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Original Contribution

Impaired Dynamic Cerebral Autoregulation in Patients With Cerebral Venous Sinus Thrombosis: Evaluation Using Transcranial Doppler and Silent Reading Stimulation

Songwei Chen [a,b,c], Hongxiu Chen [a,b,c], Jiangang Duan [d], Liuping Cui [a,b,c], Ran Liu [a,b,c], Yingqi Xing [a,b,c,*]

[a] Department of Vascular Ultrasonography, Xuanwu Hospital, Capital Medical University, Beijing, China
[b] Beijing Diagnostic Center of Vascular Ultrasound, Beijing, China
[c] Center of Vascular Ultrasonography, Beijing Institute of Brain Disorders, Collaborative Innovation Center for Brain Disorders, Capital Medical University, Beijing, China
[d] Department of Emergency, Xuanwu Hospital, Capital Medical University, Beijing, China

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Objective: Cerebral venous sinus thrombosis (CVST) may impair dynamic cerebral autoregulation (dCA) of the middle cerebral artery (MCA). However, most studies have focused on dCA of the MCA; a few studies are based on the posterior cerebral artery (PCA) during silent reading and neurovascular coupling (NVC). This study explored the effects of CVST on dCA of the MCA and PCA during silent reading and NVC.
Methods: From January 2021 to August 2022, 60 CVST patients and 30 controls were enrolled in this study. Non-invasive continuous beat-to-beat blood pressure, cerebral blood flow velocity and other associated information on the MCA and PCA during silent reading were collected using a transcranial Doppler. NVC assessment was performed by opening and closing the eyes periodically based on voice prompts, and eye-opening visual stimulation was achieved by silently reading Chinese tourism materials. Visual stimulation signals can selectively activate Brodmann's areas 17, 18, and 19 of the occipital when reading silently with open eyes, prompting them to release neurotransmitters and dilate PCA. dCA was determined by transfer function analysis.
Results: In dCA of the PCA during silent reading, the CVST group's very low frequency phase was lower than that of the control group ($p = 0.047$). In NVC, the difference in the indexes of the cerebrovascular conductance and visually evoked flow response of the CVST group were lower than those of the control group ($p = 0.017$ and $p = 0.019$, respectively).
Conclusion: Compared with the control group, dCA and NVC of the PCA during silent reading were impaired in CVST patients.

Introduction

Cerebral venous sinus thrombosis (CVST) is a unique cerebrovascular disease that most frequently occurs in young people and children and can present with various clinical signs and symptoms. The most common symptom is headache, which is reported in 90% of cases [1]. When arterial blood pressure (BP) varies, the ability of the brain to sustain relatively invariable cerebral blood flow (CBF) is known as dynamic cerebral autoregulation (dCA) [2,3]. The mechanism underlying dCA includes mainly myogenic, neurogenic, endothelial and metabolic reactivity theories [4], among which the myogenic theory is the most typical [5]. Neurogenic mechanisms, also known as neurovascular coupling (NVC) [6], refer to the role of neuronal activity changes in regulating the contraction and relaxation of blood vessels via the secretion of vasoactive neurotransmitters, thereby regulating local blood flow [7]. Cytoarchitectonically, the cerebral cortex has been classified into 52 cortical Brodmann areas (BAs) [8]. When reading silently with open eyes, visual cortex BAs 17, 18 and 19 in the occipital lobe can be selectively stimulated to release corresponding neurotransmitters and dilate the PCA [7–9].

Currently, researchers have developed various methods for evaluating dCA, such as correlation coefficient analysis, the autoregulatory index and transfer function analysis (TFA) [3]. According to the white paper of the International Cerebral Autoregulation Research Network (CARNet), TFA can analyze the real-time changes between BP and CBF and is a commonly used method for evaluating dCA [3]. Chen et al. [10] studied 23 patients with CVST and found that the middle cerebral artery (MCA) in the CVST group had a lower phase than the control group

* Corresponding author. Department of Vascular Ultrasonography, Xuanwu Hospital, Center of Vascular Ultrasonography, Beijing Institute of Brain Disorders, No. 45 Changchun Road, Beijing 100053, China.
E-mail address: xingyq2009@sina.com (Y. Xing).
Songwei Chen and Hongxiu Chen contributed equally to this work.

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($p = 0.03$), indicating that patients with CVST may have impaired dCA. Previous studies have reported that the obstruction of cerebral venous blood flow return and the reduction of cerebrospinal fluid absorption in patients with CVST can lead to increased intracranial pressure, cerebral venous hemorrhage or cerebral infarction; can damage cerebral arterioles and capillaries; and can affect dCA through myogenic, neurogenic and other mechanisms [10,11].

Currently, most studies on dCA in patients with CVST focus only on the MCA; a few studies have evaluated the posterior cerebral artery (PCA) during silent reading and NVC [12]. Therefore, this study was aimed at exploring the effects of CVST on dCA of the MCA, PCA during silent reading and NVC while considering the deficiencies of previous studies.

Methods

Study design and participants

Patients clinically diagnosed with CVST at Xuanwu Hospital of Capital Medical University were consecutively enrolled between January 2021 and August 2022. The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Xuanwu Hospital of Capital Medical University (LYS[2022]137). Informed consent was obtained from all participants involved in the study.

Two radiologists diagnosed each patient using magnetic resonance venography and magnetic resonance black-blood imaging [1,13,14]. On the basis of their magnetic resonance imaging staging, 60 patients with CVST were grouped into the chronic (n = 43) or non-chronic (n = 17) phase. We calculated the CVST score based on the distribution of venous sinus thrombosis on magnetic resonance imaging. Except for the superior sagittal sinus thrombosis, which was 3 points, the other venous sinus thrombosis was 1 point, and the total score for each patient was calculated [15]. Those with a total score >2 points were included in the "large range group" (n = 45), while the rest were included in the "small range group" (n = 15). The exclusion criteria were (i) inability to cooperate with the transcranial Doppler (TCD) examination because of restlessness; (ii) poor penetration or closure of the temporal window; (iii) atrial fibrillation or other heart valve diseases; and (iv) carotid stenosis [16], diabetes [17], stroke [18,19] and other diseases affecting dCA. Thirty volunteers of the same age and sex without cerebrovascular disease were recruited from the hospital as the control group. All participants underwent carotid ultrasonography and transcranial color-coded ultrasound to rule out carotid and intracranial artery stenosis.

We collected data on all participants' age, sex, heart rate, BP and end-tidal carbon dioxide ($ET\text{-}CO_2$) and the following information from the CVST group: (i) demographic data, such as hyperlipidemia, smoking and body mass index; (ii) relevant clinical symptoms, signs, risk factors [13,14,20] and the location of thrombosis; (iii) coagulation function indicators at admission, ìncluding activated partial thromboplastin time, thrombin time, prothrombin time, d-dimer and fibrinogen; and (iv) imaging data at admission, such as magnetic resonance venography and black-blood imaging.

dCA and NVC measurement

Functional TCD can measure the cerebral blood flow velocity (CBFV) of the PCA induced by visual stimulus signals non-invasively and continuously, which is a common method for evaluating NVC [21]. TCD (EMS-9D Pro; Delica Medical, Shenzhen, China) was used uniformly to examine the bilateral MCA and PCA with a 1.6 MHz probe. According to the suggestions in "Transfer Function Analysis of Dynamic Cerebral Autoregulation: A White Paper From the International Cerebral Autoregulation Research Network", dCA was evaluated by vascular ultrasound specialists [3]. The internal temperature was 22°C–24°C. Patients and volunteers were instructed to avoid alcohol, caffeinated drinks and exercise for at least 6 h before the examination. Before the Omron sphygmomanometer was used to measure the baseline BP of the brachial artery in the supine position, participants were instructed to rest for 15 min. Subsequently, they were fitted with a monitor head frame, the probe was fixed in the position of the temporal window and the TCD machine was adjusted to a dual-channel single-depth mode to monitor CBFV at depths of 50–65 and 60–70 mm in the bilateral MCA and PCA, respectively. Non-invasive continuous beat-to-beat BP (NIBP), including finger artery BP, was recorded synchronously and corrected with the measured value of brachial artery BP; the nasal catheter was used to monitor the $ET\text{-}CO_2$ continuously; and precordial electrocardiography leads were synchronously monitored. CBFV, NIBP, electrocardiography and $ET\text{-}CO_2$ were recorded in real time during the entire time the patient was in the supine position (MCA for 10 min and PCA for 10 min). During PCA silent reading and NVC monitoring, participants were asked to periodically open and close their eyes based on voice prompts. They were first instructed to close their eyes for 2 min and then performed six cycles of 24 s with eyes open (visual stimulation: silent reading) followed by 24 s with eyes closed. The reading material was a Chinese introduction to tourist attractions, such as the Forbidden City, the Great Wall and the Summer Palace. The NVC protocol was performed in accordance with published guidelines [7]. The BP and mean CBFV (V_m) of each cycle were collected during rest and silent reading [7], and the following values were calculated: index of cerebrovascular conductance, $CVCi = V_m/\text{mean arterial pressure (MAP)}$ [22]; $\Delta CVCi = CVCi_{\text{silent reading}} - CVCi_{\text{rest}}$; and visually evoked flow response, $VEFR = (V_{m,\text{silent reading}} - V_{m,\text{rest}})*100/V_{m,\text{rest}}$ (%) [23].

dCA and NVC analysis

Transfer function analysis can be used to quantitatively evaluate the dynamic relationship between the input variable (BP) and output variable (CBF) and further analyze dCA parameters (*i.e.,* gain, phase and coherence) [3]. The white paper published by CARNet members in 2016 recommends the parameters and settings of TFA to improve and standardize the TFA evaluation method [3]. The latest white paper published by CARNet members in 2022 has further updated and enhanced the parameters and settings of TFA [12]. Therefore, this study applied TFA to analyze dCA data. On the basis of the TFA method, NIBP and stable TCD raw monitoring data of 5 min were selected from the monitoring data of 10 min each for MCA and PCA for analysis. The length of the Hanning window is 100 s, and the superposition is 50% [8,24]. We calculated the phase, coherence, absolute gain (cm/s/mm Hg) and normalized gain (%/mm Hg) of very low frequency (VLF) (0.02–0.07 Hz), low frequency (LF) (0.07–0.20 Hz) and high frequency (HF) (0.20–0.50 Hz). Further analysis was conducted using the average values of dCA parameters in both brain hemispheres. The gain represented the amplitude relationship between the input signals (BP) and the output signals (CBFV), whereas the phase represented their relationship with time. A higher gain and lower phase indicated that dCA was impaired. Moreover, the reliability of the phase was higher than that of other dCA parameters [12]. A high coherence indicated that BP and CBFV had a linear relationship [3]. When coherence is < 0.5, BP and CBFV may be non-linear [25]. Therefore, this study collected only data with a coherence > 0.5. Additionally, in NVC, ΔCVCi and VEFR indicated the change in PCA velocity (PCA_v) when silent reading was compared with rest. A low value indicated that the NVC was impaired.

Statistical analysis

SPSS 26.0 software (IBM, Armonk, NY, USA) was used for all statistical analyses. Normally distributed quantitative data are expressed as the mean ± standard deviation. The *t*-test was used for comparison; the median and interquartile range were used to express non-normally distributed quantitative data, and the Mann–Whitney *U*-test was used for comparison. Percentages were used to express the qualitative data, and

the χ^2-test was used for comparison. All statistical tests were performed bilaterally. Statistical significance was set at $p < 0.05$.

Results

In this study, 75 patients with CVST were initially recorded; 9 patients for whom dCA of the MCA and/or PCA could not be measured because of the temporal window and 6 with diabetes were excluded. Therefore, 60 patients with CVST and 30 healthy volunteers were finally included in this study. Figure 1 is the participant recruitment flowchart. No statistical differences were found in age, sex, systolic BP and diastolic BP between the CVST and control groups; however, significant differences were observed in heart rate and body mass index. The most prevalent clinical symptom among all patients was headache (86.7%), followed by blurred vision (25.0%), decreased consciousness (23.3%), seizures (18.3%) and focal neurological deficits (8.3%). The most common risk factors were infection (43.3%) and systemic disease (43.3%). Forty-three (71.7%) patients had superior sagittal sinus thrombosis, whereas 55 (91.7%) had transverse or sigmoid sinus thrombosis. Table 1 outlines the clinical characteristics of the participants.

The VLF and LF phases of the CVST group in dCA of the MCA were lower than those of the control group; however, the differences were not statistically significant ($p = 0.701$ and $p = 0.893$, respectively; Table 2, Fig. 2).

In dCA of the PCA during silent reading, the VLF phase of the CVST group was lower than that of the control group ($p = 0.047$; Table 3, Fig. 2). In the NVC, the results of resting PCA_v, resting MAP, silent reading PCA_v and silent reading MAP did not differ statistically significantly between the CVST and control groups. The ΔCVCi and VEFR of the CVST group were lower than those of the control group ($p = 0.017$ and $p = 0.019$, respectively; Table 4, Fig. 2). When reading silently with eyes open, the amplitude of changes in CBFV in the control group was significantly greater than that in the CVST group (Fig. 3). There were no statistical differences ($p > 0.05$) in the VLF phase, ΔCVCi and VEFR of PCA between patients with chronic and those with non-chronic CVST. For the distribution range of CVST, the VEFR of the "large range group" was lower than that of the "small range group", and the difference was statistically significant (23.54% ± 9.24% vs. 30.79% ± 12.06%, $p = 0.018$).

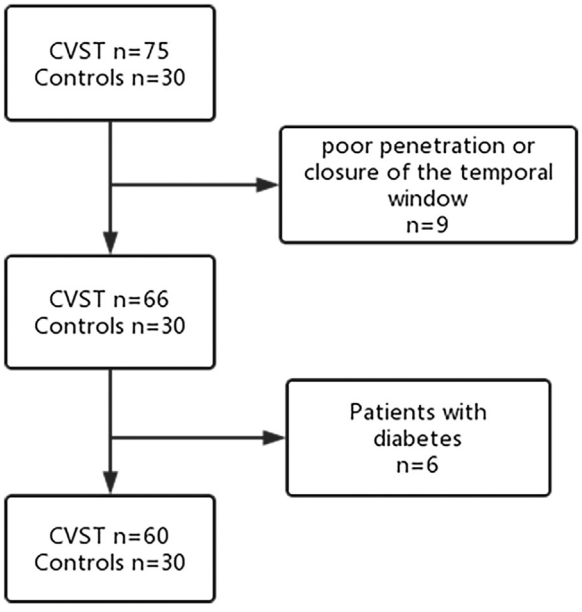

Figure 1. Flowchart of recruitment of participants. CVST, cerebral venous sinus thrombosis.

Table 1
Clinical characteristics of 60 patients with CVST and 30 controls

Characteristic	Patients with CVST (n = 60)	Controls (n = 30)	*p*
Male, n (%)	33 (55.0)	15 (50.0)	0.654
Age, y	31.50 (20.00, 44.25)	34.00 (28.50, 39.25)	0.161
Systolic blood pressure (mm Hg)	119.65 ± 12.90	114.67 ± 13.89	0.096
Diastolic blood pressure (mm Hg)	68.63 ± 10.24	70.03 ± 11.17	0.555
Heart rate (bpm)	79.50 (68.25, 90.75)	65.00 (59.75, 74.25)	<0.001[a]
Body mass index (kg/m^2)	24.50 (21.30, 27.50)	21.88 (20.41, 25.16)	0.027[a]
Smoking, n (%)	15 (25.0)	2 (6.7)	0.036[a]
Hyperlipidemia, n (%)	22 (36.7)	0	<0.001[a]
Lumbar puncture pressure (mm H_2O)	250.00 (170.00, 317.50)		
Prothrombin time (s)	13.45 (12.90, 14.40)		
Activated partial thromboplastin time (s)	37.55 (33.53, 44.43)		
Fibrinogen (g/L)	3.70 (2.95, 4.45)		
Thrombin time (s)	16.40 (15.33, 17.53)		
D-dimer (μg/mL)	0.54 (0.23, 1.47)		
Clinical symptoms, n (%)			
Headache	52 (86.7)		
Focal neurological deficits	5 (8.3)		
Seizures	11 (18.3)		
Decreased consciousness	14 (23.3)		
Blurred vision	15 (25.0)		
Risk factors, n (%)			
History of thrombosis	6 (10.0)		
Sex-specific risk factors	6 (10.0)		
Systemic disorders	26 (43.3)		
Hematologic disorders	23 (38.3)		
Anemia	16 (26.7)		
Infections	26 (43.3)		
Other	11 (18.3)		
Superior sagittal sinus thrombosis, n (%)	43 (71.7)		
Transverse or sigmoid sinus thrombosis, n (%)	55 (91.7)		

Focal neurological deficits included paralysis, aphasia, hemianopia and hypoesthesia; sex-specific risk factors include oral contraceptives, oral hormone substitutes, pregnancy and puerperium; systemic factors included Behcet's disease, antiphospholipid syndrome, ulcerative colitis, systemic lupus erythematosus, thyroid disease and rheumatoid arthritis; infections include mastoiditis, otitis media, sinusitis and syphilis; other factors included head trauma, iatrogenic risk factors, oral or vulval ulcers and dural arteriovenous fistula.
CVST, cerebral venous sinus thrombosis.
[a] $p < 0.05$, compared with normal controls.

Discussion

Analysis of dCA in 60 patients with CVST and 30 healthy volunteers revealed that after adjusting for ET-CO_2, the CVST group had a lower ΔCVCi, VEFR and PCA VLF phase during silent reading than the control group, and the difference was statistically significant, indicating that dCA in patients with CVST was impaired.

In dCA of the MCA, the CVST group had lower VLF and LF phases than the control group; however, the difference was not statistically significant. In patients with CVST, the proportion of transverse sinus or sigmoid sinus thrombosis was significantly higher than that in patients with superior sagittal sinus thrombosis. We consider transverse or sigmoid sinus thrombosis to affect primarily the cerebellum and cerebral occipital lobe blood reflux (PCA-supplied territory), while superior sagittal sinus thrombosis affects mainly cerebral hemisphere blood reflux (MCA-supplied territory). This may explain why dCA is impaired in the PCA during silent reading but normal in the MCA.

Our results indicate that whether it was the PCA during silent reading or the MCA, the CVST group had lower VLF, LF and HF gains than the control group; this is contrary to the expected impairment of dCA in

Table 2
Comparison of dCA parameters of MCA between the CVST and control groups

	Patients with CVST (n = 60)	Controls (n = 30)	*t/z*	*p*
VLF gain (cm/s/mm Hg)	0.63 (0.51, 0.95)	0.71 (0.63, 0.88)	1.18	0.237
LF gain (cm/s/mm Hg)	0.83 (0.67, 1.03)	0.96 (0.81, 1.11)	2.07	0.038
HF gain (cm/s/mm Hg)	0.87 (0.66, 1.09)	0.99 (0.79, 1.19)	1.69	0.092
VLF gain (%/mm Hg)	1.10 (0.96, 1.39)	1.14 (1.00, 1.44)	0.66	0.513
LF gain (%/mm Hg)	1.44 ± 0.41	1.55 ± 0.34	1.24	0.218
HF gain (%/mm Hg)	1.40 (1.20, 1.58)	1.46 (1.33, 1.64)	1.54	0.124
VLF phase (°)	68.84 ± 22.21	70.70 ± 20.43	0.39	0.701
LF phase (°)	43.39 ± 12.18	43.78 ± 13.84	0.14	0.893
HF phase (°)	23.20 ± 13.44	12.40 ± 11.59	3.76	<0.001
ET-CO_2 (mm Hg)	37.88 (36.73, 39.65)	38.08 (36.38, 39.70)	0.56	0.578

CVST, cerebral venous sinus thrombosis; dCA, dynamic cerebral autoregulation; MCA, middle cerebral artery; VLF, very low frequency; LF, low frequency; HF, high frequency; ET-CO_2, end-tidal carbon dioxide.

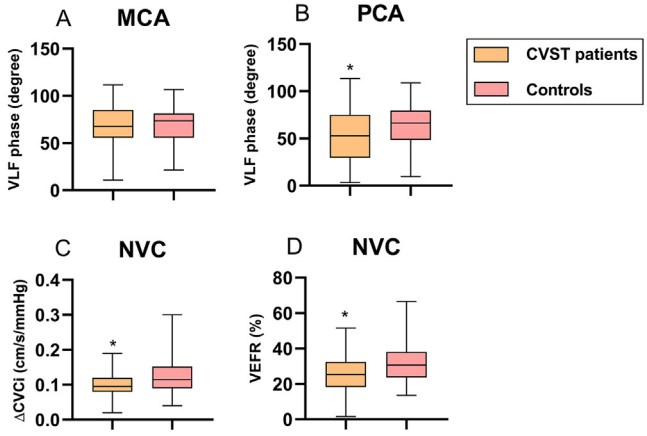

Figure 2. Comparison of the VLF phase and NVC parameters between the CVST and control groups. (A) The CVST group had a lower VLF phase of MCA than the control group; however, the difference was not statistically significant. (B) The VLF phase of the PCA during silent reading in the CVST group was significantly lower than that in the control group. (C, D) The ΔCVCi and VEFR were significantly lower in the CVST group than in the control group. *$p < 0.05$, compared with normal controls. ΔCVCi, difference in the index of cerebrovascular conductance; CVST, cerebral venous sinus thrombosis; NVC, neurovascular coupling; PCA, posterior cerebral artery; VEFR, visually evoked flow response; VLF, very low frequency.

In the NVC, no statistical difference was found between the CVST and control groups with respect to resting PCA_v, silent reading PCA_v, resting MAP or silent reading MAP. However, the CVST group had lower ΔCVCi and VEFR values than the control group, indicating that the CVST group had an impaired NVC response to visual stimulation. A guideline for standardizing the evaluation of human NVC states that NVC can be assessed using various methods, such as reading, flashing screens and eye tracking of the tester's moving hand. Among these, reading silently with eyes open is a relatively simple and feasible method [7]. Therefore, this study adopted this method based on the guideline. PCA can supply blood to the visual cortex in the occipital lobe [21]. When reading silently with eyes open, visual stimulation signals selectively activate neurons in the occipital lobe [7], prompting them to release related neurotransmitters, causing the diameter of PCA to expand and the CBFV of PCA to increase [7]. TCD has become a common approach for evaluating NVC because of its high time resolution and non-invasive nature [7]. Lin et al.'s study [26] on ischemic stroke indicated that compared with the control group, the stroke group's response to visual stimuli significantly decreased, indicating NVC was impaired. Previous research has suggested that hypotension impairs the ability of cerebral vessels to contract or expand, consequently affecting the NVC [7]. Phillips et al. [9] studied the relationship between spinal cord injury and NVC and discovered that when midodrine (a receptor agonist, vasopressor) was not used, the mean CBFV and ΔCVCi of PCA during visual movement were lower in the spinal cord injury group than in the control group. However, when

Table 3
Comparison of dCA parameters of PCA during silent reading between the CVST and control groups

	Patients with CVST (n = 60)	Controls (n = 30)	*t/z*	*p*
VLF gain (cm/s/mm Hg)	0.68 (0.52, 0.92)	0.89 (0.67, 1.27)	3.00	0.003
LF gain (cm/s/mm Hg)	0.67 (0.53, 0.85)	0.84 (0.71, 0.96)	3.05	0.002
HF gain (cm/s/mm Hg)	0.61 (0.52, 0.71)	0.68 (0.61, 0.79)	2.43	0.015
VLF gain (%/mm Hg)	1.68 (1.28, 2.52)	2.24 (1.82, 2.98)	3.15	0.002
LF gain (%/mm Hg)	1.70 (1.45, 2.01)	2.18 (1.82, 2.28)	3.08	0.002
HF gain (%/mm Hg)	1.66 (1.35, 1.97)	1.72 (1.51, 2.25)	1.34	0.179
VLF phase (°)	52.91 (29.56, 75.12)	66.43 (48.68, 79.55)	1.99	0.047[a]
LF phase (°)	39.18 ± 15.47	38.93 ± 19.38	0.07	0.947
HF phase (°)	24.94 ± 15.08	12.13 ± 11.27	4.11	<0.001
ET-CO_2 (mm Hg)	37.49 (36.70, 40.50)	38.06 (37.08, 39.83)	0.48	0.628

CVST, cerebral venous sinus thrombosis; dCA, dynamic cerebral autoregulation; PCA, posterior cerebral artery; VLF, very low frequency; LF, low frequency; HF, high frequency; ET-CO_2, end-tidal carbon dioxide.
[a] $p < 0.05$, compared with normal controls.

patients with CVST. Chen et al. [11] also discovered that patients with idiopathic intracranial hypertension had a lower gain than the control group. However, the relevant mechanisms require further study. In addition, because the phase is more reliable than the gain, we believe that dCA of patients with CVST is impaired.

midodrine was used to increase BP, the mean CBFV in patients with spinal cord injury increased by 70%, and the ΔCVCi of PCA also increased.

The mechanism underlying dCA and NVC impairment in patients remains unclear. Researchers believe that the obstruction of cerebral venous blood flow return and the reduction of cerebrospinal fluid

Table 4
Comparison of NVC parameters between the CVST and control groups

	Patients with CVST (n = 60)	Controls (n = 30)	*t/z*	*p*
Resting PCA_v (cm/s)	33.52 ± 6.30	32.57 ± 7.24	0.65	0.521
Resting MAP (mm Hg)	82.43 ± 9.63	85.96 ± 10.80	1.58	0.119
Silent reading PCA_v (cm/s)	42.32 ± 8.53	43.11 ± 9.40	0.40	0.688
Silent reading MAP (mm Hg)	83.74 ± 9.89	85.10 ± 10.53	0.60	0.547
ΔCVCi (cm/s/mm Hg)	0.09 (0.08, 0.12)	0.12 (0.09, 0.15)	2.40	0.017[a]
VEFR (%)	25.36 (18.28, 32.43)	30.73 (23.67, 38.18)	2.35	0.019[a]
ET-CO_2 (mm Hg)	37.45 (36.58, 40.50)	38.06 (37.08, 39.83)	0.74	0.459

ΔCVCi, index of the cerebrovascular conductance; CVST, cerebral venous sinus thrombosis; ET-CO_2, end-tidal carbon dioxide; MAP, mean arterial pressure; NVC, neurovascular coupling; PCA_v, posterior cerebral artery blood velocity; VEFR, visually evoked flow response.
[a] $p < 0.05$, compared with normal controls.

absorption in patients with CVST can lead to increased intracranial pressure [10]. This can cause cerebral venous hemorrhage or cerebral infarction and damage the function of the brain parenchyma, thereby causing damage to cerebral arterioles and capillaries [10], which may account for the impaired dCA. Additionally, 25.0% of the patients in the CVST group had blurred vision, suggesting that the visual center of patients with CVST is damaged. When receiving visual stimulation, the neurotransmitters released by neurons and the relaxation of blood vessels are reduced, thereby diminishing the effect of visual stimulus signals on the CBFV of the PCA.

This is the first study to simultaneously evaluate dCA and NVC of the MCA and PCA during silent reading in patients with CVST. By altering the diameter of the cerebral arteries, ET-CO_2 can affect dCA and NVC [7]. At normal temperature, every increase or decrease of 1 mm Hg in CO_2 partial pressure can lead to an increase of 3%–6% or a decrease of 1%–3% in CBF, respectively [7]. Consequently, this study measured and corrected ET-CO_2, removing its effect on NVC.

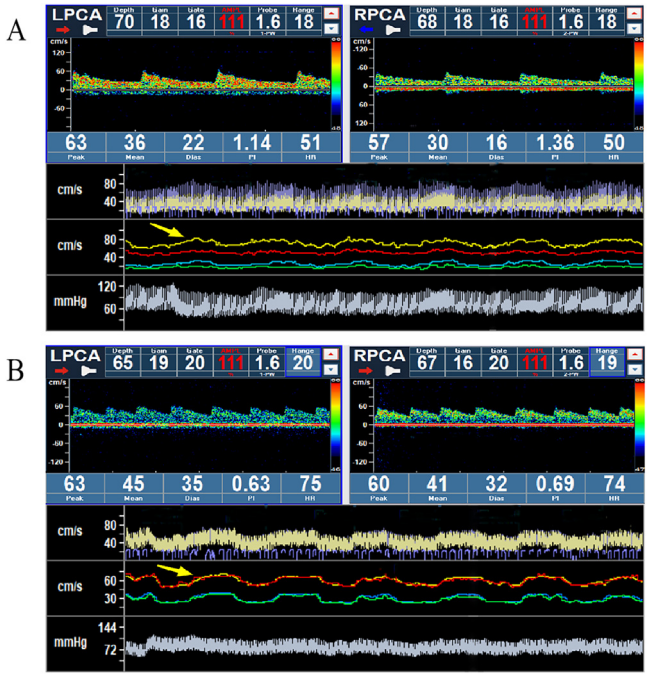

Figure 3. TCD waveforms of the CVST and control groups. (A) TCD waveform of the CVST group. (B) TCD waveform of the control group. The cerebral blood flow velocity of the control group significantly increased during silent reading with eyes open, with a greater amplitude of change than in the CVST group, indicated by the *arrow*. CVST, cerebral venous sinus thrombosis; TCD, transcranial Doppler.

This study had some limitations. First, the time between the onset of clinical symptoms and the TCD examination varied from patient to patient. The venous sinus thrombus may already have been in a subacute or even chronic phase when tested in certain individuals, which may have influenced the dCA and NVC results. Second, the study sample was small, and although the baseline data were similar for the CVST and control groups, bias could not be completely ruled out. Third, this was a cross-sectional study, and additional research is required to measure dCA and NVC again after clinical treatment and symptom improvement.

Conclusion

In patients with CVST, dCA and NVC of the PCA during silent reading were impaired, whereas dCA of the MCA was normal.

Conflict of interest

The authors declare no competing interests.

Acknowledgments

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Data availability statement

The data that support the findings of this study are available from the corresponding author on reasonable request.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.ultrasmedbio.2023.07.009.

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