Evaluation of cerebral circulation in patients with significant carotid artery stenosis

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Abstract

Background: A significant proportion of ischaemic stroke episodes are caused by atherosclerotic lesions in extracranial arteries. Assessment of haemodynamical profile of cerebral arteries in both symptomatic and asymptomatic patients with carotid artery stenosis is of clinical importance.

Aim: To assess haemodynamic changes in cerebral arteries in patients with significant internal carotid artery (ICA) stenosis.

Method: Patients (n=109) were divided into the following groups: group I (GI) – 42 subjects (64.6±9.0 years) with asymptomatic ICA stenosis ≥70%; and group II (GII) – 67 subjects (63.4±7.1 years) after stroke. The control group consisted of 30 patients (60.3±8.9 years) without significant stenoses of extracranial arteries on USG and angiography. In all cases ultrasonographic evaluation of flow velocities and directions in cerebral arteries within the circle of Willis and collateral flow was performed.

Results: The severity of ICA stenosis did not differ significantly between GI and GII. Patients in GI had flow velocity in the middle cerebral artery (MCA) increased by 15.7% and by 40.8% in the anterior cerebral artery (ACA) contralateral to the ICA stenosis ($p<0.001$ and $p<0.001$), whereas in GII no significant changes in flow velocity in these arteries were observed in comparison with the control group. Patients in the groups I and II had lower flow velocities in MCA ipsilateral to the ICA stenosis, however values for GII patients were significantly lower than in GI patients ($p<0.001$). The presence of collateral circulation through the anterior and posterior communicating arteries (ACoA and PCoA) was similar in GI and GII; however, the flow velocities in the ipsilateral MCA and ACA were significantly higher in asymptomatic patients (GI). The frequency of active collateral circulation through both ACoA and PCoA increased along with the increase of ICA stenosis severity ($p=0.003$; $p<0.001$).

Conclusions: Collateral flow in the circle of Willis in subjects with ICA stenosis occurs equally often in symptomatic and asymptomatic patients; however, it is more efficient in patients without symptoms. The rate of development of collateral circulation depends on ICA stenosis severity. The important role in maintaining collaterals within the circle of Willis is played by ACoA, although in some patients MCA may also be supplied by PCoA.

Key words: internal carotid artery stenosis, collateral flow through the circle of Willis, transcranial Doppler ultrasonography, asymptomatic patients, symptomatic patients

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Introduction

It is estimated that approximately 10-20% of all ischaemic strokes are caused by atherosclerotic lesions in the arteries arising from the aortic arch, which would lead in Poland to about 6-8 thousand cases each year [1]. Prodromal neurological symptoms such as *amaurosis fugax*, numbness and paraesthesia of limbs, transient aphasia, paresis of limb and facial muscles as well as other symptoms defined as transient ischaemic attacks (TIA), precede ischaemic strokes only in 20% of cases. Ischaemic stroke occurs as a result of two mechanisms. In the first one, critical artery stenosis reduces the flow distally to

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stenosis and causes hypoperfusion of the central nervous system structures [2, 3]. In the other mechanism, ischaemia occurs after abruption of embolic material from the thrombogenic surface of ulcerous atherosclerotic plaque and clogging of the periphery of the cerebral artery [4, 5]. Both mechanisms may coexist. Clinical consequences of stenosis as well as artery embolism may vary extensively, despite similar anatomical and morphological findings in the extracranial arteries [6].

This study attempted to assess haemodynamic changes and the adaptation process taking place in the cerebral arteries of the circle of Willis in patients with significant atherosclerotic stenoses (≥70%) of internal carotid arteries (ICA), in patients without neurological symptoms or in patients after ischaemic stroke.

Methods

Patients

The study involved 109 (89 M, 20 F) patients, mean age 64.0±7.8 (44-82) years, selected from 116 consecutive patients (91 M, 25 F) with ICA stenosis ≥70%, and without significant atherosclerotic lesions (≥50%) in the vertebral and subclavian arteries, who underwent attempted transcranial Doppler ultrasonography (TCD). The reason for exclusion of seven (6.0%) patients (2 M, 5 F) was a non-diagnostic Doppler signal through the temporal window.

Patients with ICA stenosis ≥70% were divided into two groups:

- GI: 42 patients without neurological symptoms, in whom ICA stenosis was diagnosed during Doppler ultrasonography screening or following artery murmur auscultation.
- GII: 67 patients after a neurological event such as ischaemic stroke or TIA. Additionally, G II patients were divided into two subgroups:
  IIA – 38 subjects with unilateral ICA stenosis,
  IIA – 29 subjects with bilateral ICA stenoses.

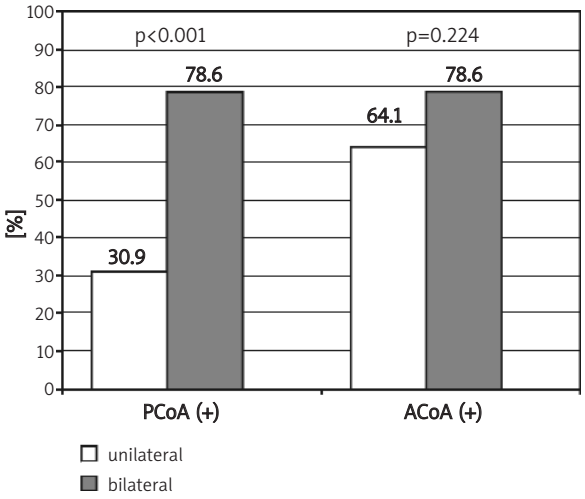

Figure 1. Rate of collateral flow through ACoA and PCoA in patients with unilateral and bilateral significant ICA stenosis

The control group (GIII) consisted of 30 individuals (22 M, 8 F), mean age 60.3±8.9 (43-78) years without a history of neurological events, and with ultrasonographically and angiographically excluded atherosclerosis of the extracranial arteries (stenoses <40%).

Ultrasonography

All patients had ultrasonography of aortic arch branches in their extracranial parts performed using duplex sonography and a 5-10 MHz linear array probe (Toshiba Aplio). On examination, flow velocities and location of atherosclerotic lesions in the common, internal and external carotid arteries as well as vertebral and proximal segments of the subclavian arteries were determined. The severity of carotid artery stenosis was established according to criteria of Bluth, adopted by the Polish Ultrasonographic Society, based on measurement of flow velocity [7].

If haemodynamically significant ICA stenosis was found (≥70%), transcranial examination of the cerebral arteries was performed via the temporal window using the Toshiba Aplio system and a 1.5-2.5 MHz convex array probe. The transcranial examination was used to measure flow velocities and directions bilaterally in the middle, anterior and posterior cerebral arteries. Additionally, the presence of collateral cerebral circulation through the anterior communicating artery (ACoA) and the posterior communicating arteries (PCoA) as well as flow direction were evaluated.

Similar ultrasonographic examination of the extracranial arteries and the circle of Willis was conducted in patients from the control group, and the flow velocities in the cerebral arteries measured in this group were the reference values for further analyses.

The severity of extracranial artery stenoses was validated and the cerebral circulation was assessed using angiography.

Statistical analysis

The statistical analysis included maximal, mean and end-diastolic flow velocities recorded with transcranial Doppler within the main cerebral arteries:

- ipsilateral to ICA stenosis – middle cerebral artery (iMCA), anterior cerebral artery (iACA) and proximal

Table I. Patient characteristics

	Group I N=42	Group II N=67	Controls N=30	p*
Age (years)	64.6±9.0	63.4±7.1	62.0±9.0	0.429
Males	30 (71.4%)	59 (88.1%)	22 (73.3%)	0.066
Hypertension	41 (97.6%)	59 (88.0%)	21 (70.0%)	0.002
Type 2 diabetes	13 (30.9%)	18 (26.9%)	8 (26.7%)	0.885
Hyperlipidaemia	38 (90.5%)	59 (88.1%)	22 (73.3%)	0.091
Smoking	28 (66.7%)	51 (76.1%)	13 (43.3%)	0.006
Coronary artery disease	36 (85.7%)	58 (86.6%)	18 (60%)	0.005
Past myocardial infarction	17 (40.5%)	34 (50.8%)	12 (40.0%)	0.470
Body mass index	27.7±3.0	27.5±3.4	28.1±3.8	0.797
ICA stenosis grade	86.6±10.2	89.8±10.2	–	0.113

**p – patients from groups I and II vs controls*

posterior cerebral artery (iP1CA) and distal posterior cerebral artery (iP2CA);
- contralateral to ICA stenosis: middle cerebral artery (cMCA), anterior cerebral artery (cACA) and proximal posterior cerebral artery (cP1CA) and distal posterior cerebral artery (cP2CA).

In the case of bilateral stenosis of ICA, the cerebral arteries supplied by the carotid artery with more severe stenosis as shown on angiography were recognised as ipsilateral.

The differences between recorded flow velocities with respect to ipsilateral cerebral arteries between the studied groups and subgroups were verified with the U-Mann-Whitney test for independent variables.

The Chi-square test was used to determine the relationship between the presence of collateral cerebral flow and neurological symptoms and the location of atherosclerotic lesions in the carotid arteries.

Statistical analyses were carried out using Statistica 5.5 software. A statistical significance level of $p<0.05$ was used.

Results

The characteristics of the studied groups are shown in Table I. There were no significant differences between the groups with reference to age, sex, incidence of myocardial infarction, diabetes mellitus, hyperlipidaemia, or body mass index (Table I). Patients in GI and GII had arterial hypertension, coronary artery disease and were smokers more often than patients in the control group.

There was no significant difference in severity of ICA stenosis in GI and GII. In patients with bilateral stenoses, the mean grade of contralateral ICA stenosis was 86.8±10.7% (70-100%). On angiography, significant stenosis of one or both ICAs was confirmed in all patients included in the study.

In the control group the mean flow velocities in the middle cerebral artery were 78.5±17.5 cm/sec, in the anterior cerebral artery 75.3±17.6 cm/sec, and in the posterior cerebral artery 57.4±13.3 cm/sec. On TCD, the blood flow directions within the ipsilateral middle and anterior cerebral arteries were opposite, which excluded the presence of active collateral flow through ACoA (Figure 2). In no patient was flow in PCoA documented, either.

Patients in GI had a significant increase in mean flow velocities in the middle cerebral artery by 15.7% and in the anterior cerebral artery by 40.8% contralaterally to ICA stenosis in comparison to flow velocities in the control group ($p<0.001$ and $p<0.001$ respectively, see also Table II). In GI patients, mean flow velocities in the middle cerebral artery ipsilateral to ICA stenosis were significantly lower than velocities measured in the control group ($p=0.025$); however, mean flow velocities in the anterior cerebral artery did not differ significantly ($p=0.255$).

In GII patients, no statistically significant differences were observed with respect to flow velocities in the middle ($p=0.358$) and anterior cerebral arteries ($p=0.075$) contralateral to ICA stenosis, as compared to control group. Nevertheless, a significant reduction by 33.2% ($p<0.001$) of mean flow velocities in the middle cerebral artery ipsilateral to ICA stenosis was found, whereas the velocities in the anterior cerebral artery were lower by 7.6% in comparison to the control group ($p=0.058$).

The comparison of flow velocities between GI and GII revealed that in both middle cerebral arteries as well as in iACA the flow velocities were significantly lower in patients with past neurological events than in subjects with asymptomatic stenosis ($p<0.001$; $p<0.001$ and $p=0.008$ respectively, see Table II).

In GIIA (symptomatic, unilateral ICA stenosis) lower velocities were seen in the ipsilateral middle cerebral artery ($p<0.001$) and similar velocities in the

Table II. Flow velocities in the ipsilateral (iMCA. iACA. iP1CA. iP2CA) and contralateral (cMCA. cACA. cP1CA. cP2CA) cerebral arteries in the studied groups

Artery	Velocity [cm/sec]	Group I N=42	Group II N=67	U Mann Whitneyp*	Controls (group III) N=30	U Mann Whitney p*
iMCA	PSV	71.1±16.4	52.4±21.3	<0.001	78.5±17.5	I *vs* III=0.025
	EDV	28.6±7.6	21.8±9.2			
	mean	49.9±12.0	37.1±15.3			II *vs* III <0.001
cMCA	PSV	90.8±22.9	75.1±27.2	<0.001		I *vs* III=0.001
	EDV	33.9±9.0	32.0±15.7			
	mean	62.4±16.0	53.6±21.5			II *vs* III=0.358
iACA	PSV	87.3±35.6	69.5±34.1	0.008	75.3±17.6	I *vs* III=0.255
	EDV	33.8±16.4	29.1±16.7			
	mean	60.5±26.2	49.3±25.4			II *vs* III=0.058
cACA	PSV	105.9±47.5	91.3±43.7	0.090		I *vs* III <0.001
	EDV	41.2±19.8	42.3±24.7			
	mean	73.6±33.7	66.8±34.2			II *vs* III=0.075
iP1CA	PSV	59.7±19.1	62.8±33.3	0.901	57.1±13.0	I *vs* III=0.532
	EDV	22.7±7.5	25.9±15.4			
	mean	39.4±13.0	44.4±24.4			II *vs* III=0.728
cP1CA	PSV	60.6±16.8	57.1±22.6	0.180		I *vs* III=0.368
	EDV	23.2±5.9	24.5±15.9			
	mean	41.9±11.4	40.8±19.3			II *vs* III=0.314
iP2CA	PSV	54.3±14.0	50.4±16.8	0.145	57.4±13.3	I *vs* III=0.468
	EDV	20.9±6.1	20.1±6.9			
	mean	37.6±10.0	35.3±11.9			II *vs* III=0.008
cP2CA	PSV	58.7±17.4	50.8±17.9	0.014		I *vs* III=0.588
	EDV	22.9±8.8	22.3±15.5			
	mean	40.8±13.1	36.6±16.7			II *vs* III=0.009

** p values were given for peak systolic velocities: PSV – peak systolic velocity; EDV – end-diastolic velocity*
Abbreviations: see "Methods" section

contralateral middle cerebral artery were observed (p=0.312) in comparison to controls (Table III).

In GIIB (symptomatic, bilateral ICA stenosis) significantly lower flow velocities were found bilaterally in the middle cerebral arteries than in the control group (p<0.001; p=0.004, see Table III).

The differences in the mean flow velocities between the studied groups resulted particularly from variable efficiency of the collateral circulation.

In 35 (81%) GI patients the flow direction recorded in the anterior cerebral artery ipsilateral to ICA stenosis was consistent with the one in the ipsilateral middle cerebral artery (Figure 2), which indicates the opening of collateral circulation through ACoA supplied from the contralateral ICA; moreover, flow velocities in all patients exceeded 44.8 cm/sec (i.e. the lowest velocity measured in the control group). In eight (19%) GI patients collateral circulation through ACoA was not observed (Figure 3); however, flow velocities within iMCA were not significantly different from those in patients with open ACoA (66.9±11.8 cm/sec vs. 72.1±17.2 cm/sec; p=0.427).

In GII, collateral circulation through ACoA was seen in 49 (73.1%) patients (p=0.273, for comparison with the rate of collateral circulation through ACoA in GI). The flow velocities in iMCA found in these patients were 55.4±21.5 cm/sec, despite opened collateral circulation. Moreover, in GI patients the flow velocities in iACA were significantly higher than in GII subjects (Table II). Thus, lower flow velocities recorded in iMCA in patients with a history of neurological events might be associated with insufficiently developed collateral circulation through ACoA from normal to the affected side in comparison to GI patients (p=0.009, see Table IV). However, it cannot be precluded that the observed reduction of flow velocity is a result of prior stroke due to embolisation and occlusion of distal branches.

Table III. Flow velocities in the cerebral arteries in Group II with ipsilateral and contralateral ICA stenosis

Artery	Velocity [cm/sec]	Group IIA N=38	Group IIB N=29	p* G IIA *vs* G IIB	Controls N=30	p* Controls *vs* G IIA	p* Controls *vs* G IIB
	PSV	54.6±23.1	49.6±18.8				
iMCA	EDV	22.1±10.1	21.5±8.1	0.390		<0.001	<0.001
	mean	38.4±16.6	35.6±13.4		78.5±17.5		
	PSV	83.9±29.2	63.7±19.3		30.8±9.9		
cMCA	EDV	35.2±18.9	27.8±9.0	0.002	54.7±13.7	0.312	0.004
	mean	59.6±24.1	45.8±14.2				
	PSV	65.7±28.4	74.5±40.3				
iACA	EDV	27.2±13.6	31.7±17.9	0.657		0.084	0.180
	mean	46.5±21.0	53.1±29.1		75.3±17.6		
	PSV	94.2±44.1	87.4±43.6		30.2±9.1		
cACA	EDV	43.9±25.9	40.0±23.2	0.511	52.7±13.4	0.039	0.447
	mean	69.1±35.0	63.7±34.4				
	PSV	57.1±15.4	70.3±44.2				
iP1CA	EDV	22.6±8.5	30.3±20.7	0.414		0.997	0.526
	mean	39.8±12.0	50.3±32.4		57.1±13.0		
	PSV	57.2±22.6	56.9±22.9		23.6±7.7		
cP1CA	EDV	26.2±19.7	22.4±8.6	0.960	40.3±10.4	0.595	0.236
	mean	41.7±21.2	39.7±15.8				
	PSV	54.2±18.1	44.7±13.0				
iP2CA	EDV	21.4±7.4	18.2±5.6	0.027		0.565	<0.001
	mean	37.8±12.8	31.5±9.3		57.4±13.3		
	PSV	54.8±20.1	45.7±13.2		23.4±7.7		
cP2CA	EDV	24.9±19.3	18.7±7.0	0.072	40.4±11.5	0.345	<0.001
	mean	39.9±19.7	32.2±10.1				

** p values were given for peak systolic velocities: PSV – peak systolic velocity; EDV – end-diastolic velocity*
Abbreviations: see "Methods" section

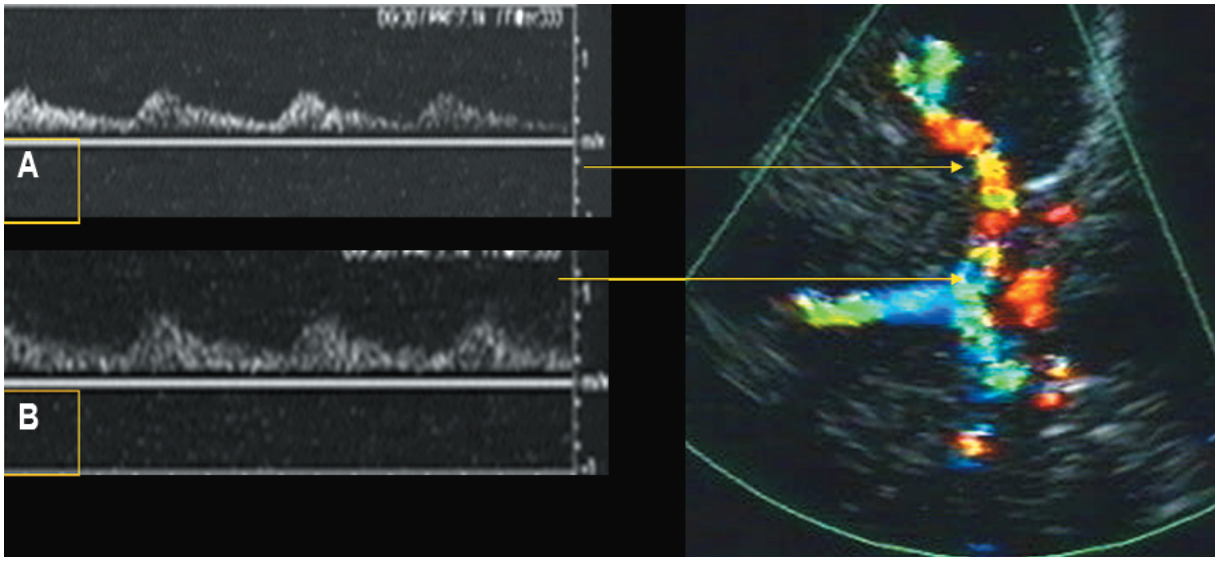

Figure 2. Active collateral flow through ACoA. Flow directions A (in the middle cerebral artery) and B (in the anterior cerebral artery ipsilateral to ICA stenosis) are parallel.

Table IV. Collateral flow in patients with ICA stenosis and in patients without significant atherosclerotic lesions in the extracranial arteries

	No. of patients	AcoA			p	PCoA		p
		None N [%]	Velocity <44.8 cm/sec* N [%]	Velocity ≥44.8 cm/sec* N [%]		None N [%]	Present N [%]	
ICA ≥70%								
Group I	42	8 (19.0)	0 (0)	35 (81.0)	0.009	24 (57.1)	18 (42.9)	0.844
Group II	67	18 (26.9)	11 (16.4)	38 (56.7)		37 (55.2)	30 (44.8)	
Controls	30	30 (100)	0 (0)	(0)		30 (100)	0 (0)	

** minimal flow velocity in the middle cerebral artery in the control group*

The statistical analysis showed that the incidence of collateral circulation through ACoA in patients with ICA stenosis ≥70% depended on the severity of ICA stenosis (no collateral circulation – mean stenosis of 84.6±11.3%; presence of collateral circulation – mean stenosis 90.7±9.1% (p=0.003)).

In 48 (44.0%) GI and GII patients the presence of at least one active PCoA was found, including 11 patients with both PCoAs, ensuring blood flow from the posterior cerebral artery to the site of division of the stenosed ICA into the middle and anterior cerebral arteries.

In GI flow in the PCoA was recorded in 42.9%, and in GII – in 44.8% (p=0.844). It was found that the rate of PCoA presence increases along with the severity of ICA stenosis. Out of 62 subjects with ICA stenosis ≤90%, the presence of flow in PCoA was diagnosed in 18 (29.0%) patients, and out of 47 patients with stenosis of >90% – in 30 (63.8%) patients (Chi^2; p<0.001). The occurrence of open collateral circulation through PCoA was also dependent on bilateral atherosclerotic lesions in ICA. In patients with bilateral stenoses active PCoA was observed more frequently (p<0.001, see Figure 3).

Discussion

Transcranial Doppler examination of the circle of Willis enables both assessment of flow velocity and directions in separate cerebral arteries, as well as observation of anastomoses connecting them. The usefulness of this method has been proved in recent years [8-11]. However, in some percentage of patients, more often in women (up to 30%), this examination is non-diagnostic due to increased calcification of the temporal squama [12, 13]. A similar situation took place in our patients: in 7 of 116 (6.0%) subjects, including 5 (20%) of 25 women, a diagnostic signal via the temporal window was not obtained. The flow velocities in the cerebral arteries differ with respect to gender (faster flow occurs in women), and decrease along with ageing [14, 15]. The flow velocity reference values worked out so far vary, which may result from the use of a range of ultrasonographic devices, the method of examination and demographic differences [16, 17]. Therefore, our study involved a group of 30 individuals without significant atherosclerotic lesions in the intra- and extracranial arteries in order to establish our own reference values with TCD examination.

Patients with ICA stenosis are characterised by a very distinct clinical course of the disease, from asymptomatic to ischaemic stroke [8]. The adaptive changes in the cerebral arteries in patients with ICA stenosis were the subject of postmortem examinations and imaging studies such as magnetic resonance imaging, computed tomography and TCD [18-20].

In asymptomatic patients, an increase of flow velocity in the anterior cerebral artery of 41% and in the middle cerebral artery of 16% contralateral to ICA stenosis were found. Other authors observed a significant increase of blood flow velocities in arteries contralateral to stenosis ranging from 18% to 78% for the anterior cerebral artery and of several points (7.3%) for the middle cerebral artery [18]. In patients with efficient cerebral collateral circulation the increase in flow velocity ipsilateral to normal ICA enables some portion of the blood through ACoA to be supplied to the anterior cerebral artery ipsilateral to ICA stenosis and further to the middle cerebral artery located on the same side, maintaining a relatively high flow rate; this was also confirmed by other investigators [21-23]. On the other hand, patients with neurological symptoms had clearly reduced flow velocities within the middle and anterior cerebral arteries ipsilateral to ICA stenosis, and no flow velocity increase within cerebral arteries was observed contralaterally.

The presence or lack of neurological symptoms in patients with significant ICA stenosis is associated mainly with the normal circle of Willis, and in particular with collateral circulation via ACoA.

Autopsy examinations have revealed that in patients dying from ischaemic stroke associated with ICA stenosis, insufficiently developed ACoA was more often seen [24-26]. The diameter of ACoA differs significantly between individual patients and ranges from 0.1 to 4.9 mm [19, 27,

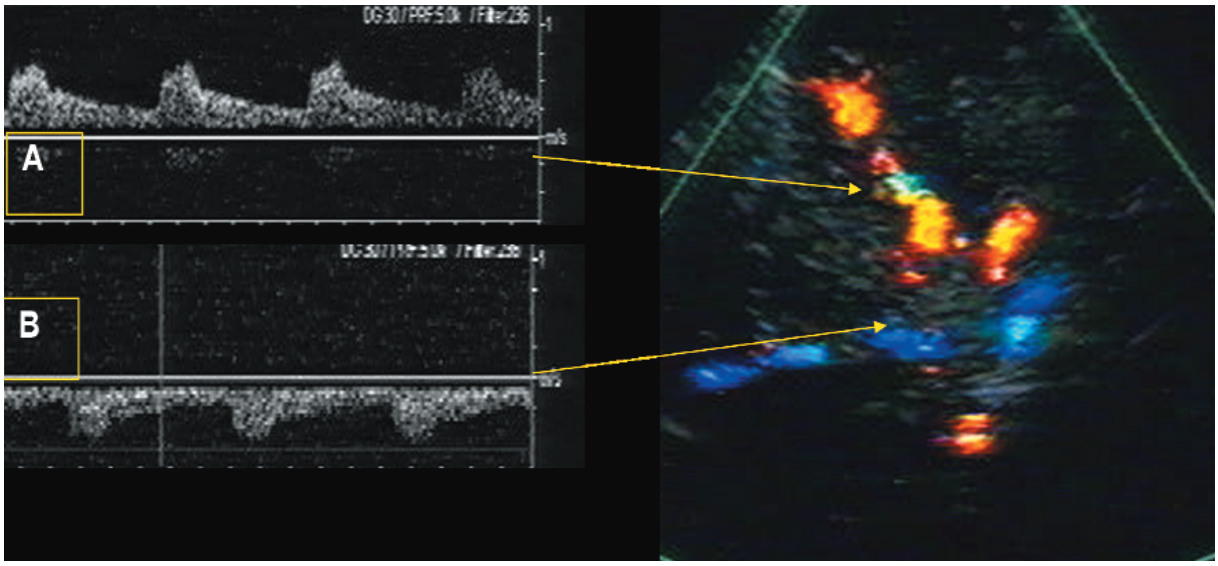

Figure 3. Collateral flow through ACoA not developed. Flow directions A (in the middle cerebral artery) and B (in the anterior cerebral artery ipsilateral to ICA stenosis) are opposite.

28]. Recently published studies by Cassot et al. using an experimental model of the cerebral circulation showed that the collateral circulation was fully efficient when the ACoA diameter was >1.6 mm, whereas ACoA with a diameter below 0.4 mm performed as an occluded blood vessel [29]. Within the range of 0.4 and 1.6 mm small changes in ACoA diameter were found to have a marked effect on the haemodynamics of intracerebral flows. Postmortem examination and magnetic resonance imaging demonstrated that an ACoA diameter of ≤0.4 mm was found in 2% of patients, a diameter within the range of 0.5-1.5 mm in 80% of patients and a diameter of ≥1.6 mm in 18% of patients [30].

With the current ultrasonography spatial resolution, the ACoA may occasionally be seen with TCD examination. Its patency with coexisting stenosis of ICA is confirmed by consistent flow directions within the anterior and middle cerebral arteries, and flow velocities enable evaluation of collateral flow efficiency. According to literature findings, the sensitivity and specificity of correct evaluation of anastomoses are respectively 98% and 100% for ACoA and 84% and 94% for PCoA [21].

There are also other collateral flow pathways in patients with ICA stenosis. Of note, in our series eight (19%) asymptomatic patients had no active collateral flow through ACoA. However, flow velocities in iMCA, though significantly lower than in the control group, remained considerably higher than in symptomatic patients. This finding may be explained by collateral blood flows with different pathways, i. e. from the posterior cerebral artery through PCoA (in our material 2 of 8 patients), through the ophthalmic artery and supratrochlear artery or epidural branches.

On the other hand, in some patients with neurological symptoms efficient collateral flow through ACoA and relatively high flow velocities in iMCA were observed (Table IV). A potential explanation of the presence of symptoms may be the embolic mechanism of stroke and not stroke resulting from reduction of cerebral blood flow. Embolus is one of two mechanisms leading to stroke. It may occur even with atherosclerotic plaques that mildly or moderately narrow the cervico-cephalic artery. Numerous studies, including studies by El-Barghouty and Nicolaides, have shown that lesions rich in lipids and containing little collagen are more frequently the cause of embolisation than collagen-rich and highly calcified lesions [31]. Currently, studies on the relationship between the atherosclerotic plaque structure evaluated with different methods and the risk of embolic stroke are being carried out. It seems, however, that in the case of stable and, in particular, calcified atherosclerotic lesions significantly narrowing the vessel, the mechanism of cerebral flow reduction and the presence of collateral circulation play an important role in the pathophysiology of stroke.

The ACoA holds an important place in cerebral flow autoregulation [20, 22, 30]. However, the middle cerebral artery may be supplied from another vessel – PCoA [32-33]. We found in our study a similar rate of active PCoA in asymptomatic patients and in subjects after ischaemic stroke. Our observations, which are consistent with the data presented by Reutern et al. [34], revealed that the rate of presence of collateral flow from ipsilateral PCoA increased along with the severity of stenosis, and was more common in patients with bilateral critical ICA stenoses.

No compensating mechanisms are effective when critical ICA stenosis coexists with contralateral carotid artery stenosis exceeding 55% [29]. Indeed, we found a decrease in flow velocities in both middle cerebral arteries, even despite the presence of collateral flow in patients with bilateral ICA stenoses.

Conclusions

1) Transcranial Doppler examination allows evaluation of the cerebral circulation in 94% of patients.
2) The rate of active collateral blood flow in symptomatic and asymptomatic patients is similar. However, it is more effective in asymptomatic patients.
3) The anterior communicating cerebral artery plays an important role in maintaining correct collateral flow within the circle of Willis, although in nearly half of patients the middle cerebral artery ipsilateral to ICA stenosis may be supplied from the posterior communicating cerebral artery.
4) The frequency of the active anterior and posterior communicating cerebral arteries' presence increases along with the severity of ICA stenosis.

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Ocena krążenia mózgowego u chorych z istotnym zwężeniem tętnic szyjnych

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Streszczenie

Wstęp: Istotny odsetek udarów niedokrwiennych spowodowany jest miażdżycowym zwężeniem w tętnicach zewnątrzczaszkowych. Ocena profilu hemodynamicznego tętnic wewnątrzczaszkowych u chorych ze zwężeniem tętnic szyjnych, zarówno bezobjawowych jak i objawowych, jest istotna.

Cel: W pracy podjęto próbę oceny zmian hemodynamicznych w tętnicach mózgowych u chorych z istotnym zwężeniem tętnic szyjnych wewnętrznych (ICA).

Metoda: 109 chorych podzielono na grupy: grupa I (GI): 42 chorych (64,6±9,0 lat) z bezobjawowym zwężeniem ICA ≥70% oraz grupa II (GII): 67 chorych (63,4±7,1 lat) po przebytym udarze mózgu. Grupę kontrolną stanowiło 30 chorych (60,3±8,9 lat) bez istotnych zwężeń w tętnicach dogłowowych w USG i angiografii. U wszystkich oceniono ultrasonograficznie prędkości oraz kierunki przepływu w tętnicach mózgowych koła Willisa oraz drogi krążenia obocznego.

Wyniki: Stopień zwężenia ICA w GI i GII nie różnił się istotnie. W porównaniu z grupa kontrolną, chorzy z GI wykazywali zwiększenie przepływu o 15,7% w tętnicy środkowej (MCA) i o 40,8% w tętnicy przedniej (ACA) mózgu po stronie przeciwnej do zwężenia ICA ($p<0,001$ oraz $p<0,001$), podczas gdy w GII nie obserwowano istotnych różnic w prędkości przepływu w tych tętnicach. Po stronie zwężenia ICA, chorzy z obu grup wykazywali niższe prędkości w MCA, z tym że prędkości u chorych z GII były znamiennie niższe niż w GI ($p<0,001$). Krążenie oboczne przez tętnicę łączącą mózgu przednią (ACoA) oraz tylną (PCoA) występowało równie często w GI i GII, jednak wśród chorych bezobjawowych (GI) prędkości przepływu w ipsilateralnej MCA i ACA były istotnie wyższe. Częstość otwierania się krążenia obocznego zarówno przez ACoA, jak i PCoA wzrastała ze wzrostem stopnia zwężenia ICA ($p=0,003$; $p<0,001$).

Wnioski: W zwężeniu ICA krążenie oboczne w kole Willisa występuje równie często u chorych objawowych i bezobjawowych, jednak u bezobjawowych jest ono bardziej wydolne. Częstość wytwarzania krążenia obocznego zależy od stopnia zwężenia ICA i podstawową rolę w jego utrzymaniu w obrębie koła Willisa odgrywa ACoA, jakkolwiek u części chorych MCA może być zaopatrywana również przez PCoA.

Słowa kluczowe: zwężenie tętnicy szyjnej wewnętrznej, krążenie oboczne przez koło Willisa, przezczaszkowa ultrasonografia dopplerowska, chorzy bezobjawowi, chorzy objawowi

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