

Samarsky Igor. Reperfusion syndrome in carotid artery surgery. *Journal of Education, Health and Sport*. 2015;5(8):219-225. ISSN 2391-8306. DOI <http://dx.doi.org/10.5281/zenodo.28075>  
<http://ojs.ukw.edu.pl/index.php/johs/article/view/2015%3B5%288%29%3A219-225>  
<https://pbn.nauka.gov.pl/works/607781>  
Formerly *Journal of Health Sciences*. ISSN 1429-9623 / 2300-665X. Archives 2011–2014  
<http://journal.rsw.edu.pl/index.php/JHS/issue/archive>

Deklaracja.

Specyfika i zawartość merytoryczna czasopisma nie ulega zmianie.  
Zgodnie z informacją MNIŚW z dnia 2 czerwca 2014 r., że w roku 2014 nie będzie przeprowadzana ocena czasopism naukowych; czasopismo o zmienionym tytule otrzymuje tyle samo punktów co na wykazie czasopism naukowych z dnia 31 grudnia 2014 r.

The journal has had 5 points in Ministry of Science and Higher Education of Poland parametric evaluation. Part B item 1089. (31.12.2014).

© The Author (s) 2015;

This article is published with open access at Licensee Open Journal Systems of Kazimierz Wielki University in Bydgoszcz, Poland and Radom University in Radom, Poland Open Access. This article is distributed under the terms of the Creative Commons Attribution Noncommercial License which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author(s) and source are credited. This is an open access article licensed under the terms of the Creative Commons Attribution Non Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>) which permits unrestricted, non commercial use, distribution and reproduction in any medium, provided the work is properly cited.

This is an open access article licensed under the terms of the Creative Commons Attribution Non Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>) which permits unrestricted, non commercial use, distribution and reproduction in any medium, provided the work is properly cited.

The authors declare that there is no conflict of interests regarding the publication of this paper.

Received: 05.06.2015. Revised 15.07.2015. Accepted: 12.08.2015.

## Reperfusion syndrome in carotid artery surgery

Samarsky Igor

Military Medical Clinical Centre of South region, Odesa, Ukraine

### ABSTRACT

**Reperfusion syndrome in carotid artery surgery.**

**Samarsky Igor**

**Military Medical Clinical Centre of South region, Odesa, Ukraine**

The study was aimed to evaluate the frequency of reperfusion injury after carotid artery surgery. It was found that reperfusion syndrome appeared in 27,9% patients when intraoperative administration of creatine phosphate was not applied. The use of intraoperative neuroprotection cut the frequency of reperfusion injury syndrome to 3.4% cases. The feasibility of intraoperative administration of creatine phosphate could improve the results of the surgical treatment of patients with stenotic lesions of carotid arteries is discussed.

**Key words: carotid artery, reperfusion injury, surgery, treatment, prevention.**

Carotid stenosis is a common complication of systemic atherosclerosis which could affect up to 5-7% of the total population [1]. The surgical treatment considered to be a "gold standard" for carotid stenosis however it has a risk of reperfusion injury of brain tissue [2, 3]. Reperfusion injury is released when blood supply returns to the tissue after a period of ischemia. The absence of oxygen and nutrients from blood during the ischemic period creates a condition in which the restoration of circulation results in inflammation and oxidative damage through the induction of oxidative stress rather than restoration of normal function [3].

Reperfusion of ischemic tissues is often associated with microvascular injury, particularly due to increased permeability of capillaries and arterioles that lead to an increase of diffusion and fluid filtration across the tissues. These "activated" endothelial cells produce more reactive oxygen species but less nitric oxide following reperfusion, and the imbalance results in a subsequent inflammatory response [2, 3]. The inflammatory response is partially

responsible for the damage of reperfusion injury. White blood cells, carried to the area by the newly returning blood, release a host of inflammatory factors such as interleukins as well as free radicals in response to tissue damage [3] The restored blood flow reintroduces oxygen within cells that damages cellular proteins, DNA, and the plasma membrane. Damage to the cell's membrane may in turn cause the release of more free radicals. Such reactive species may also act indirectly in redox signaling to turn on apoptosis. White blood cells may also bind to the endothelium of small capillaries, obstructing them and leading to more ischemia [2, 4].

Reperfusion syndrome in terms of revascularization of the brain is not so much unforeseen complication as programmed and executed an essential condition after surgery. Unfortunately this condition is quite common complication of carotid artery surgery and it can lead to significant neurological deficit is the cause of 35% of all deaths in the intervention of the carotid arteries [5].

The study was aimed to evaluate the frequency of reperfusion injury after carotid artery surgery.

#### Material and methods.

Retrospective analysis of medical records was conducted at the MMCCSR (Odessa, Ukraine) in retrospect for the period from 2008-2014. The total number of entries was 172 clinical cases, including 29 patients where neuroprotective medication NEOTON (creatine phosphate) was administered during surgery.

These patients were included in the I group. The remaining patients (n = 143, group II) was treated according to conventional guideline without using neuroprotective agents.

Following criteria of inclusion were applied: the presence of carotid stenosis of more than 60% in combination with transient ischemic attacks or amaurosis fugas; previous ischemic stroke or chronic cerebrovascular insufficiency.

Exclusion criteria: patients with restenosis after previously performed carotid endarterectomy or carotid stenting, carotid stenosis post-radiation; primarily involving the vertebral-basilar basin (vertebral lesions and subclavian arteries); severe arrhythmias (atrial fibrillation, atrial flutter and atrial and ventricular); severe respiratory failure; heart failure III-IV FC. Also not included in the study, patients had contraindications associated with high perioperative risk (acute coronary syndrome, the presence of hemodynamically significant twisting or kinking of target carotid artery calcification of atherosclerotic plaque).

Statistical analysis of the data obtained by means of contingency tables analysis and analysis of variance using the software insurance Statistica 10.0 (StatSoft Inc., CIIA) [6].

Research results and discussion.

It was established that the average age of patients was  $58,5 \pm 1,1$  years, among patients males were predominated (73.3%).

The main manifestations of reperfusion syndrome were represented with cephalgia in the early postoperative period, arterial hypertension and mild focal cerebral symptoms. This condition appeared in 48 (27.9%) patients of group II and only one (3.4%) patient in the group I. In the analysis of long-term results of surgery there was found that almost every patient had signs of mild reperfusion syndrome, marked by the phenomenon of residual neurological symptoms. However, the use of drug neyprotektsiyi prevented the development of this complication.

There are ways to prevent reperfusion syndrome after surgery for occlusive lesions of brachiocephalic arteries, consisting usually in the medication impact on the already incurred reperfusion syndrome, systemic hypertension, spasm of intracranial vessels through the use of antihypertensive anesthetics, calcium antagonists, antioxidants, alpha-blockers, nootropics, vasodilator drugs, steroid hormones, osmotic diuretics, drugs improving venous outflow, antihypertensive drugs, systemic or craniocerebral hypothermia, "stepped" dosing in cerebral blood flow by applying an intraluminal graft [6, 7].

All of these methods are not without drawbacks, since the correction is made has arisen syndrome reperfusion injury, vascular reactions it is often difficult to predict, diagnose, and accordingly treated. Drug correction syndrome is not always effective. Use of intraluminal shunt - protection method of intraoperative hypoperfusion of the brain - in all cases, the projected

hyperperfusion is not justified, and may be accompanied by complications, it complicates and lengthens the operation, the effect of his questionable [6].

Creatine phosphate (NEOTON) plays a key role in ensuring energetic supply of muscle contraction, intracellular transport carrying energetic metabolites from the places of production to places of use. With the depletion of phosphocreatine cells lose their ability to contract, even if there is enough ATP. Neoton positive effect on the natural mechanisms of energy homeostasis of nerve tissue. These effects prevent the occurrence of adverse effects of cerebral hypoxia and decrease risk of reperfusion brain injury. After introduction of the blood, exogenous phosphocreatine inactivated significantly slower than ATP. Furthermore, the compound has the property of selectively accumulate in tissues with elevated levels of metabolism.

#### Conclusion.

The data provided above demonstrates the feasibility of intraoperative administration of creatine phosphate could improve the results of the surgical treatment of patients with stenotic lesions of carotid arteries, particularly, decrease the frequency of reperfusion syndrome from 27.9% to 3.4%.

#### References:

1. Abd-Allah F, Kassem HH, Hashad A, Shamloul RM, Zaki A.  
Prevalence of intracranial atherosclerosis among patients with

- coronary artery disease: a 1-year hospital-based study. *Eur Neurol.* 2014;71(5-6):326-30
2. Sanderson TH, Reynolds CA, Kumar R, Przyklenk K, Hüttemann M. Molecular mechanisms of ischemia-reperfusion injury in brain: pivotal role of the mitochondrial membrane potential in reactive oxygen species generation. *Mol Neurobiol.* 2013 Feb;47(1):9-23.
  3. White BC, Sullivan JM, DeGracia DJ, O'Neil BJ, Neumar RW, Grossman LI, Rafols JA, Krause GS. Brain ischemia and reperfusion: molecular mechanisms of neuronal injury. *J Neurol Sci.* 2000 Oct 1;179(S 1-2):1-33
  4. DeGracia DJ, Kumar R, Owen CR, Krause GS, White BC. Molecular pathways of protein synthesis inhibition during brain reperfusion: implications for neuronal survival or death. *J Cereb Blood Flow Metab.* 2002 Feb;22(2):127-41
  5. Pratschke J, Tullius SG, Neuhaus P. Brain death associated ischemia/reperfusion injury. *Ann Transplant.* 2004;9(1):78-80
  6. Simon R. Post-conditioning and reperfusion injury in the treatment of stroke. *Dose Response.* 2014 Jul 7;12(4):590-9.
  7. Thompson BJ, Ronaldson PT. Drug delivery to the ischemic brain. *Adv Pharmacol.* 2014;71:165-202