EDITORIAL COMMENTARIES

Are two heads better than one?

In the paper by Wardlaw et al (this issue, pp 155–160) the authors consider the question as to whether or not the severity of carotid bifurcation stenosis has some bearing on reader accuracy when interpreting magnetic resonance angiography. The answer is yes, assessment becoming less accurate with increase in severity of the stenosis. If we accept that there is an increased risk of complication from conventional angiography in patients with more severe stenoses then the importance of accurate non-invasive diagnosis to triage patients before carotid endarterectomy is realised.

The fact that there are so many different ways of performing magnetic resonance angiography implies that there is no one single best technique. As the authors point out, even the widely held “gold standard” of intra-arterial angiography is flawed.

Despite all the above variables, the attempt of Wardlaw et al to report interobserver variability in the detection of signal gap and % stenosis in a Doppler prescreened symptomatic population, raises some interesting questions and recommendations for radiologists. Quality assurance issues are raised in this paper in addition to stressing the importance of familiarity with variations in technology, leading to a previously published recommendation to calibrate different systems against conventional angiography. This paper hints at the importance of knowing local between reader variability and suggests that consensus opinion may be helpful when assessing the degree of carotid stenosis; having first decided which method—CCA (preferred by the authors), NASCET, or ECST—to use when interpreting magnetic resonance angiography. There are definite recommendations for the more widespread availability of picture archival systems (PACS), in the United Kingdom, for the electronic transmission of magnetic resonance angiography images which constitute large data sets.

The paper confirms an association, previously described in the literature, between signal drop out and a greater degree of stenosis, most likely on the basis of turbulent flow—with the caveat that signal drop out may also be seen with tortuous vessels alone. Taken in association with digital flow signal in the cervical internal carotid artery signal drop out may be used to help distinguish stenosis from occlusion.

The authors again show that magnetic resonance angiography consistently overestimates the percentage stenosis and that further studies still need to be done to see if magnetic resonance angiography is more accurate, if more expensive, than say a second ultrasound examination, in identifying those patients with an 80%–99% stenosis who might most benefit from carotid endarterectomy.

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Diagnosis of chronic peripheral neuropathy

“The golden rule is that there are no golden rules.” (George Bernard Shaw: Maxims for Revolutionists)

The annual incidence of peripheral neuropathy is at least 118/100 000, sufficiently common to justify investigating more efficient strategies for its assessment. The Amsterdam Neuromuscular Centre (pp 205–209 of this issue) are to be congratulated on their pioneering audit of their own guidelines for diagnosing chronic polyneuropathy. They have castigated themselves for doing neurophysiological studies, which did not contribute to the diagnosis in 48%, and ancillary investigations, which were unhelpful in 51%. They conclude that in the presence of a known diagnosis—for instance, of diabetes mellitus—the occurrence of a typical polyneuropathy does not require further investigation. If the diagnosis is not obvious, investigations (or, one might say, more thorough history taking) should be aimed at revealing diabetes, alcohol misuse, renal failure, drug toxicity, and possibly HIV infection. In their revised guidelines the Amsterdam group defer neurophysiological studies until after these investigations, considering their value to be in identifying demyelinating neuropathies and the presence of subclinical motor involvement in clinically pure sensory neuropathies.

No one would disagree that the process should begin with a thorough clinical assessment in which the history and examination will often reveal the diagnosis, as with the history of pes cavus in a parent or the stigmata of liver disease in the alcoholic. However there is a danger in accepting a coincident systemic illness as the cause of a neuropathy. For example, both vasculitic neuropathy and chronic inflammatory demyelinating polyradiculoneuropathy may occur in diabetes mellitus. Many would argue that the neurophysiological distinction of axonal from demyelinating neuropathy helps to direct the subsequent
investigations and should be retained at an earlier stage in the process and not relegated as now proposed by the Amsterdam group. Furthermore the value of the negative investigation should not be underestimated. The symptoms of peripheral neuropathy are distressing and patients want to be reassured that all treatable causes have been considered.

The authors set out guidelines for chronic polyneuropathy assuming that the first step in the assessment, the identification of a multiple mononeuropathy, has been undertaken. Unfortunately vasculitic neuropathy may present as a symmetric polyneuropathy rather than a multiple mononeuropathy and the Amsterdam algorithm would risk missing such a diagnosis before disabling deficits have accumulated. Pointers to a diagnosis of vasculitis would be asymmetry, stepwise progression, pain, systemic illness and a raised erythrocyte sedimentation rate but sometimes all these are absent and the diagnosis can only be made by nerve biopsy. However, sural nerve biopsy should only be performed if the diagnosis cannot be achieved by other means because of the risk of persistent pain at the biopsy site in a third of patients.

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