Identifying Clinically Relevant Carotid Disease

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With the advent of magnetic resonance angiography (MRA) the diagnosis of carotid artery disease has clearly become a polymodal process, one that can be done by several methods. Twenty-five years ago x-ray arteriography (XRA) was the only practical test for carotid stenosis, and it was performed primarily by direct carotid puncture. Today not only can we also assess the carotid artery using noninvasive ultrasound and magnetic resonance techniques, but each modality can be applied in several different ways. In addition, preliminary studies suggest that spiral computed tomography for carotid evaluation is a promising addition to the noninvasive armamentarium.

The North American Symptomatic Carotid Endarterectomy Trial (NASCET) has convinced skeptics that identifying and operating on appropriately severe common carotid bifurcation lesions can lead to a risk reduction for stroke. Attention now focuses on which methodologies are the best for identifying clinically relevant carotid artery disease. This investigative process is necessary, for one cannot — nor will the health-care system in the future — allow multiple costly diagnostic studies to select a patient for carotid endarterectomy. In this issue of Stroke a neuroradiological and vascular surgical research team from the Hospital of the University of Pennsylvania (HUP) reports its blinded-reader comparison of MRA and duplex ultrasonography (DUS) in the diagnosis of carotid artery luminal diameter stenosis in the range of 70% to 99%.

The NASCET findings are too often treated as the end of the road in our quest for understanding what constitutes risk for stroke in patients with carotid artery disease, when in fact the results provide an important beginning. A stenosis of 70% was never presented as a threshold by the NASCET investigators but as the lower limit for the percent stenosis category for which the authors had meaningful results at the time of their report. Although study of their 30% to 69% stenosis group continues and the results of the Asymptomatic Carotid Atherosclerosis Study are still pending, too many clinicians are taking a 70% stenosis as the threshold for clinical action in symptomatic and asymptomatic patients. Moreover, noninvasive identification of this degree of disease has been (over)simplified for some workers by the suggestion that at the point of stenosis MRA signal dropout indicates sufficient hemodynamic change to represent at least a 70% stenosis. The data of the HUP authors and our experience are in agreement that this "ain't necessarily so." A quarter of the cases with signal dropout in the HUP report had less than a 70% stenosis, including one patient with a 55% stenosis. It should be kept in mind that the presence and extent of segmental signal loss is a function both of physiological and imaging factors.

Selective use of NASCET data to provide a rationale for acting on what are probably nonspecific MRA signs creates the risk of promoting an epidemic of endarterectomy that could rival that which precipitated NASCET in the first place. Already there has been a major rise in the number of endarterectomies in some centers. At the Massachusetts General Hospital from 1985 to 1989 the number fell from 231 to 138 and has now risen to about 250 annually. This represents almost a doubling since before the NASCET report. At another Boston hospital the number has increased from 34 in the year before the release of the NASCET report to 112 for the first 7 months of 1993. The projected total for all of 1993 is 134, or a fourfold increase since 1990. A neuroradiologist at a suburban Boston hospital recently said that he no longer knows what the MRA findings in advanced carotid disease actually mean, because the surgeons operate without an XRA on any patient with signal dropout, presuming the stenosis to be at least 70%.

The NASCET data confirm what had been suggested previously, that progression to more severe carotid disease represents increased risk of ischemic events. Within the 70% to 99% stenosis category the NASCET investigators found increasing benefit of endarterectomy with increasing severity of disease. The actual degree of stenosis that constitutes the necessary environment to initiate the stroke process remains unknown. It is possible that it will be less than a 70% stenosis; current ongoing studies should tell us. How-
ever, it also is possible that only anatomically tighter stenoses are clinically relevant; new studies of different design may be required to clarify this issue. Of the two possibilities, our experience favors the latter, and the issue might only be resolved by using residual lumen diameter measurements, rather than percent stenosis calculations, to determine degree of disease.

Calculation of percent stenosis, as used by NASCET, takes the residual lumen diameter at the point of maximal stenosis (M) as a percentage of the diameter of the normal distal segment (D). Percent stenosis = \(1 - \frac{M}{D}\) \times 100. This measurement creates a problem in comparing one patient or physiological situation to another. The distal internal carotid artery diameter is not the same in all individuals; radiographically in any stroke-age individual it may vary along its distal course, and it narrows with very severe disease. When the distal internal carotid measures 5, 6, and 7 mm, a 70% stenosis correlates with a 1.5-, 1.8-, and 2.1-mm residual lumen diameter, respectively. While a lumen is so tight that flow falls, the distal internal carotid artery segment will narrow, which makes the calculated percent stenosis less severe. We wonder if some of the cases in NASCET that were thought to have a 70% stenosis might actually have had more severe disease, but because of narrowing of the distal segment the degree of disease might have been underestimated.

Following the lead of C. Miller Fisher we routinely measure residual lumen diameters. We rarely see a stroke in association with a carotid artery lesion that has a residual lumen diameter greater than 1.5 mm, and we uncommonly see a stroke when it is greater than 1.0 mm. Thanks to the circle of Willis, embolic events account for the majority of strokes in patients with carotid disease rather than compromise of flow per se.

Our current observations suggest that stroke from carotid stenosis may occur commonly in association with an internal carotid artery lumen that is so tight that flow falls, which presumably abets sludging, thrombus formation, and embolism. By carotid Doppler frequency and velocity measurements we find that internal carotid artery flow begins to fall when the residual lumen diameter is 0.5 to 0.7 mm. This is a long way from a 70% stenosis.

Because a percent stenosis measurement does not have a uniform anatomic substrate, it should not have the same hemodynamic or pathophysiological relevance in all patients or the same relation to stroke risk. Moreover, because MRA and ultrasound findings are derived from physiological events, one would not be surprised to find at least occasional discrepancy between the arteriographic percent stenosis measurements and the noninvasive findings. Residual lumen diameter determinations, however, if appropriately corrected for magnification, should reflect the same anatomic situation between patients and more likely be associated with similar hemodynamic changes. It would seem to be the most physiologically meaningful measurement to use for any future study where precision is important. This comment is not made without failing to realize that correcting for magnification (or minification in digital subtraction angiographic studies) is not always straightforward.

As the NASCET data emphasize, the tightness of stenosis has clinical relevance and is important to document. Therefore, the imperative in carotid diagnosis should be to determine as accurately as possible the precise degree of stenosis, not a range. Only then will one be able to make the appropriate clinicopathological correlation that will demonstrate if a threshold for stroke related to lumen size exists and to routinely identify it if one does. MRA cannot yet provide a precise degree of stenosis, either as a percent or actual millimeter measurement. It cannot, therefore, be used reliably to give more than a range of disease, and within this range it certainly is limited in providing data on progression. The HUP report has adequately addressed the diagnostic accuracy of MRA for a range of disease, but we believe more precise diagnostic information about severity of stenosis is required if one is to build on the NASCET data.

We also consider that DUS does not represent the full power of carotid ultrasound studies. With the introduction of duplex imaging and color-flow Doppler, many laboratories have dropped continuous-wave Doppler examinations as well as indirect testing (eg, periorbital Doppler and oculoplethysmography studies). We believe that this is a mistake. A full ultrasound battery can be used to provide relatively precise information on the degree of carotid stenosis, not just a range, and to monitor the carotid system for lesions both at the bifurcation and beyond it that put a patient at risk for stroke. To adequately assess the extracranial vessels for lesions that can cause ocular or hemispheric ischemic events, one cannot restrict his or her practice to “bifurcology” but must use direct tests, which monitor the bifurcation, and indirect tests, which assess distal circulatory beds.

In a number of situations the indirect tests are an important complement to the direct tests in helping to decipher the actual severity of disease at the common carotid bifurcation, such as when a partially hypoechoic plaque limits the B-mode information, shadowing or tortuosity confound the color-flow image, and/or a stenosis is so tight that Doppler-generated frequencies and velocities have fallen toward or back to normal values. When the periorbital Doppler study, for example, shows reversal of flow in the supraorbital vessels at the orbital rim and the direct tests indicate an advanced but indeterminate degree of internal carotid artery stenosis, the overall findings are essentially pathognomonic of a residual lumen diameter of 1 mm or less. The indirect tests also can identify lesions distal to the bifurcation that cause hemodynamic changes that might not be detected by the standard direct test tools. These include distal cervical carotid dissection, fibromuscular disease, siphon atheromatous stenosis, and giant cell arteritis.

In our experience continuous-wave Doppler has several features that make it an important complement to pulsed-wave Doppler duplex and color-flow imaging. With a continuous-wave probe one can rapidly scan the carotid almost in its entire cervical extent; to insonate the same area with a duplex probe is a more laborious stepwise process, so that the examiner may not routinely sample the distal cervical carotid. Continuous-wave Doppler does not have the same aliasing problems as does pulsed-wave Doppler, so that continuous-wave Doppler can provide
more accurate peak systolic measurements in areas of severe stenosis. Lastly, pulsed-wave Doppler velocity determinations require operator-dependent selection of an angle of insonation. Especially in patients with tortuous vessels, this might result not only in erroneous velocity values but introduce a potential reproducibility problem. Indeed, we find in some patients that continuous-wave frequency determinations are more consistent over time than are pulsed-wave Doppler velocity measurements and therefore are more useful in following a stenosis for evidence of progression, particularly to very severe degrees of disease.

By using a battery of indirect and direct tests that includes direct continuous-wave Doppler, one begins to realize the full potential of noninvasive carotid ultrasound studies and to provide the capability for relatively precise noninvasive estimation of residual lumen diameter. In our Neurovascular Laboratory, when the stenosis is less than 2.5 mm we use the combined physiological data to derive an estimated residual lumen diameter that we report to the nearest .25 mm or .5 mm (depending on the internal consistency of the data). As part of a recent study we found excellent correlation between ultrasound/arteriographic residual lumen diameter determinations. For 30 internal carotid arteries \( r = 0.96 \); for 10 of these vessels with residual lumen diameters 1.5 mm or less by arteriography, the blinded ultrasound findings were correct within .25 mm in 80% of the cases and within .5 mm in 100%.

"Today the position of the stroke field relative to carotid disease is similar to that of Gowers in 1885 relative to the exploding knowledge about the functional anatomy of the brain. To borrow his words, our knowledge "is at once more than we can use and far less than we need."16 What we believe NASCET makes clear is the need to know how the precise degree of carotid stenosis correlates with stroke risk and how to use our invasive and noninvasive tests to obtain the most precise data. Because MRA currently lacks precision and is subject to many signal loss and signal-look-alike artifacts, it can provide misleading diagnostic information if used alone in patients with advanced stenotic disease.17 When MRA and appropriate noninvasive results correlate, one can have greater certainty of the results. Of course, arteriography remains the criterion.14 Whether it can be omitted if the MRA and DUS results are concordant will depend on the quality and comprehensiveness of the two studies, as well as the patient's suitability for arteriography. We agree with our HUP colleagues that concordant MRA and DUS alone will not necessarily obviate the need for catheter arteriography, but in some patients concordant MRA and a complete ultrasound battery might.

References


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