

# Monitoring of Venous Hemodynamics in Patients With Cerebral Venous Thrombosis by Transcranial Doppler Ultrasound

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**Objectives:** To test the assumption that transcranial Doppler ultrasound (TCD) is able to detect and to monitor intracranial venous blood flow velocities in patients with confirmed cerebral venous thrombosis (CVT).

**Design:** Prospective case study in 18 patients.

**Setting:** Inpatient neurologic service in a university hospital.

**Subjects and Methods:** Serial TCD examinations were performed in 18 consecutive patients with CVT (14 females, 4 males) aged 16 to 64 years (mean  $\pm$  SD,  $36.8 \pm 13.1$  years) during a mean follow-up ranging from 34 to 783 days (mean  $\pm$  SD,  $201 \pm 185$  days) between 1993 and 1997. Venous TCD was performed with a 2-MHz range-gated transducer.

**Results:** Venous blood flow velocities were successfully measured in all patients. The highest measured velocities in the monitored intracranial venous vessels ranged from 20 to 150 cm/s (mean  $\pm$  SD,  $58.9 \pm 38.8$  cm/s), and the lowest were from 9 to 84 cm/s (mean  $\pm$  SD,  $27.9 \pm 17.0$  cm/s).

Fifteen patients (83%) showed a decrease of velocities—2 of them after a transient increase during cessation of heparin therapy. The percentage of velocity decrease ranged from 34% to 73% (mean  $\pm$  SD,  $56.4\% \pm 10.9\%$ ). A plateau phase, defined as no further decrease in velocities, was reached in these patients within 4 to 314 days (mean  $\pm$  SD,  $59.9 \pm 73.7$  days). Three patients (17%) showed no changes in velocities as defined by a limit of velocity variation of 30% during the course of CVT. High venous velocities were significantly associated with altered consciousness ( $P = .001$ ). A nonsignificant relationship was observed with affliction of the superior sagittal sinus. No correlations were noted for onset of disease, initial motor deficits, and presence of bleeding. No predictive value was gained from analyzing the outcome in relation to absolute velocities or their decrease.

**Conclusions:** Serial TCD studies allow monitoring of venous hemodynamics and collateral pathways in patients with CVT. Normal venous velocities in serial measurements, however, do not exclude a diagnosis of CVT.

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**D**ESPITE THE evident importance of venous collateral pathways in cerebral venous thrombosis (CVT), the interest in the assessment of the collateral pathways was limited. One major reason for this neglect has been the invasiveness of digital subtraction angiography (DSA) impeding serial examinations. Magnetic resonance (MR) imaging and MR angiography have focused new interest in the evaluation and monitoring of the thrombotic process.<sup>1-3</sup> Their usefulness for analyzing hemodynamic properties, however, has been limited until now. Doppler ultrasound techniques have become the standard modality in the diagnosis and follow-up of thrombotic lesions of other body regions, especially in deep venous thrombosis of the legs.<sup>4</sup> Their application to the cerebral cir-

culation was hindered mainly by bony limitations. In the past few years, however, attempts have been made to assess the normal cerebral venous circulation by means of conventional transcranial Doppler ultrasound (TCD),<sup>5-8</sup> and transcranial color-coded duplex sonography.<sup>9-12</sup> Furthermore, elevated venous blood flow velocities have been reported in single adult cases with thrombotic occlusion of the major venous outlets.<sup>9,13,14</sup> In some instances, follow-up examinations were performed that demonstrated dynamic changes in venous vessels.<sup>15-17</sup> The ability to monitor venous hemodynamics over longer periods has not yet been shown in a larger group of patients. We intended to prove in a prospective study the assumption that TCD allows the detection and monitoring of blood flow velocities in venous vessels of patients with confirmed CVT. In addition, we

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## PATIENTS AND METHODS

Eighteen consecutive patients (14 females, 4 males) aged 16 to 64 years (mean  $\pm$  SD age,  $36.8 \pm 13.1$  years) with CVT confirmed by DSA and/or MR angiography were examined between 1993 and 1997. Doppler ultrasound was performed with a 2-MHz range-gated transducer (Multidop-X and Multidop-X4; DWL, Sipplingen, Germany). Almost all studies were performed by the same examiner (J.M.V.). The examination was performed in the supine position when using the transtemporal and transorbital approaches. The suboccipital access was performed with the patient in the supine position with mild elevation of 1 shoulder and the head slightly rotated. Most of the venous vessels were identified by concomitant insonation of specific arteries. The basal vein of Rosenthal (BVR) was insonated through the posterior temporal window at a depth of about 60 mm together with the P2 segment of the posterior cerebral artery. The deep middle cerebral vein (DMCV) was defined by its proximity to the middle cerebral artery. In both vessels, flow is directed away from the probe.<sup>6</sup> Venous vessels in the anterior parasellar region were insonated through the anterior temporal window at a depth of 50 to 60 mm or by using the transorbital route at a depth of 70 mm with a flow away from the probe. They were defined by their proximity to the internal carotid artery siphon and considered to correspond to the main inflow vessels to the cavernous sinus (CS), especially to the sphenoparietal sinus or to a sylvian vein.<sup>7</sup> The inferior petrosal sinus (IPS) was detected by using the suboccipital approach by its proximity to the basilar artery at a depth of about 80 to 90 mm with a flow directed toward the probe.<sup>8</sup> The  $V_{mean}$  (the time-averaged mean velocity over the cardiac cycle of the spectral outline) was automatically recorded in centimeters per second. Reference values were derived from our own series,<sup>6-8</sup> with a  $V_{mean}$  ( $\pm$ SD) of  $11.1 \pm 2.7$  cm/s in the DMCV,  $10.1 \pm 2.3$  cm/s in the BVR,  $27.3 \pm 17.4$  cm/s in the anterior CS inflow region, and  $20.8 \pm 9.2$  cm/s in the IPS. A definite increase or decrease of velocity was defined by a change of at least 30%. The pulsatility index was defined as systolic velocity minus diastolic velocity divided by  $V_{mean}$ . For statistical evaluation, paired *t* tests were performed. A *P* value less than .05 was considered significant.

studied the relationship between venous blood flow velocities and severity of disease, site of thrombosis, presence of bleeding, and outcome.

## RESULTS

Patient data, neuroimaging results, and TCD findings are summarized in the **Table**.

### CLINICAL AND NEUROIMAGING FINDINGS

Eighteen patients with proven CVT were examined. A sudden onset of symptoms was found in 6 patients. Ten

patients presented with a subacute course (up to 4 weeks) and the remaining 2 patients had a chronic course. Digital subtraction angiography and/or MR angiography revealed thrombosis of the superior sagittal sinus (SSS) in 14 patients, of the lateral sinuses in 17, of the galenic system in 7, and of cortical veins in 2. Nonhemorrhagic venous infarcts were found in 6 patients, and 5 patients had computed tomographic evidence of hyperdense lesions. Therapy with heparin with doubling of the partial thromboplastin time, followed by warfarin sodium therapy for at least 6 months, was the standard treatment.

### TCD FINDINGS

All patients underwent 3 to 25 serial TCD examinations (mean  $\pm$  SD,  $10.4 \pm 6.8$  times) during a follow-up ranging from 34 to 783 days (mean  $\pm$  SD,  $201 \pm 185$  days). Venous signals were detected in all patients. We intended to insonate a maximum of venous vessels in each patient. One dominant and frequently insonated venous vessel was selected for follow-up monitoring. The inflow pathways to the CS were selected in 11 patients: in 4 of them using the transorbital approach, the IPS in 3, the BVR in 2, and the DMCV and a vessel considered to represent the superior petrosal sinus each in 1 patient. There was a marked increase in  $V_{mean}$  in all vessels of the patients with CVT compared with the normal reference values: in the inflow area of the CS,  $68.7 \pm 41.7$  cm/s ( $n = 11$ ) vs  $27.3 \pm 17.4$  cm/s (*P* = .007); in the BVR,  $23.0 \pm 3.0$  cm/s ( $n = 2$ ) vs  $10.1 \pm 2.3$  cm/s (*P* = .07); in the DMCV,  $34.0$  cm/s ( $n = 1$ ) vs  $11.1 \pm 2.7$  cm/s; and in the IPS,  $40.7 \pm 16.4$  cm/s ( $n = 3$ ) vs  $20.8 \pm 9.2$  cm/s (*P* = .1). A definite decline of  $V_{mean}$  was obvious during follow-up in all selected vessels. The measured steady state values, defined as no further decrease of velocities, were found to be  $33.1 \pm 21.2$  cm/s in the inflow of the CS (*P* = .007),  $9.5 \pm 0.5$  cm/s in the BVR (*P* = .06),  $10.0$  cm/s in the DMCV, and  $23.3 \pm 4.7$  cm/s in the IPS (*P* = .09). There was no difference between the plateau velocities in patients and the results of the normal population. Taking all examined vessels into account, the highest measured velocities throughout the follow-up ranged from 20 to 150 cm/s (mean  $\pm$  SD,  $58.9 \pm 38.8$  cm/s), and the lowest were from 9 to 84 cm/s (mean  $\pm$  SD,  $27.9 \pm 17$  cm/s).

Fifteen patients (83%) had a decrease of velocities. In 2 of them, a transient increase of velocities occurred during intermittent cessation of high-dose heparin therapy due to extracranial hemorrhagic complications. The decrease of velocities over time showed a wide variability. The steady state phase was reached within 4 to 314 days (mean  $\pm$  SD,  $59.9 \pm 73.7$  days). Seven patients reached the plateau phase within 1 month (2 of them during the first week), 6 further patients within 3 months, 1 patient within 6 months, and 1 within 1 year. The fall of velocities ranged from 34% to 73% (mean  $\pm$  SD,  $56.4\% \pm 10.9\%$ ). No relevant changes, defined as a limit of velocity variation of 30% during the course of disease, were noted in 3 patients (17%). A clear envelope for recording the pulsatility index could be obtained in about 50% of recordings. The pulsatility index was low, usually ranging between 0.2 and 0.6. During episodes of elevated intra-

## Summary of Patient, Clinical, and Doppler Data\*

| Patient No./Sex/Age, y | Outcome        | Site of Thrombosis  | Follow-up, d | Maximum V <sub>mean</sub> , cm/s | Minimum V <sub>mean</sub> , cm/s | Mean Fall of V <sub>mean</sub> , % | Steady State Phase, d |
|------------------------|----------------|---------------------|--------------|----------------------------------|----------------------------------|------------------------------------|-----------------------|
| 1/F/31                 | Total recovery | SSS, R-TS, SS, DV   | 97           | 150                              | 60                               | 60                                 | 4                     |
| 2/F/36                 | Total recovery | L-TS, CV            | 60           | 20                               | 12                               | 40                                 | 30                    |
| 3/F/16                 | Total recovery | SSS, R-TS           | 305          | 20                               | 9                                | 55                                 | 76                    |
| 4/M/32                 | Total recovery | R-TS                | 286          | 24                               | 20                               | 17                                 | †                     |
| 5/F/22                 | Total recovery | TS, SS, DV          | 190          | 25                               | 15                               | 40                                 | 110                   |
| 6/F/64                 | Major sequelae | SSS, R-TS, L-TS, CV | 87           | 50                               | 24                               | 52                                 | 21                    |
| 7/F/23                 | Total recovery | SSS, R-TS, SS       | 31           | 75                               | 20                               | 73                                 | 16                    |
| 8/M/52                 | Minor sequelae | SSS, R-TS           | 8            | 24                               | 20                               | 17                                 | †                     |
| 9/M/28                 | Minor sequelae | SSS, SS, DV         | 34           | 35                               | 23                               | 34                                 | 34                    |
| 10/F/27                | Total recovery | SSS                 | 453          | 63                               | 30                               | 52                                 | 314                   |
| 11/F/43                | Minor sequelae | L-TS, SS, DV        | 265          | 103                              | 40                               | 61                                 | 53                    |
| 12/F/60                | Total recovery | SSS, R-TS, L-TS     | 185          | 58                               | 24                               | 58                                 | 50                    |
| 13/F/27                | Total recovery | SSS, L-TS, SS       | 43           | 35                               | 20                               | 62                                 | 43                    |
| 14/F/45                | Major sequelae | SSS, R-TS, L-TS, SS | 37           | 120                              | 48                               | 60                                 | 7                     |
| 15/F/33                | Minor sequelae | SSS, R-TS, L-TS     | 783          | 26                               | 10                               | 61                                 | 26                    |
| 16/F/53                | Total recovery | SSS, R-TS           | 70           | 95                               | 84                               | 10                                 | †                     |
| 17/F/34                | Total recovery | SSS, R-TS           | 142          | 34                               | 10                               | 71                                 | 86                    |
| 18/M/36                | Total recovery | SSS, R-TS           | 155          | 104                              | 34                               | 67                                 | 29                    |
| Mean ± SD              |                |                     |              |                                  |                                  |                                    |                       |
| 36.8 ± 13.1 (age)      | ...            | ...                 | 201 ± 185    | 58.9 ± 38.8                      | 27.9 ± 17.0                      | 56.4 ± 10.9†                       | 59.9 ± 73.7           |
| Range                  |                |                     |              |                                  |                                  |                                    |                       |
| 16-64 (age)            | ...            | ...                 | 34-783       | 20-150                           | 9-84                             | 34-73                              | 4-314                 |

\*V<sub>mean</sub> indicates time-averaged mean velocity over the cardiac cycle of the spectral outline; R, right; L, left; SSS, superior sagittal sinus; SS, straight sinus; TS, transverse sinus; DV, deep veins; CV, cortical veins; and ellipses, not applicable.

†Not considered because the variation of velocity was <30%.

‡The values of patients 4, 8, and 16 are not included.

cranial pressure, the arterial Doppler spectra revealed a higher pulsatility transiently. The arterial blood flow velocities usually remained in the normal range.

In cases of SSS thrombosis, higher velocities were found than in cases of thrombosis at other locations (63.5 vs 43.0 cm/s). Velocities were also higher in complete occlusions as compared with partial occlusions (71.6 vs 59.0 cm/s). However, these differences did not reach statistical significance. Initial impairment of consciousness, on the other hand, was significantly associated ( $P = .001$ ) with higher venous velocities (119.3 vs 41.1 cm/s). No obvious relationship was seen between venous velocities and the presence of motor deficits or bleeding. Outcome was not correlated to the initial range of velocities or to the degree of regression.

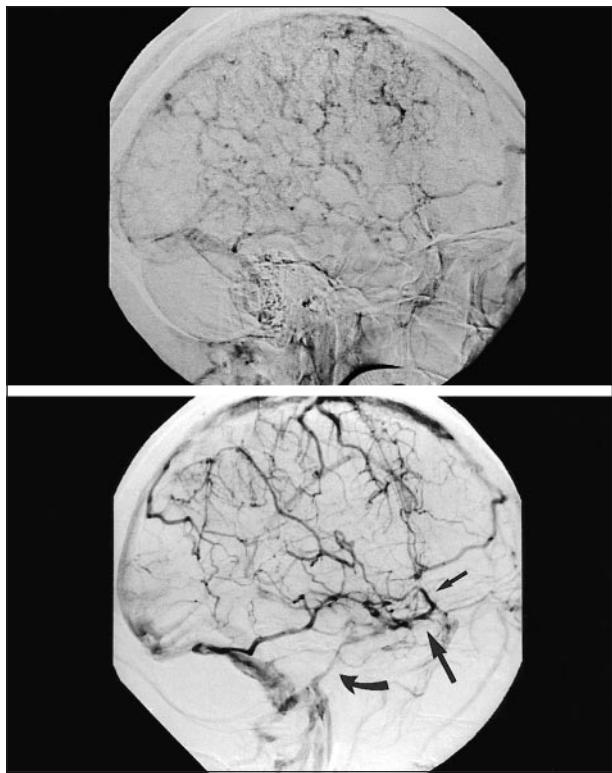
### ILLUSTRATIVE CASE

A 31-year-old woman (patient 1) presented with a 5-day history of elevated intracranial pressure. On admission, she complained of headaches and double vision. Clinically, she was alert and had a left-sided hemiparesis. A native computed tomographic scan was unremarkable. Digital subtraction angiography revealed CVT with major affliction of the SSS, the straight sinus, the right transverse sinus, and the deep internal veins (Figure 1, top). On transtemporal TCD examination, a prominent venous signal with a velocity of 150 cm/s and a flow directed away from the probe, considered to represent an inflow vessel to the CS, was found in the left parasellar region at a depth of 52 to 62 mm. Partial thromboplastin time-adjusted high-dose heparin therapy was followed by clinical normalization within 48 hours. Dur-

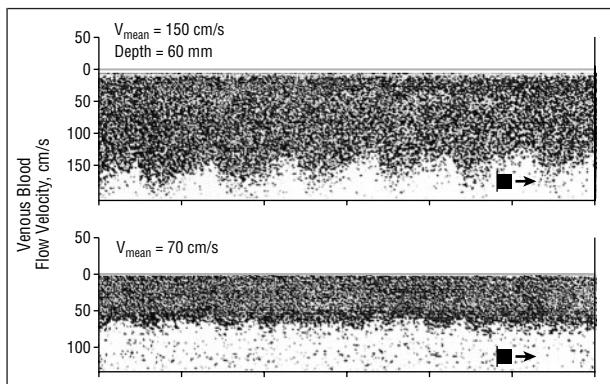
ing the first 4 days, the venous V<sub>mean</sub> dropped to values between 60 and 70 cm/s and remained in this range during the following week (Figure 2). Two weeks after admission, she developed clinical signs of tentorial herniation. A right-sided subdural hematoma was seen on computed tomography with midline shift and marked brain edema. Venous blood flow velocities remained unchanged during that time, suggesting a nonthrombotic cause of the subdural hematoma, which was confirmed on repeated angiography (Figure 1, bottom). During cessation of heparin treatment after surgery, the velocities increased transiently to 105 cm/s, reaching again steady state values of 60 to 70 cm/s during adjusted anticoagulation (Figure 3). The patient's condition normalized within 6 weeks and she was discharged from the hospital with warfarin medication. During a follow-up of 97 days, the venous velocities remained substantially unchanged.

### COMMENT

Many aspects regarding the pathophysiology of CVT are not well understood.<sup>18,19</sup> Considering similar mechanisms as in deep vein thrombosis, however, an evolving process with instantaneous changes of venous hemodynamics has to be assumed due to continuous recanalization and subsequent new thrombus formation. The severity of the disease seems—apart from the site of the occlusion—to depend on the time course of the thrombus formation and the capacity to build up a compensatory venous circulation. This may be by using preexisting nonaffected veins and sinuses and/or by opening up of collaterals. In contrast to the dural sinuses, the cere-

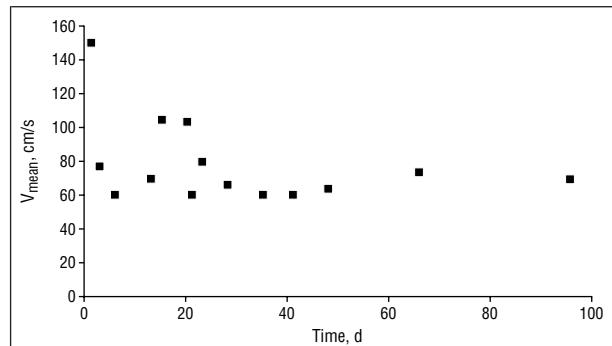


**Figure 1.** Angiogram of lateral venous phase of left carotid artery demonstrating severe cerebral venous thrombosis in patient 1 with extensive filling defect of the superior sagittal sinus and the galenic system (top). A marked improvement is seen on repeated angiography after 2 weeks of anticoagulation therapy with a prominent venous drainage into the sphenoparietal sinus (small arrow), cavernous sinus (large arrow), and inferior petrosal sinus (curved arrow) (bottom).



**Figure 2.** Venous transcranial Doppler ultrasound revealing mean venous blood flow velocities ( $V_{mean}$ ) of 150 cm/s in patient 1 (top) that decreased to a plateau of about 70 cm/s within 4 days of anticoagulation therapy (bottom). A transient elevation occurred after evacuation of subdural hematoma and cessation of anticoagulant therapy. Arrows indicate flow away from the probe.

bral veins are able to extend their walls. Thus, dilation of the veins may be seen as an indirect sign of CVT on DSA examination. The dynamic aspects of CVT, however, cannot be studied in follow-up examinations by DSA due to its invasiveness. Magnetic resonance imaging and MR angiography offer an important advance as diagnostic and follow-up instruments. Visualization of vessels, however, does not necessarily correspond to the vessels' functional status. The additional application of MR flow



**Figure 3.** Time course of mean venous blood flow velocities ( $V_{mean}$ ) in patient 1.

measurements of the venous system will probably further contribute to the assessment of the dynamic aspects of CVT.<sup>20,21</sup>

#### NORMAL VENOUS BLOOD FLOW VELOCITIES

In the past years, normal venous  $V_{mean}$  values were established in various cerebral veins and sinuses in adults by Doppler ultrasound methods. The recognition of the venous character of an intracranial Doppler signal can be assured by its typical low pulsatile flow with a pulsatility index ranging between 0.2 and 0.5, its reaction pattern during the Valsalva test demonstrating marked transients of flow velocities, and the whispering sound clearly different from an arterial signal. Using TCD, venous vessels are identified by their known anatomical relationship to certain arteries, eg, the DMCV to the middle cerebral artery, the BVR to the posterior cerebral artery, the inflow vessels to the CS to the carotid siphon, and the IPS to the basilar artery. Normal venous  $V_{mean}$  values have been reported to be  $11.1 \pm 2.7$  cm/s in the DMCV,  $10.1 \pm 2.3$  cm/s in the BVR,<sup>6</sup>  $27.3 \pm 17.4$  cm/s in the anterior CS inflow<sup>7</sup> through a transtemporal approach, and  $20.8 \pm 9.2$  cm/s in the IPS via a suboccipital approach.<sup>8</sup> Similar values in the DMCV and the BVR have been reported using transcranial color-coded duplex sonography.<sup>12</sup> The straight sinus was not studied in our series because of its limited transoccipital accessibility. On transoccipital insonation of the straight sinus, a  $V_{mean}$  of the straight sinus of  $20.0 \pm 3.0$  cm/s using TCD<sup>5</sup> and a peak velocity of 35.0 cm/s using transcranial color-coded duplex sonography<sup>11</sup> were obtained. By using a transtemporal approach, the straight sinus revealed a lower peak velocity of  $19.1 \pm 6.1$  cm/s.<sup>9,12</sup>

#### VENOUS VELOCITIES IN CVT

In SSS thrombosis, a  $V_{mean}$  in the BVR ranging from 19.5 to 43.0 cm/s was reported in 3 and 4 patients, respectively, by single TCD measurements.<sup>13,17</sup> Using color-coded transcranial color-coded duplex sonography via a temporal access, venous peak velocities of 83.0 and 92.0 cm/s were found in the straight sinus.<sup>9</sup> Significant differences of venous blood flow velocities in patients with thrombosis of the transverse sinus were reported using echocontrast-enhanced transcranial color-coded du-

plex sonography. Peak velocities were found to be  $28.4 \pm 6.5$  cm/s in the nonaffected transverse sinus and  $9.4 \pm 4.0$  cm/s in the hypoplastic or partially occluded transverse sinus.<sup>14</sup>

The interpretation of venous hemodynamics from single observations, however, may be limited by the high variability of normal venous blood flow velocities, which is of special relevance in the inflow area of the CS. In our series of long-term follow-up examinations, a marked decrease of velocities during the course of the disease was noted in 15 patients (83%). Elevated venous velocities were thought to correspond to the venous collateral pathways that were also documented on angiography. Especially in patients with marked venous signals at the CS and affliction of the SSS, DSA depicted a prominent sphenoparietal sinus, CS, and IPS in the majority of cases. A steady state was achieved after 4 to 314 days (mean, 59.9 days), which indicates that the hemodynamic changes may last several months, but may also be resolved within a few days. A variety of morphologic changes over time has also been found in MR studies: the thrombus may be resolved or partially reopened within several weeks.<sup>22,23</sup> Complete recanalization has been observed in 2 series after 6 months in 3 of 10 patients<sup>1</sup> and 5 of 8 patients.<sup>2</sup> Abnormalities, however, may be present for years.<sup>3</sup> Considering the MR data, a steady state of venous blood flow velocities seems to represent the point in time when adequate collateralization is achieved rather than indicating complete resolution of the thrombus.

Two of our patients experienced a rise of velocities during perioperative interruption of intravenous heparin therapy. In at least 1 of them, the interruption was followed by a severe progress of CVT, which adds further evidence of the importance of anticoagulation.<sup>19,24</sup> No changes were noted in 3 patients, with 1 of them exhibiting symptoms of pseudotumor cerebri starting 3 months before admission, and the other 2 experiencing mild symptoms and clinical signs. Thus, normal blood flow velocities in examined venous vessels do not exclude CVT. This was also shown in 2 of 6 patients with SSS thrombosis by analyzing the BVR and DMVC.<sup>13</sup> It seems, however, that velocities in the normal range are associated with a less severe clinical presentation and more chronic evolution. On the other hand, high venous velocities—especially measured at the CS inflow—are not necessarily a sign of compromised venous hemodynamics.

In 11 patients, the area of the CS was chosen as the reference vessel for follow-up. Using DSA and MR angiography, this area was identified as the most relevant drainage pathway in CVT independent of the site of thrombosis. The IPS was selected in 3 patients. It usually communicates with the sphenoparietal sinus via the CS and has the advantage of only moderate variations of normal velocities. However, because of the necessity to manipulate the head position during the examination, this approach cannot be recommended in severely affected patients. The BVR was selected for monitoring purposes in 2 patients. This vessel is not difficult to insonate. From angiographic studies in cases of SSS thrombosis, it is evident that the basal veins form a part of the compensatory circulation. However, due to the abil-

ity of the vein to dilate, the rise in venous blood flow velocity may be limited. Consequently, normal velocities in the BVR do not exclude elevated flow in this vessel. In our series, only 7 of 14 patients with a clearly demonstrable BVR and otherwise elevated venous velocities had elevated velocities in the BVR. In 1 patient with occlusion of the straight sinus, the BVR revealed a reverse flow. This type of collateralization is shown on DSA in patients with occlusion of the straight sinus due to meningiomas<sup>25</sup> and in deep-seated arteriovenous malformations<sup>26</sup> and has also been observed in 1 infant with thrombosis of the straight sinus.<sup>27</sup> One prominent signal was found using a posterior temporal approach that could not be assigned to a specific vessel. From the selected Doppler settings and angiographic findings, insonation of the superior petrosal sinus was assumed.

## LIMITATIONS

Increasing bony thickness in elderly patients may hinder the insonation of intracranial vessels. As CVT is usually encountered in patients of younger age, this will be a minor limitation. In our series, only in 1 patient was there no detectable signal transtemporally. Follow-up studies should require a reliable anatomical identification of the insonated vessels. This was not achieved in many patients when monitoring the inflow region of the CS. Vessel identification problems may be overcome by transcranial color-coded duplex sonography. However, a significantly lower detection rate using transcranial color-coded duplex sonography has to be considered.<sup>28</sup> By using echo-contrast agents, the detection rate of intracranial venous vessels will improve. Furthermore, raised intracranial pressure may alter venous hemodynamics and flow velocities. Localized brain edema may also cause vessel displacement, leading to uncertainties in vessel identification. For follow-up examinations, however, it seems more important to define a reference venous vessel using constant Doppler settings, ie, probe position, depth, sample volume, and relationship to arterial signals, than to be sure of its designation. Finally, the large variation in normal venous velocities, especially in the inflow area of the CS, is also a major obstacle hindering a simple interpretation of velocity data.

## CONCLUSIONS

The present study provides further support that TCD enables serial venous velocity measurements in venous collateral pathways in patients with CVT. There was an obvious relationship between initially high venous flow velocities and severity of disease, but not with outcome. Early regression of velocities was not found to indicate a favorable prognosis, but could be observed in the majority of cases. Transcranial Doppler ultrasound cannot replace the diagnostic value of angiography or MR modalities; however, it may offer the possibility to evaluate noninvasively dynamic changes in the venous circulation of the brain in normal and altered states. Whether the assessment of functional parameters of venous hemodynamics in CVT will influence decisions on therapy and its duration has to be investigated in future studies.

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