Color Duplex Scanning on Other Miscellaneous Carotid Topics

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Abstract

Although patients should be evaluated by careful history and physical examination, our policy tends to rely on noninvasive vascular testing (duplex ultrasound) as an initial step in the diagnosis of carotid artery disease. The results of noninvasive tests may also help in obtaining optimal angiograms. An example is the patient with noninvasive evidence of severe stenosis who has no significant stenosis demonstrated in standard views of the carotid artery bifurcation. The results of the noninvasive tests indicate the need for additional projections, and if the bifurcation region does not show the expected lesion, there is a strong indication for obtaining adequate siphon views. This chapter will summarize the clinical implications of the vascular laboratory in the diagnosis of cerebrovascular insufficiency including the role of magnetic resonance angiography, computed tomography angiography, color duplex ultrasound, and catheter-based digital subtraction arteriography, in single or combination, asymptomatic carotid bruit, management of patients with transient ischemic attacks (TIA), intraoperative duplex ultrasound assessment of carotid endarterectomy, and post-carotid endarterectomy stroke. It will also cover the value of duplex ultrasound after carotid endarterectomy, post-carotid artery stenting, and the diagnosis of temporal arteritis.

Keywords

Cerebrovascular insufficiency • Diagnosis • Noninvasive vascular testing

Various noninvasive tests for the evaluation of cerebrovascular insufficiency have been described in previous chapters. While early forms of noninvasive testing depended on the presence of severe disease, the current techniques, especially carotid artery imaging, demonstrate the opposite characteristic. Carotid imaging is able to detect minimal disease that is not hemodynamically significant; in fact, overestimation of the degree of stenosis in some cases has been a consistent problem. Nevertheless, any test intended for screening must have a high degree of sensitivity to be used appropriately in the initial assessment of disease. Noninvasive assessment, therefore, combines low risk, low cost, and high sensitivity.

Although we agree that patients should be evaluated by careful history and physical examination, our policy tends to rely on noninvasive vascular testing as an initial step in the diagnosis of carotid artery disease. The results of noninvasive tests may also help in obtaining optimal angiograms. An example is the patient with noninvasive evidence of severe stenosis who has no significant stenosis demonstrated in standard views of the carotid artery bifurcation. The results of the noninvasive tests indicate the need for additional projections, and if the bifurcation region does not show the expected lesion, there is a strong indication for obtaining adequate siphon views.

Prior to the advent of digital techniques, standard angiograms were routinely used in the evaluation of patients with

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cerebral ischemic attacks in order to determine whether vascular reconstructive surgery was indicated. Standard angiography was of limited clinical value, particularly as a means of diagnostic screening in asymptomatic patients, because of prohibitive costs, poor patient acceptance, and the risk of arterial catheterization. As a result, noninvasive vascular tests became established as the preferred means of diagnostic screening in asymptomatic patients, because they provided an objective method of determining the hemodynamic significance of carotid disease in a safe and relatively cost-efficient manner.

Recent studies have questioned the role of arteriography as the "gold standard" in the evaluation of carotid artery occlusive disease [1–3]. Contrast arteriography has also been noted to have a 1–4% incidence of neurologic complications with about a 1% incidence of stroke reported in the Asymptomatic Carotid Atherosclerosis Study (ACAS) [4]. Other complications of arteriography that were reported include complications at the arterial puncture site (5%) and contrast-induced renal dysfunctions in 1–5%. With this in mind, it would be beneficial and cost-effective if these patients could be safely evaluated without invasive arteriography. Color duplex ultrasonography of the carotid arteries, computed tomography angiography, and magnetic resonance angiography are noninvasive/minimally invasive modalities that can detect and grade carotid artery stenosis. The role of each modality in the diagnosis of carotid artery disease is discussed in detail in Chap. 18.

Role of Carotid Imaging in Patients with Asymptomatic Carotid Bruit

Carotid bruits may be helpful in detecting carotid artery stenosis. Carotid bruits must be differentiated from venous hums which are found in one-fourth of young adults or cardiac murmurs [5]. The venous hum typically increases in quality with the neck turned away from the auscultated side and often disappears with Valsalva maneuver or when patient lies down. Meanwhile, there is no definitive way to differentiate a radiated cardiac murmur or bruit originating from intrathoracic vessels from a cervical carotid bruit; however cardiac murmurs are more frequently bilateral and more audible in the chest and lower neck than in the mid to upper neck [6]. Patients on dialysis can also have cervical bruits that radiate into the neck secondary to arteriovenous fistula placement [7].

Fell et al. [8] reported on 100 patients with 165 asymptomatic carotid bruits. Duplex scanning showed a normal internal carotid artery in 12 cases (7%), <50% stenosis in 83 (50%), ≥50% stenosis in 61 (37%), and occlusion in 9 (6%). Thus, although the majority of neck bruits were associated with some degree of carotid stenosis, only 43% had ≥50% stenosis, which may justify further work-up in selected patients.

Once a neck bruit is determined to be carotid in origin, the next stage is to determine whether this bruit is asymptomatic or symptomatic. This can be determined by the presence or absence of TIA symptoms or stroke as indicated earlier. It should be noted that symptoms referable to the contralateral carotid artery, even in the absence of bruit on that side, may prompt evaluation of patient for symptomatic carotid stenosis on the contralateral side. Carotid bruit may also be absent in patients with severe carotid stenosis in 20–35% of patients [9]. To be noted, a previously audible bruit may disappear if carotid stenosis progresses to severe/tight stenosis or occlusion. Carotid bruits can also originate from the external carotid artery or in patients with an occluded internal carotid artery because of increased blood flow in the external carotid artery.

Several studies have analyzed the significance of carotid bruits. In a subset analysis of the NASCET trial in which the presence of carotid bruits was compared with angiographic imaging of the carotid system, the presence of focal ipsilateral carotid bruits had a sensitivity of 63% and a specificity of 61% for high-grade carotid stenosis (70–99%), and the absence of a bruit did not significantly change the probability of significant stenosis in this group of patients (pretest 52%, posttest 40%) [10]. Ratchford et al. found in a diverse group of patients that the sensitivity of bruit auscultation was low at 56%, with a specificity of 98%, a PPV of 25%, and a NPV of 99% [11]. They concluded that prevalence of carotid bruits is low in the general population. Also, if bruit is heard in an asymptomatic patient, 25% will have a >60% stenosis; however the ability to predict plaque by ultrasound was 89%.

The absolute risk of stroke is generally increased in the presence of carotid bruit. In population-based studies, the annual risk of stroke was 2.1% (95% confidence interval [CI], 0.6–8.5) for persons who had a carotid bruit versus 0.86% (95% CI, 0.8–0.9) for those who did not [12–14]. This represents an absolute risk increase for stroke of 1.24% a year and a relative risk for stroke of 2.4. The presence of a carotid bruit remained an independently significant variable, with a relative risk of 2.0, even after adjustment for various risk factors, including hypertension, age, and sex [12].

Carotid bruits have also been shown in meta-analysis to be a useful indicator of systemic atherosclerosis and a prognostic indicator of myocardial infarction and death [15]. MI and death occurred twice as often in patients with versus patients without carotid bruits [15].

With these facts in mind, and given the low absolute risk of stroke in patients with asymptomatic carotid bruits, and the low prevalence of surgically relevant significant carotid stenosis in these patients with a relatively small absolute benefit of CEA, most clinicians pursue further investigations only in patients who carry a high risk of carotid stenosis and

stroke and who also carry a low operative risk for CEA [16, 17]. Further evaluation in these patients should only be done if the patients prefer to undergo carotid intervention, whether CEA or stenting; otherwise no further evaluation is indicated.

As indicated earlier, the ACAS study concluded that patients with ≥60% stenoses who were treated medically had a higher stroke rate over a 5-year period in comparison to patients who underwent a CEA. Figure 20.1 summarizes a practical approach in patients with asymptomatic carotid bruits or non-hemispheric symptoms. After the initial step of carotid DUS, if <60% stenosis was detected, it is recommended that the test be repeated in 6–12 months.

If stenosis of ≥60–99% was detected and the patient is a good risk, a magnetic resonance angiogram/CTA can be done to complement the findings of the ultrasound, and if confirmed, a CEA is recommended for patients at high risk for stroke. If the magnetic resonance angiogram/CTA was not conclusive or contradicted the ultrasound findings, then angiography may be considered in centers with a minimal stroke risk rate from angiography. However, in patients with a good-quality carotid DUS, an endarterectomy may be considered based on ultrasound findings only. Several authorities would not recommend CEA in asymptomatic patients unless carotid stenosis exceeds 70–80%. In patients who had a good-quality ultrasound showing total occlusion, no further follow-up is needed. However, if the quality of the ultrasound was limited, a magnetic resonance image/CTA is recommended to confirm occlusion.

Another indication for studying asymptomatic patients is to screen patients with advanced coronary artery disease or peripheral vascular diseases. Due to the diffuse nature of atherosclerosis, many of these patients have occult carotid bifurcation lesions with a resulting increased risk of stroke. This type of screening is carried out most often in patients who are being considered for cardiac or major peripheral arterial operations in order to detect carotid stenoses that may substantially increase the risk of intraoperative and postoperative stroke. Carotid screening is covered in depth in Chap. 9.

Role of Carotid Imaging in Patients with Atypical or Non-hemispheric Symptoms

Patients with atypical or non-hemispheric symptoms often do not have a clear indication for angiography. Some of these patients' symptoms include dizziness, blackouts, bilateral visual disturbances, or bilateral motor or sensory deficits. Since a variety of nonvascular causes, such as orthostatic hypotension, cardiac arrhythmias, and medications, may be responsible for these symptoms, noninvasive carotid testing is important in identifying these patients with hemodynamically significant carotid stenosis. Our management protocol for this group of patients is outlined in Fig. 20.1.

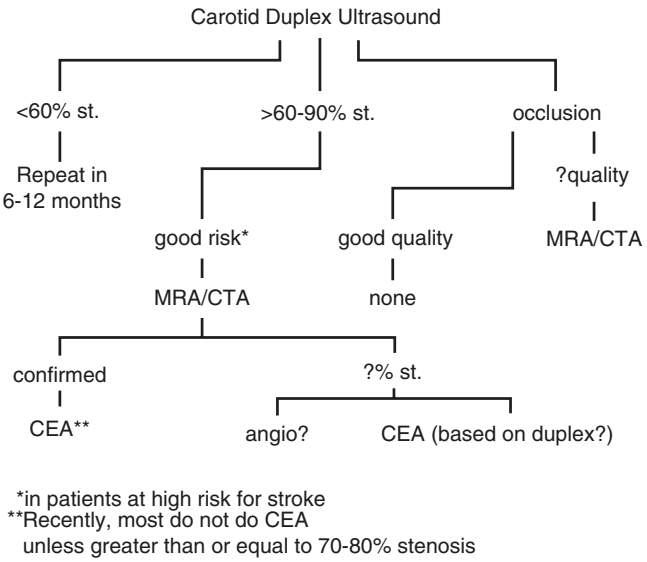


Fig. 20.1 Management protocol for patients with asymptomatic carotid bruit or non-hemispheric symptoms. *MRA* magnetic resonance angiography, *CEA* carotid endarterectomy, *st.* stenosis

Role of Carotid Imaging in Patients with Focal Neurologic Deficits (Transient Ischemic Attacks or Strokes)

A good proportion of transient ischemic attacks (TIAs) or permanent focal neurologic deficits in hemispheric distribution or with amaurosis fugax is caused by embolization from ulcerations and atheromatous plaques. Therefore, the purpose of carotid screening in patients with hemispheric neurologic symptoms is to identify lesions that could be the source of cerebral emboli or could reduce cerebral hemispheric blood flow. In the North American Symptomatic Carotid Endarterectomy Trial (NASCET) study [18], carotid endarterectomy was highly beneficial for patients with recent (<3–6 months) hemispheric TIAs or mild strokes and >70–99% and 50–69% stenoses of the ipsilateral internal carotid artery. Based on these results, patients with symptoms of severe stenoses of the carotid artery should be treated by CEA unless their medical condition makes the risk of surgery prohibitive where carotid artery stenting (CAS) may be considered. Our management protocol for this group of patients is outlined in Fig. 20.2. As noted in Fig. 20.2, the initial step is to obtain a color DUS, and if the study is diagnostic and shows <50% stenosis, the patient is treated medically (e.g., antiplatelet therapy, statins, risk modifications, etc., and repeat color DUS in 12 months). If the stenosis is ≥50%, the ultrasound is of good quality, and the patient has classical focal hemispheric symptoms, a CEA can be done

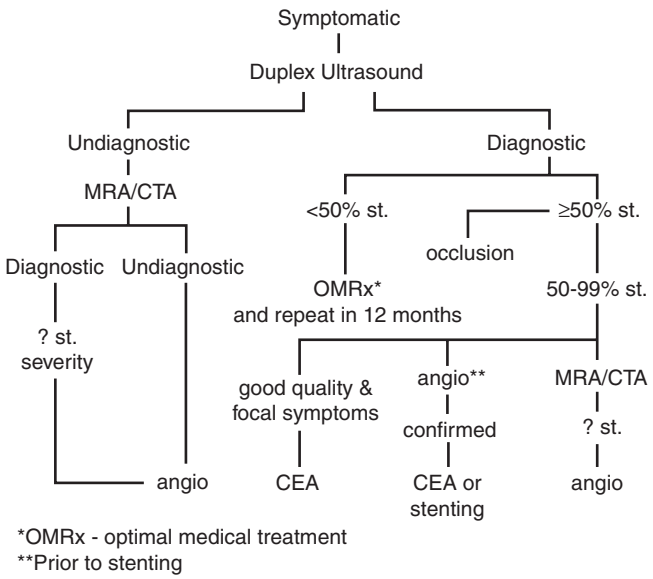


Fig. 20.2 Management protocol for patients with suspected hemispheric symptoms (cerebrovascular disease). For <50% stenosis, repeat ultrasound in 12 months. *MRA* magnetic resonance angiography, *CEA* carotid endarterectomy, *OMRx* optimal medical treatment, *st.* stenosis

based on the carotid DUS findings alone; or MRA/CTA can be done to complement the ultrasound findings, and if the diagnosis is confirmed, surgery may be considered without angiography. Angiography is reserved for patients with a marginal quality DUS or CTA/magnetic resonance angiogram or in patients with contradictory magnetic resonance angiogram and DUS results. If the DUS shows total occlusion and the ultrasound was of good quality, no further workup is usually necessary. For patients with a DUS that is not diagnostic, magnetic resonance angiogram/CTA is done, and if it is diagnostic and the severity of stenosis is established, surgery can be done accordingly. If the MRA/CTA is not diagnostic, angiography is recommended.

Specific Duplex Criteria for Specific Clinical Situations

In choosing our criteria for peak systolic velocity and end diastolic velocity, we chose the values that gave the highest overall accuracy. However, which criteria to use should depend on the "outcome" desired by the clinician. Although some surgeons have advocated CEA based on duplex criteria alone [3, 19, 20], the decision to proceed with an arteriogram is based on the duplex findings in the majority of patients. The mortality and morbidity of arteriography vary from institution to institution but can be significant [4, 21]. We propose that vascular laboratories

Table 20.1 Selected optimal criteria with best PPV (≥95%) and overall accuracy in detecting ≥60–99% and 70–99% ICA stenosis

Best PPV	PPV (%)	Overall accuracy (%)	Sensitivity (%)	Specificity (%)	NPV (%)
For ≥60% ICA stenosis					
ICA PSV ≥220 cm/s	96	82	64	98	76
ICA EDV ≥80 cm/s	96	87	79	97	84
ICA/CCA PSV Ratio ≥4.25	96	71	41	99	65
ICA PSV & EDV 150 & 65[a]	96	90	82	97	86
For ≥70% ICA stenosis					
ICA PSV ≥300 cm/s	97	80	48	99	76
ICA EDV ≥110 cm/s[a]	100	91	75	100	87
ICA/CCA PSV ≥ none	–	–	–	–	–
ICA PSV & EDV 150, 110[a]	100	91	75	100	87

[a]These values have the best PPV and overall accuracy

at institutions with significant mortality and morbidity in relation to carotid arteriography use duplex criteria with 95% or greater PPV and the best overall accuracy in order to minimize the number of patients undergoing unnecessary arteriography (Table 20.1). These criteria can also be utilized when CEA is performed without preoperative arteriography. In those institutions where arteriography does not significantly add to the mortality and morbidity of the overall treatment of carotid disease, we suggest using the criteria described in Table 20.2. These criteria have the highest negative predictive value to ensure that only a minimum number of patients with equal to or greater than 60% or 70% stenoses are missed.

A duplex classification was proposed by us which would consist of lesions <30% stenosis, ≥30–49% stenosis, ≥50–59% stenosis, ≥60–69% stenosis, and ≥70% stenosis. This duplex classification would fit into the existing trials [NASCET and ACAS] and may be of benefit as new conclusions are released [22]. By reporting results using these criteria, the clinician will be better able to make decisions regarding the need for CEA or arteriogram based on the risks and benefits for individual patients. With the added risks of arteriography, decisions to operate would be better based on duplex findings alone. Having PPVs of 90–97% and accuracies of 87–93% can eliminate many unnecessary arteriograms. For those vascular laboratories who may be using the

Table 20.2 Selected optimal criteria with best NPV (≥95%), and overall accuracy in detecting ≥60–99% and 70–99% ICA stenosis

Best PPV	NPV (%)	Overall accuracy (%)	Sensitivity (%)	Specificity (%)	PPV (%)
For ≥60% ICA stenosis					
ICA PSV ≥135 cm/s[a]	99	80	99	64	71
ICA EDV—none	–	–	–	–	–
ICA/CCA PSV Ratio ≥1.62	95	71	97	47	62
ICA PSV & EDV—none	–	–	–	–	–
For ≥70% ICA stenosis					
ICA PSV >150 cm/s[a]	99	80	99	69	65
ICA EDV ≥60 cm/s	96	83	94	77	71
ICA/CCA PSV ≥ none	–	–	–	–	–
ICA PSV & EDV—none	–	–	–	–	–

[a]These values have the best NPV and overall accuracy

consensus duplex criteria, specific velocities can be used accordingly, as indicated in Chap. 19.

It is important to note that the data obtained by individual vascular laboratories will vary as a result of differences in equipment, abilities, and consistencies of vascular technicians and reader interpretations [22]. Therefore, each laboratory must adapt a method that employs the equipment they use and has validated their method when using proposed new duplex criteria.

Intraoperative Duplex Ultrasound Assessment of Carotid Endarterectomy

Intraoperative use of the B-mode ultrasound imaging system for completion evaluation of the CEA has been advocated by Sigel et al. [23]. The development of smaller scanning heads and probes together with techniques of sterilization has made this application feasible. The ultrasound examination can be performed quickly and, unlike angiography, requires no delay for film processing. Nor is it necessary to inject contrast material. Angiography is also associated with the risks of subintimal injections, thromboembolic complications, and allergic reactions.

Despite careful operative techniques, certain vascular defects can be missed, e.g., intimal flaps, luminal thrombus/platelet aggregation, stricture, etc., that occur in the course of carotid repair. These defects can escape visual inspection and palpation of the repair. If these defects are left undetected, they can result in stroke secondary to thrombus formation, platelet aggregation, or arterial thrombosis; or they may result in postoperative recurrent carotid stenoses [24–31].

Most authorities rely on imaging or Doppler flow detection technique to exclude technical defects. The diagnostic signal analysis is highly sensitive and specific (>90%), particularly if pulse Doppler analysis is performed. This technique is simple, widely available, and relatively inexpensive. Although abnormalities of the Doppler flow signal are readily apparent by audible interpretation, quantitative spectral analysis is preferable. With flow and pressure reducing lesions, a spectral broadening is present throughout the pulse cycle, and a peak systolic velocity (PSV) exceeding 150 cm/s is noted. Visual inspection of velocity spectra and calculation of PSV can be obtained by a high-frequency pulse Doppler probe or duplex scanning, which permits classification of flow patterns into three categories: normal flow, mild to moderate flow disturbance, and severe flow disturbance [25]. When a significant residual flow abnormality is identified, angiography is usually recommended to delineate the abnormality before reexploration of the repair.

Intraoperative duplex ultrasonography has been advocated because of its ability to provide both anatomic and hemodynamic information [29–33]. Improvements in linear ray scan head design and electronic signal processing, including color-coded velocity display, have made duplex scanning feasible in the operating room and an ideal modality for intraoperative assessment of CEA. Duplex scanning has an advantage over Doppler flow analysis alone, in that the structure of the anatomic defects associated with severe flow disturbance can usually be determined. B-mode imaging is sensitive in detecting small intimal defects in flaps; however, most authorities have not repaired these minor lesions, and the outcome of the procedure has not been adversely influenced [31].

A comparison of intraoperative and early postoperative duplex findings after CEA indicated that a majority of these abnormalities identified by duplex scanning within 3–6 months of CEA represent residual rather than recurrent stenoses [30].

Color duplex scanning with a 7.5- to 10-MHz linear ray transducer has been used for intraoperative studies. These studies are conducted with the transducer covered by a sterile disposable plastic sleeve that contains acoustic gel (Fig. 20.3). The probe is generally positioned in the cervical incision directly over the exposed carotid repair. A sterile solution is instilled into the incision for acoustic coupling. As the surgeon scans the arterial repair, the technologist adjusts the instrument to optimize the Doppler angle, sample

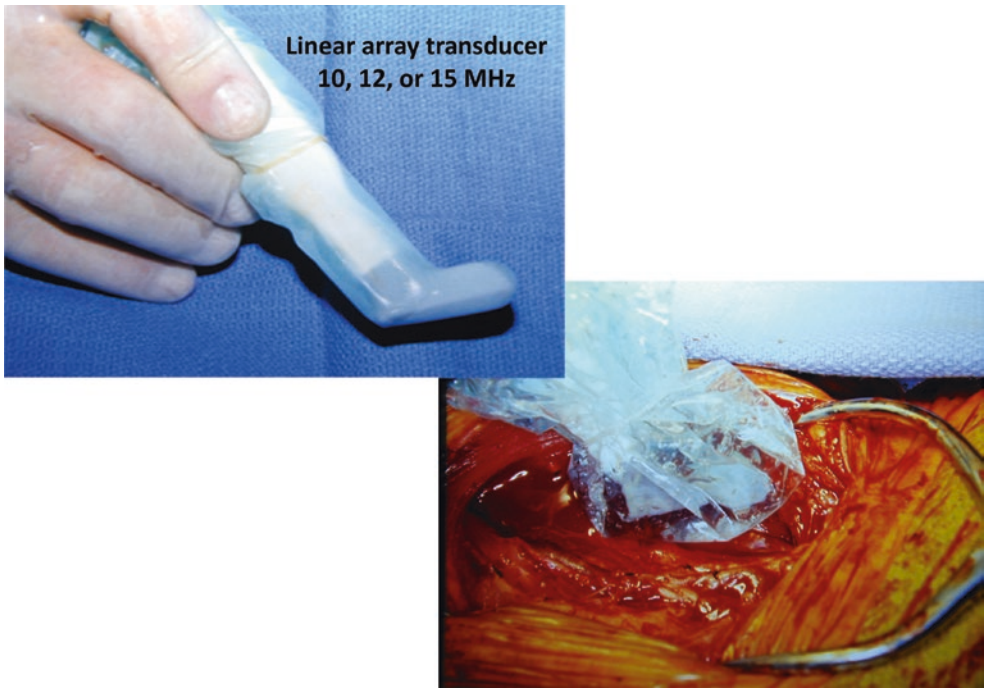


Fig. 20.3 A "hockey stick" linear array (10–15 MHz) transducer suitable for intraoperative carotid repair site imaging. The small-footprint transducer is enclosed in a sterile plastic sheath for scanning within the neck incision directly on the carotid endarterectomy site

volume, color-coded image, and recorded velocity spectra. Vessel walls are imaged at 90°, but blood flow patterns should be evaluated at Doppler angles of <60°.

For CEAs with primary closure, the entire CEA segment should be examined with duplex ultrasound. The point in the CCA at which the lesion is transected should be examined. Normally, this should leave a distinct shelf, which can be appreciated on B-mode imaging. This can be easily visualized in both transverse and longitudinal views. The velocity data proximal to, in, and distal to the endarterectomy site should also be done in longitudinal view, and sampling of the PSVs in the endarterectomy site should be obtained. Similarly, scanning of the proximal ICA in the bulb and beyond it should be done, and attention should be called to the point of the transaction of the plaque or the end of the plaque distally. The external carotid artery should also be examined for the first few centimeters, looking for residual plaques or areas of thrombus. In patients who have CEAs closed with a patch, either polytetrafluoroethylene (PTFE) or Dacron, it is impossible to scan through the patch itself because of the air within the wall of the patch. However, it is possible to scan along the side of the artery, either posterior or anterior to the patch, which may yield the necessary information (Figs. 20.4 and 20.5).

The limitations of this technique are largely related to lack of experience, correct measurement of duplex derived flow velocities, recognition of abnormal flow patterns, and

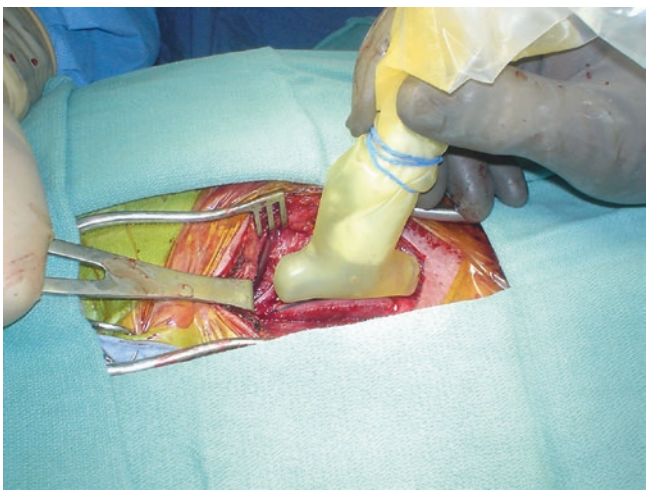


Fig. 20.4 Probe scanning position for carotid endarterectomy closed with a patch. It is impossible to scan through PTFE patch, but operator can scan along the side of artery, either posterior or anterior to the patch

transducer size. Intraoperative duplex imaging has the following advantages over angiography: comparable or higher accuracy, safety, ease of repeated use after reexploration, and low cost. Color duplex scanning is also sensitive to variations in anatomy and minor vascular defects that may alter blood flow streamlines. Certain flow patterns produced by carotid patch angioplasty should be noted and should not be regarded as abnormal. Some authorities have reported vascular defects in as many as one-third of their repairs, but only

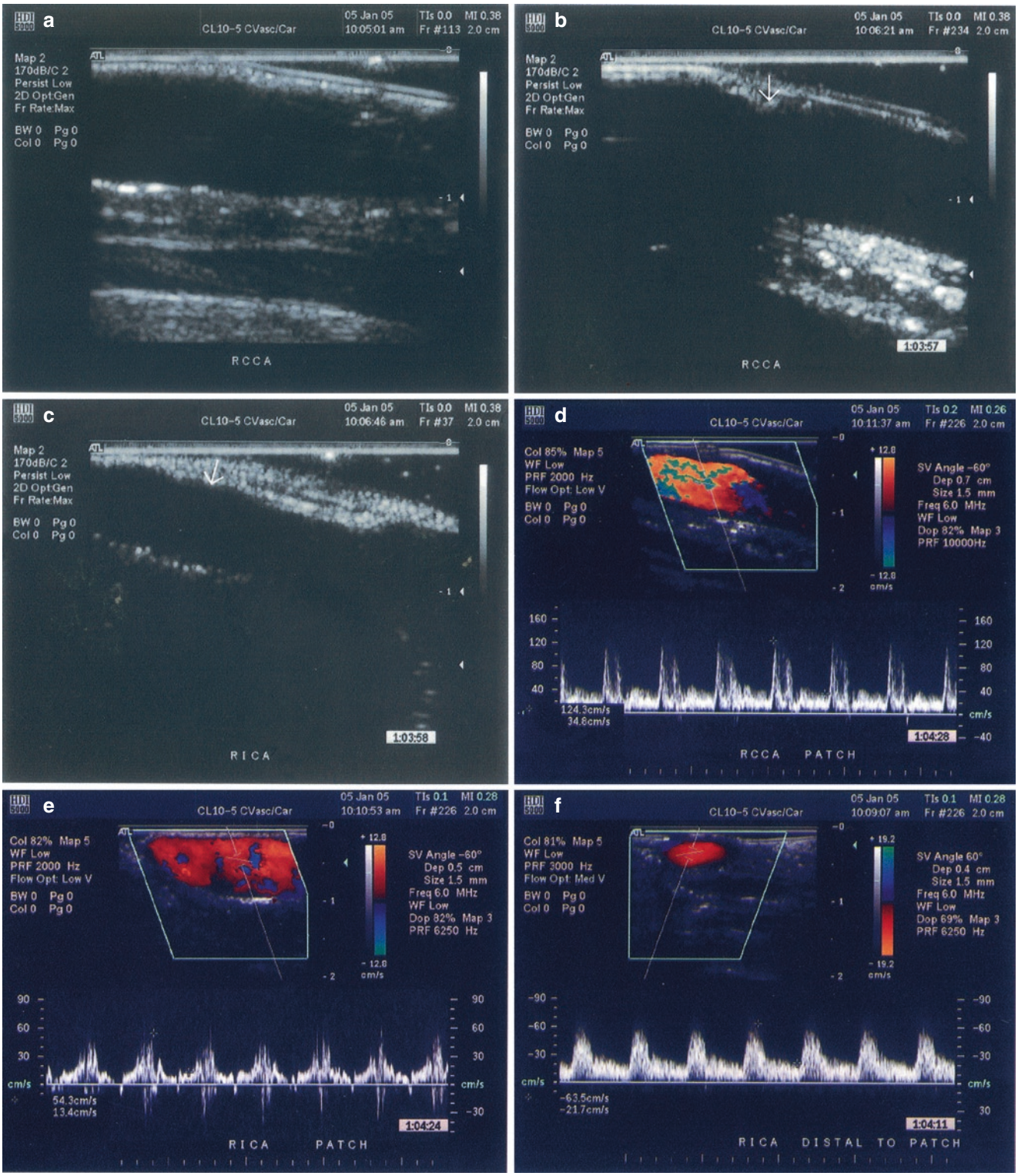


Fig. 20.5 Intraoperative duplex ultrasound of carotid endarterectomy: (**a**) common carotid artery in gray scale. Note the shelf of the proximal end of carotid endarterectomy (*arrow*), (**b**) common carotid artery bifurcation in gray scale (*arrow*), (**c**) internal carotid artery in gray scale, (**d**) common carotid artery with color flow, (**e**) internal carotid artery with patch with color flow, (**f**) distal internal carotid artery with color flow

one-third of these appear to justify reexploration. Further details can be seen in Chap. 11.

Intraoperative Monitoring of Carotid Endarterectomy with Transcranial Doppler Sonography

Transcranial Doppler (TCD) sonography has the advantage of allowing monitoring of both hemodynamic and embolic events, primarily in the middle cerebral artery distribution during CEA. One of the common uses of TCD is intraoperative monitoring to determine whether shunting is necessary and whether the shunt is malfunctioning [34]. TCD can be a useful indicator that early carotid clamping is necessary if multiple emboli were detected [34]. TCD can also be helpful in patients where shunts are not being used since a TCD signal will give an idea of the flow through the middle cerebral artery. The middle cerebral artery cannot be insonated in 5–15% of patients, most commonly because of the lack of a window for Doppler signal penetration of the skull. Severe cerebral ischemia is considered present in the first minute after carotid occlusion if the middle cerebral artery velocity decreases to 15% of the baseline or lower and mild ischemia if it drops to 15–40% of the baseline. An adequate perfusion is present if the velocity is >40% of the baseline [35]. Following insertion of the shunt or upon de-clamping, a brisk recovery in middle cerebral artery velocity should be seen, usually >80%. Absolute mean velocities of 15 cm/s or even 30 cm/s have been alternatively suggested. A middle cerebral artery velocity of 30cm/s has correlated roughly with a carotid artery stump blood pressure of 50 mmHg. Some authorities reported that TCD detects critically low flow that results in neurologic deficits, even in the absence of electroencephalographic changes. The converse is also true: a pronounced drop in mean velocity has been observed in conjunction with a normal EEG and no resultant cerebral infarction, the cortex surviving from the other cerebral and leptomeningeal vessels.

In a recent study, Ackerstaff et al. [36] concluded that in CEA, TCD-detected microemboli during dissection and wound closure, ≥90% middle cerebral artery velocity decrease at cross-clamping, and ≥100% pulsatility index increase at clamp release are associated with operative stroke. In combination with the presence of preoperative cerebral symptoms and ≥70% ipsilateral ICA stenosis, these four TCD monitoring variables can reasonably discriminate between patients with and without operative stroke. This supports the use of TCD as a potential intraoperative monitoring modality to alter the surgical technique by enhancing a decrease of the risk of stroke during or immediately after the operation.

TCD can also be used in the postoperative period to detect early thrombosis of the carotid artery, continued embolization, or the hyperperfusion syndrome. Another useful indication for TCD monitoring is in the early postoperative period since more than one-half of patients develop emboli in the first 3 hours after carotid endarterectomy [37] and a majority of these will stop without further treatment; however if the TCD indicates an increasing number of these emboli, treatment may be necessary (e.g., dextran infusion). TCD can also be useful in postoperative monitoring by measuring the middle cerebral artery velocities. If these velocities decrease, it may indicate compromising of the carotid endarterectomy site; meanwhile increasing velocities may be indicative of hyperperfusion syndrome. Presently, there is no level 1 evidence that TCD is essential in the routine practice of carotid artery surgery.

It has been reported that markedly increased mean velocity (150% of the baseline) may herald an intracranial hemorrhage. The use of TCD monitoring during CEA has led some surgeons to modify their operative techniques based on hearing a distressing frequency of emboli while operating with the continuously audible TCD. Further details on TCD are discussed in Chap. 15.

Role of Carotid Imaging in Patients Who Develop a Neurologic Deficit After Leaving the Operating Room

If patients wake up well after CEA and then develop a neurologic deficit, emergent reexploration is indicated. If the deficit proves to be a TIA as symptoms resolve prior to the return to the operating room, heparin anticoagulation followed by immediate duplex scan is preferred. A thrombosed ICA may be treated operatively or medically (anticoagulation), particularly in patients with dense deficits. A patent carotid without apparent pathology is immediately followed by brain CT/CTA to identify intracranial hemorrhage or other pathology and assess the intracranial vasculature. If negative for hemorrhage, oral anticoagulation is started. Thromboembolism of inaccessible intracranial vasculature may be treated with selective catheterization and lytic therapy. Blood clots found at the endarterectomy site are treated by emergency reexploration.

Post-carotid Endarterectomy Surveillance

Restenosis is a known entity that occurs after CEA and may vary between 12 and 36%, but the frequency of restenosis varies depending on the diagnostic method used and the frequency of follow-up examinations [38–48]. Several studies have reported on the value of postoperative carotid

duplex surveillance, but no consensus has been reached [38–47]. The advantages that have been cited are detection of significant restenosis prior to the onset of neurologic events, which aids in the prevention of potential strokes, and follow-up on the contralateral carotid artery to document the development of surgically correctable stenosis. Opponents of routine postoperative carotid duplex surveillance claim that restenosis is benign in nature; therefore, a large number of strokes may not be prevented by surveillance [40, 41, 43, 44, 47]. Despite the high rate of restenosis, symptoms attributed to restenoses are rare; therefore several authorities have suggested that routine surveillance of patients after CEA is not efficacious [39, 41, 44].

Several factors were associated with restenosis: continued smoking, small internal carotid artery diameter, operative defect detected at intraoperative assessment, and primary closure after CEA. Moore et al. [49] prospectively determined the incidence of restenosis using Doppler ultrasound follow-up to 5 years in ACAS patients who underwent CEA. The aggregate incidence of residual and recurrent carotid stenosis for all time intervals was 13%. Early restenosis (<2 years) in this group of patients was found in 8% and late restenosis in 2%. Of the 136 patients who were felt to have restenosis, only 8 (5.9%) underwent reoperation, only one of whom was for symptoms. There was also no correlation between late stroke and recurrent stenosis. Similarly, Cao et al. [50] randomized 1353 who underwent CEA using the eversion technique (678) or standard CEA (primary closure in 419 and patch closure in 256). The life table estimate of the cumulative risk of restenosis at 4 years was 4% in the eversion CEA group and 9% in the standard CEA group. Almost all of these patients (98%) were asymptomatic.

Mattos et al. [41] described their experience with postoperative carotid duplex surveillance and found an equal stroke-free survival at 5 years between patients with or without >50% restenosis. In addition, only 1 of 380 patients suffered a stroke in their study, suggesting a benign clinical significance of recurrent carotid artery stenosis. Mackey et al. claim a low rate of clinically significant restenosis [40]. Their retrospective series of 258 patients (348 arteries) show a potential 4% incidence of late strokes, but this included all patients who underwent repeat CEA for asymptomatic restenosis. They also noted that the majority of restenoses (53%) remained asymptomatic and did not progress to occlusion throughout follow-up. Of ten documented late occlusions, eight did not result in stroke. Eight patients with operable restenosis had TIAs and underwent reoperation. They found that even patients with 75–99% restenosis most often remained asymptomatic (37%) or had TIAs (32%). Only 2 (11%) of 19 patients with 75–99% restenosis had an unheralded stroke. They felt that postoperative carotid duplex surveillance was not justified due to the low incidence of symptomatic restenosis.

In spite of these findings, investigators have been reluctant to advise that postoperative carotid duplex surveillance be abandoned because the cost-effectiveness of this surveillance has not been formally investigated. Others have reported that high-grade stenosis (>75%), whether caused by myointimal hyperplasia of the CEA site or progressive atherosclerosis of the contralateral carotid artery, is associated with an increased risk of late stroke [28, 47].

Ouriel et al. reported an 11% incidence of restenotic lesions greater than 80%. Although the incidence of symptoms with restenotic lesions was low (12%), the onset of symptoms at the time of occlusion was significant [42]. Forty-two percent of patients became symptomatic at the time of occlusion, with 33% resulting in a stroke. This led to the observation that critical restenoses are precursors to stroke, even if asymptomatic, and, therefore, the detection of >80% restenosis allows future stroke prevention, if operative intervention is undertaken [42]. Mattos et al. also described the outcome for >80% restenosis. In their group, one of three patients with >80% restenosis suffered a stroke, one had a TIA, and one remained asymptomatic. This suggests a more serious course once restenosis reaches >80% [41].

Recently, Kallmayer et al. reported on ultrasound surveillance after CAS and CEA. They reviewed multicenter randomized controlled trials (RCTs) published between 1990 and 2013 in regard to DUS surveillance intervals, recurrent ipsilateral stroke rates, and recurrent stenoses rates. They also performed a Medline literature search from January 1990 to February 2014 using the key words: carotid endarterectomy, carotid stenting, surveillance, carotid artery surveillance, and carotid artery stenosis. They also analyzed carotid-related guidelines published between 2006 and 2013 for recommendations on DUS surveillance after CAS or CEA. Nine RCT protocols (NASCET, ECST, ACST, ACAS, CAVATAS, SAPPHIRE, EVA-3S, CREST, and SPACE) showed similar follow-up intervals (at 1 month, at 3 or 4 and at 6, and at 12 months after CEA and CAS and then annually). The recurrent carotid stenosis (≥50%) or occlusion rates were 6% at 4 years after CAS or CEA. The annual ipsilateral cerebral ischemic event rate was about 1% and 0.5% after CEA for symptomatic or asymptomatic stenosis, respectively. Since the overall carotid restenosis and postprocedural stroke rates were low, the necessity of DUS was questioned in post-CAS and post-CEA patients in prospective single-center series. However, some patients (women, diabetics, patients with dyslipidemia, smokers) may have increased restenosis rates after CAS or CEA. They also felt that data on DUS surveillance intervals following CAS is rare. Long-term DUS surveillance was only recommended in 3 out of 21 identified guidelines since the benefits were considered to outweigh the risks. However, the level of evidence for any recommendation on DUS surveillance was consistently low. They concluded that their literature review

revealed little evidence to support routine DUS after CEA within short intervals. They felt that one periprocedural DUS and one DUS after 12 months after CEA seems to be reasonable. However, further DUS surveillance seems appropriate in patients with ipsilateral restenosis ≥50% and contralateral disease progression ≥50% and in patients who are considered high risk for restenosis. Since the long-term data on surveillance after CAS was inconsistent, imaging at 6 months and then annually seems reasonable [51].

So far, a consensus has not yet been reached in the surgical literature regarding the usefulness, cost-effectiveness, or timing of postoperative carotid duplex surveillance.

Timing of Postoperative Carotid Duplex Surveillance

Several authors have recommended an initial surveillance duplex on the operative carotid system within the first 6 months [38, 41–43, 47] to detect residual stenosis from the operative procedure or early restenosis [42]. Roth et al. [47] recommended an initial DUS to ensure a technically successful CEA, with subsequent postoperative carotid duplex surveillance at 1–2 years, as long as restenosis and contralateral stenoses remain <50%. More frequent follow-up (every 6 months) is warranted if >50% stenosis is noted or with the onset of symptomatic disease [47].

Several studies have reported that the majority of restenoses occurs during the first 1–2 years after CEA. Mattos et al. [38] noted that 70% of restenoses were detected within 1 year after the CEA and 96% developed within 15 months. Similar observations were noted by us previously [45].

Ricco et al. [52] reported on the need for follow-up duplex scan 1 year after CEA was performed with prosthetic patching and intraoperative completion arteriography. A total of 605 CEA procedures with prosthetic patch closure and intraoperative completion arteriography were performed in 540 patients. All patients underwent duplex scan at 4 days and then yearly after the procedure. Intraoperative completion arteriography showed abnormalities in 114 cases, including 17 involving the ICA and 73 involving the external carotid artery. Successful revision was achieved in all cases and confirmed by repeat arteriography. Postoperative duplex scans at 4 days detected three abnormalities involving the ICA (0.5%), including asymptomatic occlusion in one case and residual stenosis >50% in two cases. Ninety-eight percent of patients were stenosis-free at 1 year. Actuarial stroke-free survival was 98.3% at 3 years. Diameter reduction of the contralateral carotid artery progressed over 70% within 1 year after CEA in 22.9% of patients with contralateral carotid stenosis over 50% at the time of the initial intervention. The findings of this study indicate that duplex scan follow-up 1 year after CEA with intraoperative completion arteriography is unnecessary unless postoperative duplex scan demonstrates residual stenosis of the ICA. However, duplex scan at 1 year is beneficial for patients presenting with contralateral carotid artery disease with diameter reduction >50% at the time of CEA.

Lovelace et al. [53] conducted a study on optimizing duplex follow-up in patients with an asymptomatic ICA stenosis of <60%. All patients who underwent initial carotid duplex examination for any indication since January 1, 1995, with at least one patent, asymptomatic, previously non-operated ICA with <60% stenosis, with 6 months or greater follow-up, and with one or more repeat duplex examinations were entered into the study. On the basis of the initial duplex examination, ICAs were classified into two groups: those with a PSV <175 cm/s and those with a PSV of 175 cm/s or more. Follow-up duplex examinations were performed at varying intervals to detect progression from <60% to 60–99%. A total of 407 patients (640 asymptomatic ICAs with <60% stenosis) underwent serial duplex scans (mean follow-up, 22 months). Three ICAs (0.5%) became symptomatic and progressed to 60–99% ICA stenosis at a mean of 21 months, whereas four other ICAs occluded without stroke during follow-up. Progression to 60–99% stenosis without symptoms was detected in 46 ICAs (7%) (mean, 18 months). Of the 633 patent asymptomatic arteries, 548 ICAs (87%) had initial PSVs <175 cm/s, and 85 ICAs (13%) had initial PSVs of 175 cm/s or more. Asymptomatic progression to 60–99% ICA stenosis occurred in 22 (26%) of 85 ICAs with initial PSVs of 175 cm/s or more, whereas 24 (4%) of 548 ICAs with initial PSVs <175 cm/s progressed ($p < 0.0001$). The Kaplan–Meier method showed freedom from progression at 6 months, 12 months, and 24 months was 95%, 83%, and 70% for ICAs with initial PSVs of 175 cm/s or more versus 100%, 99%, and 95%, respectively, for ICAs with initial PSVs <175 cm/s ($p < 0.0001$).

They concluded that patients with <60% ICA stenosis and PSVs of 175 cm/s or more on initial duplex examination are significantly more likely to progress asymptomatically to 60–99% ICA stenosis and progression is sufficiently frequent to warrant follow-up duplex studies at 6-month intervals. Patients with <60% ICA stenosis and initial PSVs <175 cm/s may have follow-up duplex examinations safely deferred for 2 years.

Cost-Effectiveness of Postoperative Carotid Duplex Surveillance

There have been reports that postoperative carotid duplex surveillance is not cost-effective since there is such a low incidence of symptomatic restenosis. Patel et al. evaluated the cost-effectiveness of postoperative carotid duplex surveillance [46]. They concluded that postoperative carotid duplex surveillance after CEA has an unfavorable cost-effectiveness ratio. In the process of their analysis, they identified a subset of patients

in which postoperative carotid duplex surveillance may be cost-effective. These included patients in whom the rate of progression to >80% stenosis exceeded 6% per year. In their analysis, they felt that some groups of patients could potentially have a rate of disease progression that approaches or exceeds the level at which postoperative carotid duplex surveillance becomes cost-effective. Some of these include patients with multiple risk factors, e.g., smoking, hypertension, hyperlipidemia, diabetes mellitus, coronary artery disease, female gender, and a young age. In addition, they concluded that with postoperative carotid duplex surveillance, the rate of carotid artery occlusion could be reduced by 15% per year.

Our evaluation of the cost of postoperative carotid duplex surveillance agrees with these conclusions [54]. Three hundred and ninety-nine CEAs were randomized into 135 with primary closure, 134 with PTFE patch closures, and 130 with vein patch closures and followed for a mean of 47 months. Postoperative carotid duplex surveillance was done at 1, 6, and 12 months and every year thereafter (a mean of 4.0 studies/artery). A Kaplan–Meier analysis was used to estimate the rate of ≥80% restenosis over time and the time frame of progression from <50% to 50–79% and ≥80% stenosis. Greater than or equal to 80% restenosis developed in 24 (21%) with primary closure and 9 (4%) with patching. A Kaplan–Meier estimate of freedom from 50 to 79% restenosis at 1, 2, 3, 4, and 5 years was 92%, 83%, 72%, 72%, and 63% for primary closure and 99%, 98%, 97%, 97%, and 95% for patching. A Kaplan–Meier estimate of freedom from ≥80% restenosis at 1, 2, 3, 4, and 5 years was 92%, 83%, 80%, 76%, and 68% for primary closure and 100%, 99%, 98%, 98%, and 91% for patching ($p < 0.01$).

Out of 56 arteries with 20–50% restenosis, 2/28 patch closures and 10/28 primary closures progressed to 50–<80% restenosis ($p = 0.02$) and 0/28 patch closures and 6/28 primary closures progressed to ≥80% ($p = 0.03$). In primary closures, the median time to progression from <50% to 50–79%, <50% to ≥80%, and 50–79% to ≥80% was 42, 46, and 7 months, respectively. Of the 24 arteries with ≥80% restenosis in primary closures, 10 were symptomatic. Thus, assuming that symptomatic restenosis would have undergone duplex examinations anyway, there were 14 asymptomatic arteries (12%) that could have been detected only by postoperative carotid duplex surveillance (estimated cost of $139,200) and would have been candidates for redo CEA. Of the nine arteries with patch closures (three PTFE and six vein patch closures) with ≥80% restenosis, six asymptomatic arteries (four vein patch closure and two PTFE, 3%) could have been detected by postoperative carotid duplex surveillance. In patients with a normal duplex at the first 6 months, only 4/222 (2%) patched arteries (two asymptomatic) developed ≥80% restenosis versus 5/13 (38%) in patients with abnormal duplex examinations ($p < 0.001$).

Assuming a 5% stroke rate for the 14 repeat CEAs for asymptomatic ≥80% restenosis in the primary closure group in our series [55], 0.7 strokes would be associated with the 14 repeat CEAs, and approximately 4.7 strokes would have been prevented through surgical intervention prior to occlusion (assuming a similar outcome of ≥80% restenosis as described by Mattos et al. [41]). There was a net reduction of four strokes in patients with primary closure and an approximate cost of $56,150 per stroke prevented.

Also, assuming a similar outcome of >80% restenosis as described by Ouriel et al. [42], and if one-half of these >80% restenosis would progress to total occlusion (seven patients), and assuming one-third of patients with total occlusion would suffer a stroke, then approximately 2.3 strokes would be prevented by doing the 14 redo CEAs. Since 0.7 strokes would result from repeating 14 CEAs [55], the net effect would be prevention of 1.6 strokes at a cost of $224,600, i.e., $140,250 per stroke prevented. This analysis does not take into consideration the value of duplex screening of the contralateral non-operated side.

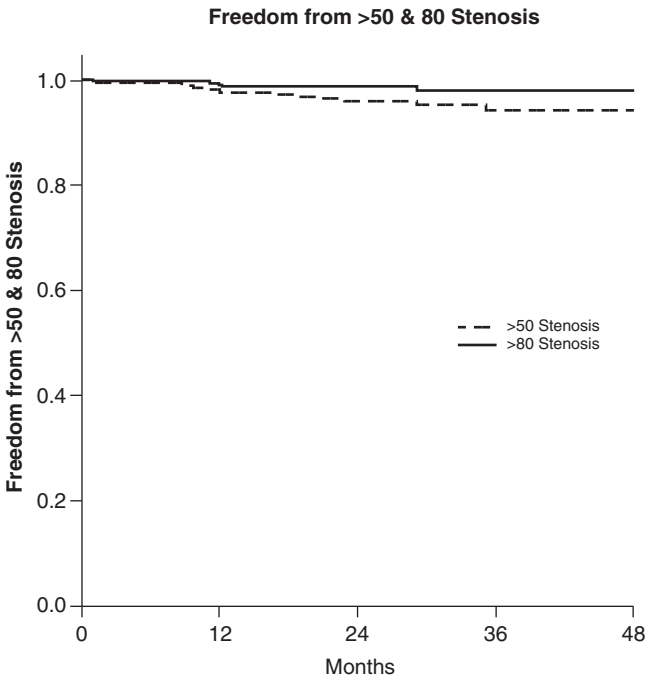
The justification for this cost is unclear without a definite estimate of the economic burden for caring for these stroke victims. Considering the low incidence of >80% restenosis in patients with patch angioplasty closure, the cost-effectiveness of postoperative carotid duplex surveillance appears to be unfavorable and, therefore, should be limited to a single DUS to detect residual stenosis. Subsequent follow-up should be dictated by the results found on the initial scan and the onset of neurologic symptoms.

Our randomized prospective studies confirm that carotid restenosis is a known entity that follows a percentage of patients who undergo carotid surgery. In the past, the clinical significance of carotid restenosis has led some investigators to conclude that postoperative carotid duplex surveillance is not warranted. We showed that based on the incidence of >80% restenosis, postoperative carotid duplex surveillance may be beneficial in patients with primary closure with examinations at 6 months and at 1–2-year intervals for several years. For patients with patching, a 6-month postoperative duplex examination, if normal, is adequate.

The Value and Economic Analysis of Routine Postoperative Carotid Duplex Ultrasound Surveillance After CEA with Patch Closure

We recently analyzed 489 out of 501 patients who underwent CEA with patch closure. Patients had immediate post-CEA duplex ultrasound examinations and were followed routinely both clinically and with duplex ultrasound at regular intervals of 1, 6 and 12 months and annually thereafter. We used a Kaplan–Meier analysis to estimate ≥50% and ≥80% post-CEA restenosis rates. We also estimated the cost of post-CEA duplex surveillance. Ten patients had residual postoperative ≥50% stenosis, and 37 patients didn't undergo a second duplex ultrasound examination, so they were

excluded from the final analysis. The mean follow-up was 20.4 months, and the mean number of duplex ultrasound examinations was 3.6. 11 of 397 patients (2.8%) with a normal finding on immediate postoperative duplex ultrasound versus 4 out of 45 (8.9%) with mild stenosis on immediate postoperative duplex ultrasound progressed to ≥50% restenosis (p = 0.055). Overall, 15 patients (3.1%) had ≥50% restenosis, 9 patients with 50–<80% and 4 with 80–99% (2 of these had carotid artery stenting reintervention), and 2 had late carotid occlusion. All of these patients were asymptomatic, except for one who had a TIA. The mean time to ≥50–<80% restenosis was 14.7 months versus 19.8 months for ≥80% restenosis after the CEA. The freedom from restenosis rates were 98%, 96%, 94%, 94%, and 94% for ≥50% restenosis and 99%, 98%, 97%, 97%, and 97% for ≥80% restenosis at 1, 2, 3, 4, and 5 years, respectively (Fig. 20.6). Freedom from myocardial infarction, stroke, and deaths was not significantly different between patients with and without restenosis (100%, 93%, 83%, and 83% versus 94%, 91%, 86%, and 79% at 1, 2, 3, and 4 years, respectively (p = 0.951, Fig. 20.7)). The estimated charge of this surveillance was 3.6 × 489 (number of CEAs) × $800 (charge for carotid duplex ultrasound), which equals $1,408,320, and the overall reimbursement was 3.6 × 399 × 489 = $702,400 to detect only four patients with ≥80–99% restenosis who may have been potential candidates for reintervention. We concluded that the value of routine postoperative duplex ultrasound surveillance after CEA with patch closure may be limited, particularly if the finding on immediate postoperative duplex ultrasound is normal or shows minimal disease [56].



>50 Stenosis				>80 Stenosis			
	At risk	%	SE		At risk	%	SE
1yr	314	0.981	0.0073	1yr	317	0.992	0.0049
2yr	208	0.962	0.0110	2yr	211	0.988	0.0058
3yr	83	0.945	0.0162	3yr	84	0.981	0.0090
4yr	25	0.945	0.0162	4yr	26	0.981	0.0090

Fig. 20.6 Freedom from ≥50% and ≥80% restenosis rates at 1, 2, 3, and 4 years. From AbuRahma AF, et al. The value and economic analysis of routine postoperative carotid duplex ultrasound surveillance after carotid endarterectomy. J Vasc Surg 2015;62:378–84. Reprinted with permission from Elsevier

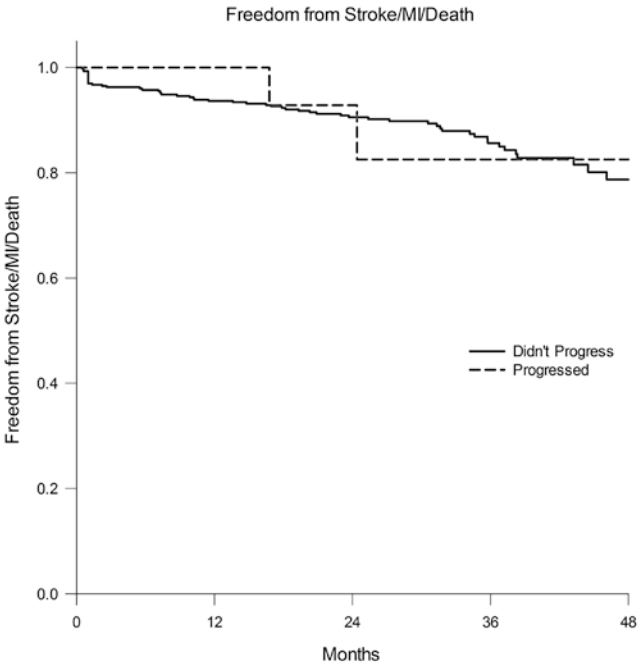


Fig. 20.7 Freedom from MI/stroke/deaths at 1, 2, 3, and 4 years. From AbuRahma AF, et al. The value and economic analysis of routine postoperative carotid duplex ultrasound surveillance after carotid endarterectomy. J Vasc Surg 2015;62:378–84. Reprinted with permission from Elsevier

Proposed Duplex Velocity Criteria for Carotid Restenosis Following CEA with Patch Closure

Duplex ultrasound velocity criteria are used to evaluate the severity of carotid stenosis; however, these standard velocities may not be applicable to carotid restenosis after CEA with patch angioplasty. We conducted a study to determine if patch angioplasty closure alters velocities just distal to CEA and to define optimal velocities for detecting ≥30%, ≥50%, and ≥70% restenosis. Our study included 200 CEAs randomized into 100 with Hemashield Finesse patch and 100 with polytetrafluoroethylene (PTFE) ACUSEAL patch. All patients underwent duplex ultrasounds immediately after

surgery, at 1 month, and every 6 months thereafter. Patients with a PSV of the ICA just distal to the patch of ≥130 c/s had a CTA. PSVs, EDVs, and ICA/common carotid artery (ICA/CCA) ratios were correlated to completion arteriograms/CTAs. ROC curve analyses were done to determine the optimal velocity criteria in detecting ≥30%, ≥50%, and ≥70% restenosis. We analyzed 195 pairs of imagings (duplex ultrasound versus CTA/angiogram). When we applied the standard velocity criteria for non-operated arteries, 37% and 10% of patients were believed to have ≥50–<70% and ≥70–99% restenosis versus 11.3% on CTA/angiography, respectively ($p < 0.001$). The mean PSV for ≥30%, ≥50%, and ≥70% restenosis were 172, 249, and 389 c/s, respectively ($p < 0.001$). An ICA PSV of ≥155 c/s was optimal for ≥30% restenosis with sensitivity, specificity, PPV, NPV, and overall accuracy of 98%, 98%, 98%, 98%, and 98%, respectively. A PSV of ≥213 c/s was optimal for ≥50% restenosis with sensitivity, specificity, PPV, NPV, and overall accuracy of 99%, 100%, 100%, 98%, and 99%, respectively. An ICA PSV of 274 c/s was optimal for ≥70% restenosis with sensitivity, specificity, PPV, NPV, and overall accuracy of 99%, 91%, 99%, 91%, and 98%, respectively (Table 20.3). ROC analysis showed that PSVs were significantly better than EDVs and ICA/CCA ratios in detecting ≥30% and ≥50% restenosis (Figs. 20.8 and 20.9). We concluded that the means PSVs of a normal ICA distal to CEA patching were higher than normal non-operated ICAs; therefore, standard duplex velocities criteria should be revised after CEA with patch closure [57].

Duplex Ultrasound Surveillance of Carotid Stents

Kupinski et al. [58] conducted a study to evaluate the DUS characteristics of carotid stents including comparing hemodynamic to B-mode and color flow imaging data in 40 carotid stents placed in the common or internal carotid arteries of 37 patients. DUS examinations included PSV and end diastolic velocity (EDV) taken proximal to the

Table 20.3 PSVs: sensitivity and specificity for ≥30%, ≥50%, and ≥70% restenosis

≥30	PSV	Sensitivity	95% CI	Specificity	95% CI	PPV	NPV	Overall accuracy
	150	99	97–100	93	88–99	95	99	96
	152	99	97–100	95	91–100	96	99	97
	153	99	97–100	96	93–100	97	99	98
	154	98	96–100	96	93–100	97	98	97
*	**155**	**98**	**96–100**	**98**	**94–100**	**98**	**98**	**98**
	157	96	93–100	98	94–100	98	95	97
	159	95	91–99	99	96–100	99	93	96
	160	92	87–97	99	96–100	99	88	96
≥50								
	191	100	100	92	84–100	97	100	98%
	197	100	100	94	87–100	98	100	99%
	199	100	100	96	90–100	99	100	99%
	201	99	98–100	98	93–100	99	98	99%
*	**213**	**99**	**98–100**	**100**	**100**	**100**	**98**	**100%**
	215	98	96–100	100	100	100	93	99%
	219	97	95–100	100	100	100	91	98%
	221	97	94–100	100	100	100	89	97%
≥70								
	263	99	97–100	77	61–93	97	91	96%
	264	99	97–100	80	64–96	97	91	96%
	270	99	97–100	83	68–98	98	91	97%
	272	99	97–100	87	73–100	98	91	97%
*	**274**	**99**	**97–100**	**91**	**79–100**	**99**	**91**	**98%**
	289	98	96–100	90	78–100	99	86	97%
	306	98	96–100	90	77–100	99	82	97%
	312	98	96–100	95	85–100	99	82	97%
	317	98	96–100	95	85–100	99	82	97%

*The values in bold have the highest overall accuracies

From AbuRahma AF, et al. Proposed duplex velocity criteria for carotid restenosis following carotid endarterectomy. J Vasc Surg 2009;50:286-91. Reprinted with permission from Elsevier Limited

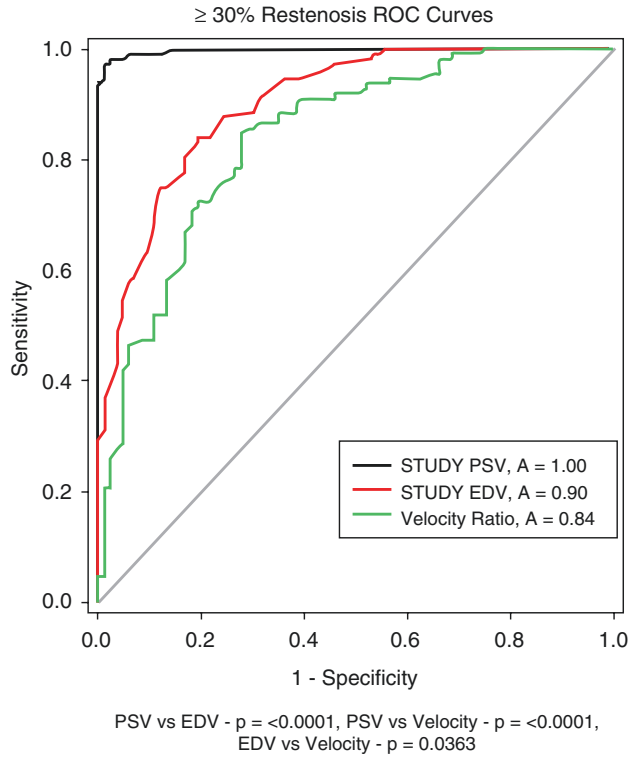


Fig. 20.8 Receiver operator curve analysis for ≥30% restenosis. From AbuRahma AF, et al. Proposed duplex velocity criteria for carotid restenosis following carotid endarterectomy with patch closure. J Vasc Surg 2009;50:286–91. Reprinted with permission from Elsevier

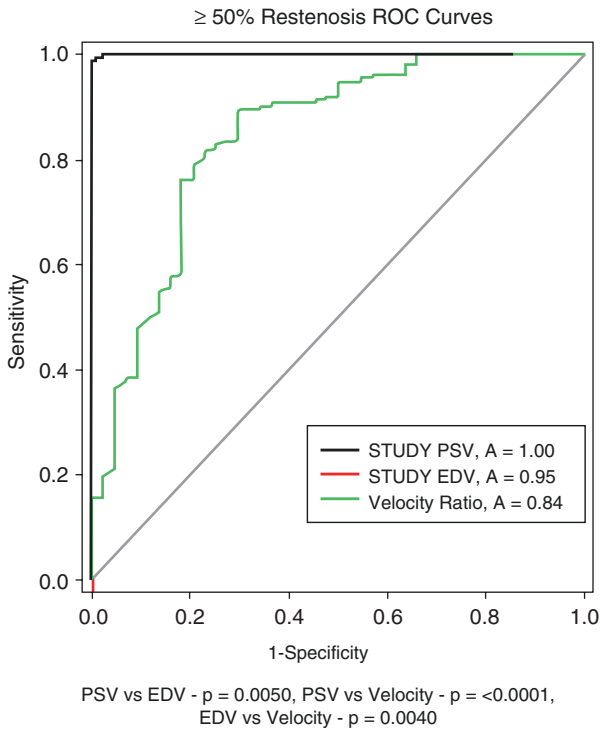


Fig. 20.9 Receiver operator curve analysis for ≥50% restenosis. From AbuRahma AF, et al. Proposed duplex velocity criteria for carotid restenosis following carotid endarterectomy with patch closure. J Vasc Surg 2009;50:286–91. Reprinted with permission from Elsevier

stent (pre-stent), at the proximal, mid, and distal regions of the stent, and distal to the stent (post-stent). The stents were evaluated at 1 day and 3, 6, and 12 months post-procedure and yearly thereafter. The average follow-up interval was 6 ± 1 month. In 31 patient ICA stents, the PSV proximally within the stent was 92 ± 6 cm/s with an EDV of 24 ± 2 cm/s. The mid stent PSV was 86 ± 5cm/s with an EDV of 24 ± 2 cm/s. The distal stent PSV was 90 ± 4 with an EDV of 26 ± 2 cm/s. Proximal to the stent, the PSV was 70 ± 3 cm/s with an EDV of 17 ± 1 cm/s. Distal to the stent, the PSV was 77 ± 4 cm/s with an EDV of 25 ± 2 cm/s. There were no defects observed on B-mode image and no areas of color turbulence. Three stents developed stenotic areas with PSVs of 251, 383, and 512 cm/s. The EDV was 50, 131, and 365 cm/s, respectively. Post-stenotic turbulence was present in each of these stents. An elevated PSV of >125 cm/s was found in 32% of the stents (9 of 28) without evidence of stenosis on B-mode image of post-stenotic turbulence. These data demonstrate that velocities within stented carotid arteries can be elevated above established ranges for normal. They concluded that velocity criteria may need to be adjusted when applied to stented carotid arteries. It has been suggested that focal velocity increase at the point of maximal narrowing >150 cm/s and a pre-stenotic (or pre-stent) to stenotic segment PSV ratio of l:≥2 are suggestive of significant in-stent restenosis [59].

Optimal Carotid Duplex Velocity Criteria for Defining the Severity of Carotid In-Stent Restenosis

We conducted a prospective study to define the optimal velocities in detecting various severities of in-stent restenosis: ≥50% and 80–99%. Our analysis included 144 CAS patients. All patients had completion arteriograms and postoperative carotid DUS imaging, which was repeated at 1 month and every 6 months thereafter. If patients had a PSV of the ICA of ≥130 cm/s, they underwent CT/angiogram. We recorded the PSVs and EDVs of the ICA and common carotid artery and the PSV of the ICA/common carotid artery ratio. A ROC curve analysis was used to determine the optimal velocity criteria for ≥30%, ≥50%, and ≥80% in-stent restenosis. The mean follow-up was 30 months (range: 1–78 months). There were 215 pairs of imagings (DUS versus CTA/angiography) available for analysis. We found that an ICA PSV of ≥154 cm/s was optimal for ≥30% stenosis with a sensitivity of 99%, specificity of 89%, PPV 96%, NPV of 97%, and overall accuracy of 96%. An ICA EDV of 42 cm/s had a sensitivity, specificity, PPV, NPV, and overall accuracy in detecting ≥30% stenosis of 86%, 62%, 87%, 60%, and 80%, respectively. An ICA PSV of ≥224 cm/s was optimal for >50% stenosis with

a sensitivity of 99%, specificity of 90%, PPV of 99%, NPV of 90%, and overall accuracy of 98%. An ICA EDV of 88 cm/s had a sensitivity, specificity, PPV, NPV, and overall accuracy in detecting ≥50% stenosis of 96%, 100%, 100%, 53%, and 96%. An ICA/CCA ratio of 3.4 had a sensitivity, specificity, PPV, NPV, and overall accuracy in detecting ≥50% stenosis of 96%, 100%, 100%, 58%, and 96%, respectively. An ICA PSV of ≥325 cm/s was optimal for >80% stenosis with a sensitivity of 100%, specificity of 99%, PPV of 100%, NPV of 88%, and overall accuracy of 99%. An ICA EDV of 119 cm/s had a sensitivity, specificity, PPV, NPV, and overall accuracy in detecting ≥80% stenosis of 99%, 100%, 100%, 75%, and 99%, respectively (Table 20.4). The PSV of the stented artery was a better predictor for in-stent restenosis than the EDV velocity or ICA/CCA ratio for >30% and >50% in-stent restenosis (Figs 20.10 and 20.11). We concluded that the optimal DUS velocity criteria for in-stent restenosis of ≥30%, ≥50%, and ≥80% were the PSVs of 154, 224, and 325 cm/s, respectively [60].

Table 20.4 The optimal cutoff values for peak systolic velocity, end diastolic velocity, and internal carotid artery/common carotid artery ratios

	Peak systolic velocity			End diastolic velocity			ICA/CCA ratio		
Stenosis	Cutoff	AUC (95% CI)	SE	Cutoff	AUC (95% CI)	SE	Cutoff	AUC (95% CI)	SE
≥30%	>154	0.97 (0.93–1)	0.02	>42	0.76 (0.68–0.84)	0.04	>1.533	0.83 (0.77–0.90)	0.03
≥50%	>224	0.95 (0.84–1)	0.05	>88	0.82 (0.69–0.96)	0.07	>3.439	0.88 (0.77–0.99)	0.06
≥80%	>325	0.88 (0.63–1)	0.12	>119	0.90 (0.72–1)	0.09	>4.533	0.86 (0.62–1)	0.12

AUC area under the curve, *CI* confidence interval, *ICA/CCA* internal carotid artery/common carotid artery, *SE* standard error
From AbuRahma AF, et al. Optimal carotid duplex velocity criteria for defining the severity of carotid in-stent restenosis. J Vasc Surg 2008;48: 589–94. Reprinted with permission from Elsevier

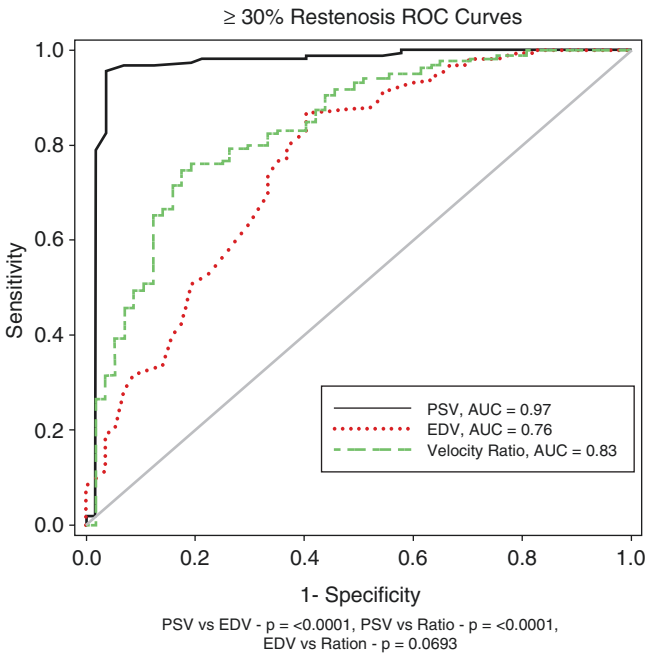


Fig. 20.10 Receiver operating characteristic (*ROC*) curve for _30% in-stent restenosis shows peak systolic velocity (*PSV*, *black line*), end diastolic velocity (*EDV*, *red line*), and velocity ratio area under the curve (*AUC*, *green line*). From AbuRahma AF, et al. Optimal carotid duplex velocity criteria for defining the severity of carotid in-stent restenosis. J Vasc Surg 2008;48:589–94. Reprinted with permission from Elsevier

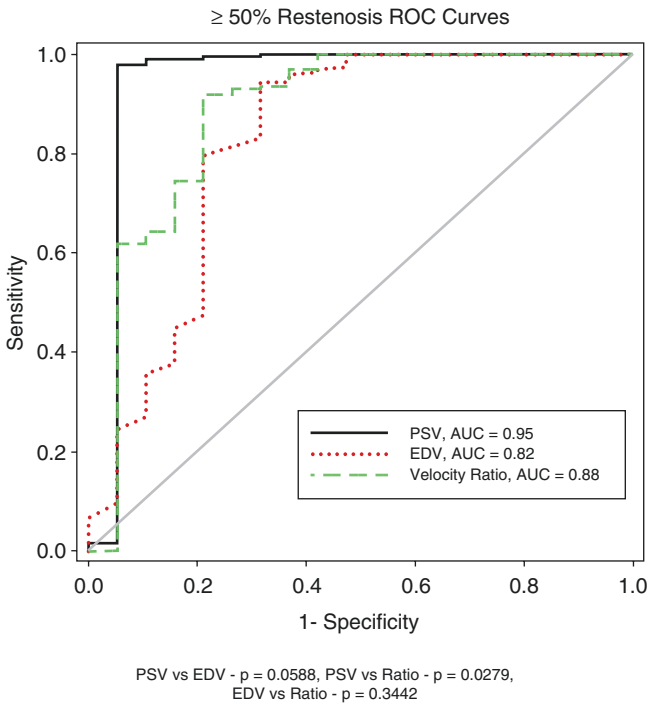


Fig. 20.11 Receiver operating characteristic (*ROC*) curve for _50% in-stent restenosis shows peak systolic velocity (*PSV*, *black line*), end diastolic velocity (*EDV*, *red line*), and velocity ratio area under the curve (*AUC*, *green line*). From AbuRahma AF, et al. Optimal carotid duplex velocity criteria for defining the severity of carotid in-stent restenosis. J Vasc Surg 2008;48:589–94. Reprinted with permission from Elsevier

Determination of Progression of Carotid Stenosis

It has now become clear that it is possible to determine major progression of disease in two different categories with duplex scanning technology. Progression of disease from a mild form (20–50% diameter reduction) to a severe form (50–99% diameter reduction) can be accurately detected based on significant changes in duplex velocities [61]. In addition, in severe stenosis, it is possible to identify the development of extreme degrees of stenosis (>80% diameter reduction) by the changes in the ratio between peak systolic and end diastolic velocities. The ability to identify such disease progression without invasive arteriographic studies will contribute to our understanding of the natural history of the disease process.

Carotid screening after CEA for the sake of detecting contralateral disease progression has been of much more value. Several studies have reported on the progression of contralateral stenosis after CEA [54, 62, 63]. Contralateral carotid stenosis progression was more frequent than ipsilateral recurrent stenosis during the long-term follow-up in these studies. Several studies have also identified that the risk of contralateral carotid artery stenosis progression is dependent on the existing disease at the time of the initial CEA [54, 62, 63]. The risk of progression for moderate stenosis at the initial surveillance to severe stenosis can be as high as five times [63].

Natural History of Carotid Artery Stenosis, Contralateral to Carotid Endarterectomy

A few nonrandomized studies have reported on the natural history of carotid artery stenosis contralateral to CEA. We analyzed the natural history of carotid artery stenosis contralateral to CEA from two randomized prospective trials [45, 64].

The contralateral carotid arteries of 534 patients who participated in two randomized trials comparing CEA with primary closure versus patching were followed clinically and had DUSs at 1 month and every 6 months. Carotid artery stenoses were classified into <50%, ≥50–<80%, ≥80–99%, and occlusion. Late contralateral CEAs were done for significant carotid artery stenoses. Progression of carotid artery stenosis was defined as progress to a higher category of stenosis.

Out of 534 patients, 61 had initial contralateral CEAs, within 30 days of the ipsilateral CEA, and 53 had contralateral occlusions. Overall, 109/420 (26%) progressed at a mean follow-up of 41 months (range: 1–116 months). Progression of contralateral carotid artery stenosis was noted in 5/162 (3%) patients who had baseline normal carotids; 56/157 (36%) patients with <50% carotid artery stenosis progressed versus 45/95 (47%) patients with 50–<80% carotid artery stenosis (p = 0.003). The median time for progression was 24 months for <50% carotid artery stenosis and 12 months for ≥50–<80% carotid artery stenosis (p = 0.035). Freedom from progression for patients with baseline <50% and ≥50–<80% carotid artery stenosis at 1, 2, 3, 4, and 5 years was 95%, 78%, 69%, 61%, 48%; and 75%, 61%, 51%, 43%, and 33%, respectively (p = 0.003). Freedom from progression in patients with baseline normal carotid arteries at 1, 2, 3, 4, and 5 years was 99%, 98%, 96%, 96%, and 94%. Late neurologic events referable to the contralateral carotid artery were infrequent in the whole series (28/420, 6.7%) and included 10 strokes (2.4%) and 18 TIAs (4.3%) (28/258, 10.9% in patients with contralateral carotid artery stenosis); however, late contralateral CEAs were done in 62 patients (62/420, 15%, in the whole series, 62/258, 24%, in patients with contralateral carotid artery stenosis). The survival rates were 96%, 92%, 90%, 87%, and 82% at 1, 2, 3, 4, and 5 years.

We concluded that progression of contralateral carotid artery stenosis was noted in a significant number of patients with baseline contralateral carotid artery stenosis. Serial carotid DUSs every 6–12 months for patients with ≥50–<80% carotid artery stenosis and every 12–24 months for ≤50% carotid artery stenosis are adequate.

Carotid Endarterectomy Based on Carotid Duplex Ultrasonography Without Angiography

In many centers, carotid evaluation by angiography is no longer done routinely, even when planning for surgery, to eliminate the risk of neurologic events during angiography. The risk of stroke from angiography is around 1% [46].

Although standard conventional angiography is still generally considered to be the definitive diagnostic test for carotid artery stenosis, there has been an increasing interest in performing CEA based on clinical evaluation and duplex scanning only [19, 65–71]. It has been estimated that up to 95% of CEA procedures are currently undertaken on the basis of carotid duplex ultrasound alone [72], again, with no evidence that reliance on this policy compromised patient safety or operability. This is generally done to minimize cost and to expedite surgery on these patients, therefore optimizing the long-term benefit conferred by CEA, specifically for symptomatic patients. However, for clinicians who advocate this policy, they must keep in mind the following considerations: carotid duplex must be done in an accredited vascular laboratory and interpreted by high-qualified physicians in this field; the method of estimating carotid stenosis must also be defined, e.g., the NASCET method; clinicians must also be aware of duplex ultrasound findings that may suggest an inflow or outflow disease, which if present; and other imaging

must be added prior to intervention [73]. This has been stimulated by improvement in the accuracy and reliability of color carotid duplex scanning, along with the increasing demands to minimize both the risk of carotid angiography and the cost of medical care. CEAs are generally indicated for high-grade stenoses of asymptomatic patients and in moderate to severe stenoses in patients with hemispheric neurologic events. These stenoses can usually be accurately detected by duplex scanning.

Dawson et al. [65] completed a prospective evaluation of 94 cases that showed that arteriography affected clinical management in only one case (1%). They indicated that while specific indications for CEA without angiography remain controversial, the results of angiography rarely alter the clinical treatment plan when a technically adequate duplex scan shows an 80–99% stenosis in asymptomatic patients or an ipsilateral 50–99% stenosis in patients with hemispheric neurologic symptoms [65].

If arteriography is not done, there is a potential to miss significant lesions in the carotid siphon or an intracranial aneurysm or tumor as the cause of TIAs. However, it is unlikely that carotid siphon disease will produce significant symptoms [74] and, therefore, does not impact the decision to perform CEA. Intracranial aneurysms occur in approximately 1–2% of patients undergoing arteriography [75], but most are small and unlikely to be affected by CEA [75]. With the advances in imaging techniques, the concern for occult brain tumors has become less relevant. These limitations can be overcome by using carotid MRA/CTA.

In addition, associated costs are significant with some institutions reporting charges for cerebrovascular arteriography as high as $5000–$6000. Strandness [76] has suggested that wider use of duplex scanning as the sole preoperative test could result in substantial savings. For instance, if 150,000 CEAs are done annually, with an average cost of angiography of $3000, the total cost of angiography alone would be $750 million dollars (not counting the costs of an estimated 7500 TIAs, 1500 strokes, and 100 deaths). If these same patients had duplex scanning alone, the total costs would be approximately $37 million; this represents a savings annually in the United States alone of $712 million [76]. We have already begun to see a shift in the testing that is done for a preoperative diagnosis. A report from the University of Vermont stated that 87% of their last 130 CEAs were performed without arteriography, with acceptable rates of stroke and death [77].

CEA should not be attempted without MRA/CTA arteriography unless the following criteria are met [65]:

1. The distal ICA is free of significant disease (disease is localized to the carotid bifurcation).
2. The CCA is free of significant disease.
3. Vascular anomalies, kinks, or loops are not present.
4. The duplex scan is technically adequate.
5. Vascular laboratory duplex accuracy is known.

Some potential pitfalls include patients with nonhemispheric symptoms, recurrent stenosis, or ICA stenosis of <50% [65, 77]. However, as experience grows, indications may be expanded.

Therefore, MRA/CTA/conventional angiography is most likely to be useful when the duplex scan is not diagnostic, in patients with atypical lesions that appear to extend beyond the carotid bifurcation, and for stenoses of <50% in patients with classical hemispheric neurologic symptoms.

Ultrasonic Carotid Plaque Morphology and Carotid Plaque Hemorrhage: Clinical Implications

The lack of neurologic symptoms in many patients with significant carotid stenosis has perplexed many scientists. It has been proposed that the character of the plaque may be as, or more, important than significant stenosis in producing neurologic events.

We [78] examined the importance of ultrasonic plaque morphology and its correlation to the presence of intraplaque hemorrhage and its clinical implications. We studied 152 carotid plaques associated with ≥50% ICA stenoses in 135 patients who had CEAs and characterized them ultrasonographically into irregular/ulcerative, smooth, heterogeneous, homogeneous, or not defined. Heterogeneous plaques were defined as a mixture of hyperechoic, isoechoic, and hypoechoic plaques. In contrast, homogeneous plaques were defined as consisting of only one of the three types of echogenic plaques. An isoechoic plaque was defined as having the echogenicity of a normal intima-media complex. A hyperechoic plaque was brighter than an isoechoic plaque, and a hypoechoic plaque was not as bright as an isoechoic plaque. An irregular plaque was defined as a plaque that lacks a smooth surface with or without an intimal layer. A smooth plaque was defined as a plaque without surface irregularities or ulcerations. All plaques were examined pathologically for the presence of intraplaque hemorrhage. The ultrasonic morphology of the plaques included 63 with surface irregularity (41%), 48 smooth (32%), 59 heterogeneous (39%), 52 homogeneous (34%), and 41 (27%) not defined. Intraplaque hemorrhage was present in 57 out of 63 (90%) irregular plaques and 53 out of 59 (90%) heterogeneous plaques, in contrast to 13 out of 48 (27%) smooth plaques and 17 out of 52 (33%) homogeneous plaques ($p < 0.001$). Fifty-three out of 63 (84%) irregular plaques and 47 out of 59 (80%) heterogeneous plaques had TIAs/stroke symptoms, in contrast to 9 out of 48 (19%) for smooth plaques and 15 out of 52 (29%) for homogeneous plaques

($p < 0.001$). Fifty-four percent of the irregular plaques and 57% of the heterogeneous plaques had ipsilateral cerebral infarcts, in contrast to 12% of the smooth plaques ($p < 0.001$) and 14% of the homogeneous plaques ($p < 0.001$). We concluded that irregular and/or heterogeneous carotid plaques are more often associated with intraplaque hemorrhage, neurologic events, and cerebral infarcts. Therefore, ultrasonic plaque morphology may be helpful in selecting patients for CEA.

In another study, we [79] analyzed the natural history of 60–<70% asymptomatic carotid stenosis according to ultrasonic plaque morphology and its implication on treatment. Patients with 60–<70% asymptomatic carotid stenosis during a 2-year period entered into a protocol of carotid duplex surveillance/clinical examination every 6 months. Their ultrasonic plaque morphology was classified as heterogeneous (Group A, 162) or homogeneous (Group B, 229). CEA was done if the lesion progressed to ≥70% stenosis or became symptomatic.

Three hundred and eighty-two patients (391 arteries) were followed at a mean follow-up of 37 months. The incidence of future ipsilateral strokes was significantly higher in Group A than in Group B: 13.6% versus 3.1% ($p = 0.0001$, odds ratio 5). Similarly, the incidence of all neurologic events (stroke/TIAs) was higher in Group A than in Group B: 27.8% versus 6.6% ($p = 0.0001$, odds ratio of 5.5). Progression to ≥70% stenosis was also higher in Group A than in Group B: 25.3% versus 6.1% ($p = 0.0001$, odds ratio 5.2). Forty-four (27.2%) late CEAs were done in Group A (16 for stroke, 21 for TIAs, and 7 for ≥70% asymptomatic carotid stenosis) versus 13 (5.7%) for Group B (5 for stroke, 7 for TIAs, and 1 for ≥70% asymptomatic carotid stenosis ($p = 0.0001$, odds ratio 6.2).

We concluded that patients with 60–<70% asymptomatic carotid stenosis with heterogeneous plaquing were associated with a higher incidence of late stroke, TIAs, and progression to ≥70% stenosis than patients with homogeneous plaquing. Prophylactic CEA for 60–<70% asymptomatic carotid stenosis may be justified if associated with heterogeneous plaquing.

In another study of the correlation of ultrasonic carotid plaque morphology and the degree of carotid stenosis [80], 2460 carotid arteries were examined using color DUS during a 1-year period. Carotid stenoses were classified into <50%, 50–<60%, 60–<70%, and >70–99%.

Heterogeneous plaques were noted in 138 of 794 arteries with <50% stenosis, 191/564 with 50–<60% stenosis, 301/487 with 60–<70% stenosis, and 496/615 with 70–99% stenosis. The higher the degree of stenosis, the more likely it is to be associated with heterogeneous plaques. Heterogeneous plaques were present in 59% of ≥50% stenoses versus 17% for <50% stenoses, 72% of ≥60% stenoses versus 24% for <60% stenosis, and 80% of ≥70% stenoses versus 34% for <70% stenoses ($p < 0.0001$ and odds ratios of 6.9, 8.1, and 8.0, respectively). Heterogeneous plaques were associated with a higher incidence of symptoms than homogeneous plaques in all grades of stenoses: 68% versus 16% for <50% stenosis, 76% versus 21% for 50–<60%, 79% versus 23% for 60–<70%, and 86% versus 31% for ≥70–99% ($p < 0.0001$ and odds ratios of 8.9, 11.9, 12.6, and 13.7, respectively). Heterogenosity of plaques was more positively correlated to symptoms than any degree of stenosis (regardless of plaque structure). 80% of all heterogeneous plaques were symptomatic versus 58% for all ≥50% stenoses, 68% for all ≥60% stenoses, and 75% for all ≥70% stenoses ($p < 0.0001$, $p < 0.0001$, and $p = 0.02$, respectively).

We concluded that the higher the degree of carotid stenosis, the more likely it is to be associated with ultrasonic heterogeneous plaquing and cerebrovascular symptoms. Heterogenosity of the plaque was more positively correlated to symptoms than to any degree of stenosis. These findings suggest that plaque heterogenosity should be considered in selecting patients for CEA [80].

Differentiating unstable from stable plaques by ultrasound has been hampered by the subjectiveness of interpreting such images [81–83]. Biasi et al. [83] conducted a study to confirm that plaque echogenicity evaluated by computer analysis, as suggested by preliminary studies, can identify plaques associated with a high incidence of strokes. A series of 96 patients with carotid stenosis in the range of 50–99% were studied retrospectively (41 with TIAs and 55 asymptomatic). Carotid plaque echogenicity was evaluated using a computerized measurement of the median grayscale value (GSM). All patients had a CT brain scan to determine the presence of infarction in the carotid territory.

The incidence of ipsilateral brain CT infarctions was 32% for symptomatic plaques and 16% for asymptomatic plaques ($p = 0.076$). It was 25% for >70% stenosis and 20% for <70% stenosis ($p = 0.52$). It was 40% in those with a GSM of <50 and 9% for plaques with a GSM of >50 ($p < 0.001$) with a relative risk of 4.6 (95% CI 1.8–11.6).

It was concluded that a computer analysis of plaque echogenicity was better than the degree of stenosis in identifying plaques associated with an increased incidence of CT brain scan infarction and consequently useful for identifying individuals at high risk of stroke.

Kern et al. [82] investigated the value of real-time compound ultrasound imaging for the characterization of atherosclerotic plaques in the ICA. Thirty-two patients with plaques of the ICA as identified by high-resolution B-mode scanning were investigated with real-time compound ultrasound imaging. Two independent observers rated plaque morphology according to a standardized protocol. The majority of plaques was classified as predominantly echogenic and as plaques of

irregular surface, whereas ulcerated plaques were rarely observed. The interobserver agreement for plaque surface characterization was good for both compound ultrasound ($\kappa = 0.72$) and conventional B-mode ultrasound ($\kappa = 0.65$). For the determination of plaque echogenicity, the reproducibility of compound ultrasound [$\kappa(w) = 0.83$] was even higher than that of conventional B-mode ultrasound [$\kappa(w) = 0.74$]. According to a semiquantitative analysis, real-time compound ultrasound was rated superior in the categories plaque texture resolution, plaque surface definition, and vessel wall demarcation. Furthermore, there was a significant reduction of acoustic shadowing and reverberations. They concluded that real-time compound ultrasound was a suitable technique for the characterization of atherosclerotic plaques, showing good general agreement with high-resolution B-mode imaging. This subject will be covered in depth elsewhere in this volume (Chap. 8).

Identifying High-Risk Carotid Plaque

Truijman et al. [84] are conducting a prospective multicenter study to improve the diagnosis of high-risk carotid plaques (Plaque At RISK [PARISK]). This is a study of patients with recent (<3 months) neurological symptoms due to ischemia in the territory of the carotid artery and <70% ipsilateral carotid artery stenosis who are not scheduled for CEA or CAS. A total of 300 patients will undergo baseline MRI, multidetector CTA, and carotid duplex ultrasound. They will also undergo MRI of the brain, ambulatory TCD recording of the middle cerebral artery, and blood work. The imaging will be repeated in 150 patients after 2 years. All patients will undergo a follow-up brain MRI and regular clinical follow-up until the end of the study. The combined primary endpoint contains ipsilateral recurrent ischemic stroke or TIAs or new ipsilateral ischemic brain lesions on follow-up brain MRIs [84].

Truijman et al. [84] also reported on intraplaque hemorrhage, fibrous cap status, and microembolic signals in symptomatic patients with mild to moderate carotid artery stenosis (PARISK study). Patients with recent TIAs or minor stroke in the carotid territory and an ipsilateral mild to moderate carotid artery plaque were included in this multicenter diagnostic cohort study. Intraplaque hemorrhage and fibrous cap status were dichotomously scored. Analysis of transcranial Doppler data was done blinded for the MRI results. A total of 113 patients were included. TCD measurements were available in 105 patients (average recording time was 219 min). A total of 26 microembolic signals were detected in 8 of 105 patients. Intraplaque hemorrhage was present in 44 of 105 plaques. Fibrous cap status was assessable in 92 of 105 plaques, and 36 of these had a thin/ruptured fibrous cap. There was no significant difference in the prevalence of microembolic signals between patients with and without intraplaque hemorrhage ($p = 0.46$) or with thick versus thin/ruptured fibrous cap ($p = 0.48$) was found. They concluded that intraplaque hemorrhage and fibrous cap status were not associated with microembolic signals in patients with a symptomatic mild to moderate carotid artery stenosis. This suggests that MRI and TCD provide different information on plaque vulnerability [85, 86].

Gupta et al. [87] conducted a systematic review and meta-analysis of carotid plaque MRI and stroke risk and concluded that the presence of intraplaque hemorrhage, lipid-rich necrotic core, and thinning/rupture of the fibrous cap on MRI of carotid plaque was associated with an increased risk of future stroke or TIAs in patients with carotid atherosclerotic disease. They also found that a dedicated MRI of plaque composition offers stroke risk information beyond measurement of luminal stenosis in carotid atherosclerotic disease [87].

However, den Hartog et al. [88] concluded that, based on current literature; it appears premature for routine application of MRI to assess carotid plaque characteristics associated with plaque vulnerability. Although MRI still holds promise, clinical application for plaque characterization would require a consensus regarding MRI settings and confirmation by histology. Predefined protocols for histology and MRI need to be established [88].

Carotid Duplex Ultrasound After Neck Trauma

DUS can be used in evaluating vascular injuries of the neck. Although carotid trauma is not strictly a disease of the carotid bifurcation, developments in this area parallel the changes seen in surgery for atherosclerotic disease. Carotid duplex following cervical trauma was prospectively evaluated by Fry et al. [89]. Fifteen patients had duplex scan and arteriography, and 11 of these had a region of interest in zone II and four in zone III. One injury was diagnosed by duplex scan in this group, and this was confirmed by arteriography; both studies were normal in the remaining 14 patients. On the next 85 patients, Fry et al. then performed duplex scan only, with arteriography reserved only for an abnormal duplex result. In this group, 62 patients had potential injuries in zone II and the remainder in zone III. Seven arterial injuries were identified by duplex scan and confirmed by arteriography. The remaining 76 patients had normal duplex scans and no sequelae up to 3 weeks post discharge. It was concluded that DUS is a valuable tool in evaluating carotid injury.

Carotid Duplex Ultrasound for Internal Carotid Artery Dissection

ICA dissection has been reported more often recently than was previously suspected. This disease can appear spontaneously or may follow traumatic events accompanied by the fully developed picture of focal ischemia with facial and neck pain and Horner's syndrome (ptosis, miosis, and anhydrosis). It can also appear with very few symptoms or may even be completely asymptomatic. Using a color flow DUS, the diagnosis can be made when the flow signal is carefully followed over the entire neck region. In the longitudinal section, forward and backward signal components in blue/red color coding are generally seen next to one another in the proximal ICA. Distally, an area free of flow signals marks the proximal end of the dissection. Corresponding Doppler signals characterize partial recanalization with systolic forward and backward signal components but with diastolic forward flow preservation [90–92]. On angiography, proximally there is a threadlike occlusion/subtotal stenosis of the ICA without a connection to the intracranial vasculature (Fig. 20.12). Monthly follow-up assessments are important, since the majority of the cases spontaneously recanalize.

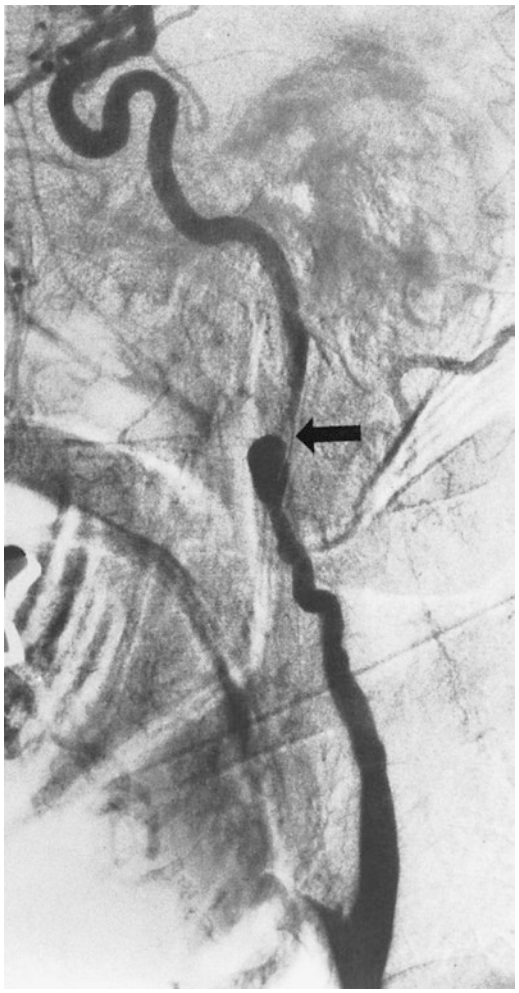


Fig. 20.12 Carotid arteriogram showing internal carotid artery dissection of the higher cervical portion, as indicated by the *black arrow*

Role of Duplex Ultrasound in Vertebrobasilar Insufficiency

Recent studies have shown that with adequate skill and patience on the operator's part, the innominate, subclavian, cervical, and prevertebral segment of the vertebral artery can be displayed with real-time duplex ultrasound. Duplex scanning appears to be the most successful and accurate technique to diagnose atherosclerotic lesions of the vertebral arteries in the neck region. With this technique, the cervical segment of the vertebral artery can be visualized, and the direction of the flow can be determined, whether antegrade or retrograde, which may be suggestive of subclavian steal. It has been reported that a reliable investigation of the prevertebral segment and the orifice of the vertebral artery is possible in more than 80% of cases. Further details on this subject are covered in Chap. 14.

Color Duplex Ultrasound in the Diagnosis of Temporal Arteritis

Temporal arteritis is sometimes diagnosed clinically, but a temporal artery biopsy is usually recommended to confirm the diagnosis [93]. The American College of Rheumatology requires three of the following five criteria to be met to establish the diagnosis: age ≥50 years, new onset of localized headache, temporal artery tenderness or decreased pulse, erythrocyte sedimentation rate ≥50 mm/h, and histologic findings. Schmidt et al. [93] examined the usefulness of color duplex ultrasonography in patients suspected of having temporal arteritis. In their prospective study, all patients seen in the departments of rheumatology and ophthalmology from January 1994 to October 1996 who had clinically suspected active temporal arteritis or polymyalgia rheumatica were examined by duplex ultrasonography. They examined both common superficial temporal arteries and the frontal and parietal rami as completely as possible in longitudinal and transverse planes to see if they were perfused, if there was a halo around the lumen, and (using simultaneous pulsed-wave Doppler ultrasonography) if there was a stenosis. Stenosis was considered to be present if flow velocity was more than twice the rate recorded in the area before the stenosis, perhaps with waveforms demonstrating turbulence and reduced velocity behind the area of stenosis. Two ultrasound studies were performed and read before the biopsies. Based on standard criteria, the final diagnoses were temporal

arteritis in 30 patients, 21 with biopsy-confirmed disease, polymyalgia rheumatica in 37, and negative histologic findings and a diagnosis other than temporal arteritis or polymyalgia rheumatica in 15. They also studied 30 control patients matched for age and sex to the patients with arteritis.

Schmidt et al. [93] found that in 22 (73%) of the 30 patients with temporal arteritis, ultrasonography showed a dark halo around the lumen of the temporal arteries. The halos disappeared after a mean of 16 days (range: 7–56) of treatment with corticosteroids. Twenty-four patients (80%) had stenoses or occlusions of temporal artery segments, and 28 patients (93%) had stenoses, occlusions, or a halo. No halos were identified in the 82 patients without temporal arteritis; 6 (7%) had stenoses or occlusions. For each of the three types of abnormalities identified by ultrasonography, the interrater agreement was ≥95%.

They concluded that there are characteristic signs of temporal arteritis that can be visualized by color duplex ultrasonography. The most specific sign is a dark halo, which may be due to edema of the artery wall. In patients with typical clinical signs and a halo on ultrasonography, it may be possible to make a diagnosis of temporal arteritis and begin treatment without performing a temporal artery biopsy.

Overall Accuracy of Noninvasive Vascular Testing

Although angiography has been the standard against which most noninvasive tests have been measured, it is far from ideal for comparison with physiologic tests designed to detect altered hemodynamics. Furthermore, any significant stenosis in the carotid artery from its origin at the aorta up to and including the ophthalmic artery can result in abnormal test results in the indirect methods of testing. In addition, long-standing collateral pathways that compensate effectively for the hemodynamic effects of the stenotic lesion can produce a normal result in an indirect carotid test. Direct methods (duplex imaging) do not detect lesions in the upper part of the internal carotid artery, where such lesions can also produce an abnormal result with an indirect test. In an unbranched artery, blood flow is determined by the cross-sectional area of its narrowest portion and by the pressure gradient across it. Accordingly, the extent of stenosis caused by a carotid bifurcation plaque should be calculated by comparing the narrowest diameter of its diseased lumen with the diameter of the undiseased distal internal carotid artery. Although the term "critical stenosis" is generally used to compare the results of noninvasive testing, the exact value necessary to cause a measurable decrease in pressure or alteration in arterial blood flow remains controversial. DeWeese et al. [94] reported that lesions that narrowed the lumen less than 47% and left a residual lumen larger than 3 mm in diameter never caused measurable pressure drops, whereas stenoses greater than 63% of the luminal diameter with residual lumens smaller than 1 mm in diameter always did. Therefore, if systolic pressure distal to an arterial stenosis is measured, lesions that reduce the diameter 50% or more, thus reducing the cross-sectional area by 75% or more, are generally detected. However, if alterations in blood flow are measured, diameter reductions in excess of 67% (more than 90% of the cross-sectional area) are necessary for abnormal test results [90]. Clinically, a stenosis greater than 75% of the diameter or 94% of the area is necessary to cause symptomatic reduction of cerebral blood flow [95]. Since various reports have used diameter reductions from 40% to 75% as their standard of comparison, some variations in the reported results can be explained on this basis.

In addition to problems in study design, many of the carotid noninvasive studies report their results in terms of diagnostic accuracy. Since diagnostic accuracy may vary with the prevalence of disease in the population, it is impossible to compare different series if this term is used. By contrast, if results of carotid noninvasive studies are expressed in terms of sensitivity, i.e., the ability to detect the presence of the disease (true-positive rate), and specificity, i.e., the ability to detect the absence of disease (true-negative rate), these terms should be independent of disease prevalence and allow comparison of one series with another. The following terms are generally used in comparing the accuracy of various noninvasive vascular tests:

1. Sensitivity is calculated by dividing the number of true-positive tests detected noninvasively by the total number of true-positive tests detected by angiography.
2. Specificity is calculated by dividing the true-negative tests detected noninvasively by the total true-negative tests detected by angiography.
3. The false-positive rate is calculated by dividing the number of false-positive tests detected noninvasively by the total number of noninvasive positive tests.
4. The false-negative rate is calculated by dividing the number of false-negative tests detected noninvasively by the total number of negative noninvasive tests.
5. The positive predictive value is the percentage of noninvasive test results that accurately predicts abnormality, in other words the percentage of positive noninvasive tests that correctly predicted disease as supported by "gold standard" arteriography. It is calculated by the number of true-positive noninvasive testing divided by the number of all positive noninvasive studies (i.e., true plus false positives).
6. Negative predictive value is defined as the percentage of noninvasive test results that accurately predicts normality,

in other words the percentage of negative noninvasive studies that correctly predicted the absence of disease as supported by "gold standard" arteriography. It is calculated by dividing the number of true-negative noninvasive tests by the number of all negative noninvasive studies (i.e., true plus false negatives).

7. The overall accuracy is defined as the sum of the true-positive and the true-negative values compared with the total number of tests performed.

Figures 20.13, 20.14, and 20.15 are simplified methods of calculating sensitivity, specificity, positive predictive values, negative predictive values, and overall accuracy. Although specificity and sensitivity possess certain advantages, they are limited to fixed threshold criteria that are taken as positive for the noninvasive carotid screening test. Expressing the result of a screening test as a receiver operator characteristic curve avoids the limitations of fixed threshold criteria [96]. This curve plots the dynamic relationship between sensitivity and specificity and allows the examiner to increase or decrease the sensitivity of the tests by varying the threshold criterion for a positive result of that particular test.

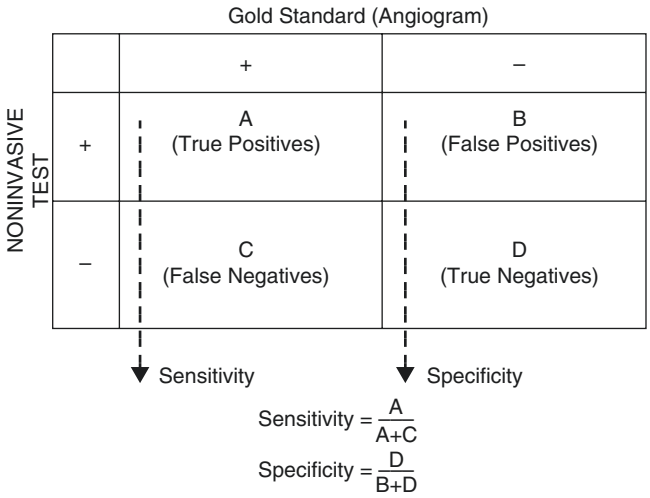


Fig. 20.13 Method for calculating sensitivity and specificity

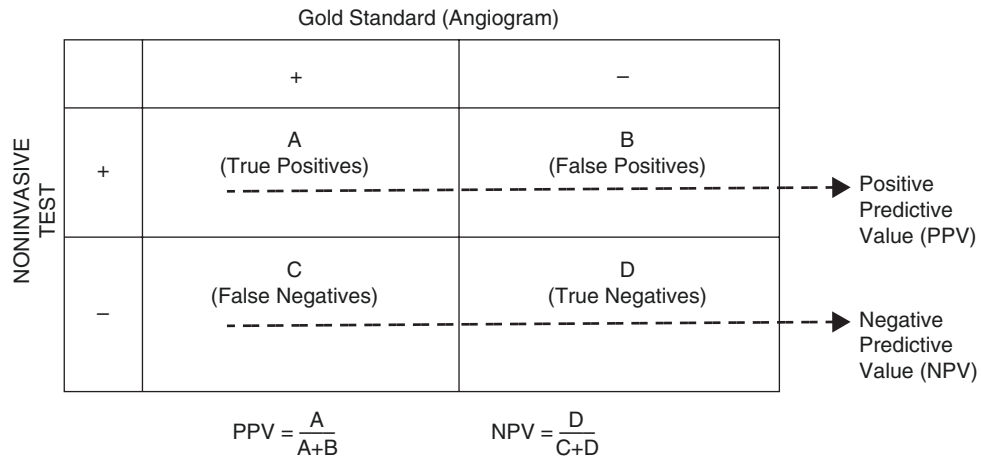


Fig. 20.14 Method for calculating positive predictive values and negative predictive values

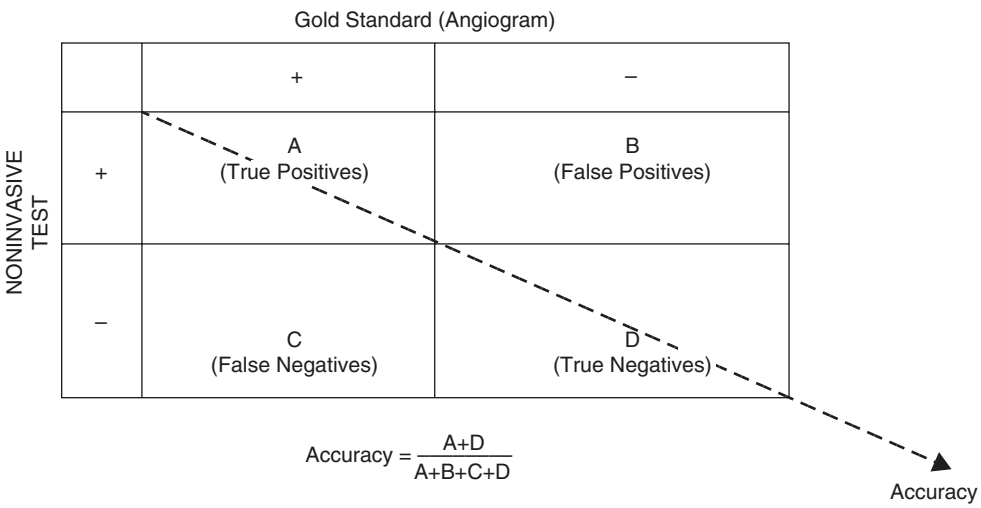


Fig. 20.15 Method for calculating overall accuracy

Review Questions

1. Vascular laboratories should use the following duplex velocity criteria to minimize the number of patients undergoing unnecessary arteriography prior to CEA:
   a. Duplex velocity criteria with 95% or greater negative predictive value
   b. Duplex velocity criteria with 95% or greater positive predictive value
   c. Duplex velocity criteria with the best overall accuracy
   d. Duplex velocity criteria with the best sensitivity
2. Carotid duplex studies for patients who underwent CEA with patching showed:
   a. The mean peak systolic velocity of a normal internal carotid artery distal to CEA patching was lower than normal non-operated internal carotid artery
   b. The mean peak systolic velocity of a normal internal carotid artery distal to CEA patching was higher than normal non-operated internal carotid artery
   c. The mean peak systolic velocity of a normal internal carotid artery distal to CEA patching was similar to a normal non-operated internal carotid artery
   d. None of the above
3. The role of post-CEA carotid duplex surveillance is more valuable in:
   a. Detecting restenosis after CEA with patching
   b. Detecting progression of existing contralateral carotid artery stenosis
   c. Post-CEA duplex surveillance has no value in any circumstances
   d. None of the above
4. CEA should not be attempted without MRA/CTA or further imaging unless the following criteria are met:
   a. The distal ICA is free of significant disease (disease is localized to the carotid bifurcation)
   b. The common carotid artery is free of significant disease
   c. Duplex scan is technically adequate
   d. All of the above

Answer Key

1. b
2. b
3. b
4. d

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