Prediction of cerebral ischemia during carotid stenting depending on the intensity of the preoperative ultrasound signal from the plaque

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Carotid stenting is an effective method for improving cerebral perfusion; risk assessment of cerebral embolism associated with this intervention remains a hot research topic.
Objective: *to identify predictors of cerebral embolism associated with carotid angioplasty with stenting (CAS).*
Material and methods. *A prospective exploratory research included 46 patients (age from 44 to 81 years, median 65 years) with atherosclerotic stenosis of the internal carotid artery, who underwent CAS and were treated at the Scientific Center of Neurology (Moscow). The study did not include patients with restenosis, stroke with severe disability, contraindications for antiplatelet therapy, statins, and MRI examination. All patients underwent preoperative ultrasonographic (US) examination and postoperative histological examination of particles in carotid stent embolic protection devices. The state of the brain was assessed before and 24 hours after CAS using diffusion-weighted MRI. To identify predictors of the development of cerebral embolism, the clinical characteristics of patients, together with ultrasonographic and morphological data, were examined in a multivariate statistical analysis.*
Results. *Preoperative US signal from an atherosclerotic plaque of high (estimated above 35 dB) intensity was associated with dense matter (in a protective device) of the "fibrosis with calcification" and "calcification" type and with a high probability (80%; 95% confidence interval 71–85%) predicted intraoperative embolization of cerebral vessels with the acute ischemic lesions (AIL) formation. In a low (not higher than 35 dB) intensity of the preoperative ultrasound signal, the probability of AIL formation was statistically significantly lower (50%).*
Conclusion. *There is a direct correlation between the intensity of the ultrasound signal and the density of the substance in the protective device. A high intensity of the preoperative ultrasound signal (estimated above 35 dB) is an unfavorable predictor of AIL, associated with CAS (with a probability of about 80%).*

Keywords: *carotid stenosis; stenting of the carotid artery; atherosclerosis; cerebral embolism; embolism prognosis.*
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One of the leading causes of cerebrovascular pathology is atherosclerotic lesions of the arteries that feed the brain. An angioreconstruction of cerebral vessels (primarily the vessels of the carotid system) can become an important tool for improving cerebral perfusion in such a situation.

In the last decade in the Russian Federation there has been a significant increase in the frequency of endovascular operations, including carotid angioplasty with stenting (CAS) [1, 2]. During this operation, there is a risk of embolic complications in the brain substance, which prompts the search for methods for their preoperative prediction. A number of studies have shown a relationship between the morphological structure (according to magnetic resonance imaging – MRI) of the components (foci of atheromatosis and hemorrhage) of atherosclerotic plaque (ASP) and cerebral embolism (according to diffusion-weighted magnetic resonance imaging – DW-MRI) [3–6]. We did not find in the available literature any works in which the relationship between ultrasonic (US) visualization of ASP during preoperative ultrasound examination and the morphological content of particles in filtering devices was investigated.

The aim of this study was to determine possible US predictors of the occurrence of cerebral embolism caused by CAS, to identify the relationship between the intensity of the US signal during preoperative ultrasound and the composition of the substance in the embolic protection devices of carotid stents.

Material and methods. The prospective exploratory study included 46 consecutive patients treated at the Research Center of Neurology (Moscow): 28 men and 18 women with the median age (equal to the mean age) of 65 years (range from 44 to 81 years) with ultrasonographically verified atherosclerotic stenosis (symptomatic and asymptomatic) of the carotid arteries, measured by the algorithm of the NASCET study [7]. The choice of the intervention (open or endovascular) was determined by a commission of vascular and endovascular surgeons with the participation of an

angioneurologist. Stenosis was considered symptomatic if within 6 months prior to CAS a patient had ipsilateral symptoms of retinal ischemia, transient ischemic attacks, or ischemic stroke [8]. The study did not include patients admitted for the treatment of carotid stenosis caused by the development of restenosis after the previous carotid endarterectomy, patients with stroke leading to severe disability, as well as patients with contraindications to taking antiplatelet drugs and statins, or ineligible to MRI examination. All patients or their legally authorized representatives signed a voluntary informed consent for surgical treatment; the study was approved by the ethical committee of the Research Center of Neurology (protocol No. 11/14 of 10.19.2020).

The majority of patients older than 60 years were males. In most patients, the course of atherosclerotic process (according to the anamnestic data and the results of a neurological examination) was asymptomatic. Almost all patients (93%) were diagnosed with arterial hypertension, every second patient had at least three risk factors for the development of vascular disease.

In accordance with the developed protocol of the study, all patients in the pre- and postoperative period underwent an ultrasound examination of the carotid arteries (Philips iE 33) using a linear probe with a frequency of 11 MHz. The standardization of the measurement protocol made it possible to minimize the possible measurement error (the error did not exceed ±2 dB). The ultrasound examination algorithm was similar to that used earlier in the work on comparing the ultrasonic and microscopic parameters of ASPs removed during carotid endarterectomy (CEA) [9].

The indication for surgery in asymptomatic patients was >70% carotid stenosis (which is consistent with the results of the SAPPHIRE study [10]). Aspirin (100 mg) and clopidogrel (75 mg) were prescribed 7 days before the endovascular intervention. All stenting procedures were performed on the same biplane angiographic unit (Innova 3131; General Electric) under local anesthesia, using a percutaneous transfemoral approach, by the same neurointerventional team of doctors. Diagnostic x-ray contrast angiography was performed before and after the stenting procedure. Embolic protection systems Emboshield NAV 6 and Filter Wire were used as protective devices (according to the diameter of the distal segment of the internal carotid artery – ICA) in 28 (61%) and 18 (39%) cases, respectively. Depending on the diameter and length of ASPs, dual-layer low-profile Casper and CGUARD stents were used in 35 (76%) and 9 (20%) cases, respectively, as well as Acculink self-expanding nitinol stents in two patients (4%). To achieve the optimal result of stenting, the majority (n=45; 98%) of patients underwent (after positioning and deployment of the stent) post-dilation using a Viatrac 14 Plus balloon catheter. After CAS, dual antiplatelet therapy continued for at least 3 months (acetylsalicylic acid was prescribed for lifetime).

The state of the brain substance before the intervention and 24 hours after it was assessed using DW-MRI (imaging system with magnetic induction 3 T, Magnetom Verio tomograph; Siemens Medical System). Hyperintense damage to the brain substance, which was absent before angioreconstruction, was regarded as a sign of embolism associated with endovascular intervention. The number of new acute ischemic lesions (AIL) according to DW-MRI was calculated by one independent neuroradiologist, who did not have information about clinical and interventional data. A neurological examination of each patient with a deficit assessment according to the National Institutes of Health Stroke Scale (NIHSS) was performed by an independent neurologist 24 hours after the stenting procedure [11, 12].

After the completion of the stenting procedure, the embolic protection system of the brain was removed, and its contents were sent for histological examination. In each case, a longitudinal section of the wall of the protection device was made, followed by making an imprint of its inner surface on a glass slide. Fragments of stony density were subjected to decalcification and subsequent embedding in paraffin with sections 5–6 μm thick on a microtome. The sections and prints were stained with Carazzi's hematoxylin and eosin. Histological preparations were digitized with a Pannoramic midi slide scanner (3D Histech) for subsequent morphometric analysis (the size of potentially embolic material detected in the protection device was assessed).

Statistical analysis included estimation of homogeneity of the compared groups of patients by categorical variables (using Fisher–Freeman–Holton test) and continuous variables (using unpaired Student's t-test or Mann–Whitney U-test, depending on the results of assessing distributions for normality), and ROC analysis. All criteria are two-sided. In the table, categorical variables are represented by absolute and relative frequencies (%), continuous variables are represented by the arithmetic mean with standard deviation. The statistical package SPSS 20.0 was used. The results were considered statistically significant at $p<0.05$.

Results. The technical success of CAS (according to control angiography and ultrasound) was achieved in all patients. In 24 (52%) patients, 24 hours after the intervention, according to DW-MRI, AILs were detected; however, neurological disorders (including transient ischemic attack and ischemic stroke) were not detected in any of the patients. The revealed foci (whose size did not exceed 5 mm) in most cases (95%) were localized in the cortical substance of the brain on the side of the intervention. The number of foci varied from 1 to 15.

We compared two groups of patients: with AILs (detected by DW-MRI 24 hours after the intervention) and without them according to the following characteristics: gender, age, body mass index, history of antiplatelet therapy, cholesterol level, degree of ICA stenosis, presence of occlusion of the contralateral ICA, time interval between the onset of symptoms and intervention, the number of previous surgical interventions on the coronary and brachiocephalic arteries, etc. (see table). Of all the characteristics considered, only the intergroup difference in the intensity of the preoperative ultrasound signal (from ASP fragments in the area of the greatest narrowing of the ICA lumen) reached statistical significance ($p=0.01$). For other characteristics, no strict statistical significance was obtained (in some cases, apparently due to insufficient sample size).

During the histological examination of the imprints of the inner surface of the filtering devices, particles of the following composition were found (Fig. 1):

- cellular components of blood (fresh erythrocytes, platelets, fibrin threads, leukocytes) without ASP fragments – 38 (83%);
- complexes of cells of the vascular wall (fibroblast-like cells 300 μm in size with single connective tissue fibers and without them) – 2 (4%);
- fragments of fibrosis with calcification, 500–1000 μm in size, calcifications;
- calcifications with a complex of cells of the vascular wall (no more than 200 microns) – 6 (13%).

The analysis of the relationship between the morphological type of the substrate in the protection device and the presence or absence of AILs in the brain substance showed that such morphological substrates as "fragments of fibrosis with calcification" and "calcification" were present mainly in patients who developed AILs. Other types of morphological substrates (cellular blood components without ASP fragments, a complex of vascular wall cells, calcification with a complex of vascular wall cells) were present in patients both with and without AILs (Fig. 1).

The morphological substrates "fragments of fibrosis with calcification" and "calcification" corresponded to the highest (>35 dB) intensity of the ultrasound signal during preoperative ultrasound examination (Fig. 2). At a lower (⩽35 dB) intensity of the ultrasound signal, the frequency of formation or non-formation of AILs was approximately the same. An intense (>35 dB) ultrasound signal during preoperative ultrasound can be considered a prognostic sign of the formation of AILs associated with CAS (at the same time, substances of the type "fragments of fibrosis with calcification" and "calcifications" are found in the protection device of the carotid stent). High (>35 dB) preoperative ultrasound signal intensity is an unfavorable predictor of the formation (with a probability of 80%; 95% CI 71%–85%) of AILs associated with CAS, with a sensitivity and specificity of 80% (p = 0.05 according to the results of ROC analysis). At a low (estimated ⩽35 dB) preoperative intensity of the ultrasound signal, the probability of formation or non-formation of AILs associated with CAS is 50% to 50%, while no significant correlation with the substance type in the protection device has been detected.

If we rank the types of morphological substrate in the protection device from less dense to denser, then the intensity of the preoperative ultrasound signal from an ASP changes from less intense to more intense: 6 (1.7–11.4) dB, 31.5 (29–34.0) dB, 39.5 (36.0–40.8) dB and 42 dB (Fig. 3).

Discussion. CAS using brain embolism protection is an established treatment option in appropriately selected patients with a predicted low postoperative rate of neurological complications and mortality [13]. Commonly used protective devices belong to the filtering type (especially in the treatment of patients with insufficient collateral circulation) since

Comparison of groups of patients with and without AIL diagnosed after CAS

Characteristics	All patients (n=46)	Patients with AIL after CAS (n=24)	Patients without AIL after CAS (n=22)
Sex, n (%):			
men	28 (61)	14 (50)	14 (50)
women	18 (39)	10 (56)	8 (44)
Age, years, median (range)	65 (44–81)	65 (44–81)	65 (57–79)
Body mass index, kg/m², M±σ	29.2±3.3	28.2±3.1	29.2±4.4
Symptomatic stenosis, n (%)	5 (11)	3 (60)	2 (40)
Asymptomatic stenosis, n (%)	41 (89)	20 (49)	21 (51)
Degree of stenosis, %, M±σ	77.0±7.6	77.0±7.1	77.0±9.2
Occlusion of the contralateral ICA, n (%)	2 (4)	1 (50)	1 (50)
Arterial hypertension, n (%)	43 (93)	23 (54)	20 (46)
Angina pectoris, n (%)	5 (11)	4 (80)	1 (20)
Myocardial infarction before CAS, n (%)	6 (13)	5 (83)	1 (17)
Operations on the coronary arteries before CAS, n (%)	5 (11)	4 (80)	1 (20)
Operations on the brachiocephalic arteries befor CAS, n (%)	13 (28)	8 (62)	5 (38)
Diabetes mellitus, n (%)	13 (28)	8 (62)	5 (38)
Atrial fibrillation, n (%)	11 (24)	9 (82)	2 (18)
Smoking, n (%)	20 (43)	13 (65)	7 (35)
Aspirin, n (%)	19 (41)	15 (79)	4 (21)
Clopidogrel, n (%)	2 (4)	2 (100)	0 (0)
Statins, n (%)	39 (85)	24 (62)	15 (38)
Hypoglycemic therapy, n (%)	12 (26)	7 (58)	5 (42)
Antihypertensive therapy, n (%)	39 (85)	24 (62)	15 (38)
LDL, mmol/l, M±σ	1.7±0.6	1.7± 0.6	1.6±0.5
Total cholesterol, mmol/l, M±σ	4.5±1.0	4.7±0.9	4.4±0.3
Intensity of the preoperative ultrasound signal from the ASP, dB, median (range)	13.7 (0.8–42.0)	29.0 (1.6–42.0)*	12.5 (0.8–34.0)*

Note. * – differences are statistically significant (p=0.01). LDL – low density lipoproteins.

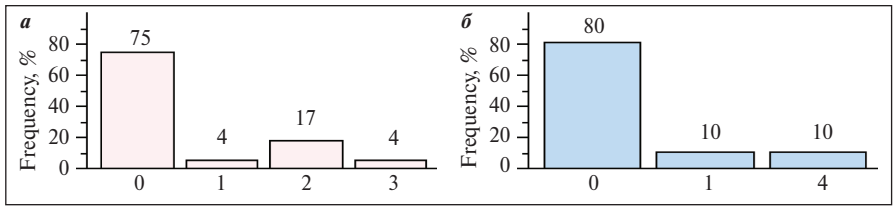

Fig. 1. *Distribution of morphological substrate types in the protective device of the carotid stent in the presence (a) and absence (б) of the CAS-related AIL: 0 – cellular components of the blood without atherosclerotic plaque (ASP) fragments: 1 – a complex of vascular wall cells, 2 – a fragment of fibrosis with calcification; 3 – calcification; 4 – a calcification with a complex of vascular wall cells*

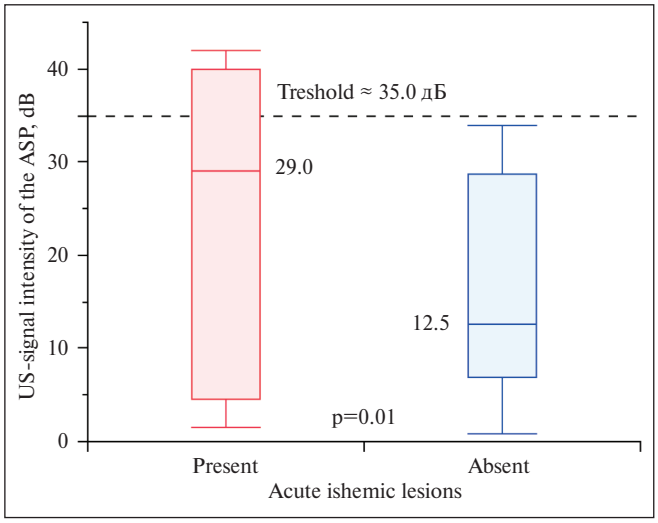

Fig. 2. *Intensity of the preoperative US-signal from ASP in groups of patients with and without AIL (detected 24 hours after CAS). With a high probability, a high (above 35 dB) intensity of the preoperative US-signal is associated with the formation of AIL during CAS*

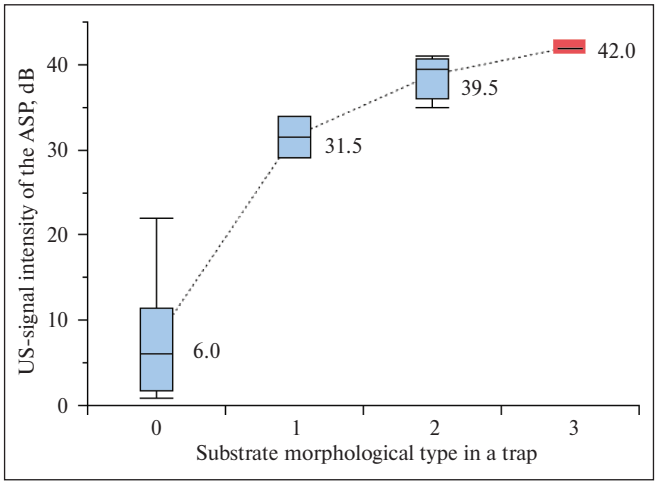

Fig. 3. *Correlation between the intensity of the preoperative US-signal from the ASP and the substance of the substrate in the protective device (ordered on the abscissa axis from less dense to more dense): 0 – cellular components of blood without fragments of ASP; 1 – a complex of cells of the vascular wall; 2 – a fragment of fibrosis with calcification; 3 – calcification. The higher the density of the substrate in the protective device – the higher the intensity of the preoperative US-signal from the ASP*

they do not require temporary occlusion of the blood flow during the procedure [14]. However, the risk of distal embolism associated with the CAS procedure remains. In the study of imprints of the inner surface of the anti-embolic protection system, particles of the substance were found by us in all cases (100%); other studies give the following data: 81% [15], 84% [16], and 100% [17]. A number of studies report that the presence of intra-ASP hemorrhage (detected by MRI imaging) can predict the risk of cerebral embolization during CAS [18, 19]. The substance in the protection device of the carotid stent consisted mainly of cellular blood components without fragments of ASP (83%), in other cases – of cells of the vascular wall, fibrosis with calcification, as well as calcifications and calcifications with a complex of vascular wall cells. We found that the intensity of the ultrasound signal during the preoperative study of ASP is directly related to the density of fragments in the protective device, and a high-intensity (>35 dB) preoperative ultrasound signal from the ASP with a high probability (about 80%) predicts intraoperative embolization of cerebral vessels (detected by DW-MRI 24 hours after CAS). A high-intensity preoperative ultrasound signal corresponded to the morphological substrates "fragments of fibrosis with calcification" and "calcification" in the protection device (Fig. 4). With a low-intensity (not exceeding 35 dB) preoperative ultrasound signal, the probability of formation or non-formation of AIL (with CAS) was approximately the same (50% to 50%). Thus, an intense (>35 dB) US signal from an ASP during preoperative ultrasound examination can be considered an unfavorable predictor of CAS-related AILs, suggesting a high (about 80%; 95% CI 71%–85%) probability of AIL formation (with sensitivity and specificity of 80%).

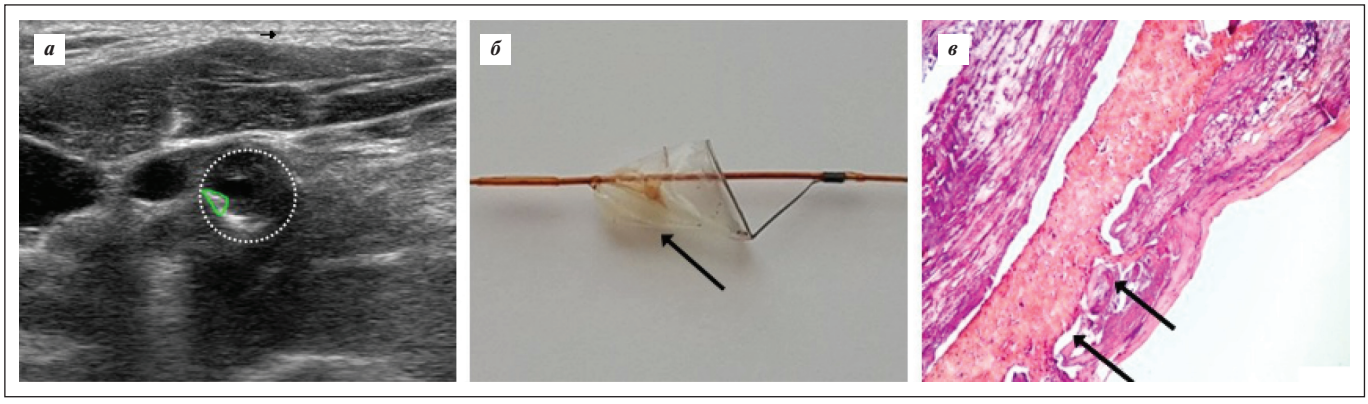

Fig. 4. *Structural features of the ASP of the ICA sinus and antiembolic protection of the brain. а – marked section of the ASP with an US-signal intensity of 41 dB; б – system of antiembolic protection of the brain with the presence of a ASP fragment (arrow); в – histological section of the ASP fragment corresponding to the area with an US-signal intensity of 41 dB: calcified fibers and small calcifications (arrows) are visible on the luminal surface of the ASP. Hematoxylin and eosin stain, magnification × 200*

REFERENCES

1. Алекян БГ, Покровский АВ, Карапетян НГ, Ревишвили АШ. Современные тенденции развития хирургического и эндоваскулярного лечения больных с артериальной патологией. *Ангиология и сосудистая хирургия*. 2019;25(4):55-63. doi: 10.33529/ANGIO2019401
[Alekian BG, Pokrovskiy AV, Karapetian NG, Revishvili ASh. Modern trends in development of surgical and endovascular treatment of patients with arterial pathology. *Angiologiya i sosudistaya khirurgiya*. 2019;25(4):55-63. doi: 10.33529/ANGIO2019401 (In Russ.)].

2. Медведев РБ, Танашян ММ, Кунцевич ГИ и др. Ишемические повреждения головного мозга после каротидного стентирования. *Ангиология и сосудистая хирургия*. 2015;21(1):65-71.
[Medvedev RB, Tanashyan MM, Kuntsevich GI, et al. Ischaemic lesions of cerebral after carotid stenting. *Angiologiya i sosudistaya khirurgiya*. 2015;21(1):65-71 (In Russ.)].

3. Yoshimura S, Yamada K, Kawasaki M, et al. High-intensity signal on time-of-flight magnetic resonance angiography indicates carotid plaques at high risk for cerebral embolism during stenting. *Stroke*. 2011;42(11):3132-7. doi: 10.1161/STROKEAHA.111.615708

4. Yuan C, Mitsumori LM, Ferguson MS, et al. *In vivo* accuracy of multispectral magnetic resonance imaging for identifying lipid-rich necrotic cores and intraplaque hemorrhage in advanced human carotid plaques. *Circulation*. 2001;104(17):2051-6. doi: 10.1161/hc4201.097839

5. Matsukawa H, Fujii M, Uemura A, et al. Pathology of embolic debris in carotid artery stenting. *Acta Neurol Scand*. 2015;131(4):197-202. doi: 10.1111/ane.12303

6. Гулевская ТС, Ануфриев ПЛ, Танашян ММ. Морфология и патогенез изменений белого вещества при хронической цереброваскулярной патологии. *Анналы клинической и экспериментальной неврологии*. 2022;16(2):78-88.
[Gulevskaya TS, Anufriev PL, Tanashyan MM. Morphology and pathogenesis of white matter changes in chronic cerebrovascular disease. *Annaly klinicheskoy i eksperimental'noy nevrologii = Annals of Clinical and Experimental Neurology*. 2022;16(2):78-88. doi: 10.54101/ACEN.2022.2.9 (In Russ.)].

7. Barnett HJ, Taylor DW, Eliasziw M, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. *N Engl J Med*. 1998;339(20):1415-25. doi: 10.1056/NEJM199811123392002

8. Goessens BM, Visseren FL, Kappelle LJ, et al. Asymptomatic carotid artery stenosis and the risk of new vascular events in patients with manifest arterial disease: the SMART study. *Stroke*. 2007 May;38(5):1470-5. doi: 10.1161/STROKEAHA.106.477091. Epub 2007 Mar 15.

9. Медведев РБ, Танашян ММ, Скрылев СИ и др. Связь ультразвуковых и морфологических характеристик атеросклеротических бляшек каротидного синуса. *Ангиология и сосудистая хирургия*. 2018;24(4):43-8.
[Medvedev RB, Tanashyan MM, Skrylev SI, et al. Relation between ultrasonographic and morphological characteristics of atherosclerotic plaques of carotid sinus. *Angiologiya i sosudistaya khirurgiya*. 2018;24(4):43-8 (In Russ.)].

10. Gurm HS, Yadav JS, Fayad P, et al. Long-term results of carotid stenting versus endarterectomy (SAPPHIRE) in high-risk patients. *N Engl J Med*. 2008 Apr 10;358(15):1572-9. doi: 10.1056/NEJMoa0708028

11. Biller J, Love BB, Marsh EE, et al. Spontaneous improvement after acute ischemic stroke. A pilot study. *Stroke*. 1990;21(7):1008-12. doi: 10.1161/01.str.21.7.1008

12. Brott T, Adams HP Jr, Olinger CP, et al. Measurements of acute cerebral infarction: a clinical examination scale. *Stroke*. 1989;20(7):864-70. doi: 10.1161/01.str.20.7.864

13. Fairman R, Gray WA, Scicli AP, et al; CAPTURE Trial Collaborators. The CAPTURE registry: analysis of strokes resulting from carotid artery stenting in the post approval setting: timing, location, severity, and type. *Ann Surg*. 2007 Oct;246(4):551-6; discussion 556-8. doi: 10.1097/SLA.0b013e3181567a39

14. Танашян ММ, Медведев РБ, Евдокименко АН и др. Прогнозирование ишемических повреждений головного мозга при реконструктивных операциях на внутренних сонных артериях. *Ангиология и сосудистая хирургия*. 2017;23(1):59-65.
[Tanashian MM, Medvedev RB, Evdokimenko AN, et al. Prediction of ischaemic lesions of the brain in reconstructive operations on internal carotid arteries. *Angiologiya i sosudistaya khirurgiya*. 2017;23(1):59-66 (In Russ.)].

15. Theron JG, Payelle GG, Coskun O, et al. Carotid artery stenosis: treatment with protected balloon angioplasty and stent placement. *Radiology*. 1996 Dec;201(3):627-36. doi: 10.1148/radiology.201.3.8939208

16. Angelini A, Reimers B, Della Barbera M, et al. Cerebral protection during carotid artery stenting: collection and histopathologic analysis of embolized debris. *Stroke*. 2002 Feb;33(2):456-61. doi: 10.1161/hs0202.102337

17. Martin JB, Pache JC, Treggiari-Venzi M, et al. Role of the distal balloon protection technique in the prevention of cerebral embolic events during carotid stent placement. *Stroke*. 2001 Feb;32(2):479-84. doi: 10.1161/01.str.32.2.479

18. Altaf N, MacSweeney ST, Gladman J, Auer DP. Carotid intraplaque hemorrhage predicts recurrent symptoms in patients with high-grade carotid stenosis. *Stroke*. 2007 May;38(5):1633-5. doi: 10.1161/STROKEAHA.106.473066. Epub 2007 Mar 22.

19. Yamada K, Yoshimura S, Kawasaki M, et al. Embolic complications after carotid artery stenting or carotid endarterectomy are associated with tissue characteristics of carotid plaques evaluated by magnetic resonance imaging. *Atherosclerosis*. 2011 Apr;215(2):399-404. doi: 10.1016/j.atherosclerosis.2011.01.002. Epub 2011 Jan 19.

20. Van Laanen JH, Hendriks JM, Verhagen HJ, van Beusekom HM. Quantity, particle size, and histologic composition of embolic debris collected in a distal protection filter after carotid angioplasty and stenting: Correlation with patient characteristics, timing of carotid artery stenting, and procedural details. *J Thorac Cardiovasc Surg*. 2013 Aug;146(2):492-5. doi: 10.1016/j.jtcvs.2013.03.024. Epub 2013 Apr 12.

21. Sorimachi T, Kakita A, Morita K, et al. Routine aspiration method during filter-protected carotid stenting: histological evaluation of captured debris and predictors for debris amount. *Acta Neurochir (Wien)*. 2011 Nov;153(11):2159-67. doi: 10.1007/s00701-011-1093-3. Epub 2011 Jul 31.

22. Sorimachi T, Nishino K, Morita K, et al. Blood flow changes caused by distal filter protection and catheter aspiration in the internal carotid artery during carotid stenting: evaluation using carotid Doppler sonography. *AJNR Am J Neuroradiol*. 2011 Feb;32(2):288-93. doi: 10.3174/ajnr.A2276. Epub 2010 Nov 4.

23. Sorimachi T, Nishino K, Shimbo J, et al. Routine use of debris aspiration before retrieval of distal filter protection devices in carotid arterial stenting: analysis of captured debris and evaluation of clinical results. *Neurosurgery*. 2010 Nov;67(5):1260-7; discussion 1267. doi: 10.1227/NEU.0b013e3181ef5e85

24. Finol EA, Siewiorek GM, Scotti CM, et al. Wall apposition assessment and performance comparison of distal protection filters. *J Endovasc Ther*. 2008 Apr;15(2):177-85. doi: 10.1583/07-2272.1

25. Maleux G, Demaerel P, Verbeken E, et al. Cerebral ischemia after filter-protected carotid artery stenting is common and cannot be predicted by the presence of substantial amount of debris captured by the filter device. *AJNR Am J Neuroradiol*. 2006 Oct;27(9):1830-3.

26. Faggioli G, Ferri M, Rapezzi C, et al. Atherosclerotic aortic lesions increase the risk of cerebral embolism during carotid stenting in patients with complex aortic arch anatomy. *J Vasc Surg*. 2009 Jan;49(1):80-5. doi: 10.1016/j.jvs.2008.08.014. Epub 2008 Oct 22.

27. Macdonald S, Evans DH, Griffiths PD, et al. Filter-protected versus unprotected carotid artery stenting: a randomised trial. *Cerebrovasc Dis*. 2010 Feb;29(3):282-9. doi: 10.1159/000275505. Epub 2010 Jan 15.

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Conflict of Interest Statement

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