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A transcranial Doppler method in the evaluation of cerebrovascular spasm

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Summary. An ultrasonic Doppler method was used to monitor flow velocities in basal cerebral arteries in 21 patients with spontaneous subarachnoid hemorrhage (SAH). The time course of vasospasm as evaluated by this technique was similar to that reported in angiographic studies. In 82% of the patients an increase in intracranial velocities to 120 cm/s or more was found during the second or third week after hemorrhage. (Normal value 62 cm/s). Arterial narrowing giving rise to velocities above 200 cm/s was classified as servere spasm. This occurred in 42% of the cases, and a significant decrease in flow velocity in the extracranial carotid artery was found in this group.

Key words: Subarachnoid hemorrhage – Vasospasm – Doppler ultrasound

The hemodynamic effect of vasospasm is similar to that of stenosis due to other causes, both producing in increase in flow velocity and a loss of pressure through the narrowed segment [4]. Cerebral blood flow (CBF) will be reduced when autoregulation in the distal vascular bed has been exhausted and is unable to compensate for the increased resistance of the spastic segment. Because of the effectiveness of cerebral autoregulation, this flow reducing effect will probably only be seen in severe spasm and indicates a critical condition. Thus, measurement of CBF cannot in principle reveal the effect of the onset and resolution of moderate spasms.

The pressure difference over a stenosis or spasm characterizes the hemodynamic effect of the narrowing and is the parameter of choice for the evalution of aortic or mitral stenosis as well as obstructive disease of peripheral arteries. However, such information is normally not available in the cerebral circulation. The increase in flow velocity through the stenosed or spastic segment is related to the loss of flow energy; but the geometrical complexity and the variable length of peripheral arterial narrowing do not permit a calculation of pressure loss from the Doppler shifts as described for stenosis of cardiac valves [7]. However, clinical studies have shown that the increase in Doppler shift through stenosed carotid segments is a useful parameter for the evaluation of the severity of the lesion [12].

We have previously described a method for recording Doppler signals from basal cerebral arteries through the intact cranium [2]. In a recent report [1] we found that patients with clear angiographic evidence of vasospasm in the middle cerebral arteries (MCA's) also had Doppler shifts corresponding to flow velocities in excess of 120 cm/s. This is 4 standard deviations from the mean value (62 cm/s in normal subjects [2]. One case even reached 240 cm/s. There was an inverse relationship between diameter as measured from angiograms and the flow velocity found in the Doppler recordings. The present study reports the results of transcranial Doppler for monitoring the natural course of vasospasm after SAH.

Materials and methods

From a consecutive series of 40 patients referred to us for spontaneous SAH, twenty-one cases were selected for the present series. Nineteen patients were rejected because they either had no aneurysm on angiography or because Doppler recordings could only be made for a period of less than 7 days. The aneurysms were located as follows: Nine from the anterior communicating artery (ACoA), seven on the termi-

nal internal carotid (ICA) posterior communicating artery, three on the MCA at the trifurcation, one on the basilar and one on the ophthalmic artery.

Five patients experienced major rebleeds during the period of observation and two of these died from this complication. One patient died on Day 9 as a result of vasospasm. (The day of the SAH being defined as Day zero.) The remaining 16 patients underwent surgery at the following times: three patients between Days 4 to 7, two patients between Days 8 to 11, four patients between Days 12 to 15, two patients between Days 16 to 19 and the remaining 5 after Day 20.

The basal cerebral arteries are located considerably deeper than the extracranial carotid and vertebral vessels, and they are therefore more difficult to reach with Doppler ultrasound. Furthermore, the cranial bones dampen ultrasound to some degree depending upon the thickness and probably also the consistency of the bone strucuter. It is necessary to use low ultrasonic frequencies (1.5 to 2.5 MHz) and locate those areas in the temporal region through which the ultrasound beam best penetrates. The intracranial arteries have small dimensions in comparison with a 2MHz ultrasonic probe. Therefore, to get a sufficient signal to noise ratio, a polystyrol lens was applied to the piezoelectric transducer to achieve a focusing effect at the required depth between 40 and 70 mm from the skin surface. The ultrasonic beam width as tested in water was 4 to 5 mm. The Doppler instrument used for the present investigation was of a pulsed range-gated type which permitted depth selection in steps of 5 mm. The burst length as well as the lowpass filter characteristics (before the sampling of the range-gate) were selected to give a length of about 10 mm of the sampling volume. The Doppler signals were displayed on a realtime spectrum analyzer with direction discrimination. The magnitude of the Doppler shift was determined with a cursor from the frozen spectral display; the reading represented the time-mean of the spectral outline or envelope. The velocity v (cm/s) corresponding to the Doppler shift f (Hz) was calculated from the formula: $v = 0.039 \times f$. The correspondence between the real flow velocity and that observed through the Doppler effect is only sufficiently accurate when the blood flow is insonated at sharp angles (below 30 degrees). In this respect, the MCA and the ACA are favourably located when the transducer is placed on the temporal region.

The transcranial recording procedure first requires the location of an "ultrasonic window" in the temporal region through which the Doppler shifts from the basal cerebral arteries can be observed. The window is usually found just above the zygomatic arch between the frontal process of the zygomatic bone and the front of the ear. If the patient has multiple windows, the anterior one is preferred for recording from the MCA (and the posterior cerebral artery PCA) and the posterior one for recording from the ACA and the terminal ICA. The search for the best window is made with the depth set to 50 or 55 mm, moving the probe over the surface in small steps, each time realigning the aiming angle to obtain the strongest Doppler signal. In some patients over 40 years of age the window may be very small and the Doppler signals weak. Then a precise positioning of the probe is necessary – we have found it useful to move the probe very slowly and to only make small movements each time it is realigned. In two of the cases in the present series, we were unable to obtain satisfactory Doppler signals through the intact cranium. After operation, however, excellent recordings could be made in these patients through the burrholes made at craniotomy.

Having located a suitable ultrasonic window, the next step is the identification of the various basal cerebral arteries. A manual scanning technique is used to differentiate between the vessels. The pulsed Doppler allows scanning both laterally (by tilting the probe) and in depth (by changing the position of the range-gate). The proximal portion of the MCA has a characteristic anatomical course, being the only main artery that comes laterally outwards from the Circle of Willis. The PCA and the terminal ICA both exhibit the same flow direction as the MCA, but the signals from the former arteries are always lost when scanning outwards below 55 to 50 mm, while those from the MCA may be followed up to about 30 mm. Sometimes two branches of the MCA are found at these shallow depths. Having identified the MCA, the Doppler signal is scanned inwards until a spectrum with a flow directed away from the probe is found: This represents the ACA. The signals from the PCA are located posteriorly and slightly caudally from the ICA bifurcation. The PCA can be scanned inwards to the brain midline where the contralateral PCA is observed with flow in the opposite direction. Brief compression of the common carotid artery, when indicated, helps to identify the arterey in question by observing the change in direction, magnitude and pulsatility of the Doppler shift [2].

The Doppler signals from the ICA in the neck were also recorded in the present series. In order to minimize the effect of the insonation angle on the readings, the same 2MHz Doppler instrument was used with the depth set to about 40 mm. The probe was held below the mandible and the depth setting and aim were adjusted so that the ultrasonic beam was as parallel as possible to the assumed course of

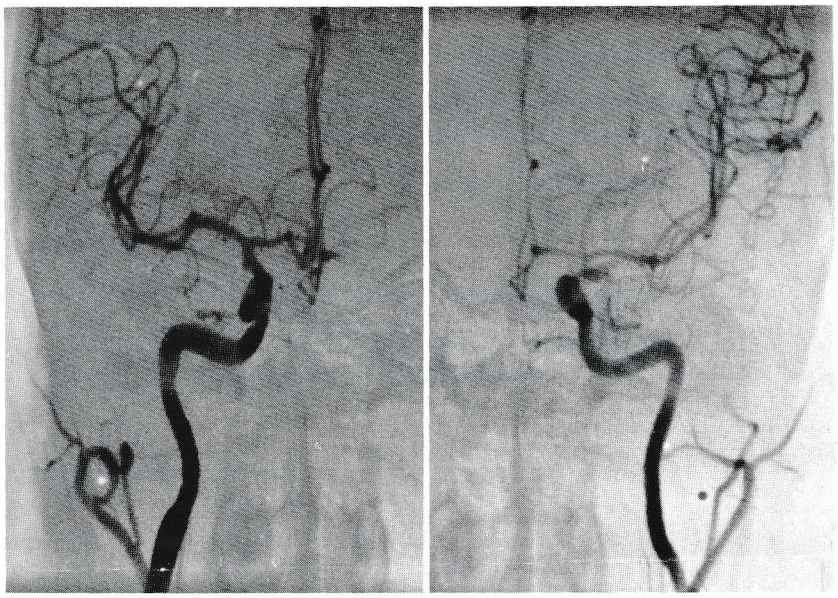

Fig. 1. Angiograms from a 30-year-old man with SAH 9 days previously. The basal cerebral arteries were clearly spastic on the left side. Also note that the left anterior cerebral artery was filled from both sides, while the right was only filled from the ipsilateral side. The cerebral arteries on the right side appear normal

the carotid arteries. The carotid bifurcation was identified and the recording was made 2 to 4 cm distal to this.

Doppler recordings were routinely taken daily in the first and second week after SAH, then the interval was increased to 2–3 days. A total of 312 transcranial Doppler investigations were made in the present series.

Illustrative case report

Angiograms from a 30-year-old man with SAH 9 days previously are shown in Fig. 1. There was an aneurysm of the left terminal ICA, and moderate vasospasm of the left MCA and ACA. The arteries on the right side showed normal diameters. The Doppler spectra from both MCA's are shown in Fig. 2. The velocity on the left side was elevated, with a time-mean of 180 cm/s: while that on the right side was within the normal range, 85 cm/s. It was possible to obtain a satisfactory spectrum from the left ACA, the right ACA had 120 cm/s. The angiogram revealed filling of both pericallosal arteries from the right side; it is therefore likely that the relatively high velocity in the right ACA was not primarily caused by narrowing of this artery, but mainly by an increase in collateral flow due to vasospasm of the left side. This case illustrates the point made by duBoulay [5] that spasm of the ACA can be less ominious than spasm of an endartery (MCA) when the Circle of Willis has sufficient collateral capacity.

Results

The findings in the entire series are shown graphically in Fig. 3. The timescale was divided into intervals of 4 days. The minimum, median, maximum and both quartiles of the highest intracranial velocity found in each patient were plotted as a trend for the series. When multiple examinations were made within each time interval of 4 days, the highest velocities found in each investigation were averaged. The values on the left represent the normal material presented previously [2]. Compared with these, the velocities were only slightly elevated during the first four days after SAH (median 80 cm/s) and velocities above 120 cm/s were seen in only one patient. In the following week (from Day 4 to Day 11), the trend was an unequivocal increase of the intracranial flow velocities reflecting the onset of vasospasm. At the end of the second week and start of the third (Days 11 to 15), 82% of the patients showed velocities of 120 cm/s in one or more arteries, and half of the cases had 160 cm/s or more. During the end of the third and the fourth week there was a marked decline in velocity trend reflecting the resolution of vasospasm. After 30 days all but one patient were below 120 cm/s; however, the group as a whole was still significantly different from the series of normals ($P < 0.05$), and the trend was still on the decrease.

One patient died from her MCA vasospasm. Her trend is shown as the maximum of the three first intervals in Fig. 3. The increase per day in this case was 36 cm/s, while the median case increased 12 cm/s per day.

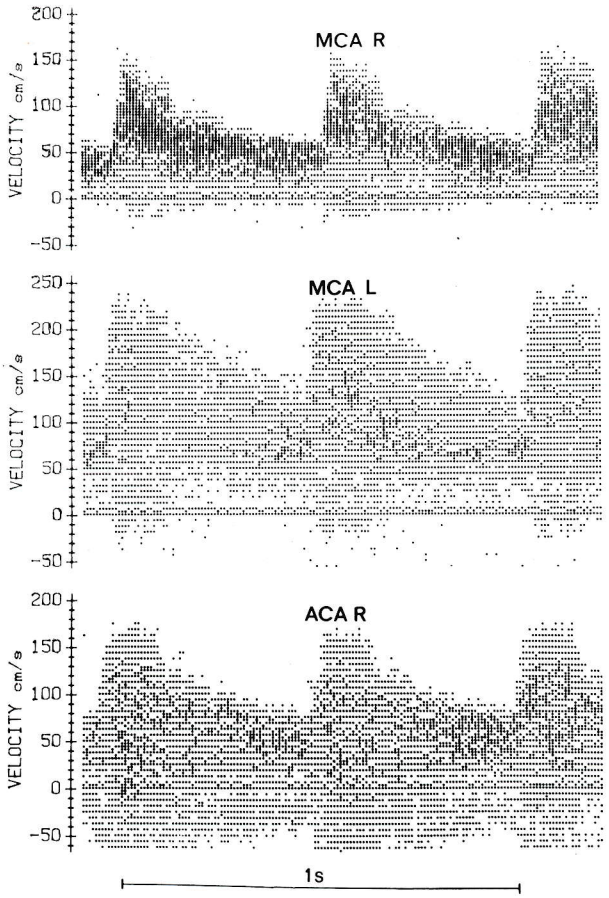

Fig. 2. Transcranial Doppler recordings from the case shown in Fig. 1. The flow velocities in the left middle cerebral artery (MCA L) were considerably elevated to a time-mean of 180 cm/s while those on the right side (MCA R) were only slightly higher (85 cm/s) than normal. The signals from the left proximal anterior cerebral artery (ACA L) could not be found, on the right side (ACA R) the flow velocity was high 120 cm/s) when compared to normal values. This probably reflects the involvement of this artery in collateral circulation (see Fig. 1)

The upper curves in Fig. 4 show the trend in the group of 9 patients who at some period had Doppler shifts exceeding 200 cm/s (circles) compared with the rest of the material (squares). The lower curves show the velocities (average of both sides) from the ICA in the neck for these groups. On days 9 to 15 there was a reduced ICA flow velocity, 31.5 cm/s, in the group with the highest intracranial velocities. This ICA velocity was significantly ($P < 0.01$) lower than that in the same groups during the fourth and fifth week when the intracranial velocites had declined. It was also significantly ($P < 0.01$) lower than the ICA velocity in the group of normals. In the patients with intracranial velocities never exceeding 200 cm/s, the reduction in ICA velocities during Days 9 to 15 was not statistically significant.

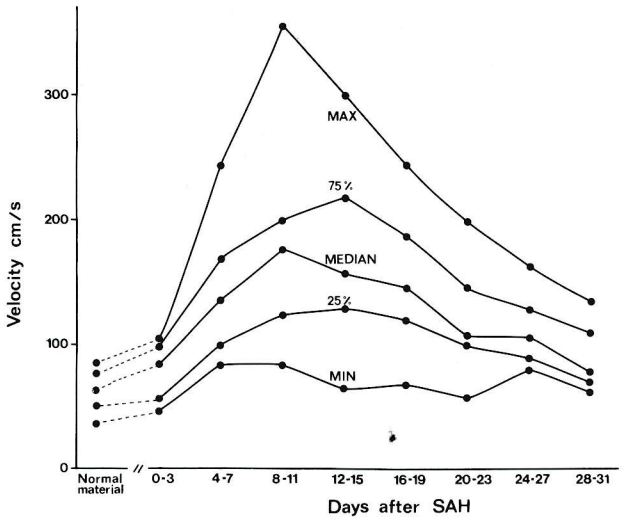

Fig. 3. Time-course of intracranial flow velocities in 21 patients with SAH. The findings are compared with a series of normal subjects. Ordinate represent velocity as determined from the Doppler shift. The artery with the highest Doppler shift was selected. Curves for the median, both quartiles and the total range of observations are shown

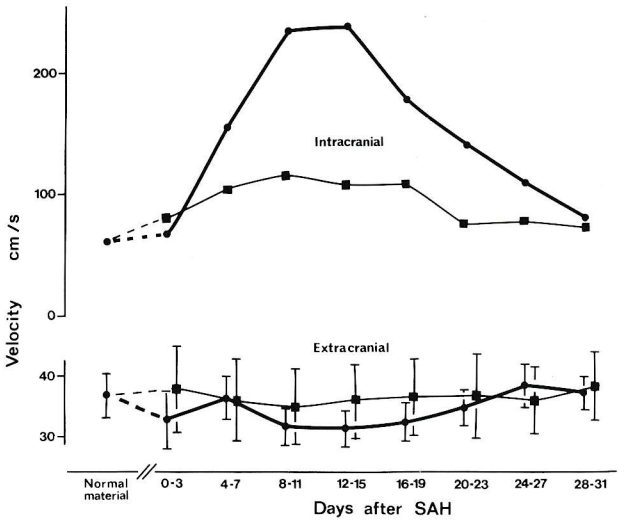

Fig. 4. Comparison of timecourses of intracranial and extracranial flow velocities in patients with severe spasm (> 200 cm/s, filled circles) and those with only moderate or no spasm *(filled squares)*. The curves represent means of each patient group with bars indicating standard devations in the lower panel

Discussion

Angiography allows direct observation of the lumen size in cerebrovascular spasm [6], while the transcranial Doppler method gives information on the velocity of flow. There is an inverse relationship between the degree of arterial narrowing and the velocity in the narrowed segment [1, 10, 12]. Preferably, each case should be compared with its own control i. e. the

intracranial flow velocities prior to the SAH. Unfortunately, this information is usually inaccessible and we have therefore compared the transcranial Doppler findings in patients with SAH to a control group reported previously [2]. Twenty percent narrowing is about the least that can be detected on a angiogram [13]. Theoretically, this reduction in arterial diameter should result in increase in intracranial flow velocity to about 97 cm/s in the MCA of a normal material. For clinical purposes, we have chosen as a preliminary threshold, a level of 120 cm/s for classification as moderate spasm and 200 cm/s for differentiation between moderate and severe spasm.

In principle, the flow velocity increase could also be caused by an augmentation in volume flow. The massive increase in the intracranial flow velocities during the second and third week after SAH was not parallelled by corresponding trend in the flow velocity of the extracranial ICA. In contrast, the latter was significantly reduced during the period of most severe spasm. This can be interpreted as a reduction in cerebral blood flow (CBF), and is in accordance with the findings of Meyer et al. [9] using the Xe inhalation method.

Figure 4 also shows that the group which later developed severe spasm, had a reduction in extracranial ICA flow velocity during the first 4 days when compared to the group that developed only moderate or no spasm. Although our material is too small to allow a conclusive discussion, it nevertheless suggests that severe spasm is preceeded by a period of cerebral blood flow impairment during the first days after SAH.

The present findings indicate that intracranial flow velocities above 200 cm/s are potentially dangerous because they herald a reduction in CBF. We have found it useful to monitor the flow velocities in the extracranial ICA, to be in a better position to evaluate the intracranial findings. One patient had a 50% reduction in the ICA flow velocity when the MCA's had Doppler shifts corresponding to over 350 cm/s – a situation which rapidly resulted in coma and cerebral circulatory arrest. On the other hand, we have observed two cases of MCA velocities of about 250 cm/s with no neurological deficits. The extracranial ICA flow velocity did not decrease significantly in these patients. It must be emphasized that the extracranial ICa Doppler recording should be made with special care to minimize the errors due to the insonation angle and variability in the placement of the sample volume. Preferably, the patients' arterial (or end-tidal) pCO2 should be measured so that variations in this parameter can be taken into consideration. This was not done routinely in the present study.

Practically all cases with clear lateralized bleedings such as that shown in Fig. 1 had considerably greater increases in the Doppler shifts on the affected side than those on the contralateral side. The results as presented in Fig. 3 are thus indicative of the severity of spasm in the most affected arterial segments.

Angiographic studies where cases of multiple hemorrhages have been eliminated, demonstrate a low incidence of spasm during the first 4 days after the SAH [8, 11]. This is in accordance with the present findings. During the first four days there was only one case that could be classified as having moderate spasm. Two cases were investigated within 10 h of the first hemorrhage; both these had rather low flow velocities (less than 40 cm/s) in all the basal cerebral arteries. Furthermore, no evidence of an early phase of spasm was found in one case that was investigated within an hour after a rebleed.

The trend of rising intracranial flow velocities during the end of the first and the start of the second week corresponds to the decrease in arterial lumen observed in angiographic studies during this phase [8, 11]. We have found it useful to calculate the rise in velocity per day to predict the severity of the spasm before the maximum is in fact reached. In our opinion patients with rapidly increasing spasm are particularly vulnerable to an ill-timed operation. The inherent day-to-day variability of the readings due to variations in pCO2 etc. do not permit calculation of the rise per day from readings only 24 h apart. Multiple readings within an interval of at least 48 h is in our opinion the minimum requirement for calculation of such a rise-per-day index. If the intracranial velocities increase by more than 25 cm/s per day in this period, the patient will usually end up with severe spasm in the second or even the third week.

The culmination of vasospasm was indicated by the velocity trend levelling out and reaching a peak between Day 12 and Day 15. This is somewhat later than reported in angiographic studies [8, 11, 14]. Furthermore, the present series indicates an even higher incidence of vasospasm, 82%, than seen in angiographic studies [8, 11].

The resolution of vasospasm was slower than the onset; the average case had a decline of 6 cm/s per day in the third and fourth weeks.

The data presented here are representative of the development of vasospasm after SAH when the patient undergoes surgery relatively late. Although the present material is not conclusive in this respect, it is our impression that surgery after Day 10 does not influence the course of vasospasm very much.

Angiography is mandatory in treating patients with SAH to localize the source of bleeding. In addi-

tion there may be an indication for a repeat angiographic examination prior to a decision to operate in order to determine the degree of vasospasm. The good correspondence between the transcranial Doppler findings and those of angiography [1] suggests that the noninvasive ultrasonic approach may replace repeat angiographic investigation for this purpose. We have also found it very useful to monitor the timecourse of the intracranial as well as the extracranial flow velocities in the individual case to improve the general handling of patients with SAH.

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