Transcranial Doppler sonography within 12 hours after subarachnoid hemorrhage

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Twenty-one patients were subjected to repeated assessment of cerebral blood flow velocities by means of transcranial Doppler sonography (TCDS) during the first 12 hours after subarachnoid hemorrhage (SAH). In 19 patients the study was performed following the first SAH, and in two after early rebleeds. Flow velocities did not indicate an early phase of arterial narrowing in any case. Following the first TCDS assessment, flows were evaluated repeatedly in the 19 survivors. Increased flow velocities suggesting arterial narrowing or vasospasm occurred only after a delay of at least 4 days. The results of this study favor the restoration of normal velocity patterns in surviving patients and do not indicate that an acute phase of vasospasm exists either immediately after or in the first 12 hours after SAH.

KEY WORDS : cerebral aneurysm . cerebral blood flow . transcranial Doppler sonography . subarachnoid hemorrhage

DURING the last two decades an immense number of studies have been undertaken to elucidate the pathophysiology of cerebral vasospasm and delayed ischemic deterioration following aneurysmal subarachnoid hemorrhage (SAH). The significance of the various mechanisms possibly involved in this complex phenomenon remains to be explained. It has been proposed that the development of vasospasm and delayed ischemia is paralleled by an increase in the concentration of vasoconstrictor agents in the blood-contaminated cerebrospinal fluid (CSF), and a large number of different substances have been suggested to account for the pathogenesis of delayed cerebral hypoperfusion in patients suffering from aneurysmal SAH.66

A variety of animal models have been designed in order to study the pathophysiology of vasospasm. Based on the results of such studies on experimental SAH, it has been claimed that vasospasm exhibits a biphasic course, consisting of an acute phase, and a late phase, reaching a maximum between 2 and 7 days after the bleed, depending upon the species.4,5,11,12,27,31,37,38,44,45,48,54,64 Is this biphasic course also true in the human situation? Since the first report in 195445 many instances of repeat rupture during angiography have been documented and the events taking place in the large cerebral arteries during rupture have been well described.2,3,6,9,10,13,15,17,19,23,24,26,28-30,32-36,42,43,46,47,52,53,55-58,63,67 In 1976, Wilkins65 reviewed 32 cases of aneurysm rupture during angiography and concluded that there was no proof that an acute phase of vasospasm occurs in humans following rupture of an intracranial aneurysm. In our experience with early angiography (within 72 hours after the bleed) in more than 500 patients with SAH, we also found no proof for an early vasospastic phase in the large cerebral arteries in humans (B Romner, et al., unpublished data).

With the introduction of noninvasive transcranial Doppler sonography (TCDS) by Aaslid, et al.,1 a new device has been added which allows assessment of blood flow velocity and flow direction in all large basal cerebral arteries.18,21,22,49-51 Recently, Grote and Hassler20 were able to study three patients during aneurysm rupture and did not observe TCDS findings indicative of vascular narrowing during extravasation of blood in the subarachnoid spaces. The present study was undertaken to determine whether, based on TCDS, there appear to be velocity changes suggesting an acute phase of vasospasm in human cerebral arteries during the first 12 hours following SAH.

Clinical Material and Methods

During a 1-year period from August, 1987, to July, 1988, 63 individuals who had suffered an SAH were admitted to the neurosurgical unit in Lund. Twenty-one patients who were admitted within hours after the
Early transcranial Doppler sonography after SAH

TABLE 1
Clinical data and MCA flow velocities in the first 12 hours after SAH*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Aneurysm Location</th>
<th>Clinical Grade</th>
<th>Subarachnoid Blood†</th>
<th>Interval SAH to TCDS</th>
<th>Mean MCA Flow Velocity (cm/sec)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>45</td>
<td>M</td>
<td>ACoA</td>
<td>IV</td>
<td>3</td>
<td>2 min</td>
<td>32</td>
<td>30</td>
</tr>
<tr>
<td>2</td>
<td>38</td>
<td>F</td>
<td>lt MCA</td>
<td>II</td>
<td>2</td>
<td>6 min</td>
<td>80</td>
<td>90</td>
</tr>
<tr>
<td>3</td>
<td>67</td>
<td>M</td>
<td>?</td>
<td>II</td>
<td>?</td>
<td>40 min</td>
<td>64</td>
<td>84</td>
</tr>
<tr>
<td>4</td>
<td>51</td>
<td>F</td>
<td>rt ICA‡</td>
<td>V</td>
<td>4</td>
<td>75 min</td>
<td>60</td>
<td>50</td>
</tr>
<tr>
<td>5</td>
<td>62</td>
<td>F</td>
<td>ACoA</td>
<td>III</td>
<td>2</td>
<td>4 hrs</td>
<td>54</td>
<td>52</td>
</tr>
<tr>
<td>6</td>
<td>41</td>
<td>M</td>
<td>ACoA</td>
<td>IV</td>
<td>4</td>
<td>5 hrs</td>
<td>24</td>
<td>26</td>
</tr>
<tr>
<td>7</td>
<td>61</td>
<td>F</td>
<td>NUD</td>
<td>I</td>
<td>1</td>
<td>5 hrs</td>
<td>32</td>
<td>32</td>
</tr>
<tr>
<td>8</td>
<td>53</td>
<td>F</td>
<td>lt ICA‡</td>
<td>V</td>
<td>3</td>
<td>5 hrs</td>
<td>64</td>
<td>66</td>
</tr>
<tr>
<td>9</td>
<td>51</td>
<td>F</td>
<td>ACoA</td>
<td>II</td>
<td>2</td>
<td>6 hrs</td>
<td>38</td>
<td>42</td>
</tr>
<tr>
<td>10</td>
<td>55</td>
<td>M</td>
<td>rt ICA</td>
<td>II</td>
<td>2</td>
<td>6½ hrs</td>
<td>64</td>
<td>58</td>
</tr>
<tr>
<td>11</td>
<td>55</td>
<td>M</td>
<td>ACoA</td>
<td>II</td>
<td>3</td>
<td>7 hrs</td>
<td>42</td>
<td>52</td>
</tr>
<tr>
<td>12</td>
<td>34</td>
<td>M</td>
<td>NUD</td>
<td>V</td>
<td>4</td>
<td>8 hrs</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>13</td>
<td>45</td>
<td>M</td>
<td>lt ICA</td>
<td>II</td>
<td>2</td>
<td>9 hrs</td>
<td>58</td>
<td>46</td>
</tr>
<tr>
<td>14</td>
<td>20</td>
<td>M</td>
<td>lt ICA</td>
<td>II</td>
<td>2</td>
<td>10 hrs</td>
<td>62</td>
<td>58</td>
</tr>
<tr>
<td>15</td>
<td>39</td>
<td>F</td>
<td>rt MCA‡</td>
<td>V</td>
<td>4</td>
<td>10 hrs</td>
<td>80</td>
<td>48</td>
</tr>
<tr>
<td>16</td>
<td>56</td>
<td>M</td>
<td>rt MCA</td>
<td>II</td>
<td>3</td>
<td>11 hrs</td>
<td>46</td>
<td>48</td>
</tr>
<tr>
<td>17</td>
<td>62</td>
<td>M</td>
<td>NUD</td>
<td>II</td>
<td>2</td>
<td>11 hrs</td>
<td>30</td>
<td>26</td>
</tr>
<tr>
<td>18</td>
<td>27</td>
<td>F</td>
<td>lt MCA</td>
<td>II</td>
<td>3</td>
<td>11 hrs</td>
<td>44</td>
<td>48</td>
</tr>
<tr>
<td>19</td>
<td>63</td>
<td>F</td>
<td>ACoA</td>
<td>III</td>
<td>3</td>
<td>12 hrs</td>
<td>42</td>
<td>48</td>
</tr>
<tr>
<td>20</td>
<td>49</td>
<td>F</td>
<td>lt MCA</td>
<td>III</td>
<td>3</td>
<td>12 hrs</td>
<td>44</td>
<td>38</td>
</tr>
<tr>
<td>21</td>
<td>26</td>
<td>M</td>
<td>rt ICA</td>
<td>III</td>
<td>3</td>
<td>12 hrs</td>
<td>44</td>
<td>44</td>
</tr>
</tbody>
</table>

* MCA = middle cerebral artery; SAH = subarachnoid hemorrhage; TCDS = transcranial Doppler sonography; ACoA = anterior communicating artery; ICA = internal carotid artery complex; NUD = negative four-vessel angiography, origin unknown; ? = not known. For explanation of clinical grade, see text.
† Evaluation according to Fisher, et al.: Group 1 = no blood detected; Group 2 = diffuse or thin sheet of blood; Group 3 = localized clot or thick layer of blood; Group 4 = diffuse or no SAH, but with intracerebral or intraventricular clots.
‡ Emergency surgery with hematoma evacuation and aneurysm clipping performed without previous angiography.
§ Autopsy finding: angiography not performed.

bleed were investigated with TCDS. Table 1 summarizes the relevant clinical data. In 19 patients (Cases 3 to 21) TCDS was performed following the first SAH and in two (Cases 1 and 2) immediately after early rebleed. Twelve patients were in a good condition (Hunt and Hess Grade I or II), three were in Grade III, and six were in a poor condition (Grade IV or V).

The TCDS examination was routinely conducted transtemporally on the middle cerebral artery (MCA) on both sides and, when possible, on the intracranial internal carotid artery (ICA), and the anterior cerebral artery (ACA) bilaterally. Mean flow velocities in both MCA's are presented in Table 1. The normal flow velocity in the MCA, measured with our equipment, is 62 ± 12 cm/sec, and arterial narrowing or vasospasm is suspected when flow velocities exceed 120 cm/sec. Eight patients (Cases 1, 3, 6 to 8, 12, 16, and 17) did not undergo surgery. The outcome for all patients is given in Table 1.

Results
Flow velocities were below the normal range in 12 patients (57%), within the normal range in eight (38%), and slightly above normal limits in only one (Table 1). This patient (Case 2) was investigated immediately after a third early rebleed, the first occurring 1 week prior to the investigation. No patient showed increased flow velocities of the type seen in patients with cerebral vasospasm.

Following the first assessment, flow velocities were repeatedly measured in the survivors. No increased flow velocities indicative of vasospasm were recorded until after a delay of at least 4 days. In Case 1, a TCDS evaluation obtained 2 minutes after an early rebleed from an anterior communicating artery aneurysm revealed flow velocities indicative of severely increased intracranial pressure (ICP). This patient remained in deep coma, and a repeat TCDS study 2 1/2 hours after the second bleed showed complete cerebral circulatory arrest (Fig. 1). In Case 2, a TCDS evaluation 6 minutes after an early MCA aneurysmal rebleed showed flow velocities in the upper normal range in both MCA’s. In our clinic, all patients with a proven aneurysmal SAH routinely receive intravenous injections of the calcium channel blocker nimodipine. None of the patients investigated in the present study received nimodipine prior to the first TCDS assessment. Angiography performed within 24 hours after the bleed showed no evidence of large-vessel narrowing in any of the 18 patients undergoing four-vessel angiography. In Case 3 (Fig. 2), the patient developed delayed ischemic deterioration on Day 6 after SAH. A TCDS study on Day 7 revealed severely increased flow velocities in both MCA’s, indicating vasospasm (Fig. 2e). Computed tomography (CT) showed cerebral infarction.
FIG. 1. Transcranial Doppler sonograms (TCDS's) in Case 1. Upper: Studies obtained 2 minutes after a second subarachnoid hemorrhage in this 45-year-old normotensive man with ruptured anterior communicating artery aneurysm and nonruptured right middle cerebral artery (MCA) aneurysm. Flow velocities in the right MCA (left) and left MCA (right) indicate severely increased intracranial pressure. Lower: The patient remained in a deep coma and a repeat TCDS 2½ hours after the second bleed showed complete arrest of the cerebral circulation.

What happens when an intracranial aneurysm ruptures? Within seconds after the rupture a rapid increase in ICP occurs. It appears that the abrupt rise in ICP is due to the direct transmission of arterial pressure to the intracranial space. Recent physiological data with direct TCDS recordings during hemorrhage confirm an abrupt rise in ICP which may be as high as or higher than the diastolic blood pressure and result in complete cerebral compression ischemia. The increased ICP ensures hemostasis and may preserve the life of the patient if the ischemic insult does not exceed a few minutes. In such cases flow is restored and the patient may appear more or less recovered from the ill effects of the first bleed.

Our TCDS recordings within minutes after aneurysm rupture are shown in Fig. 1. Upper: Studies obtained 2 minutes after a second subarachnoid hemorrhage in a 45-year-old normotensive man with a ruptured anterior communicating artery aneurysm and a nonruptured right middle cerebral artery (MCA) aneurysm. Flow velocities in the right MCA (left) and left MCA (right) indicate severely increased intracranial pressure. Lower: The patient remained in a deep coma and a repeat TCDS 2½ hours after the second bleed showed complete arrest of the cerebral circulation.

FIG. 2. Transcranial Doppler sonograms in Case 3. a-d: Studies obtained 40 minutes after a massive subarachnoid hemorrhage (SAH) in the left (a) and right (b) middle cerebral artery (MCA) and the left (c) and right (d) anterior cerebral artery (ACA). e: Study on Day 7 post-SAH when the patient showed delayed ischemic dysfunction.

Discussion

What happens when an intracranial aneurysm ruptures? Within seconds after the rupture a rapid increase in ICP occurs. It appears that the abrupt rise in ICP is due to the direct transmission of arterial pressure to the intracranial space. Recent physiological data with direct TCDS recordings during hemorrhage confirm an abrupt rise in ICP which may be as high as or higher than the diastolic blood pressure and result in complete cerebral compression ischemia. The increased ICP ensures hemostasis and may preserve the life of the patient if the ischemic insult does not exceed a few minutes. In such cases flow is restored and the patient may appear more or less recovered from the ill effects of the first bleed.

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rupture and during the subsequent 12 hours after hemorrage favor the assumption of a transitory phase of global ischemia followed by restored flow patterns in surviving patients, and do not indicate an acute phase of vasospasm. Mean hemispheric cerebral blood flow (CBF) studies after the intravenous administration of xenon-133 in patients within 6 hours post-SAH have corroborated that there is a state of hyperperfusion rather than a decreased CBF (M Jakobsen and B Romner, et al., unpublished data).

The present findings of decreased flow velocity in large conducting arteries within 12 hours after SAH may indicate either proximal arterial dilatation or decreased global blood flow as seen in severely raised ICP or hypocapnia. The good condition of most patients at TCDS assessment contradicts severely raised ICP as a cause of the low flow velocities, whereas hypocapnia cannot be excluded as a contributing etiological factor since blood gas analyses were not routinely performed during the TCDS assessments.

The reason why many of the patients who appear to have recovered after rupture of an intracranial aneurysm develop secondary ischemic cerebral deterioration many days or even weeks later remains unexplained. Fisher and coworkers 14 investigated the relationship of the amount and distribution of subarachnoid blood (as observed on CT scans) to the later development of secondary ischemic deterioration, and concluded that subarachnoid blood in sufficient amounts is the major factor determining the occurrence of delayed cerebral ischemia. Our experience of a very low incidence of delayed ischemic deterioration following early operation with evacuation of blood clots and blood-contaminated CSF supports the concept that this mechanism is underlying the "late phase of vasospasm" (the only type of SAH-induced cerebral arterial narrowing occurring in humans).

References

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