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## Многоцентровое исследование: исходы каротидной эндартерэктомии в зависимости от конфигурации Виллизиева круга

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### АННОТАЦИЯ

**Обоснование.** Виллизиев круг (ВК) является важной сетью коллатералей, способной компенсаторно перераспределить гемодинамическую нагрузку. Ряд исследований показал, что приблизительно в 50–90% случаев ВК разомкнут, а количество отсутствующих сегментов коррелирует с низкой толерантностью к ишемии головного мозга, вызванной пережатием внутренних сонных артерий (ВСА). В настоящее время существует дефицит исследований, посвященных связи различных конфигураций ВК с риском ишемического повреждения головного мозга (ГМ).

**Цель.** Анализ непосредственных результатов эверсионной каротидной эндартерэктомии (КЭЭ) у пациентов с разными типами конфигурации строения Виллизиева круга (ВК).

**Материалы и методы.** В данное многоцентровое ретроспективное исследование за период с 2010–2020 гг. было включено 641 пациент с гемодинамически значимыми стенозами (ВСА). Всем больным выполнялась мультиспиральная компьютерная томография с ангиографией экстракраниальных артерий и артерий ВК. В зависимости от вариантов строения ВК были сформированы 6 групп пациентов: группа 1 (64,9%, n = 416) — замкнутая задняя часть (ЗЗЧ) — при наличии задней соединительной (ЗСА) и P1 сегмента задней мозговой (ЗМА) артерий; группа 2 (27%, n = 173) — промежуточный тип строения задней части (ПЗЧ) — при гипоплазии ЗСА или ЗМА; группа 3 (8,1%, n = 52) — незамкнутая задняя часть (НЗЧ) — при отсутствии ЗСА или ЗМА; группа 4 (85,95%, n = 551) — замкнутая передняя часть (ЗПЧ) — при наличии передней соединительной артерии (ПСА) и A1 сегмента передней мозговой артерии (ПМА); группа 5 (7,95%, n = 51) — промежуточный тип строения передней части (ППЧ) — при гипоплазии ПСА или ПМА; группа 6 (6,1%, n = 39) — незамкнутая передняя часть (НПЧ) — при отсутствии ПСА или ПМА. Для оценки компенсаторных возможностей головного мозга (ГМ) всем пациентам проводилось измерение ретроградного давления во ВСА и интраоперационная церебральная оксиметрия.

**Результаты.** В послеоперационном периоде был зафиксирован 1 летальный исход в группе 4 (ЗПЧ) по причине формирования геморрагической трансформации в зоне ишемического инсульта, на фоне развития гиперперфузионного синдрома. Наибольшее количество ишемических инсультов по кардиоэмболическому подтипу было диагностировано в бассейне ПМА при наличии нестабильной атеросклеротической бляшки: группа 1 (ЗЗЧ) — 0%; группа 2 (ПЗЧ) — 0%; группа 3 (НЗЧ) — 0,24%, n = 1; группа 4 (ЗПЧ) — 0,18%, n = 1; группа 5 (ППЧ) — 1,96%, n = 1; группа 6 (НПЧ) — 5,10%, n = 2; p > 0,9999. Вероятная причина — эмболизация на фоне подъема артериального

давления перед пережатием ВСА. В свою очередь, большинство ишемических инсультов по гемодинамическому подтипу развилось в бассейне ЗМА: группа 1 (ЗЗЧ) — 0%; группа 2 (ПЗЧ) — 1,73%, n = 3; группа 3 (НЗЧ) — 3,80%, n = 2; группа 4 (ЗПЧ) — 0,18%, n = 1; группа 5 (ППЧ) — 0%; группа 6 (НПЧ) — 2,56%, n = 1;  $p > 0,9999$ . Данная закономерность совпала с наибольшим количеством пациентов с наличием ВК по типу ПЗЧ и НЗЧ среди всех незамкнутых вариантов строения.

**Заключение.** Показатели ретроградного давления во ВСА и интраоперационной церебральной оксиметрии не всегда демонстрируют необходимость в установке временного шунта. В виду разомкнутости ВК перераспределение кровотока идет с формированием зон гипо- и гиперперфузии, что вызывает ишемические изменения в веществе головного мозга (ГМ). Таким образом, для поддержания адекватной церебральной гемодинамики, смягчения эффекта гипо- и гиперперфузии, снижения риска развития ишемического инсульта, в показаниях к применению временного шунта необходимо рассматривать разомкнутый тип строения ВК.

**Ключевые слова:** каротидная эндартерэктомия; эверсионная каротидная эндартерэктомия; ретроградное давление; церебральная оксиметрия; разомкнутый Виллизиев круг; нестабильная атеросклеротическая бляшка; SYNTAX; временный шунт; геморрагическая трансформация

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# Multicenter study: outcomes of carotid endarterectomy depending on configuration of circle of Willis

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## ABSTRACT

**BACKGROUND:** The circle of Willis (CW) is an important network of collaterals that provide compensatory redistribution of hemodynamic load. Several studies showed that the CW is open in approximately 50%–90% of cases, and the number of missing segments correlates with low brain tolerance to ischemia in internal carotid artery (ICA) compression. Currently, studies dedicated to the relationship of different configurations of CW with the risk of ischemic brain damage.

**AIM:** The analyze the immediate results of eversion carotid endarterectomy (CEA) in patients with different configurations of the structure of the CW.

**MATERIALS AND METHODS:** We included 641 patients with hemodynamically significant stenosis of the internal carotid arteries (ICA) in a study period from 2010 to 2020. All patients underwent multispiral computed tomography with angiography of the extracranial and CW arteries. Based on the structural variants of the CW, six groups of patients were studied: group 1 (64.9%, n = 416) — closed posterior part (CPP) with the existence of posterior communicative artery (PCA) and P1 segment of the posterior cerebral artery (PCerA); group 2 (27%, n = 173) — an intermediate structure of the posterior part (IPP) with hypoplasia of the PCA or PCerA; group 3 (8.1%, n = 52) — open posterior part (OPP) with the absence of PCA or PCerA; group 4 (85.95%, n = 551) closed anterior part (CAP) with the presence of the anterior communicating artery (ACA) and A1 segment of the anterior cerebral artery (ACerA); group 5 (7.95%, n = 51) — an intermediate structure of the anterior part (IAP) with hypoplasia of ACA or ACerA; group 6 (6.1%, n = 39) — open anterior part (OAP) with the absence of ACA or ACerA. To assess the compensatory potentials of the brain, all patients underwent measurement of the retrograde pressure in the ICA and intraoperative cerebral oximetry.

**RESULTS:** In the postoperative period, 1 death was recorded in group 4 (CAP) due to a hemorrhagic transformation in the zone of ischemic stroke, on the background development of hyperperfusion syndrome. The largest number of ischemic strokes of the cardioembolic subtype was diagnosed in the ACerA territory in the presence of an unstable atherosclerotic plaque: group 1 (CPP) 0%; group 2 (IPP) — 0%; group 3 (OPP) — 0.24%, n = 1; group 4 (CAP) — 0.18%, n = 1; group 5 (IAP) — 1.96%, n = 1; group 6 (OAP) — 5.1%, n = 2; p > 0.9999. The probable cause was embolization against the background

increase in the arterial pressure before ICA clamping. In turn, the majority of ischemic strokes of the hemodynamic subtype developed in the territory of PCerA: group 1 (CPP) — 0%; group 2 (IPP) — 1.73%, n = 3; group 3 (OPP) — 3.8%, n = 2; group 4 (CAP) — 0.18%, n = 1; group 5 (IAP) — 0%; group 6 (OAP) — 2.56%, n = 1;  $p > 0.9999$ . This pattern coincided with the largest number of patients with CW of the IPP and OPP types among all open variants of the structure.

**CONCLUSION:** Parameters of retrograde pressure in the ICA and intraoperative cerebral oximetry do not always demonstrate the need for a temporary shunt (TS). Due to the opened structure of CW, the redistribution of blood flow occurs with the formation of zones of hypo- and hyperperfusion, causing ischemic alterations in the brain matter. Thus, in order to maintain adequate cerebral hemodynamics, to mitigate the effect of hypo- and hyperperfusion, and reduce the risk of ischemic stroke, the open variant of the CW structure should be considered as an indication for a TS.

**Keywords:** *carotid endarterectomy; eversion carotid endarterectomy; retrograde pressure; cerebral oximetry; open circle of Willis; unstable atherosclerotic plaque; SYNTAX; temporary shunt; hemorrhagic transformation*

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## BACKGROUND

Carotid endarterectomy (CEA) as an operation of choice in cases of hemodynamically significant internal carotid artery (ICA) stenosis gained the status of a “clique” in modern vascular surgery [1–3]. Arterial compression to perform arteriotomy and atherosclerotic plaque (ASP) removal from the carotid bifurcation was a constructive solution by M.E. DeBakey in 1953 and is still used today [4, 5]. Here the effectiveness of the compensation mechanism of the cerebral blood flow during ICA compression largely reflects the ability of the brain to adapt to artificial ischemia, which is also associated with the risk of a postoperative acute cerebrovascular event (stroke)/transient ischemic attack (TIA) [6–9]. Both intraoperative hypoperfusion and hyperperfusion of the brain after clamp removal from the ICA are critical triggers that increase the probability of ischemic stroke and accompany CEA [10, 11]. In this situation, the circle of Willis (CW) is an important network of collaterals that provide compensatory redistribution of hemodynamic load [10, 12, 13]. However, several studies showed that the CW is open in approximately 50%–90% of cases, and the number of missing segments correlates with low brain tolerance to ischemia in ICA compression [13, 14]. Currently, studies dedicated to the relationship of different configurations of CW with the risks for stroke/TIA development are limited. Thus, some works demonstrate the growth of postoperative stroke/TIA in proportion to the evidence of the CW open structure [12–15].

**Aim** — to assess hospital results of CEA in patients with different CW configurations.

## MATERIALS AND METHODS

From 2010 to 2030, 1,584 eversion CEA was performed in patients with hemodynamically significant ICA stenoses, wherein 641 patients underwent multispiral computed tomography with angiography (MSCT-AG) of the extra- and intracranial arteries. This group of patients was included in the present, cohort, comparative, retrospective open-ended study. The MSCT-AG data of the intracranial arteries identified the following types of CW structures:

**type 1:** closed posterior part (CPP) with the posterior communicating artery (PCA) and P1 segment of the posterior cerebral artery (PCerA);

**type 2:** intermediate type of structure of the posterior part (IPP) with hypoplasia of the PCA or PCerA;

**type 3:** open posterior part (OPP) without the PCA or PCerA;

**type 4:** closed anterior part (CAP) with the existence of the anterior communicating artery (ACA) and A1 segment of the anterior cerebral artery (ACerA);

**type 5:** intermediate type of structure of the anterior part (IAP) with hypoplasia of ACA or ACerA; and

**type 6:** open anterior part (OAP) without the ACA or ACerA.

The sample was divided into six groups based on the types of CW structures (Table 1).

**Table 1.** Formed Groups of Patients Depending on Circle of Willis Type

Type of Structure of Circle of Willis	Group	n	%
Closed posterior part (CPP)	1	416	64.9
Intermediate type of structure of posterior part (IPP)	2	173	27.0
Open posterior part (OPP)	3	52	8.1
Closed anterior part (CAP)	4	551	86.0
Intermediate type of structure of anterior part (IAP)	5	51	8.0
Open anterior part (OAP)	6	39	6.1

**Inclusion criteria** were indications for CEA based on the current national recommendations.

**Exclusion criteria:**

1. Contraindications for CEA;
2. Vertebral and subclavian artery stenoses; and
3. Existence of subclavian steal syndrome.

In the preoperative period, patients underwent screening color duplex scanning of the brachiocephalic bed, lower limb arteries, aortic arch (with a linear

sensor of 7–7.5 MHz frequency), and heart (with the sector probe of 2.5–4 MHz frequency) with use of Acuson 128XP (Acuson, USA) and Sonos 2500 apparatuses (Hewlett Packard, USA) to conclude the existence of multifocal atherosclerosis (MFA). The topographic characteristics of an ASP in the ICA were visualized by MSCT-AG. The degree of stenosis was determined according to the North American Symptomatic Carotid Endarterectomy Trial classification. “Unstable” ASP

was defined based on the current national recommendations and the “classification of carotid stenoses based on auscultative, ultrasound, and angiographic features”, including such characteristics as “undermined”, “with decay”, “with ulceration”, and “with intraplaque hemorrhage with or without destruction of the cap and atheroma”.

Coronary vessel lesion was assessed by coronary angiography using Innova 2100 angiographic unit (General Electric, USA). The severity of coronary atherosclerosis was calculated using the interactive SYNTAX Score calculator ([www.syntaxscore.com](http://www.syntaxscore.com)).

Compensation capacities of the cerebral blood flow during CEA were evaluated in the following way. Arterial pressure (AP) was pharmacologically elevated to 190/100 mmHg, heparin was intravenously introduced at a dose of 5,000 Units, and arteries were compressed. Invasive measurement of the retrograde pressure in the ICA was performed. At the level of AP <60% of the systemic pressure, a temporary shunt (TS) was applied. Intraoperatively, cerebral oximetry of all patients was performed using Invos 5100 C apparatus (Medtronic, США). TS was inserted when oximetry parameters were reduced to <30% of the initial level.

Control points were understood as the development of such adverse cardiovascular events as a lethal outcome, myocardial infarction (MI), stroke/TIA, thrombosis/restenosis in the reconstruction zone, bleeding of 3b type and higher on the Bleeding Academic Research Consortium scale, and combined endpoint (death + ACVE/TIA + MI).

The study was performed following the standards of Good Clinical Practice and principles of the Declaration of Helsinki.

The type of distribution was determined using the Kolmogorov–Smirnov test. Comparison between groups was performed using the Kruskal–Wallis test and Pearson’s chi-squared test. Differences were considered statistically significant at  $p < 0.05$ . Pair-wise comparison of groups was performed using the Mann–Whitney and Pearson’s chi-squared tests with Yates’ correction for differences between all groups in one of the parameters or a p-value close to 0.05. Study results were processed using the Graph Pad Prism ([www.graphpad.com](http://www.graphpad.com)) application software package.

Clinical and demographic characteristics revealed that most patients were males of middle age with stable angina. Every fifth patient had a history of MI or stroke/TIA. MFA was verified in a third of cases. In every tenth patient diabetes mellitus was determined. The severity index EuroSCORE II revealed no intergroup differences (Table 2).

## RESULTS

All angiographic parameters of patients were compared, both cerebral and coronary. SYNTAX index of severity of lesion of coronary arteries corresponded to a low level (Table 3).

TS was mostly needed in the case of CW open variants (OPP, OAP). However, in the overwhelming majority of cases, TS was already inserted during the operation on average in  $12.6 \pm 3.5$  min after ICA compression, in connection with oximetry parameter reductions by >30% of the initial level (Table 4).

The hospital follow-up period recorded one lethal outcome in group 4 (CAP). The patient had 99% symptomatic stenosis of the left ICA, without signs of unstable ASP, with CW closed structure. The patient suffered a stroke of ischemic type in the territory of the left medial cerebral artery (MCerA) 30 days before. At hospital discharge, neurological deficit fully regressed (on admission it was 6 points on the National Institutes of Health Stroke Scale). The revascularization time was identified by subjective categorical reasons given by the patient himself (harvesting in the autumn period). CEA was without peculiarities, the time of ICA compression was 27 min. After CEA termination, the patient’s consciousness was at the level of sopor. The emergency MSCT-AG of the brain revealed hemorrhagic transformation in the zone of ischemic stroke in the territory of the left MCerA. The patient was consulted by a neurosurgeon. This condition was not an indication of surgical intervention. In the intensive care unit, conservative management tactics for the patient were chosen. On the next day after CEA, the patient’s consciousness dropped to coma II. The control MSCT of the brain revealed negative dynamics in the form of brain edema buildup. After the second consultation with the neurosurgeon, decompression craniotomy was performed. In the evening, the hyperthermic syndrome was recorded ( $t = 38.0^\circ\text{C}$ ). Despite the conducted therapy, on day 5 after CEA, biological death was registered. Autopsy results revealed cardiac arrest with underlying brain edema and brainstem herniation as the cause of death. Thus, the actual trigger of the entire pathological chain of events was hyperperfusion syndrome that resulted from blood flow restart after the clamp removal from the ICA.

The parameters of stroke/TIA, MI, CEP, hemorrhagic complications were compared in all groups (Table 5).

## DISCUSSION

The study of F.B. Shukurov et al. revealed that the existence of the open CW is connected with the



**Table 2.** Clinical and Demographic Characteristics of Study Groups

Parameter	Group 1 (CPP)	Group 2 (IPP)	Group 3 (OPP)	Group 4 (CAP)	Группа 5 (IAP)	Group 6 (OAP)	P
Age, M ± m, years	63.5 ± 4.1	62.6 ± 5.1	66.4 ± 5.0	61.4 ± 6.1	62.9 ± 5.2	62.5 ± 5.7	> 0.9999
Male gender, n (%)	317 (76.2)	108 (62.4)	35 (67.3)	395 (71.6)	(66.6)	25 (64.1)	> 0.9999
1–2 functional class angina pectoris, n (%)	365 (87.7)	117 (67.6)	38 (73.0)	407 (73.8)	40 (78.4)	29 (74.3)	> 0.9999
Postinfarction cardiosclerosis, n (%)	74 (17.7)	42 (24.2)	12 (23.0)	92 (16.6)	11 (21.5)	8 (20.5)	> 0.9999
Diabetes mellitus, n (%)	39 (9.3)	21 (12.1)	3 (5.7)	51 (9.2)	5 (9.8)	4 (10.2)	> 0.9999
Chronic obstructive pulmonary disease, n (%)	5 (1.2)	3 (1.7)	0	6 (1.0)	0	1 (2.5)	> 0.9999
chronic renal insufficiency, n (%)	8 (1.9)	4 (2.3)	2 (3.8)	13 (2.3)	3 (5.88)	2 (5.1)	> 0.9999
Multifocal atherosclerosis with lesion of three arterial beds, n (%)	81 (19.4)	48 (27.7)	21 (40.3)	94 (17.0)	13 (25.4)	23 (58.9)	3 vs 1: 0.0088 6 vs 1: < 0.0001 4 vs 2: 0.0454 6 vs 2: 0.0003 4 vs 3: 0.0015 6 vs 4: < 0.0001 6 vs 5: 0.0021
Left ventricle ejection fraction, M ± m, %	59.7 ± 6.3	57.8 ± 6.9	59.6 ± 6.2	59.1 ± 6.7	58.8 ± 7.4	59.2 ± 6.3	> 0.9999
Aneurysm of left ventricle, n (%)	1 (0.2)	0	0	1 (0.1)	0	0	> 0.9999
EuroSCORE II, M ± m	4.2 ± 2.1	4.3 ± 2.5	4.7 ± 2.2	5.1 ± 2.6	4.5 ± 2.2	4.7 ± 2.9	> 0.9999
Percutaneous coronary intervention in history, n (%)	69 (16.5)	47 (27.1)	13 (25)	89 (16.1)	12 (23.5)	10 (25.6)	2 vs 1: 0.0411 4 vs 2: 0.0181
Coronary artery bypass surgery, n (%)	7 (1.6)	1 (0.5)	0	3 (0.5)	0	1 (2.5)	> 0.9999
Stroke/ transient ischemic attack in history, n (%)	86 (20.6)	35 (20.2)	41 (78.8)	112 (20.3)	16 (31.3)	14 (35.8)	3 vs 1: < 0.0001 3 vs 2: < 0.0001 4 vs 3: < 0.0001 5 vs 3: < 0.0001 6 vs 3: < 0.0001

**Table 3.** Angiographic Characteristics of Study Groups

Parameter	Group 1 (CPP)	Group 2 (IPP)	Group 3 (OPP)	Group 4 (CAP)	Группа 5 (IAP)	Group 6 (OAP)	P
% of internal carotid artery stenosis	86.3 ± 5.7	86.2 ± 5.5	88.2 ± 3.5	87.0 ± 5.5	86.9 ± 5.2	86.2 ± 5.3	> 0.9999
Unstable atherosclerotic plaque, n (%)	98 (23.5)	56 (32.3)	7 (13.4)	121 (21.9)	9 (17.6)	5 (12.8)	3 vs 2: 0.0688 4 vs 2: 0.0694
Contralateral occlusion of internal carotid artery, n (%)	94 (22.6)	36 (20.8)	6 (11.5)	118 (21.4)	13 (25.5)	2 (5.1)	> 0.9999
Hemodynamically significant internal carotid artery stenoses on both sides, n (%)	47 (11.3)	15 (8.7)	5 (9.6)	78 (14.1)	6 (11.7)	6 (15.4)	> 0.9999
SYNTAX taking into account revascularization of myocardium in history, M ± m	6.8 ± 3.4	7.1 ± 4.2	7.8 ± 5.3	6.8 ± 4.8	7.8 ± 2.4	6.7 ± 3.1	> 0.9999

**Table 4.** Intraoperative Characteristics of Study Groups

Parameter	Group 1 (CPP)	Group 2 (IPP)	Group 3 (OPP)	Group 4 (CAP)	Группа 5 (IAP)	Group 6 (OAP)	P
Use of temporary shunt (in total), n (%)	117 (28.1)	69 (39.9)	46 (88.5)	149 (27.0)	17 (33.3)	36 (92.3)	2 vs 1: 0.0909 3 vs 1: < 0.0001 6 vs 1: < 0.0001 3 vs 2: < 0.0001 4 vs 2: 0.0279 6 vs 2: < 0.0001 4 vs 3: < 0.0001 5 vs 3: < 0.0001 6 vs 4: < 0.0001 6 vs 5: < 0.0001
Reduction of cerebral oximetry parameters by >30% from the initial level that required temporary shunt installation, n (%)	0	26 (15.0)	38 (73.0)	1 (0.2)	1 (2.0)	33 (84.6)	2 vs 1: < 0.0001 3 vs 1: < 0.0001 6 vs 1: < 0.0001 3 vs 2: < 0.0001 4 vs 2: < 0.0001 5 vs 2: 0.0322 6 vs 2: < 0.0001 4 vs 3: < 0.0001 5 vs 3: < 0.0001 6 vs 4: < 0.0001 6 vs 5: < 0.0001
Duration of internal carotid artery compression, min	26.5 ± 4.2	27.1 ± 6.1	25.4 ± 4.0	26.1 ± 4.4	26.4 ± 3.3	27.1 ± 5.3	> 0.9999

**Table 5.** Hospital Results in Study Groups

Parameter	Group 1 (CPP)	Group 2 (IPP)	Group 3 (OPP)	Group 4 (CAP)	Группа 5 (IAP)	Group 6 (OAP)	P
Death, n (%)	0	0	0	1 (0.2)	0	0	> 0.9999
Stroke/transient ischemic attack (in total), n (%)	0	3 (1.7)	3 (5.7)	2 (0.4)	1 (2.0)	3 (7.7)	2 vs 1: 0.7 3 vs 1: 0.0007 6 vs 1: <0.0001 6 vs 2: 0.0073 4 vs 3: 0.0017 6 vs 4: <0.0001 6 vs 5: 0.0777
	In total: 6 (0.9)			In total: 6 (0.9)			1.0
Stroke/transient ischemic attack with presence of unstable atherosclerotic plaque, n (%)	0	0	1 (0.2)	1 (0.2)	1 (2.0)	2 (5.1)	6 vs 1: <0.0001 6 vs 2: <0.0001 6 vs 4: <0.0001
	In total: 1 (0.2)			In total: 4 (0.7)			0.37
Stroke/transient ischemic attack without unstable atherosclerotic plaque, n (%)	0	3 (1.7)	2 (3.8)	1 (0.2)	0	1 (2.6)	3 vs 1: 0.0059 4 vs 3: 0.0092
	In total: 5 (0.8)			In total: 2 (0.3)			0.45
Myocardial infarction, n (%)	1 (0.2)	0	0	1 (0.1)	0	0	> 0.9999
3b and higher type of bleeding on Bleeding Academic Research Consortium scale	3 (0.7)	0	1 (1.9)	5 (0.9)	0	0	> 0.9999
Combined endpoint (death + stroke/ transient ischemic attack + myocardial infarction)	1 (0.2)	3 (1.7)	3 (5.7)	4 (0.7)	1 (1.9)	3 (7.6)	3 vs 1: 0.0072 6 vs 1: 0.005 6 vs 2: 0.0269 4 vs 3: 0.0186 6 vs 4: 0.0014



development of intraoperative hypotension and postoperative persistent reduction of the AP, which also lead to hypoperfusion of the brain and development of stroke/TIA [16]. R.S. Tarasov et al. found that the open CW is a predictor of the development of hospital complications after CEA in patients with MFA ( $p = 0.000011$ ; OR 0.335; 95% CI 0.203–0.552) [14]. However, the classification of CW to “open” and “closed” is conventional and does not reflect the full depth of the contribution of different variants of its structure to stroke/TIA formation [17].

The work of E.R. Lebedeva et al. devoted to anomalies of the CW structure concluded that the most common variants are OPP and IPP that was also confirmed by our study results (OPP was 2% more common than OAP; IPP was three times more common than IAP) [18]. This, in its turn, is associated with a higher frequency of stroke/TIA just in the posterior parts of the brain. The authors think that this relationship is due to insufficient self-regulation of cerebral hemodynamics in response to different factors that cause circulation changes [18]. Our work showed that the most common localization of intraoperative stroke/TIA in patients without unstable ASP is the territory of the PCerA. Thus, without PCA and in ipsilateral ICA compression, this part of the brain will receive blood from the contralateral arteries. In case of failure of the latter and long-term ICA compression, ischemic alterations will develop, accompanied by edema and vasoconstriction [19]. This, in turn, leads to gradual intraoperative reduction of oximetry parameters of the ipsilateral part of the brain. Our study results showed that despite a 60% ICA retrograde pressure of the systemic pressure, the oximetry parameters of the brain reduce in some cases (14.5%;  $n = 89$ ), especially in the parts of the brain with hypoplasia or absence of the artery on average by  $12.6 \pm 3.5$  min of the operation. Finally, all cases of the intraoperative hemodynamic stroke in the territory of the PCerA were recorded in patients with reduced oximetry parameters and a variant structure of the CW of OPP or IPP type. However, the CW CAP and OAP type had one hemodynamic stroke in each variant, but on the contralateral side. This phenomenon was due to the presence of hemodynamically significant stenosis of the contralateral ICA. Here, parameters of the retrograde pressure and oximetry at the moment of compression of the ipsilateral ICA were within the norm and did not require TS insertion. Thus, to avoid hemodynamic stroke/TIA, preventive TS is necessary for patients with open types of the CW (OPP, IPP, OAP, and CAP) despite the satisfactory parameters of the retrograde pressure in the ICA and of cerebral oximetry. Recently, the consistency of CW is not a

determining factor for TS insertion according to the national recommendations. TS should be selective and based on the parameters of the ICA retrograde pressure, and blood flows velocity measurement in the MCerA and of cerebral oximetry (B level of evidence) [1]. Thus, a conclusion based on the results of our work considered an additional recommendation for TS application as a preventive step to intraoperative hemodynamic stroke/TIA reduction.

According to our observations, the embolus gets to the MCerA-ACerA pool three times more often, causing the subsequent ischemic alterations in unstable ASP. Here, stroke/TIA of the cardioembolic subtype most commonly develops in OAP and IAP. This tendency was confirmed by the study of L.M. Tibekina et al. and was associated with compensatory collateral blood flow insufficiency in the variant structure of the CW [19]. The embolus movement from ICA to MCerA and ACerA is due to the peculiarities of blood flow direction in the cerebral arteries [20]. Thus, the main direction of the flow is from the posterior (vertebral arteries, VA) and medial (ICA) parts of the brain. Therefore, the blood flows to the center of the CW (territory of the MCerA) and ACerA. However, one stroke of the cardioembolic subtype was recorded in a case with OPP in our study. This patient had a missing P1 segment of PCerA on the right combined with hypoplasia of both VA, which led to blood flow rerouting from the right ICA (where unstable ASP was visualized) to this area. If VA were not hypoplastic, adequate blood flow resistance is developed from the PCA, and embolus displacement would most likely occur to the ACerA. Thus, the direction of the embolus is always determined by the existence of the region with the lowest hemoperfusion. From here, predicting the regions with the most probable development of stroke of cardioembolic subtype is possible at the preoperative stage. However, it is extremely difficult to prevent embolization with unstable ASP in the ICA. Embolus detachment is provoked by elevation of AP before arterial compression. Without this measure, CAE is impossible; however, TS insertion does not require AP elevation before vascular clamp application, since the compression of ICA itself is short-term. Thus, TS in unstable ASP prevents embolization and the development of stroke of the cardioembolic subtype.

## CONCLUSION

The most common variants of the circle of Willis are configurations with the intermediate type of structure of the posterior part (hypoplasia of the posterior communicating or posterior cerebral

arteries) and with the open posterior part (absence of the posterior communicating or posterior cerebral arteries). Optimal parameters of the retrograde pressure in the internal carotid arteries and of oximetry in these patients do not provide a full guarantee of compensation of the cerebral blood flow in arterial compression that is associated with the identified reduction of the tolerance of the brain to ischemia during carotid endarterectomy. In this situation, the preventive insertion of temporary shunt is reasonable. In addition, temporary shunt is used in unstable atherosclerotic plaque that will exclude pharmacological rise of the blood pressure and associated risk of embolization. Thus, additional indications for temporary shunt insertion are the existence of the open circle of Willis and unstable atherosclerotic plaque in the internal carotid artery.

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