Noninvasive Techniques for Diagnosis of Carotid Artery Disease

A VARIETY of noninvasive techniques have been advocated for the detection of carotid artery disease. These techniques may be divided into 2 major groups: 1) those that assess the carotid bifurcation directly (phonoangiography, audio-frequency analysis of bruits, Doppler ultrasonic scanning, and real-time B-scan imaging) and 2) those that assess the hemodynamics of the carotid system distal to the bifurcation and that indirectly provide information about its morphology (ophthalmodynamometry, thermography, oculopneumoplethysmography, and directional Doppler flow studies).

An increasing number of reports suggest that these techniques can be utilized in varying combinations to obtain information about the extent of atherosclerosis at the carotid bifurcation, particularly for lesions that produce 60 to 75% or greater stenosis — in which hemodynamic changes might be expected. There are certain situations in which this information may be of clinical merit, but one must be cautious about allowing this information to indicate one type of therapy over another. It is not with the studies themselves that there is concern but rather with the way in which the information obtained from these studies is currently being used. It is in this area, especially, that technologic advances have outpaced clinical knowledge.

Why is there so much interest in these noninvasive studies? The major reasons seem relatively clear: 1) the belief that carotid atherosclerosis is bad, 2) a bias that one form of therapy (that is, surgery) is more effective for certain types of lesions than are other methods, and 3) the belief that these studies may be useful in the selection of patients for angiography and in planning medical or surgical treatment. In determining the clinical usefulness of these techniques, certain observations about carotid atherosclerosis must be considered: namely, it is a relatively common disorder, symptoms may occur when only minimal disease is present, and severe disease may be present and not produce any symptoms. These observations suggest that there must be more to evaluating patients (and the worth of any noninvasive instrument) than merely being able to describe the pathologic anatomy of the carotid arteries. The pathologic change may be, and likely is, responsible for some of the symptoms we see, but its presence alone cannot be used as the major criterion for judging the need for any treatment modality (medical or surgical), although this clinical practice is becoming more widespread.

The problem is not only whether these studies can be used to detect carotid atherosclerosis that can be treated, but also whether these studies can be utilized to predict which patients should have their carotid atherosclerosis treated. If not, physicians will continue to find themselves in the position of detecting many patients with atherosclerosis and not knowing what to do with them.

Although it may be a logical conclusion that severe atherosclerosis poses a risk for subsequent infarction, the number of studies that focus critically on this issue are few. Some clinicians have taken as fact that severe atherosclerosis imposes a significant risk, and that the risk of surgical treatment is less than that of the natural history of the disease. That may be so, but the proof is lacking, especially in view of the reported morbidity of carotid endarterectomy which approaches 21% in some community hospitals.

Clinical Applications

At present, the applicability of these studies to clinical practice will vary, and depends on the clinician’s predetermined bias as to the efficacy of the various available treatment modalities. This may be illustrated by reviewing three common clinical situations — 1) evaluation of symptomatic patients, 2) routine screening of asymptomatic patients, and 3) screening of asymptomatic patients before surgery.

For patients with clear, recurrent carotid system transient ischemic attacks (TIA), these studies are of little help, because no single study or combination of studies yet devised can detect minimal atherosclerosis with ulceration, differentiate severe stenosis from occlusion, or detect intracranial atherosclerosis. Any of these conditions may be present and each may require a different therapeutic approach. For carotid system transient ischemic attacks most clinicians would recommend carotid arteriography regardless of the results of the noninvasive studies. Noninvasive studies may be of some value for the patient with a negative neurovascular examination and a single carotid system TIA (which may have a variety of pathogenetic mechanisms). For such patients, the studies may help direct the clinician’s attention to the responsible lesion, and they may — depending on the
bias of the attending physician — be of help in determining which patients need to have angiography.

The usefulness of these studies in patients with non-hemispheric TIAss is uncertain as these episodes are often nonfocal in character and they are encountered in an age group in which carotid atherosclerosis is common. Are the symptoms and the atherosclerosis merely coincidental unrelated findings, or are they causally related? Unless noninvasive studies are utilized in a prospective and controlled manner, the question may never be answered — yet this knowledge is of vital importance to the clinician.

In routine screening of asymptomatic patients, the problem becomes increasingly complex, for although noninvasive studies can demonstrate severe atherosclerosis, they have not been used in a prospective manner to determine which patients are at risk. Clinical practice, when such patients are encountered, varies with the physician. If one believes that the evidence is clear enough to warrant treating all such patients (medically or surgically), then these studies may be of help. It is more rational first to employ these techniques to study the natural history of asymptomatic disease and obtain the data necessary to make a clinical judgment about the need for treatment.

A similar problem is the screening of asymptomatic patients before other major surgical procedures. This problem is compounded because the actual risk of cerebral infarction following surgery for other conditions is not known. In 2 studies of aorto-iliac surgery, the cerebral infarction rate seemed to be low, and in several studies involving open-heart surgery, it is higher than was previously thought.

The clinical usefulness of these studies depends on the predetermined bias of the physician.

Perspective for the Future

To design the ideal noninvasive study, in addition to providing the obvious factors of safety and economy, it would be necessary to assess accurately the carotid bifurcation for all degrees of disease (from none to occlusion) and to provide information about the morphology and dynamics of the intracranial circulation. In asymptomatic patients such studies must be proven useful in determining which group of patients is at greatest risk for symptoms of cerebral ischemia and infarction.

Studies assessing these various noninvasive techniques must be reported so that clinicians can judge how best to make use of them. Initially, interest is centered on obtaining pathologic-angiographic correlations. Studies of these procedures must make clear: 1) the percentage which is technically unreliable; 2) the accuracy in assessing the carotid bifurcation with normal versus minimal disease, ulceration, stenosis of less than 50%, stenosis of more than 50%; and severe stenosis versus occlusion; and 3) the ability of the study to assess the morphology and dynamics of the intracranial circulation. Most studies to date indicate that these techniques are useful in detecting hemodynamically significant lesions but are of little value in detecting minimal stenosis or ulceration of atherosclerotic plaques or in differentiating severe stenosis from occlusion.

The potential value of these studies is great, but enthusiasm must be tempered by the observation that they are being applied in clinical situations prematurely. As clinical instruments, they can provide information about the presence of moderately severe carotid atherosclerosis, and are tools that can potentially provide much-needed data concerning the natural history of a common disease process. Used in this manner, these new techniques will provide a rational basis on which to make important therapeutic decisions, and therein lies the most promising clinical application of noninvasive techniques.

Burton A. Sandok, M.D.
Associate Professor of Neurology
Mayo Medical School
Rochester, Minnesota 55901

References
3. Reid JM, Spencer MP: Ultrasonic Doppler technique for imaging blood vessels. Science 176: 1235-1236 (June 16) 1972
10. Capistrant TD, Gummit RJ: Detecting carotid occlusive disease by thermography. Stroke 4: 57-64 (Jan-Feb) 1973
17. Kartchner MM, McRae LP: Noninvasive evaluation of
A REVIEW of the currently available animal models for cerebral ischemia and stroke was the first topic of the conference. Dr. Erland Nelson, Chairman of the Conference, mentioned that Cooper in England in 1836 had tied an animal’s carotid arteries to find what happened and therefore should be given credit for establishing the first stroke model. An animal model for stroke should have the following characteristics: sudden onset, progression and regression of symptoms as seen in humans, be economical and usable prospectively. In the human, stroke is associated with many variables, including age, atherosclerosis, and high blood pressure and some of these variables can be reproduced in animals, many cannot.

Dr. Gaetano Molinari reviewed the history of stroke models and mentioned that the ideal model should resemble, as closely as possible, the clinical picture seen in humans with cerebrovascular accidents. Most stroke models used healthy young animals but stroke in humans largely occurs in elderly individuals. In older experimental animals, especially older gerbils and possibly the stroke-prone hypertensive rat, there may be a higher incidence of stroke. Aged animals apparently have less elasticity in their cerebral vessels and higher cerebrovascular resistance. Dr. Molinari concluded that no current animal model for stroke perfectly mimicked the human situation. In the usual models, cerebral ischemia is produced by a variety of means including embolization, hypotension, either by reducing general blood pressure or occluding all neck vessels to the head, and by individual cerebral vessel occlusion. In most experimental animals, vessel occlusion produces highly variable results depending on the animal. Many of the animals have wide variations in cerebral collateral circulation. Dr. Molinari expressed belief that clot emboli perhaps have the advantage in producing cerebral infarction as they do not require invasive procedures; their disadvantage is that they are unpredictable in the amount and site of cerebral infarction produced.

Duty of Pathologist

Dr. John Moossy reviewed the role of the morphological pathologist in the production of experimental stroke and indicated that it was the duty of the pathologist to validate the results of experimental stroke in each model, using all the facilities available including macroscopic, microscopic and electron microscopic examination. He said the electron microscope is best for the early detection of cerebral ischemia as it can distinguish reversible from irreversi-
Noninvasive techniques for diagnosis of carotid artery disease.
B A Sandok

Stroke. 1978;9:427-429
doi: 10.1161/01.STR.9.5.427

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/9/5/427.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/