Ultrasound of the carotid and vertebral arteries

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Ultrasound plays an important role in the assessment of carotid arterial disease, complimentary to other imaging modalities. However, ultrasound does have limitations, not least the requirement of a high degree of operator skill. Recent advances in ultrasound technology will strengthen its role by improving accuracy. This review discusses the role of ultrasound in assessing the carotid arterial system with emphasis on evaluating stenosis

Historically, non-invasive ultrasound (US) of the extra-cranial carotid circulation became possible with oculopneumoplethysmography, in which alterations in peri-orbital vascular beds distal to the carotid bifurcation were detected. Although highly specific, the technique gave no indication as to the site of the abnormality[1]. Increased sophistication of ultrasound, in particular Doppler probes, has allowed direct interrogation of the neck vessels to detect stenotic lesions. Doppler and B-mode US were first linked as 'duplex machines' in 1974[2]. Colour Doppler is now an integral part of the US examination and enables the assessment of flow in vessels and spectral waveform analysis at the point of maximum stenosis[3]. Doppler-derived velocity parameters may be used to detect and quantify stenoses of the internal carotid artery. Indeed, colour Doppler ultrasound (CDUS) is the most cost-effective method of evaluating patients with symptoms of carotid transient ischaemic attacks[4]. This review discusses the role of ultrasound in assessing the carotid arterial system with emphasis on evaluating stenosis.

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Ultrasound assessment of stenosis: technique

The common carotid artery divides into its internal and the external branches at the level of the fourth cervical vertebrae, although the level of this division may vary from T2 to C1[5]. Several centimetres of the internal carotid artery and external carotid artery may be examined

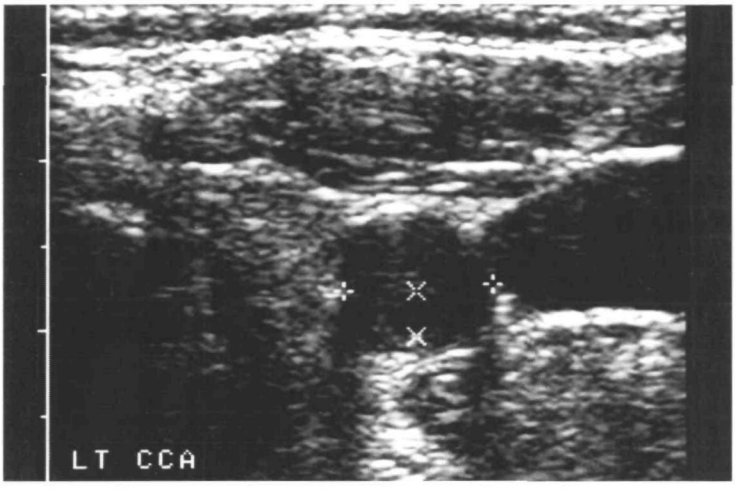


Fig. 1 Transverse section through the distal left common carotid artery (LT CCA) demonstrating a measured diameter reduction of the artery (x–x, residual lumen, +—+, vessel walls with internal circumferential atheroma). This method of calculation of a stenosis is rarely possible

when the bifurcation is at the normal level. In the majority of patients, the internal carotid artery lies posterior and lateral to the external carotid artery. Both vessels have a number of distinguishing US features which aid differentiation. Ideally, the area reduction of an internal carotid artery stenosis should be estimated in the transverse plane with the probe perpendicular to the vessel walls (Fig. 1). However, for a variety of reasons not all internal carotid artery luminal stenoses are amenable to assessment in this manner. A measurement of the velocity of flow in the longitudinal direction is invariably required and constitutes an indirect estimate of the degree of luminal narrowing.

Grey-scale imaging in the longitudinal direction should precede colour Doppler imaging to avoid obscuring subtle areas of plaque. The common carotid artery is generally imaged as far proximal as possible and followed distally to the level of the bifurcation. In most patients, it is not possible to examine the external carotid artery, internal carotid artery and carotid bulb at the same time and each vessel should be examined sequentially. It may be possible to identify the region of maximal internal carotid artery narrowing on grey-scale images thereby allowing accurate placement of the pulsed-Doppler sample volume. However, an area of colour turbulence on CDUS allows accurate placement of the pulsed-Doppler sample volume at the point of maximum narrowing (Fig. 2) and improves the sensitivity and speed of the examination[6,7]. A spectral waveform is obtained from which a velocity measurement is generated using the Doppler equation (Appendix 1).

The angle of insonation must remain constant (*i.e.* less than 60°) for velocity measurement, since small changes in angle may affect absolute velocity readings. The extent of atherosclerotic narrowing is calculated from measurements of the peak systolic and end diastolic velocities in

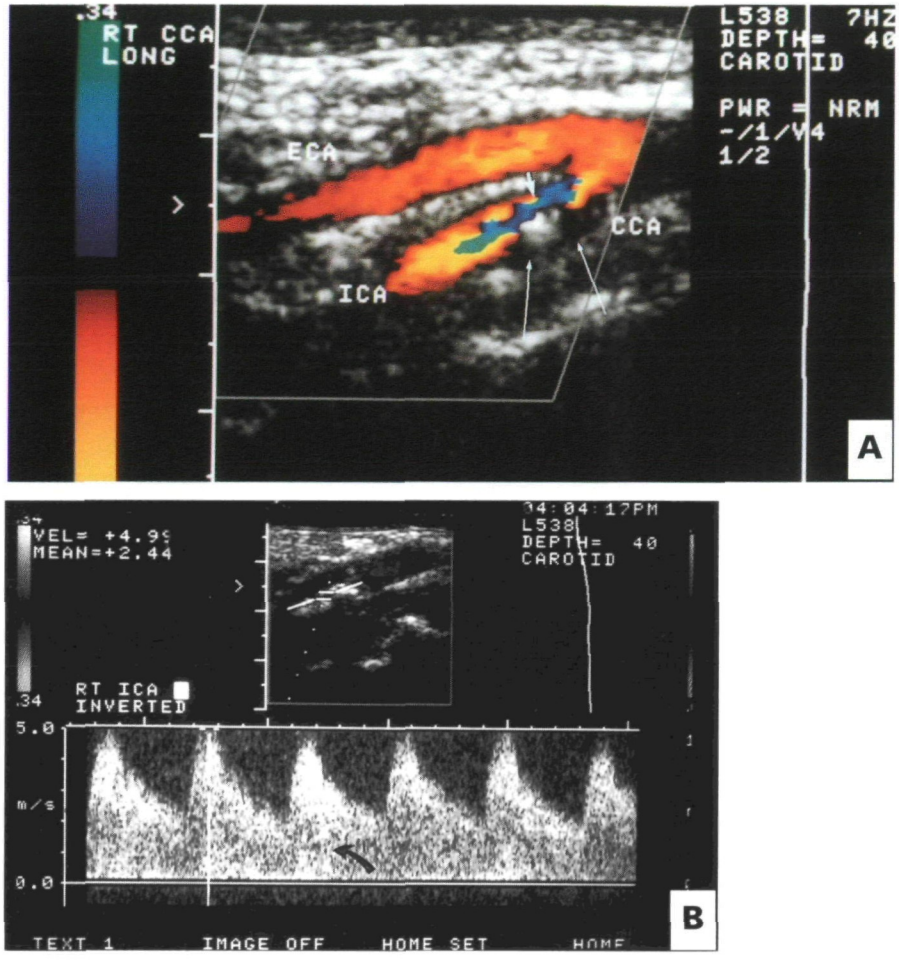


Fig. 2 (**A**) Colour disturbance (blue, arrow) in the proximal internal carotid artery (ICA). This indicates an area of stenosis caused by heterogeneous plaque disease in the carotid bulb (long arrow, ECA; external carotid artery, CCA; common carotid artery). (**B**) With the spectral gate placed at the region of greatest colour disturbance, a spectral Doppler waveform shows an elevated velocity of 4.99 m/s (normal < 1.0 m/s). The 'in-filling' beneath the outline of the spectral trace (curved arrow) is called spectral broadening and represents a multitude of different velocities.

the common carotid artery and internal carotid artery. It is important to identify and record the spectral waveform in the external carotid artery, so that it is clear that the velocities have been measured in the correct vessels. Flow reversal, manifest as colour change from red to blue separated by a thin black line in the carotid bulb opposite to the origin of the external carotid artery, is commonly seen and should not be mistaken for turbulence.

The vertebral arteries may also be interrogated during the examination. By moving the probe laterally beyond the common carotid artery, the arteries are identified between the transverse processes of the cervical spine. The vertebral artery can normally be examined in three segments: the proximal (pre-transverse) portion, the inter-transverse portion and the atlas loop[8]. The more anteriorly located vertebral vein should be readily identified on spectral and colour Doppler. Failure to show the vertebral arteries may be due to absence or occlusion which will not be

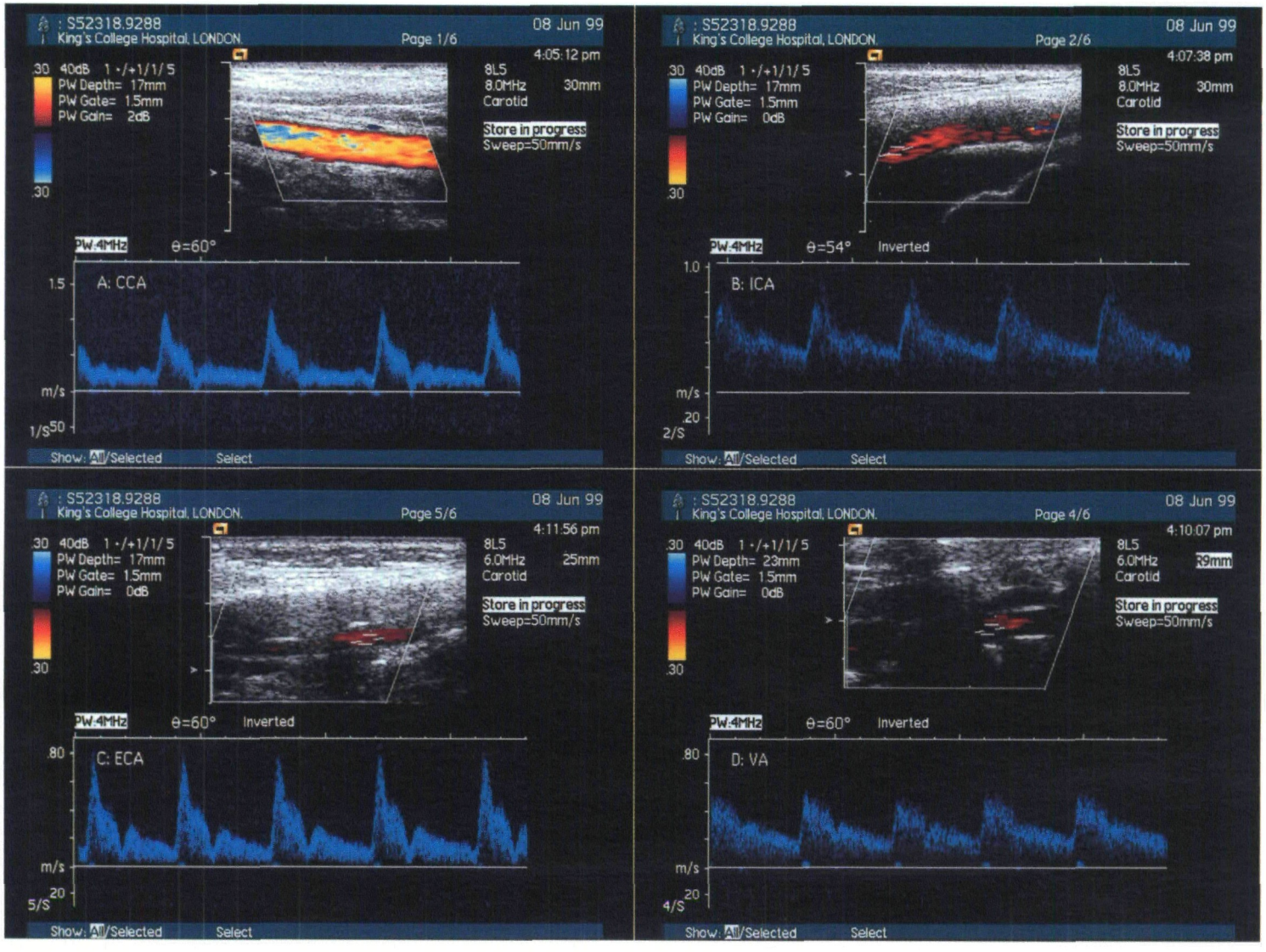


Fig. 3 Normal spectral Doppler waveform patterns (A) Common carotid artery (CCA). (B) Internal carotid artery (ICA) (C) External carotid artery (ECA). (D) Vertebral artery (VA)

clear from the ultrasound examination. The calibre of the vertebral arteries may be asymmetrical in up to 25% of normal individuals, with the left usually the larger[9].

The normal spectral waveform patterns of the common carotid artery, internal carotid artery, external carotid artery and the vertebral artery are shown in Figure 3. In general, both the internal carotid artery and the vertebral artery, which directly supply brain parenchyma, have a 'low resistance' pattern on spectral analysis which contrasts with the 'high resistance' external carotid artery, which supplies the facial muscles and the scalp. There is a wide systolic peak and high diastolic flow in the internal carotid artery and vertebral artery, whereas the external carotid artery has a narrow systolic peak and absence of diastolic flow. The common carotid artery is a hybrid of the two patterns with a narrow systolic peak and some forward diastolic flow. Velocity measurements are calculated using the Doppler equation from the frequency shift (Appendix 1)[10]. However, it should be remembered that the accuracy and reproducibility of velocity measurements between ultrasound machines has been questioned; differences of up to 15% have been documented[11].

Numerous velocity measurements to grade stenoses have been suggested[12–15]. The most commonly used criteria are the internal carotid peak systolic velocity (ICPSV) and the ICPSV to common carotid peak systolic (CCPSV) ratio, with the end diastolic velocity used to discriminate in borderline measurements.

Velocity parameters, in the crucial 60–70% diameter reduction range, have been re-assessed in line with the conclusions of the North American Symptomatic Carotid Endarterectomy Trial (NASCET)[16] and European Carotid Surgery Trial[17]. In one retrospective study, a variety of ultrasound parameters were compared with those at angiography[18]. The authors concluded that the single most useful measurement was the ICPSV, which when greater than a measurement of 230 cm/s, indicated a diameter reduction of greater than 70%. Moneta *et al* have prospectively studied 100 angiograms, measuring the degree of diameter reduction based on NASCET criteria and correlating this with findings on CDUS[19]. An ICPSV/CCPSV ratio of greater than 4.0 provided the best combination of sensitivity, specificity, positive predictive value, negative predictive value and overall accuracy for detection of a 70–99% stenosis. An ICPSV of greater than 130 cm/s and an ICPSV/CCPSV ratio of greater than 3.2 suggests a reduction in diameter of greater than 60%. A guide to these measurement indices is shown in Table 1.

In practice, these figures should only be the basis from which to develop criteria specific for individual vascular departments and the type of equipment used. Ideally each centre should be constantly auditing CDUS findings, comparing these to angiography and findings at surgery. The particular criteria chosen to assess a stenosis should be carefully

Table 1 Suggested duplex Doppler ultrasound criteria for grading internal carotid artery (ICA) diameter reduction, based on derived figures[19,105]

Diameter reduction (%)	PSV	EDV	PSV_{ICA}/PSV_{CCA}
0–29	< 100	< 40	< 3 2
30–49	110–130	< 40	< 3 2
50–59	> 130	< 40	< 3 2
60–69	> 130	40–110	3 2–4 0
70–79	> 230	110–140	> 4 0
80-95	> 230	> 140	> 4 0
96–99	'String flow'		
100	'No flow'		

CCA, common carotid artery, PSV, peak systolic velocity, EDV, end diastolic velocity, PSV_{ICA}/PSV_{CCA}, ratio of the velocities Velocity measurements are in cm/s

considered. For example, employing a single ICPSV measurement instead of a ratio measurement does not eliminate the problems of variable cardiac output, cardiac arrhythmia, the presence of a proximal common carotid artery narrowing (the so-called 'tandem' lesion[20]) and interval changes of myocardial function. Often a combination of measurements will increase the accuracy of estimation of the stenosis.

Comparison of imaging modalities

Conventional arteriography, magnetic resonance arteriography and contrast enhanced dynamic computed tomography provide an assessment of the luminal size but are unable to characterize the vessel wall or associated plaques. Early plaque formation is accompanied by compensatory arterial enlargement, a phenomenon seen both in the coronary[21] and the carotid arteries[22], and significant plaque formation may occur before detectable luminal narrowing on arteriography. A comparison between ultrasound and arteriography in 900 patients demonstrated that half of the 345 arteries considered normal at arteriography were shown to have a lesion at ultrasound[23]. It would seem, therefore, that ultrasound may be of value in evaluating atherosclerosis in the carotid arteries. The accuracy of colour Doppler ultrasound in comparison with conventional angiography is not disputed[7,24].

Limitations of carotid Doppler ultrasound

A number of technical factors may limit the value of ultrasound examination. With extensive plaque disease, particularly if calcified, acoustic shadowing may hamper insonation of the area distal to the

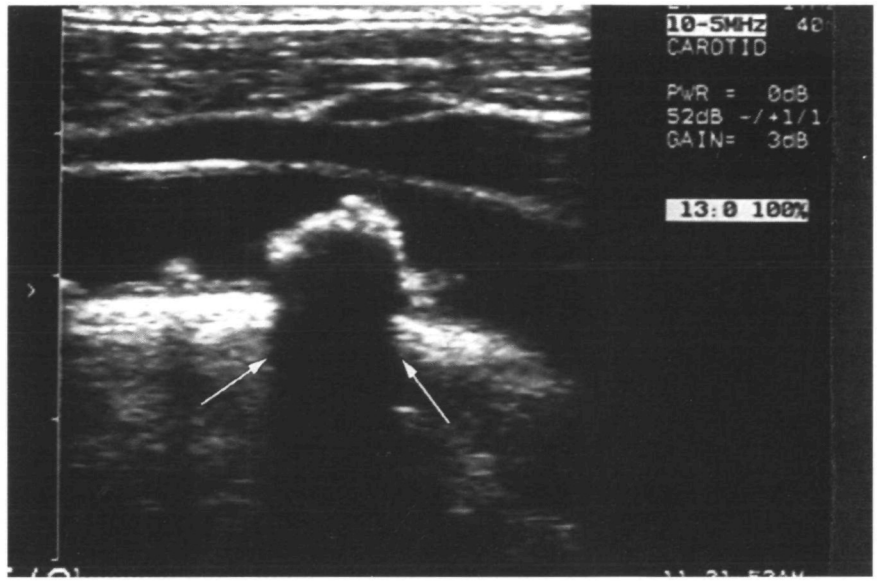


Fig. 4 An area of plaque in the common carotid artery containing a substantial amount of calcification causes marked distal acoustic shadowing (arrows). The acoustic shadowing precludes interrogation of the area beneath the plaque

calcification (Fig. 4). Significant narrowing of the underlying vessel may be present in the absence of a high velocity jet. Varying the angle of insonation (*e.g.* posteriorly) may be helpful, but often the examination is inconclusive and another imaging modality is necessary. Similarly, a bifurcation at the level of the mandible may be obscured. Finally, a tortuous vessel may result in a spurious increase in velocity. In practice, this is less of a problem with colour Doppler imaging since the vessel is completely filled with colour and no narrowing is demonstrated.

Detection of carotid occlusion

The distinction between a total occlusion and a 99% diameter reduction is crucial, since the former is a contra-indication to surgery[25]. In high-grade internal carotid artery lesions, a reduction in volume flow causes the velocity measured at the stenosis to decrease and normal velocity criteria no longer apply. Internal carotid artery occlusion on US can be inferred on the basis of the lack of pulsation or expansion of vessel walls but this is unreliable[26]. The diagnosis of occlusion based on the detection of a thrombus-filled lumen, the absence of wall motion characteristics and the lack of Doppler flow signal has a high reported accuracy[27]. Characteristic flow reversal, with dampening of flow in the common carotid artery, may also be present in the patent vessel just proximal to the occlusion. With long-standing occlusion, the external carotid artery may become 'internalized' with the development of collaterals, particularly around the orbit, producing a hypertrophied external carotid artery with a high forward

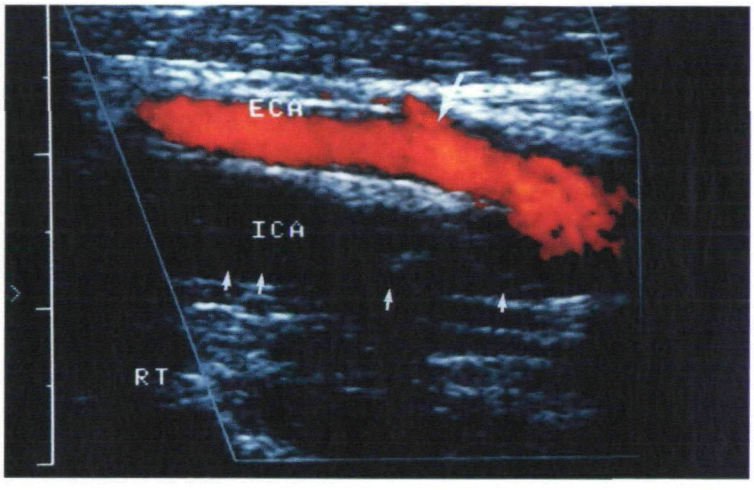
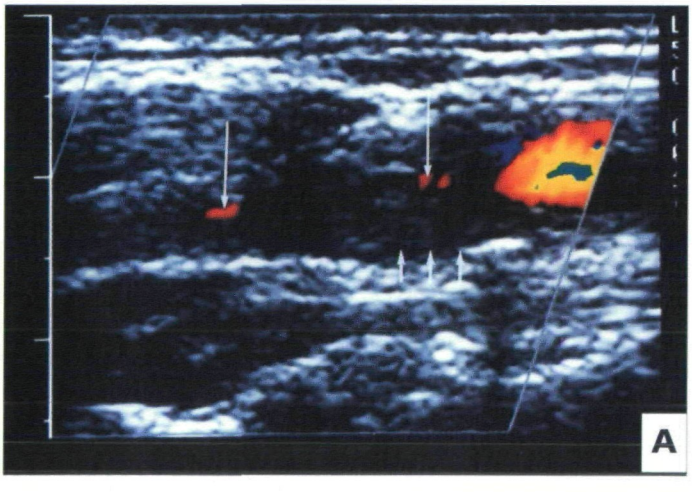


Fig. 5 No colour signal is obtained from the lumen of the internal carotid artery (ICA) which is filled with echo poor thrombus (arrows) The external carotid artery (ECA) is hypertrophied with a prominent branch (long arrow) These appearances are those of a long-standing ICA occlusion

diastolic component in the spectral waveform (Fig. 5). The ipsilateral vertebral artery may then become dominant.

Colour Doppler has improved the ability of US to distinguish between an occlusion and severe stenosis by allowing a narrow channel to be identified. However, technical difficulties remain (Fig. 6). The accuracy in detection of a very narrow patent channel of severe stenosis and the distinction from a total occlusion is likely to improve in the future with the use of 'power' Doppler and intravenous ultrasound contrast media[28].



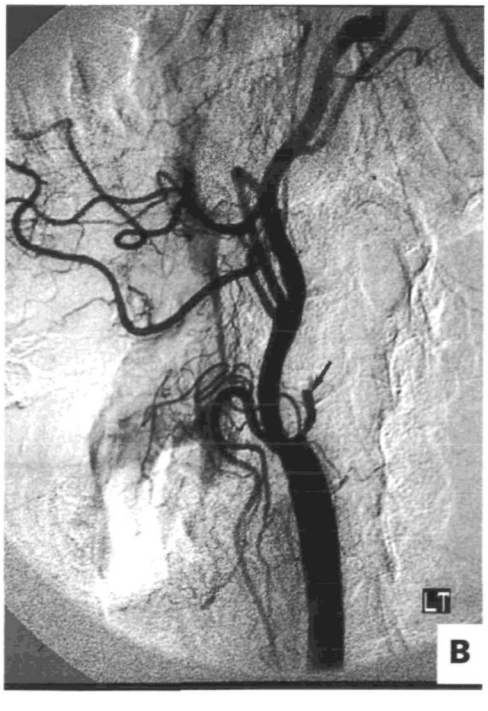


Fig. 6 (**A**) The lumen of the internal carotid artery (short arrows) is filled with echo-poor thrombus but some flow is demonstrated on colour Doppler ultrasound (long arrows) with optimized flow settings, indicating a near occlusion. (**B**) Angiography confirms a short patent segment of internal carotid artery (arrow).

Plaque morphology

The extent of plaque disease has been positively correlated with an increasing likelihood of cerebrovascular events[29–31]. Plaque disease associated with haemorrhage and ulceration may be associated with cerebrovascular symptoms[32]. Many patients with symptoms of carotid disease do not have a significant stenosis of the ipsilateral internal carotid artery and those with a significant stenosis do not necessarily have symptoms. In one study, the number of patients with a significant stenosis and symptoms of stroke attributable to the stenosis was estimated at 20% of the total number of stroke patients[33]. The potential for atherosclerotic plaques to produce cerebral ischaemic events is dependent not only on flow-reduction through the stenosis but also the embologenic property of atherosclerotic plaque[34].

The importance of plaque morphology in assessing risk has received less attention than quantifying stenosis. The morphological characteristics of plaque, as seen on US, may provide important information on the type of plaque and the risk for neurological events. As discussed above, arteriography, magnetic resonance arteriography and contrast enhanced dynamic computed tomography provide an assessment of the luminal size but are unable to characterize the vessel wall or associated plaque[35].

Classification of plaque

A classification of plaque morphology has been suggested[36]. Four categories are recognized: predominantly echolucent (type 1), uniformly echogenic (type 4), with the intermediate forms being more echolucent (type 2) and more echogenic (type 3). Calcification may occur in all plaque types[37]. More recently, a classification of plaque has been proposed by the Committee on Standards in Non-invasive Vascular Testing of the Joint Council of the Society for Vascular Surgery and the International Society for Cardiovascular Surgery[26]. Put simply, plaques may be characterized as homogeneous or heterogeneous (Fig. 7). The division is subjective with heterogeneous plaques being those in which major differences in echogenicity are identified.

In an attempt to classify carotid bifurcation disease to take account of both the grade of diameter stenosis and plaque characteristics, the same committee has suggested a classification system modelled on the TNM classification of tumours. The plaque is characterized by 'P' where P1 = homogeneous and P2 = heterogeneous, the surface character by 'S' where S1 = smooth, S2 = irregular and S3 = ulcerated and the luminal narrowing by 'H' (H1–H5: degree of stenosis), taking into account all clinically important features.

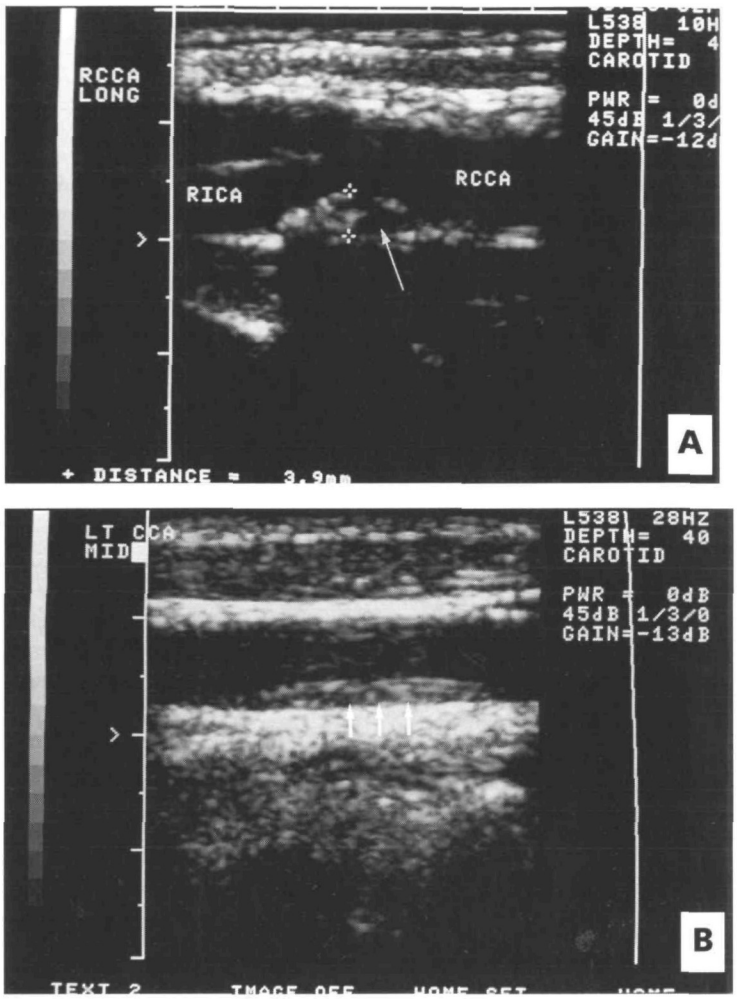


Fig. 7 (**A**) Heterogeneous plaque (cursors) in the right carotid bulb. Echolucent areas (arrow) correspond to lipid deposits or areas of intraplaque haemorrhage. (RCCA, *right common carotid* artery, RICA, right internal carotid artery (**B**) Homogenous plaque with a uniform echogenic appearance (arrows).

Complications of plaque disease

Clinicopathological studies of carotid atheroma have implicated plaque structure, more specifically intraplaque haemorrhage, as an aetiological factor in symptomatic carotid artery disease[38–40]. However, disagreement exists about the relationship between the histological nature of plaque and US features. A study which correlated histology with US suggested that fibrous plaques are highly echogenic. As the lipid content increased, plaque became more echolucent[41]. It is suggested that the increased lipid and cholesterol content of echolucent plaque renders these plaques unstable[42]; echogenic plaques contain significantly more fibrin and collagen, rendering them more stable[43,44]. Other studies have suggested that low echogenic areas in plaque demonstrate a high correlation with areas of intraplaque

haemorrhage on histological examination[37,45]. These low echogenic areas, thought to represent intraplaque haemorrhage, have been associated with an increased risk of cerebrovascular events[34]. A recent study found a specificity of only 78% and a sensitivity of only 44% for detecting intraplaque haemorrhage as echolucent areas on US[46]. However, the concept of a haemorrhage-containing plaque, has been questioned[47]. Indeed, the value of US in the detection of plaque haemorrhage remains controversial.

Whatever the histological correlate of the echolucent areas within atherosclerotic plaques, this ultrasound feature seems to identify patients with a greater risk of symptomatic cerebrovascular disease. Heterogeneous plaques have been shown to be the dominant plaque type in symptomatic patients with a greater than 70% stenosis[48]; these patients have an increase in neurological deficits over a 3 year follow–up period[49]. Heterogeneous plaques are also the dominant plaque in asymptomatic patients who are subsequently symptomatic[50]. Fibrous plaques may predict a tendency to remain clinically stable[51].

The surface morphology of plaque may be categorized as smooth, irregular or ulcerated (Fig. 8)[52]. Ulcerated plaques have been associated with an increase in the number of infarcts detected on computed tomography and, therefore, represent a more sinister finding than a smooth fibrotic plaque[53]; plaque ulceration predisposes to the development of thrombus and subsequent emboli. Lipid emboli may also arise from the ulcerated surface[54]. A comparison of US and arteriography with surgical specimens, in the assessment of plaque surface ulceration and irregularity, has shown that US detection is superior to that of arteriography[55,56]. However, even US does not consistently evaluate ulceration[57]. It is, therefore, better to classify lesions seen on US as either smooth or irregular and only if a crater depth of greater than 2 mm is seen can ulceration be considered present and more likely to initiate an embolus[26].

Intima-medial thickness

The development of plaque disease and the consequent arterial narrowing are late manifestations of atherosclerotic disease; minor alterations in the inner arterial wall predate these changes. Ultrasound can be used to image the boundaries of the inner arterial wall with clarity[58]. The inner echogenic line represents the luminal–intimal interface and the outer echogenic line represents the media-adventitia interface; the distance between the two lines is a measure of the thickness of the combined intima and media[59]. These interfaces can be seen on both the near and far walls of larger arteries when the US beam is perpendicular to the wall, but more clearly on the far wall in vessels

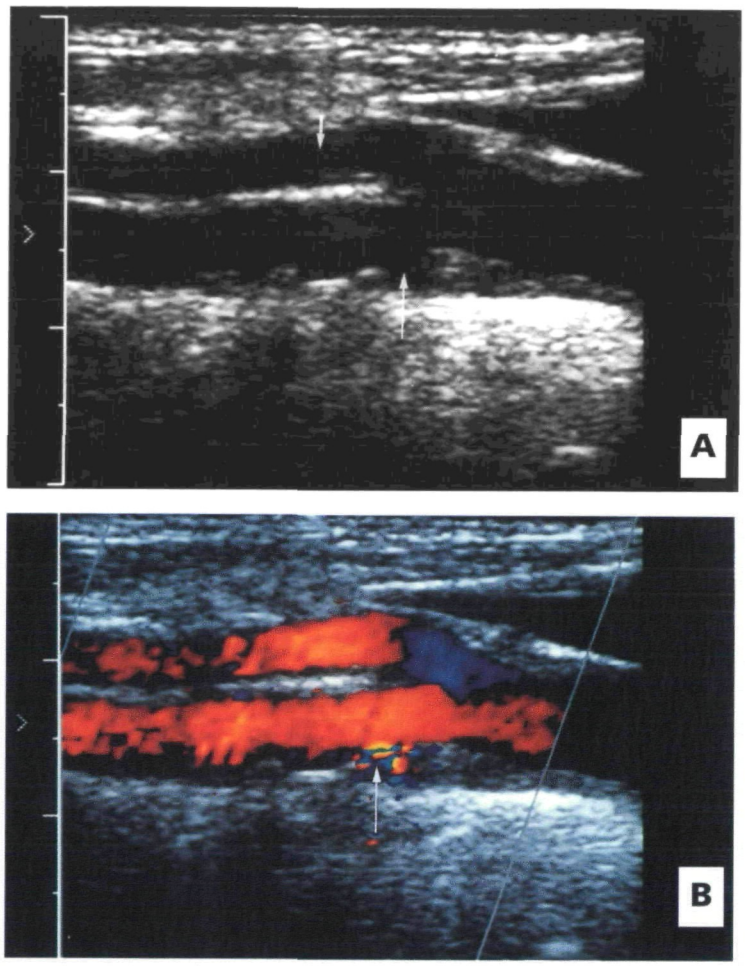


Fig. 8 (**A**) An ulcerated plaque (arrow) is demonstrated in the carotid bulb (short arrow; external carotid artery) (**B**) The ulcer crater fills with turbulent flow on colour Doppler ultrasound (arrow).

running parallel to the skin surface (Fig. 9). An increase in the distance between these two lines, the intima-media thickness (IMT) predicts atherosclerosis in other vessels, especially the coronary arteries[60].

A number of risk factors have been associated with an increased IMT in the carotid artery. Patients with hypercholesterolaemia are at higher risk of developing cardiovascular disease and elevated levels of cholesterol, independent of other risk factors, have been related to an increased IMT[61,62]. Other factors have been linked to increased IMT measurements and are detailed in Table 2[61–69]. It has been demonstrated that HDL-cholesterol levels have a negative correlation with IMT measurements suggesting a protective effect at the arterial wall level[70]. Increased IMT is associated with the presence of severe angiographically-detected coronary heart disease[63] and has been related to the presence of coronary artery calcification, itself a marker of early coronary artery disease, on ultra-fast computed tomography[71].

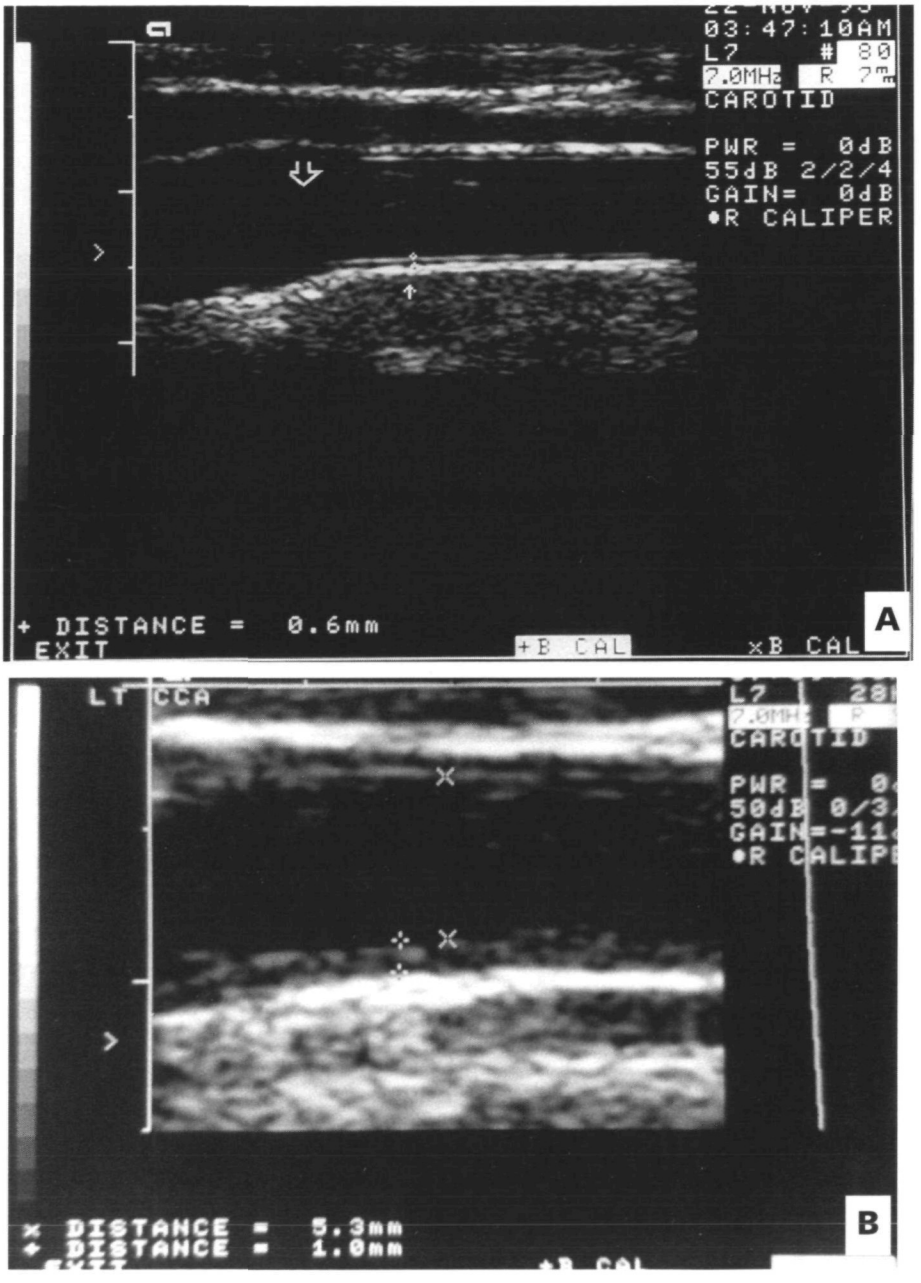


Fig. 9 (**A**) Longitudinal image of the distal common carotid artery and bulb (open arrow) demonstrating the inner arterial wall complex (arrow, between cursors) in a normal young person. (**B**) Same position in the distal common carotid artery in a patient with hypercholesterolaemia. The intima-media layer is thickened (+—+, 1.0 mm). The lumen of the common carotid is between the cursors (x–x).

More recently[72], the measurement of IMT has been used to document the regression of atherosclerotic disease in patients treated with the 3-hydroxy-3-methylglutaryl-coenzyme-A reductase inhibitors, such as lovastatin™. A lowering of serum cholesterol has a direct effect on the arterial wall, suggesting that increased IMT may be used as a marker of regression or progression of atherosclerosis.

Table 2 Risk factors for an increase in intimal-medial thickness (IMT) of the inner arterial wall as seen on ultrasound

Age
Familial hypercholesterolaemia
LDL-cholesterol
Lipoprotein (a)
Active and passive smoking
Homocysteine
Chronic exposure to elevated levels of serum angiotensin converting enzyme
Hypertension
Diabetes mellitus

Currently, there is no consensus on the ideal location for the measurement of IMT. In practice, it is appropriate to measure at a site that is readily visualized so that reproducible measurements may be made. Typically, IMT is measured along the far wall of the common carotid artery bilaterally, within 1 cm proximal to the carotid bulb[73]. The median wall thickness in adults ranges between 0.5–1.0 mm, with an increase with advancing age[74]. In general, IMT is thicker in men[75]. Values of greater than 1.0 mm are considered abnormal by most observers[76].

Vertebral artery

Reversal of flow in the vertebral artery ipsilateral to a proximal subclavian artery stenosis was demonstrated separately by Contorni[77] and Reivich[78] in 1960, and termed the subclavian steal syndrome by Fisher[79]. Initially, a large spectrum of symptoms were thought to be a result of this disorder, arising from brainstem ischaemia and stroke; these were considered to occur spontaneously or secondary to arm exercise[80]. More recent observations have raised doubts as to the significance of retrograde vertebral artery flow in producing cerebrovascular events. Like internal carotid artery stenosis, the subclavian steal phenomenon represents generalized atherosclerosis and may be a harmless haemodynamic phenomenon[81,82].

Demonstration of reversed flow in the vertebral artery by CDUS is accepted practice and is a valid substitute to arteriography as the first line investigation. However, arteriography is still sometimes necessary to delineate the proximal subclavian artery abnormality[83]. Routine examination of the vertebral arteries during a CDUS examination is rapid and ascertains the presence of vertebral artery flow reversal. The detection of reversal or biphasic flow at rest in the vertebral artery allows the diagnosis of subclavian steal to be made without any further investigation. In some cases, steal from the basilar artery does not occur at rest and blood flow to the arm must be increased to demonstrate vertebral flow reversal[84].

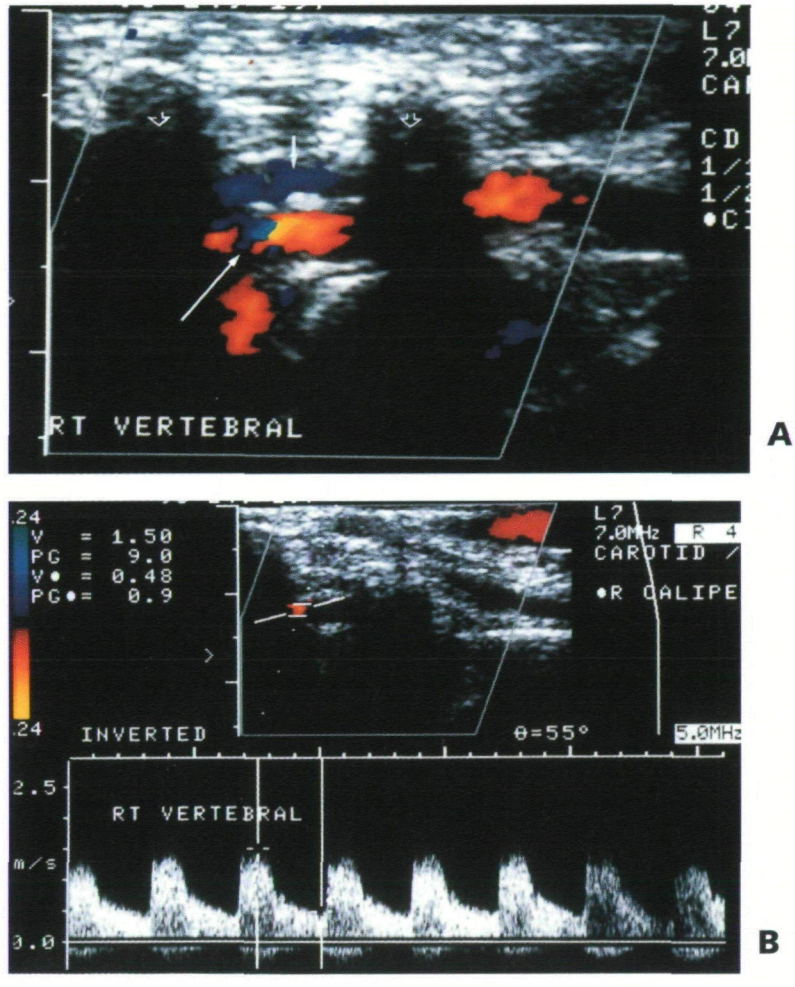


Fig. 10 (**A**) Colour Doppler ultrasound of the vertebral right vertebral artery demonstrating an area of colour disturbance corresponding to a stenosis (arrow). The open arrows indicate the transverse processes of the cervical vertebral bodies through which the vertebral courses (short arrow, vertebral vein) (**B**) Spectral Doppler waveform indicates a velocity of 1 50 m/s, confirming a stenosis

Stenosis on vertebral artery US may be seen as turbulent flow or waveform dampening[85]. The relationship between the severity of a vertebral artery stenosis and the peak systolic velocity has not been fully assessed. The average peak systolic velocity in the normal vertebral artery is estimated at 56 cm/s (range 19–98 cm/s)[9]; a focal velocity greater than 100 cm/s, accompanied by disturbed flow is suggestive of a stenosis (Fig. 10). Transcranial US is able to image the distal vertebral arteries to the level of the basilar artery and this may extend the value of US in the assessment of the posterior fossa circulation[86].

Dissection of the carotid and vertebral artery

Angiography has been the method of choice for imaging an internal carotid artery dissection[87]. The most common angiographic feature is a tapered

stenosis, less often an occlusion or an aneurysm is seen[88]. Ultrasound is capable of imaging the flow dynamics of a dissection, establishing the patency of a false lumen as well as defining the extent of thrombus in the vessel wall. Imaging with dynamic contrast enhanced spiral computed tomography and magnetic resonance, do not allow the interrogation of flow dynamics; demonstrating a peri-arterial rim of intramural haematoma surrounding either a normal or narrowed flow void[89].

In two series of internal carotid artery dissections imaged with US, the commonest spectral Doppler finding was either a bi-directional high resistance pattern or absence of a signal in a total occlusion[90,91]. Furthermore, resolution occurred in a high proportion of patients – 68% over a mean time of 51 days. Flow reversal[92], bi-directional flow[93] and forward flow[94] may occur in the false lumen during the cardiac cycle. Vertebral artery dissections are less common, occurring secondary to neck trauma and spontaneously[95]. Vertebral artery dissections have been analyzed using both transcranial and extracranial US, but patterns are non-characteristic[96,97].

Other vascular diseases

A non-specific response to terminal internal carotid artery occlusion of any cause is the development of a fine system of collateral vessels around the base of the brain, the angiographic features described as a 'puff of smoke'[98]. Classically, these appearances are associated with moya-moya disease[99]. In an assessment of the ultrasound appearances associated with moya-moya disease, abnormal spectral waveforms were obtained from the ipsilateral internal carotid artery showing either no flow or a high-resistance flow pattern[100]. The US appearances in the extracranial internal carotid artery can mimic a dissection[101].

In Takayasu's disease, US demonstrates a characteristic circumferential arterial wall thickening, described as a 'macaroni-like' diffusely thickened intima-medial complex[102]. Ultrasound was found to be superior to arteriography in delineating this abnormality and, therefore, able to detect disease at an earlier stage[103]. Furthermore, US was found to be more sensitive than magnetic resonance in resolving wall thickening. Spectral Doppler usually shows a high resistance pattern[103]. Follow-up with US allows the monitoring of the regression of the wall abnormalities while on treatment, greatly reducing the need for repeated angiography.

Conclusion

Ultrasound plays an important role in the assessment of carotid arterial disease, complimentary to other imaging modalities. However, ultrasound

does have limitations, not least the requirement of a high degree of operator skill. Recent advances in ultrasound technology will strengthen its role by improving accuracy[104]. Carotid Doppler ultrasound is an essential tool in the armamentarium of the stroke physician.

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Appendix 1

Doppler equation

$$f_D = 2v (\cos \theta) f/c$$

Where, f_D = difference between received and transmitted ultrasound frequency; v = speed of the target; θ = angle between the direction of the ultrasound beam and the target; c = speed of sound in tissue; and f = frequency of the transmitted ultrasound.