at the site with maximum stenosis with the one of the normal ICA downstream of the obstructive lesion, according to the NASCET criteria [2]. Different systemic conditions, such as hypertension, can autonomously affect the peak velocities along the whole carotid system. The

reduction in wall elasticity may lead to an increase in arterial pulsatility and elevated peak systolic velocity throughout the entire circulation, including the carotid system. In the presence of focal acceleration, the use of the internal carotid artery: common carotid artery velocity ratio (Vmax ICA:Vmax CCA) can effectively diagnose and classify carotid stenosis severity and rule out diffuse non-specific velocity increases. The ICA/CCA ratio is independent from general blood flow alterations. The ratio varies between 1.5 and 2 in cases of 50–70% stenosis, between 2 and 4 in cases of 70–89% stenosis and is above >90% in cases of >90% stenosis (33,36,37). Moneta et al [9] found that for an ICA:CCA PSV ratio of >4, there is a sensitivity of 0.91 and a specificity of 0.87 for a >70% stenosis according to the NASCET method. Grant et al [33] found in their series that only 80% of all stenoses were correctly classified as >70% by an ICA:CCA PSV ratio >4. A very tight stenosis causes PSV to fall (Figure 11 and Table 2). When both spectral Doppler and colour flow examination fail to detect any signal at the site of arterial examination a total occlusion should be diagnosed. However, the distinction between near occlusion and total occlusion cannot always be easily assessed by ultrasound examination alone; if trickle flow is not detected, stenosis may be incorrectly diagnosed as an occlusion. Low velocity can be detected with colour Doppler as a narrow channel in the ICA corresponding to the "string sign" on arteriography and can be confirmed by pulsed Doppler sampling. Power Doppler or contrast imaging can help to detect slow flow. Apparent occlusion warrants confirmation by angio CT.

*Table 2 Consensus panel greyscale and Doppler ultrasound criteria for diagnosis of ICA stenosis guidelines SRU radiology 2003;229:340-46*

**Carotid Artery Stenosis: Gray-Scale and Doppler US Diagnosis - Society of Radiologists in Ultrasound Consensus Conference**

Consensus Panel Gray-Scale and Doppler US Criteria for Diagnosis of ICA Stenosis

| | Primary Parameters | | Additional Parameters | |
|---|---|---|---|---|
| Degree of Stenosis (%) | ICA PSV (cm/sec) | Diameter Reduction (%) 2D Plaque Estimate* | ICA/CCA PSV Ratio | ICA EDV (cm/sec) |
| Normal | < 125 | None | < 2.0 | < 40 |
| < 50 | < 125 | < 50 | < 2.0 | < 40 |
| 50 – 69 | 125 – 230 | ≥ 50 | 2.0 – 4.0 | 40 – 100 |
| ≥ 70 but less than near occlusion | > 230 | ≥ 50 | > 4.0 | > 100 |
| Near occlusion | High, low or undetectable | Visible | Variable | Variable |
| Total occlusion | Undetectable | Visible, no detectable lumen | Not applicable | Not applicable |

* Plaque estimate ( diameter reduction) with gray-scale and Color Doppler US.

The velocity criteria adopted in different vascular laboratories and that reported in the literature are often different. Therefore, every vascular laboratory should apply its own velocity criteria but only if they have developed and validated it through parallel angiographic evaluation in a large study. In all other conditions, as recommended by the consensus Conference of the Society of Radiologists in Ultrasound [38], or more recently by a joint consensus statement of the American Society of Neuroimaging and the society of Vascular and Interventional Neurology (39), each laboratory should comply with the velocity parameters that have been discussed and shared by the components of these consensus panels (Table 2).

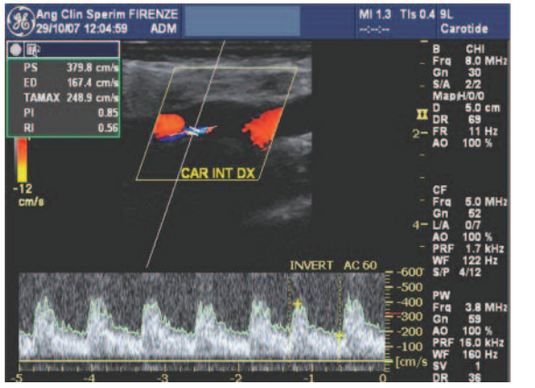




As well as PSV values, diastolic velocities can be very useful in grading stenosis severity. End diastolic velocity (EDV) only significantly rises in high grade stenosis (diameter reduction of >60%), therefore, EDV is a good marker for high grade stenosis (Table 2). The characteristics of the arterial flow upstream and downstream to the stenotic vessel can reflect the severity of the obstructive disease (19). The total occlusion of the distal (intracranial) internal carotid artery may not be directly visible by colour flow imaging at the cervical level but it invariably leads to an increased resistive index in the proximal part of the homolateral internal carotid artery and in the common carotid artery. In the severe haemodynamic stenosis (80–95%) the pressure drop caused by the lesion can lead to a decrease in the downstream peak velocity and to a delay in the increase of the velocity curve [40]. However, these changes can be variable according to the pressure characteristics of the downstream vascular bed and to the individual extent of the collateral blood flow.

A typical example of severe stenosis according with the SRU criteria is given in Figure 12.

*Figure 12 Spectral Doppler sampling at the level of maximum lumen reduction and in the middle of a focal turbulence (colour flow): the high peak systolic and diastolic velocity are consistent with a high grade ICA stenosis.*




## Vertebral arteries

The vertebrobasilar system is responsible for 20–30% of intracranial blood flow. Although ischaemic stroke in the vertebrobasilar system is less common than in the carotid system, abnormal Doppler and duplex examination are at least as frequent in the vertebrobasilar system as in the carotid system [41]. Dizziness, ataxia and drop attacks are the most common signs and symptoms of a defect in the posterior circulation. However, the clinical picture can be extremely variable and the potential contribution of the vertebrobasilar artery occlusive disease to vertebrobasilar insufficiency can be difficult to evaluate on clinical grounds alone [42].

Indeed, while neurological defects due to a lesion in the carotid system cause highly specific hemispheric symptoms, the clinical effects of a vertebrobasilar insufficiency are much less specific.

Vertebral or basilar artery disease, extrinsic vertebral compression in the spinal canal or subclavian or innominate steal syndrome can typically cause visual disturbances and diplopia, vertigo, parasthesae, impaired coordination or drop attacks. Extrinsic vertebral artery compression usually causes transient, stereotypical symptoms. Although the obstruction of a larger vessel (i.e. vertebral arteries or basilar artery) more commonly leads to a more specific clinical syndrome, when ischaemic events involve only limited sections of the vertebrobasilar compartments, the clinical syndromes can be extremely variable according to the vascular territory.

Although typical vertebrobasilar syndrome includes dizziness in association with other specific signs or symptoms (i.e. ataxia, impaired coordination, visual disturbances and diplopia), it has also been reported, that up to 62% of patients with vertebrobasilar insufficiency exhibit at least one episode of isolated vertigo in the natural history of the disease [43]. Isolated vertigo as a sign of vertebrobasilar insufficiency has been observed in studies [44]. In addition, dizziness can also be found in other conditions for example gastroenteritis or myocardial infarction. In cases of acute vertigo, differentiation between peripheral and central vestibular causes can be difficult. Apart from atherosclerotic lesions, acute symptoms of vertebrobasilar insufficiency can be due to dissection, which typically occurs after trauma. For differential diagnosis, an interdisciplinary approach is needed because the vestibular, neurological and psychiatric disorders can have a common role in the development of symptoms and further overlapping can occur. There are no strict and specific indications to the systematic ultrasound evaluation of the vertebrobasilar system, but it may be indicated in the presence of clinical suspicion of recurrent ischaemia in the vertebrobasilar artery territory, and in patients with significant difference in arterial blood pressure values between the upper arms.

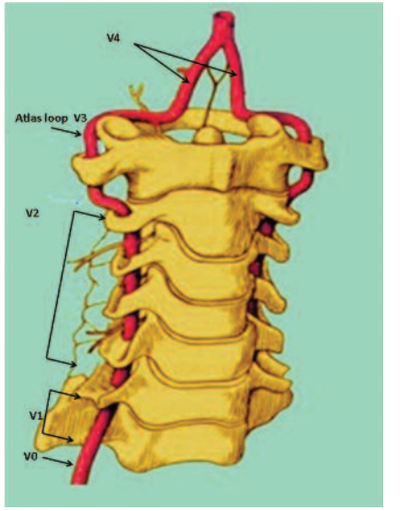
## Anatomy

The two vertebral arteries originate from the ipsilateral subclavian arteries at the level of the sixth cervical vertebra (V0). In 4% of all patients, the left vertebral artery originates directly from the aortic arch [41,45]. The pre-vertebral section of the vertebral artery (V1) is defined as reaching from the subclavian artery to where it enters the spine through the costotransversal foramina at the sixth vertebrae. Both vertebral arteries proceed upwards along the posterior aspect of the neck, where their course is partly intra-osseous within the foramina transversaria of the corresponding vertebrae (V2). The vertebral arteries course superiorly and wind around the atlas, at the side of the medulla oblongata and at the level of the atlanto-occipital inter-space. This segment is, therefore, also referred to as atlas loop (V3). Muscular branches arising from the V3 segment form anastomoses with the occipital artery from the external carotid artery [41]. Finally, the vertebral arteries proceed cephalad and anteriorly until they reach the pontomedullary level, where they join and form the basilar artery (V4) (Figure 13).

*Figure 13 Anatomical representation of the different segments of the vertebral artery:*
*V0 - at the origin from the subclavian artery*
*V1 - from the origin to the site of entry into the transverse foramen of the 6th cervical vertebra*
*V2 - the cervical intraforaminal segment*
*V3 - the upper segment winding around the atlas (atlas loop)*
*V4 - the terminal intracranial segment joining the contralateral vertebral artery to originate the basilar artery*

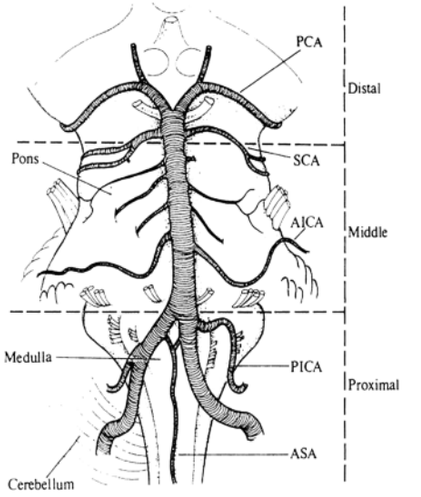







Branches of the basilar artery supply the entire pons, and the superior and anterior aspects of the cerebellum. Branches of the vertebral arteries supply the medulla and the interior surface of the cerebellum (Figure 14) [46,47].

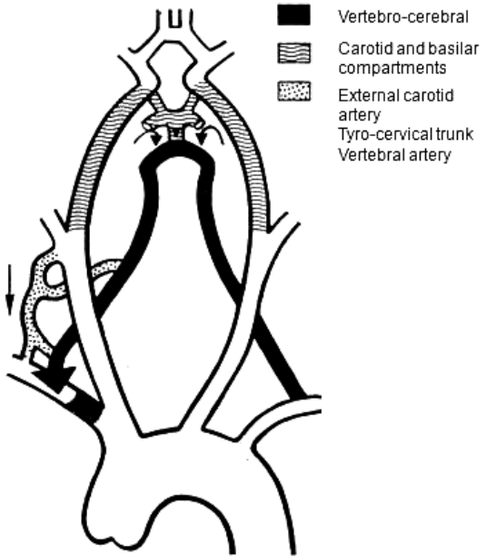
*Figure 14 Arterial branches originating from the V4 segments of vertebral artery give rise to branches that supply the entire pons and the superior and anterior aspects of the cerebellum.*




The lack of symptoms, even in the presence of severe stenosis or occlusion of a subclavian or proximal vertebral artery is often due to the presence and efficiency of a thyrocervical collateral vessel arising from the ipsilateral external carotid artery (Figure 15).

In a small number of patients one of the vertebral arteries can end in the posterior inferior cerebellar artery instead of joining the contralateral vertebral artery. In a few patients one vertebral artery can supply a muscular compartment of the neck. The finding of a high resistance pattern at spectral Doppler examination can be misleading because it may be interpreted as the consequence of a distal obstruction rather than a benign anatomical variant.

*Figure 15 Schematic representation of a thyro-cervical collateral artery feed by the external carotid artery in the presence of an obstruction of the ipsilateral subclavian artery or of the proximal vertebral artery.*




## Methodological aspects

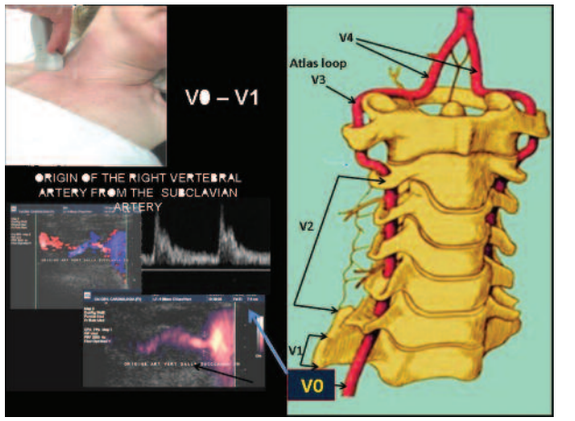
### Proximal segments (V0-V1)

The V0 and V1 segments of the vertebral artery course behind and underneath the clavicula and their visualisation is not always easy. However, the examination of these proximal segments should not be over sought especially in the presence of abnormal V2 velocity patterns. Indeed, the pre-vertebral segments are the preferred site of stenosis. Virtually all stenoses of the vertebral artery occur at its origin [46,48]. The origin of the vertebral arteries, and their proximal intrathoracic segment (V0 and V1), can be seen by placing the probe at the level of the supraclavicular fossa (Figure 16).





*Figure 16 The origin of the vertebral artery (V0) from the subclavian artery can be studied with a supraclavicular approach. The power angio imaging shows the proximal part of the left subclavian giving rise to the vertebral artery.*




The assessment of the artery, especially at its origin, can be often difficult due to calibre variation in the presence of congenital hypoplasia. Finally, the origin and proximal segment of the vertebral artery may be confused with other large branches arising from the proximal subclavian artery, such as the thyrocervical trunk. Holding the probe on the supraclvicular fossa, an oscillating compression of the V3 segment (between the tip of the mastoid and the transverse process of the atlas) allows the distinction between the origin of vertebral artery and other contiguous vessels. The compression leads to an unmistakable signal modulation of the V0 vertebral waveform and assumes a typical saw-tooth appearance. No variation can be detected in the contiguous arterial vessels. The detection of a turbulent flow at V0 level is indicative of significant stenosis in the presence of pronounced focal lesion, which increases the velocity value compared with those found in more distal segments (>50% difference). A high-grade stenosis is identified on the basis of a marked increase in peak systolic velocity (≥150 cm/s) [48].

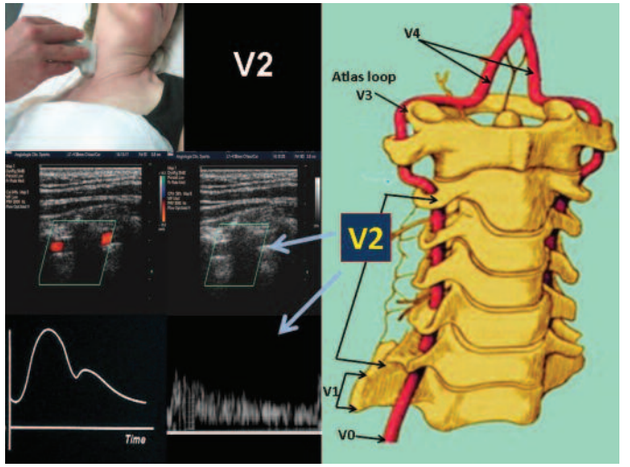
### The V2 segment

A more reliable approach to assessment of the vertebral arteries is to begin the examination by scanning the vessel near its mid-segment (region V2). This segment is more easily seen, its course is typically straight and it does not usually exhibit any tapering or diameter changes. In addition, it is rarely affected by atherosclerotic lesions. It is possible to obtain adequate imaging and quantitative spectral Doppler velocity data from a portion of the mid-segment of the extracranial vertebral arteries in more than 98% of patients and vessels [47]. The examination is easily accomplished by first obtaining a good longitudinal view of the common carotid artery at the approximate level of the third to fifth cervical vertebrae. Once this image has been obtained, angling the probe laterally and inferiorly will bring the vertebral artery into view (Figure 17). Vertebral bodies can be easily recognised as hyperechoic transverse bars. Colour Doppler imaging identifies the artery flow by the pulsatile pattern of colour flow interrupted by a series of anechoic bands due to the shadowing effect of the transverse processes of the cervical spine.

## Normal findings

The normal diameter of the vertebral artery is 3–5mm, but in the presence of an hypolapstic vessel the contralateral one may exhibit a compensatory hyperplasia, thus resulting in a more than 2mm difference between the diameter of the two sides.

*Figure 17 The figure shows how to scan cervical region to explore the V2 segment of the vertebral artery:*
*- Apply the transducer longitudinally anteriorly, along the steronocleidomastoideal muscle*
*- Angle the probe parallel to the carotid artery, laterally and inferiorly*
*- Look for vertebral bodies: hyperechoic transverse bars*
*- The flow velocity profile in the normal subject has a typically low resistance pattern*




Almost 50% of patients have a dominant vertebral artery with a higher flow than the contralateral one. The smaller non-dominant artery often shows a flow pattern of increased vascular resistance, and a decrease in both peak and diastolic velocity [45].

In cases of lateral differences in flow velocity and poor visualisation of the origin, an unchanged waveform, possibly with a reduced diastolic flow helps to diagnose hypoplasia and to rule out stenosis [48]. The peak systolic velocity in the vertebral artery can be very variable; quantitatively it should exhibit a scaled-down version of flow in the internal carotid artery, ranging from 20–60 cm/s whereas end-diastolic velocity values ranges between 5–30 cm/s. [48]. The resistive index varies from 0.62–0.75 [49]. The velocity profile shows a typical low-resistance, continuous and monophasic pattern (Figure 17). The occurrence of a stenotic lesion of the V2 segment is rare and when present the same criteria of V0 segment are applied. Most turbulence in this vertebral section is usually in association with a tortuous vessel. In addition, given the wide range of normal velocities in healthy individuals, no real cut-off velocity has been defined to discriminate between low-grade and haemodynamically significant stenosis, as seen in the carotid. Rarely, high-velocity turbulent flow patterns can be detected in the mid-segment of a vertebral artery because of extrinsic compression from the bone spine (often associated with changes in head or neck position).



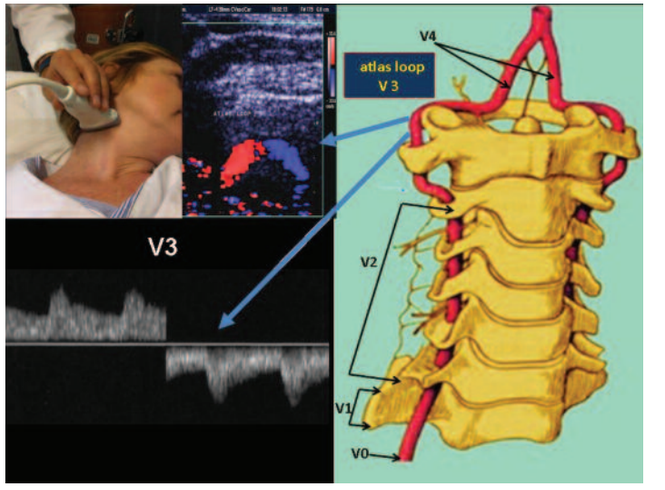


However, the examination of the velocity profile of the V2 segment is very important because the finding of a dampened and slow rising velocity curve at this level suggests the presence of an haemodynamic stenosis within the more proximal segments (V0 or V1). Doppler interrogation of the V0–V1 segment in this case must be performed carefully.

## V3 segment (atlas loop)

To examine this segment the patient is asked to turn his or her head and the transducer is placed in a transverse position on the posterior aspect of the neck under the mastoid prominence (Figure 18). Insonating rates of 76% on the right and 86% on the left have been reported [49]. 2D examination alone usually fails to detect the artery. Colour mode appearance of the V3 segment is typically comma-shaped and it is easily seen (Figure 19). The rounded course of the vessel does not allow an appropriate angle correction for velocity measurements. No systematic values have been reported; therefore, the velocity data collected in the V2 segment should be used as reference values [50].

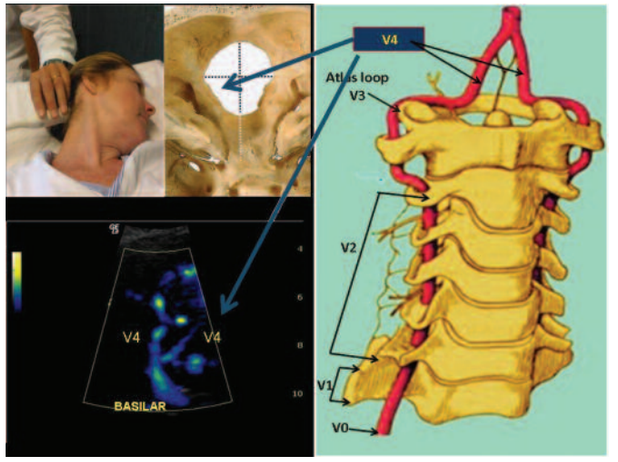
*Figure 18 Typical "comma"- like appearance of the V3 segment at colour Doppler examination with characteristic bi-directional profile of spectral Doppler.*




## V4 (intracranial) segment

The confluence of the distal part of the vertebral arteries is the origin of the basilar artery can only be examined by transcranial colour-coded sonography (TCCS) using a low frequency (2.5–3.5 MHz) sector probe. Through the posterior suboccipital approach, the vertebral artery and the origin of the basilar artery are examined by placing the probe over the midline in a skin impression between the occipital bone and the atlas. Having the patient in a supine position and the head rotated laterally by 30–45° [41]. The foramen magnum and the hyperechoic clivus serve as the anatomical landmarks, with both vertebral arteries located at their edges. The direction of the sector beam is tilted upwards from the root of the nose to the frontal prominence to enable the visualisation of the typical colour Doppler Y-shaped image of V4 segments at their confluence in the basilar artery (Figure 19).

*Figure 19 Typical power imaging Y-shaped appearance of the distal segments of the vertebral arteries (V4) where they join to originate the basilar artery. The depth of insonation of the vertebral-basilar junction usually ranges between 70 and 95mm.*




In the healthy patients all segments are seen in blue because their flow is directed away from the transducer. The origin of the basilar artery can be visually identified at a 65–70 mm depth; the depth of its superior intracranial end ranges from 95–125 mm. Very rarely the vertebral artery can end in the postero-inferior cerebellar artery (PICA) instead of joining the contralateral vertebral artery.

## Common distribution of the obstructive lesions of the vertebral arteries

Stenoses and occlusions of the vertebral arteries are mostly found at the origin (V0) or the intracranial segment (V4) [46,48]. Dissections appear with declining frequency in V3, V2, V1 [41]. The methodological aspects and the diagnostic criteria to identify the obstructive lesions of the V0 to V2 tract have already been described.

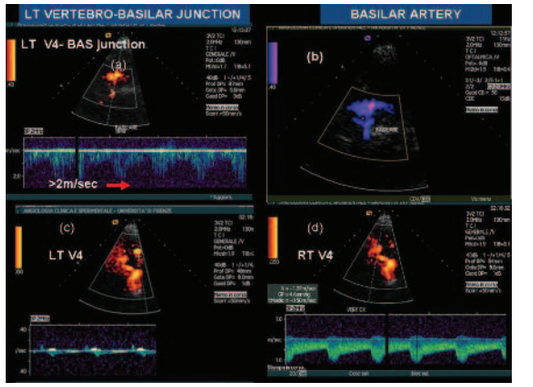
## V4 vertebral artery stenosis

The threshold value for a >50% stenosis of the intracranial tract is 120cm/s with a reported 100% sensitivity, specificity and positive and negative predictive values [50]. The colour appearance of a focal stenosis can be readily seen by colour flow imaging and confirmed by spectral Doppler ultrasound (Figure 20a).

*Figure 20 Power imaging and spectral Doppler of the left vertebral-basilar junction: the detection of a high velocity at the vertebral-basilar junction (top left panel) is consistent very low velocity and high resistance profile, due to the downstream obstruction (lower left panel).*





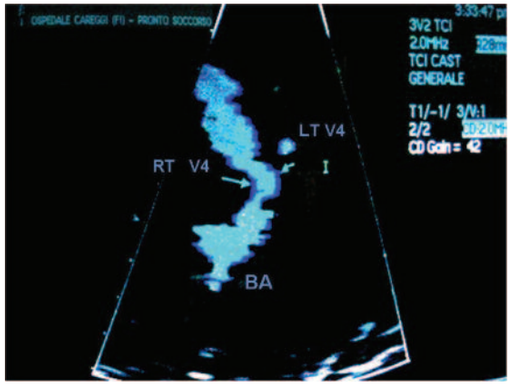



## V4 occlusion

Transcranial colour-coded Doppler diagnostic confidence of TTCS for intracranial vessel occlusion is up to 100% [50]. The direct evidence of an occlusion of the V4 tract is a lack of colour signal from the corresponding segment (Figure 21).

As previously indicated, the finding should not be mistaken with a V4 tract ending in the posterior inferior cerebellar artery, which should be carefully excluded. When the occlusion is associated with the activation of collateral vessels, the effects on the flow in the downstream vascular compartments can be extremely variable according to the location and site of collateral vessels and depending on the location of the obstruction (V0 segment, V4 segment) [41]. Occlusion or a severe stenosis of the V0 segment can translate as an undulating or even antegrade flow pattern in V4 due to the development of cervical collaterals connecting the vertebral artery with the costocervical and thyrocervical trunks [41]. A distal intracarnial V4 occlusion and sometimes a severe stenosis may cause a stump signal or a highly pulsatile flow signal in the upstream V4 portion of the vessel (Figure 21c) with an almost absent end-diastolic component [48].

*Figure 21 Power imaging of the vertebral-basilar junction; the only RT V4 segment can be visualized; no flow signal is observed from the LT V4 due to complete occlusion of this segment.*



## Subclavian steal syndrome

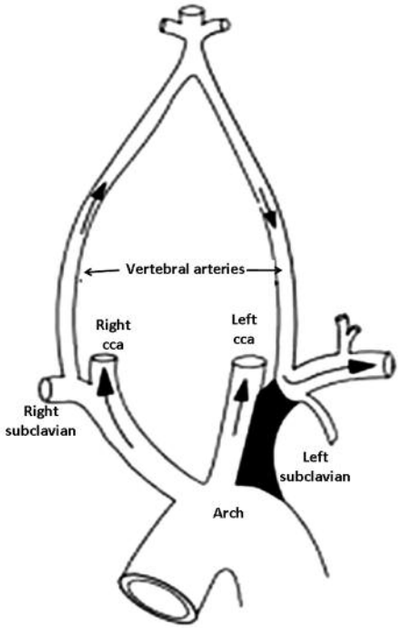
The obstruction of the origin of a subclavian artery is usually the consequence of atherosclerotic disease [41]. The obstructive lesion may be due to an occlusion or severe stenosis leading to a compensatory reaction consisting of an inversion of the arterial flow through the ipsilateral vertebral artery (Figure 22).

Other collateral pathways can also be present, such as the internal thoracic artery, a costocervical trunk, or the thyrocervical trunk (Figure 15). Patients with subclavian steal have a systolic pressure difference greater than 15–20mmHg between the normal and the affected arms.

The clinical signs (dizziness, ataxia and drop attacks) associated with this condition are paradigmatic of the vertebrobasilar insufficiency and they typically occur during exertion of the ipsilateral arm and rarely at rest. In most cases subclavian steal is asymptomatic [51].

Symptoms may occur only in the presence of simultaneous obstructive lesion of intra- or extracranial vessels [52]; in addition, the progression of a stenosis of the subclavian artery is slow and it is not a common cause of embolic events in posterior circulation [46].

*Figure 22 Schematic representation of the haemodynamic changes induced by the occlusion of the proximal part of the left subclavian artery: the ipsilateral vertebral artery exhibits an inversion of the flow direction.*




For these reasons, the detection of an occlusion of the subclavian artery may not have any therapeutic relevance if no neurological symptoms or clinical complaints are present.

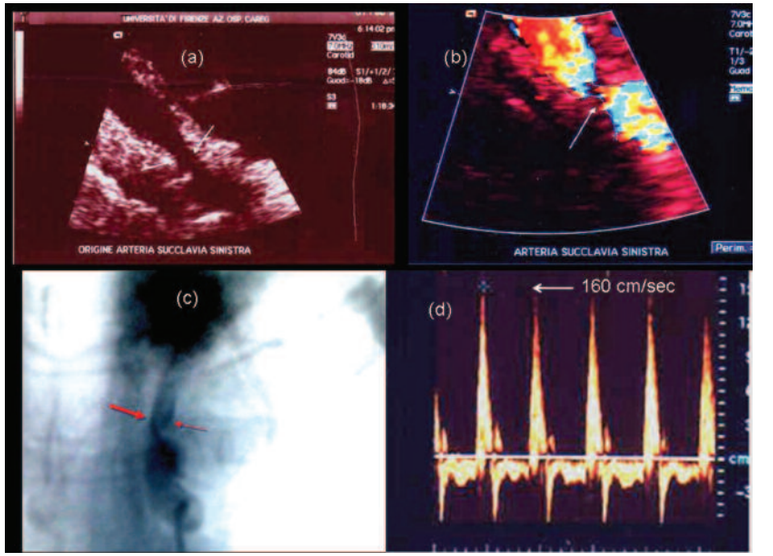




### Ultrasound evaluation

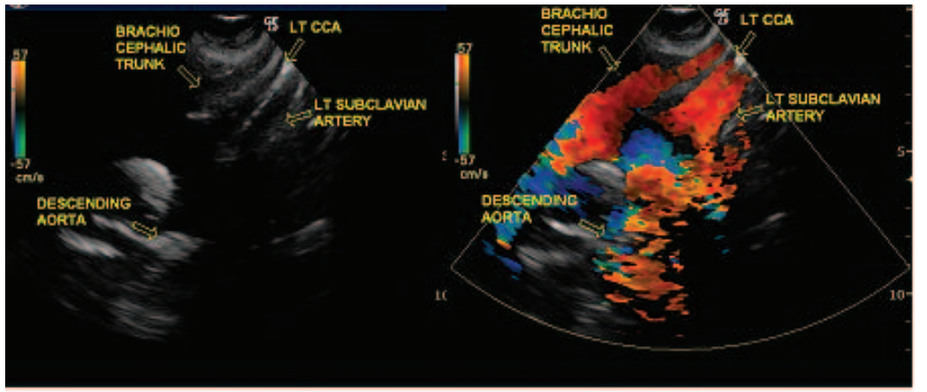
The examination of the subclavian artery can be performed by placing a linear transducer on the supraclavicular fossa in a posteroinferior direction. A pulsatile flow away from the transducer and a characteristic velocity reduction during compression of the brachial arterial allows for quick vessel identification. The velocity profile has a characteristic muscular high-resistance pattern. Right side stenosis is more readily visible due to the more superficial location of the subclavian arteries. However, most stenoses (85%) occur on the left side [53]. When the lumen is visibly narrowed on 2D imaging and a focal turbulence is detected by colour flow imaging, the spectral Doppler sampling can demonstrate high-systolic velocity values at the site of the stenosis (Figure 23).

*Figure 23 A focal turbulence is detected by colour-flow examination of the proximal subclavian artery (b) at the site of lumen reduction (a). The spectral Doppler sampling (d) shows an increase in peak systolic velocity. The angiography confirmed the US finding (c).*



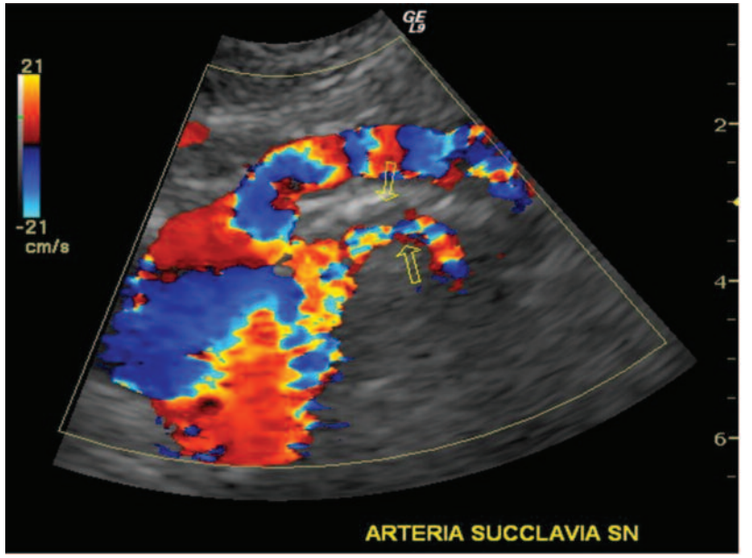

An alternative acoustic window can sometimes be obtained using a sector probe at the suprajugular level: the probe must be first rotated clockwise, towards the left shoulder, and slightly tilted downwards. In this way, it is possible to obtain a single view of the aortic arch and the origin of the brachiocephalic trunk, the left common carotid artery and the subclavian arteries (Figure 24).

*Figure 24 2D and colour flow representation of the thoracic aortic arch by Tran jugular approach: from left to right: the origin of the brachio-cephalic trunk, left common carotid and left subclavian arteries.*




Although the transjugular acoustic window is not satisfactory in many patients, it can be very convenient to directly visualise an obstructive lesion of the origin of the subclavian artery throughout its extension (Figure 25).

*Figure 25 Colour flow representation of the origin of the left common carotid and left subclavian artery from the aortic arch (transjugular approach): the subclavian artery shows an extensive diameter reduction and an extensive intraluminal turbulence due to stenosis.*

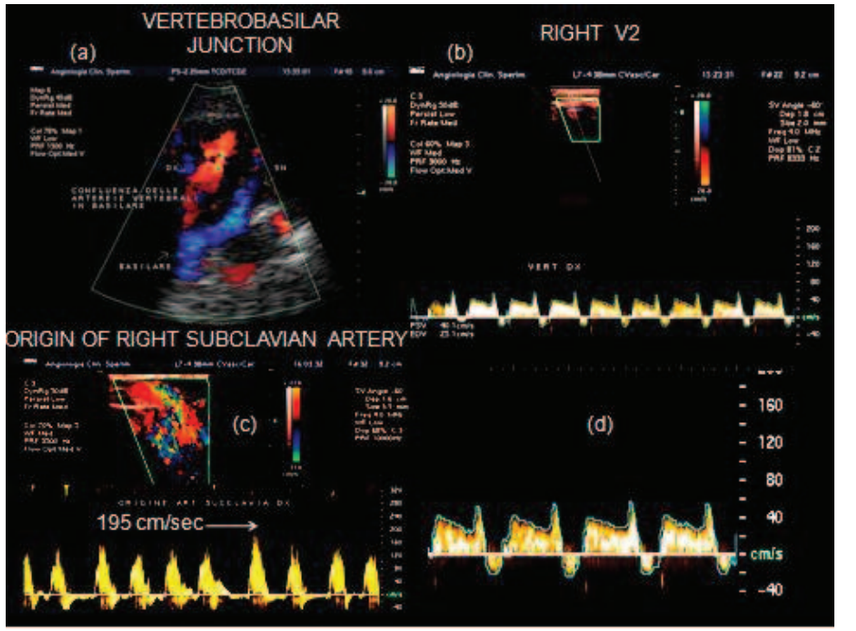






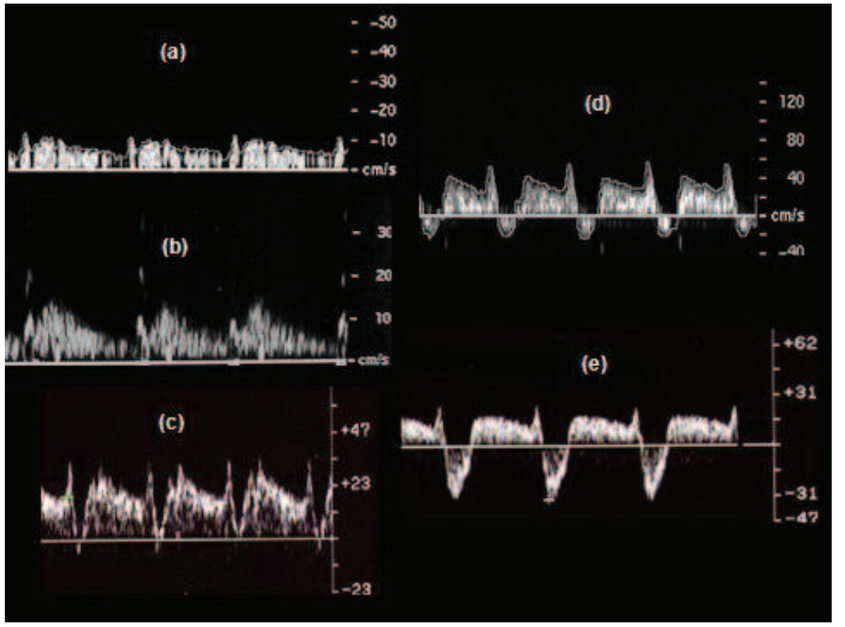

The diagnosis of a subclavian steal is the result of an integrated investigation of the vertebral arteries and the proximal subclavian arteries. The suspicion of subclavian obstructive disease is rarely raised on clinical grounds given the low number of symptomatic patients. More often than not the first abnormal finding is the demonstration of an inverted flow velocity through one of the vertebral arteries during a routine examination of the carotid and vertebral arteries, or during a transcranial study of the vertebral (V4) and basilar arteries (Figure 26).

*Figure 26 Transcranial transformational evidence of a subclavian steal: the distal segment of the right vertebral artery (V4) coloured in red (a) was consistent with an inversion of arterial flow. The finding was confirmed by the partial flow reverse in the V2 segment (spectral Doppler) (b) and was associated with a focal acceleration of the proximal subclavian artery due to stenosis.*




Depending on disease severity, the inversion of flow can vary widely. It may involve only the early systolic hesitation or the entire cardiac cycle (Figure 27). Compensation through ipsilateral vertebral arterial inversion is seen by progressive changes in the vertebral velocity profile, which ranges from increasing systolic deceleration, through to-and-fro flow with retrograde systolic flow and antegrade diastolic flow, to complete retrograde flow (Figure 27).

*Figure 27 The spectral profile of the vertebral artery with early systolic reverse: progressive increase in duration of systolic flow inversion from (a) to (e) are usually associated with different degrees of collateralisation provided by the vertebral artery and to the severity of subclavian obstruction.*



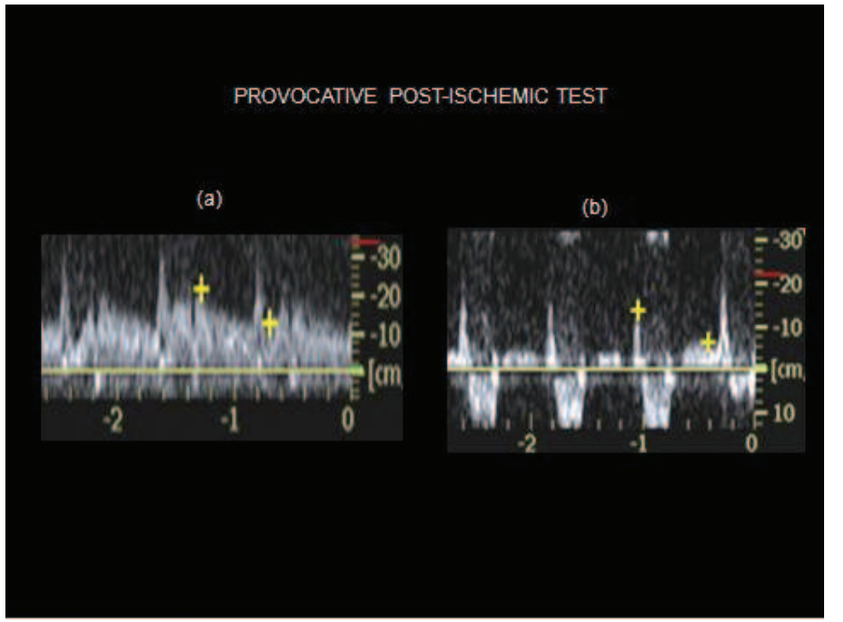

An increase in the steal phenomenon may be deliberately provoked during the hyperaemic reaction following short ischaemia of the arm. The post-ischaemic provocative test is performed by a 3min inflation of an arm cuff (>200mmHg) followed by rapid cuff-release (Figure 28).

At baseline, the vertebral velocity profile shows only a partial systolic reverse. The demonstration of more pronounced changes in the duration of the retrograde flow in the post-ischaemic phase confirms the diagnosis of subclavian steal syndrome and rules out positive pre-test results.





*Figure 28 Post-ischaemic provocative test: the velocity profile of the vertebral artery becomes negative (b) after the release of the arm-cuff and confirms a steal phenomenon caused by a significant obstruction of the subclavian artery.*




## Conclusions

Ultrasound techniques are today the examination of choice for the screening of the atherosclerotic diseases of the carotid and vertebral arteries. In experienced hands they provide valuable information, not only for diagnostic purposes, but also for surveillance of patients following surgical or interventional procedures and in stroke patients. The accuracy, reproducibility and reliability of examination requires a full knowledge of the anatomy of intra- and extracranial circulation. Significant disease at one site should be interpreted, not only for its local effect, but also taking into account its interaction with cardiac activity and intracranial haemodynamics. The best results may be obtained when the operator is fully aware, not only of the potentials offered by the technique, but also of several pitfalls and limitations.

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Ultrasound Evaluation of Cerebrovascular Obstructive Diseases