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REVIEWS

Clinical Applications of Noninvasive Carotid Artery Testing

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The management of patients with cerebral transient ischemic attacks and carotid artery stenosis remains controversial. Noninvasive techniques help to determine which patients require surgical intervention without exposing the majority of patients to the risk and discomfort of invasive procedures. Measurement of ophthalmic artery pressure by ophthalmodynamometry or oculoplethysmography gives a representation of perfusion pressure in the internal carotid artery circulation. Doppler ultrasound studies can define the extent of obstruction to flow at the carotid artery bifurcation and assess collateral flow from the external carotid artery. Real time B-mode ultrasonography can detect nonobstructive ulcerated plaque in the carotid artery bifurcation.

Employing these examinations in a test battery can identify hemodynamically significant lesions, which are more likely to precipitate a stroke. The information obtained from these studies can be utilized in patients with episodes of cerebral transient ischemic attacks, asymptomatic carotid artery bruits and vertebrobasilar insufficiency. Noninvasive carotid artery testing is also useful in screening patients with nonspecific symptoms, such as dizziness or light-headedness, which may be related to decreased flow in the carotid circulation. Noninvasive carotid artery testing can provide valuable anatomic and physiologic information required in the appropriate management of patients with cerebrovascular disease. It is of particular value in managing patients with heart disease who are at high risk for complications from invasive procedures.

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Atherosclerotic disease at the bifurcation of the cervical carotid artery has been recognized as a potential source of cerebral ischemia and stroke (1). Hemodynamically significant obstruction of blood flow to the ipsilateral cerebral hemisphere can occur, particularly with occlusion of the internal carotid artery. In addition, fragments of atherosclerotic plaque can embolize to distal vasculature in the cerebral circulation, resulting in an area of focal ischemia (2).

A great deal of interest has been focused on disease at the carotid artery bifurcation because atherosclerotic plaque in this region can be removed surgically (3). Patients can be identified clinically before the occurrence of a stroke. Patients with episodes of cerebral transient ischemic attacks (1–3) or nonspecific symptoms of light-headedness (4) are often found to have atherosclerotic plaque at the carotid artery bifurcation. Flow disturbances at the carotid bifurcation can be detected by auscultation of a vascular bruit in the neck.

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Although there is a significant association between atherosclerotic disease at the carotid bifurcation and stroke, it is not always clear how to manage an individual patient with carotid artery disease or transient ischemic attacks appropriately. Most patients with carotid artery disease do not develop a stroke (5). This is true even for patients with cerebral transient ischemic attacks (6–8). The incidence of a stroke occurring in patients with antecedent transient ischemic attacks is reported to be as high as 33% over a 5 year period (7). However, the incidence of stroke in patients with antecedent transient ischemic attacks who also have angiographically demonstrable carotid artery disease is 5% in those with unilateral lesions and 14 to 18% in those with bilateral lesions (6). Moreover, only 50% of patients with transient ischemic attacks have angiographically demonstrable disease at the carotid artery bifurcation (9). The remainder have transient ischemic attacks from other causes of cerebral ischemia.

Because of the emphasis on carotid artery disease as a cause of stroke, the other major sources of cerebral ischemia are often overlooked. Thrombosis of intracranial arterioles by proliferative changes in vessel walls occurs in patients with hypertension (10) and diabetes (11). This intracranial disease accounts for up to 50% of strokes (10). Recent evidence has indicated that the heart is a major source of

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cerebral emboli in patients with cardiac arrhythmia (12), valvular heart disease (13) and myocardial infarction (14). Intracranial thrombosis and cardiogenic emboli can cause a transient ischemic attack as well as stroke.

The appropriate management of carotid artery disease is complicated because removal of atherosclerotic plaque by carotid endarterectomy is not without risk. Some surgical series (3) report excellent results for carotid endarterectomy, with complication rates as low as 1%. In other series (6), the incidence of serious cerebrovascular or cardiac complications approaches the natural history of the disease itself. Cerebral arteriography is also associated with a small but significant incidence of serious complications, particularly in patients with cerebrovascular disease (15). Even digital intravenous subtraction angiography involves a certain amount of pain and morbidity, while a satisfactory image is obtained in only 60% of studies (16). Patients with myocardial or renal disease do not tolerate the large amounts of contrast medium required to obtain satisfactory images. Therefore, clinicians have sought means of delineating a subset of patients with transient ischemic attacks or carotid artery disease, or both, in whom the benefits of endarterectomy outweigh the risks. Noninvasive means of assessing the nature and extent of atherosclerotic disease at the carotid artery bifurcation have evolved to identify these patients without subjecting all patients to the discomfort and possible complications associated with invasive procedures.

Methods of Noninvasive Carotid Artery Testing

Two categories of noninvasive tests are employed to assess atherosclerotic disease at the carotid artery bifurcation: 1) indirect tests of perfusion in the internal carotid artery distal to the bifurcation; and 2) direct imaging of the carotid artery bifurcation by ultrasonographic techniques. The indirect methods were developed initially and still provide important physiologic information.

Indirect Tests

Ophthalmic artery pressure. *Ophthalmodynamometry.* Historically, the first test developed to assess carotid artery function was ophthalmodynamometry (17). The ophthalmic artery is the first major branch of the intracranial internal carotid artery. Ophthalmodynamometry is performed by applying external pressure to the eye with a strain gauge while simultaneously visualizing the arteries in the ocular fundus. The pressure at which the vessels begin to collapse in diastole and refill in systole is recorded as the diastolic pressure. The pressure at which complete collapse of the arteries occurs is recorded as the systolic pressure. If there is a significant decrease in perfusion pressure in the internal carotid artery because of occlusive disease at the bifurcation or further distally, a reduced ophthalmic artery pressure will be observed. Normally, the ophthalmic artery pressure is 75% of the brachial artery pressure (17). Ophthalmodynamometry is technically difficult to perform, highly subjective and requires patient cooperation. If there is retinal vascular disease, such as diabetic or hypertensive retinopathy, it is difficult to visualize the vessels.

Oculoplethysmography. This test was developed by Gee et al. (18) to provide a more objective means of measuring ophthalmic artery pressure. A vacuum is applied to the surface of the eye at gradually increasing pressure. This reduces the distortion of the globe produced by the pulsations of the ophthalmic artery. The pulse wave is recorded by a plethysmograph. The pressure required to obliterate the pulsations is calibrated to represent the systolic pressure in the ophthalmic artery (Fig. 1). The normal ophthalmic pressure as measured by this technique is also 75% of the brachial artery pressure, with a standard deviation of ± 5% (4,18). The ophthalmic pressure is normal in central retinal artery occlusion (19), but is sometimes reduced with ophthalmic artery stenosis (20).

Measurement of ophthalmic artery pressure provides a means of determining the perfusion pressure in the intracranial internal carotid artery. However, to reduce systolic perfusion pressure in the carotid artery by 10%, the diameter of the lumen must be reduced to 1.0 mm (21). Only high grade stenosis or complete occlusion of the internal carotid artery can be detected. There is considerable collateral flow to the internal carotid artery by way of the ophthalmic artery from the supraorbital vessels of the external carotid circulation. Therefore, if good collateral circulation is present, ophthalmic artery pressure will be normal, even though there is hemodynamic obstruction to flow in the internal carotid artery at the bifurcation. Thus, the specificity of oculoplethysmography and ophthalmodynamometry in detecting

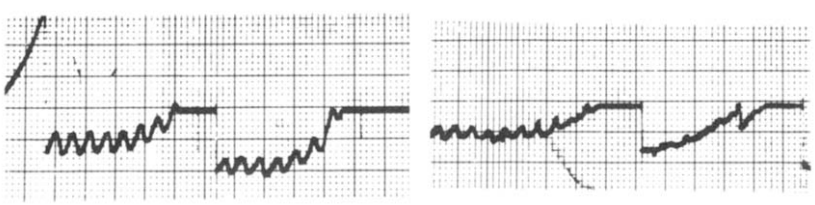


Figure 1. Pulse wave recorded from the right and left ophthalmic artery by oculoplethysmography. Gradual obliteration of the pulse wave indicates the systolic pressure in the ophthalmic artery. (Reprinted from Weinberger J, et al. [20], with permission of the American Heart Association, Inc.)

carotid artery stenosis of greater than 50% is as high as 98%, with sensitivity ranging from 70 to 95% (22).

Pulse delay oculoplethysmography. Another type of oculoplethysmography was developed by Kartchner et al. (23) to take advantage of differential flow in the internal and external carotid arteries. In this method, the time of arrival of the systolic pulse in the eye from the ophthalmic artery is compared with the time of arrival of the pulse to the pinna of the ear from branches of the external carotid artery. If there is a delay in arrival of the pulse to the eye, it is suggestive of internal carotid artery disease. Some authors (23) have reported a high degree of correlation between pulse delay oculoplethysmography and carotid angiography, but others (24) have had less satisfactory results with this technique. The specificity of the technique for detecting lesions with greater than 50% stenosis is 84%, with sensitivity ranging from 52 to 90% (22). Poor correlation with angiography usually occurs when there is disease in the carotid sinus below the origin of both the internal and external carotid arteries. Unfortunately, a large proportion of atherosclerotic disease forms in this region.

Supraorbital Doppler ultrasound examination. The supraorbital and supratrochlear branches of the ophthalmic artery provide a major anastomosis between the internal and external carotid arteries. When the internal carotid artery is not obstructed, flow is anterograde from the internal to the external carotid artery. With obstruction of the internal carotid artery, flow is retrograde from the external to the internal carotid artery.

The direction and velocity of flow in the supraorbital artery can be detected with Doppler ultrasonography. High frequency sound waves of 8 to 10 MHz are emitted from the Doppler instrument and reflected off the red blood cells flowing in the vessel. The returning echo is of a slightly lower frequency than the emitted Doppler wave because of the motion of the red blood cells. The frequency shift is in the audible range. The degree of frequency shift is directly proportional to the velocity of the red blood cells; the faster the cells are moving, the higher the frequency of sound that is audible (25–27).

The direction of flow in the supraorbital artery can be detected by applying the Doppler probe below the supraorbital ridge until flow is heard. Bidirectional Doppler instruments indicate whether the flow is anterograde or retrograde (28). However, a correct determination is dependent on probe placement. Therefore, the superficial temporal and facial branches of the external carotid artery are compressed. With retrograde flow there is diminution or obliteration of flow when external branches are compressed (29). Most patients show no change in flow with compression of the external carotid artery branches (30). False positive studies are encountered in 10% of cases and false negative studies are encountered in 15% of cases when results of supraorbital Doppler ultrasonography are compared with those from carotid angiography (30). Nevertheless, supraorbital Doppler flow provides valuable information as to the extent of available collateral circulation to the internal from the external carotid artery. It is also a method for determining whether obstruction to flow at the carotid artery bifurcation is hemodynamically significant.

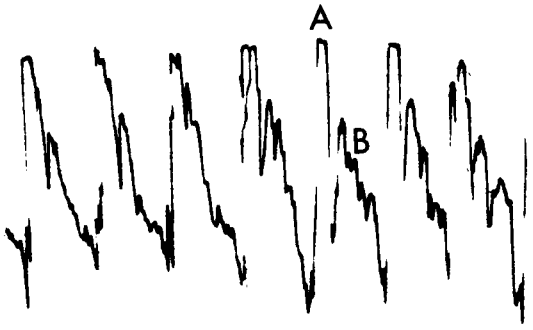
Direct Methods of Detecting Disease at the Carotid Artery Bifurcation

Carotid phonoangiography. The original method of detecting vascular disease at the carotid artery bifurcation was auscultation of a vascular bruit over the neck. As a vessel narrows, laminar flow is lost. A smaller volume of blood flows through the stenotic area at a higher rate. Eddy currents develop both proximal and distal to the stenosis. This turbulent flow produces a bruit that can be auscultated with a stethoscope. However, it is often difficult to distinguish a carotid bruit from a transmitted cardiac flow murmur. In addition, with high grade carotid stenosis, flow can be reduced sufficiently to eliminate turbulence, and the bruit disappears. Therefore, auscultation of a bruit correlates with angiographic findings of carotid stenosis in approximately 60% of cases (31).

Carotid phonoangiography is a method of quantitating the frequency of turbulent flow at the carotid artery bifurcation (32). Kistler et al. (33) demonstrated a linear correlation between degree of stenosis and the frequency of turbulence. However, the method could not detect high grade stenoses in which the bruit was too soft to be appreciated. These are, of course, precisely the lesions that one wishes to identify. Therefore, the value of carotid phonoangiography as a diagnostic test is limited.

Doppler examination of the cervical carotid artery. Doppler ultrasonography can be employed to measure flow in the cervical carotid artery. As mentioned previously in discussing supraorbital Doppler examination (25–27), the

Figure 2. Normal pulse waveform in the internal carotid artery with systolic peak (**A**) and gradual decrease in diastole (**B**). (Reprinted from Weinberger J, et al. [30] with permission from *New York State Journal of Medicine*, copyright by the Medical Society of the State of New York.)



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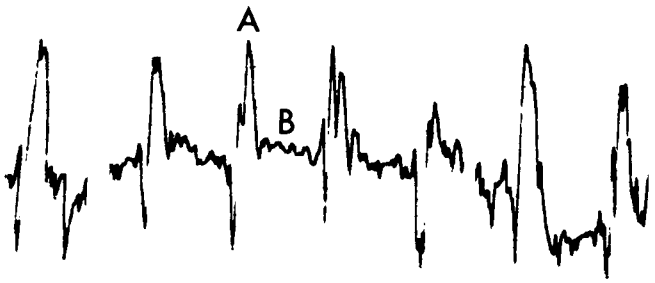

Figure 3. Reduced flow in the common carotid artery with attenuation of upsweep of the systolic phase (**A**) and flattening of the pulse wave in diastole (**B**). (Reprinted from Weinberger J, et al. [30], with permission from *New York State Journal of Medicine*, copyright by the Medical Society of the State of New York.)

frequency of sound produced by the Doppler instrument is directly proportional to the velocity of flow of the red blood cells in the carotid artery. The frequencies of sound can be auscultated by the examiner and plotted as a pulse waveform (Fig. 2) (30,34). Flow in the internal carotid artery can be differentiated from flow in the common and external carotid arteries because the declining slope of the pulse wave in diastole is less abrupt (30,34). The bifurcation can be readily identified in most instances.

Rate of blood flow. Obstructive lesions at the carotid artery bifurcation can produce a decrease in the rate of diastolic flow and a slowing of the upsweep of systolic flow (Fig. 3). However, to produce a 10% reduction in blood flow, the cross-sectional area of the carotid artery must be reduced to 4.5 mm^2 (35). Thus, only high grade stenosis or complete occlusion of the carotid artery produces a reduction in flow. If there is good runoff of flow into the external carotid artery, flow in the common carotid artery may be normal even in the presence of a complete occlusion of the internal carotid artery. In this instance, the pulse waveform in the external carotid artery becomes similar to that of the normal internal carotid artery. This causes difficulty in identifying complete occlusions of the internal carotid artery. Usually, the indirect tests of carotid hemodynamic measurements can identify these lesions (30).

Turbulence of flow. Doppler examination of the carotid artery bifurcation can also detect turbulence of flow. Low frequency turbulence correlates with lesions causing a 40 to 70% stenosis on angiography (30). High frequency turbulence occurs when there is greater than 70% stenosis (30). Eddy currents from turbulence passing by a nonobstructive ulcerated plaque can also be detected as flutter turbulence (30,36,37). Doppler frequencies can be plotted on an analog computer by fast Fourrier transform analysis to give a spectrum in real time (plotted as a pulse wave) (Fig. 4) or a histogram. Turbulence produces a spread of frequencies, with more frequencies at both the higher and the lower end of the spectrum (25,26). Employing spectral analysis of Doppler frequencies correlates with angiography in more than 90% of cases in lesions with greater than 50% stenosis (25,26).

Auditory versus spectral analysis. Several authors (30,36) report that auditory determination of the types of turbulence gives a more subtle delineation of the flow abnormalities than spectral analysis. In my experience, this has been the case, with 7% false positive studies and 2% false negative studies (30). Turbulence from eddy currents near a plaque can be discriminated from turbulence due to stenosis. This is of value in the assessment of hemodynamics. Cardiac flow murmurs can be differentiated from carotid artery bruits. Even a dissecting aneurysm of the aorta that involved the common carotid artery and was not demonstrated on angiography has been diagnosed by this technique (38).

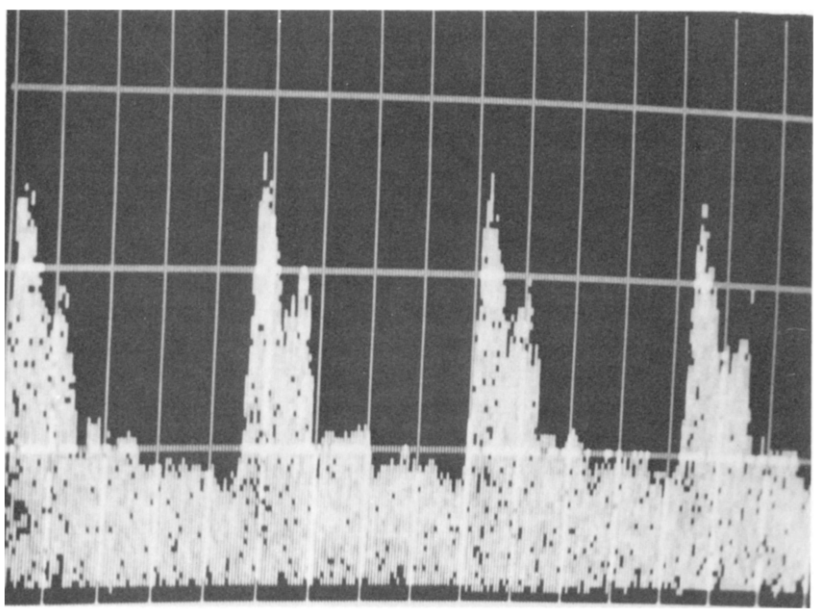

Figure 4. Spectral analysis of Doppler flow frequencies indicates a pulse waveform. Note the midsystolic deflection in this recording from a patient with mitral valve prolapse.

Limitations. Clearly, there are difficulties in assessing carotid artery flow by direct Doppler examination. The interpretation of auscultatory findings is subjective. The study should be performed by the physician taking responsibility for the interpretation of the test. It is not always possible to identify the carotid artery bifurcation or an area of stenosis that occupies only a small region of the vessel. It is sometimes difficult to distinguish stenosis of the internal from stenosis of the external carotid artery. Complete occlusion of the internal carotid artery can be missed. To overcome these drawbacks, ultrasonic techniques have been developed to image the carotid artery bifurcation.

Imaging of the carotid artery bifurcation. Doppler ultrasound can produce a visual image of the carotid bifurcation. The carotid artery can be scanned with either a continuous wave (27) or a pulsed (25,26) Doppler wave device. A minimal threshold of flow velocity is determined. All points across the lumen of the carotid artery with flows higher than this threshold are plotted on an oscilloscope. This produces a line at each level of the carotid artery scanned. The internal and external carotid artery can be visualized because the septum dividing them is seen as an area of no flow. Thus, an image of the bifurcation is produced (Fig. 5). Narrowing of the lumen can also be visualized (Fig. 6). This method has shown a correlation of 95% with angiography for stenoses greater than 70%, but has not been useful for demonstrating nonobstructive plaques (27,39,40). The method is enhanced by spectral analysis or audible interpretation of Doppler frequencies (25–27).

Real time B-mode ultrasonography. This method is the most recent advent in ultrasonic imaging of the carotid bifurcation. Sound waves are emitted by a piezoelectric crystal at frequencies of 5 to 7.5 MHz. Echoes are received by the transducer. The transducer rotates through an arc of up to 120° so that tomographic sections are made. The echoes are displayed on a cathode-ray tube screen. The X and Y axes represent the spatial location of the source of the echoes. The density of the reflecting structure is represented by the intensity of brightness on the screen. Blood does not produce any echo, whereas calcium produces a very bright echo (41). Vessel walls are of intermediate density. When a satisfactory image is obtained, the carotid artery bifurcation can be visualized in great detail. The intima can be distinguished from the media and small nonobstructive plaques can be visualized that sometimes are not identified by angiography (37,41).

As with all noninvasive techniques, however, this method is not always accurate. When there is high grade stenosis, it is sometimes difficult to identify the bifurcation. A complete occlusion of the internal carotid artery cannot always be distinguished from a high grade stenosis. When the bifurcation is high or tortuous, it cannot always be visualized clearly. Low echo density plaques are difficult to detect, but often contain elements of higher echo density, so that they are usually identified but sometimes underestimated. Most of these difficulties are surmounted by duplex scanning: combining real time B-mode ultrasonography with concomitant Doppler examination.

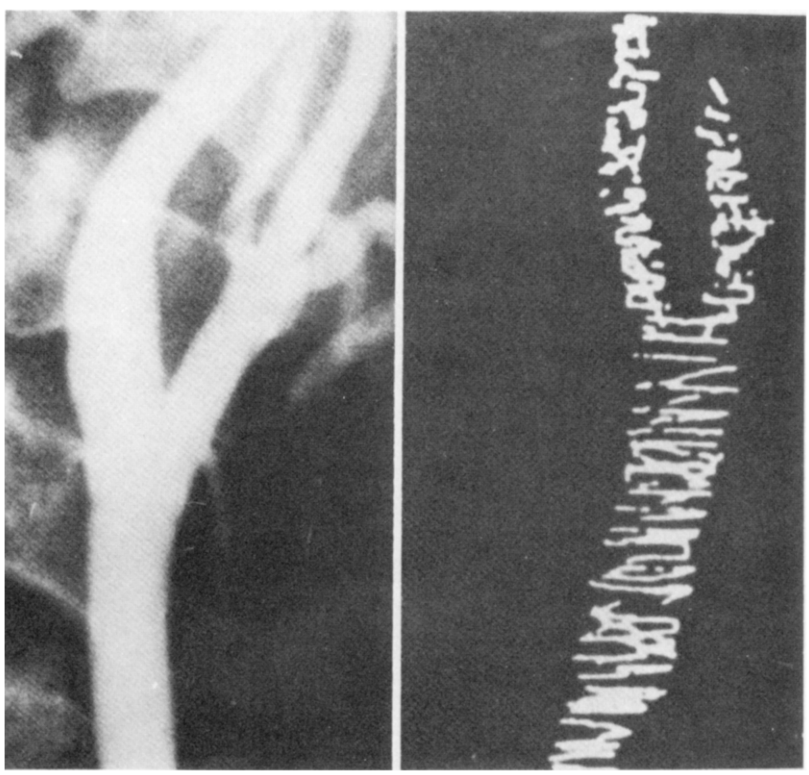

Figure 5. A normal Doppler angiogram of the carotid bifurcation (**right**) and angiographic appearance (**left**). (Reproduced from Bloch S, Baltaxe HA, Shoumaker RD [27], with permission.)

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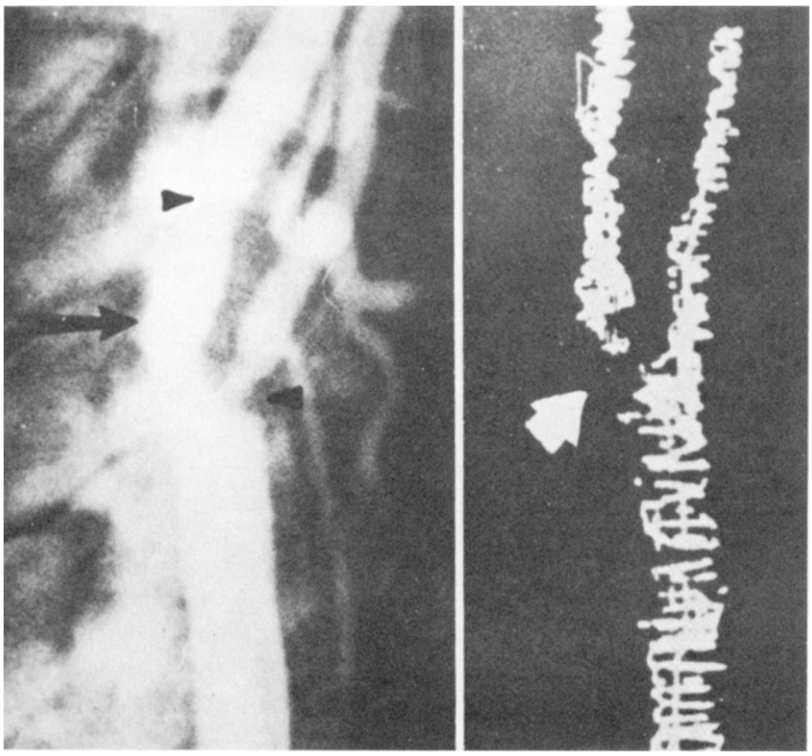

Figure 6. Doppler angiogram of the carotid bifurcation with high grade stenosis of the internal carotid artery (**arrow**) (**right**) and angiographic appearance (**left**). (Reproduced from Bloch S, Baltaxe HA, Shoumaker RD [27], with permission.)

Duplex Doppler scanning. Duplex scanning has an advantage over direct carotid Doppler examination because an area of stenosis can be identified by the B-mode scan and the flow through the lesion can be measured. Flow proximal and distal to the stenosis can also be ascertained. This is valuable in assessing the hemodynamics of the lesion. The flow characteristics and turbulence patterns measured by the Doppler examination give a better estimation of the degree of obstruction than measurements of lumen size on the sonogram. Changes in flow are directly proportional to the area of the lumen of the vessel. Since the carotid artery is a tortuous vessel rather than a perfect cylinder, different tomographic ultrasound views of the same region will show varying degrees of narrowing.

Most duplex B-scan Doppler instruments employ a 4 MHz Doppler probe. This low frequency probe provides greater penetration of tissue to identify deeper vessels. The 9.5 MHz Doppler probe employed in direct carotid artery flow studies provides a more precise resolution of spectral frequencies and is of greater value in determining the hemodynamics of turbulent flow. Therefore, both techniques should be employed in assessing flow in the carotid bifurcation.

The Noninvasive Test Battery

From the preceding description of noninvasive techniques, it is apparent that each has its advantages and disadvantages. Combining several studies in a battery enhances diagnostic accuracy (39,40). The sensitivity of the battery is increased if a positive result on any one test is interpreted as demonstrating a significant lesion, but the specificity is reduced (40). If a positive result is required on all studies in a test battery for a lesion to be considered significant, the specificity is increased but the sensitivity is reduced (40).

Accuracy of the four test battery. The noninvasive test battery can also be employed by using the information from each test in assessing the characteristics of disease at the bifurcation (30,37). A battery of four tests is employed in our laboratory. Real time B-mode ultrasonography is used to demonstrate the anatomic relations of plaque at the carotid bifurcation. Doppler ultrasound studies are employed to determine the hemodynamics of flow and degree of obstruction. These studies complement each other. Doppler ultrasound studies are more accurate in demonstrating hemodynamic lesions (30). Real time B-mode ultrasonography is more accurate in demonstrating nonobstructive plaques (37). Oculoplethysmography indicates the perfusion pressure of flow in the intracranial internal carotid artery (30). The supraorbital Doppler study is useful in determining the extent of collateral circulation and also provides some data on hemodynamics (30). The four studies serve to cross-check each other. Even though any one study may have up to 10% false positive results and 8% false negative results when compared with angiography, hemodynamic lesions can be identified in more than 95% of cases (30).

It is important to recognize that almost all of the four tests are subjective and accuracy is dependent on the skill of the examiner. Interpretation of the different studies to

make a diagnosis requires considerable judgment when the results do not concur. In most instances, a correct diagnosis can be obtained (30). The hemodynamic effects of a carotid lesion can be determined in more than 95% of cases even if the lesion itself cannot be visualized accurately. This confers a unique role of the noninvasive test battery in the clinical management of patients.

Clinical Applications of the Noninvasive Test Battery

Noninvasive carotid artery testing can provide both a physiologic and anatomic description of the extent and significance of carotid artery disease at the carotid bifurcation. These data can be applied to a number of clinical situations.

Transient Ischemic Attacks

The appropriate management of patients with transient ischemic attacks is controversial. Two previous reviews on noninvasive carotid artery testing (39,40) recommended that the evaluation of patients with a verified attack of cerebral transient ischemia should begin with carotid or digital intravenous angiography. This subjects many patients to unnecessary risk and discomfort because 50% of patients with a cerebral transient ischemic attack do not have appreciable disease at the carotid artery bifurcation (9). In addition, it is not clear that all lesions at the carotid artery bifurcation, even those producing transient ischemic attacks, require surgical extirpation.

Risk of stroke. Considerable evidence exists that hemodynamically significant obstruction at the carotid bifurcation carries a much greater risk of stroke than does nonobstructive plaque. Unselected autopsy studies from both the United States (42) and England (43) have revealed that most people older than 65 years of age have some degree of atheroma at the carotid artery bifurcation. A significant association between cerebral infarction and disease at the carotid bifurcation was noted only in individuals with high grade stenosis or complete occlusion (42,43). A similar study (44) was carried out in 100 consecutive patients with stroke who underwent cerebral angiography. When there was disease in the carotid artery ipsilateral to the affected cerebral hemisphere, the lesion was almost always a hemodynamically significant stenosis or complete occlusion.

In a prospective study (45), patients with arteriographically demonstrable carotid artery disease who were not surgically treated were followed up for a mean of 36 months. There was a 2% incidence of stroke when the ophthalmic artery pressure was normal and a 16% incidence of stroke when this pressure was reduced. Two other studies (30,37) utilizing a noninvasive carotid testing battery demonstrated a significantly higher ($p < 0.01$, $p < 0.001$) incidence of cerebrovascular symptoms in patients with hemodynamic carotid disease than in patients with nonobstructive plaques. In those patients whose clinical symptoms were correlated with angiography as well as with the noninvasive battery, cerebrovascular symptoms occurred ipsilateral to a hemodynamic lesion in 88% of cases, whereas they occurred ipsilateral to a nonobstructive plaque or a normal bifurcation in 35% of cases (30).

Hemodynamic factors appear to play a role, even in embolic stroke. A study of cerebral blood flow and electroencephalography during carotid endarterectomy suggests that transient cerebral ischemia arises from embolic events. Permanent deficits occur when there was an embolic event in an area of decreased cerebral perfusion (46).

Determination of a hemodynamically significant lesion. Noninvasive carotid artery testing is able to determine whether a lesion is hemodynamically significant in 95% of cases (25,27,30,36). Obstruction to flow can be determined by Doppler examination when there is high frequency turbulence or distortion of the pulse waveform (30). A decrement in perfusion pressure can be ascertained by oculoplethysmography (30). The extent of collateral circulation can be judged by the direction of flow in the supraorbital region (30).

If a hemodynamic lesion is noted, carotid endarterectomy can be considered when the medical condition of the patient is satisfactory. At this point, cerebral angiography can be performed to document the lesion at the carotid bifurcation and delineate the intracranial circulation. It is also advisable for the patient to have computed tomography of the brain and an electroencephalogram because transient episodes of neurologic dysfunction may be due to cerebral mass lesions or seizure phenomena.

Role of ulcerated carotid plaques. Some authors (2) maintain that ulcerated plaques in the carotid artery bifurcation are as important a source of cerebrovascular disease as are hemodynamic lesions. Other authors (47) even suggest that atherosclerotic lesions too small to be seen on angiography are found at carotid endarterectomy and may be responsible for transient ischemic attacks. Real time B-mode ultrasonography is an excellent method for visualizing these small nonobstructive plaques. When the plaques are in the carotid sinus and do not involve the origin of the internal carotid artery, the B-mode scan can identify plaques not seen on angiography (Fig. 7) (37). There is some evidence to suggest that the B-mode scan can distinguish nonobstructive lesions that are more prone to cause embolization (Fig. 8) (37). Further studies are in progress to determine whether B-mode sonography can demonstrate the pathologic constituents of atherosclerotic plaques. On the whole, most patients with transient ischemic attacks ipsilateral to a nonobstructive plaque can be managed by platelet antiaggregant therapy with aspirin (48). If ischemic episodes continue, angiography and endarterectomy can be performed.

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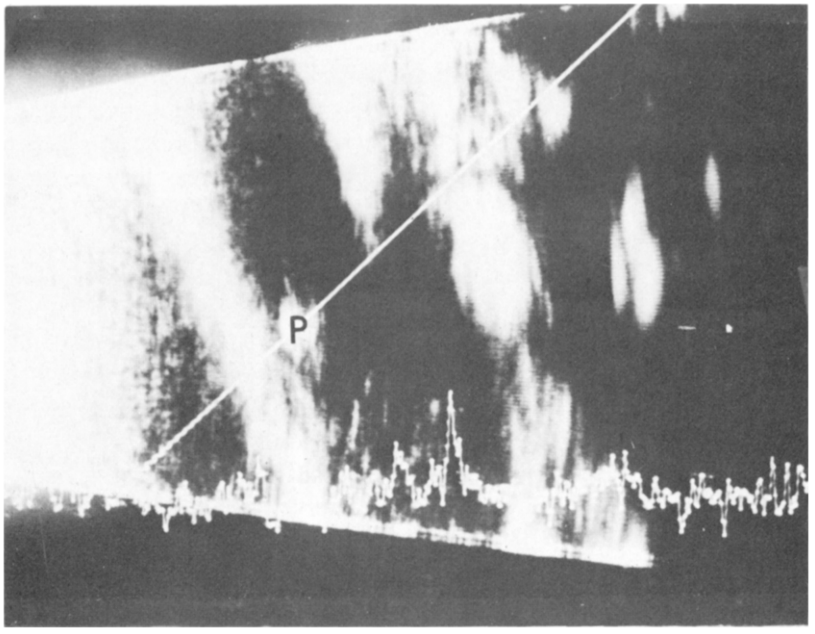


Figure 7. Real time B-mode ultrasonography demonstrates a small nodular plaque (P) in the carotid sinus of a patient with a cerebral transient ischemic attack. The lesion was not detected by digital intravenous subtraction angiography. (Reprinted from Weinberger J, et al. [37], with permission from *Archives of Neurology*, copyright by the American Medical Association.)

Irreversible Neurologic Deficits

Patients with mild hemispheric neurologic deficits secondary to an ischemic cerebrovascular accident can be managed in the same manner as patients with a transient ischemic attack (49). Patients with more severe deficits or appreciable lesions on computed tomographic scan of the brain can be studied acutely with noninvasive testing to determine whether the carotid bifurcation is likely to be the source of the stroke. Endarterectomy can be performed when indicated, but is usually deferred until at least 6 weeks after the stroke because of the risk of hemorrhage into the infarction when surgery is performed acutely (50).

Vertebrobasilar Insufficiency

Vertebrobasilar insufficiency is often difficult to categorize. Almost every elderly patient complaining of dizziness or presyncope is diagnosed as having vertebrobasilar insufficiency at one time or another. In most instances, vertebrobasilar occlusive disease is not the cause of the symptoms (51). The Joint Committee for Stroke Facilities requires two symptoms or signs of focal dysfunction arising from structures supplied by the posterior circulation in order to establish a diagnosis of vertebrobasilar insufficiency (49); one of these symptoms can be vertigo or dizziness (49).

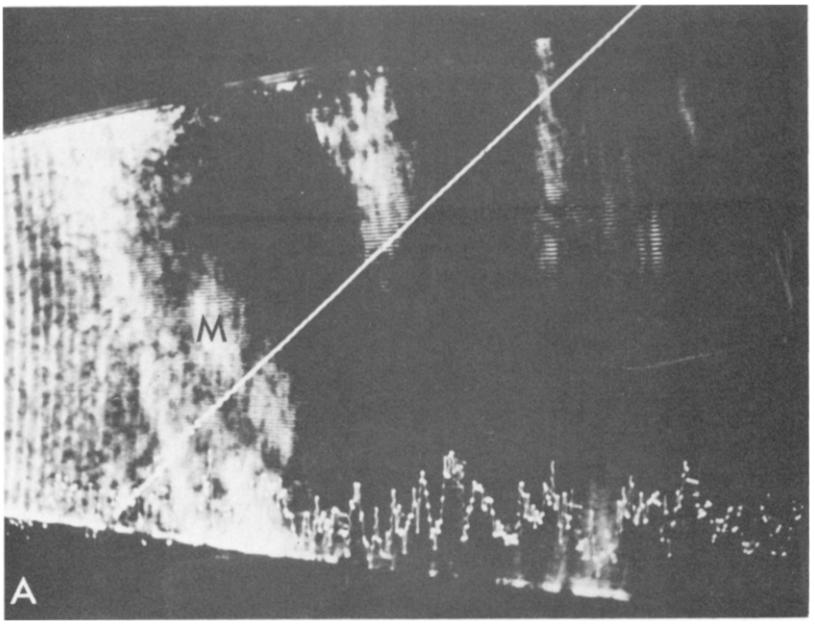
The natural history of vertebrobasilar insufficiency is somewhat different from that of transient ischemic attacks in the carotid flow territory. Most patients have continued episodes of transient ischemia rather than a completed stroke (8). When a stroke does occur secondary to vertebrobasilar occlusive disease, there is often associated carotid occlusive disease (52).

Combined carotid and vertebrobasilar disease. The carotid and vertebrobasilar circulations form collateral channels through the posterior communicating arteries. Noninvasive carotid artery testing can determine whether there is inadequate collateral blood supply because of hemodynamic obstruction to flow in the carotid artery circulation. In this instance, carotid endarterectomy sometimes alleviates the transient ischemic episodes (6) and also may reduce the incidence of stroke.

Many patients with nonspecific symptoms of light-headedness or dizziness are also referred for noninvasive carotid testing. Twenty-one percent of these patients are found to have hemodynamically significant obstruction to flow in the carotid circulation (4). Whether carotid endarterectomy alleviates the symptoms or reduces the incidence of stroke in these patients has not been determined.

Asymptomatic Carotid Bruits

Risk of stroke. Two major prospective studies (53,54) demonstrated that patients with an asymptomatic carotid bruit are not at increased risk for stroke in the ipsilateral cerebral hemisphere. Heyman et al. (54) found an incidence of stroke of 13.9% in 72 patients with an asymptomatic bruit followed up for 6 years, compared with 3.4% in 1,548 patients without a bruit. However, there was no significant increase in the risk of stroke in the cerebral hemisphere ipsilateral to the bruit. Wolf et al. (53) found a stroke rate twice that expected for age in subjects followed up in the Framingham study, but more than half of the strokes were not related to the bruit. There are two major flaws in these studies. The first is that only 60% of patients with a cervical bruit have carotid artery stenosis on angiography (31). This



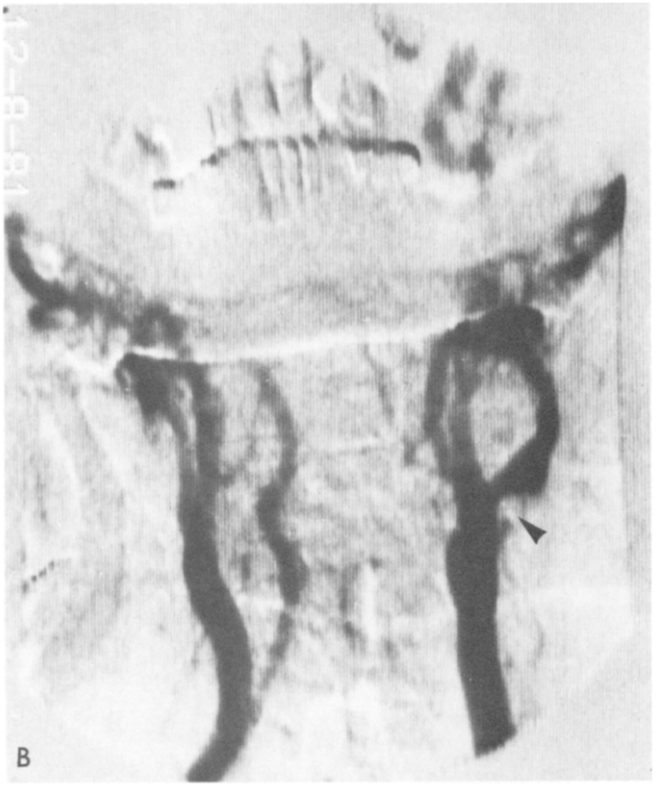


Figure 8. **A**, Real time B-mode ultrasonography demonstrates a mural plaque (M) lining the wall of the carotid sinus of a patient with a cerebral transient ischemic attack. This type of lesion was found to be associated with a similar proportion of cerebral ischemic symptoms as high grade stenosis. (Reprinted from Weinberger J, et al. [36], with permission.) **B**, The mural plaque visualized as a stenotic ulcer (**arrow**) on digital intravenous subtraction angiography. (Reprinted from Weinberger J, et al. [37] with permission from *Archives of Neurology*, copyright by the American Medical Association.)

dilutes the population studied that is at risk for stroke from carotid disease. Second, patients with an asymptomatic bruit on one side may have silent hemodynamic disease on the contralateral side (30). This reduces the percent of stroke ipsilateral to an asymptomatic bruit, while not accounting for some strokes ipsilateral to significant carotid artery disease.

Thompson et al. (55) followed up 138 patients with an asymptomatic bruit and angiographically demonstrated carotid artery stenosis. The incidence of stroke in these patients was 17.4% for 18 months compared with 4.5% in patients with an asymptomatic bruit who had carotid endarterectomy. However, the group not operated on contained patients who were too ill to undergo surgery and who also had other risk factors that could have precipitated stroke.

Role of carotid endarterectomy. Many clinicians observe patients with an asymptomatic carotid bruit until symptoms of transient cerebral ischemia develop. At this point, the patient is referred for carotid endarterectomy. This may not be appropriate management. Fifty percent of patients with stroke ipsilateral to a hemodynamic carotid stenosis or occlusion did not report episodes of transient cerebral ischemia before the stroke (43). Their first ischemic episode resulted in permanent neurologic deficit.

Kartchner and McRae (56) studied 1,287 patients presenting with an asymptomatic bruit, using a noninvasive test battery of carotid phonoangiography and pulse-time oculoplethysmography. Patients were followed up for 6 to 70 months (average 24). There was a 1.9% incidence of stroke in patients with negative noninvasive studies and an 11.9% incidence of stroke when both noninvasive studies were positive. This suggests that noninvasive carotid testing can determine the potential risk of stroke for an individual patient with an asymptomatic carotid bruit. With this information, the relative risks and benefits of carotid endarterectomy can be weighed. Patients can be followed up serially with noninvasive testing to determine whether there is progression of disease.

Evaluation of the Carotid Circulation Before Coronary Artery Bypass or Peripheral Vascular Surgery

General major surgery. Several centers (57,58) recommend that carotid endarterectomy be performed on pa-

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tients with carotid stenosis before major surgery to prevent a reduction in cerebral perfusion should a large decrease in blood pressure occur. However, the incidence of perioperative cerebral infarction in general surgical procedures is less than 1.0% (59). Even though intraoperative hypotension is frequent, strokes generally develop postoperatively and are most often related to cardiogenic emboli rather than carotid artery occlusive disease (59). The presence of a carotid bruit does not predispose patients undergoing surgery to stroke (60). Ropper et al. (60) found an overall incidence of postoperative stroke of 0.7% in 735 patients undergoing elective surgery. Only one stroke occurred in 104 of these patients with an asymptomatic bruit. However, a bruit does not always indicate carotid stenosis, while high grade stenoses or carotid occlusions may not produce a bruit.

Coronary or vascular surgery. Turnipseed et al. (61) studied 330 patients with carotid Doppler imaging and spectral analysis before peripheral vascular or coronary artery bypass surgery. They found no relation between the incidence of carotid occlusive disease and perioperative stroke in the 170 patients with peripheral vascular disease or the 160 patients with coronary artery disease. Barnes and Marszalek (62) studied 314 patients with noninvasive carotid artery testing before coronary artery bypass surgery. Carotid artery stenosis greater than 50% was found in 13.1% of the patients. Only one-third of these patients had a bruit. There was only one stroke and one transient ischemic attack during surgery, and neither was related to a carotid lesion. Therefore, prophylactic carotid endarterectomy does not appear to be valuable in preventing stroke before major vascular or coronary artery surgery. The one exception may be in patients with a significant decrement in perfusion pressure at the ophthalmic artery. Kartchner and McRae (63) observed a 1% incidence of perioperative stroke in patients with an asymptomatic bruit and normal pulse delay on oculoplethysmography, whereas patients with an abnormal oculoplethysmogram had a 17% incidence of perioperative stroke. Brener et al. (22) noted a 22% incidence of stroke during coronary artery bypass surgery ipsilateral to an occluded carotid artery with reduced ophthalmic artery pressure measured by oculoplethysmography.

Fifty percent of patients with peripheral vascular disease have coincident carotid artery disease (61,64). Screening these patients with noninvasive carotid artery testing is indicated to assess the extent of carotid disease. These patients should be managed on the basis of their neurologic symptoms and hemodynamic factors, but carotid endarterectomy need not precede peripheral vascular surgery in all patients with carotid artery lesions.

There is only a 10% coincidence of carotid artery disease with coronary artery disease (61). This difference between patients with coronary artery disease and peripheral vascular disease probably reflects differences in the pathogenesis of atherosclerotic disease in large peripheral arteries and in the coronary arteries. Peripheral vascular disease has a higher association with hypertension and diabetes, whereas coronary artery disease has a higher association with hyperlipidemia (61). Since the incidence of carotid disease is relatively low in patients with coronary artery disease, all of these patients need not be screened with noninvasive carotid artery testing. Noninvasive carotid artery testing is indicated for patients with cerebrovascular symptoms or a cervical bruit. These patients should also be managed on the basis of their symptoms and the extent of carotid disease.

Indications and risks of carotid endarterectomy. The incidence of fatal myocardial infarction when carotid endarterectomy is performed before coronary artery bypass surgery has been reported to be as high as 33%, whereas simultaneous coronary artery bypass surgery and endarterectomy resulted in no mortality, but up to a 20% incidence of postoperative stroke (65). When both operations are indicated, coronary artery bypass surgery should precede carotid endarterectomy or the two procedures should be performed simultaneously (58,65). In fact, the incidence of myocardial infarction in patients with cerebral transient ischemic attacks is 20%, equal to the incidence of stroke (66), so that these patients should be screened for coronary artery disease as well.

Summary

Noninvasive carotid artery testing can provide physiologic and anatomic information to delineate the extent of atherosclerotic disease at the carotid artery bifurcation. This information can help to decide whether to manage a patient medically or surgically. A noninvasive test battery can be used to discriminate hemodynamically significant obstruction to flow from nonobstructive plaque by assessing flow dynamics at the carotid bifurcation with Doppler ultrasound studies and measuring distal perfusion pressure at the ophthalmic artery. Real time B-mode ultrasonography also visualizes the anatomic configuration of plaque in the carotid artery sinus. Noninvasive testing can be used to screen patients before angiography or as an adjunct to angiography since the two procedures complement one another. Noninvasive testing is of particular value in assessing carotid artery disease in patients with cardiologic disease who are at high risk for complications from invasive procedures.

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References

1. Fisher CM. Transient monocular blindness associated with hemiplegia. Arch Ophthal 1952;47:167–203.
2. Moore WS, Hall AD. Importance of emboli from carotid bifurcation

in pathogenesis of cerebral ischemic attacks. Arch Surg 1970; 101:708–11.

3. Thompson JE, Talkington CM. Carotid endarterectomy. Ann Surg 1976;184:1–15.
4. Weinberger J, Biscarra V, Weisberg MK. Hemodynamics of the carotid artery circulation in the elderly "dizzy" patient. J Am Geriatric Soc 1981;29:402–6.
5. Hennerici M, Aulich A, Sandmann W, et al. Incidence of asymptomatic extracranial arterial disease. Stroke 1981;12:750–8.
6. Fields WS, Maslenikov V, Meyer JS, et al. Joint Study of Extracranial Arterial Occlusion: V. Progress report of prognosis following surgery or nonsurgical treatment for transient cerebral ischemic attacks and cervical carotid artery lesions. JAMA 1970; 211:1993–2003.
7. Marshall J. The natural history of transient ischemic cerebrovascular attacks. Quart J Med 1964;33:309–24.
8. Ziegler DK, Hassanein RS. Prognosis in patients with transient ischemic attacks. Stroke 1973;4:666–73.
9. Pessin MS, Duncan GW, Mohr JP, Poskanzer DC. Clinical and angiographic features of carotid transient ischemic attacks. N Engl J Med 1977;296:358–62.
10. Marshall J. The Management of Cerebrovascular Disease. 2nd ed. Boston: Little, Brown, 1968.
11. Alex M, Baron EK, Goldenberg S, Blumenthal HT. An autopsy study of cerebrovascular accident in diabetes mellitus. Circulation 1962;25:663–73.
12. Friedman GD, Loveland DB, Ehrlich SP Jr. Relationship of stroke to other cardiovascular disease. Circulation 1968;38:533–41.
13. Kane WC, Aronson SM. Cardiac disorders predisposing to embolic stroke. Stroke 1970;1:164–72.
14. Thompson PL, Robinson JS. Stroke after acute myocardial infarction: relation to infarct size. Br Med J 1978;2:457–9.
15. Faught E, Trader SD, Hanna GR. Cerebral complications of angiography for transient ischemia and stroke: prediction of risk. Neurology 1979;29:4–15.
16. Little JR, Farlan A, Modic MT, et al. Intravenous digital subtraction angiography in brain ischemia. JAMA 1982;247:3213–6.
17. Smith JL, Logan DG. The ophthalmodynamometric posture test. Am J Ophthal 1959;48:735–40.
18. Gee W, Oller DW, Amundsen DG. The asymptomatic carotid bruit and the ocular pneumoplethysmography. Arch Surg 1977;112:1381–8.
19. Smith JL. Central retinal and internal carotid occlusions: ophthalmodynamometric differentiation. Arch Ophthal 1961;65:550–2.
20. Weinberger J, Bender AN, Yang WC. Amaurosis fugax associated with ophthalmic artery stenosis: clinical simulation of carotid artery disease. Stroke 1980;11:290–3.
21. DeWeese JA, May AG, Linchik EO, et al. Anatomic and hemodynamic correlations in carotid artery stenosis. Stroke 1970;1:149–57.
22. Brener BJ, Brief DK, Alpert J, et al. A four-year experience with preoperative non invasive carotid evaluation of two thousand twenty-six patients undergoing cardiac surgery. J Vasc Surg 1984;1:326–38.
23. Kartchner MM, McRae LP, Crain V, et al. Oculoplethysmography: an adjunct to arteriography in the diagnosis of extracranial carotid occlusive disease. Am J Surg 1976;132:728–32.
24. Ginsberg MD, Greenwood SA, Goldberg HI. Limitations of quantitative oculoplethysmography and of directional ultrasonography in cerebrovascular diagnosis: assessment of an air-filled OPG system. Stroke 1981;12:27–32.
25. Blackshear WM, Thiele BL, Chikos PM, et al. Detection of carotid occlusive disease by ultrasonic imaging and pulsed Doppler spectrum analysis. Surgery 1979;86:698–706.
26. Keller HM, Meier WE, Anliker M, Kumpe DA. Noninvasive measurement of velocity profiles and blood flow in common carotid artery by pulsed Doppler ultrasound. Stroke 1976;7:370–7.
27. Bloch S, Baltaxe HA, Shoumaker RD. Reliability of Doppler scanning of the carotid bifurcation: angiographic correlation. Radiology 1979;132:687–91.
28. Bone GE, Barnes RW. Clinical implications of the Doppler cerebrovascular examination: a correlation with angiography. Stroke 1976;7:271–4.
29. Barnes RW, Russell HE, Bone GE, et al. Doppler cerebrovascular examination: improved results with refinements in techniques. Stroke 1977;8:468–71.
30. Weinberger J, Biscarra V, Weitzner I, Sacher M. Non-invasive carotid artery testing. Role in management of patients with transient ischemic attacks. NY State J Med 1981;81:1463–8.
31. Ziegler DK, Zileli T, Dick A, et al. Correlation of bruits over the carotid artery with angiographically demonstrated lesions. Neurology (Minneap) 1971;21:860–5.
32. Duncan GW, Gruber JO, Dewey CF Jr, et al. Evaluation of carotid stenosis by phonoangiography. N Engl J Med 1961;292:1124–8.
33. Kistler JP, Lees RS, Miller A, et al. Correlation of spectral phonoangiography and carotid angiography with gross pathology in carotid stenosis. N Engl J Med 1981;305:417–9.
34. Rutherford RB, Hiatt WR, Kreutzer EW. The use of velocity wave form analysis in the diagnosis of carotid artery occlusive disease. Surgery 1977;82:695–702.
35. Brice JG, Dowsedt DJ, Lowe RD. Haemodynamic effects of carotid artery stenosis. Br Med J 1964;2:1363–6.
36. Barnes RW, Nixl L, Rittgers SE. Audible interpretation of carotid Doppler signals: an improved technique to define carotid artery disease. Arch Surg 1981;116:1185–9.
37. Weinberger J, Robbins A. Neurologic symptoms associated with nonobstructive plaque at carotid bifurcation. Analysis by real-time B-mode ultrasonography. Arch Neurol 1983;40:489–92.
38. Weinberger J. Doppler pulse wave form analysis of carotid artery flow in dissecting aortic aneurysm. Arch Neurol 1981;38;256–7.
39. Ackerman RH. A perspective on noninvasive diagnosis of carotid disease. Neurology 1979;29:615–31.
40. Cebul RD, Ginsberg MD. Noninvasive neurovascular tests for carotid artery disease. Ann Intern Med 1982;97:867–72.
41. Comerota AJ, Cranley JJ, Cook SE. Real time B-mode carotid imaging in diagnosis of cerebrovascular disease. Surgery 1981;89:718–29.
42. Fisher CM, Gore I, Okabe N, White PD. Atherosclerosis of the carotid and vertebral arteries—extracranial and intracranial. J Neuropathol Exp Neurol 1965;24:455–76.
43. Schwartz CJ, Mitchell JRA. Atheroma of the carotid and vertebral arterial systems. Br Med J 1961;2:1057–63.
44. Pessin MS, Hinton RC, Davis KR, et al. Mechanisms of acute carotid stroke. Ann Neurol 1979;6:245–52.
45. Busuttil RW, Baker JD, Davidson RK, et al. Carotid artery stenosis: hemodynamic significance and clinical course. JAMA 1981; 245:1438–41.
46. Sundt TM, Sharbrough FW, Piepgras DG, et al. Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy. Mayo Clin Proc 1981;56:533–43.
47. Allen GS, Preziosi TJ. Carotid endarterectomy: a prospective study of its efficacy and safety. Medicine 1981;60:298–309.
48. The Canadian Cooperative Study Group. A randomized trial of aspirin and sulfinpyrazone in threatened stroke. N Engl J Med 1978;299:53–9.
49. Toole JF, Truscott BL, Anderson WW, et al. Report of the Joint Committee for Stroke Facilities. VII. Medical and surgical management of stroke. Stroke 1973;4:269–320.
50. Blaisdell WF, Clauss RH, Galbraith JG, et al. Joint Study of Extracranial Arterial Occlusion. IV. A review of surgical considerations. JAMA 1969;209:1889–95.
51. Fisher CM. Vertigo in cerebrovascular disease. Arch Otolaryng 1967;85:529–34.

52. Fisher CM. Occlusion of the vertebral arteries. Arch Neurol 1970;22:13–9.

53. Wolf PA, Kannel WB, Sorlie P, McNamara P. Asymptomatic carotid bruit and risk of stroke. The Framingham Study. JAMA 1981; 245:1442–5.

54. Heyman A, Wilkinson WE, Heyden S, et al. Risk of stroke in asymptomatic persons with cervical arterial bruits: a population study in Evans County, Georgia. N Engl J Med 1980;302:838–41.

55. Thompson JE, Patman RS, Talkington CM. Asymptomatic carotid bruit: long-term outcome of patients having endarterectomy compared with unoperated controls. Ann Surg 1978;188:308–16.

56. Kartchner MM, McRae LP. Noninvasive evaluation and management of the "asymptomatic" carotid bruit. Surgery 1977;82:840–7.

57. Treiman RL, Foran RF, Shore EH, et al. Carotid bruit: significance in patients undergoing an abdominal aortic operation. Arch Surg 1973;106:803–5.

58. Rice PL, Pifarre R, Sullivan HJ, et al. Experience with simultaneous myocardial revascularization and carotid endarterectomy. J Thorac Cardiovasc Surg 1980;79:922–5.

59. Hart R, Hindman B. Mechanisms of perioperative cerebral infarction. Stroke 1982;13:766–73.

60. Ropper AH, Wechsler LR, Wilson LS. Carotid bruit and the risk of stroke in elective surgery. N Engl J Med 1982;307:1388–90.

61. Turnipseed WD, Berkoff HA, Belzer FO. Postoperative stroke in cardiac and peripheral vascular disease. Ann Surg 1980;195:365–8.

62. Barnes RW, Marszalek PB. Asymptomatic carotid disease in the cardiovascular surgical patient: is prophylactic endarterectomy necessary? Stroke 1981;12:497–500.

63. Kartchner MM, McRae LP. Carotid occlusive disease as a risk factor in major cardiovascular surgery. Arch Surg 1982;117:1086–8.

64. Weinberger J, Biscarra V, Weisberg MK, Jacobson JH. Factors contributing to stroke in patients with atherosclerotic disease of the great vessels: the role of diabetes. Stroke 1983;14:709–12.

65. Gorman LC. The preoperative patient with an asymptomatic cervical bruit. Med Clin North Am 1979;63:1335–41.

66. Heyman A, Wilkinson WE, Hurwitz BJ, et al. Risk of ischemic heart disease in patients with TIA. Neurology 1984;34:626–30.