ISSN 2304-3415, Russian Open Medical Journal
2018. Volume 7. Issue 4. Article CID e0411
DOI: 10.15275/rusomj.2018.0411

Original article

Near-infrared spectroscopy to predict cerebral hyperperfusion after carotid endarterectomy

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Received 11 May 2018, Revised 23 August 2018, Accepted 27 August 2018

Abstract: The *aim*: to conduct retrospective study of cerebral regional oxygen saturation (rSO2) at all stages of carotid endarterectomy (CEA) to define ischemia and cerebral hyperperfusion predictors.
Material and Methods — rSO_2 were registered in 169 patients under general anesthesia after induction, before carotid artery clamping, 2 minutes after and each 10 minutes after clamping, before, 2 and 5 minutes after reperfusion and at the end of the operation. We estimated baseline values and intraoperative changes of rSO_2 in terms of clinical and instrumental findings. Logistic regression analysis was aimed to define significant risk predictors of cerebral ischemia and hyperperfusion and ROC-curve analysis – to set a cut-off point of rSO_2 reduction and elevation.
Results — Arterial hypertension III grade (HR 9.5%; CI 95%: 1.1-82.7) appeared to be an independent predictor according to the results of multivariate analysis. It was revealed that the most significant predictor of hyperperfusion syndrome is absolute increase of rSO_2 after reperfusion by more than 11.3. Sensitivity, specificity, positive and negative predicative value of the defined parameter were 87.4%, 83%, 35% and 98.4% respectively.
Conclusion — The conducted multivariate analysis demonstrated that only long-term arterial hypertension is a significant risk factor for hyperperfusion syndrome development after CEA. In our study the most accurate parameter for Fore-Sight oximeter is maximum value of absolute increase in rSO_2 after reperfusion by more than 11.3. Cerebral oximetry is a non-invasive method which is easy to use and interpret and enables to estimate both ischemia and cerebral hyperperfusion during CEA and in the early postoperative period.

Keywords: carotid endarterectomy, cerebral oximetry, near-infrared spectroscopy, cerebral hyperperfusion syndrome.

Cite as Shchanitsyn IN, Larin IV, Bakharev RM, Lukin OYu. Near-infrared spectroscopy to predict cerebral hyperperfusion after carotid endarterectomy. *Russian Open Medical Journal* 2018; 7: e0411.

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Introduction

The incidence of neurological complications after carotid endarterectomy (CEA) occurs in 3-5% [1, 2]. In most cases perioperative stroke develops as a result of long-term carotid artery clamping, thrombosis or embolization [2-5]. Cerebral hyperperfusion syndrome (CHS) after CEA was reported in 0-12%. Most common methods used for neuromonitoring include electroencephalography, somatosensory evoked potentials, carotid stump pressure, transcranial Doppler (TCD) ultrasound or combinations of these; as well as assessing the development of neurological signs during endarterectomy performed under local anaesthetic [6]. In recent years a lot of studies came out on the use of cerebral oximetry (CO) for neuromonitoring during CEA.

In 1977 Franz Jöbsis gave first description of near-infrared spectroscopy (NIRS) method for estimating blood circulation parameters in myocardial and brain tissue [7]. In 1985 Ferrari et al. reported the use of CO in humans [8] and in 1993 first commercial cerebral oximetry was maintained by FDA (Food and Drug Administration) in the USA. Nowadays there are several devices of CO: INVOS (Somanetics Corporation, USA), CAS Medical Systems (Branford, USA), Nonin EQUANOX (Nonin Medical Inc., USA), Critikon Cerebral Redox Monitor (Johnson and Johnson Medical Ltd, USA), TOS (Tostec, Japan), NIRO 300 (Hamamatsu Photonics, Japan), OM220 (Shimadzu Co., Japan), et al. In Russian Federation there are two CO devices available working on the basis of source-detector separated spectroscopy: Fore-Sight (CASMED, USA), Invos (Somanetics, USA).

None of the neuromonitoring methods in CEA demonstrated significant advantage over the others [9]. CO appeared to have high correlation with other monitoring methods and advantages of being a non-invasive, continuous and easily interpreted method. Nevertheless CO allows only for indirect assessment of blood flow in the frontal area of cerebral cortex while injuries of parietal lobe and deep structures cannot be assessed, cut-off points for critical ischemia and hyperperfusion are not yet defined and there are few studies estimating the influence of various clinical and demographic factors on CO results.

The aim of this study was to conduct retrospective analysis of cerebral regional oxygen saturation at all stages of carotid endarterectomy to define ischemia and cerebral hyperperfusion predictors.

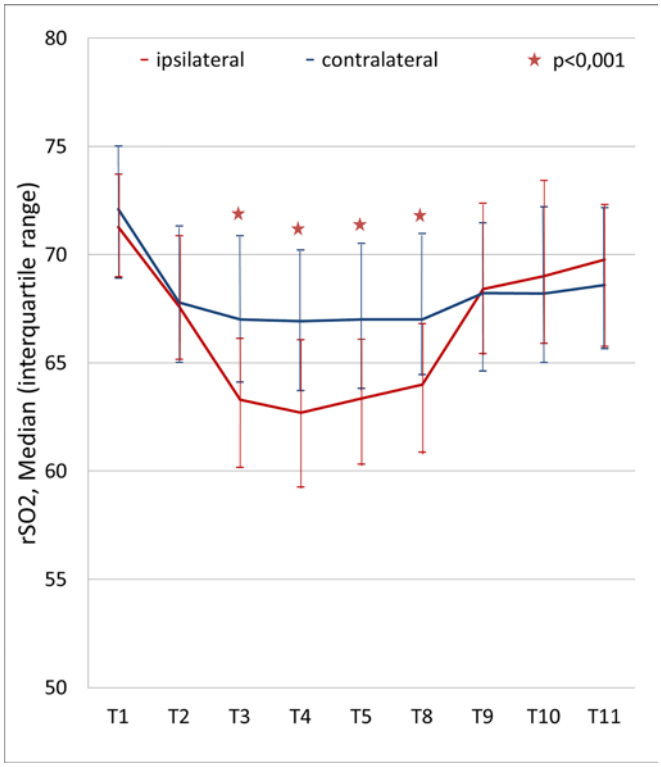

Figure 1. Intraoperative changes in regional cerebral oxygen saturation (rSO_2) in all patients on the surgical and contralateral side.

Material and Methods

Patients

We analysed regional oxygen saturation (rSO_2) of brain tissue in the course of 169 operations on carotid arteries. All operations were performed in the Department of Neurosurgery of Research Institute of Traumatology, Orthopedics and Neurosurgery of Saratov State Medical University (Saratov, Russia) from February 2013 to November 2017.

Intraoperative technique and monitoring

All operations were performed under general anaesthesia (fentanyl, propofol). Suxamethonium chloride or pipecuronium bromide in standard dose were given to reach myorelaxation. Mean arterial blood pressure was sustained in the range from +20% to -20% from the preoperative level with the change of anaesthetic depth. Artificial ventilation was performed in the normoventilation mode with 40% oxygen to sustain partial carbon dioxide pressure in the 30-36 mmHg. Routine monitoring included electrocardiography, indirect measurement of arterial pressure, pulse oximetry and capnography.

In most cases we used standard anterior approach from sternocleidomastoid muscle. Heparin 5000 U was administered five minutes before carotid cross clamping. Eversion carotid endarterectomy (eCEA) was performed in 77.5% (131/169), eCEA with internal carotid artery (ICA) tortuosity resection – in 7.1 % (12/169), endarterectomy from common carotid artery – in 3.5% (6/169), classical carotid endarterectomy – in 2.4% (4/169), ICA tortuosity resection in 9.4% (16/169).

During CEA parameters of rSO_2 were registered each 20 sec. The sensors of cerebral oximeter were applied to the hairless scalp overlying the frontal lobe (cerebral oximeter Fore-Sight, CASMED). Mean values for 1 min. at different stages: after anesthesia (T1), before carotid artery clamping (T2), 2 minutes after (T3) and each 10 minutes after clamping (T4-7), before reperfusion (T8), 2 minutes after (T9) and 5 minutes after (T10) reperfusion and at the end of the operation (T11) were considered in the analysis. We estimated baseline value and analyzed intraoperative changes of rSO_2 based on clinical and instrumental findings.

The neurologic status was evaluated before the operation, after awakening and before discharge. CHS was diagnosed in presence of transitory mental disorders and severe cephalgia without any additional ischemic injury on computed tomography (CT) or magnetic resonance imaging (MRI). CT perfusion or single-photon emission CT (SPECT) in postoperative period was not performed.

Statistical analysis

Statistical analysis was performed using Microsoft Office Excel 13 (Microsoft, USA) and SPSS Statistics 23 (IBM, USA). Data are presented as median and interquartile range – Me (LQ, UQ).

To calculate the differences between cerebral hyperperfusion (CHS) and non-CHS patients, the Mann-Whitney U test was used. To compare groups for qualitative indexes we used Pearson chi-square test (when total number of observations was less than 5 applied Fisher's exact test). The Wilcoxon signed rank test was used to compare rSO_2 changes at different time periods. Differences were deemed statistically significant if $p<0.05$. Logistic regression analysis was used to determine the joint effect of multiple variables on hyperperfusion after CEA. The optimal cut-off points was chosen using the receiver operating characteristic (ROC) curve and Youden's index (sensitivity + specificity – 1). Variables with p-values <0.2 from univariate analysis were included in multivariate analysis. Direct step-by-step approach with inclusion probability 0.05 and exclusion 0.10 and more was used. Odds ratio with confidence interval 95% was calculated. Predictive values are shown as sensitivity, specificity, positive predictive value (PPV) and negative predicative value (NPV), positive likelihood ratio (LR+), negative likelihood ratio (LR-) and diagnostic odds ratio (DOR).

All procedures performed in study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration.

Results

Changes of rSO_2 in all patients on the surgical and contralateral side are shown on *Figure* 1. Statistically significant reduction of rSO_2 level on the operated side during carotid clamping was stated in comparison with contralateral side ($p<0.001$).

Neurological complications (transitory ischemic attack, TIA) after the operation were present in two patients (1.2%, 2/169). While estimating the most effective parameter predicting TIA development we revealed several parameters in univariate analysis, however, these parameters did not demonstrate significance in multivariate analysis. rSO_2 reduction ipsilaterally 2 minutes after clamping had largest area under the curve (AUC). The defined cut-off 2-min-post-clamping points of more than 9.1 had 100% sensitivity and 90.4% specificity. However PPV was only 11.1% which was associated with rare incidence of complications.

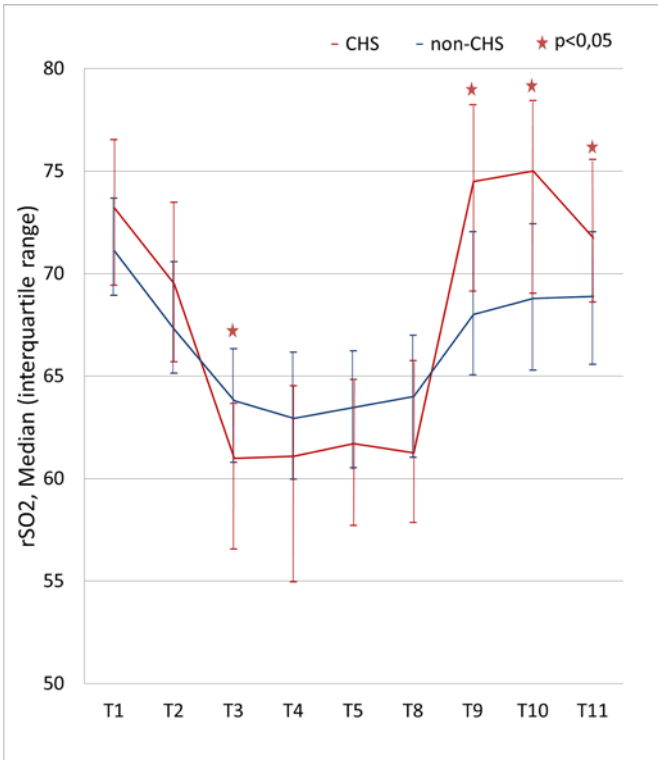

Figure 2. Intraoperative changes in ipsilateral regional cerebral oxygen saturation (rSO2) in the cerebral hyperperfusion (CHS) and the non-cerebral hyperperfusion (non-CHS) group.

In postoperative period CHS developed in 16 patients (9.5%, 16/169). 10 patients (5.3%, 9/169) had severe cephalgia and 6 patients – transitory mental disorders (2.9%, 5/169). Clinical manifestations of hyperperfusion completely vanished by discharge. In one case (0.6%, 1/169) there was significant increase of preoperative neurological deficit (aphasia). All patients with signs of hyperperfusion development underwent MRI. There were no signs of additional ischemic brain injury.

Clinical data of 169 patients is presented in *Table* 1. *Figure* 2 presents the comparison of rSO_2 changes on the surgical side in patients with and without hyperperfusion. Initially before and during clamping CO results in patients with and without hyperperfusion did not differ (p>0.05). Significant differences in rSO_2 were revealed only 2 minutes after clamping (p=0.018) and after reperfusion (p=0.014).

Univariate analysis demonstrated that body mass index (BMI) of more than 27 kg/m^2 and arterial hypertension III grade are predictors of hyperperfusion syndrome. However, multivariate analysis specified only long-term arterial hypertension III grade to be an independent predictor (LR 9.5, CI 95%: 1.1-82.7). Mean intraoperative arterial blood pressure did not correlate with rSO_2 changes.

In order to define the most significant CO parameters allowing to predict hyperperfusion development we conducted multivariate analysis with the specification of cut-off points considering ROC-curves (*Table* 2). The following parameters appeared to be independent predictors of hyperperfusion: rSO_2 value ipsilaterally 5 minutes after reperfusion >76.5 (LR 7.2, CI 95%: 1.1-45.4), maximum interhemispheric difference rSO_2 during clamping >7.4 (LR 16.4, CI 95%: 2.5-107.3), maximum difference between rSO_2 value during clamping and after reperfusion >11.3 (LR 15.9, CI 95%: 1.6-152.2). The last parameter had the largest AUC and DOR (*Table* 3).

Table 1. Clinical and instrumental data

Variables	*Hyperperfusion* No (n=153)	Yes (n=16)	*P-value*
Clinical data			
Age, years	64 (58, 68)	64 (60, 70)	0.964
BMI, kg/m^2	28 (25, 32)	31 (28, 35)	0.035
Women	61 (40)	8 (50)	0.438
Atherosclerosis	138 (90)	15 (94)	0.537
Tortuosity	15 (10)	1 (6)	
Symptomatic patients	71 (46)	8 (50)	0.799
MoCA (n=73)	23 (19, 25)	21 (19, 24)	0.332
CCI (A.V. Pokrovsky):			0.530
- I	10 (6)	2 (12)	
- II	11 (7)	2 (12)	
- III	80 (52)	6 (37)	
- IV	52 (34)	6 (37)	
Comorbidity			
Ischemic heart disease	112 (73)	10 (62)	0.386
Chronic heart failure	28 (18)	3 (19)	0.593
Arrhythmia	18 (12)	2 (12)	0.592
Hypertension III degree	90 (59)	15 (94)	0.006
Chronic renal failure	14 (9)	3 (19)	0.206
Diabetes mellitus	17 (11)	4 (25)	0.118
Instrumental data			
Degree of ipsilateral stenosis, %	75 (70, 80)	75 (70, 80)	0.755
Occlusion	5 (3)	0	0.604
Linear velocity of blood flow, m/s	1.8 (1.5, 2.1)	1.6 (1.4, 2.0)	0.407
Left side, n (%)	66 (43)	7 (44)	0.582
Degree of contralateral stenosis:			0.252
- <50%	88 (57)	6 (37)	
- >50%	60 (39)	10 (62)	
- occlusion	5 (3)	0	
Incompleteness of the C.W. (n=48)	11 (25)	1 (25)	0.697
Intraoperative data			
Operation time, minutes	90 (80, 90)	90 (80, 94)	0.840
Cross-clamp time, minutes	31 (26, 36)	32 (25, 38)	0.755
Shunt use, n (%)	3 (2)	1(6)	0.331

MoCA, the Montreal Cognitive Assessment; CCI, chronic cerebrovascular insufficiency; C.W., Circle of Willis.
Binary data are presented as frequencies and percentages – n (%). Continuous data are presented as median with interquartile range – Me (LQ, UQ).

Discussion

In a meta-analysis of Guay et al. involving 320 patients undergoing CEA (5 studies) sensitivity of cerebral oximetry in revealing ischemia was 74% (CI 95%: 54-89), specificity – 82% (CI 95%: 76-88) [9]. rSO_2 cut-off points were in the range of 15-20%. In our study rSO_2 reduction 2 minutes after clamping of more than 9.1 had 100% sensitivity and 90.4% specificity. However PPV was only 11.1%. Therefore the results of our study should be taken cautiously in the respect of prognostic value of cerebral oximetry in cerebral ischemia and larger cohorts are needed.

Hyperperfusion symptoms include strong headache, convulsions, consciousness disturbances or focal neurological signs in absence of additional ischemic injury on CT or MRI. It is manifested by cerebral oedema, intracerebral hemorrhage as well as increased asymmetrical blood flow in ipsilateral cerebral hemisphere on CT-perfusion or SPECT [13] and TCD [14]. Although the incidence of intracerebral hemorrhage is only 0.4-1.8% [13], considering increased risk of lethality and neurological deficit the significance of timely diagnostics and treatment of this syndrome is undoubtable. Furthermore, even subclinical hyperperfusion syndrome after CEA may result in cognitive disorders [10, 13].

Table 2. Significant predictors of postoperative hyperperfusion. Univariate and multivariate analysis

Variables	*Univariate analysis*			*Multivariate analysis*		
	OR	CI 95%	P-value	OR	CI 95%	P-value
rSO_2 value 2 minutes after reperfusion >75.7	14	4.4-46.1	<0.001			NS
rSO_2 value 5 minutes after reperfusion >76.5	16	4.8-52.5	<0.001	7.2	1.1-45.0	0.035
rSO_2 value at the end of the operation >68.1	6,7	1.5-30.6	0.007			NS
rSO_2 decrease 2 minutes after crossclamping >5.5	17	3.6-76.9	<0.001			NS
rSO_2 decrease 2 minutes after crossclamping >8.5%	19	4.0-84.8	<0.001			NS
The maximum rSO2 decrease during clamping >7.9	25	5.5-117	<0.001			NS
The maximum rSO2 decrease during clamping >11.6%	19	5.1-72.4	<0.001			NS
rSO_2 increase 2 minutes after reperfusion >6.0		*	<0.001			NS
rSO_2 increase 2 minutes after reperfusion >11.8%	33	7.0-152	<0.001			NS
rSO_2-ratio ischemia <0.885	29	6.2-133	<0.001			NS
rSO_2-ratio hyperperfusion >1.184	29	6.1-133	<0.001			NS
Interhemispheric rSO_2 difference 2 minutes after crossclamping >6.9	20	5.3-78.2	<0.001			NS
Interhemispheric rSO_2 difference 2 minutes after crossclamping >9.8%	17	4.5-66.4	<0.001			NS
The maximum interhemispheric rSO_2 difference during clamping >7.4	23	6.1-87.9	<0.001	16	2.5-107.0	0.004
The maximum interhemispheric rSO_2 difference during clamping >11.5%	21	6.2-72.3	<0.001			NS
The maximum interhemispheric rSO_2 difference after reperfusion >6.9	14	4.1-46.7	<0.001			NS
The maximum difference between rSO_2 value during clamping and after reperfusion >11.3	34	7.3-159	<0.001	16	1.6-152.0	0.016
The maximum difference between rSO_2 value during clamping and after reperfusion >18.4%	29	6.2-133	<0.001			NS

rSO_2, regional cerebrovascular oxygen saturation; OR, odds ratio; CI, confidence interval; NS, not significant (p>0.05); SO_2-ratio ischemia, the lowest rSO_2 value during clamping divided by the mean rSO_2 value before clamping; SO_2-ratio hyperperfusion, the highest rSO_2 value after reperfusion to the minimum value during clamping.

Variables with P<0.2 from univariate analysis were included in multivariate analysis. * – OR was not calculated when one of the cells in table had a value 2 or less.

Table 3. Diagnostic impact of significant predictors for hyperperfusion syndrome after carotid endarterectomy

Variables	*p**	*AUC*	*Juden Index*	*Cut-off point*	*Se*	*Sp*	*LR+*	*LR-*	*DOR*
rSO_2 value 5 minutes after reperfusion	0.002	0.74	0.44	76.5	50	94	8.5	0.5	16
The max interhemispheric rSO_2 difference during clamping	<0.001	0.86	0.66	7.4	81	84	5.2	0.2	23
The max difference between rSO_2 value during clamping and after reperfusion	<0.001	0.88	0.71	11.3	87	83	5.1	0.1	34

* – Mann-Whitney U-test. rSO_2, regional cerebrovascular oxygen saturation; max, maximum; AUC, area under the curve; Se, sensitivity (in %); Sp, specificity (in %); LR+, positive likelihood ratio; LR-, negative likelihood ratio; DOR, diagnostic odds ratio.

Table 4. The cut-off points for rSO_2 value in hyperperfusion syndrome prediction after carotid endarterectomy

Parameters	*Authors*				
	Ogasawara et al.	Komoribayashi et al	Matsumoto et al.	Pennekamp et al.	Our study
Year	2003	2006	2009	2012	2017
Number	50	89	64	151	169
Operation	CEA	CEA	CAS	CEA	CEA
Detection	SPECT	SPECT, TCD	SPECT	TCD	Clinical data, MRI
Oximeter	TOS 96	TOS 96	INVOS 5100	INVOS 5100	FORE-SIGHT MC 2030
Variable	ΔrSO_2 before clamping and after reperfusion	SO_2-ratio	ΔrSO_2 before and after procedure	ΔrSO_2 before clamping and after reperfusion	The maximum ΔrSO_2 during clamping and after reperfusion
Cut-off value	> 5%	< 0,9	> 10%	> 2%	> 11,3
Sensitivity, %	100	90	100	100	87,4
Specificity, %	86,4	91,1	85,5	58	83
PPV, %	50	56,3	18,2	11	35
NPV, %	100	98,6	100	100	98,4

CEA, carotid endarterectomy; CAS, carotid artery stenting; SPECT, single-photon emission computed tomography; TCD, Transcranial Doppler ultrasound; PPV, positive predictive value; NPV, negative predictive value; ΔrSO_2, regional cerebrovascular oxygen saturation difference; SO_2-ratio, the lowest rSO_2 value during clamping divided by the mean rSO_2 value in the last 2 minutes before clamping.

In our study 16 patients (9.5%) had hyperperfusion signs in postoperative period: severe cephalgia, transitory mental disorders and neurological deficit increase without additional ischemic changes and intraoperative hemorrhage on MRI. 73 patients were assessed by Montreal Cognitive Assessment before and 6 months after operation. All patients demonstrated statistically significant improvements (p<0.001). But at the same time there were no significant differences depending on the presence of hyperperfusion (p=0.459).

There are only a few studies on risks factors of hyperperfuion syndrome after CEA. Various authors have defined the following risk factors: bilateral ICA stenosis and previous stroke [10], long-term arterial hypertension, high-grade stenosis, insufficient collateral blood circulation, contralateral artery occlusion [15], postoperative hypertension [14], decreased cerebrovascular

reactivity on preoperative TCD [16]. In our study multivariate analysis proved only log-term arterial hypertension III degree to be an independent predictor (LR 9.5, CI 95%: 1.1-82.7).

Intraoperative TCD (linear blood velocity assessment in medial cerebral artery) for hyperperfusion syndrome prediction is described by various authors [14, 17]. But this method requires requires skilled operators for consistent results, not in all patients it can be applied due to the lack of acoustic window. Transcranial cerebral oximetry has a range of advantages: easy to use, providing continuous real-time information, possible to use in the postoperative period [16]. Only a few authors estimated rSO_2 cut-off points for the prediction of cerebral hyperperfusion after reperfusion during CEA [12, 14, 16, 18] (*Table* 4) These studies are conducted with the use of various oximeter types and hyperperfusion assessment parameters. Our study revealed absolute reperfusion values of rSO_2 elevation of more than 11.3 to be the most significant predictor of hyperperfusion. Sensitivity, specificity, positive and negative prognostic value were 87.4%, 83%, 35% and 98.4%, respectively.

We assume that early detection of cerebral hyperperfusion with cerebral oximetry allows for preventive treatment, particularly more precise control of arterial pressure in the postoperative period and of anticoagulation therapy length. We reckon that patients with long-term arterial hypertension and hyperperfusion predictors defined by cerebral oximetry are at high risk of hyperperfusion syndrome and require closer monitoring and arterial pressure control (not higher than 120 mmHg) for several postoperative days. We routinely used enoxaparin on the first day after operation. But after the consideration of this research results we decided to suspend this therapy in order to prevent hemorrhagic complications. Apart from that we suppose it reasonable to use CO in the early postoperative period in intensive care unit.

Conclusion

The multivariate analysis demonstrated that only long-term arterial hypertension III grade is a independent risk factor of hyperperfusion after CEA. At present there are no well-defined predictors of rSO_2 changes to predict the development of hyperperfusion. Cut-off points depend on the cerebral oximeter type and the criteria for hyperperfusion on the CO-monitor. Out study report the elevation of absolute rSO_2 value after reperfusion compared to the clamping value of more than 11.3 to be the most accurate parameter to predict hyperperfusion when Fore-Sight oximeter is used.

Limitations

Our study has several limitations. Firstly, we did not apply perfusion CT or TDC for the confirmation of cerebral hyperperfusion. Secondly, patients with significant increase of rSO_2 after reperfusion had more aggressive antihypertensive treatment which influenced the study results. 26 patients had elevated rSO_2 values of more than 11.3 and at the same time did not have any hyperperfusion signs postoperatively. It significantly reduced specificity and positive prognostic value of cerebral oximetry. Thirdly, cerebral oximeter sensors were applied on the forehead and it only monitored a superficial area of the frontal lobe but perfusion changes in other brain areas might have escaped detection. Nevertheless according to previous research SPECT results demonstrate that hyperperfusion was revealed in the whole ipsilateral hemisphere including controlled frontal lobe [13, 14, 16]. Fourthly, high variability of absolute rSO_2 values reduced specificity of hyperperfusion predictors. Relative ratio (percentages of reperfusion to clamping or baseline values) might help to overcome this limitation. And yet taken into account the fact that rSO_2 values before clamping and at the end of the operation differed in most patients ($p<0.001$) we assume that estimation of rSO_2 changes after reperfusion in comparison with baseline values is not informative. Cerebral oximeter Fore-Sight (Casmed, USA) with 4 near-infrared laser light waves allows assessing absolute rSO_2 values. Moreover, our analysis demonstrated high specificity of absolute rSO_2 values. Fifthly, in most cases we did not use intraluminar shunt and cerebral ischemia on the background of long-term ICA clamping may have increased hyperperfusion risk postoperatively. However in our study clamping time did not correlate with hyperperfusion rate postoperatively. This stands with the other researches reporting hyperperfusion and clamping time as non-correlating parameters [16]. rSO_2 is also influenced by extracranial blood flow, arterial pressure level, blood oxygen saturation, partial carbon dioxide pressure in gas mixture, bilirubin and hematocrit level [19]. In the present study, all patients had blood saturation level of more than 99%, partial carbon dioxide pressure was sustained on the constant level, blood loss was minimal (50 ml and less), bilirubin and hemoglobin levels did not correlate with rSO_2. Two-detector sensor Fore-Sight provides the exclusion of light reflected from superficial extracranial tissues and the obtained signal contains only brain tissue saturation data. Thus it is slightly possible that these factors had any significant influence on rSO_2 measurements.

Conflict of interest: none declared.

Ethical approval

All procedures performed in study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration.

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