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Assessment of Cerebral Autoregulation and Optimal Arterial Pressure with Near-Infrared Spectroscopy in Traumatic Brain Injury Patients

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Abstract

In patients with severe traumatic brain injury (TBI), simultaneous measurement of intracranial and arterial blood pressure (ICP and ABP) allows monitoring cerebral perfusion pressure (CPP) and assessing cerebral autoregulation (CA). CPP, a difference between ICP and ABP, is the pressure gradient that drives oxygen delivery to cerebral tissue. CA is the cerebral vasculature ability to maintain stable blood flow despite changes in CPP and thus is an important homeostatic mechanism. Pressure reactivity index (PRx), moving Pearson correlation between slow waves in ICP and ABP, has been most frequently cited in literature over the past two decades as a tool for CA evaluation. However, in some clinical situations, ICP monitoring may be unavailable or contraindicated. In such cases, simultaneous mean arterial pressure (MAP) monitoring and Near-Infrared Spectroscopy (NIRS) can be used for CA assessment by cerebral oximetry index (COx), allowing calculation of the optimal blood pressure (MAPOPT). The purpose of this study was to compare regional oxygen saturation (rSO2)-based CA (COx) with ICP/ABP-based CA (PRx) in TBI patients and to compare MAPOPT derived from both technologies. Three TBI patients were monitored at the bedside to measure CA using both PRx and COx. Patients were monitored daily for up to three days from TBI. Averaged PRx and COx, and PRx and COx –based MAPOPT were compared using Pearson's correlation. Bias analysis was performed between these same CA metrics. Correlation between averaged values of COx and PRx was $R = 0.35$, $p = 0.15$. Correlation between optimal MAP calculated for COx and PRx was $R = 0.49$, $p < 0.038$. Bland–Altman analysis showed moderate agreement with a bias of 0.16 ± 0.23 for COx versus PRx and good agreement with a bias of $0,39 \pm 7.89$ for optimal MAP determined by COx versus PRx. Non-invasive measurement of CA by NIRS (COx) is not correlated with invasive ICP/ABP-based CA (PRx). However, the determination of MAPOPT using COx is correlated with MAPOPT derived from PRx. Obtained results demonstrate that COx is not an acceptable substitute for PRx in TBI patients. But in some TBI cases, NIRS may be useful for optimal MAP determination.

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1 Introduction

In patients with severe traumatic brain injury (TBI), simultaneous measurement of intracranial and arterial blood pressure (ICP and ABP) allows monitoring cerebral perfusion pressure (CPP) and assessing cerebral autoregulation (CA) [1,2]. CPP, a difference between ICP and ABP, is the net pressure gradient that drives oxygen delivery to cerebral tissue [3,4]. Cerebral autoregulation (CA) is a crucial mechanism for maintaining stable cerebral blood flow (CBF) [5,6]. Compromised cerebral autoregulation is main reason for hypo- or hyper- perfusion and a key element of secondary brain damage in patients with traumatic brain injury (TBI) [2,7]. Currently, clinicians have access to various methods for assessing the status of autoregulation [8,9]. According to Consensus (expert opinion), none of the existing techniques can be considered a standard for autoregulation assessment [10]. Nevertheless, the most straightforward and accessible method of surrogate evaluation of cerebral autoregulation in patients with acute cerebral injury remains the pressure reactivity index (PRx) [9–11]. PRx calculates as a moving correlation coefficient between intracranial pressure (ICP) and arterial blood pressure (ABP) signals [12]. However, in some clinical situations, ICP monitoring may be unavailable or contraindicated. In such cases, simultaneous ABP monitoring and Near-Infrared Spectroscopy (NIRS) can be used for CA assessment by cerebral oximetry index (COx) [13,14], allowing calculation of the optimal blood pressure (MAPOPT) [15]. The purpose of this study was to compare the regional oxygen saturation (rSO2)-based CA (COx) with ICP/ABP-based CA (PRx) in TBI patients and to compare the MAPOPT derived from both technologies.

2 Methods

In this work, we present retrospective data of multimodal neuromonitoring of 3 patients with severe TBI (Table 1, 2). The study was conformed to the Declaration of Helsinki standards and was approved by the Burdenko Institute Ethics Committee. At the time of hospitalization, two out of three patients were in a coma, Glasgow Coma Scale (GCS) 7, one patient was confused (GCS 12), but then worsened to coma. All patients required mechanical ventilation and had direct arterial blood pressure (ABP) monitoring using a catheter in the radial artery. All patients were treated according to international guidelines [16]. The “Codman” ICP probes (Codman & Shurtleff Inc., Raynham, MA) were installed at the Kocher point to a depth of 2 cm and connected to “ICP Express Codman” and bedside “Philips IntelliVue MP60”(Philips Medical Systems, Best, The Netherlands) monitors.

The patients were connected to NIRS INVOSTM 5100 (cerebral/somatic oximetry monitor, Covidien, Boulder, CO) using self-adhesive sensors attached to each side of the forehead. NIRS monitoring was carried out only for the first 3 days after hospitalization. COx coefficient was carried out in a time window of 6 hours, with a successive repetition of 6 times (table 3). After excluding artifacts, we compared six 6-hour epochs (36 hours in total) of ABP, ICP, rSO2 simultaneous recordings for each patient. All raw and calculated signals (ABP, ICP, rSO2, COx, PRx) were analyzed using ICM+ software (University of Cambridge, Cambridge, UK) [17]. The COx was calculated as moving correlation between the slow waves of rSO2 and MAP [13]. Averaged COx within each 10-s window was collected as 30 data points to monitor each COx in a 300-s window. Right and left sides

were monitored on all patients, and an average COx from both sides was calculated for correlation analysis. The PRx was calculated as a coefficient between slow waves of ICP and MAP [12]. The COx and PRx measurements from the monitoring onset were binned into 5-mmHg increments of MAP for analysis. Optimal MAP for each patient was defined as the MAP with the best autoregulation (i.e., MAP with the lowest COx and PRx). Statistical analyses were performed using Statistica10.0 (StatSoft, USA). The Blunt-Altman method was used to compare PRx and COx. Correlations between COx and PRx values were done using Pearson's correlation coefficient. All data are presented as mean ± standard deviation.

3 Results

All patients were male, 23,6 +/− 6,6 years old (Table 1). According to the Marshall CT classification, two patients had diffuse brain injury DI II, and one patient1 had DI I [18]. Data for all period of monitoring are presented in Table 2. MAP was 89 +/− 7,6 mmHg, ICP was 12 +/− 7,7 mmHg, CPP was 78 +/− 8,9 mmHg, rSO2-R was 74+/− 4,3%, rSO-L was 68,3 +/− 3,8 %. Correlation between the averaged values of COx and PRx was not significant $R = 0.35$, $p = 0.15$ (Figure 1a). Correlation between optimal MAP calculated for COx and PRx was $R = 0.49$, $p < 0.038$ (Figure 1c).

Bland–Altman analysis showed moderate agreement with a bias of 0.16 ± 0.23 for COx versus PRx (Figure 1b). Good agreement with a bias of 0,39 ± 7.89 for optimal MAP determined by COx versus optimal MAP determined by PRx (Figure 1d).

4 Discussion

In the presented clinical observations, we compared invasive (PRx) and non-invasive (Cox) methods for autoregulation evaluation. We specifically selected similar patients with severe TBI and diffuse brain injury (DI I and DI II) [18], with unclear or questionable indications of ICP monitoring [1,19]. Nevertheless, in the absence of indications for invasive ICP monitoring, blood pressure control remains an essential option in the management of such patients; thus, the presence of target optimal blood pressure is probably beneficial. A number of studies have shown that high deviation from optimal blood pressure is associated with worse outcomes in patients with traumatic brain injury [20–22]. At the same time, in patients after cardiac arrest, maintaining perfusion close to optimal blood pressure improved brain oxygenation [23]. We await the final results of the COGiTATE Study [24] and hope that the protocol for optimizing ABP/ CPP will take an important place in the management of patients with acute brain damage. We hope that wavelet analysis and controlled generation of slow waves will improve the quality of autoregulation monitoring by reducing signal noise and expand the possibilities of non-invasive assessment of autoregulation.

5 Conclusions

The obtained results demonstrate that NIRS-derived COx is not an acceptable substitute for ICP/ABP-derived PRx monitoring in TBI patients. But in some TBI cases, NIRS may be useful for optimal MAP determination.

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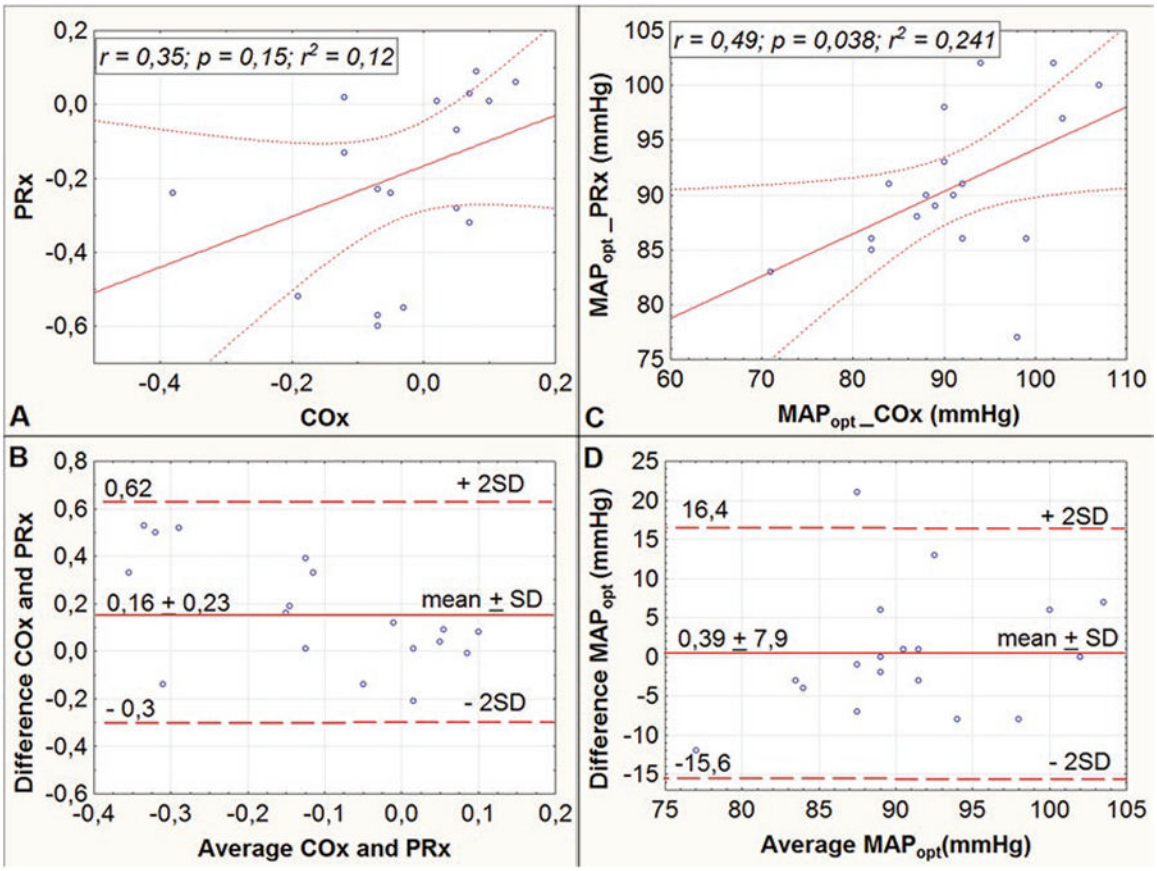

Fig. 1.

(**a, b**) – correlation and Bland Altman plot Cox and PRx; (**c, d**) – correlation and Bland Altman plot MAPopt_PRx and MAPopt_ORx

Table 1

Clinical characteristics of the patients

Pts no.	Age(years)	Sex	CT Marshall	GCS	GOS
1	16	M	DI II	7	4
2	27	M	DI I	7	5
3	28	M	DI II	12[a]	1

CT Marshall classification of TBI pts. based on computerised tomography, *DI* diffuse injury

[a]Patient N° 3 was GCS 12 at the time of hospitalisation, but later deteriorated to GCS 6

Table 2

Physiologic variables (mean ± SD) for all periods of monitoring

Pts no.	MAP(mmHg)	ICP(mmHg)	COx-R	COx-L	PRx
1	92 ± 8	14 + 3	0.02 + 0.32	0.04 + 0.32	−0.32 + 0.33
2	89 ± 7	9.8 + 3	0.08 + 0.25	0.09 + 0.25	0.11 + 0.29
3	86 ± 8	9.5 + 5	0.02 + 0.24	0.03 + 0.28	0.27 + 0.35

MAP mean arterial pressure, *ICP* mean intracranial pressure, *COx-R, and COx-L* cerebral oximetry index on the right and left side, *PRx* pressure reactivity index

Table 3

Data from six sequential 6-h periods of monitoring

Pts no.	6 h-period	COx	PRx	MAP_{opt}_COx	MAP_{opt}_PR
1	1	−0.19	−0.52	94	102
	2	−0.03	−0.55	107	100
	3	−0.07	−0.6	91	90
	4	−0.07	−0.57	103	97
	5	0.07	−0.32	98	77
	6	0.05	−0.28	92	86
2	1	0.07	0.03	84	91
	2	0.02	0.01	88	90
	3	0.14	0.06	99	86
	4	0.05	−0.07	82	86
	5	0.1	0.01	82	85
	6	0.08	0.09	87	88
3	1	−0.12	0.02	102	102
	2	−0.38	−0.24	90	98
	3	−0.07	−0.23	89	89
	4	−0.05	−0.24	71	83
	5	−0.12	−0.13	90	93
	6	−0.09	0.12	92	91

Cox averaged cerebral oximetry index, *PRx* averaged pressure reactivity index, MAP_{opt}*_COx and* MAP_{opt}*_PRx* optimal blood pressure estimated by COx and PRx