RISK OF DEVELOPMENT OF NEUROLOGICAL COMPLICATIONS IN PROSTHETIC REPAIR OF THE AORTIC ASCENDING PORTION AND ARCH

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The study comprised a total of 68 patients (presenting) with chronic dissection of the aortic ascending portion and arch, undergoing surgery and subjected to measuring at various stages of the operation the level of cerebral oxygenation (rSO_2) of the right and left hemispheres by means of bilateral transcranial spectroscopy.

The aim of the study was to examine the risk for the development of neurological complications in patients with chronic dissection of the aortic ascending portion and arch in various methods of cerebral protection during aortic prosthetic reconstruction.

Group One consisted of thirty-one 40-to-61-year-old (mean age 51 years) patients who during circulatory arrest (CA) were as cerebral protection subjected to antegrade cerebral perfusion (ACP) on the background of moderate hypothermia (23–24°C). Group Two included thirty-seven 40-to-58-year-old (mean age 48 years) patients who during CA were subjected to cerebral protection consisting in craniocerebral hypothermia on the background of total deep hypothermia (18°C). Prior to surgery and in the immediate period thereafter, all patients underwent clinical and instrumental examination of the neurological status.

During CA while aortic arch repair in Group One patients at the expense of maintaining cerebral perfusion a decrease in rSO_2 registered in the right and left hemispheres amounted to only 11.8 and 8.7%, respectively, compared with the baseline values. In Group Two patients during CA a decrease in rSO_2 along the right and left hemispheres amounted to 29.6 and 30.9% compared with the initial values, which was statistically significantly more than in Group One ($p=0.002$ and $p=0.003$). Thus, in Group Two patients during CA cerebral hypoperfusion resulted in a considerable decrease in oxygen supply of the brain, in spite of systemic deep hypothermia and craniocerebral hypothermia, promoting reduction of cerebral metabolism. Using ACP during CA in Group One patients maintained the oxygen status of the brain at an optimal level.

In Group One patients, in the early postoperative period neurological complications were registered in 12.9% of cases. In Group Two, neurological complications were noted in 35.1% of cases.

The univariate logistic regression analysis demonstrated that the risk for the development of any neurological complications depended on the degree of a decrease in rSO_2 during CA while prosthetic repair of the aortic arch relative to the previous values – OR 1.25; 95% CI 1.11–1.65; $p=0.02$.

Hence, deep hypothermia and craniocerebral hypothermia used as cerebral protection during CA turned out to be less effective compared with ACP, because despite reduction of metabolic requirements of the brain, cerebral hypoperfusion substantially of neurological status impairments in the early postoperative period.

Key words: *aortic prosthetic repair, neurological complications, cerebral protection.*

INTRODUCTION

Prosthetic repair of the aortic ascending portion and arch occupies a special place position amongst operative interventions on the aorta. Such operations appear to have still been associated with high risk of neurological complications and a lethal outcome [1, 2].

Lesions of the central nervous system are known to be the most severe complications encountered in patients after endured surgery on the proximal portion of the aorta, and often predetermine the outcome of the operative intervention itself. To them belong not only stroke as the most severe complication but also transitory neurological deficit and hypoxic encephalopathy [3].

Currently, there is no common opinion concerning selection of methods aimed at protecting the brain from ischaemic and reperfusion lesions. In order to reduce the risk of neurological complications during interventions on the arch, there have been worked out various methods, including: cessation of artificial circulation (AC) in the conditions of deep hypothermia, retrograde cerebral perfusion via the superior vena cava, and antegrade cerebral perfusion (ACP) [4, 5].

Hitherto published by various authors outcomes of surgical interventions on the proximal aortic portion, including the data on postoperative transitory and persistent neurological complications differ

Table 1

Clinical and functional characteristics and perioperative data of patients with chronic aortic dissection

Parameters, units of measurement		Group 1, n=31	Group 2, n=37	p
Body mass index (kg/m^2), (Me, 25–75%)		27.1 (21.4–31.8)	28.3 (21.5–32.2)	0.28
Hypertension, n (%)		25 (80.6)	29 (78.4)	0.53
Aortic valve disease, n (%)		4 (12.9)	4 (10.8)	0.54
Atrial fibrillation, n (%)		5 (13.5)	6 (16.2)	0.62
Chronic obstructive pulmonary disease, n (%)		3 (9.7)	3 (8.1)	0.57
Chronic renal insufficiency, n (%)		3 (9.7)	4 (10.8)	0.60
Diabetes mellitus, n (%)		3 (9.7)	4 (10.8)	0.60
Perioperative findings				
Supracoronary prosthetic repair of the ascending aorta +	Prosthetic repair of the aortic arch by means of applying an aggressive oblique anastomosis, n (%)	9 (29)	12 (32.4)	0,48
	Prosthetic repair of the ascending portion of the aorta and arch according to the “elephant trunk” technique, n (%)	8 (25.8)	10 (27)	0,56
	Stenting of the aortic arch with the Djumbodis system	10 (32.3)	11 (29.8)	0,51
Prosthetic repair of the aortic valve and ascending portion of the aorta with a valve-containing conduit + aortic arch repair by means of application of an aggressive oblique anastomosis, n (%)		4 (12.9)	4 (10.8)	0.54
Duration of artificial circulation (min) (Me, 25–75%)		207.4 (160.2–217.2)	249.5 (219.2–287.5)	0.04*
Duration of cooling (min), (Me, 25–75%)		59.8 (48.2–71.5)	95.2 (81.0–125.8)	0.003*
Duration of circulatory arrest (min), (Me, 25–75%)		-	51.3 (36.7–72.1)	
Duration of antegrade cerebral perfusion (min), (Me, 25–75%)		53.0 (38.5–65.4)	-	
Duration of aortic occlusion (min), (Me, 25–75%)		141.2 (117–175)	139.7 (113–183)	0.28
Time of rewarming (min), (Me, 25–75%)		75.4 (62.2–91.3)	93.2 (73.7–103.4)	0.18

*Note: * – statistically significant differences between the groups.*

significantly even with similar perfusion conditions of cerebral protection [6, 7].

In view of the aforesaid, the aim of the present study was to examine the risk for the development of neurological complications in patients with chronic dissection of the aortic ascending portion and arch in various methods of cerebral protection during prosthetic repair of the aorta.

PATIENTS AND METHODS

The study included a total of 68 patients averagely aged 50±2.3 years and diagnosed with DeBakey type I chronic aortic dissection. Of these, 49 (72%) patients were men and 19 (28%) were women. The patients underwent prosthetic repair of the aortic ascending portion and arch both with preservation of the aortic valve and with the use of a valve-containing conduit.

By aetiology of the process, systemic atherosclerosis was diagnosed in 61 (89.7%) patients, with Marfan syndrome revealed in 7 (10.3%) cases.

The inclusion criteria were as follows:

1. Presence of DeBakey type I chronic aortic dissection with the indications for operative treatment in the form of prosthetic repair of the aortic ascending portion and arch;
2. Presence of an anatomically complete circle of Willis, as determined by the findings computed angiography.

The exclusion criteria were as follows:

1. A haemodynamically significant lesion of the brachiocephalic and intracranial arteries;
2. Dissection propagating to the brachiocephalic arteries;
3. Presence of neurological disorders.

Surgical treatment was carried out in the conditions of AC in the nonpulsatile mode, with the volumetric rate of perfusion amounting to 2.5 l/min/m^2. The first stage consisted in performing prosthetic repair of the aortic ascending portion on the background of AC. Depending on involvement of the aortic valve and root in the pathological process, we performed either supracoronary prosthetic repair of the ascending aorta or prosthetic reconstruction of the aortic valve and the aortic ascending portion with a valve-containing conduit.

Table 2

Cerebral oxygenation in patients with chronic dissection of the aortic ascending portion and arch at various stages of surgical treatment

Parameters, units of measurement		Initial narcosis	Artificial circulation before CA	CA	Reperfusion	Rewarming	Cessation of artificial circulation	End of operation
Group 1	rSO_2 of the right hemisphere, %	56.9 (51–63)	57.0 (52–67)	50.3 (46–57)	59.5 (51–65)	62.4 (54–70)	56.0 (51–64)	60.1 (53–67)
	rSO_2 of the left hemisphere, %	59.0 (53–66)	56.3 (51–64)	51.4 (46–60)	60.0 (53–72)	61.5 (54–71)	55.2 (51–63)	58.6 (52–65)
Group 2	rSO_2 of the right hemisphere, %	55.2 (50–59)	53.1 (50–63)	37.4* (31–43)	54.8 (49–60)	71.2 (62–81)	52.4 (48–57)	55.0 (49–63)
	rSO2 of the left hemisphere, %	54.8 (52–62)	52.4 (49–61)	36.2* (30–42)	57.4 (51–63)	68.1(61–76)	50.5 (46–57)	52.1 (47–61)

*Note: rSO_2 – cerebral oxygenation; CA – circulatory arrest; * – statistically significant differences with rSO_2 values in Group One patients, $p<0.05$.*

Table 3

Postoperative parameters and complications in patients with chronic dissection of the aortic ascending portion and arch

Parameters, units of measurement	Group 1, n=31	Group 2, n=37	p
All neurological complications, n (%)	4 (12.9)	13 (35.1)	0.03*
Encephalopathy, n (%)	3 (9.7)	9 (24.3)	0.10
Acute cerebrovascular accident, n (%)	1 (3.2)	4 (10.8)	0.23
Atrial fibrillation, n (%)	3 (9.7)	5 (13.5)	0.45
Cardiopulmonary insufficiency, n (%)	2 (6.5)	6 (16.2)	0.19
Renal insufficiency, n (%)	4 (12.9)	4 (10.8)	0.54
Multiple organ failure syndrome, n (%)	3 (9.7)	5 (13.5)	0.45
Artificial pulmonary ventilation, (hours) (Me, 25–75%)	45.7 (24.3–56.5)	59.3 (35–65)	0.18
Length of stay in the intensive care unit, (days) (Me, 25–75%)	4.0 (3–6)	5.6 (4–7)	0.15
Resternotomy, n (%)	1 (3.2)	4 (10.8)	0.23
Length of hospital stay, (days) (Me, 25–75%)	25.8 (19–31)	32.7 (25–39)	0.24
In-hospital lethality, n (%)	3 (9.7)	4 (10.8)	0.60

*Note: * – statistically significant differences between the groups.*

Achieving the required level of hypothermia was followed by the second stage, i. e., prosthetic repair of the aortic arch. Depending on involvement of the distal and proximal portions of the arch into the pathological process, we decided upon the policy of the intervention. The following variants of aortic arch reconstruction were used: establishment of an aggressive oblique hemiarch-type anastomosis; prosthetic repair of the aortic arch according to the “elephant trunk” technique by a vascular graft with reimplantation of brachiocephalic arteries into the graft on the common landing zone; stenting of the aortic arch with the Djumbodis system. Patients having received multiple-branched grafts during operations on the aortic arch were not included in our study.

Depending on differences of cerebral protection during prosthetic repair of the aortic arch, the patients were divided into two groups. Group One comprised thirty-one 40-to-61-year-old (mean 51 years) patients (22 men and 9 women) in whom during circulatory arrest (CA) cerebral protection consisted in ACP on the background of moderate hypothermia (23–24°C). Group Two was composed of thirty-seven 40-to-58-year-old (mean 48 years) patients subjected during CA to cerebral protection performed as craniocerebral hypothermia (cooling of the head with a cloth helmet filled with ice) on the background of total deep hypothermia (18°C). Patient’s cooling was carried out with a thermal gradient “coolant-body” of 7–8°C. Once prosthetic repair of the aorta terminated, the patient was slowly rewarmed on the background of AC up to a nasopharyngeal temperature of 36°C.

During surgery we registered oxygen supply of the brain by means of bilateral transcranial spectroscopy [device unit cerebral oximeter INVOS 5100 (Somanetics, USA)]. The method of cerebral oximetry registers saturation of haemoglobin with O_2 primarily in blood cerebral venous vessels, with the aim of assessing the degree of cerebral ischaemia. The normal values of rSO_2 correspond to the normal values of central venous saturation – 63–75% [8].

The level of cerebral oxygenation (rSO_2, %) of the right and left hemispheres was analysed at the following stages: 1) initial narcosis, 2) during AC prior to CA, 3) during CA, 4) beginning of reperfusion after CA, 5) period of rewarming on the background of AC, and 7) end of the operation.

During the operation we registered the dynamics of the indices of glucose and lactate in venous blood.

Prior to surgery and in the immediate period after operative treatment all patients underwent clinical and instrumental study of the neurological status using the Mini-mental State Examination (MMEE) – a questionnaire consisting of 30 items and used for assessing the state of cognitive functions. The maximal score of this test is 30, corresponding to high cognitive abilities. Instrumental methods of the study included magnetic resonance tomography and electroencephalography. Prior to surgery, all patients included in the study were free from neurological impairments.

The obtained findings were statistically analysed by means of the statistical programme package Statistica 6.1 (USA), using both parametric and nonparametric methods of statistics with the calculation of the following: mean value – M and mean error – m, median – Me with the interquartile range (25^{th} and 75^{th} percentiles, %), as well as expressed in numerical values and percents. Significance of differences of dependent variables was determined by the Wilcoxon criterion, and that of independent variables – according to the Mann–Witney criterion. Intergroup comparison of the categorical values was carried out by means of χ^2 test with Yates correction or Fisher’s exact test. Risk factors for the development of neurological complication in the early postoperative period were determined using the model of the univariate logistic regression. This model included both quantitative attributes (rSO_2 values at the beginning of the operation, degree of rSO_2 decrease during CA in relation to the previous values, duration of a decrease in the absolute values of rSO_2 during CA below 40%, age, body mass index, duration of AC, duration of CA, aortic occlusion time) and qualitative attributes (presence of accompanying obstructive pulmonary disease, diabetes mellitus, renal pathology, atrial fibrillation), also indicating the odds ratios (ODs) and 95 % confidential intervals (DIs). The threshold values for predictors of an unfavourable prognosis were determined by the ROC analysis. The predictive power of prognostic markers was evaluated and compared proceeding from the area under the ROC-curve (AUC). Differences were regarded as statistically significant if $p<0.05$.

RESULTS

Table 1 shows the clinical and functional characteristics and perioperative parameters of patients of the both groups with chronic aortic dissection.

Group Two patients were found to have longer duration of AC compared with Group One at the expense of the stage of cooling which is associated with the necessity of reaching deep hypothermia.

Table 2 shows the dynamics of the values of rSO_2 of the both hemispheres at various stages of the operation, depending on the method of cerebral protection.

At the stage of initial narcosis and during AC while prosthetic reconstruction of the ascending aortic portion, the absolute values of rSO_2 amounted to more than 50% in the both groups of patients. During CA while prosthetic repair of the aortic arch in Group One patients owing to maintaining cerebral perfusion, a decrease in rSO_2 along the right and left hemispheres was registered to decrease only by 11.8 and 8.7%, respectively, as compared with the baseline values. In Group Two patients during CA, a decrease in the rSO_2 values amounted to 29.6 and 30.9 % compared with the baseline levels, which was statistically significantly more than in Group One patients ($p=0.002$ and $p=0.003$, respectively). The absolute values of rSO2 along the right and left hemispheres in Group Two patients during CA were also lower than in Group One patients ($p=0.01$ and $p=0.003$, respectively) and amounted to less than 40%.

Hence, in Group Two patients during CA cerebral hypoperfusion resulted in a considerable decrease in oxygen provision of the brain, despite systemic deep hypothermia and craniocerebral hypothermia, contributing to reduction of cerebral metabolism. Using ACP during CA in Group One patients maintained the oxygen status of the brain at an optimal level. A decrease in cerebral perfusion on the background of a drop of systemic arterial pressure (AP) during CA in Group Two patients and subsequent reperfusion on the background of cold vasospasm upon completion of CA may promote the development of disordered cerebrovascular autoregulation. According to the data of oxygen supply of the brain, a considerable decrease in rSO_2 during CA was changed by a sharp growth of these values during reperfusion and reached the highest values at the stage of rewarming at the expense of increased metabolic requirements of the brain. Hence, Group Two patients demonstrated a big gradient of rSO_2 from 37 to 71% along the right cerebral hemisphere and from 36 to 68% along the left one, which is not physiological for cerebrovascular regulation. Group One patients did not show a considerable gradient of rSO_2 in the interval between the end of CA and the stage of rewarming, since cerebral blood flow was maintained with the help of ACP.

At the end of the operation, in both groups the level of rSO_2 amounted to more than 50%, as was at the beginning of the operation.

During the whole operation, except CA, mean AP was maintained within the range of 70 mm Hg.

During initial narcosis in Group One and Group Two patients the level of glucose amounted to 5.4 (4.8–5.7) and 5.3 (4.7–5.8) mmol/l, respectively, the lactate level – 1.0 (0.7–1.2) and 0.9 (0.5–1.3) mmol/l, respectively, with no statistically significant differences. At the end of the operation, in Group One and Two patients the glucose level increased statistically significantly up to 9.1 (8.1–9.6) mmol/l ($p=0.01$) and 10.1 (9.8–11.5) mmol/l ($p=0.001$), the level of lactate increased up to 5.1 (4.1–6.2 mmol/l ($p=0.003$) and 7.8 (6.8–9.1) mmol/l ($p=0.002$), respectively. The above-stated indices were statistically significantly higher in Group Two patients – ($p=0.01$ and $p=0.004$), thus strongly suggesting the development of more pronounced acidosis.

The postoperative parameters and complications in patients of the both groups are shown in Table 3.

In the early postoperative period, there were three lethal outcomes in Group One patients. One 46-year-old patient developed uncontrolled haemorrhage on the background of disseminated intravascular coagulation, requiring resternotomy. Two 48- and 52-year-old patients died on the background of multiple organ failure. There were four lethal outcomes in Group Two. In two 38- and 50-year-old women lethal outcomes occurred resulting from uncontrolled haemorrhage on the background of disseminated intravascular coagulation. In two 51- and 53-year-old men death resulted from the development of multiple organ failure with acute cerebral circulation impairments in the basins of the left and right internal carotid arteries.

In Group One patients, in the early postoperative period neurological complications were registered in 12.9% of cases. Posthypoxic encephalopathy was revealed in three patients (the composite score by the MMSE scale ranged from 25 to 26).One 63-year-old patient was found to have acute cerebrovascular accident in the basin of the right internal carotid artery. During prosthetic repair of the aortic arch this patient showed a decrease in rSO_2 as compared with the baseline value by 15.4%, with type 2 diabetes mellitus revealed as concomitant pathology.

In Group Two, neurological complications were noted in 35.1% of cases. Nine patients were found to have posthypoxic encephalopathy [the average composite score by the MMSE scale amounting to 26 (range 23–27)]. Four patients were diagnosed as having acute cerebrovascular accidents in the basins of the left and right internal carotid arteries. During CA, a decrease in rSO_2 in those patients was noted to range from 27 to 36% as compared with the baseline values.

The univariate logistic regression analysis demonstrated that the risk for the development of any neurological complications depended on the degree of a decrease in rSO_2 during CA while prosthetic repair of the aortic arch as compared with the previous values (OR – 1.25; 95% CI 1.11–1.65; p=0.02).

The findings of the ROC analysis demonstrated that a decrease in the value of rSO_2 by 32% and more at the stage of CA [AUC 0.77 (0.59–0.95); p=0.016] possessed the highest sensitivity and specificity in relation to the risk for the development of disordered neurological status.

At the same time, the regression analysis revealed no statistically significant dependence of the risk for the development of neurological complications from the rSO_2 values at the beginning of the operation, duration of the decrease in the absolute values of rSO_2 during CA below 40%, age, body mass index, duration of CA, duration of aortic occlusion and the presence of any accompanying disease (diabetes mellitus, obstructive pulmonary disease, renal pathology, atrial fibrillation).

Hence, the obtained findings make it possible to conclude that during prosthetic repair of the aorta special attention should be paid to the dynamics of oxygen supply of the brain. First of all, it concerns those moments when deep hypothermia and craniocerebral hypothermia are used as cerebral protection during CA while prosthetic repair of the aortic arch. Such being the case, despite a decrease in metabolic requirements of the brain, the presence of cerebral hypoperfusion during CA leads to an increased the risk for the development of neurological complications in the early postoperative period as compared with the method of ACP.

DISCUSSION

Modern trends in providing the most efficient "physiological protection" of organs lead to the necessity of determining an optimal method of cerebral protection during prosthetic repair of the aorta from alteration by ischaemia factors [9].

Currently, deep hypothermic CA as the most convenient and simplest technique remains dominating in prosthetic repair of the aortic arch, decreasing lowering intensity of metabolic processes in the body and promoting increased tolerance to hypoxia [10].

According to other authors' opinion, in complicated cases envisaging a prolonged period of manipulations in the area of the arch (more than 60 minutes) preference should be given to ACP, since this method decreases the level of neurological complications and improves the early postoperative period as compared with deep hypothermic CA [11]. In our study, relying on studying peculiarities of the dynamics of oxygen supply of the brain in two methods of cerebral protection we defined the method of ACP as the most physiological one because of no considerable "drop" of oxygen supply of the brain during CA at the expense of maintaining cerebral perfusion. In the group of patients operated on using deep hypothermic CA with craniocerebral hypothermia besides a more considerable decrease in the level of rSO_2 we also observed a sharp increase of these indices after the end of CA discontinued on the background of lower metabolic demands of the brain and cold vasospasm.

We should enlarge upon some pathophysiological aspects. AC in itself triggers a stress reaction of the body with participation of humoral and cellular components of inflammation. There develops a systemic inflammatory reaction which is one of the factors of damaging the central nervous system leading to damage of the brain blood barrier [12]. In our work, in the group of patients treated using hypothermic CA at the expense of the necessity of longer and deeper cooling, the duration of AC was higher as compared with the patients managed using ACP on the background of moderate hypothermia.

On the background of prolonged CA in the conditions of hypoxia anaerobic glucolysis is activated accompanied and followed by glycolysis whose one of the products of which is lactate. The development of acidosis decreases intensity of capillary cerebral blood flow, and accumulation of suboxidised products induce oedema, which in its turn may additionally enhance disordered perfusion of cerebral tissues, especially in cortical layers [13]. Reperfusion after completion of CA may lead to ischaemically reperfusional lesions of the brain, to loss of integrity of the brain blood barrier and outflow of fluid into brain tissues [14]. These aspects first of all exert an unfavourable effect on the patients' neurological status and quality of life [15]. In our study, the above-mentioned mechanisms are reflected by a higher blood content of glucose and lactate at the end of the operation in the group of patients treated using hypothermic CA compared with patients in whom cerebral protection was carried out by means of ACP on the background of moderate hypothermia. An abrupt drop of the level of rSO_2 during hypothermic CA and a considerable rise of cerebral oxygenation after the end of CA on the background of cold vasospasm increased the "load" on the autoregulatory mechanisms of cerebral vessels. Taking into consideration that the patients of this cohort in the majority of cases are found to have arterial hypertension forming elevated rigidity of vessels, this yet to a larger degree impairs cerebrovascular autoregulation. According to our data, during prosthetic repair of the aortic arch in patients with hypothermic CA a decrease in oxygen supply of the brain averagely reached 30%

in relation to baseline values and was accompanied by the highest rate of neurological impairments during the in-hospital period compared with the group of patients treated using ACP. On the background of ACP a decrease in rSO_2 during CA did not exceed 11%. The logistic regression analysis demonstrated direct dependence of the development of any neurological complications from the degree of a decrease in rSO_2 during AC.

Hence, the risk for the development of neurological complication depends on the degree of a decrease in oxygen supply of the brain during CA. The use of deep hypothermia and craniocerebral hypothermia as cerebral protection in CA is less effective compared with ACP, because despite reduction of metabolic requirement of the brain, cerebral hypoperfusion substantially increases the chances of the development of neurological status impairments in the early postoperative period. Therefore, once there is a technical possibility of using ACP, this method of cerebral protection should be considered as advantageous.

ЛИТЕРАТУРА/REFERENCES

1. ***Bokeria L.A., Garmanov S.V.*** Surgical management of aneurysms of the aortic ascending portion and arch in conditions of selective antegrade cerebral perfusion. Annals of Surgery. 2013; 3: 23–30 (in Russian).
2. ***Chernyavsky A.M., Alsov S.A., Lyashenko M. M., et al.*** Analysis of neurological complications following_after surgical reconstruction of the aortic arch in patients with proximal dissection. Circulatory Pathology and Cardiac Surgery. 2013; 2: 35–40 (in Russian).
3. ***Tsetsou S., Eeckhout E., Qanadli S.D., et al.*** Nonaccidental arterial cerebral air embolism: a ten-year stroke center experience. Cerebrovasc. Dis. 2013; 35: 4: 392–395.
4. ***Bokeria L.A., Malashenkov A.I., Rychin S.V., et al.*** Prosthetic repair of the aortic ascending portion and arch in conditions of bihemispheral perfusion of the brain in various level of hypothermia. Annals of Surgery. 2012; 2: 38-45 (in Russian).
5. ***Bachet J.*** What is the best method for brain protection in surgery of the aortic arch? Selective antegrade cerebral perfusion. Cardiol. Clin. 2010; 28: 2: 389–401.
6. ***Angeloni E., Benedetto U., Takkenberg J.J., et al.*** Unilateral versus bilateral antegrade cerebral protection during circulatory arrest in aortic surgery: a meta-analysis of 5100 patients. J. Thorac. Cardiovasc. Surg. 2014; 147: 1: 60–67.
7. ***Zierer A., Risteski P., El-Sayed Ahmad A., et al.*** The impact of unilateral versus bilateral antegrade cerebral perfusion on surgical outcomes after aortic arch replacement: A propensity-matched analysis. J. Thorac. Cardiovasc. Surg. 2014; 147: 4: 1212–1218.
8. ***Axelrod B.A.*** Regional oxygenation in ensuring safety of cardiosurgical operations. Circulatory Pathology and Cardiac Surgery 2014; 3: 53–58 (in Russian).
9. ***Misfeld M., Mohr F., Etz C.*** Best strategy for cerebral protection in arch surgery – antegrade selective cerebral perfusion and adequate hypothermia. Ann. Cardiothorac. Surg. 2013; 2: 3: 331–338.
10. ***Tian D., Wan B., Bannon P., et al.*** A metaanalysis of deep hypothermic circulatory arrest alone versus with adjunctive selective antegrade cerebral perfusion. Ann. Cardiothorac. Surg. 2013; 2: 3: 261–270.
11. ***Averina T.B., Shundrov A.S., Mokrinskaya L.Yu., et al.*** Perfusion protection of the brain and visceral organs in reconstructive interventions on the aortic ascending portion and arch. Clinical Physiology of Circulation. 2014; 2: 14–19 (in Russian).
12. ***Svetlova N.Yu.*** Pathophysiology of cerebral lesion in operations with assisted circulation. Anaesthesiology and Resuscitation. 2006; 3: 24–27 (in Russian).
13. ***Nicole T.J., Mak Sameena Iqbal, Benoit de Varennes, et al.*** Outcomes of post-cardiac surgery patients with persistent hyperlactatemia in the intensive care unit: a matched cohort study. Journal of Cardiothoracic Surgery. 2016; 11: 33–41.
14. ***Kamenskaya O.V., Cherniavsky A.M., Klinkova A.S., et al.*** Efficiency of various cerebral protection techniques used during the surgical treatment of chronic pulmonary thromboembolism. J. Extra Corpor. Technol. 2015; 47: 2: 95–102.
15. ***Matthias E., Marwan H., Christopher G., et al.*** Long-term outcome and quality of life in aortic type A dissection survivors. Thorac. Cardiovasc. Surg. 2016; 64: 2: 91–99.

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