

The Role of Cerebral Venous Dyshemias in the Formation of Chronic Cerebral Circulation Failure

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ABSTRACT

The arterial and venous components of the vascular bed of the brain are an interconnected system, the venous segment of which is a highly organized reflexogenic zone, which is responsible for the development of many complex reactions that ensure a normal level of cerebral blood flow. Despite the close relationship between the arterial link and the venous bed of the brain, the study of the venous component of the cerebral circulation has lagged significantly behind studies of arterial hemodynamics in normal and pathological conditions. The degree of compensation of venous dysgemia depends on collateral circulation and the rate of development of venous stasis inside the skull. Underestimation of the role of cerebral venous circulation in the formation of vascular cerebral pathology interferes with the correct understanding of the pathokinetic mechanisms of the development of chronic cerebral ischemia.

An important condition for the successful management of patients is the timely recognition and early diagnosis of chronic venous cerebral dyscirculation (VCD), which can lead to effective therapy.

KEYWORDS

Cerebrovascular Pathology, Chronic Cerebral Venous Discirculation.

Introduction

Vascular diseases of the brain are an urgent medical and social problem that occupies a leading place in the structure of morbidity and mortality in economically developed countries. In recent years, the structure of diseases of the nervous system has been changing due to the increase in ischemic forms. This is due to an increase in the proportion of arterial hypertension and atherosclerosis as the main cause of vascular pathology of the brain [1. 2].

Chronic cerebral ischemia (CCI) is a single pathological process that includes a variety of mechanisms arising from the inappropriateness of cerebral blood flow to the metabolic needs of the brain tissue, the leading place in the etiology of which belongs to arterial hypertension and atherosclerosis of cerebral vessels [3.4].

It has now been proven that in CCI, along with impaired blood flow to the brain in the affected artery basin, obstruction of venous outflow from the brain has an important pathogenetic significance [5.6-7]. In CCI, the state of the main arteries of the head is usually studied, while studies of the brain have shown that approximately 85% of the volume of the vascular bed of this organ is accounted for by venous vessels, 10% - by arteries, and about 5% - by capillaries.

Nevertheless, in clinical neurology by the 60-70s of the twentieth century, sufficient material was accumulated to suggest that cerebral venous circulation disorders have a certain proportion in the structure of vascular lesions of the brain.

Currently, there is no doubt about the relevance of issues of etiology, pathogenesis, clinical picture, diagnosis and treatment of cerebral venous blood circulation disorders, although the literature on this problem is not numerous.

Until now, no detailed description has been received of the relationship between the arterial and venous links of cerebral hemodynamics and cerebrovascular reactivity in the entire cerebral blood flow system at various stages of cerebrovascular insufficiency. The criteria for predicting the restoration of brain functions and cerebral hemodynamics in cerebral ischemia remain insufficiently developed, the information content of the existing markers of pathology is not defined, and the control of the effectiveness of the therapy is carried out mainly on the basis of clinical data (2. 3).

Therefore, conducting a study on the diagnosis and treatment of chronic cerebrovascular disorders in combination with cerebral venous dysfunction is timely.

Purpose of the study:improving the results of diagnosis and treatment of intracranial venous insufficiency.

Materials and Methods

A total of 282 patients with CCI stages I and II were studied against the background of Hypertension (HD) and Atherosclerosis (ATH) or their combination, in the period from 2015 to 2018. Among the studied there were 134 (47.5%) men and 148 (52.5%) women aged 50 to 76 years (average age 54.7 ± 5.1 years).The patients were examined and treated in the neurological department of the Somatic Hospital in Tashkent. During the REG study in 237 (84.0%) patients, venous cerebral dyscirculation (VCD) was revealed.All patients were divided into groups depending on the presence of cerebral venous dysfunction (VCD). According to the results of a study using a questionnaire to identify VCD (8), all patients were divided into 3 groups depending on the severity of their VCD... Group I consisted of 61 patients (21.6%) with grade 1 VCD, group II consisted of 101 patients (35.8%), group III consisted of 75 patients with grade 3 VCD (26.6%).The control group (CG) consisted of 45 people with CCI without signs of VCD from those examined in whom VCD was not detected.

All patients underwent a standard neurological examination (analysis of patient complaints, an objective examination, including the study of the neurological status), study of the clinical features of headache (HD) using the original questionnaire, determination of the intensity of headache using a ten-point visual analogue scale (VPS).

Along with clinical and neurological studies, in addition to EEG recording, studies of cognitive evoked potentials (P300) were included in the block of studies. Cognitive evoked potentials (CEP) were chosen due to the fact that they allow assessing endogenous events occurring in the brain and associated with the recognition and memorization of the presented stimuli (9).

Also, the patients underwent neurophysiological research methods: duplex scanning (DS) in the modes of color Doppler mapping and pulsed Doppler ultrasonography of extra- and intracranial vessels.

To analyze the data obtained, the Statistics software package was used using the Statistics 8.0 and Excel applied programs. The distribution of characteristics for normality was assessed using the Kolmogorov-Smirnov test.To identify the differences between the indicators in the compared groups with a normal distribution, the Student's t test was used, in cases where the distribution did not meet the criteria of normality, its nonparametric analogue, the Mann-Whitney test, was used. The critical level of significance (p) when testing statistical hypotheses in the study was taken equal to 0.05.When describing the results of the study, quantitative data are presented in the form $M (\pm \sigma)$, where M is the arithmetic mean, σ is the standard deviation, qualitative data are presented as absolute values, percentages and shares.

The Results of the Study

Analysis of 282 cases of chronic cerebral ischemia showed that the main etiological factors were: cerebral vascular atherosclerosis (24.1%), hypertension (38.4%) and their combination (37.6%), vasomotor dystonia (8.5%).(Extra systole, atrial fibrillation, paroxysmal tachycardia) were observed in 2.6% of patients, and in 31.6% - coronary heart disease. Concomitant degenerative-dystrophic changes in the cervical spine were detected in about half of the patients (74.4%), varicose veins of the lower extremities (57.7%), chronic obstructive pulmonary disease (COPD) - 41.0%, chronic heart failure (CHF) - 29.5%.A history of 16.5% of patients had indications of acute circulation accident (TIA, hypertensive crises, stroke).

Neurological examination in patients of the main group revealed small-focal disseminated symptoms. The most common symptom is soreness of the exit points of the V pair of cranial nerves. Most of these patients were found to have vegetative-vascular and neurosis-like syndromes. 68.2% of patients in the main group (more often women) were worried about morning swelling of the face after a night's sleep.When examining patients with VCD, a pronounced pallor of the skin in combination with a cyanotic color of the face was noticeable. In some cases, local cyanosis of the lipsthe puffiness of the face and eyelids in the morning was determined in the overwhelming number of patients (71.2%). With physical activity, the swelling decreased in the evening.

In the control group, the patients did not have “venous symptoms”. Clinical and neurological symptoms in patients in this group were less pronounced than in patients in the main group.

All the examined patients showed rather distinct focal symptoms. In the majority of patients, it was presented by the syndrome of pyramidal insufficiency, often in combination with the initial manifestations of the vestibulo-ataxic and cephalic syndromes. Along with signs of focal lesions, there were quite pronounced emotional-affective disorders and mnestic disorders. In patients of the main group, in comparison with the control group, such neurological syndromes as asthenic, cephalgic, vestibulo-ataxic pyramidal insufficiency were significantly more frequent in 82.3%, 100.0%, 53.6% and 62.0% of cases, respectively (table one).

Table1. Clinical and neurological symptoms in patients with CCI depending on the availability of VCD

Neurological syndromes	Groups of examined			
	main group (237)		control group (45)	
	n	%	n	%
Asthenic	195	82,3%*	15	33,3%
Cephalgic	237	100%*	11	24,4%
vegetative	186	78,5%*	21	46,7%
Vestibular-atactic	127	53,6%*	2	4,4%
Amiostatic	19	8,0%	0	0,0%
Pyramidal insufficiency	147	62,0%	7	15,6%
Pseudobulbar	23	9,7%	1	2,2%

Note: * - differences between groups, $p < 0.05$;

After analyzing the data obtained during the examination, we came to the conclusion that the formation of CCI depends not only on violations of arterial inflow, but also on the degree of inconsistency of venous cerebral outflow with arterial inflow. This imbalance reduces the hemodynamic reserve of cerebral circulation, contributing to the development of chronic cerebral ischemia and the formation of structural brain pathology in general both arterial and venous insufficiency - vascular discirculatory encephalopathy.

Cephalgic syndrome had distinctive features. Thus, in patients of the main group, headache (HD) was more often bilateral, localized in the parietal-occipital (45.3%) or frontotemporal (48.1%) areas. In 35.9% of cases, local headache became diffuse. Most had constant, people complained about the feeling of heaviness in the head. In 28.5% of cases, paroxysmal intense pain occurred, which was characterized as compressing, bursting, pressing and pulsating. It intensified against the background of psycho-emotional or physical stress, when working in an inclined position (36.2%).

The quantitative severity of HD in the examined patients according to VPS was, on average, determined in points. There were intergroup differences in the nature of the headache. So, in the main group, the headache had a severity of 5.1 points, which is significantly higher compared to the same indicator in the control group. All patients in the main group had a headache that occurs in the morning upon waking up, decreased after getting out of bed and disappeared by mid-day.

In patients of the control group, hypertension was more often paroxysmal (53.2%), bilateral (41.8%), with a projection in the occipito-parietal region (42.2%). HD increased against the background of psychoemotional or physical stress, when working in an inclined position (39.8%). Among the patients of the control group, HD in 27.4% of cases was disturbed at night. According to the VPS, this HD was defined as moderate - 4.3 points. Morning HD was rare and was observed in 18.4% of cases.

The assessment of the comorbid background of the patients of the two groups is presented in Table 2. According to the presented data, all patients had comorbidities, but among the patients of the main group in most cases (74.4%) metabolic syndrome, osteochondrosis of the cervical spine (94.9%), varicose veins of the lower extremities (57.8%), chronic obstructive pulmonary disease (41.4%), chronic heart failure (CHF) - 30.4%, which is significantly higher than these indicators, respectively, compared with patients in the control group.

Table 2. The structure of comorbidity in patients with CCI depending on the presence of VCD

comorbid diseases	main group (237)		control group (45)	
	n	%	n	%
Increased blood pressure	221	93,2% **	11	24,4%
Metabolic syndrome	176	74,3% **	12	26,7%
Osteochondrosis of the cervical spine	225	94,95% *	23	51,1%
Chronic obstructive pulmonary disease	98	41,4% **	3	6,7%
Varicose veins of the lower extremities	137	57,8% *	14	31,1%
Tension headache	148	62,4% **	8	17,8%
Cardiovascular insufficiency	72	30,4% *	5	11,1%

Note: * - differences between groups, $p < 0.05$; ** - differences between groups, $p < 0.005$.

Based on the data obtained, we formulated a questionnaire to identify the degree of venous cerebral dysfunction (VCD) (Fig. 1). The subject underlines the appropriate answer “Yes” or “No”. For a quantitative assessment of the available signs, an expert assessment of the “venous” symptoms was carried out by scoring each sign according to its specific gravity among the various symptoms of VCD (venous cerebral dysfunction). On this basis, an appropriate score was assigned to each symptom in case of a positive answer to the corresponding question. On this basis, an appropriate score was assigned to each symptom in case of a positive answer to the corresponding question [10].

Date	S.N.P.	age
Analyze		
circle the circles if you have such condition	Patient Questionnaire for Symptoms of Venous Cerebral Dysfunction	scores
	symptoms	
1	Headache on waking	2
2	Symptom of tight collar	2
3	Asymptom of gritty eyes	1
4	Pasty face and eyelids in the morning	2
5	Symptom of a high pillow	1
6	Decreased corneal reflexes	1
7	Soreness of exit points and hypesthesia in the innervation zone of the first branch of the	1
8	Sleep disturbance	1
9	Hypokinesia	1
	Comorbid background	
10	Increased blood pressure	2
11	Methodical syndrome	1
12	Osteochondrosis of the cervical spine	1
13	Chronic obstructive pulmonary disease	2
14	Varicose veins of the lower extremities	1
15	Tension headache	1
16	Cardiovascular insufficiency	2
	Interpretation	Scores
VCD 1 degrees	Mild severity	6-11
VCD 2 degrees	Moderate severity	12-17
VCD 3 degrees	Severe severity	18-22

Figure 1. Questionnaire for detecting clinical signs of cerebral venous dysfunction

The study design for identifying signs of VCD, which is usually filled in by a doctor: the resulting score from 6 to 11 determines the mild degree of VCD, from 12 to 17 points - the average severity of VCD, and the score from 18 to 22 is interpreted as severe.

According to this questionnaire, all patients were divided into 3 groups, depending on the severity of their VCD. The control group (CG) consisted of 45 people with CCI without signs of VCD.

In the study of neurophysiological features in patients with venous cerebral dysfunction, the following results were obtained. Comparison of the indicators obtained in the REG study, there was a difference in cerebral blood flow in patients with CCI, depending on the presence of VCD. In the study of the blood filling of the basin of the carotid arteries, it was revealed that the patients of the compared groups practically differed in terms of the parameters of pulse blood filling and the main rheographic indices. A qualitative analysis of the rheograms revealed that all patients with VCD showed changes in the form of an increase in the amplitude and shape of the venous wave.

Difficulty of venous outflow of the 1st degree significantly prevailed ($p < 0.045$) in patients with VCD II in 58.0% of cases, compared with patients with VCD I, where this indicator was lower and amounted to 36.5%. Difficulty of venous outflow of the 2nd degree prevailed in patients with VCD III in 63.4% of cases, which was significantly higher than this indicator in groups 1 and 2 ($p < 0.005$).

Intergroup comparisons of the data obtained in the REG study also had differences. Thus, in patients with VCD I, the following types of REG were identified - normo-hypertensive (32.5%) and arterial-hypertensive type of REG (51.7%); patients with VCD II - arterial-hypertensive type REG occurred in 65.7% of patients, vertebrogenic effect on the vertebral arteries was found in 35.2% of patients, a decrease in pulse blood filling - in 35.8% of cases. In patients with VCD III, the arterial-hypertensive type of REG was mainly detected (89.2%), 65.7% of patients with vertebrogenic effects on the vertebral arteries were significantly higher than in the second group.

All patients with VCD showed a significant decrease in all groups of patients in comparison with the control group in both leads of the rheographic index, the maximum rate of rapid blood filling, and the average rate of slow blood filling; an increase in the peripheral resistance index, diastolic and diastolic indices, venous outflow index (diagram 1).

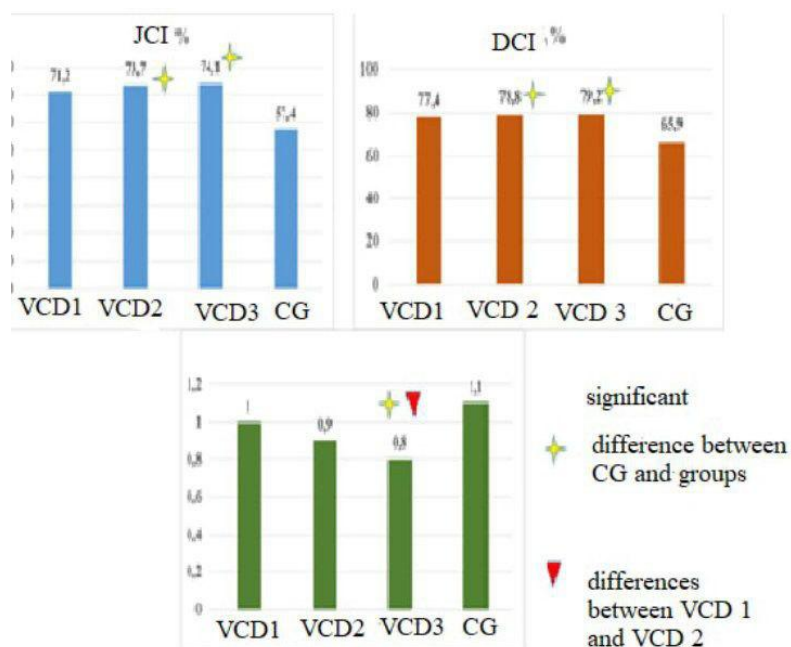


Diagram 1. REG indices in the carotid system, F-M derivations

Duplex sanitation (DS) and magnetic resonance angiography (MRA) revealed the presence of atherosclerotic changes in 25.8% of patients. At the same time, compaction of the artery wall was noted, atherosclerotic plaques, loosening and enlargement of the vascular intima were detected. The scatter of indices of the size of the intima-media

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complex was significant and ranged from 0.6 to 2.7 mm with an average of 1.4 ± 0.4 . The tortuosity of the carotid arteries at VCD II Art occurred in 31.4%; at VCD III Art. - in 38.2% of patients.

Thus, tortuosity of the carotid arteries was characteristic of a significant number of patients with CCI, regardless of the VCD stage. No statistically significant difference was obtained between the parties ($p > 0.5$). Evaluating LBFV, we can state that as the disease progresses, it decreases. It is noteworthy that these processes are more characteristic of the left CCA for VCD II and VCD III.

In order to assess hemodynamic changes during VCD against the background of CCI, we studied the volumetric blood flow rate Q (in ml / min) in the CA and VA, as well as the total Q sum ml. (Table 3).

Table 3. Data on the volumetric blood flow rate (Q, ml / min) of extracranial arteries depending on the VCD stage, $M \pm \sigma$.

Q	VCDI	VCDII	VCD III	Control group
CCA	$545,2 \pm 71,5$	$495,8 \pm 96,1^*$	$468,2 \pm 91,4^*$	$523,6 \pm 89,3$
ICA	$274,1 \pm 48,4$	$246,2 \pm 39,4^*$	$213,6 \pm 48,0^{* \wedge}$	$292,6 \pm 68,5$
PA	$92,5 \pm 26,1$	$81,6 \pm 29,3$	$54,9 \pm 240,4^*$	$96,3 \pm 35,5$
Qsumml.	$754,7 \pm 15,1$	$634,4 \pm 106,1^*$	$578,9 \pm 78,2^{* \wedge}$	$798,4 \pm 84,7$

The study of the volumetric blood flow rate in the main arteries of the head showed a significant decrease in the volumetric blood flow rate in all extracranial vessels (CCA, ICA, VA), as well as the total volumetric velocity Q sum ml with increasing severity of ICP.

When comparing the indicators of the state of the left common carotid arteries in patients with varying degrees of severity of VCD are presented in Table 4. In patients with VCD I, in comparison with the control group, there were no statistically significant differences. However, there was a tendency to a decrease in the linear blood flow velocity and an increase in the diameter and thickness of the intima-media complex, while an increase in vascular tone (IR) and peripheral resistance (IP) was noted, these indicators were significant for patients with VCD II in comparison with CG and VCD I. The S / D ratio increases in VCD III due to a greater decrease in the maximum diastolic blood flow velocity, which indicates the onset of diastolic dysfunction already with VCD I and VCD II.

Table 4. Comparison of the state of ultrasound triplex scanning of the left common carotid artery with varying degrees of venous cerebral dysfunction, $M \pm \sigma$

Indicator, units	VCDI	VCDII	VCDIII	Control group
Common carotid artery (CCA) diameter, mm	$6,08 \pm 0,14$	$6,18 \pm 0,15$	$7,11 \pm 0,19^*$	$5,99 \pm 0,23$
Intima-media complex (IMK) thickness, mm	$0,89 \pm 0,11$	$0,95 \pm 0,19^*$	$0,98 \pm 0,12^{* \#}$	$0,75 \pm 0,17$
Average blood flow velocity (Vmed), cm / s	$41,9 \pm 9,5$	$40,2 \pm 12,5$	$39,4 \pm 18,2^*$	$45,1 \pm 9,5$
Maximum blood flow velocity (Vmax), cm / s	$85,7 \pm 16,2$	$81,5 \pm 10,4$	$80,7 \pm 14,1$	$86,3 \pm 12,9$
Minimum blood flow velocity (Vmin), cm / s	$26,4 \pm 4,9$	$21,6 \pm 8,2^*$	$19,3 \pm 7,6^*$	$29,4 \pm 5,7$
Purselo circulatory resistance index (IR)	$0,7 \pm 0,05$	$0,75 \pm 0,04^{* \#}$	$0,77 \pm 0,01^{* \#}$	$0,68 \pm 0,06$
Gosling Pulsator Index (IP)	$1,41 \pm 0,54$	$1,50 \pm 0,36^*$	$1,54 \pm 0,31^{* \#}$	$1,31 \pm 0,3$
Volumetric average velocity (Vvolmed), ml / min	$286,2 \pm 48,1$	$274,2 \pm 50,1$	$258,5 \pm 49,9^*$	$290,8 \pm 50,4$
Ratio of maximum systolic blood flow velocity to maximum diastolic velocity (S / D)	$3,26 \pm 0,21$	$3,89 \pm 0,21^*$	$4,11 \pm 0,27^*$	$2,99 \pm 0,34$

In patients with VCD against the background of CCI, there is an increase in the diameter, thickness of the intima-media complex, linear blood flow velocity and a decrease in the volumetric mean blood flow velocity, with an increase in IR and IP against the background of developing diastolic dysfunction, due to an increase in vascular tone and rigidity. However, already with VCD III, a structural and functional change in the common carotid arteries occurs - the diameter of the common carotid artery increases, the thickness and elasticity of its walls increase, the muscle tone of the vascular wall increases, against the background of a statistically insignificant decrease in the volumetric blood flow velocity, peripheral resistance to blood flow increases. As a result of the revealed changes in the common carotid arteries, conditions for malnutrition of the brain structures appear in the VCD.

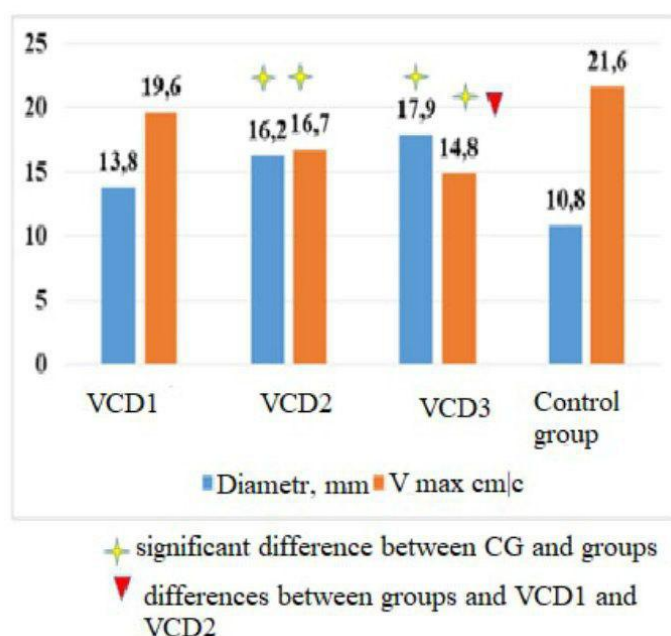


Diagram 2. Diameter and flow rate in the inner jugular vein depending on the VCD stage.

In the study of cerebral venous blood flow in the studied patients, a respiratory stress test was performed to assess the viability of the valve apparatus of the internal jugular vein (IJV): in response to a deep breath, the closure of the valve leaflets was observed, accompanied by a significant reduction in blood flow in the IJV. In 149 (62.9%) cases, a functional test showed a reversal of blood flow in valvular insufficiency. There were no cases of valve insufficiency in the control group.

Considering the role of the common jugular vein as a collector of venous outflow from the cranial cavity, venous outflow from the cranial cavity was studied in the vertebral and jugular veins. The decrease in the volumetric blood flow velocity in the internal jugular vein is most likely associated with a significant decrease in the linear blood flow velocity (LBFV) in the extracranial vessels in comparison with age norms (Diagram 2). In patients with VCD, higher values of the IVJ diameter and a comparative decrease in blood flow intensity are determined. The diagram shows that there is a tendency to a decrease in the intensity of blood flow depending on the stage of VCD.

The examination of the vertebral veins (VV) was performed in the supine position, and the VV blood flow was determined in patients with VCD II - in 26.4% of cases VCD III - in 42.7%. During the transition to orthostasis - in 100.0% of cases in both groups VV were inconsistent in a larger number of patients with ICP III - in 62.1% of cases ($p < 0.05$).

At the same time, according to the data of ultrasound examination, an increase in the maximum blood flow velocity (Vmax) in the basal veins of the brain (Rosenthal, Galen and the rectus sinus), as well as pseudopulsation in these vessels (the pulsatory index Gosling -RI) was noted. In patients of the control group, the blood flow in the basal vein of Rosenthal from both sides and in the straight sinus had a monophasic character compared with the main group, where a biphasic pattern of blood flow was observed.

When assessing the quantitative parameters of cerebral venous blood flow in all patients with VCD, difficulty in blood flow was revealed during the usual localization of intracranial veins. At the same time, in the deep venous system, a significant increase in LBFV values was noted, PI values were decreased.

Table 5. Blood flow parameters in intracranial veins depending on the VCD stage, $M \pm \sigma$

Groups	VCDI		VCDII		VCDIII
Parameters	V max, cm / s	RI	V max, cm / s	RI	V max, cm / s

Vienna Rosenthal	12,8 ± 5,4	0,41 ± 0,08	21,8 ± 7,2	0,28 ± 0,08	23,4 ± 7,2*
Straight sine	20,1 ± 6,2	0,48 ± 0,07	29,6 ± 7,8	0,32 ± 0,07	31,2 ± 6,2*

Conclusion

When diagnosing CCI, one should take into account the presence of venous discirculation of the brain. It is necessary to evaluate the “venous” complaints of patients and the peculiarities of the neurological status caused by venous cerebral disjunction. The use of the REG study technique and the use of the questionnaire developed by the authors – “Questionnaire for a patient to identify the presence of symptoms of Venous Cerebral Dysfunction (VCD)” - should be a screening study. For an objective assessment of the functional state of cerebral blood flow in patients with CCI, in addition to the study of the arterial component using CDS and TCD, it is necessary to study its venous component. This helps to clarify the stage of the disease, predict its course, and assess the effectiveness of the therapy. For differential diagnosis of the VCD stage, correction of treatment and prediction of the course of chronic cerebral ischemia in the practice of neurologists, it is necessary to investigate the state of the venous system of the brain.

References

- [1] Gusev, E.I., &Skvortsova, V.I. (2001). *Ischemia of the brain*. Moscow, 328.
- [2] Kotov, S.V., Isakova E.V., Kozyakin, V.V. (2014). On the issue of the prevention of cerebral stroke. *Russian medical journal*, 22(22), 1582–1585.
- [3] Suslina, Z.A. (2015). *Vascular diseases of the brain* / Z.A. Suslina, Yu. Varakin, N.V. Vereshchagin. Moscow: MED press-inform, 356.
- [4] Van Popele, N.M., Grobbee, D.E., Bots, M.L., Asmar, R., Topouchian, J., Reneman, R.S., & Witteman, J.C. (2001). Association between arterial stiffness and atherosclerosis: the Rotterdam Study. *Stroke*, 32(2), 454-460.
- [5] Belova, L.A. (2010). Venous Cerebral Discirculation at Chronic Cerebral Ischemia: Clinics, Diagnostics, Treatment. *Neurological Bulletin*, 42(2), 62-67.
- [6] Manvelov, L.S., &Kadykov, A.V. (2007). Venous insufficiency of cerebral circulation. *Atmosphere. Nervous diseases*, 2,18-21.
- [7] Ivanov A. Yu., Panuntsev A.N., Kondratyev A.N., Ivanova N.E., Petrov A.E., Komkov D.Yu., Panuntsev G.K., Cherepanova E.V., Vershinina E A.A., Popova E.V., &Ustaeva I.G. (2010). Features of venous outflow from the brain *Neurological bulletin*, 5–10.
- [8] Gafurov, B.G.(2018). Clinical and functional features of venous cerebral circulation in chronic cerebral ischemia. *Journal of Neurology*, 4, 45-48.