Letters to the Editor

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Evaluation of Carotid Artery Stenosis by Power Doppler Imaging

To the Editor:

Steinke and coworkers[1] recently reported on the possible advantages of power Doppler imaging (PDI) for quantification of stenoses of the internal carotid artery (ICA): similar to angiography, it should be possible to assess the degree of stenosis of the ICA as the percentage of the diameter reduction from the longitudinal image.

We have tested this examination procedure by comparison with the usual, validated sonography criteria on 40 cases of ICA stenosis. Included in the observation period of the study were all consecutive stenosis findings for which color-coded duplex sonography (CCDS) fulfilled the following criteria: (1) detection of a local flow acceleration in the ICA, (2) peak flow velocity of $\geq$1 m/s (measurement of the jet flow at the stenosis maximum or, in cases of sound extinction there, directly distal from it), and (3) detection of flow disturbances. Quantification was achieved using the known Doppler criteria.[2]

In 22% of the cases an adequate evaluation by PDI was not possible: in 6 stenoses the residual lumen could not be demonstrated because of sound extinction; in an additional 3 stenoses, the vessel wall could not be imaged adequately for determination of the degree of stenosis. In the remaining cases the degree of stenosis was regularly underestimated in comparison with the Doppler criteria (Figure 1). Figure 2 illustrates this for the example of a high-grade stenosis of the ICA: the stenosis is very poorly demonstrated in the PDI (panel A). Angiography and conventional CCDS findings with determination of the peak systolic velocity were in agreement, and both revealed the high-grade stenosis (panels B and C).

The problem of underestimation of stenoses in color Doppler images is known from conventional CCDS. Technical factors are responsible for the fact that the residual lumen of a high-grade stenosis is mostly too widely demonstrated: the color signal shown on the monitor of the ultrasound apparatus is not a direct representation of the detected flow but rather the result of an extensive electronic image processing.[2] As a consequence of spatial processing, measured volumes in the proximity of a rapidly perfused stenosis canal in which no flow is actually measured can in fact appear to give rise to flow signals. Since this function of the image processing may produce differing results with different ultrasound systems, the findings of independent investigators may vary. For our investigations we used the same type of apparatus (Acuson 128 XP) as Steinke and coworkers, and thus this argument cannot be used to explain the differing results. We also used the same apparatus settings as recommended by this group as far as the appropriate information was given.

The conclusion of Steinke et al that "PDI further improves the assessment of ICA stenosis" cannot be confirmed, even when our results are not taken into account. (1) There is only a moderate correlation between PDI and angiography findings,[1] whereas good correlations have been demonstrated previously in several studies on comparison of Doppler sonography findings with those of angiography[3–5] or endarterectomy specimens.[6,7] (2) The PDI findings are not validated: angiography was held to be responsible for the only modest correlation between PDI and angiography. What then can be taken as the reference standard? (3) The local degree of stenosis was determined sonographically according to the European Carotid Surgery Trial, thus only those angiographic methods—not an amalgamation of various procedures—may be taken for comparison. (4) Sonographic longitudinal images are in principle poorly reproducible because they are not obtained under standardized conditions. If the method is to be used in spite of this, then first of all the reproducibility of the findings and the agreement between different observers must be confirmed. (5) The finding presented as an example by Steinke et al in their Figure 1 confirms our reservations: according to PDI the degree of stenosis amounts to 38%; according to Doppler criteria at a peak flow velocity of 1.7 m/s (illustration of the same finding in another article by Steinke and Hennerici[8]), a stenosis of almost 70% exists.

In conclusion, the method of Steinke et al[1] thus rather adds to the already existing "chaos in methodology" for measuring carotid stenosis.[9] CCDS is—also according to our experience with a large number of cases—currently able to replace preoperative angiography in the majority of cases of carotid stenosis; however, this is possible only through use of validated criteria for stenosis.

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Stenosis grade according to PDI

80-89							
70-79							3
60-69						3	2
50-59				2	3	2	1
40-49				3	3	1	
30-39				4	1	1	
20-29				2			
	20-29	30-39	40-49	50-59	60-69	70-79	80-89

Stenosis grade according to Doppler criteria

Figure 1. Comparison of PDI and Doppler criteria for estimation of the degree of ICA stenosis.

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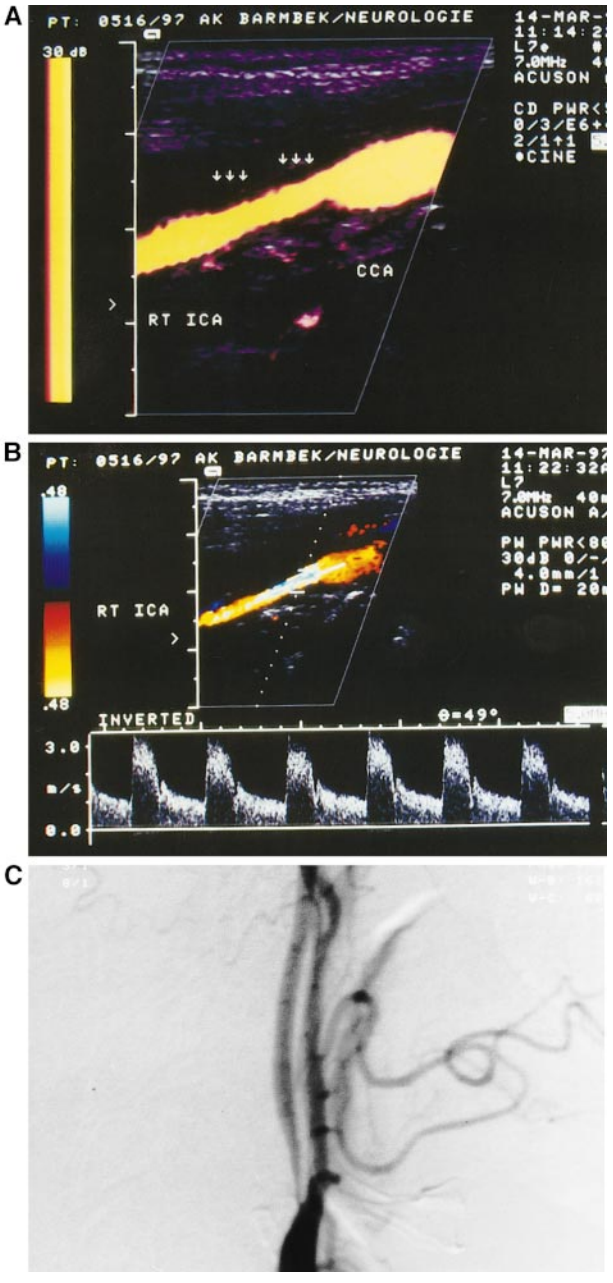

Figure 2. Comparison of PDI, Doppler criteria, and angiography for a high-grade stenosis of the ICA (sonography was performed before and after angiography and revealed identical results). A, Power Doppler mode. B, CCDS in velocity mode with determination of the peak systolic velocity. C, angiography (selective digital subtraction angiography). (We are grateful to Prof Dr H. Zeumer, head of the Department of Neuroradiology, University Hospital Hamburg-Eppendorf, for kindly providing the angiography image.)

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Response

We appreciate the interest of Dr Arning in the results of our recent study.[1] In an attempt to reevaluate the diagnostic significance of PDI, he has compared this technique to "usual Doppler criteria" in a sample of selected cases but without angiographic confirmation and found its utility less convincing than concluded in our study. Unfortunately, the Doppler criteria used by Arning and coworkers have not been published in an original scientific paper but refer to personal experiences of the author of the letter published in a German textbook.[2] The criteria also do not correspond with those recently proposed by a large board of experts in an international consensus meeting.[3] Without vigorous testing and exactly defined criteria, however, any classification of the degree of carotid stenosis based on different criteria such as hemodynamics (ie, Doppler) and morphology (ie, duplex ultrasound and angiography) is misleading; eg, a peak systolic velocity of 170 cm/s alone cannot simply be considered a valid criterion for a 70% stenosis.

In contrast to our study, Arning does not provide systematic data but instead argues from a small collection of cases with casual angiograms, such as those illustrated in the Figure. Although he claims to use the same instrumental setting as we did in our study, it is obvious from the illustration that the gain of power for Doppler color signals was inadequately adjusted, leading to overestimation of the intrastenotic lumen diameter and to underestimation of the degree of stenosis, respectively. The observation of a somewhat lower rate of adequate visualization of the intrastenotic lumen (85% in 40 ICA stenoses versus 92% in 128 stenoses in our trial) and the reported difficulty in displaying high-grade stenosis may simply reflect selection bias and problems in technology. Because corresponding data from both velocity and amplitude modes are not reported, the direct comparison with our study is impossible, and the discussion missed the crucial difference made between measurement of the local degree of stenosis in sonography and angiography. The latter uses an approximation of the distance between the vessel walls whereas the former directly images wall and plaque texture.[4] This was demonstrated by the inclusion of different reference methods to validate PDI results in our trial and should not be misinterpreted as "amalgamation of various procedures."

Arning is correct when he states that the correlation of PDI and angiography was only moderate. However, angiographic overestimation using the local degree of stenosis (European Carotid Surgery Trial) and underestimation using the distal degree of stenosis (North American Symptomatic Carotid Endarterectomy Trial) were not accounted for.[5] However, if angiography is referenced and the procedure of measurement defined, PDI studies correlate very closely with angiographic images. Different ultrasound methodologies must also be defined and carefully used for classification purposes: procedures imaging structural or flow conditions (2-D and 3-D tests) should not be mixed with Doppler recordings from selected sample volumes (1-D). Such inappropriate dimensional comparisons have repeatedly shown a wide variability of results and have often contributed to the common view of ultrasound as a subjective procedure with poor reproducibility. "Homemade" Doppler criteria actually bear a great responsibility in supporting this poor reputation, whereas

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appropriately used objective imaging ultrasound procedures are a strong shield in the battle against methodological chaos.

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Two Chinese Patients With Vertebrobasilar Dolichoectasia

To the Editor:

We read with great interest the article, "Posterior Circulation Infarcts in Patients With Vertebrobasilar Dolichoectasia," by Passero and Filosomi.[1] We would like to make the following comments and describe 2 Chinese patients with vertebrobasilar dolichoectasia (VBD) who presented with features of vertebrobasilar stroke. (There is a minor printing mistake in Table 1 of the article, since 12 patients instead of 11 had prior transient ischemic attacks.[1])

First, the authors suggested the following 2 pathophysiological mechanisms of infarction in patients with VBD: (1) infarcts in distal territories (including posterior cerebral artery territory) associated with artery-to-artery embolism and (2) brain stem/cerebellar infarcts associated with branch atheromatous disease. Nevertheless, the authors postulated in the following paragraph that slow flow and distortion were related to infarcts in the posterior cerebral artery territory and that infratentorial infarcts were related to distortion and stretching of the branches of the basilar artery. Finally, the authors also pointed out the importance of superimposed atheromatous changes in precipitating ischemia in pateints with VBD. Thus, we remain confused as to the most likely pathophysiological mechanisms for ischemia in VBD.

Second, VBD is an uncommon vascular anomaly, but the authors were able to study 40 consecutive stroke patients with associated VBD and compare them with another 40 VBD patients without stroke.[1] We are interested in knowing the frequencies of VBD in both stroke patients and patients without stroke. In addition, we would like to know how the authors collected the 40 VBD patients without stroke. Was the diagnosis of VBD made in postmortem examinations? If the VBD was diagnosed by (CT) or MRI, what were the presenting symptoms and why were neuroimaging tests indicated?

The Queen Mary Hospital of Hong Kong is a regional hospital serving a population of one-half million. Stroke patients are routinely assessed by a member of the Division of Neurology, who collects prospective data for our stroke database and recommends appropriate management. Our stroke database has collected information from 358 stroke patients between January 1997 and May 1998, and ischemic stroke affected 275 of these patients. Brain stem/cerebellar infarctions occurred in 25 patients. During this period, we have encountered 2 Chinese patients with VBD. The first was a 73-year-old hypertensive woman who had postradioiodine hypothyroidism on replacement therapy. She presented with sudden onset of right-sided weakness and numbness, dysarthria, dysphagia, and vertigo. Initial examination showed normal consciousness, horizontal nystagmus, left sixth nerve palsy, dense right hemiplegia, and right upgoing plantar response. CT of the brain revealed calcified VBD and possible left pontine infarct. MRI and MR angiography confirmed VBD with possible dissection, extensive brain stem infarct, and asymptomatic right middle cerebral artery aneurysmal dilatation. She was given subcutaneous injections of low-molecular-weight heparin. Her consciousness deteriorated during the first few days, but her condition later improved. She survived with significant neurological deficits.

A 75-year-old woman with history of hypertension and diabetes mellitus presented with progressive slurring of speech and limb weakness for 10 days. Initial examination revealed dysarthria, left-sided hypertonus, mild generalized weakness, and ataxia of both arms. CT showed calcified VBD only. She became confused, with agitation, over the next 2 days and had 2 episodes of generalized tonic-clonic convulsions on day 3 after admission. Repeat CT showed no interval change, and treatment with phenytoin was commenced. Her consciousness deteriorated further, and she became unresponsive following 2 more episodes of generalized seizures. Her seizure did not recur after add-on therapy with sodium valproate. When neurological examination was repeated, we found that the withdrawal response to noxious stimuli was impaired on her left side and her gaze was deviated to the left. Thus, our diagnosis was evolving brain stem infarction related to her VBD. MRI of her brain was performed 3 weeks later. Although the VBD was confirmed, MRI did not show any infarction in her brain. Follow-up EEG revealed periodic, 1-Hz generalized sharp waves typical of Creutzfeldt-Jakob encephalopathy. Her cerebrospinal fluid was normal. She was subsequently sent to a convalescent hospital for long-term care. These 2 cases illustrate the fact that stroke associated with VBD is uncommon (1 in 25 patients with brain stem/cerebellar infarcts) in Chinese and that VBD or other aneurysmal arterial dilatation may be an incidental finding unrelated to the neurological symptoms.

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1. Passero S, Filosomi G. Posterior circulation infarcts in patients with vertebrobasilar dolichoectasia. *Stroke*. 1998;29:653–659.

Response

We thank Drs Cheung and Mak for their interest in our article, and we regret the printing error in Table 1. It is impossible to name the "most likely pathophysiological mechanisms for ischemia in VBD." Several mechanism may be involved, and every patient has different features related to the degree of ectasia, vertical elongation, and lateral displacement of the basilar artery and to the presence of atheromatous changes of the posterior

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circulation. Common mechanisms (arteriolar lipohyalinosis, obstruction by atheroma or intraluminal thrombus, and artery-to-artery embolism) may be operating, and some have been regarded as being responsible for ischemia associated with VBD.[1–6] Our patients may be classified according to classic criteria and assigned to one of these mechanisms.

Because in VBD other specific mechanisms such as distortion of branches of the basilar artery (BA) and hemodynamic factors may contribute to ischemia,[5,7] we analyzed some aspects of the BA (degree of ectasia, vertical elongation, and lateral displacement). The results of this analysis showed that location of infarcts was linked to some characteristics of the BA, which suggests certain pathophysiological mechanisms (reduced blood flow velocity and possible distortion of the posterior cerebral arteries and their small branches, and distortion and/or stretching of the branches of the BA).

The importance of superimposed atheromatous changes in precipitating ischemia in patients with VBD emerges from a comparison of VBD patients with and without stroke. Ischemic complications of VBD were more often observed in patients who had superimposed atheromatous changes of the posterior circulation.

In conclusion, (1) the presence of atheromatous changes of the posterior circulation, with its consequences (artery-to-artery embolism, branch atheromatous disease) is an important factor for ischemic complications in VBD patients but not a necessary condition, since some patients with stroke had no atheromatous changes; and (2) pathophysiological mechanisms closely linked to the characteristics of the BA operate in VBD patients with ischemic complications together with any atheromatous changes.

VBD is regarded as "uncommon" and often asymptomatic, and many papers on the topic, including mine,[8,9] open with this premise. Actually, the true incidence of VBD and the frequency with which this vascular anomaly becomes symptomatic are still uncertain. Being infrequent, the means used to detect it and the type of patients investigated are important. The most suitable means are undoubtedly MRI and MR angiography. In our experience and that of others,[10] CT scan may not be sufficient. With regard to the type of patient, if we look for VBD in populations at risk (ie, patients with vertebrobasilar ischemia, hemifacial spasm, cranial nerve syndromes, auditory-vestibular symptoms, and trigeminal neuralgia), we have a greater probability of finding patients with this anomaly. We recently found a close association between VBD and idiopathic bilateral vestibular loss.[11] On this point the literature is clear: in studies of patients who underwent cerebral angiography, the incidence of VBD ranged from 0.17% to 5.8%[12–14]; in selected series of patients, the rates of VBD were 2% to 7% in patients with trigeminal neuralgia,[15,16] 4.8% in patients with cranial nerve syndromes,[17] 2.5% in patients with lateral medullary[18] or thalamic[19] infarcts, 2.8% in patients with pontine infarcts,[20] 3.3% in patients with cerebellar infarcts,[21] 7.1% in patients with lower brain stem infarcts,[22] 12.5% to 14.3% in patients with vertebrobasilar infarcts,[23,24] 26.7% in patients with vertigo and slowing of vertebrobasilar flow,[25] and 78.3% in patients with hemifacial spasm.[26]

We began gathering our population in 1980 in collaboration with the Institutes of Radiology, Otolaryngology and Neurosurgery. We created a type of VBD register for the pupose of studying clinical and imaging data and, in particular, follow-up. Our register now includes 124 patients who on the basis of clinical presentation may be grouped as follows: 47 with acute cerebrovascular ischemic events (transient ischemic attack or stroke), 17 with intracerebral hemorrhage (ICH), 39 with cranial nerve syndromes and/or auditory-vestibular symptoms, 2 with hydrocephalus, and 19 with incidental VBD. It is difficult to evaluate the epidemiology; however, patients with cerebrovascular ischemic events belong to a series of approximately 6300 patients hospitalized for acute cerebrovascular ischemic disease; those with ICH belong to a series of 1005 patients with ICH; and patients with other types of presentation were among those hospitalized in our institute or in the Institute of Otolaryngology and those seen in the respective outpatient clinics. For patients with cerebral ischemic or hemorrhagic events, the incidence of VBD (0.75% and 1.7%, respectively) can be calculated since all did at least CT scan. It cannot be calculated for other patients because not all of them did imaging studies.

As clearly stated in our article, all patients were diagnosed by CT scan, cerebral angiography, or MRI studies, and not by autopsy. Of our 40 VBD patients without stroke, 14 had impairment of one or more cranial nerves, associated in 8 with auditory-vestibular symptoms; 11 had vestibular symptoms (imbalance, vertigo, oscillopsia); 2 had auditory symptoms (tinnitus and hearing loss); 3 had combined auditory and vestibular symptoms; and 10 had symptoms unrelated to the VBD. Patients with cranial nerve impairment had involvement of the facial nerve (paresis with nuclear/peripheral pattern or hemifacial spasm; n=5), trigeminal nerve (neuralgia or sensory disturbances with peripheral pattern; n=4), or both (n=5). Additional cranial nerves were involved in 3 cases. In patients with cranial nerve syndromes or with auditory-vestibular symptoms, the need for imaging is evident and requires no further comment.

Drs Cheung and Mak found 1 case of VBD among 25 patients with brain stem/cerebellar infarcts (4%) or 2 cases among 275 stroke or presumed stroke patients (0.72%). These percentages are in line with the above, and it may be premature to suggest that VBD is rarer in Chinese patients. It is incidental and well known that VBD may be asymptomatic or unrelated to the neurological symptoms.[11,13,15,27]

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