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Experimental Medicine

Brain Focal Impairment and Hemodynamic Parameters in Relation to the Type of Collateral Supply

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ABSTRACT. The aim of the paper was to study the relationship between collateral flow via different pathways and cerebral hemodynamic parameters in patients with unilateral high-grade internal carotid artery (ICA) changes.

Materials and Methods: 41 patients with severe stenosis and 30 patients with occlusion of ICA underwent brain MRT, 3D TOF-MR-angiography, Color Doppler of extra-intracranial vessels to investigate collateral flow via the circle of Willis and via the ophthalmic artery (OphA). Maps of the cerebral perfusion parameters were calculated.

Results: In 50(70%) cases "symptomatic" cerebral ischemia was noted. In symptomatic patients cortical MCA infarctions (13(26%)), and border-zone infarctions-10(20%) prevailed. In cases of unilateral ICA occlusion compensatory dilatation of contralateral ICA and enhancement of flow volume by 60%, additionally enhancement of flow in the vertebral arteries by 18% was observed. Patients without collateral flow via the circle of Willis or flow via the PComA only have higher incidence of brain infarction (13(85%)) and impaired hemodynamic parameters in the MCA(V mean-38cm/s, PI-0.69) than patients with collateral flow via the AComA (2 infarctions, Vmean-44cm/s, PI-0.77). Patients with reversed OphA could prove an additional risk for infarction. Patients with collateral flow via both AComA and PComA had less increased rCBV than those without.

Conclusion: Patients with collateral flow via the PComA and reversed OphA flow have more impaired hemodynamic parameters and a higher risk of brain infarctions than patients with collateral flow via the AComA. Complex use of TCCD, 3D TOF-MR-angiography and PWI gives all neccessary information about the type and hemodynamic parameters of collateral supply in high-grade ICA changes. *© 2008 Bull. Georg. Natl. Acad. Sci.*

Key words: *atherosclerosis, carotid artery, collateral supply.*

Severe atherosclerosis of the Internal Carotid artery (ICA) may lead to symptoms of transient retinal or cerebral ischemia and an increased risk of stroke. Therefore, knowledge of hemodynamic status may be important to elucidate the hemodynamic contribution of the symptoms.

In patients with occlusive disease of the internal carotid artery (ICA), collateral circulation is important to maintain adequate cerebral perfusion. The primary collateral pathway is the circle of Willis, with the possibility of redistributing flow from the contralateral ICA via the anterior communicating artery or from the vertebrobasilar arteries via the posterior communicating artery. Secondary collateral pathways include the external carotid artery via the ophthalmic artery and leptomeningeal anastomoses at the brain surface. When these collateral pathways are not adequate to maintain normal blood flow, vasodilatation of arterioles occurs and reduces cerebrovascular resistance for sustaining normal cerebral perfusion [1-3].

The results of several studies have demonstrated that adequate collateral circulation may prevent the development of hemodynamic failure. In contrast, findings from different studies showd that the presence of leptomeningeal collateral flow was associated with an increased risk of future ischemic stroke [1, 4-6]. The actual

contribution of the individual collateral pathways is difficult to assess and quantify. Assessment of cerebral hemodynamics can be performed with different techniques. The noninvasive evaluation of the collateral circulation status became possible only after introduction into clinical practice of several neuro-angioimaging tools, as Computed Tomography angiography (CTA), Magnetic-resonance angiography (MRA), Color Doppler sonography (CDUS), Transcranial Color Doppler (TCCD) modalities. All above-mentioned modalities give valuable information about the presence and efficiency of collateral supply in patients with ICA occlusive changes. Several studies have reported significant correlation (r=0.64) between TCD and MRI findings in the identification and evaluation of collateral supply sources in ICA occlusive changes patients [5-9].

The aim of our study was the relationship between collateral flow via different pathways and cerebral hemodynamic parameters in patients with unilateral high-grade internal carotid artery (ICA) changes.

MATERIALS AND METHODS: 71 consecutive symptomatic or asymptomatic subjects **(**44 men, 27 women) with occlusion or severe (>70%) stenosis of the extracranial segment of the ICA were included in the study. Patients ranged in age from 39 to 73 years (mean age, 62±11yers). 41 patients had severe ICA stenosis (>70%) and 30 had unilateral occlusion (Figs. 1,2).

All patients underwent a careful neurological and cardiological examination, ECG, transthoracic echocardiography, brain MRT, 3D TOF-MR-angiography, Color Doppler of extra-intracranial vessels to investigate collateral flow via the circle of Willis (anterior communicating (AComA) and posterior communicating (PComA) arteries) and via the ophtalmic artery (OA).

In all cases contralateral ICA was normal, or a mild stenosis (mean 31±24%) was noted. Percent of ICA stenosis was determined by the use of NASCET criteria.

The control group consisted of 20 subjects (mean age, 52 ± 9 years) without hemodynamically significant ICA obstruction**.**

MR imaging was performed by using a 1.5-T whole-body system (Magnetom Avanto Siemens Medical Systems, Erlangen, Germany). Flow territory imaging was achieved by using regional perfusion imaging sequences. Contrast enhancement by 5% Magnevist (Schering) was used. Evaluation of intracranial vessels was performed by Tof-fl3d-multiple-tra TR 56ms. TE 10.4ms. F.A.40 programs, for the extracranial vessels tof -fl2d-tra-traw-sat. TR 52ms, TE 10ms, F.A. 70 program was used.

Color Doppler ultrasonography (CDUS) of the extracranial carotid and vertebral arteries was performed on the unit Acuson CV 70 and Acuson X 300, with 5-10MHz linear probe. Carotid artery disease was assessed and defined according to standardized criteria. Transcranial Doppler sonography (TCD) was performed on the same Acuson CV 70 and Acuson X 300 unit 2.1MHZ probe.

Both ICA and VA diameter, peak systolic velocities, blood flow volumes (FV) were studied. The FV was calculated by the following formula FV= pDx(V/4)x60, in which *D* is the diameter of the artery, V- mean flow velocity.

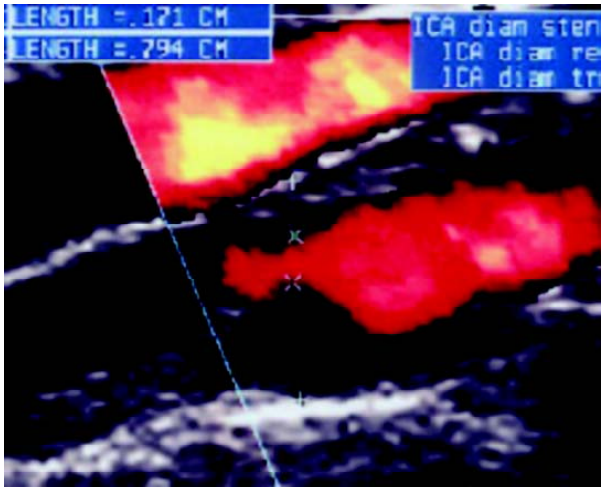

Fig. 1. Internal carotid artery severe (85%) stenosis. Color Doppler ultrasound. Severe narrowing of artery lumen.

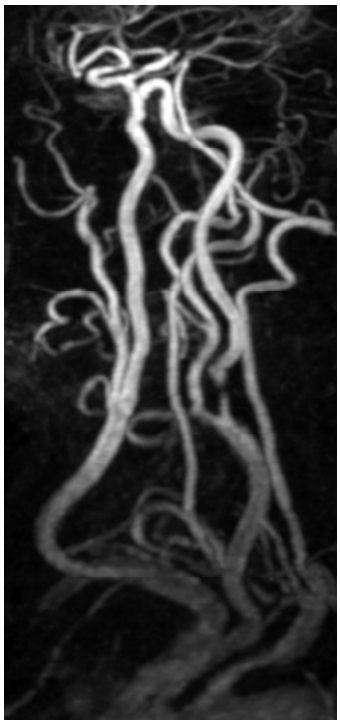

Fig. 2. Internal carotid artery severe (85%) stenosis. MR-angiography, gad-fl3d-tof-MIP. Interrupt of flow signal from ICA proximal site.

Examination of vessels of the circle of Willis was performed as described by Aaslid et al. Blood flow velocities (Vcm/s) in the middle, anterior, posterior cerebral arteries (MCA, ACA, PCA) and pulsatile indexes (PI) were measured. The patency of major collateral vessels, namely OA, anterior (ACoA), and posterior (PCoA) communicating arteries, was evaluated.

Results. Of 71 eligible patients, 50 were symptomatic in the vascular territory of the middle cerebral artery (MCA) ipsilateral to the carotid artery pathology. Of these, 21(30%) had a transient retinal or cerebral ischemia (TIA), and 29(41%) suffered from stroke (12 of them minor stroke, Rankin Scale score 1 or 2). Symptomatic side was left 40(56%) and right 31(44%) in cases. Patients with bilateral ICA high-grade changes were not included in the study. 21(30%) patients with nonfocal brain discirculation were defined as "asymptomatic" and were also enrolled.

According to the brain pathology site, infarctions were divided as cortical/subcortical, deep white-matter, and near to the basal ganglia. The main vascular supply areas and so-called "border-zone" areas were defined.

As is seen from Table 1, in asymptomatic patients MRT revealed only 2 cases of so-called "silent" infarctions: one in the MCA supply area, one in the PCA supply area. In this group prevalence of lacunar infarctions (8(38%)) and subcortical leukoencephalopathy (6(29%)) was noted.

In symptomatic (n=50) patients MRT found prevalence of cortical or cortical\subcortical infarctions of the MCA supply area – 13(26%), and 10(20%) cases of border-zone infarctions, of these 4 cortical (MCA\ACA or MCA\PCA supply area) and 6 deep white-matter watershed infarctions (Table 1).

In the majority of patients with MCA area border-zone infarction by TCCD significant decrease of blood flow at the MCA was revealed: V mean-29.8±8.4cm/s. Furthermore, there was also a significant decrease of pulsatile or resistive indexes: PI mean-0.68. In contrast, in patients with cortical infarctions, lacunar infarctions or subcrortical leucoencephalopathy hemodynamic changes were not so impaired: V mean MCA-37.5±9.2cm/s.

To assess collateral flow in the circle of Willis we studied the presence and character of collateral flow in the so-called primary pathway via Anterior communicating artery (AComA) and secondary pathway via ophthalmic artery (OA) (Table 2).

TCCD revealed presence of flow in the OA in all cases. In a majority of cases with ICA critical stenosis flow direction in the OA was antegrade. In contrast to ICA occlusion cases, prevalence of OA reversed flow was noted (22(76%)).

Table 1

Brain changes in patients with unilateral ICA high-grade stenosis or occlusion

	Asymptomatic n=21	Symptomatic n=50
Cortical infarction		
MCA	1(4.7%)	13(26%)
ACA	0	5(10%)
PCA	1(4.75)	1(2%)
Border-zone infarction	0	10(20%)
Lacunar infarction	8(3.8%)	9(18%)
White matter infarction	0	5(10%)
Leukoaraiosis	6(29%)	6(12%)
Cortical atrophia	5(24%)	1(2%)

Investigation revealed collateral flow via the primary collateral pathway, AComA in 56 cases, and only via the PComA in 15 (21%) cases.

TCCD showed that in 16(22%) patients with collateral flow via the AComA flow parameters in the ipsilateral MCA ware near to normal levels- V mean-44cm/s, PI-0.77. In this group only 2 cases on infarctions (1-cortical, 1- deep white matter infarction) were observed. In other 14 patients prevalence of multiple lacunar infarctions and subcrortical leucoencephalopathy were distinguished.

We found that patients without collateral flow via the circle of Willis or flow via the PComA only (n=15) have a high incidence of brain infarction. Despite that in 11(71%) cases PComA was patent, in 13(85%) patients presence of infarction was noted- 8(53%) border-zone and 5(33%) cortical infarctions of the MCA supply area. TCCD showed significant decrease of flow in the MCA(V mean-38cm/s, PI-0.69) in this group.

Table 2

Presence of collateral pathways in patients with unilateral ICA high-grade stenosis or occlusion

Circle of Willis	**Stenosis n=41**	**Occlusion n=30**
ACoA(-)	7(19%)	8(27%)
ACoA (+)	33(81%)	23(77%)
PcoA(-)	13(31%)	8(27%)
PcoA(+)	28(69%)	22(73%)
Ophthalmic artery		
Antegrade flow	23(56%)	8(24%)
Reversed flow	18(44%)	22(76%)

In other 2 patients with multiple lacunar infarctions and subcrortical leucoencephalopathy beneficial function of PComA and compensatory enhancement of flow in vertebral artery were noted. Patients with reversed flow at the ophthalmic artery could prove an additional risk for brain infarction. Of 40 patients with reversed flow at the ophthalmic artery, in 20(50%) cases brain infarctions were revealed of them 7(17.5%)-border-zone. In 31 patients with antegrade ophthalmic artery flow infarction was detected only in 9(33%) cases.

Hemodynamic status tended to be worse in this group of patients. In patients with noncompetent AComA or with revesed flow in the OA significant decrease of pulsatile index (PI) in the MCA was marked- 0.66-0.72(mean 0.69±.27)

TCCD showed that in patients with antegrade ACA flow, flow velocity at the ACA (V ACA) was normal or slightly low (Vmean-44cm/s, range-34-53cm/s), hemispheric ratio of velocities in the ACA and MCA was normal- V ACA / V MCA=0.76; but where ACA flow was reversed, the flow velocity -V ACA increased (Vmean-67.5cm/s, range-59-73cm/s) and hemispheric ratio tended to be abnormal- V ACA / V MCA=1.35.

In contrast to MCA, in ACA correlation of pulsative index changes with type of collateral supply and infarction site were not revealed.

In order to assess the circle of Willis anatomy all patients underwent 3D time-of-flight (TOF) MR-angiography. Investigation revealed the presence of (classic)circle of Willis in 38(54%) patients; however, it must be mentioned that in 10 cases one of the PComA was projected by thin (0.8-1.2mm) line and was interpreted as hypoplasia of PComA (Figs. 3,4).

One of the main aims of our study was to determine whether there are changes in the hemodynamic parameters in the contralateral to occlusion ICAs and vertebral arteries.

Table 3 shows the comparison of the mean ±SD values for diameter BFVs of both ICAs and VAs and side-to-side differences for the patients with unlilateral ICA occlusion.

As is seen from Table 3, in cases of ICA occlusion (n=30), significant dilatation of the contralateral ICA (mean 7.5±2,4mm) and enhancement of BFV (mean 428±45ml/min) was noted; this seems about 60% higher compared to the control group ($p<0.05$). We did not find significant side-to-side difference in the diameters and BFVs of the ICAs and diameters of the VAs.

In patients with unilateral ICA occlusion the mean BFV in the ipsilateral VA was higher than that on the contralateral side (mean 108 ±38ml/min. vs. 96±32ml/min). Furthermore, there was also significant increase of BFV as compared to the control group.

In all cases of the occlusion of the ICA, revealed by CDUS diagnosis, was confirmed by 2D time-of-flight (TOF) MR-angiography (100% confirmation). In all cases of ICA occlusion dilatation of the contralateral ICA lumen and enhancement of the signal intensity as in the contralateral ICA and in the system of posterior circulation, as a sign of recruitment of posterior circulation flow was observed (Fig. 5).

Conclusion. The cerebral hemodynamic status of patients with severe occlusive carotid artery disease has been reported to play a significant role in the occur-

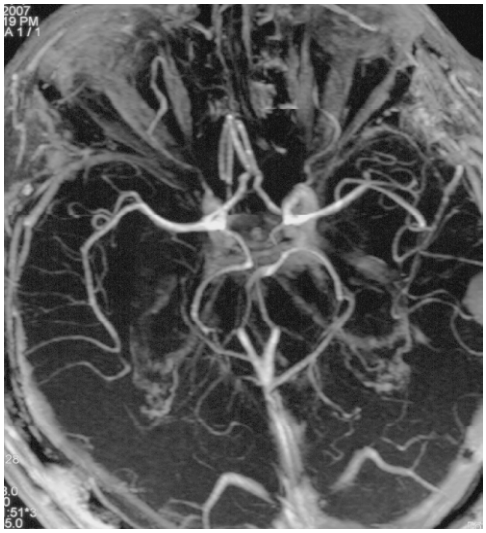

Fig. 3. Competent (closed) circle of Willis. MR-angiography, gad-fl3d-tof-MIP.

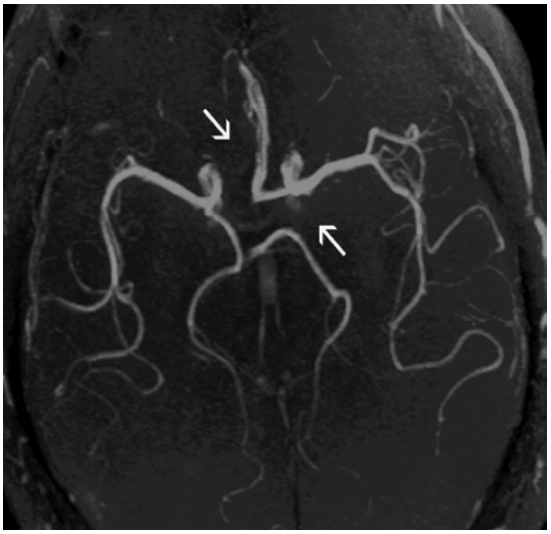

Fig. 4. Noncompetent circle of Willis. MR-angiography, gad-fl3d-tof-MIP. Right Anterior Commucating artery and left Posterior Commucating artery are absent.

Table 3

Values of diameters and blood flow volumes in the ICAs and VAs in cases of unilateral ICA occlusion

	Diameter, mm				Q ml/min			
	ICA dex	**ICA sin**	**VA dex**	**VA sin**	**ICA dex**	**ICA sin**	**VA dex**	**VA sin**
Control (n=25)	*5.6±2.3*	*5.82±2.9*	*3.38±1.4*	*3.56±1.8*	*252±56*	*266±73*	*88±32*	*97± 41*
ICA dex occlusion	_	7.8± 3.2	4.3±2.0	3.9±1.8	_	454±52	106±44	98 ±28
ICA sin occlusion	7.4± 3.4	_	3.7±1.6	4.4±1.8	414±32	_	94±36	115± 42

rence of stroke. Two mechanisms of cerebral ischemia in this disease have been identified: vascular occlusion from an embolism or propagating thrombus from an atherosclerotic plaque of the carotid artery (artery-to-artery embolism), and watershed or border-zone ischemia caused by critically reduced perfusion pressure (hemodynamic stroke) [2, 4, 10, 11].

Patients with complete carotid artery occlusion may show no evidence of intracranial hemodynamic compromise, because collateral circulation compensates for the decrease in cerebral blood flow (CBF). When these collateral pathways are not adequate to maintain normal blood flow, vasodilatation of arterioles occurs and reduces cerebrovascular resistance in order to sustain normal cerebral perfusion. This phenomenon is known as cerebrovascular autoregulation. Compromised CBF plays an important role in causing ipsilateral ischemic events in patients with occlusion of the internal carotid artery (ICA). As a matter of fact, cerebral ischemia frequently develops in areas of collateral pathways for blood supply from bordering vascular territories. As a result, our study showed prevalence of cortical (19/38%) and border-zone infarctions (10/20%) than in cases of ICA occlusion [3, 8, 12, 13].

In our opinion, somewhat frequent cases of deep white matter infarctions may be the result of hypoperfusion by carotid stenoocclusive disease. The reason of the deep white matter diffuse and focal changes may be hypoperfusion of corticomedullar arteries and transformation of their supply area in the "deep border-zone" [3, 10, 12].

In fact, there is evidence that cerebral hemodynamic status can predict the outcome of ICA occlusion. Anterior and posterior communicating arteries are considered the primary collateral pathways; the ophthalmic artery (OA) and blood flow via leptomeningeal vessels are considered the secondary pathways.

Our study shows that the most important compensatory path in patients with unilateral ICA occlusion is collateral flow from contralateral ICA via AComA. CDUS shows that the mean BFV in the contralateral ICA is increased almost by 55%. The results of our study are in overall agrement with previously published results. On the other hand, our data show that the mean net flow volume in the VAs is increased by almost 18% when compared with the control ones, confirming that the

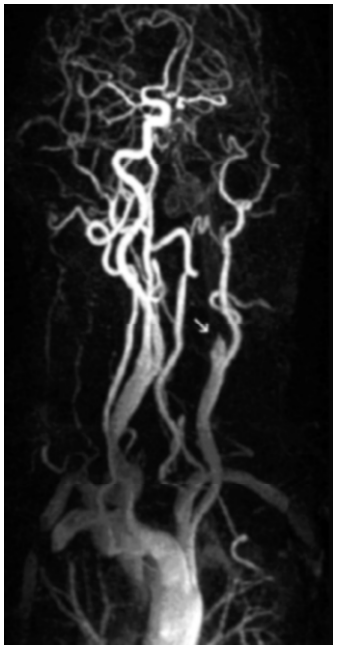

Fig. 5. Left Internal carotid artery occlusion. MR-angiography, gad-fl3d-tof-MIP. Absence of flow signal from ICA. Compensatory enhancement of signal intensity at the contralateral carotid artery and vertebrobasilar system.

vertebrobasilar circulation is also important in collateral supply in cases of ICA occlusion. Our data suggest that the anterior circle is a preferential mode of collateral supply in patients with ICA unilateral occlusion. Patients with collateral flow via the anterior communicating artery have less impaired hemodynamic parameters than those with collateral flow via the PComA (85% infarction in group without AComA vs. 12% infarction in patients, with patent AComA). Secondary collateral pathways include the external carotid artery via the ophthalmic artery. Other leptomeningeal or extracerebral anastomoses play a minor role in brain compensatory supply [13-15].

Our study shows that patients with reversed ophthalmic artery flow have more impaired hemodynamics parameters than those without. In patients with OA reversed flow brain infarction was revealed in 51% of cases, compared to 32% in patients with antegrade OA flow. Hemodynamic status tended to be worse in patients with reversed flow of the ophthalmic artery. This finding corresponds with the hypothesis that the OA is a secondary collateral pathway, which is only recruited when the primary pathways fall short [14-17].

Our study allows us to conclude that adequate collateral circulation may prevent the development of hemodynamic failure. Collateral circulation via the AComA or both AComA and PComA is a sign of well-compensated hemodynamic status. However, recruitment of the PComA as the only primary pathway or recruitment of the OA can be regarded as an indication of an impaired perfusion status of the brain.

ექსპერიმენტული მედიცინა

თავის ტვინის კეროვანი ცვლილებები და ჰემოდინამიკური პარამეტრები კოლატერალური მიმოქცევის მდგომარეობასთან მიმართებაში

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ავტორებმა შეისწავლეს თავის ტვინის კოლატერალური მიმოქცევის მდგომარეობა პაციენტებში შიგნითა საძილე არტერიის ცალმხრივი კრიტიკული სტენოზითა და ოკლუზიით. შესწავლილ იქნა 71 პაციენტი (41(58%)—კრიტიკული სტენოზი, 30(42%)—ოკლუზია). პაციენტთა ასაკი მერყეობდა 41-78წწ. ფარგლებში (საშუალო ასაკი-62±12წ). 40(56%) შემთხვევაში დაზიანებული იყო მარცხენა, ხოლო 31(44%) შემთხვევაში მარჯვენა შიგნითა საძილე არტერია.

ყველა პაციენტს ჩაუტარდა რუტინული ნევროლოგიური კვლევა, თავის მაგნიტურ-რეზონანსული ტომოგრაფია, მაგისტრალური ექსტრაკრანიული და ინტრაკრანიული სისხლძარღვების დუპლექს-სკანირება და 3D TOF მაგნიტურ-რეზონანსული ანგიოგრაფია. 50(70%) შემთხვევაში გამოვლინდა სიმპტომური ცერებრალური იშემია. სიმპტომატურ პაციენტებში ჭარბობდა შუა ცერებრული არტერიის აუზის კორტიკალური ინფარქტები 13(26%) და ე.წ. "მოსაზღვრე ზონის" ინფარქტები-10(20%). შიგნითა საძილე არტერიის ცალმხრივი ოკლუზიის პირობებში ადგილი ჰქონდა კონტრალატერალური შიგნითა საძილე არტერიის კომპენსატორულ დილატაციას და ნაკადის მოცულობითი სიჩქარის დაახლოებით 60%-ით კომპენსატორულ მომატებას, ასევე ნაკადის მოცულობითი სიჩქარის დაახლოებით 18%-ით მატებას ვერტებრულ არტერიებში.

პაციენტებში, სადაც არ მოქმედებდა წინა შემაერთებელი არტერია, აღინიშნება როგორც ნაკადის ინტენსივობის დაქვეითება იფსილატერალურ ინტრაკრანიულ კაროტიდულ აუზში (V mean-38cm/s, PI-0.69), ასევე საგრძნობლად მეტი ინფარქტის შემთხვევები(13(85%); მოფუნქციონირე წინა შემაერთებელი არტერიის მქონე პირებში — 2 ინფარქტი, Vmean-44cm/s, PI-0.77.

ყოველივე ზემოთ აღნიშნულიდან შეგვიძლია ვფარაუდოთ, რომ წინა შემაერთებელი არტერია წარმოადგენს ექსტრაკრანიული სტენო-ოკლუზიური პროცესის საკომპენსაციო ყველაზე მნიშვნელოვან კოლატერალს. თვალბუდის ანასტომოზი ამ მხრივ შედარებით ნაკლებეფექტურია. პაციენტებში, რომლებთანაც არ აღინიშნება ვილიზიის წრის შემაერთებელი არტერიების ფუნქციონირება, ან სადაც ფუნქციონირებს მხოლოდ უკანა შემაერთებელი არტერია, ვლინდება ინფარქტის (უპირატესად მოსაზღვრე ზონის) განვითარების მაღალი რისკი. ასევე შედარებით არაკეთილსაიმედო ფაქტორად არის მიჩნეული რეტროგრადული ნაკადის გამოვლენა თვალბუდის არტერიაში. მაგნიტურ-რეზონანსული ტომოგრაფიის, მრ-ანგიოგრაფიისა და ექსტრა-ინტრაკრანიული სისხლძარღვების დუპლექს-სკანირების კომპლექსური გამოყენება იძლევა პრაქტიკულად სრულ ინფორმაციას კოლატერალური მიმოქცევის ფუნქციური სტატუსის შესახებ საძილე არტერიის მნიშვნელოვანი სტენოზების შემთხვევაში.

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