fast Fourier spectral analysis, but greater reliability has been achieved experimentally using the Wigner method of spectral analysis. Nevertheless, fast Fourier analysis does allow a differentiation that is practically applicable, and appreciation of this enabled us to diagnose three cases of early postoperative carotid thrombosis based on the detection of persistent particulate embolization in the immediate postoperative period. TCD detection of these emboli made possible an early diagnosis of this condition before the development of neurological signs and enabled early operative intervention. This minimized the neurological deficit in two patients and averted deficit in the third, and we believe that this represents an important clinical application of TCD monitoring during CEA. In addition, we were able to determine that the majority of emboli occurring during other stages of the operation were predominantly characteristic of air emboli and not associated with postoperative neurological deficits.

In conclusion, we agree with Jansen et al that intraoperative TCD monitoring during CEA is valuable in detecting conditions of inadequate collateral cerebral blood flow and significant episodes of intraoperative embolization. However, we contend that appreciation of the character of the emboli and continuing TCD monitoring into the early postoperative period may be of significant benefit in reducing perioperative mortality and morbidity associated with the operation.

M.E. Gaunt, FRCS
J.L. Smith, BSc
P.R.F. Bell, MD, FRCS
Department of Surgery

P.J. Martin, MRCP
Department of Neurology
Leicester Royal Infirmary
Leicester, England

A.R. Naylor, MD, FRCS
Department of Surgery
Aberdeen Royal Infirmary
Aberdeen, Scotland

References
3. Brass et al. have reported an inverse correlation between the number of microemboli delivered during surgery; even more than 1000 microemboli were detected in some patients. Although in a recent series of 301 patients we did not perform neurophysiological function tests, we found a relation between intraoperative focal ischemic symptoms and the occurrence of more than 10 microemboli, especially in the dissection phase of CEA.

In our opinion, the analysis of embolic events is still hindered by inadequate signal processing techniques. The stage of the operation can suggest the nature of microemboli, air bubbles being extremely unlikely when the artery has not yet been opened. Because the fundamental paradox of the Fourier transform technique is that the best temporal resolution that can be achieved is inversely proportional to the frequency resolution, the Wigner method seems promising but the technique is still under study. In our hospital we were not equipped to discriminate between different microemboli.

Persistent particulate embolization in the early postoperative period is an important observation. In some cases this phenomenon heralds an ischemic complication; unchanged embolization immediately after the operation should therefore prompt reassessment of the operated artery, although an increase in the number of microemboli in the first postoperative hours, without clinical consequences, has been described. For this reason, postoperative embolization should be interpreted carefully to avoid unnecessary reexploration. We agree with the statement made by Gaunt et al in their letter that postoperative TCD monitoring should be undertaken to study the cause of morbidity in the first days after the operation.

C. Jansen, MD, PhD
R.G.A. Ackerstaff, MD, PhD
Department of Clinical Neurophysiology
St Antonius Hospital
Nieuwegein, The Netherlands

Role of Transcranial Doppler Sonography in the Differentiation of Multi-infarct and Alzheimer-Type Dementia

To the Editor:

In their interesting article, Ries et al concluded that the effective pulsatility range is a noninvasive additional criterion in the differential diagnosis of dementia. However, I have doubts that the methodology used in the article was adequate for this differentiation; there are physiological variables affecting transcranial Doppler (TCD) velocities that were not taken into consideration in the statistical analysis. According to Hagen-Poiseille's law, blood viscosity has an important influence on cerebral vasomotion and on hemodynamic changes in the proximal part of the intracranial supplying vessels. If all other factors remain constant, blood flow is inversely proportional to viscosity, and blood viscosity of which hematocrit is the most important factor, is a major determinant of blood velocity. Brass et al have reported an inverse correlation between hematocrit and TCD velocity.

The principal metabolic factors that affect cerebral blood flow and that may be reflected in TCD measurements are PO2 and PCO2. Cerebral blood flow does not begin to rise because of hypoxia until
PO2 falls below 50 mm Hg, and it is unaffected by P02 levels above normal. For this reason, the consideration of PO2 is probably unnecessary for most TCD applications. By contrast, TCD velocity increases sharply, by 65% to 150% of baseline velocity, at a PO2 of 40 mm Hg or above. In several studies a linear relation has been demonstrated between maximal Doppler velocity and end-expiratory PO2 of 20 to 60 mm Hg.

Other factors that influence TCD measurements are age and sex. Vriend et al reported that females up to 50 years of age had significantly higher TCD velocities than age-matched males. Arnold et al demonstrated that middle cerebral artery TCA velocity declined with advancing years.

In summary, I think it is necessary to compare patients undergoing TCD with an age- and sex-matched control group, not only an age-matched control group, and that the alterations in PO2 (eg, end-tidal PO2 concentrations) as well as the hematocrit should be taken into consideration when interpreting TCD data obtained in a variety of clinical settings.

Antonio Alayon Fumero, MD
Department of Neurology
Complejo Hospitalario Nuestra Señora de la Candelaria Santa Cruz de Tenerife Canary Islands

References

6. Arnold et al demonstrated that middle cerebral artery TCA velocity declined with advancing years.

Response

We thank Dr Alayon Fumero for his comments on our article on differentiation of multi-infarct and Alzheimer dementia by intracranial hemodynamic characteristics.

The doubts expressed by Dr Alayon Fumero about the adequacy of methods used in this study refer mainly to the possible effects of changes in hematocrit and PO2 on the measured flow velocities. The effects of extreme pathological hematocrit and of hyperviscosity in general have been described, but suspicion of a false positive result in a TCD investigation should be limited to upper or lower extreme values not seen in either the patient group or the control group, according to exclusion criteria of the study protocol. For a subject to be included, absolute flow velocity in his or her middle cerebral artery had to be within the normal range to exclude main stem stenosis. Moreover, the effect of hyperviscosity on flow velocities in the directly insonated vessel segment should be partly counterbalanced by the indirect assessment of peripheral flow resistance on the basis of pulsatility criteria.

The effects of PO2 changes on measured TCD velocities are due to the reaction of the peripheral vessels, i.e., vasoconstriction or vasodilatation influencing flow velocities in the supplying basal vessels. This reaction serves as a criterion in testing the vasomotor reserve capacity. However, this effect is limited in time by the test procedure as well as by the side effects experienced by the patient in case of a longer lasting PO2 imbalance, which is not maintained during a steady-state routine examination as performed in our study. Results of a TCD study, including testing of the cerebrovascular reserve capacity in normal probands and in patients with signs of subclinical and clinically manifest microangiopathy, did not show a significant difference in pulsatility parameters such as the Gosling pulsatility index and the effective pulsatility range (EPR), compared with the measured PO2 values at rest. In the study under discussion, the TCD investigation was performed in all patients with dementia and in control subjects under stabilized conditions at rest. Possible cardiopulmonary diseases leading to relevant and permanent imbalances in blood gas pressure were excluded by the study protocol, because this could have represented a different cause of the dementia syndrome. Potential effects of extracranial or intracranial stenosis or occlusion in any group, implying a possibly different reaction pattern to PO2, were excluded by the study protocol.

The influences of age and sex on the measured flow velocities and pulsatility were largely limited by the similarities in the groups’ male-female ratios, age ranges, and mean ages, which for all subgroups were within the same decade, as is usually seen in reference studies defining normal range values. In fact, pulsatility values, defined as EPR and calculated on the basis of data from two other studies with matching age groups, gave normal-range EPR values highly corresponding to our data. Additionally, the influence of declining flow velocities with increasing age would be the same in all the groups, and would be abolished by the definition of EPR as a ratio of absolute flow velocities. In conclusion, the interpretation of specific pulsatility characteristics as an additional, not a single, method of differentiating between a vascular cause and a degenerative cause of dementia is valid.

Fernand Ries, MD
Department of Neurology
University Clinic, Bonn
Bonn, Germany

References


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A Alayon Fumero

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