surgical treatment? It is stated that they refused operation but one does not know what information they were given to come to this conclusion. In our experience it is vanishingly rare for patients to refuse a treatment that has been presented to them as the most efficacious option. We suspect, but cannot prove, that many radiosurgically treated AVMs are appropriate for microsurgical excision.

We must also respectfully disagree with Dr. Coffey's comments regarding the tautological nature of AVM grading scales. In our study the Spetzler and Martin scale was very good at doing what it was designed for: predicting surgical complications. The fact that it works does not make it tautological. A tautology (all bachelors in our series were found to be unmarried males) must be true by definition. It is conceivable that factors other than the Spetzler and Martin grade might have better predicted a new neurological deficit following surgery. The fact that they did not confirms the value of the scale; it does not make it tautological. This scale may or may not be worthwhile for radiosurgical case selection or risk analysis but that is irrelevant for the purposes of our paper unless there is something about Spetzler and Martin Grade I through III AVMs that makes them particularly poor lesions to treat radiosurgically. Of course, cerebrovascular surgeons will use much information in addition to the patient's rank on a grading scale in determining the best treatment options. This brings us back to the selection bias argument.

We strenuously object to the charge that we "used erroneous denominators to ensure minuscule obliteration rates for the radiosurgical series." One could make the charge that radiosurgical reports often use erroneous denominators to ensure maximum obliteration rates for radiosurgical series. If one treated 100 patients with AVMs, obtained angiograms in 50 (because it is suspected that the AVM is gone), and finds that 45 lesions are obliterated the definite obliteration rate is 45%. One might wish it were 90% but this really is not the case. Because we believed that the definite obliteration rate might be unrealistically low in the radiosurgical series we added a category of probable obliteration. We most certainly did not attempt to decrease the efficacy of radiosurgery artificially by using inappropriate denominators. Furthermore, it remains to be seen how "minuscule" these radiosurgical obliteration rates are. The definite obliteration rates with radiosurgery treatment in our review ranged from 36 to 65% with a mean of 45%. Our estimation of probable obliteration rates following radiosurgery ranged from 66 to 74%, with a mean of 71%. The multicenter Japanese study reported at the 1998 American Association of Neurological Surgeons annual meeting by Yamamoto and colleagues[2] found a 50% definite AVM obliteration rate in 885 patients who had been followed for more than 3 years after gamma knife radiosurgery. If only those patients undergoing follow-up angiography are used as the denominator the obtained rate rises to 65%.[2] We stand by our methodology.

Dr. Coffey points out the fallacy involved in pitting the results of microsurgery against radiosurgery 1 day after treatment. This is not what we did in our analysis but it is precisely what is done to advertise the benefits of radiosurgical treatment of AVMs. Many patients with AVMs are candidates for either microsurgery or radiosurgery. We believe that AVMs treated microsurgically assume a higher immediate risk of complication in exchange for lower cumulative morbidity and mortality rates that become obvious only over time. This may be a difficult concept for many patients to appreciate. The absence of immediate risk from radiosurgery is obvious to patients. It is more difficult for them to understand that a period of several years will be necessary to determine whether their AVMs are obliterated and that during this time they continue to be at risk; or that their AVMs may persist and they will be in jeopardy indefinitely unless further treatment is done; or that the complications of radiosurgery are similar to the complications of microsurgery but delayed in onset. The advantages of "have your treatment today and go out to dinner tonight" begin to pale when these factors are considered.

Finally, we agree with Dr. Coffey that neurosurgeons can no longer justify the performance of any particular AVM treatment, even their favorite one, based solely on local customs, the expectations of referring physicians, or the wishes of misinformed patients. That is why we wrote our paper.

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Doppler Ultrasound in Subarachnoid Hemorrhage

TO THE EDITOR: The recent article by Wardlaw and colleagues (Wardlaw JM, Offin R, Teasdale GM, et al: Is routine transcranial Doppler ultrasound monitoring useful in the management of subarachnoid hemorrhage? *J Neurosurg 88:*272–276, February, 1998) sets forth a very interesting question that, in my opinion, the authors do not completely answer.

From a clinical point of view, transcranial Doppler (TCD) ultrasound monitoring in the management of subarachnoid hemorrhage patients would prove useful if by early warning and by early aggressive therapy it enhanced our ability to fight deficits caused by vasospasm. Assuming that a therapy would be effective against the reduction in regional flow caused by vasospasm, prompt therapy could benefit the patient before a clear clinical deficit had been established. In this paper, the usefulness of TCD monitoring seems to have been established by correlation with the late ischemic neurological deficit and with changes in management; unfortunately, no adequate data are available to prove that an elevated velocity of the TCD correlates with the reduction in flow causing the

neurological deficit. According to Clyde, et al.,[1] the increased blood velocity revealed by TCD ultrasonography does not always correspond to reduced cerebral blood flow (CBF); they do, however, correlate high blood velocity with increased CBF, not with ischemia. A key point not addressed by the study is, therefore, the pathophysiological meaning of an increase in TCD velocity: if the correspondence between high TCD velocity and critical reduction in flow cannot be supported, any clinical intervention based on these data becomes questionable. The paper shows that there is virtually complete agreement between TCD results and angiographic data and that may prove that angiographic vasospasm has a direct correlation with TCD velocity. Unfortunately, two factors reduce the potential of these findings. First, angiographic vasospasm is not equivalent to clinical vasospasm. Second, angiography is usually performed on admission, or early during the patient's hospital stay, to allow early surgical or interventional procedures; vasospasm is more frequent in the late phases, peaking at 7 to 10 days. Therefore, the probabilities of confirming high TCD velocity on the basis of angiography are very low and cannot validate the usefulness of TCD examination in detecting critical reductions in CBF. If we look at cases that have demonstrated neurological deficit without increase in TCD velocity, we may draw information about the weakness of TCD monitoring as a useful tool in the early detection of ischemia. Additionally, intervention before the clinical signs of ischemia develop seems critical in terms of potential benefits for the patient. It is possible that performing TCD examination only three times per week does not maximize the potential of these studies to provide early warning of ischemia.

The authors must be commended for their very careful review of the large amount of data recorded in several cases. It is my view, and one acknowledged by the authors themselves, however, that further work is necessary to clarify the usefulness of routine TCD monitoring in management. For this reason, I cannot agree that the study's findings are strong enough to support the value of routine TCD monitoring. On the other hand, I fully concur with the authors that a trial study, in which patients are randomly assigned TCD monitoring, is highly valuable.

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To The Editor: We read with enthusiasm the paper by Wardlaw and colleagues. The authors have provided clinical evidence supporting the value of routine TCD monitoring in patients with SAH. The observations that they have made are perceptive; however, we would like to add a few points to the methodology of TCD usage in SAH management.

The accuracy of TCD in detecting SAH-related cerebral vasospasm is restricted by its low specificity rate, that is, the downside of TCD monitoring of SAH patients is the high rate of false-negative results.[1–4] The TCD specificity of vasospasm detection has been determined to be as low as 52%.[3] The use of relative changes of Doppler velocity instead of absolute velocity values increases the clinical usefulness of TCD technology for the management of patients with SAH.[2] An improved methodology incorporating a new and more dependable grading system minimized the false-negative results in at least three clinical situations we studied.[2]

It is worth noting that false-negative low TCD velocities were observed in patients with proximally evolving vasospasm. Patients with stenosis in their common carotid arteries demonstrate a decreased range of normal Doppler velocity values in their middle cerebral arteries, causing a false-negative assessment of vasospasm due to SAH. The rate of increase of Doppler velocity is more important than its absolute value. Therefore, a patient with lower initial Doppler velocity values and a 35-cm/second increase in Doppler velocity within 24 hours may have a statistically higher risk of vasospasm than another patient with higher blood flow velocity values and the same increase in velocity over a longer time period. Evaluations based on absolute velocity values would demonstrate false-negative results in the first patient, who is actually at higher risk of vasospasm.

To benefit from TCD monitoring of SAH, we must know how specifically and sensitively TCD reveals the clinical diagnosis of vasospasm-related ischemic deficit. In a recent article,[2] we concluded that TCD grading according to the absolute velocity monitoring values resulted in 17% false-negative and 5% false-positive information, whereas the new grading system resulted in 8% false-negative and 2.5% false-positive information rates. It would have been beneficial if Wardlaw's study had included information about the clinical sensitivity and specificity of TCD.

We recommend that TCD examinations be performed daily as a part of the routine workup of a patient with SAH (Wardlaw, et al., performed TCD examinations three times per week). In the monitoring of SAH-related vasospasm, TCD technique must be regarded as a neurosurgical diagnostic tool, and to overcome the technician-dependent inherent errors of Doppler technique, the same neurosurgeon should perform all of the TCD examinations of a patient with SAH. By considering these clinical principles of TCD monitoring, one can even use this technique to predict vasospasm-related ischemic deficit.[1,2,4]

In our opinion, these suggestions should be considered in any further studies assessing the clinical dependability of TCD in the management of subarachnoid hemorrhage. Recent developments in endovascular methods to reverse or prevent cerebral vasospasm–related neurological deficit continue to increase the importance of TCD determination and prediction of vasospasm.

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