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Impact of contralateral carotid stenosis on brain tissue oxygenation during carotid endarterectomy

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SOUHRN

Kontext: Cerebrální oxymetrie (rSO_2) se jeví jako slibná metoda pro sledování adekvátnosti dodávky kyslíku do mozku v průběhu karotické endarterektomie (CEA). Cílem studie bylo zhodnotit vztah mezi závažností stenózy kontralaterální (neoperované) karotidy a změnami rSO_2 v průběhu CEA.
Metodika: rSO_2 byla sledována u 38 pacientů podstupujících CEA v regionální anestezii. Pacienti byli následně rozděleni podle přítomnosti (Gdef) nebo absence (Gnodef) nového neurologického deficitu. Výchozí rSO_2 před naložením svorky (rSO_2base), průměrné (rSO_2avg) a minimální (rSO_2min) hodnoty během tří minut po svorce a změna rSO_2 během tří minut po naložení svorky (ΔrSO_2avg a ΔrSO_2min) byly porovnány mezi oběma skupinami. Vliv stenózy kontralaterální karotidy na rSO_2 byl hodnocen pomocí korelace. Data jsou uvedena jako medián (interkvartilové rozpětí).
Výsledky: Nový neurologický deficit se vyskytl u čtyř pacientů. Neprokázali jsme významný rozdíl rSO_2 měřené na operované straně mezi Gdef a Gnodef: rSO_2base bylo 79 (75–87) v Gnodef a 79 (64–90) v Gdef (p = 0,84), rSO_2min 74 (65–81) v Gnodef a 75 (59–90) v Gdef (p = 0,70) a ΔrSO_2min 6 (2–13) v Gnodef a 2 (0–6) v Gdef (p = 0,15). Podobně jsme nezaznamenali významné rozdíly mezi skupinami pro rSO_2 měřené na kontralaterální straně. Stenóza neoperované karotidy byla rizikovým faktorem pro rozvoj perioperačního neurologického deficitu, ale neprokázali jsme její vliv na hodnoty rSO_2.
Závěr: Neprokázali jsme závislost mezi závažností kontralaterální stenózy karotidy a změnami rSO_2 v průběhu CEA. Cerebrální oxymetrie nebyla schopná předpovědět rozvoj neurologického deficitu během CEA.

Keywords:
Carotid endarterectomy
Cerebral ischemia
Cerebral oximetry
Cerebrovascular monitoring
Near-infrared spectroscopy

ABSTRACT

Background: Cerebral oximetry (rSO_2) seems to be a promising technique for assessment the adequacy of cerebral oxygen delivery during carotid endarterectomy (CEA). The purpose of this study was to evaluate the association of the severity of contralateral (non-operated) carotid artery stenosis and the magnitude of rSO_2 changes during CEA.
Methods: rSO_2 was monitored in 38 patients undergoing CEA under regional anesthesia. Patients were retrospectively assigned to one of two groups: with (Gdef) or without (Gnodef) change in neurological status. Baseline rSO_2 values (rSO_2base), average (rSO_2avg) and minimal (rSO_2min) values during 3 minutes after clamping, and rSO_2 change after clamping (ΔrSO_2avg and ΔrSO_2min) were compared between both groups. The influence of contralateral carotid artery stenosis on rSO_2 was assessed by correlation. Data are presented as median (IQR).
Results: Neurologic deterioration has developed in 4 patients. We did not find any statistical difference in rSO_2 values between Gdef a Gnodef on operated side – rSO_2base was 79 (75–87) in Gnodef and 79 (64–90) in Gdef (p = 0.84), rSO_2min 74 (65–81) in Gnodef and 75 (59–90) in Gdef (p = 0.70), and ΔrSO_2min 6 (2–13) in Gnodef and 2 (0–6) in Gdef (p = 0.15). Similarly, there was not any difference between groups if rSO_2 was measured on contralateral side. We showed that non-operated ACI stenosis was a risk factor for developing of perioperative neurological deficit, but we did not demonstrate any influence of non-operated ACI stenosis severity on rSO_2 values.
Conclusion: We did not prove any association between the severity of contralateral carotid artery stenosis and the magnitude of rSO_2 changes during CEA. Cerebral oximetry was not able to predict the development of neurologic deficit during CEA.

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Introduction

Carotid artery disease causes approximately 10 to 20% of strokes.[1,2] Carotid endarterectomy (CEA) has been established as an effective treatment for patients with atherosclerotic carotid stenosis. This procedure can significantly reduce risk of subsequent stroke in symptomatic patients and in selected group of asymptomatic patients.[3] However, according to current literature, the perioperative stroke occurs in more than 2% of patients undergoing CEA.[4] This is usually caused by either cerebral hypoperfusion or embolism during surgery. Routine periprocedural placement of an intraluminal shunt to prevent cerebral ischemia carries its own risk of intimal damage or stroke caused by embolism.[5,6] Therefore, it is important to identify patients who are at high risk of developing severe neurological complications during carotid clamping and who are likely to benefit from selective shunting. The real-time clinical evaluation in awake patient still remains the simplest and the most accurate way to monitor the change in neurologic status.[7,8] However, this approach can be applied only when regional anesthesia is used. Monitoring devices, such as transcranial doppler (TCD), electroencephalography (EEG) or somatosensory evoked potentials (SSEP) have been tested for many years but have serious logistic limitations and disadvantages. So far, no single monitoring technique is reliable enough to predict perioperative stroke during CEA under general anesthesia.[9]

Various studies have indicated that the use of cerebral oximetry, based on the principles of near-infrared spectroscopy (NIRS), is useful in determining cerebral ischemia during CEA. As first described by Jöbsis in 1977,[10] NIRS is a non-invasive technique that allows continuous monitoring of cerebral hemoglobin oxygenation (rSO_2). There are suggestions that information provided by this device may significantly improve patient outcome by preventing neurological dysfunction.[11,12] Unfortunately, the evidence to define clear cut-off value for presence of perioperative cerebral ischemia is limited. Therefore, at this time NIRS cannot be used as a standard monitoring technique for prevention of perioperative stroke from CEA.[13]

Many patients undergoing CEA for their symptomatic internal carotid artery disease have multiple atherosclerotic lesions throughout their extracranial cerebral arteries.[14] Consequently, significant contralateral carotid stenosis could be an important factor that affects the magnitude of change in rSO_2. The circle of Willis (CW), the most important vascular structure, functions as collateral source of perfusion following occlusion of either internal carotid system or basilar system, but its compensatory potential in keeping the cerebral perfusion at a sufficient level can be blunted in individuals with diffuse atherosclerosis. It has been previously demonstrated that the functional configuration of the CW reflects the degree of internal carotid artery obstruction.[15] To our knowledge, no studies describing the impact of significant contralateral carotid stenosis on intraoperative cerebral ischemia development have been done.

The aim of this prospective observational study was to evaluate the influence of contralateral carotid artery disease on intraoperative changes in rSO_2 in patients undergoing elective CEA. In addition, we investigated the reliability of rSO_2 changes during CEA to detect clinical neurological deterioration.

Material and methods

Patients and monitoring

The study was approved by the Ethics Committee of St. Anne's University Hospital. Since this is an observational study, the Ethics Committee waived the requirement for obtaining informed consent. Consecutive patients undergoing elective CEA in regional anesthesia were enrolled into the study. All patients received standard medication; antihypertensive drugs were continued on the day of surgery, with the exception of angiotensin-converting enzyme inhibitors. No preoperative sedation was administered.

An ipsilateral single-shot cervical plexus block (deep and superficial) was performed using 20 ml and 10 ml of 0.5% bupivacaine.[16] If the patient complained of pain during surgery, supplemental 1% trimecaine was administered directly into the wound by the surgeon. If this measure was not sufficient, 5–10 µg of intravenous sufentanil was administered. Any other sedation that could potentially affect intraoperative evaluation of neurological function was avoided. Invasive blood pressure, five-lead electrocardiogram with ST segment analysis, peripheral hemoglobin saturation, and regional cerebral oxygen saturation (rSO_2) were continuously monitored during operation.

The INVOS 5100c cerebral oximeter (Somanetics Corporation, Troy, MI, USA) was used for rSO_2 monitoring from the time of admission to the operating room to discharge to the postoperative care unit. According to the manufacturer's instructions, two cerebral oximeter sensors were placed on the forehead, one on each side of the midline for bilateral recordings. By this placement, signal is received from tissue perfused by the anterior (ACA) and the middle cerebral artery (ACM). Measured rSO_2 values were continuously recorded for later offline analysis. The use of cerebral oximeter device did affect neither standard surgical technique nor anesthesia management and no change in clinical management occurred based on the rSO_2 readings.

During the time of carotid occlusion, neurologic function was assessed regularly by determining the level of consciousness and by patient's ability to respond to verbal commands and exhibit normal motor strength in the contralateral upper extremity. Within the first 3 minutes after clamping, neurological examination was performed every 20 seconds. After that, testing interval was extended to 5 minutes. Inability to respond appropriately to verbal commands, unconsciousness, slurring of speech, or development of motor weakness were considered as a new neurologic deficit and led to insertion of shunt and conversion from regional to general anesthesia.

Data collection and statistical analysis

From the cerebral oximeter recordings for each and every patient the following rSO_2 parameters were calculated: baseline rSO_2 value was defined as the average rSO_2 over a 3 minutes period before the common carotid artery

(CCA) clamping (rSO_2base), minimal rSO_2 value detected during the first 3 minutes after clamping (rSO_2min), while mean cross clamp was the average rSO_2 value over this period (rSO_2avg). To suppress inter-subject variability in rSO_2base values and to allow comparison of rSO_2 variations after carotid clamping, rSO_2 change (ΔrSO_2) from baseline to rSO_2min and rSO_2avg were calculated using the formulas: $\Delta rSO_2min = rSO_2base - rSO_2min$; $\Delta rSO_2avg = rSO_2base - rSO_2avg$

The magnitude of the computed change after cross-clamp is expressed in percentage points. After completion of the study, patients were assigned to one of two groups: those who did not show a change in neurological status (Gnodef), and those who did (Gdef). Statistical analysis was performed using the Statistica 13 software (Statsoft Inc., Tulsa, OK, USA). A non-parametric Mann-Whitney U test was used for post hoc comparisons of all the measured and calculated rSO_2 values between both groups of patients. The impact of contralateral carotid stenosis severity on rSO_2 values and changes after clamping was evaluated by correlation. A p value of < 0.05 was considered significant. ROC (receiver operating characteristic) curves were created for each rSO_2 parameter and AUC (area under curve) were calculated. All data are presented as median (IQR).

Results

38 consecutive patients (28 men and 10 women) aged between 61 and 78 years were enrolled. Patient's comorbidities and risk factors are presented in Table 1. No neurologic changes were observed in 34 patients (Gnodef, 89.5%),

Table 1 – Comorbidities and risk factors

Male/Female	28/10
Risk factors	
Hypertension	32 (84.2%)
Diabetes mellitus	12 (31.6%)
Hypercholesterolemia	31 (81.6%)
Smoking	18 (47.4%)
Atherosclerosis presentation	
Coronary artery disease	18 (47.4%)
Peripheral arterial disease	16 (42.1%)
Cerebrovascular disease (CEA indication)	
Asymptomatic	18 (47.4%)
Transient ischemic attack	2 (5.2%)
Stroke	18 (47.4%)
Degree of ipsilateral stenosis	
< 70%	38 (100%)
≥ 70%	0 (0%)
Degree of contralateral stenosis	
< 70%	27 (71.1%)
≥ 70%	11 (28.9%)

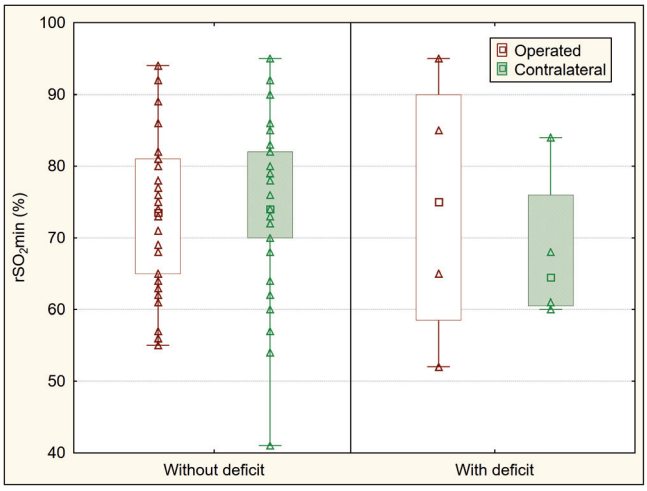

Fig. 1 – Comparison of the lowest rSO_2 values during the first 3 minutes after carotid clamping (rSO_2min) between patients with and without neurologic deficit. Box-and-whisker plots represent median, quartiles and range; triangles represent individual values.

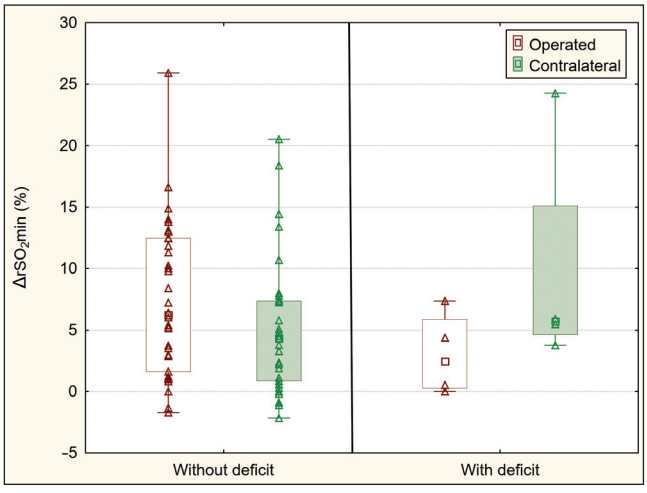

Fig. 2 – Comparison of drop in rSO_2 during the first 3 minutes after carotid clamping (ΔrSO_2min) between patients with and without neurologic deficit. Box-and-whisker plots represent median, quartiles and range; triangles represent individual values.

whereas neurologic deterioration occurred in 4 patients (Gdef, 10.5%) during the first 3 minutes after carotid clamping. In two of these patients change in neurologic status resolved shortly after declamping and the operation was completed after conversion from regional to general anesthesia and insertion of intravascular shunt. Surgery was cancelled in the remaining two patients due to persisting neurologic symptoms. Overall, CEA was completed successfully in 36 patients. The duration of surgery was 135 minutes (123 to 143). All the 38 patients left the hospital without a clinically detectable, new neurologic deficit.

The numerical values of rSO_2 and AUC for all 38 patients with bilateral monitoring are shown in Table 2. We have found no significant difference in any of rSO_2 values among patients with neurological deficit versus those without neurological deficit. In addition, there was no significant difference between the two groups when either ipsilateral or contralateral values of rSO_2 were com-

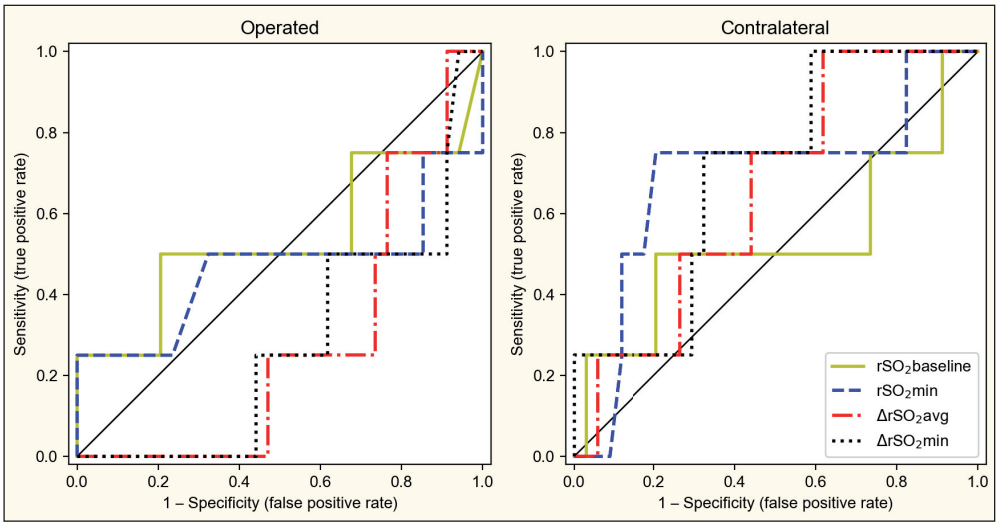

Fig. 3 – Ability of rSO_2 parameters measured on operated and contralateral side to predict neurologic deficit during CEA. Expressed as receiver operating characteristic curves.

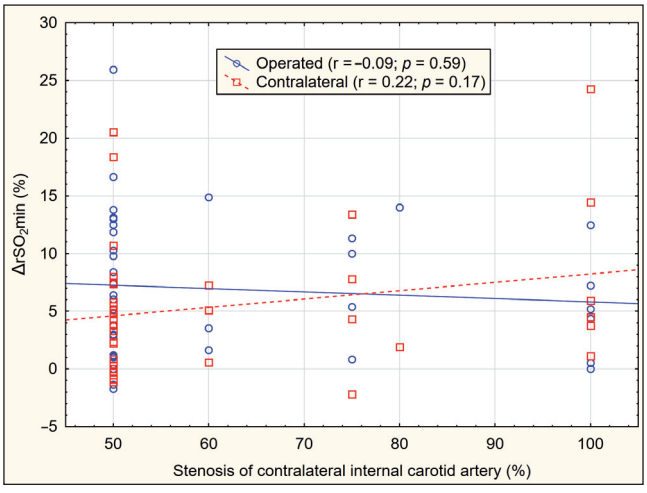

Fig. 4 – Correlation between drop in rSO_2 during the first 3 minutes after carotid clamping (ΔrSO_2min) and severity of non-operated carotid artery stenosis.

pared. Fig. 1 shows the comparison of minimal rSO_2 measured during the first 3 minutes after clamping in patients developing and not developing cerebral ischemia, Fig. 2 shows the comparison of rSO_2 drop (ΔrSO_2min) and ROC curves are shown in Fig. 3.

From the four patients who developed cerebral ischemia after carotid artery clamping, three had contralateral ACI occlusion (> 70% stenosis; detected by ultrasound). In such patients with more than 70% stenosis of contralateral ACI, we have observed a trend to develop perioperative neurological deficit (OR 9.75, CI 0.89–107.3, p = 0.0627). Conversely, no significant relationship was found between increasing degree of contralateral carotid stenosis and the baseline value or the magnitude of rSO_2 change on both frontal lobes. For illustration, the correlation between the minimal value of rSO_2 and severity of non-operated carotid artery stenosis is shown in Fig. 4. Both hospital and ICU length of stay were significantly lower in the group without intraoperative neurological deficit. The median length of hospital stay in the group without deficit was 8 days (7 to 9) and 12 days (9 to 18) in the deficit group (p = 0.043). The median length of ICU stay in the group without deficit was 3 days (2 to 4) and 10 days (5 to 17) in the deficit group (p = 0.008).

Discussion

To our knowledge, this is the first study reporting a relationship between a degree of non-operated carotid stenosis and a change of rSO_2 during CEA. Although, non-operated ACI stenosis was a risk factor for develop-

Table 2 – Comparison of cerebral oximetry (rSO_2) values, both measured and calculated, between patients with and without change in neurological status during carotid endarterectomy

	Operated side (ipsilateral)				Non-operated side (contralateral)			
	Without neurological deficit	*With neurological deficit*	*p*	*AU ROC*	*Without neurological deficit*	*With neurological deficit*	*p*	*AU ROC*
rSO_2base	79 (75–87)	79 (64–90)	0.8365	0.54	77 (74–87)	79 (69–88)	0.8725	0.53
rSO_2min	74 (65–81)	75 (59–90)	0.6966	0.47	74 (70–82)	65 (61–76)	0.4201	0.69
rSO_2avg	76 (67–82)	77 (62–91)	0.8365	0.46	76 (74–82)	72 (67–79)	0.3198	0.66
ΔrSO_2min	6 (2–13)	2 (0–6)	0.1519	0.28	4 (1–7)	6 (5–15)	0.2166	0.70
ΔrSO_2avg	4 (0–10)	0 (0–3)	0.1667	0.28	2 (0–5)	3 (2–9)	0.3434	0.65

ing perioperative neurological deficit, we did not demonstrate any influence of contralateral carotid artery stenosis on rSO_2 values measured neither on ipsilateral nor contralateral side of the forehead. Our expectations that patients with severe degree of contralateral stenosis have lower baseline rSO_2 and more pronounced decrease of rSO_2 after carotid cross-clamping have not been confirmed. Insufficient sensitivity of rSO_2 in cerebral ischemia detection is supported by a fact that three of four patients with developed neurological deficit had confirmed contralateral ACI stenosis. This finding is consistent with results of meta-analysis published in 2013 which showed on more than 28 thousands of patients, that contralateral carotid stenosis increases the risk of perioperative stroke (OR 1.65) and death (OR 1.76).[17] Moreover, we did not find any difference in rSO_2 values between patients with and without intraoperative neurological deficit and thus, we were not able to define a critical threshold which could reliably predict development of cerebral ischemia.

In fact, our findings are consistent with other previously published studies. A major challenge of most studies utilizing intraoperative NIRS during CEA has been defining the sensitivity and specificity of changes in rSO_2 as correlated with either clinical signs of cerebral ischemia or other neuromonitoring modalities.[9,18–21] The main area of research interest was to determine the threshold rSO_2 during CEA that correlates with clinical signs of cerebral ischemia and allows to predict the need for shunt placement. So far, several studies of NIRS have failed to identify an ischemic threshold with adequate sensitivity and positive predictive value. In 99 patients undergoing CEA in regional anesthesia Samra et al. have established a 20% relative decrease of rSO_2 from baseline as a threshold for prediction of adverse neurological events. This cut-off point was determined to have a sensitivity of 80% and a specificity of 82%.[18] Similarly, Hirofumi et al. determined that reduction in rSO_2 of 16–18% from baseline was a predictor of cerebral ischemia during CEA.[19] The results of a large retrospective study analyzing data from 594 patients undergoing CEA in general anesthesia showed that previously described 20% drop in rSO_2 has a high specificity of 98% but concurrently a very poor sensitivity of 30%. The authors concluded that optimal cut-off is 12% decrease in rSO_2, but this threshold provided only a mediocre sensitivity of 75% and positive predictive value of 37%.[20]

There are several potential factors explaining the discrepancy between cerebral oximetry and clinical findings in carotid surgery. Firstly, it is the location of the sensor on the forehead over the frontal lobes. Since rSO_2 reflects predominantly tissue perfusion of prefrontal cortex, regional hypoperfusion affecting ACM circulation may be hardly detectable.[22] Secondly, NIRS has a limited ability to distinguish between intracranial and extracranial changes in oxygenation. In other words, the signal can be contaminated by blood flow in extracranial tissues.[23] Finally, a threshold for hypoxic injury, and thus for intervention, is difficult to precisely define because of a wide normal baseline range varying between 50% and 85%.[24]

A major limitation of this study was the low number of included patients (n = 38). Similarly, a relatively high frequency of neurological complications observed in this study was also caused by the small sample size. The long-term rate of periprocedural neurologic complications in a combined cohort of elective and early CEA in our center is less than 2%, which is comparable with a value of 1.5% found in a recent meta-analysis of elective CEA.[25]

Conclusions

Although non-operated ACI stenosis was a risk factor for development of perioperative neurological deficit, we were not able to demonstrate any association between the severity of contralateral ACI stenosis and the magnitude of rSO_2 changes during CEA. Moreover, the cerebral oximetry failed to detect the occurrence of perioperative cerebral ischemia. Therefore, we cannot recommend this method for routine monitoring in CEA surgery.

Conflict of interest
None.

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