Is routine transcranial Doppler ultrasound useful in the management of subarachnoid hemorrhage?


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In this prospective observational study, the authors assess the impact of routine transcranial Doppler (TCD) ultrasound monitoring on the diagnosis, management, and outcome of delayed ischemic neurological deficit complicating subarachnoid hemorrhage (SAH). Over a 10-month period 186 patients admitted to a regional neurosciences center were included in the study. Three times a week, routine TCD examinations performed by neuroradiographers made an important positive contribution to the diagnosis of delayed ischemic neurological deficit in 72% of patients with this complication and altered management for the benefit of the patient in 43%. In 9% of patients with recent SAH, it was believed that the outcome might have been better if the TCD result had been acted upon appropriately. The TCD results did not adversely influence management or outcome and were accurate when compared with those obtained on angiography. The authors conclude that a routine TCD service provided by neuroradiographers is accurate and useful in diagnosing and managing elevated blood velocities and ischemic neurological deficit following SAH. In addition, it is possible that if the information gleaned from TCD findings was used more often in patient management, outcome might be improved; however, a randomized controlled trial is necessary to assess this definitively.

Key Words * transcranial Doppler ultrasound * vasospasm * subarachnoid hemorrhage * cerebral ischemia

Delayed ischemic neurological deficit, still a frequent complication of subarachnoid hemorrhage (SAH), is probably multifactorial in origin,[14,16] but its development correlates with angiographically demonstrated vasospasm[8] and elevated blood velocities measured using transcranial Doppler (TCD) ultrasound monitoring.[1,13] It has been suggested that TCD studies may enable identification of patients with a high risk of developing neurological deficits.[7] Nevertheless, the value of TCD ultrasonography in routine clinical practice remains controversial, and the reported studies have made use of observations by clinicians or specifically dedicated research technicians. At the Institute of Neurological Sciences, Glasgow, a service was established in which TCD ultrasonography is routinely performed by a team of neuroradiographers in patients admitted with SAH. We prospectively assessed the service to answer the following questions: 1) does TCD monitoring influence diagnosis, management, or outcome; 2) is the diagnosis of increased blood velocity by TCD accurate; 3) is the timing of TCD monitoring optimum; and 4) for what period of time is it useful to record TCD after SAH?

CLINICAL MATERIAL AND METHODS
Between May 1, 1992, and February 28, 1993, all patients with the presumptive diagnosis of aneurysmal SAH who were admitted to the Institute of Neurological Sciences in Glasgow were identified by one of the authors and/or the neuroradiographers. We recorded details of the patient's admission clinical state and assessed the patient's condition by using the World Federation of Neurological Surgeons (WFNS) system, which includes the Glasgow Coma Scale (GCS),[5,15] in addition to the results of all computerized tomography (CT) scans, angiograms, complications, outcome, and evidence of action by the neurosurgical staff in response to the results of the TCD investigations. Similar information was recorded in patients admitted for elective aneurysm surgery. The clinical details were obtained from: the patient's chart, the admitting neurosurgeons, ward nurses, and direct observation of the patient (by one of the authors and the radiographers). The collection of clinical data was done unobtrusively so as not to influence the attitude of the neurosurgeons to TCD results during the review.

The admission and any follow-up CT scans were reviewed prospectively by a consultant neuroradiologist blinded to other data and classified by the initial amount of blood according to the method of Hijdra, et al.,[9] and for hematoma, hydrocephalus, and parenchymal low-density areas suggestive of an ischemic lesion. The four-vessel intraarterial digital angiograms were correlated "blind" to the TCD and CT results, according to the number and site of aneurysms, the presence of spasm (judged as normal, possible, or definite arterial narrowing to at least 50% of the normal size) in the middle cerebral artery (MCA), anterior cerebral artery (ACA), or posterior cerebral artery, the presence of atheromatous stenosis which might lead to the erroneous diagnosis of vasospasm on TCD monitoring, and the presence of anomalies of the circle of Willis (such as an absent A1 segment of the ACA). Spasm was indicated by irregular narrowing of the arteries but was not measured objectively because of the recognized difficulties and impracticality of routinely obtaining accurate angiographic measurement of arterial caliber.[6]

All patients were offered the TCD monitoring service consisting of a bedside TCD examination on Monday, Wednesday, and Friday from the time of admission until discharge (this continued the arrangement in the existing TCD SAH monitoring service). An EME TC 64b (Eden Medical Electronics GMBH, Germany) with a 2-MHz probe connected to a personal computer mounted on a trolley was used. A TCD examination was performed more frequently if requested. The TCD findings were written in the patient's chart on a "results" sheet. The time between admission to the neurosurgical unit and the first TCD study, the maximum blood velocity recorded by TCD monitoring and the date on which it occurred, the greatest increase in blood velocity in any 2-day period, and the velocity increase in the 2 days prior to any neurological deterioration were extracted from the TCD record sheet after the patient's hospital discharge. Vasospasm was defined as an increase in mean blood velocity above the upper limit of the normal range for the patient's age;[2,4] angiographically visible vasospasm is thought to correlate with a mean blood velocity of 110 cm⁻¹ or more in the MCA.[13] Any patient who did not undergo TCD monitoring was noted, as were the reasons.

Clinical Follow-Up Evaluation

Clinical follow-up data were obtained from the case notes for each patient at hospital discharge and at the first postdischarge clinic visit, which was usually 3 months later. Their outcome was classified according to Glasgow Outcome Scale (GOS) score.[11] The assessment of complications during the admission and any associated neurological deterioration was based on information obtained from the attending doctors and nurses, the patient's neurological observation charts, observations by the neuroradiographers on their TCD rounds, and knowledge of the patient's attendance in the x-ray department for repeated CT scans. Clinical deterioration was defined as a reduction of at least two points on the GCS score lasting for at least 8 hours or the development of a focal neurological deficit such as dysphasia or hemiparesis. We relied as much as possible on the observations of the radiographers or one of the authors (J.M.W.) and routine nursing
observations, so as not to raise the profile of the audit and influence clinicians' attitudes. In some cases it was
necessary to rely on the case notes or on direct questioning of ward staff to obtain details about complications
and their likely cause.

Classification of the cause of deterioration was based on the mechanism considered most likely at the time
and included operative complication, rebleeding documented by repeated CT scan, or postmortem
examination, as well as other medical complications such as pulmonary edema, or ischemic neurological
deficit. Ischemic neurological deficit was diagnosed when: 1) other likely causes had been excluded; 2) CT
scanning revealed a new low-density area in an arterial distribution suggestive of ischemia; and 3) the
cerebral arterial blood velocities were elevated on TCD studies. Usually reasons 1 and 2 carried more weight
than 3, and the first reason was the main method of diagnosing ischemic neurological deficit in this review. If
more than one cause of clinical deterioration was likely, the most important cause was put first, and other
causes were considered to be secondary.

Using all information available in the patient's chart as well as the observations of the doctors and nurses'
actions at the time, a decision was made after the patient's discharge as to whether the TCD result had
influenced the diagnosis of vasospasm, the management of the patient, and the clinical outcome. In making
the determination for diagnosis, it was decided whether the TCD result had 1) contributed significantly to
establishing the diagnosis (presence or absence) of vasospasm (and therefore the likelihood of ischemic
neurological deficit), 2) not contributed, 3) provided misleading information, or 4) was irrelevant (for
example, if the patient had died within hours of admission). In making the determination for management, it
was decided whether the TCD result had 1) influenced management for the better, 2) not influenced
management (that is, TCD result and management decisions were compatible), 3) led to a series of
detrimental management decisions, or 4) been irrelevant. For outcome, evaluation centered on whether 1)
outcome might have been better had the TCD result been acted on, 2) TCD findings had no influence on
outcome (the two were compatible), 3) TCD result adversely influenced outcome, or 4) it was not relevant.
All information germane to how management decisions were made was carefully sought, and, when
necessary, additional information from the attending doctors was obtained. In cases of doubt, we erred on the
side of the TCD result being less, rather than more, influential, and more, rather than less, harmful.

All the data were entered into a database, taking care to maintain patient anonymity. Analysis of the results
was performed using a commercially available statistical package (SPSS-PC). Statistical advice was given by
the University of Glasgow Department of Statistics and the Greater Glasgow Health Board Audit Office. The
advice of the local area ethics committee was sought on the study, and permission for it to proceed was
granted.

RESULTS

During the 10-month study period there were 165 emergency admissions following suspected aneurysmal
SAH and 21 elective admissions for aneurysm surgery. There may have been a few additional patients who
died soon after admission and, therefore, could not be included; some patients admitted for elective aneurysm
surgery have not been included because of difficulties in identifying them.

The mean age of the group was 49 years (± 12.7 years). Of the patients admitted on an emergency basis, 71
were admitted within 24 hours of the suspected bleed (43%), 44 within 48 hours (cumulative total 70%), and
15 were admitted on the 3rd day (9%). Thus, 79% of emergency admissions were admitted within 3 days of
the SAH, and 90% were admitted within 6 days. The majority of patients were classified as being in good
clinical condition on admission: 40% received WFNS Grade I (GCS score 15); 34% Grade II or III (GCS
score 14 or 13), 19% Grade IV (GCS score 12 to 7); and only 7% received a WFNS Grade V (GCS score 6 or
less). Seventy percent of the patients admitted after a recent bleed underwent procedures to prevent
bleeding, with nearly all undergoing an operation to clip the aneurysm suspected of causing the SAH. Thirty percent did not undergo an operation or an interventional neuroradiological procedure because the source of hemorrhage was not identified (34 patients, 21% of patients with recent SAH), the patient died (11 patients), or because the patient did not recover sufficiently from the initial hemorrhage or its complications to be fit enough for surgery (16 patients). Of the patients who did undergo operative intervention, 31% underwent surgery 3 days after SAH and 50% within 5 days of the SAH.

During the course of the admission, 82 (44%) of 186 patients deteriorated clinically, the main causes of which are shown in Table 1. Ischemia (in association with increased blood velocities) was considered to be the main cause in 29 (39%) of the 82 patients (16% of the total 186 patients) and was the most frequent complication (26 emergency and three elective admissions). Ischemic neurological deficit was considered to be a secondary cause of deterioration in an additional four patients. Of the 26 emergency patients who suffered ischemia, 12 (46%) had a neurological deficit lasting between 24 hours and 7 days, and 50% had a neurological deficit lasting more than 7 days. Two of the three patients undergoing elective surgery developed ischemia and had a neurological deficit lasting between 24 hours and 7 days, and the other one had a neurological deficit lasting more than 7 days.

<table>
<thead>
<tr>
<th>Cause of Deterioration</th>
<th>Emergency Admission (%)</th>
<th>Elective Admission</th>
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<tbody>
<tr>
<td>delayed ischemic neurological deficit</td>
<td>26 (35)</td>
<td>3</td>
</tr>
<tr>
<td>rebleed</td>
<td>10 (13.5)</td>
<td>0</td>
</tr>
<tr>
<td>periop problem</td>
<td>19 (26)</td>
<td>3</td>
</tr>
<tr>
<td>medical problem</td>
<td>5 (7)</td>
<td>1</td>
</tr>
<tr>
<td>hydrocephalus</td>
<td>13 (18)</td>
<td>1</td>
</tr>
<tr>
<td>uncertain</td>
<td>1 (1)</td>
<td>0</td>
</tr>
</tbody>
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Of the 165 emergency admission patients, 149 underwent angiography. There was complete agreement between angiographic findings and those of the TCD studies (that is, angiographically demonstrated vasospasm and elevated blood velocities on TCD studies) in the 114 patients in whom angiography was performed within 2 days of the TCD examination that established the maximum blood velocity during the admission. That is, if TCD findings demonstrated the MCA mean velocity to be greater than 120 cm/second, the angiogram showed spasm. In an additional 35 patients, the time interval between the angiogram and the highest recorded velocity on TCD monitoring was more than 3 days, and comparisons of these studies were not considered to be valid. Transcranial doppler ultrasound correctly identified components of the circle of Willis in all but seven patients: in one of these the ACA (A1 segment) was present, but missed on TCD; in the other six, the A1 was absent on angiography but was considered to be present based on findings of TCD, presumably because the internal carotid artery (ICA) siphon was mistaken for the ACA. In retrospect, in these cases the velocities were lower than appropriate for the ACA. Patients admitted after a recent SAH underwent between one and 12 TCD studies over a mean period of 7 days. No late increases in blood velocity were recorded more than 2 weeks after operation.

The maximum mean flow velocity recorded during admission, the maximum increase in mean velocity in any 2-day period, and the increase in mean velocity in the 2 days before any clinical deterioration (and the probable cause) are shown in Table 2. Both the highest average mean velocities and the greatest increase in mean velocity over any 2 days occurred in the patients thought to have ischemic neurological deficit as the cause of the deterioration. The maximum increase in MCA blood velocity over the 2 days prior to onset of a clinical deterioration was greatest in patients with probable ischemic neurological deficit (rise of 39
cm/second/2 days compared with a maximum of 15 cm/second/2 days in patients considered to have another cause for deterioration).

The influence of TCD monitoring on clinical protocol is shown in Table 3. The TCD results were confusing in one emergency patient, unhelpful in two, and not relevant in 27% of the emergency patients (who had an early recurrent aneurysmal SAH or who died soon after admission); they were also irrelevant in 19% of patients seeking elective surgery who were only admitted for an angiogram and in whom surgery was deemed unnecessary. The TCD results appeared to alter management beneficially in 37% and 48% of patients, respectively. In the patients admitted on an emergency basis this was often because TCD monitoring demonstrated elevated blood velocities and influenced (delaying or advancing) the timing of surgery or discharge or even led to simple actions such as continuing bed rest or administration of intravenous fluids for an additional 24 hours.

<table>
<thead>
<tr>
<th>Maximum Mean Blood Velocities (cm/sec)</th>
<th>Main Causes of Neurological Deterioration</th>
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<tbody>
<tr>
<td>rt MCA</td>
<td>None</td>
</tr>
<tr>
<td>during the study</td>
<td>95.9 ± 54.5</td>
</tr>
<tr>
<td>lt MCA</td>
<td>92 ± 52.8</td>
</tr>
<tr>
<td>rise over 2-day period</td>
<td>20.2 ± 30.4</td>
</tr>
<tr>
<td>rise in the 2 days prior to deterioration</td>
<td>19.7 ± 29.8</td>
</tr>
<tr>
<td>rt MCA</td>
<td>0</td>
</tr>
<tr>
<td>lt MCA</td>
<td>0</td>
</tr>
<tr>
<td>no. of patients</td>
<td>104</td>
</tr>
</tbody>
</table>

* Values are expressed as the mean ± standard deviation of the mean.
The TCD result did not adversely affect management in any patient. The TCD findings were considered to have made a useful management contribution in 43% of all patients. In 9% of patients it was believed that improved outcome at 3 months might have been attained had the result of the TCD study been incorporated into the patient's clinical management; that is, more intensive, active measures to correct ischemic neurological deficit instituted sooner in the presence of blood velocities that were persistently and markedly elevated.

**DISCUSSION**

Performing serial TCD observations after SAH has two objectives: to detect elevated blood flow velocities and to identify patients who are at risk of ischemic neurological deficit. The cause of ischemic neurological deficit after SAH is complex. Angiographically demonstrated vasospasm and elevated blood velocities recorded by TCD monitoring have been shown to correlate with ischemic neurological deficit, but there are discrepancies. For example, the artery in which blood velocity elevation is maximum is not necessarily the territory in which ischemic symptoms arise, nor does the site of greatest blood accumulation in the subarachnoid space necessarily relate to the ischemic territory.[10] Nevertheless, increased blood velocities in the intracerebral circulation can be noninvasively identified by TCD studies, and this correlates with a greater risk of ischemic deficit.

We have shown that TCD ultrasound findings correspond closely to the results obtained using intraarterial angiography. There was no patient with a high blood flow velocity in whom angiography demonstrated normal arterial caliber, and no patient who had normal velocity in whom angiography revealed vasospasm. In the present study, the upper limit of normal blood velocity was taken from previously published studies,[2,4] and our results, obtained by neuroradiographers, are at least as good as those from previous studies in which specially trained, dedicated technicians were used.

We did not measure the MCA/ICA velocity ratio,[13] mainly because measurement of ICA velocities and calculation of the ratio would have prolonged the examination time by approximately 30%. The original work correlating MCA/ICA ratios with vasospasm was based on only 51 patients and, to the best of our
Assessment of the influence of the TCD results in the diagnosis of ischemic neurological deficit was straightforward: the TCD data either confirmed or excluded elevated blood velocities. In patients with possible raised intracranial pressure (ICP), the pulsatility index was taken into account in the interpretation of mean blood flow velocities, because significant angiographically demonstrated vasospasm may be present, but with only moderately elevated blood velocity when the ICP is raised.[12] An ICP monitoring device or intraventricular drain was often inserted when there was clinical suspicion of raised ICP or when there was evidence on CT scanning of obstructive hydrocephalus, in which case the true ICP reading was known, aiding the interpretation of blood velocity.

In the present study, the patients who developed an ischemic deficit had a mean maximum rise in MCA blood velocity of approximately 50 cm/second for 2 preceding days. This is approximately half of the change (that is, a rise of 50 cm/second/day) that was reported in a previous study to predict an ischemic deficit.[7] It is probable that patients can develop ischemic neurological deficit with much less substantial increases in mean blood velocities than 50 cm/second/day, and smaller increases should not preclude the diagnosis of ischemic deficit.

Assessment of the influence of TCD findings on either management or outcome was necessarily somewhat arbitrary. If there was doubt about the contribution of the TCD result, we erred on the side of suggesting that it was not helpful; therefore, our assessment may be an underestimation of the benefit of TCD results. Some of the actions that were taken in response to the results of TCD monitoring are based on well-established principles rather than definitive evidence of benefit on outcome in randomized trials. Nevertheless, the use of volume expansion and hypertension, modification of the timing of angiography or surgery, and timing and rate of mobilization and discharge have a rational pathophysiological basis and are integral parts of the conventional care of patients with SAH. The results of TCD monitoring influenced these aspects of management in approximately 40% of patients, and in an additional 9% an appropriate, effective response to the TCD result might have led to improved outcome.

Serial TCD observations were most useful in patients with recent aneurysmal SAH who received a good clinical grade when admitted. In these patients the development of a new/delayed ischemic neurological deficit was the most serious and most frequent complication. They were less useful, however, in patients who received a poor clinical grade, because there were other medical or neurosurgical problems.

Although the technology to make TCD measurements has been available for some 15 years, their value in the management of SAH is still debated. This may in part be because uncertainty exists about the best method of preventing or treating ischemic neurological deficit.[3,17] Observational studies of the efficacy of interventions, such as the present one, are prone to bias. The extent of any effect of TCD ultrasound on studies of outcome after SAH can only be determined definitively in a trial in which patients are randomly allocated to undergo TCD monitoring or not. At the time of this study, this option was considered to be unacceptable by some clinicians, in part because there are so many other factors that influence outcome after SAH that conclusive evidence of benefit solely from TCD monitoring was unlikely. Until such evidence is obtained, the findings of this study support the value of providing a service for routine TCD monitoring in patients with SAH.

Acknowledgments

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References


