Critical Closing Pressure of Cerebral Circulation at Concomitant Moderate-to-Severe Traumatic Brain Injury

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Abstract

Background: Critical closing pressure (CrCP) is the pressure below which local pial blood pressure is inadequate to prevent blood flow cessation. The state of cerebral CrCP in patients with concomitant moderate-to-severe traumatic brain injury (cTBI) after brain lesions surgery remains poorly understood.

Aim: The aim of our study was to establish the dynamics of CrCP after intracranial surgery in traumatic brain injury (TBI) patients with polytrauma. Material and methods: Results of the treatment of 70 patients with moderate-to-severe cTBI were studied (Male: Female – 39:31, mean age –33.2 ± 12.2 years). Depending on intracranial surgery, patients were divided into 2 groups. All patients were subjected to transcranial Doppler of both middle cerebral arteries, and evaluation of mean arterial pressure (MAP). Based on the data obtained, CrCPs were calculated. Significance was preset to $P < 0.05$. Results: Mean CrCP values in each group were significantly higher than a reference range ($p < 0.01$). There was no significant difference in CrCP values between the left and right hemispheres in the group 1 ($p = 0.789$). In the group 2, mean CrCP values on the unoperated side remained significantly lower than on the operated side ($p = 0.000011$) even after intracranial surgery. In group 1, mean CrCP values were significantly lower than on the surgery side in the group 1 ($Z = 3,4$; $p = 0.043$). Conclusion: CrCP values in concomitant moderate-to-severe TBI after removing brain lesions and without surgery were significantly higher than referral data. Even after removal of brain

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F. Scholkmann et al. (eds.), *Oxygen Transport to Tissue XLIII*, Advances in Experimental Medicine and Biology 1395, https://doi.org/10.1007/978-3-031-14190-4_27

lesions volumes in patients with concomitant moderate-to-severe TBI, CrCP values on the surgery side remained markedly higher than on the side opposite to the removed lesion volumes.

Keywords

Intracranial hematoma · Intracranial pressure (ICP) · Cerebral perfusion pressure (CPP) · Microvascular perfusion

1 Introduction

According to current guidelines, based on a large body of research, adequate microvasculature perfusion maintenance is an essential component of intensive therapy of head injury. However, cerebral perfusion pressure (CPP) stabilisation at severe multiple trauma and shock, as caused by road traffic accidents, often resembles a Sisyphean task.

CPP can be calculated as a difference between mean arterial pressure (MAP) and brain parenchymal pressure, which is usually considered equal to intracranial pressure (ICP) [1].

Furthermore, ICP gradients development in the injured brain can seriously complicate the calculation of local CPP, which was confirmed by Sun et al. [2].

According to Kaczmarska et al., cerebral microvascular perfusion might be more accurately described by the closure margin, or 'effective' perfusion pressure, defined as the difference between MAP and the pressure below which arterial and arteriolar vessels collapse and local blood flow ceases [3].

This parameter has been defined as zero flow pressure or critical closing pressure (CrCP) [4]. It is believed that when arterial blood pressure reaches the CrCP value, the pulse fluctuations smooth out, followed by an avalanche-like collapse of the microvascular bed [5]. Previously, Richards et al. showed that CrCP is highly correlated with invasively established CPP and ICP values [6].

Therefore, the measurement of CrCP is of practical importance, since it allows a non-invasive assessment of the cerebral perfusion state when invasive monitoring of ICP is difficult or impossible [7].

It should be noted that the state of cerebral CrCP in patients with concomitant moderate-to-severe TBI (cTBI) after brain lesions volume surgery remains poorly understood.

The aim of our study was to establish the dynamics of CrCP after intracranial surgery in TBI patients with polytrauma.

2 Methods

This non-randomised single-centre study complies with the Declaration of Helsinki. The protocol was approved by the local Ethics Committee of Nizhny Novgorod Clinical Hospital named after N.A. Semashko. All patients and their families provided written informed consent. The inclusion and exclusion criteria are in Table 1.

2.1 Population

The study involved 39 men and 31 women with concomitant moderate-to-severe TBI; mean age of 33.2 ± 12.2 years treated at Nizhny Novgorod Regional Trauma Centre Level I of Regional Clinical Hospital named after N.A. Semashko. The recruitment period was from January 2013 to

Table 1 Inclusion and exclusion criteria

Inclusion criteria	Exclusion criteria
1. Between 16 and 70 years old	1. Less than 16 and more than 70 years old
2. Polytrauma with moderate-to-severe TBI within 3–5 days after a brain injury	2. CT Marshall grade III, VI (any non-evacuated intracranial hematoma, parenchymal volume lesions, etc.)
3. GCS less than 12 on admission	3. Injury severity score less than 9 and more than 36
4. Injury severity score between 9 and 36	4. Any cardiovascular injury/diseases history
5. CT Marshall grades I, II, V	

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December 2020. All patients were polytraumatised. They all received therapy according to the Guideline of TBI treatment and Advanced Trauma Life Support [8]. The patients were divided into two groups. The first group included 37 TBI patients without volume lesions. The second group comprised 33 TBI patients after intracranial surgery within the first 3 days of the injury. Glasgow Coma Scale (GCS) severity was 10.4 ± 1.5 and 9.7 ± 1.7 in the first and second group, respectively. The injury severity scores (ISS) were 31 ± 9 and 32 ± 8 in the first and second group, respectively. Among 33 patients that underwent meningeal hematomas surgery, the epidural hematomas were removed in 7 patients, subdural hematomas in 32, and multiple hematomas in 4 patients.

2.2 Critical Closing Pressure Calculation

Cerebral circulation CrCP was calculated using Eq. 1 proposed by Ogoh [9]:

$$\mathrm{CrCP} = \mathrm{ABPs} - \frac{\mathrm{ABPs} - \mathrm{ABPd}}{\mathrm{Vs} - \mathrm{Vd}} \times \mathrm{Vs} \quad (1)$$

where ABPd – diastolic arterial pressure (mmHg), ABPs – systolic arterial pressure (mmHg), CrCP – critical closing pressure (mmHg), Vd – diastolic cerebral blood flow velocity (cm/s), Vs – systolic cerebral blood flow velocity (cm/s).

Reference range CrCP was chosen according to Ogoh S. as 33 ± 2 mmHg.

Diastolic and systolic arterial blood pressure was monitored noninvasively (IntelliView MP5, Philips Medizin Systeme, Germany). Cerebral blood flow velocity (CBFV) in both middle cerebral arteries (MCA) was bilaterally measured using ultrasound Doppler with a 2-Mhz probe for 20 minutes (Sonomed 300 M, Spectromed, Russia) [9]. We used the intensive care monitor 'Centaurus-CrCP' (Ver. 3.0, Privolzhsky Research Medical University, Russia). PaO_2, $PaCO_2$, and core temperature were within normal ranges. All patients did not require sedation or pharmacological support of blood pressure and were normotensive.

2.3 Statistical Analysis

Continuous variables are expressed as median [interquartile range]. The data were evaluated for normality distribution using the Shapiro-Wilk test. The differences in CrCP values were assessed using a nonparametric Sign test. The significance level was set at 0.05. All analyses were performed using the software package Statistica 7.0 (Statsoft Inc., USA).

3 Results

Mean CrCP values in each group were significantly higher than a reference range ($p < 0.01$).

We did not find a significant difference in CrCP values between the left and right hemispheres in the first group (94 [86;107] mmHg vs. 94 [87;108] mmHg, respectively, $Z = -0.267$; $p = 0.789$).

In the second group, mean CrCP values on the unoperated side ('non-surgery' side) remained significantly lower than on the operated side ('surgery' side) (91 [85;110] mmHg vs. 87 [78;110] mmHg, respectively, $Z = 4.4$; $p = 0.000011$) even after intracranial surgery.

There was no statistically significant difference between mean CrCP on the 'non-surgery' side compared to mean CrCP values in the first group ($Z = 0.182$; $p = 0.855$).

In addition, in the first group, mean CrCP values were significantly lower than on the surgery side in the second group ($Z = 3,4$; $p = 0.043$).

CrCP values in the 'surgery' side at various types of removed intracranial volume lesions showed no significant differences ($p > 0.05$).

Also, we did not find significant effects of patient age on CrCP value ($p > 0.05$).

4 Discussion

For the first time, a theoretical model that describes the collapse of small vessels with a regional arterial fall was proposed by Burton in 1951 [4].

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It has been noted that a drop in CrCP by half leads to perfusion collapse in half of the total number of capillaries proportionally. Thus, the importance of CrCP is evident [11, 12].

The aim of our study was to establish the dynamics of CrCP after intracranial surgery in TBI patients with polytrauma.

We have shown that CrCP values were significantly higher than normal in all concomitant moderate-to-severe TBI patients.

We assume that one of the possible reasons may be a decrease in the catecholamines concentration due to shock relief in the subacute stage of polytrauma, which leads to cerebral vasodilation. This CrCP rise is confirmed by previous studies [7].

Another possible reason might be related to the increase of pleural cavities pressure due to the lung contusion, haemo- or pneumothorax, which was found in our study in all patients, and is consistent with previous research [13].

We have shown that CrCP significantly differs in TBI patients after intracranial surgery.

Significant CrCP increase ($p = 0.00032$) on the side of surgical intervention apparently has several reasons. We suggest that cerebral microvascular vasospasm or microthrombosis in the craniectomy area is the most possible reason [5]. Although evident signs of 'dopplerographic' vasospasm were found in only 27 patients (81.2%), the putative CBF turbulence caused by proximal vasospasm may lead to an underestimation of its frequency [14].

Postoperative CT scans did not reveal any volume brain lesion, which reflects the ICP constancy as well the absence of any ICP gradients [2, 15]. Therefore, CrCP increase probably relates to vascular wall tone increase, which was reported in previous research [13, 15].

5 Conclusion

CrCP values in concomitant moderate-to-severe TBI after removal volume lesions and without surgery were significantly higher than referral data.

Even after removing volume brain lesions in the patients with concomitant moderate-to-severe TBI, CrCP values on the surgery side remained significantly higher than the side opposite to where volume lesions were removed.

Acknowledgments KT and AT were supported by a Grant-in-Aid for Exploratory Research from the Privolzhsky Research Medical University. OSG was supported by RSF 20-15-00090, RSF 19-15-00201, grant 075-15-2019-1885; DB was supported by NIH R01 NS112808.

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