

Assessment of Intracranial Venous Hemodynamics in Normal Individuals and Patients With Cerebral Venous Thrombosis

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Background and Purpose—Despite ongoing improvements in noninvasive imaging techniques, transcranial color-coded duplex sonography (TCCS) has so far been used only on a limited basis in patients with cerebral venous thrombosis. We evaluated the diagnostic value of both noncontrast and contrast-enhanced TCCS by comparing normal volunteers and patients with acute cerebral venous thrombosis.

Methods—In 75 healthy volunteers (aged 45.8 ± 17.4 years), normal values for the deep cerebral veins (DCVs) and the posterior fossa sinuses were established by transtemporal insonation. Eight patients with cerebral venous thrombosis were assessed by TCCS, through which the hemodynamics of the DCVs were measured, and the patients were followed-up over a period of between 33 and 387 days after examination. MR angiography served as the “gold standard” technique for confirming the venous status in all 8 patients.

Results—No side differences in flow velocities were detected in the paired venous structures in normal volunteers. As indirect signs of (and diagnostic criteria for) cerebral venous thrombosis, pathologically increased flow velocities or significant side differences in the DCVs were registered in 5 of the 8 patients; the other patients showed nonsignificant increases in flow velocity which decreased over time. During follow-up, the status of the posterior fossa sinuses could be diagnosed correctly in seven patients after contrast enhancement when these results were compared with those of venous MR angiography. In 1 patient, a partial recanalization was mistakenly diagnosed as an occlusion.

Conclusions—TCCS allows a reliable evaluation of the major DCVs and posterior fossa sinuses. The anterior and mid portions of the superior sagittal sinus and cortical veins cannot be assessed. Increased venous blood flow velocity can be used as an indirect criterion for indicating a cerebral venous thrombosis. Clinical recovery coincided with decreases in blood flow velocity in the series of patients investigated in this study. (*Stroke*. 1999;30:70-75.)

Key Words: ultrasonography, Doppler ■ TCCS ■ cerebral veins ■ cerebral sinus ■ dural sinus thrombosis

Cerebral venous thrombosis is considered a differential diagnosis in patients with headache and cerebrovascular disease. Because of the wide spectrum of clinical manifestations of this disease, which range from no symptoms at all to severe venous infarction, an invasive diagnostic approach is not preferable and noninvasive techniques are urgently required to allow the best clinical decisions to be made. Unlike both venous MR angiography (MRA) and CT venography,¹ transcranial ultrasound has not until now played a significant role in this area. Reports on the application of ultrasonographic techniques in cerebral venous thrombosis remain largely anecdotal.

The main disadvantage of conventional transcranial Doppler sonography is the need for arterial landmarks to locate the venous structures and the inability to reliably insonate the dural sinuses. Encouragement for a new approach is now provided through the use of transcranial color-coded sonography (TCCS) combined with contrast enhancing agents. Such agents can elevate low-intensity Doppler signals from

intracranial venous vessels above the detection threshold and allow an unambiguous anatomic allocation of the backscattered signals. Only a limited number of patients have been studied to date; these studies have indicated either disturbed venous hemodynamics²⁻⁶ or the absence of venous segments,⁷ both of which suggest the presence of thrombosis. Furthermore, studies of venous hemodynamics are also intriguing, because our knowledge about the pathophysiology of congestive cerebral bleeding is still rather preliminary.

For these reasons we monitored venous flow velocities in the deep cerebral veins (DCVs) of 8 consecutive patients with cerebral venous thrombosis.

Subjects and Methods

TCCS Examination Technique

All examinations were performed according to previously published examination protocols.^{6,8} The temporal bone window was insonated with use of a TCCS system (Hewlett Packard, Sonos 1000 and 2000)

Received July 21, 1998; final revision received September 18, 1998; accepted September 29, 1998.

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equipped with a 2.0-MHz sector transducer. All subjects were examined in the supine position.

The color program was optimized to achieve the highest sensitivity for low blood flow velocities: after the arteries of the circle of Willis were identified, pulse repetition frequency was reduced to the lowest possible setting and color gain was adjusted to the optimal signal-to-noise ratio.

The deep middle cerebral vein (dMCV) was located slightly posterior to the middle cerebral artery (MCA) at an insonation depth of 40 to 60 mm, with a flow direction away from the transducer. The basal vein of Rosenthal (BV) follows the course of the posterior cerebral artery (PCA) and was insonated posteriorly and cranially to the P2 segment. Flow was directed away from the probe. The depth of the examination window was then adjusted so that the contralateral skull became visible. After an upward tilt of the probe to the level of the third ventricle, the vein of Galen (VG) could be examined in the midline just posterior to the pineal region. The anterior tip of the transducer was then rotated upward to align the apex of the cerebellar tentorium and the internal occipital protuberance in the insonation plane. In this position, the straight sinus was located by following the flow direction of the VG away from the transducer. The straight sinus was insonated in its middle portion to distinguish it from the VG and the confluens sinuum. The superior sagittal sinus was located above the internal occipital protuberance, where the direction of flow was toward the probe. For visualizing the confluens sinuum and transverse sinus, the anterior tip of the transducer was then rotated downward to the nose saddle again and the probe as a whole was tilted downward to the cranial base (Figure 1A). The contralateral transverse sinus displayed a flow direction away from and the ipsilateral transverse sinus one toward the transducer.

Venous peak-systolic blood flow velocity (PSV) and end-diastolic velocity (EDV) as well as identification rates were recorded. Because of difficulties with the reliable construction of an envelope curve around the venous Doppler spectra, the measurement of mean flow velocities was not performed.

Reference Values

To establish normal values for the DCVs and posterior fossa sinuses, 75 healthy volunteers (aged 45.8 ± 17.4 years; median, 44 years; range, 14 to 76 years; 32 females and 43 males) were examined according to the above protocol. For further evaluation, the following age groups were defined: ≤ 40 years ($n=30$; mean, 28.2 ± 6.9 years), 41 to 60 years ($n=26$; mean, 49.3 ± 6.1 years), ≥ 60 years ($n=19$; mean, 68.9 ± 5.2 years). All volunteers were examined without ultrasound contrast enhancement. Venous flow velocities and identification rates were recorded. To prevent inaccurate measurements, angle correction was performed only if the insonation angle did not exceed 60° .

Patients

Blood flow velocities in the DCVs were assessed in 8 patients with acute cerebral venous thrombosis: (3 with complete superior sagittal sinus thrombosis, 1 with partial superior sagittal sinus (anterior and middle portion) and transverse sinus thrombosis, 1 with cortical vein thrombosis, 1 with transverse and sigmoid sinus thrombosis, 1 with superior sagittal sinus and bilateral transverse sinus thrombosis, and 1 with partial confluens sinuum thrombosis (mean \pm SD age, 41 ± 15 years; range, 23 to 66 years) by TCCS, digital subtraction angiography, and MRA. Detailed patient characteristics are given in Table 1. The follow-up interval ranged from 33 to 387 days. TCCS examinations were performed every 1 ± 1.4 days ($n=17$) during the first week after admission; further examinations followed between days 9 and 92 after admission (mean, 34 ± 32 days; $n=11$) and during the later course of clinical follow-up (mean, 215 ± 54.3 days; $n=7$). Seven patients were treated with dose-adjusted intravenous heparin so that the initial PTT was at least doubled. One patient received low-dose heparin treatment.

All patients were followed-up by contrast-enhanced TCCS (ce-TCCS) in order to optimize the signal-to-noise ratio for identifying recanalization or persistent thrombosis of the venous vessels. Levovist (Schering AG) was used for contrast enhancement in all patients;

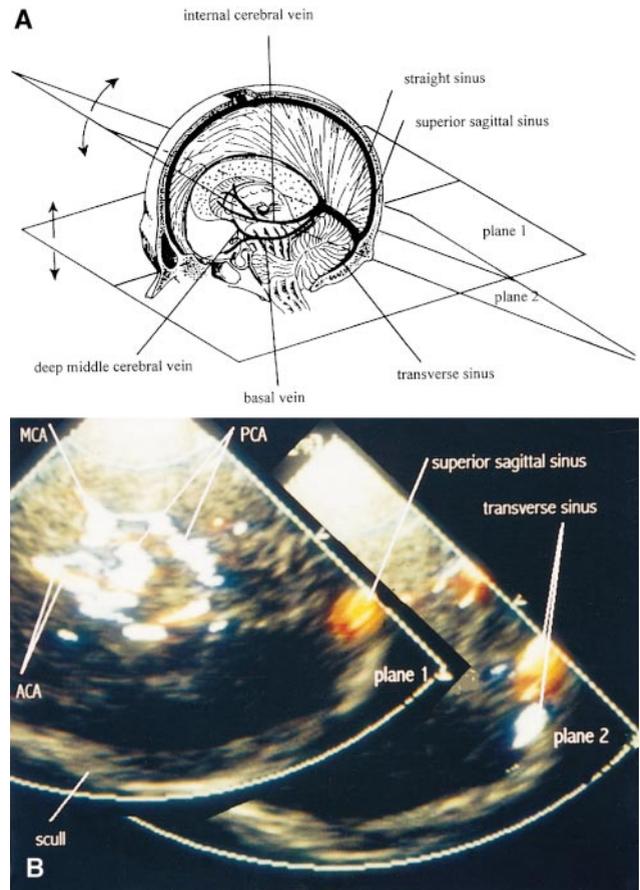


Figure 1. A, Schematic drawing of the topographical anatomy of the DCVs and sinuses that can be insonated reliably through a transtemporal bone window. Plane 1 represents the insonation plane of the cerebral veins and the superior sagittal sinus; plane 2 shows the insonation plane for the straight and the transverse sinuses, respectively. The arrows indicate movement of the transducer during the different stages of the examination. B, Transtemporal imaging of the ipsilateral and contralateral transverse sinuses and of the superior sagittal sinus after contrast enhancement. Plane 1 shows the dorsal part of the superior sagittal sinus; plane 2 marks the projection plane for examination of the transverse sinuses. The ipsilateral transverse sinus is coded red, the contralateral blue. Planes 1 and 2 correspond to the planes in panel A.

intravenous injections of 10 mL of the agent were given at concentrations of 300 to 400 mg/mL. Venous MRA served as a noninvasive morphological reference technique. During the first 6 weeks of follow-up, ce-TCCS data could be compared with 14 venous MRAs. In the time interval 6 weeks to 6 months after admission, 6 venous MRAs and ce-TCCS examinations could be correlated. Two reference examinations were obtained >6 months after admission.

At follow-up the examiner was blind to the venous MRA findings obtained between 0 and 2 days after the contrast-enhanced TCCS examination. The study was carried out in accordance with institutional ethical guidelines.

Statistical Analysis

For data analysis, the software package Turbo Statistik 3.0 was used. The normal range of PSV and EDV was defined as the mean ± 2 SDs. Side differences in peak-systolic, end-diastolic, or mean flow velocities of the paired venous structures of the deep cerebral venous system of $\geq 50\%$ were considered pathological. For comparison of flow velocities between different age and sex groups, nonparametric ANOVA (Mann-Whitney U test) was used; for evaluation of side

TABLE 1. Patient Characteristics

	Pt 1	Pt 2	Pt 3	Pt 4	Pt 5	Pt 6	Pt 7	Pt 8
Age, y	36	38	23	54	27	50	66	33
Sex	F	F	F	F	F	F	M	F
Duration of symptoms, d	8	1	18	14	1	10	1	36
Symptoms at admission	L HP, headaches	L HP, seizure	Global APH, seizures, headaches	Headaches	R HP, global APH, hemianopia, headaches	Headaches	Global APH, hemianopia, seizures, headaches	R HP, sensory APH
Diagnostic method	MRA	MRA	MRA	MRA	DSA	MRA	MRA, DSA	MRA
Thrombosed venous vessel	SSS	SSS	SSS	SSS, L TS	CV	CS	L TS, L SIS	SSS, bi TS
Hemorrhagic infarct	R parietal	R parietooccipital	L parietotemporal	None	L Occipital	None	L parietooccipital	L temporal
Presumed cause	OC	IV corticosteroids	OC	Neck dissection	Postpartum	OC excicciosis		OC
Hypercoaguable state	No	No	No	No	No	No	Heterozygote F V Leiden mutation	No
Time of follow-up, d	132	124	205	134	173	387	33	84
Symptoms at last follow-up	Normal	Normal	Normal	Normal	Normal	Normal	Slight aphasia	Slight R HP

Pt indicates patient; L, left-sided; R, right-sided; bi, bilateral; HP, hemiparesis; APH, aphasia; SSS, superior sagittal sinus; TS, transverse sinus; CV, cortical veins; CS, confluens sinuum; SIS, sigmoid sinus; OC, oral contraceptives.

differences, a Wilcoxon matched-pairs test was used. To compare identification rates of venous vessels, we used Fisher's exact test. For correlation of venous and arterial blood flow velocities, we used a linear regression model. As quality control of the follow-up examinations, the PSVs in the MCA were used to assess the intraobserver repeatability over the observation period.

Results

Normal Values

Details of normal values of flow velocities are given in Table 2. Statistically significant side differences of flow velocities in the paired venous structures could not be detected (paired U test, $P>0.05$). For this reason, a side difference of flow velocities in the DCVs of $>50\%$ was arbitrarily defined as pathological. The rate of visualization of the DCVs was highest for the BV and for

the VG (both at 89%) and was lowest for the dMCV at 76%. The sinuses (the transverse sinus, straight sinus, and the dorsal part of the superior sagittal sinus) could also be detected with variable success rates: 71%, 67%, and 52%, respectively. Overall, the identification rate was higher for cerebral veins than for the dural sinuses (Fisher's exact test, $P<0.01$). The identification rate of the venous vessels was high in the age group ≤ 40 years (dMCV, 87%; BV, 90%; VG, 90%; straight sinus, 77%; transverse sinus, 82%; and superior sagittal sinus, 63%) but tended to drop with increasing age. This finding reached the level of significance for the cerebral veins when comparing the age group ≤ 40 years versus >60 years (Fisher's exact test, $P<0.05$) and was more pronounced for the dural sinuses (age ≤ 40 years versus 41 to 60 years, $P<0.05$; age group 41 to 60 years versus >60 years,

TABLE 2. Normal Values of Venous Flow Velocities

	Flow Velocities, cm/s		n	Angle Correction, Degree	Insonation Depth, cm	Success Rate, %
	Peak Systolic	End Diastolic				
Deep middle cerebral vein	10.3±3.9	7.1±2.6	114	28.4±15.4	5.2±0.5	76
	8.5±2.9	5.7±1.9	114	0		
Basal vein	13.9±5.1	10.0±3.6	133	22.3±9.5	6.3±0.4	89
	12.4±4.0	8.9±3.0	133	0		
Great cerebral vein of Galen	19.4±11.3	13.6±7.4	25*	53.8±5.9*	8.1±0.4	89
	10.6±3.7	7.5±2.8	67	0		
Straight sinus	20.9±9.6	15.6±8.0	39*	47.4±7.2*	9.3±0.4	71
	13.1±5.1	9.4±4.0	53	0		
Transverse sinus	19.3±10.8	13.6±8.0	101	27.7±21.8	11.6±1.0	67
	14.9±6.7	10.4±5.3	101	0		
Superior sagittal sinus	10.6±3.6	6.7±2.6	39	0	10.9±0.9	52

Values are mean±SD.

Only measurements with an insonation angle of $\leq 60^\circ$ were included.

$P < 0.05$). In the age group >40 years, the number of identified vessels was higher in men than in women, without statistical significance. Venous flow velocities decreased with age. This finding was significant ($P < 0.05$) for the age group >60 years compared with those ≤ 40 years for the BV, transverse sinus, and superior sagittal sinus. Women tended to have higher flow velocities than men, which reached the level of significance for the dMCV and BV (for the dMCV, a mean PSV of 11.5 versus 9.4 cm/s; for the BV, a mean PSV of 14.7 versus 13.3 cm/s, respectively; $P < 0.05$).

In the same collective, the rate of identification of the different segments of the basal cerebral arteries was highest for the M1 segment of the MCA at 87%, declined for the P1 segment of the PCA at 85% and the P2 segment of the PCA at 79%, and was lowest for the A1 segment of the anterior cerebral artery (ACA) at 78%. The arterial identification rate tended to drop with increasing age. This finding was significant in comparison of the age groups ≤ 40 years and 41 to 60 years ($P < 0.05$). This decline of identification rate was more pronounced for the cerebral veins than for the arteries ($P < 0.05$). The overall identification rate of the M1 segment of the MCA was significantly higher than that of the dMCV ($P < 0.05$); however, the BV was identified more frequently than the P2 segment of the PCA ($P < 0.05$), comparing those arterial and venous vessels that lie in close proximity. Arterial identification rates were higher in men than in women ($P < 0.05$).

PSV and EDV for the different arterial segments of the circle of Willis were as follows: M1 MCA, 110.6 ± 33.1 and 47.2 ± 14.8 cm/s; A1 ACA, 85.6 ± 22.1 and 38.0 ± 10.8 cm/s; P1 PCA, 67.3 ± 20.5 and 29.0 ± 9.1 cm/s; and P2 PCA, 63.9 ± 14.5 and 28.8 ± 8.6 cm/s, respectively. Arterial flow velocities decreased with age. This finding was significant ($P < 0.05$) for the MCA and the PCA. Women tended to have higher flow velocities than men, which reached the level of significance for the MCA and PCA ($P < 0.05$).

A paired comparison of venous and arterial flow velocities showed only weak correlations. Overall correlation was better for the EDVs than for the PSVs. A linear regression analysis of flow velocities in the MCA related to flow velocities in the dMCV showed no correlation (PSV, $r = 0.06$, $P = 0.53$; EDV, $r = 0.12$, $P = 0.22$). However, EDVs in the PCA and the BV correlated significantly (P1 PCA, $r = 0.22$, $P < 0.05$; P2 PCA, $r = 0.22$, $P < 0.05$). Best correlation was reached for the flow velocities in the PCA and transverse sinus (for the P2 PCA: PSV, $r = 0.27$, $P < 0.01$; EDV, $r = 0.36$, $P < 0.001$; for the transverse sinus PCA: PSV, $r = 0.26$, $P < 0.05$; EDV, $r = 0.51$, $P < 0.001$).

Venous Thrombosis

Four patients (1 with superior sagittal sinus and unilateral transverse sinus thrombosis, 1 with transverse and sigmoid sinus thrombosis, 1 with superior sagittal and bilateral transverse sinus thrombosis, and 1 with cortical vein thrombosis) displayed pathologically increased flow velocities in the DCVs ipsilateral to the side of a hemorrhagic infarct (Figure 2) that normalized within 3, 8, 13, and 252 days, respectively, resulting in a reduction of flow velocities of 46% (left dMCV in patient 5), 52% (left BV in patient 7), 57% (left dMCV in patient 8), and 82% (left dMCV in patient 4) (the numbering of patients follows that in Table 1). In these patients, despite

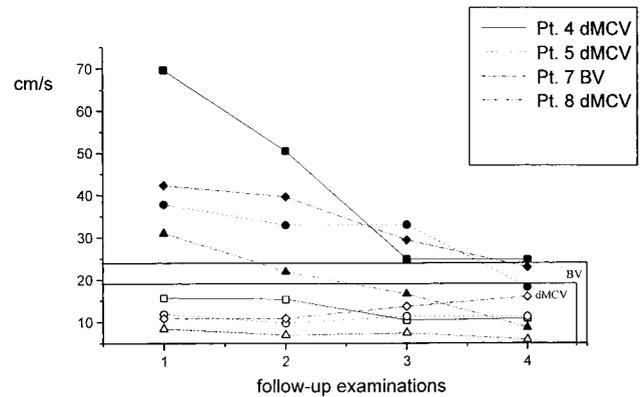


Figure 2. Time course of systolic flow velocities in the DCVs in patients with pathologically increased flow velocities on admission. The solid symbols represent the veins ipsilateral to the side of thrombosis or a venous infarct, the hollow symbols the nonaffected side. The rectangular areas give the normal range of systolic flow velocities, defined as the mean ± 2 SDs for the BV and dMCV. The x axis gives selected time points during follow-up: patient 4, days 1, 3, 12, and 134 after admission; patient 5, days 1, 3, 17, and 172 after admission; patient 7, days 1, 2, 9, and 32 after admission; and patient 8, days 1, 2, 6, and 26 after admission. The numbering of patients follows that in Table 1.

the fact that absolute flow velocity values were normal on follow-up, pathological side differences in flow velocities within the dMCV or BV persisted that resolved in 2 of the patients within 195 and 266 days, respectively. In 1 patient with superior sagittal sinus thrombosis, a significant and persistent side difference in flow velocities within the dMCVs could be demonstrated in the acute stage of illness, despite normal absolute flow velocities. All other patients displayed no significantly increased flow velocities in the DCVs or significant side differences, although there was a tendency for the flow velocities in the dMCV and BV to decrease with time: a reduction of flow velocities of 22.6% (BV in patient 6), 24% (dMCV in patient 1), 35% (dMCV in patient 3), and 59% (dMCV in patient 2) was found. In all patients we found a gradual decrease of flow velocities over time in the great VG, although even in the acute stage of illness PSVs and EDVs lay within the normal range, as defined by the mean ± 2 SDs rule (PSVs of 24.0, 20.1, 19.8, 18.4, 16.8, 13.2, 12.0, and 10.8 cm/s, without angle correction).

In patients with transverse sinus thrombosis and partial confluens sinuum thrombosis, angle-corrected PSVs in the straight sinus gradually decreased during the observation period. In the acute stage of illness in 3 of these patients, flow velocities still lay within the normal range on admission; in 1 case they were pathologically increased (48 cm/s, insonation angle $< 50^\circ$). In this case, flow velocities normalized within 3 days after admission, concurrent with the reappearance of flow signals in the initially occluded transverse sinus. One of the patients with unilateral transverse sinus thrombosis displayed pathologically increased PSVs (51 cm/s on admission) and a strong color signal in the contralateral sinus as sign of compensatory blood flow, which normalized within 3 days after admission. At this time, a flow in the initially occluded vessel was detectable. One day after admission the patient with bilateral transverse sinus thrombosis showed a stringlike color signal in 1 transverse sinus with PSVs of 68 cm/s, interpreted as venous stenosis in a partially

recanalized transverse sinus. Flow velocities normalized within 6 days, concurrent with a partial recanalization confirmed by venous MRA. For all patients, flow velocities within the arteries of the circle of Willis remained normal. During the follow-up period, flow velocities in the MCA showed a fluctuation of 0.09 ± 12.2 cm/s.

On follow-up with contrast-enhanced TCCS (ce-TCCS), recanalization of the affected posterior fossa sinus could be diagnosed correctly in 4 of the 8 patients (Figure 1B). At least partial recanalization could be confirmed by venous MRA within the first 6 weeks after admission in these patients. The patient with cortical vein thrombosis displayed normal posterior fossa sinuses. The cortical venous system was not accessible by contrast-enhanced TCCD. In the patient with transverse and sigmoid sinus thrombosis, persistent occlusion was diagnosed that could be confirmed by MRA. In the patient with partial superior sagittal sinus thrombosis, the posterior part of the sinus was diagnosed correctly as nonoccluded on admission; however, the occlusion of the middle portion of the sinus was not recognized. One high-grade residual stenosis of the superior sagittal sinus thrombosis recognized by MRA up to 10 months after admission was mistaken for persistent occlusion on contrast-enhanced TCCS examinations.

Discussion

Normal Values

In contrast to the advances that TCCS has made in evaluation of the intracranial arterial system, the cerebral venous system has been the subject of systematic evaluation in only 5 studies.⁵⁻⁹ Becker et al⁵ reported the successful insonation with TCCS of the straight sinus in 73% and of the confluens sinuum in 17% of 30 healthy adults. In a frequency- and power-based TCCS study by Baumgartner et al,⁶ the dMCVs could be identified in 88% and the basal veins in 97% of individuals aged between 20 and 59 years. The straight sinus and transverse sinus were successfully insonated in 65% and 44% of the cases, respectively. The dorsal part of the superior sagittal sinus was frequently seen but not evaluated quantitatively because of insonation angles $>60^\circ$.

In our series the dorsal part of the superior sagittal sinus was detected at high success rates, but reliable angle-corrected measurements could not be made because of the perpendicular insonation angles, similar to those encountered in the study by Baumgartner et al.⁶ Our results from healthy volunteers agree well with values reported in the literature^{5,6,9} and demonstrate that the DCVs and posterior fossa sinuses are detectable by transtemporal TCCS with success rates ranging between 50% and 90%. Angle-corrected flow velocity measurements of the VG, the straight sinus, and the superior sagittal sinus are not likely to improve the accuracy of measurement, owing to the unfavorable insonation angles. Differences in detection rate may be partly explained on the one hand by the age dependence of the identification rate and on the other by the slightly different examination protocol. The decrease of the identification rate of cerebral veins and, more pronounced, of the dural sinuses imposes no major drawback for the clinical application of the method, because patients with cerebral venous thrombosis are usually of younger age. The reliable accessibility of the deep

middle cerebral and basal veins predisposes them for follow-up studies in pathological states.

Paired venous and arterial correlations have not been reported thus far. The arterial blood flow velocities in the circle of Willis of our normal collective are in good accordance with the values reported in the literature.^{10,11} We noted a decline in the identification rate of the intracranial vessels that was, overall, more pronounced for the cerebral veins than the arteries. When we compared the identification rates of those arterial and venous vessels in close anatomical proximity, the rates were higher for the MCA than the dMCV because of the frequent problem of separating the venous from the arterial Doppler spectrum. However, identification rates were higher for the BV than for the P2 segment of the PCA. The reason for this may be the slightly curved and superiorly concave¹² course of the P2 segment, which makes it difficult to align the insonation plane with the course of the artery. Parallel to the venous side we found a reduction of flow velocities with increasing age and found higher flow velocities in men than in women. Linear regression analysis of paired venous and arterial flow velocities showed overall a poor correlation. The level of significance was reached with comparison of the EDVs in the P2 segment of the PCA and the BV, and PSVs and EDVs in the P1 and P2 segments of the PCA and the transverse sinus. Correlation was better for the EDVs than the PSVs, since the former are more closely related to the arterial inflow resistance¹³ that influences venous outflow. Regression coefficients, however, were low. This finding is explained by the poor overlap of the territories of arterial supply and venous outflow.

Venous Thrombosis

Conventional TCD has been used for diagnosis in small patient collectives with cerebral venous thrombosis, revealing pathologically increased venous blood flow velocities in the dMCV or the basal cerebral vein in some but not all patients as indirect signs of thrombosis.²⁻⁴ The main disadvantage of conventional TCD in the evaluation of intracranial veins is the need for arterial reference points to identify the venous structures. In 3 patients with superior sagittal sinus thrombosis, TCCS was able to demonstrate increased venous flow velocities in the dMCV, the straight sinus, and the confluens sinuum.^{5,6} A flow reversal in the basal cerebral veins was shown by TCCS in 2 patients with a thrombosis of the straight sinus.⁶

Taking into consideration both our findings and the reports from the literature, the following indirect ultrasonographic signs of cerebral venous thrombosis can be summarized: (1) pathologically increased flow velocities in the DCVs; (2) a pathological side difference of flow velocities in the paired DCVs; and (3) flow reversal in the BVs.

Normalization of elevated flow velocities in the DCVs during follow-up is frequently reported.^{2,3,6} In our patients, significant side differences ($>50\%$) in the DCVs persisted during follow-up if they were present at the initial examination; however, absolute flow velocities returned to the normal range within days to several months. In all but 1 patient (with transverse and sigmoid sinus thrombosis), a recanalization of the affected venous structures could be confirmed by MRA; in 1 case, a high-grade residual stenosis of the dorsal part of the superior sagittal sinus persisted. Monitoring flow veloci-

ties in the straight and transverse sinuses can give additional information on the complex venous hemodynamics in cerebral venous thrombosis and changes caused by recanalization.

It cannot be clearly stated whether there is a causal relationship between clinical status and venous flow velocities, because to date only a few patients have been closely followed-up. The increase of venous flow velocities did not correlate with the severity of our patients' symptoms on admission. Normalization or decrease of flow velocities on follow-up coincided with an improvement of the initial neurological deficits. This, of course, does not prove any causal relationship, but it may indicate either recanalization of the affected venous structures or collateralization.

The occurrence of pathological flow velocities in the DCVs is determined by the venous structure occluded and the anatomy of the venous collaterals, whereas changes in flow velocities in the sinuses accessible by TCCS seem to be more closely related to the thrombotic process itself. We found pathologically increased venous flow velocities only in patients who presented with extensive hemorrhagic infarcts, suggesting significant cortical venous obstruction as the underlying defect that forces venous blood toward the deep cerebral venous system.¹⁵ The latency (which usually cannot be determined exactly) between onset of thrombosis, the neurological symptoms, and the first ultrasound investigation influences the extent of observable flow pathology in the DCVs. Recanalization may occur within the first days after heparinization. Therefore, the timing of the first ultrasound examination in relation to treatment onset is also of importance for the detection of raised venous blood flow velocities. No registrations have been reported that were obtained during propagation of thrombosis or during the phase of clinical deterioration. More information is also needed concerning the temporal relationship between venous hemodynamics and the development of venous hemorrhage.

The repeatability of our follow-up examinations was good, considering the variations in PSVs in the MCA. The observed variations were only slightly higher than in a controlled TCCS validation study.¹⁴

Application of Echo Contrast Agents

As shown in previous studies, using transtemporal TCCS,^{5,7,16} the application of echo contrast-enhancing agents improves the identification of intracranial venous vessels and facilitates the measurement of venous flow velocities. Ries and coworkers⁷ systematically used contrast-enhanced TCCS (ce-TCCS) in 14 patients for the diagnosis of suspected transverse sinus thrombosis. After application of the echo contrast enhancer, an absence of blood flow was found in 1 transverse sinus in 4 cases, in which complete transverse sinus occlusion was confirmed by MRI and MRA in 3 cases and transverse sinus aplasia in 1 case. One thrombotic occlusion was missed on ce-TCCS. Side-to-side asymmetry of blood flow in the transverse sinus was correctly diagnosed in 10 of the 14 patients, 6 of whom displayed only residual color flow signals after contrast enhancement. In these patients, MRI and MRA showed a partial transverse sinus thrombosis in 4 patients due to the demonstration of an intraluminal clot signal, and in 1 case a transverse sinus hypoplasia was revealed.

In our study, contrast enhancement was used for follow-up. The status of the posterior fossa sinuses could be assessed in 7 of the 8

patients regarding recanalization or persistent occlusion, which confirmed the results of venous MRA. However, despite contrast enhancement, neither cortical veins nor the middle and anterior portion of the superior sagittal sinus could be identified. In 1 patient, a high-grade partial residual stenosis of the dorsal part of the superior sagittal sinus was mistakenly identified as occlusion (when taking into consideration the results of MRA). Considering the difficult accessibility of the superior sagittal sinus, contrast enhancement may improve the signal-to-noise ratio but will not improve the unfavorable anatomic localization. In agreement with the results reported by Ries and coworkers,⁷ we found direct ce-TCCS criteria for the diagnosis of cerebral sinus thrombosis in the form of absent color signals. Using these direct ultrasonographic criteria alone, echo contrast enhancement was decisive for the sonographic diagnosis in 7 of our 8 patients.

However, sinus thrombosis cannot be ruled out by either TCCS or ce-TCCS. TCCS is not suitable as a screening method for cerebral venous thrombosis; however, it proved useful as a noninvasive bedside technique for the follow-up of such patients.

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Stroke. 1999;30:70-75

doi: 10.1161/01.STR.30.1.70

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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