Intracranial Venous Hemodynamics Is a Factor Related to a Favorable Outcome in Cerebral Venous Thrombosis

Erwin Stolz, MD; Tibo Gerriets, MD; Rolf H. Bödeker, MD, PhD; Monika Hügens-Penzel, MD; Manfred Kaps, MD

**Background**—In recent studies, coma, cerebral hemorrhage, older age, and infectious origin have been identified as prognostic factors in cerebral venous thrombosis (CVT). However, no studies of the prognosis of CVT have evaluated hemodynamic factors. However, it is conceivable that the presence or absence and the efficiency of venous collaterals, as well as recanalization, may have an impact on brain tissue damage and hence on the prognosis of acute CVT.

**Methods**—Twenty-six patients with acute CVT (mean age, 40±15 years) were recruited prospectively. All patients were treated with intravenous heparin, followed by oral anticoagulation for 12 months, except for 2 patients who were lost to follow-up after hospital discharge. Neurological deficits were graded on the National Institute of Health Stroke Scale on admission, at hospital discharge, and at 90±14 days after admission. The functional clinical outcome was graded on the modified Rankin Scale on day 90 after admission. All patients received a venous transcranial duplex sonography (TCCS) on admission and were followed up in case of a pathological result until normalization was recorded (mean follow-up, 316±395 days; range, 13 to 1180 days).

**Results**—Initial TCCS was pathological in 18 of 26 patients (69%). Four distinct venous drainage types were identified: increased drainage to the cavernous sinus and to the deep cerebral veins, flow reversal in the basal veins, and either compensatory increased or reversed flow in the transverse sinus. Initially normal venous TCCS or normalized TCCS within 90 days was significantly related to favorable outcome.

**Conclusions**—TCCS can be used to evaluate venous drainage patterns in acute CVT. Furthermore, initially normal and normalization of initially pathological venous TCCS within 90 days is related to a favorable outcome in this disease. *(Stroke. 2002;33:1645-1650.)*

**Key Words:** cerebral veins ■ prognosis ■ sinus thrombosis ■ thrombosis ■ ultrasonography, Doppler, color

Cerebral venous thrombosis (CVT) is a disease with a wide spectrum of symptoms and a large range of severity. More and more incidental and asymptomatic cases have been recognized with improved noninvasive imaging techniques. Thus, a debate on an individualized treatment strategy is justified, with current treatment options ranging from local thrombolysis to nothing. Although intravenous heparin treatment in the acute stage of CVT is widely accepted, there currently are no data to give any guidance on the length of oral anticoagulation in the postacute stage of CVT. However, any individualized treatment has to be based on sound data from prognostic factors. This applies for both the acute and postacute stages of the illness; identification of poor prognostic factors may help in the selection of patients for local thrombolytic therapy with an adequate ratio of risk to benefit, and factors for a favorable prognosis may save patients from potentially harmful therapies. Furthermore, predictive factors for long-term outcome may be considered variables when a controlled trial on the length of oral anticoagulation in CVT is planned.

Coma, cerebral hemorrhage, older age, and central nervous system infection have been identified as independent prognostic factors in acute CVT in recent case series. However, no studies on the prognosis of CVT have evaluated hemodynamic factors. However, it is conceivable that the presence or absence and the efficiency of intracranial venous collaterals, as well as early recanalization, may have an impact on the extent of brain tissue damage and hence the prognosis of acute CVT. This study analyzed intracranial venous hemodynamics in relation to the clinical outcome of acute CVT.

**Patients and Methods**

**Patients and Clinical Variables**

Twenty-six patients (mean age, 40±15 years; 18 women, 8 men) with acute CVT diagnosed in all cases by either digital subtraction angiography or MRI and MR angiography (MRA) were prospectively recruited for this study. Table 1 summarizes clinical, ultrasound, and imaging data.

Neurological deficits were graded on the National Institute of Health Stroke Scale (NIHSS) on admission, at hospital discharge, and at 90±14 days after admission. The functional clinical outcome was graded on the modified Rankin Scale on day 90 after admission. All patients received a venous transcranial duplex sonography (TCCS) on admission and were followed up in case of a pathological result until normalization was recorded (mean follow-up, 316±395 days; range, 13 to 1180 days).
All 26 patients underwent venous TCCS on admission. The sonographers were not blinded to the digital subtraction angiography or MRI diagnosis. In all patients, the venous TCCS protocol included the evaluation of the sphenoparietal sinus and superior petrosal sinus draining toward the cavernous sinus, deep cerebral veins (deep middle cerebral vein, basal vein, vein of Galen), and posterior fossa sinuses (straight sinus, transverse sinus, posterior third of the superior sagittal sinus) concerning flow velocities (FVs) and flow direction. Intravenous administration of an echo-contrast–enhancing agent (Levovist 7 mL in a concentration of 300 mg/dL) was necessary to visualize the dural sinuses in 10 patients, including the 2 oldest patients (age, 67 and 78 years).

In case of an initially pathological venous TCCS, the patients were followed up at least until the TCCS findings were normalized. Two patients were lost to follow-up. Mean TCCS follow-up was 316±395 days (range, 13 to 1180 days). During the hospital treatment, follow-up intervals were 4±2 days. For classification of the TCCS results into normal or pathological, previously published normative data on FVs and flow directions have been used.5 For a given vessel, a pathological result was assumed when flow direction was reversed or the FV was increased beyond the mean plus 2 SD.

### TCCS Examination Technique

For ultrasound examinations, a phased-array color-coded ultrasound system (Hewlett Packard, Sonos 5500) equipped with a 2.0-MHz system (Hewlett Packard, Sonos 5500) equipped with a 2.0-MHz system. For intravenous contrast used TCCS, 9 mL of a contrast agent (Levovist 7 mL in a concentration of 300 mg/dL) was used. The contrast agent was administered during the initial TCCS examination. In the case of an initially pathological venous TCCS, the patients were followed up at least until the TCCS findings were normalized. Two patients were lost to follow-up. Mean TCCS follow-up was 316±395 days (range, 13 to 1180 days). During the hospital treatment, follow-up intervals were 4±2 days. For classification of the TCCS results into normal or pathological, previously published normative data on FVs and flow directions have been used.5 For a given vessel, a pathological result was assumed when flow direction was reversed or the FV was increased beyond the mean plus 2 SD.

### Table 1. Demographic, Clinical, Ultrasound, and Imaging Data

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NIH indicates NIHSS score on admission; LOC, level of consciousness (0=fully awake, 1=somnolent, 2=soporuous, 3=comatose); DRT, drainage type; FV, highest or most pathological venous flow velocity (negative flow velocities denote reversal of flow direction); STH, site of thrombosis; VNF, venous infarct on cerebral imaging; mRS 90, NIHSS score on admission; NIH 90, NIHSS score on day 90; TCCS 90, venous TCCS findings on day 90; FU, follow-up; DVD, deep cerebral vein, drainage type; COV, cortical veins; rec, recanalization; TSD, transverse sinus drainage type; SSS, superior sagittal sinus; TS, transverse sinus; SigS, sigmoid sinus; CSD, cavernous sinus drainage type; RBV, reversed flow in the basal veins; SRS, straight sinus; IVC, internal cerebral veins; and CS, confluens sinuum. The last 2 patients were lost to follow-up.
transducer was used. The intracranial venous system was insonated through a temporal acoustic bone window as described earlier.\(^5,6\) In short, after identification of the arteries of the circle of Willis, the pulse repetition frequency was reduced, and the color gain was adjusted to the optimal ratio of signal to noise.

Examination of the deep cerebral veins was performed at an insonation window depth of 10 cm. The deep middle cerebral vein (dMCV) was examined adjacent to the middle cerebral artery. The basal vein of Rosenthal was insonated cranioposteriorly of the posterior cerebral artery in its postpeduncular course. The sphenoparietal sinus was located on the rim of the lesser wing of the sphenoid bone; similarly, the superior petrosal sinus was insonated on the rim of the major sphenoid wing with the high echogenicity of the bony structures serving as landmarks. Then, depth was adjusted so that the contralateral skull became visible. After upward tilt of the probe to the level of the third ventricle, the vein of Galen was found in the midline immediately posterior of the echogenic pineal gland. From this position, the anterior tip of the transducer was rotated upward to align the insonation plane with the plane of the cerebellar tentorium so that the straight sinus could be found in extension to the flow direction of the vein of Galen and the internal occipital protuberance. Above the internal occipital protuberance, a cross section through the superior sagittal sinus was obtained. Downward tilt of the transducer resulted in the visualization of the contralateral transverse sinus. With this protocol, all examined venous structures displayed a flow direction away from the transducer except for the superior sagittal sinus. Angle correction was applied only for measurements in the dMCV and basal vein because angle correction for other venous vessels resulted in unacceptably high confidence intervals for interobserver and intraobserver reliability.\(^6\)

### Data Evaluation

In the first step, we tried to define different venous collateral drainage types using the initial TCCS examination, knowing that TCCS can provide only indirect evidence of venous collateral flow. The association of the different drainage types with the site of venous occlusion and clinical symptoms (disturbed consciousness, hemiparesis or tetraparesis, aphasia, papilledema, initial seizure) was analyzed with a \(\chi^2\) test or Fisher’s exact test when appropriate. Kendall’s rank correlation was used to examine a possible dependence of recanalization on follow-up MRI/MRA examinations and the TCCS results on day 90 in patients with initially pathological ultrasound examinations. The highest or most pathological venous peak systolic FV in each patient on admission (Table 1) was correlated with the presence of the above-mentioned clinical symptoms (Kendall’s nonparametric rank correlation).

Using complete normalization of venous FVs and flow direction as target variables, we analyzed the time course of TCCS normalization using a Kaplan-Meier approach. For comparison of the NIHSS at different time points, a Friedman test was used; a pairwise comparison was carried out with a Wilcoxon test for matched pairs.

In a posthoc analysis, potentially prognostic factors were analyzed. An mRS \(\leq 1\) was defined as the dependent variable. The variables summarized in Table 2 were evaluated with a \(\chi^2\) test or Fisher’s exact test when appropriate. Age, NIHSS on admission, and the individually highest or most pathological initial FV were dichotomized as less than or equal to median or greater than median. In an additional step, the variables in Table 2 were entered into a bivariate nonparametric correlation model with Kendall’s \(\tau\) used to describe a possible relationship with the actual mRS on day 90. Statistical calculations were carried out with SPSS for Windows.

### Results

### Clinical Course

Five patients presented with no measurable deficit on the NIHSS. Reasons for admission in these patients were epileptic seizures (\(n=2\)), persistent headaches (\(n=2\)), and recent development of tinnitus (\(n=1\)). The site of thrombosis, level of consciousness, and mRS on day 90 are summarized in Table 1. During the observation period, the NIHSS improved significantly (\(P<0.001\)) (Figure 1), with the most extensive improvement during the time of inpatient treatment (NIHSS on admission: median, 5; range, 0 to 19) compared with NIHSS at hospital discharge (median, 1; range, 0 to 12; \(P<0.01\); NIHSS at hospital discharge versus NIHSS on day 90: median, 0; range, 0 to 12; \(P<0.01\)). On day 90, excellent functional outcome was reached in 19 patients, with mRS=0 and mRS=1 in 2 patients. Two patients remained severely disabled (mRS=5).

![Figure 1. Time course of the NIHSS in individual patients.](image-url)
Venous TCCS

Taking into account the number of thrombosed straight sinus (n=2), distal segments of the superior sagittal sinus (n=8), and proximal occlusions of the transverse sinus (n=5), the initial identification rate of venous vessels was 100% for the dMVCV (52 of 52), basal vein (52 of 52), and vein of Galen (26 of 26); 77% for the sphenoparietal sinus (40 of 52) and superior petrosal sinus (40 of 52); 83% for the straight sinus (20 of 24); 94% for the transverse sinus (44 of 47); and 89% for the superior sagittal sinus (17 of 19). An initially pathological venous TCCS regarding FVs and flow directions was obtained in 18 of the 26 patients (69%). The individually highest or most pathological venous peak systolic FVs on admission are summarized in Table 1. These FVs significantly correlated with the presence of headaches (P<0.01, τ=0.55) and papilledema (P<0.05, τ=0.37).

Four major drainage types could be differentiated: (1) increased FVs in the sphenoparietal and superior petrosal sinuses as the major contributors to the cavernous sinus (cavernous sinus drainage, 6 of 18 patients, 33%; Figure 2A), (2) increased FVs in the deep cerebral veins (deep cerebral veins drainage, 10 of 18 patients, 56%; Figure 2B), (3) reversal of flow direction in the basal veins (2 of 18 patients, 11%; Figure 2C), and (4) either compensatory increased FVs in the contralateral transverse sinus in case of a proximally located occlusion of the ipsilateral transverse sinus (Figure 2D) or an ipsilaterally reversed flow in the proximal part in case of a distal thrombosis (Figure 2E) (transverse sinus drainage, 10 of 18 patients, 56%).

Headaches were frequently associated with the cavernous and deep cerebral vein drainage types (both P=0.08); disturbed consciousness correlated significantly with the transverse sinus drainage type (P<0.05).

Concerning the site of thrombosis, superior sagittal sinus thrombosis tended to be associated with the deep cerebral veins drainage type (P=0.08), sigmoid sinus thrombosis was significantly associated with the cavernous sinus drainage type (P=0.09), straight sinus occlusion correlated significantly with reversed flow in the basal vein (P<0.05), and transverse sinus thrombosis correlated significantly with the transverse sinus drainage type (P<0.05).

Figure 3 shows the time course of venous TCCS normalization. Fifty percent of patients had a normal venous TCCS within 50 days. In 11 of 16 patients (69%) with complete follow-up, a normal venous TCCS was recorded within 90 days. However, normalization of TCCS has been observed as late as 800 days after diagnosis.

In the cases of initially pathological TCCS, normalization correlated significantly with recanalization on MRI/MRA (P<0.05, τ=0.42).

Prognostic Factors

Complete clinical recovery (mRS=0) was more frequently observed in patients with normalized TCCS within 90 days compared with those with a pathological venous TCCS on day 90 (P=0.04). This finding was even more striking when patients with initially normal TCCS and those with a rapid normalization within 90 days were grouped together (P=0.03). Also, initially normal or normalized TCCS within 90 days was significantly related to an mRS ≤1 (P<0.01). Even when the analysis was restricted to patients with initially pathological TCCS, normalization within 90 days was significantly related to an mRS ≤1.

As already mentioned, potential prognostic factors related to an mRS ≤1 were screened with either a χ² or Fisher’s exact test. These results are summarized in Table 2. In a bivariate nonparametric correlation, initially normal or normalized TCCS within 90 days (τ=-0.54, P<0.001), reversed flow in the basal vein (τ=-0.65, P<0.001), disturbed consciousness (τ=0.60, P<0.01), NIHSS on admission (τ=0.38, P<0.05), headaches on admission (τ=-0.46, P<0.05), recanalization on follow-up MRA (τ=-0.46, P<0.05), venous infarct (τ=0.45, P<0.05), and age (τ=0.52, P<0.01) significantly correlated with the mRS on day 90.

Discussion

In this study, we examined the influence of cerebral venous hemodynamics on clinical outcome in acute CVT. Although TCCS has disadvantages in this respect, the most important being that not all the intracranial venous structures can be examined, it offers unlimited repeatability because of its noninvasiveness and cost effectiveness. Venous TCCS has been validated with extensive sets of normative data showing excellent agreement on normal FVs and flow directions between the different studies. In patients <50 years of age, the identification rates are high even without echo-contrast enhancement. Venous TCCS has a high interobserver and intraobserver repeatability and was therefore best suited for our purposes.

Increased venous FVs in acute CVT have been described in previous reports. Sixty-nine percent of patients in this study displayed pathological venous FVs or directions. This has implications for TCCS as a diagnostic tool; the method may positively confirm CVT in more than two thirds of cases but cannot rule out the condition. Yet, the golden standard of diagnosis remains digital subtraction angiography. Valdueza and coworkers were the first to link venous hemodynamics to clinical parameters in CVT. They reported that the extent of venous FV increase was related to the degree of disturbed consciousness. In our patient cohort, we were not able to observe this relationship but found an association with the presence of headaches and papilledema.

In this study, we tried to systematize the indirect signs of collateral venous outflow and were able to define distinct venous drainage patterns in the acute stage of illness. Sonographic drainage patterns are summarized in Figure 2, together with details on the presumed anatomy of the venous collaterals. Information on venous collaterals cannot routinely be obtained by time-of-flight MRA, so TCCS may be used as a complementary examination technique providing functional hemodynamic information on CVT. Furthermore, TCCS merits the advantage of bedside capability and virtually unrestricted repeatability. In this study, drainage pattern correlated with the presence of headaches and disturbed consciousness. Despite controversies with the results of Valdueza and coworkers, our data show that the extent of venous flow disturbance and drainage type are related to the initial clinical picture in acute CVT.
Figure 2. Venous drainage patterns defined by TCCS and presumed collateral pathways. A, Cavernous sinus drainage type: 1 indicates superior sagittal sinus; 2, straight sinus; 3, vein of Galen; 4, thrombosed superior sagittal sinus; 5, superior anastomotic vein; 6, cavernous sinus; 7, superficial middle cerebral vein; 8, sphenoparietal sinus; 9, superior petrosal sinus; 10, dMCV; 11, inferior anastomotic vein; 12, basal vein; and 13, transverse sinus. Presumed venous collaterals are alternative runoffs for the central and postcentral cortical veins via the superior anastomotic vein to the superficial middle cerebral vein and the sphenoparietal sinus toward the cavernous sinus and for the posterior group of the cortical veins via the inferior anastomotic vein and the superior petrosal sinus to the cavernous sinus. B, Deep cerebral venous drainage type. Presumed venous collaterals are transcortical anastomoses connecting basal ganglia veins with the internal cerebral veins and anastomotic connections of the superficial and deep middle cerebral veins in the sylvian fissure. C, Retrograde perfusion of the basal veins. These provide an alternative runoff in straight sinus occlusion. D, Compensatory flow increase in the transverse sinus when the thrombosis is located in the proximal contralateral transverse sinus. E, Retrograde perfusion of the proximal transverse sinus when the thrombosis is located in the distal ipsilateral transverse sinus. Venous structures in which pathological FVs or directions on TCCS were detected are shown in black; presumed collateral vessels, dark gray; and site of thrombosis, dotted white. Structures in light gray were found normal on TCCS. See elsewhere14,15 for venous collateral pathways.
The clinical outcome in this study was excellent despite a frequency of venous infarcts of 50%. Only 2 of our patients remained dependent. This observation is in line with data from a recently published study on outcome in acute CVT. However, these data show that despite a high frequency of venous infarcts, an mRS of 2, which would be regarded as an acceptable outcome in arterial ischemic stroke, has to be regarded as a poor outcome in CVT, considering that 79% of our patients had full restitution on day 90. This finding also may affect the design of future therapeutic studies.

In bivariate analysis, age, initial NIHSS, disturbed consciousness, and the presence of venous infarcts were found to positively correlate with the mRS on day 90 in accordance with recent studies evaluating prognostic factors in acute CVT. Furthermore, we found headaches, which themselves were related to the extent of venous flow disturbance and the cavernous and deep cerebral veins drainage type, correlated with the mRS on day 90. A numerically stronger correlation existed for initially normal or normalized TCCS within 90 days and a reversed flow direction in the basal veins, which we found only in straight sinus occlusion, with the mRS on day 90. These findings highlight that venous hemodynamics may affect the design of future therapeutic studies.

In this study, we observed a correlation between normalized TCCS on day 90 and MRA recanalization after 6±2 months. On the other hand, normalized TCCS may also reflect functionally sufficient venous collateral pathways. Although not all possible venous collateral pathways can be surveyed with TCCS, initially normal venous TCCS results may be associated with thromboses without widespread collaterals and hence less impedance of venous outflow. It is less likely that TCCS is not able to pick up potential collaterals because venous FVs in CVT are usually high and can easily be recorded by TCCS. In respect to hemodynamics, the situation is comparable to the arterial circulation in which competent collaterals via the ophthalmic artery and the circle of Willis can prevent or attenuate brain tissue damage.

TCCS may serve as a noninvasive, cost-effective examination technique in the follow-up of patients with CVT that provides informations on venous hemodynamics and collateral pathways and is related to outcome. TCCS can serve as a technique complementary to MRI.

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Intracranial Venous Hemodynamics Is a Factor Related to a Favorable Outcome in Cerebral Venous Thrombosis
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