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Original Article

The correlation of cerebral tissue oxygenation index and mean blood pressure in moyamoya patients

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Abstract: Moyamoya patients underwent cerebral revascularization surgery had higher risk for postoperative complications. Cerebral autoregulation impaired may be the main mechanism for postoperative complications. In our study, we used correlation coefficient *r* between cerebral tissue oxygenation index (TOI) and mean arterial blood pressure (MAP) to assessing of cerebral autoregulation. Total 30 moyamoya patients measured MAP and TOI. Spearman rank correlation coefficient *r* was calculated between MAP and TOI. 8 (26.67%) patients had postoperative neurological deterioration. The absolute value of *r* above 0.414 was adopted as diagnostic standard to predict postoperative neurological deterioration. Its sensitivity was 0.875, and specificity was 0.864. Correlation coefficients *r* was best predictors of postoperative neurological deterioration in our study. The meaning of correlation coefficient *r* need further study in a larger sample.

Keywords: Blood pressure, cerebral autoregulation, cerebral blood flow, moyamoya disease, near-infrared spectroscopy

Introduction

Moyamoya disease (MMD) is a chronic, occlusive cerebrovascular disease with unknown etiology characterized by bilateral steno-occlusive changes at the terminal portion of the internal carotid artery and an abnormal vascular network at the base of the brain [1]. The histopathology of MMD shows the large vessels of the circle of Willis display fibrocellular thickening of the tunica intima with excessive proliferation of the vascular smooth-muscle cells, marked tortuousness of the internal elastic lamina, and attenuation of the tunica media [2]. The collateral vascular networks that are characteristic of MMD show an irregular-shaped lumen and abnormal angioarchitecture [2]. The clinical features of MMD include ischemic strokes, intracerebral hemorrhages, seizures, and debilitating headaches [3]. Medical treatment with antithrombotics or vasodilators is ineffective [3]. Surgical revascularization for moyamoya disease including superficial temporal artery-middle cerebral artery (STA-MCA) anastomosis with or without indirect bypass is generally used as the standard surgical treatment for moyamoya disease [1, 4-7]. Cerebral revascularization surgery is a safe and effective treatment [7, 8], which prevents cerebral ischemic attack by improving cerebral blood flow [1]. However, the incidence of complications is higher in MMD patients who undergo surgery. Postoperative complications were defined as new neurologic events occurring within 30 days of each bypass operation [3]. Complications can be organized as ischemic injury [9], symptomatic hyperperfusion [10], and intracerebral hemorrhage [4], according to pathophysiological distinctions. The incidence of symptomatic hyperperfusion is 38.2% for adult-onset moyamoya patients [1]. According to the duration of complications, surgery complications divided into transient neurologic deterioration (TND) and permanent neurological deficit [5]. TND resolved between a few hours to 14 days [3]. Hyperperfusion after surgery [4, 6, 11, 12] and cerebral autoregulation impaired [5, 13] are the main mechanisms for TND. Patients with poorer cerebrovascular reactivity (CVR) are known to have potentially higher risk for hyperperfusion syndrome [1, 4]. CVR defined as an increase in CBF in response to a given vasodilatory stimulus [8], and is solely in connection with vasodilation or vasoconstriction [14]. Cerebral autoregulation can be affected by a number of physiological parameters, such as transmural pressure, carbon dioxide, autonomic function, and etc [14]. CVR measurements using various imaging techniques have been used to assess cerebral autoregulation of moyamoya patients [15]. Cerebral autoregulation is mediated by CVR [15]. So cerebral autoregulation impaired may be the main mechanism for TND.

Cerebral autoregulation can be explained by a tight coupling between oxygen supply and demand of the brain [16], and is essential to maintain a constant cerebral blood flow (CBF) in the context of changes in cerebral perfusion pressure [17]. Transcranial Doppler measurement of cerebral blood flow velocity in the middle cerebral artery has been widely to assess cerebral autoregulation [18]. Steiner *et al* studied correlation between the index of cerebral autoregulation assessed with blood flow velocity using transcranial Doppler, and cerebral tissue oxygenation index (TOI) recored with Near-infrared spectroscopy (NIRS) [19]. There is a good correlation between the two indexes. Transcranial Doppler has the characters of bedside availability, non-invasiveness and high temporal resolution in measuring changes in cerebral blood flow velocity [20]. However, transcranial Doppler can not measure the diameter of the vessel directly, the validity of using this technique is based on the assumption that changes in cerebral blood flow velocity represent changes in cerebral blood flow volumetric, it is assuming that the diameter of basal cerebral arteries does not change significantly in the face of changes in blood pressure [20]. NIRS is a neuroimaging tool for studying evoked hemodynamic changes within the brain, which also has the characters of bedside availability, non-invasiveness and higher temporal resolution [21]. NIRS has used to investigation of the changes in cerebral hemodynamic level. In our study, we used NIRS to calculating index of cerebral autoregulation of MMD and assessing the relationship between the autoregulation index and TND.

NIRS is a noninvasive technology that real-time monitors TOI [22, 23]. The technique is based on the Beer-Lambert law [23]. Oxygenated and deoxygenated hemoglobin have characteristic and different absorption spectra in near infrared light (700-950 nm) as it passed through tissue [22]. Their concentrations in tissue can be calculated by their relative absorption of light at these wavelengths [23]. The ratio of oxygenated hemoglobin to total hemoglobin represents the oxygen saturation of tissue under the sensor [23]. NIRS has the potential to identify impaired cerebral autoregulation and to detect cerebral hypoperfusion [22, 23]. The rationale of using NIRS to monitor cerebral autoregulation is based on the assumption that the oxygen content of brain is positively related to arterial oxygen saturation, cerebral blood flow (CBF), and oxygen tissue diffusivity, and negatively associated with the cerebral metabolic rate for oxygen [19]. NIRS measures hemoglobin oxygenation in a combination of arterial, venous and capillary blood [22, 23]. TOI measured by NIRS represents true tissue oxygen saturation and can identify cerebral ischemia [22]. TOI may give similar information as CBF with presumed stable arterial saturation, stable metabolism, and stable oxygen tissue diffusivity [19, 24]. The advantage for TOI monitoring CBF is noninvasive, continuous signal, and close proxy for CBF [24]. Under general anesthesia, with presumed stable anesthesia depth, stable metabolism, stable inspired oxygen concentration, and arterial carbon dioxide partial pressure ($PaCO_2$), comparisons of changes in local cerebral oxygen saturation taking place in response to changes in blood pressure, maybe give similar information as a comparison of changes in CBF and blood pressure. Cerebral autoregulation of moyamoya patients impaired, a fall in MAP will result in a decrease in CBF with resultant cerebral ischemia and infarction [8, 10]. It is important to perioperatively maintain the blood pressure at or above the preoperative baseline to prevent cerebral ischemia and infarction [10]. During revascularization procedures the patient's blood pressure is tent to decrease because of the anesthesia. The question is which blood pressure limits are best for moyamoya patients to increase CBF. The aim of this study was to investigate how blood pressure influences cerebral oximetry using NIRS. We also analyzed the incidence of postoperative complications.

Material and methods

Subjects

Data were prospectively collected during the clinical study. Total 30 moyamoya patients (range 23-61 years, mean 41.2 years), 18 men and 12 women, underwent STA-MCA anastomosis with general anesthesia in Tiantan hospital, from February 2012 until October 2012. Inclusion criteria: age >18 years, and adult-onset moyamoya patients. All patients underwent STA-MCA anastomosis. Exclusion criteria: anemia, diabetes mellitus, other serious systemic diseases, and postoperative infection. The protocol approved by the Ethical Committee. Informed consent was obtained from patients or their families.

Measurements and protocol

Anesthesia was induced with propofol (2-2.5 mg/kg), sufentanyl 0.3 μg/kg and rocnium 0.6 mg/kg, maintained with propofol 6-8 mg/kg/hour and remifentanyl 0.1-0.2 μg/kg/min. Depth of anesthesia maintained at bispectral index 40-50. Ventilation was controlled to obtain a target $PaCO_2$ 38-40 mm Hg [25]. Fractioned inspired oxygen is 60%. The blood pressure was maintained at or above the preoperative baseline, if necessary, by intravenous phenylephrine [26].

Mean arterial blood pressure (MAP) was measured directly from the radial artery using a standard pressure transducer (DELTRAN II Disposable pressure transducer, Utah Medical Products, United States). TOI of the frontal cortex was measured by NIRS (FORE-SIGHT MC-2000 Monitor, CAS Medical System, U.S.A.). The probe for TOI measurement was placed on the contralateral forehead because of the operative field. Monitoring frequency of FORE-SIGHT MC-2000 Monitor is 0.5 Hz. MAP and NIRS data were recorded simultaneously for 40-50 minutes, from the beginning of operation to separating STA.

Sensory evoked potential recording and motor evoked potential recording were performed during the operations.

Computed tomography (CT) was routinely measured in all patients 1 day after surgery. In some patients presenting with postoperative neurological deficit, magnetic resonance imaging (MRI) and/or CT were performed during or just after symptoms manifested. We examined the incidence of postoperative complications until hospital discharge.

Statistical analysis

Nonparametric statistical methods were used, because the majority of variables did not have significantly normal distributions. Spearman rank correlation coefficient, termed *r*, was calculated between MAP and TOI for each subject. The correlation coefficient *r* has a standardized value (range, -1 to +1). It indicates a close relationship that absolute value of correlation coefficient *r* is greater than 0.5. The larger the absolute value of *r* indicates that MAP and TOI has more close relationship. Statistical analysis in this article used the absolute value of *r*.

To detect diagnostic significance of correlation coefficient *r*, receiver-operating characteristics (ROC) with the area under the curve (AUC) were calculated.

For data analysis, patients were assigned to one of two groups: those with postoperative neurological deficit (group POND) and those without any signs of neurological change (group NORM). The values were expressed as the median ± standard deviations (SD). The data were tested for the normality (Kolmogorov-Smirnov test) at the group level. T Test and Fisher's exact test was used to present differences in characteristics between group POND and NORM. SPSS 17.0 for Windows (SPSS Inc., Chicago, Illinois, USA) was used to analyze the data. Significance was set at $P<0.05$ for all statistical tests.

Results

Total 30 adult-onset moyamoya patients with 30 surgeries in this study, mean age was 41.2 years. There were 12 (40%) females. 8 (26.67%) patients had postoperative neurological deterioration. 1 (3.33%) patient had postoperative cerebral infarction. His postoperative MRI showed fresh area of infarction. 7 (23.33%) patients suffered from temporary neurological deterioration. Their postoperative MRI/CT showed no ischemic changes, and the symptoms improved within 2 weeks. Sensory evoked potential and motor evoked potential unchanged during the operation for all patients.

Spearman rank correlation coefficient *r*, was calculated between MAP and TOI for every patient. Complications and Spearman correlation coefficient *r* were present in **Table 1**. It indicated a close relationship that absolute value of correlation coefficient *r* was greater than 0.5. There were 5 patients whose absolute value of correlation coefficient *r* was greater than 0.5. They developed neurological complications after the by-pass surgery.

Table 1. Spearman correlation coefficient *r* and clinical characteristics of the patients

Case No.	Sex/age (yr)	On-set type	BMI	MAP (mm Hg)	TOI	HR (beats/min)	*r*	Postoperative Symptoms
1	M/42	ischemic	27.0	57.8±1.4	81.9±5.2	55.0±1.3	-0.483	no
2	F/50	ischemic	26.7	67.1±3.7	86.7±3.1	55.6±3.5	0.243	dysarthria
3	F/42	ischemic	28.7	76.6±2.6	96.9±6.1	70.1±3.4	0.397	no
4	M/40	ischemic	26.0	60.4±2.2	95.6±5.4	60.1±2.6	0.582	aphasia
5	M/23	ischemic	21.5	73.1±3.0	92.1±4.8	78.9±1.3	-0.234	no
6	M/37	ischemic	26.4	65.2±0.7	100.7±3.1	61.3±1.8	-0.028	no
7	F/45	ischemic	23.4	65.1±1.2	101.5±4.9	66.4±1.8	0.134	no
8	F/38	hemorrhagic	22.7	70.8±3.1	81.1±3.8	72.1±5.1	-0.484	no
9	M/61	ischemic	26.8	80.8±3.7	64.2±1.3	56.0±3.7	0.431	dysarthria, hand motor
10	M/30	ischemic	28.7	76.3±4.1	89.1±11.0	66.1±5.9	0.130	no
11	M/36	hemorrhagic	22.5	58.2±1.5	86.0±5.9	63.8±4.2	0.280	no
12	F/59	ischemic	25.8	62.4±2.6	92.1±4.9	51.0±2.2	-0.049	no
13	M/36	ischemic	27.0	66.0±0.8	97.95±6.8	55.3±3.1	0.471	hemiparesis
14	F/52	ischemic	23.7	64.1±0.6	105.4±6.4	51.4±2.1	-0.548	seizure, hemiparesis
15	M/43	ischemic	27.0	58.9±5.8	96.1±8.1	65.0±3.3	-0.032	no
16	F/46	ischemic	25.0	58.4±0.2	78.3±2.3	53.1±1.5	-0.150	no
17	M/42	ischemic	21.8	66.0±1.0	84.5±2.8	72.3±1.9	0.133	no
18	F/44	ischemic	26.7	60.63±0.6	98.7±3.0	61.9±1.4	-0.690	infarction, hemiparesis
19	M/59	hemorrhagic	26.6	74.4±0.9	84.1±5.1	63.9±1.3	-0.355	no
20	M/28	hemorrhagic	18.3	59.1±1.1	75.1±2.3	79.5±1.2	-0.304	no
21	M/43	ischemic	24.2	63.8±0.8	87.0±1.5	68.5±1.6	0.445	no
22	F/43	hemorrhagic	22.3	60.5±1.6	71.4±8.3	71.0±3.9	-0.141	no
23	M/29	ischemic	24.7	66.8±3.5	91.9±5.1	58.2±1.3	0.215	no
24	M/41	ischemic	34.4	62.1±1.6	95.6±9.1	56.8±4.2	-0.681	aphasia, seizure
25	F/40	hemorrhagic	26.4	58.2±0.6	80.6±3.8	60.6±1.7	0.218	no
26	F/45	hemorrhagic	25.0	59.4±0.3	87.7±4.7	60.0±1.1	0.196	no
27	M/37	ischemic	26.6	66.5±0.5	73.6±4.2	52.5±1.6	-0.128	no
28	M/36	ischemic	23.2	70.3±2.5	90.0±2.7	58.9±1.6	0.095	no
29	F/26	hemorrhagic	22.6	74.4±1.6	82.5±5.2	55.7±1.6	0.085	no
30	M/44	ischemic	22.5	59.7±1.0	101.0±5.6	59.1±2.9	0.546	seizure

BMI, body mass index. MAP, mean arterial blood pressure. TOI, cerebral tissue oxygenation index. Values are listed as mean ± SD for MAP, TOI and HR.

For data analysis, patients were assigned to one of two groups: those with postoperative neurological deficit (group POND) and those without any signs of neurological change (group NORM). **Table 2** showed the analyzed results of the different factors between the groups. Absolute value of correlation coefficient *r* for group POND was higher than group NORM, and the difference was statistically significant. No Statistical significances were found between the groups with regard to sex, age, body mass index, and onset type.

In order to analyze the accuracy of correlation coefficient *r* in predicting postoperative neurological deterioration, receiver operating characteristic (ROC) analysis was performed. The area under the ROC is quantitative measure of the selectivity. It is the best selectivity when area under the curve (AUC) is 1.0, and it is the worst selectivity when AUC is 0.5. **Figure 1** showed the ROC plot of correlation coefficient *r*. AUC was 0.932 (P=0.01), and 95% confidence interval was 0.838 to 1.026. The best fitting cutoff point was 0.414. Its sensitivity was 0.875, and specificity was 0.864.

Discussion

STA-MCA anastomosis is a safe and effective treatment for moyamoya disease [6, 8]. TND following extracranial-intracranial bypass sur-

Table 2. Characteristics of group POND and group NORM

	POND	NORM	Statistical results (*P*)
Sex (F/M)	5/3	13/9	1.0
Age (yr) (mean ± SD)	46.00±7.98	39.50±9.12	0.086
BMI (mean ± SD)	26.73±3.51	24.56±2.58	0.076
Onset type			
Ischemic onset	7	15	0.391
Hemorrhagic onset	1	7	
Absolute value of *r*	0.52±0.14	0.21±0.14	0.001

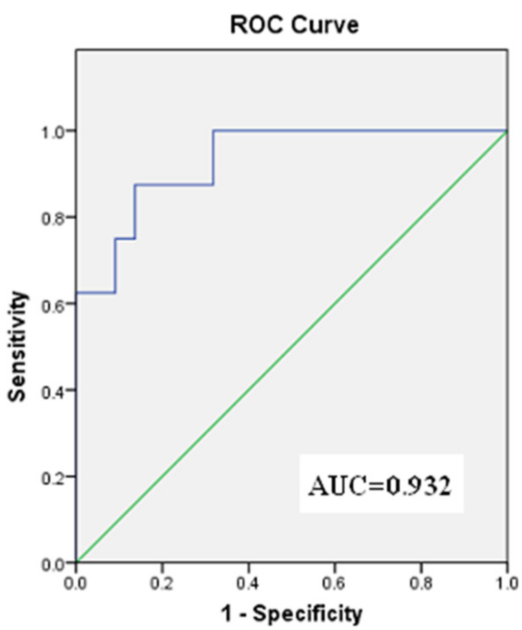

Figure 1. The receiver operating characteristic (ROC) curve for correlation coefficient *r*.

gery is a reversible focal neurological deficit lasting for a few days, without observable abnormalities in postoperative CT or MRI [11]. TND occurs in 4-38.2% [5, 6, 11]. Fresh area of infarction occurs in 1.7-8.3% [5, 11, 27]. In our study, incidence of postoperative cerebral infarction was 3.33%; incidence of TND was 23.33%. This frequency corresponds well with the references.

The mechanism of TND in patients with bypass surgery is unknown [6]. TND may be related to the following factors: focal hyperperfusion after surgery [5, 6, 11, 12], cerebral autoregulation impaired [5, 13], younger patients [5, 12], and on-set type [12]. Treatment of hyperperfusion is opposite to that for postoperative ischemia. If TND is caused by postoperative hyperperfusion, systemic arterial pressure must be strictly controlled to within the normal or lower range [5, 6, 13]. In our study, we simultaneously recorded MAP and TOI, and calculated their correlation coefficients *r*. Absolute value of correlation coefficient *r* in patients with postoperative neurological deterioration was higher than patients without neurological complications, and the difference was statistically significant. We took the absolute value of *r* above 0.414 as diagnostic standard to predict postoperative neurological deterioration. Its sensitivity was 0.875, and specificity was 0.864. Correlation coefficients *r* was the best predictor of postoperative neurological deterioration in our study, but it could not differentiate between infarction and TND. It may be a screening tool in the future.

How dose Spearman rank correlation coefficient *r* between MAP and TOI reflect cerebral autoregulation? Human and animal studies showed that time-domain correlation of MAP and TOI could assess of cerebral autoregulation [19, 24]. Steiner *et al* [19] calculated the index of cerebral autoregulation between TOI and MAP. They report positive values around +1 indicate disturbed autoregulation, and values around zero and negative values indicate good autoregulation. The correlation coefficient *r* has a standardized value (range, -1 to +1) [28]. In our study, absolute value of correlation coefficient *r* around 1 indicates disturbed autoregulation. It mean *r* around -1 indicate disturbed autoregulation, too. I could not explain this mechanism. In our study 3 patients had $r<-0.5$, and they all developed postoperative neurological deterioration. Czosnyka *et al* [29] reported similar case. They found a negative correlation between cerebral perfusion pressures and flow velocity [29]. In normal individuals, CBF remains nearly constant between cerebral perfusion pressure about 50 and 150 mm Hg [16]. Intact autoregulation absents correlation between cerebral perfusion pressure and CBF [15]. A positive $r>0.414$ signified a positive association between MAP and TOI, indicating a passive nonreactive behavior of the vascular bed. A negative value of $r<-0.414$ maybe reflect cerebrovascular excessive contraction. It was the higher MAP, the lower TOI. If so, raising blood pressure may aggravate cerebral ischemia. In our study, it means cerebral autoregulation impaired that absolute value of correlation coefficient *r* is above 0.414.

During anesthesia, raising blood pressure to improve cerebral perfusion is based on the assumption that cerebral autoregulation impaired and the positive correlation between MAP and TOI in surgery for MMD. However, the higher cerebral perfusion pressures maybe cause cerebral edema. If the correlation is negative, rising blood pressure would reduce cerebral perfusion pressure, The range of blood pressures matching to metabolic needs are narrowed for MMD patients with impaired cerebral pressure autoregulation. It seems that we will titrate and get the optimal cerebral perfusion pressure for each MMD patient. It has been postulated that continuous calculation correlation coefficient *r* allows to detecting an optimal blood pressure range that maximized improving cerebrovascular reactivity and avoiding cerebral ischemia or edema.

Disclosure of conflict of interest

None.

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