Published in final edited form as:

Acta Neurochir Suppl. 2018 ; 126: 21–24. doi:10.1007/978-3-319-65798-1_5.

Cerebral Arterial Compliance in Traumatic Brain Injury

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Abstract

Objective.—The main role of the cerebral arterial compliance (cAC) is to maintain the stiffness of vessels and protect downstream vessels when changing cerebral perfusion pressure. The aim was to examine the flexibility of the cerebral arterial bed based on the assessment of the cAC in patients with traumatic brain injury (TBI) in groups with and without intracranial hematomas (IH).

Materials and Methods.—We examined 80 patients with TBI (mean 35.7 ± 12.8 years, 42 men, 38 women). The first group included 41 patients without IH and the second group included 39 polytraumatized patients with brain compression by IH. Dynamic ECG-gated computed tomography angiography (DHCTA) was performed in 1-14 days after trauma in the first group and in 2-8 days after surgical evacuation of the hematoma in the second group. Amplitude of ABP, systole and diastole duration were measured noninvasively. Transcranial Doppler were measured simultaneously with DHCTA. The cAC was calculated by the formula proposed by Avezaat.

Results.—The cAC was significantly decreased ($p < 0.001$) in both 1st and 2nd group in comparison with normal data. The cAC in the 2nd group was significantly decreased than in the 1st group, both on the side of the former hematoma ($p = 0.017$).

Conclusion.—The cAC in TBI gets significantly lower as compared to (the conditional norm) ($p < 0.001$). After removal of the intracranial hematomas compliance in the perifocal zone remains much lower ($p = 0.017$) as compared to compliance of the other brain hemisphere.

Keywords

brain injury; intracranial hematoma; cerebral arterial compliance

1 Introduction

The secondary insults to patients with traumatic brain injury (TBI) is greatly affected by changes in the compliance and stiffness of cerebral vessels, the walls of downstream vessels

have no external elastic membrane, therefore the cerebral capillary network becomes vulnerable to intracranial and intravascular pressure surges [1].

One of the features characterizing the flexibility of the vascular network and its resistance to the said changes is the cerebral arterial compliance (cAC) [2],

The state of the cAC is of great importance for the brain microcirculation. Since the brain is located within an inextensible cranial cavity and is surrounded by an incompressible fluid, the compensation of intracranial pressure surges caused by the pulse wave passage through the brain blood vessels occurs also through the reciprocal changes in arterial lumens [3],

Thus, the higher is the cAC, the greater is the compliance of a vascular wall and, respectively, the better is the capacity of a vessel to change its lumen (i.e. vasomotion phenomenon) and thereby to maintain the adequate capillary bed perfusion [4],

Information on the compliance and stiffness of the cerebral vascular bed in the damaged brain is currently rather inconsistent, [2,5] and the aspects of the cAC reaction to the IH development and the disturbed cerebral blood flow (CBF) in the case of TBI still remain underinvestigated [3,6],

The main objective of this study was to examine the flexibility of the cerebral arterial bed based on the assessment of the cAC in TBI groups with and without IH.

2 Materials and Methods

The study complies with the Declaration of Helsinki (adopted in June 1964 (Helsinki, Finland) and revised in October 2000 (Edinburgh, Scotland)) and was approved by the local Ethics Committee. All the patients gave informed consent to participate in the study. We examined 80 TBI patients who were treated at the Departments of Neurosurgery in 2013–2016. All patients were divided into 2 groups. The 1st group included 41 patients with TBI without the development of IH. The 2nd group included 39 patients with TBI and IH.

Dynamic Helical Computed Tomography Angiography

All patients were subjected to dynamic helical computed tomography angiography (DHCTA) [7] by tomograph Philips Ingenuity CT (Philips Medical systems, Cleveland, USA). DHCTA was performed 1–12 days after TBI (mean 4 ± 3 days) in the 1st group and 2–8 days (mean 4 ± 2 days) after trauma and surgery of the hematoma in the 2nd group.

DHCTA was performed of 16 volume of the data, 160 mm in thickness, within 60 s with a contrast agent (Ultravist 370, Schering, Germany) administered [7], During or immediately after DHCTA the monitoring of the transcranial Doppler (TCD) of the MCA was recorded bilaterally with 2-MHz probes within 10 minutes [8],

Amplitude of arterial blood pressure (ABPamp) and ECG-gated duration of the systole (Tsyst) and the diastole (Tdia) were measured noninvasively (IntelliView MP5, Philips Medizin Systeme, Germany). The system appearance is shown in

The data volume was transferred to the workstation Philips Extended Brilliance Workspace (Philips Healthcare Netherland B.V., Best, the Netherlands) and MATLAB 2013b (The MathWorks Inc., Natick, MA, USA).

The CBF and cerebral blood volumes (CBVs) were calculated from the DHCTA data with complex mathematical procedures, using the “direct flow model” algorithm. [9],

The systolic–diastolic values of the MCA diameters (Dsys and Ddia) were determined in CTA series in the proximal part of the M1 of the both MCA.

Amplitude of regional CBVs oscillation (ΔCBV) was calculated as the difference between CBVs, which flowed through the MCA in systole (CBVsys), and diastole (CBVdia). We used the formulas (1, 2 and 3) proposed by de Jong, Alexandrov and Avezaat [8,9,10],

$$\Delta CBV = CBV_{sys} - CBV_{dia}, \quad (1)$$

$$\Delta CBV = \frac{\pi}{4} \times D_{sys}^2 \times CBFV_{sys} \times T_{sys} - \frac{\pi}{4} \times D_{dia}^2 \times CBFV_{dia} \times T_{dia}. \quad (2)$$

$$cAC = \Delta CBV \div ABP_{amp}. \quad (3)$$

Reference range cAC was chosen according Ikdip K. as $0.105 \pm 0.043 \text{ cm}^3/\text{mmHg}$ [11],

Statistical analysis.—The t-test for dependent samples was utilized to analyze differences in means of parameters between the ipsilateral and contralateral sides of the temporal lobes. The program Statistica 7.0 (StatSoft Inc., USA) was used for the analysis. Data are presented as Mean $\pm$ SEM. A significance level was preset to $p < 0.05$.

3 Results

Sex distribution had a male predominance (38 women, 42 men). Mean age was 35.7 ± 12.8 years (range 17–87). The wakefulness level according to GCS averaged 9.7 ± 2.5 in the 1st group and 10.1 ± 2.5 in the 2nd group.

The acquired and analyzed data are summarized in Table 1.

The cAC was significantly decreased ($p < 0.001$) in both the 1st and 2nd group TBI (with or without IH) in comparison with normal data ($p < 0.001$).

The cAC in the second group was significantly decreased than in the first group, both on the side of the former hematoma ($p = 0.017$)

There was no significant difference in cAC between the perifocal zone of the former hematoma and the same locus of the contralateral hemisphere ($p = 0.172$).

4 Discussion

It is currently shown that the disturbed microcirculation play a key role in the development of hypoperfusion episodes in patients with TBI. The cAC is deemed to be one of the most important indices, which reflects the degree of the compliance and resistance to deformation of the arterial network in response to spontaneous fluctuations in systemic hemodynamics [12],

The dynamics of the cAC in TBI remains to date poorly studied. At the same time the cAC assessment is required as it may serve as a predictor for an ischemic brain injury [2],

In our study we have shown that the cAC in TBI is significantly and statistically reliably reduced as compared with the norm.

In our opinion, there may be several reasons for such cAC dynamics but all of them are more or less associated with the development of a brain edema [1].

Firstly, the development of a mixed cerebral edema increases the arterial wall stiffness, which affects the cAC [13],

Secondly, an edema development causes the diastolic compression of a pial bed, thus, significantly reducing the capillary bed capabilities to retain its lumen, and accordingly, to maintain the vasomotor activity [9].

It should be noted that the development of the IH changes even more the cAC value.

Here we have shown that even after the removal of an IH the cAC in it perifocal zone still remained significantly lower than with the TBI without the IH development.

This effect may be explained by Behzadi data, which have shown that the CBF is dependent not only on cAC but on the diameter of blood vessels as well, which may considerably vary in case of TBI because of both the macro- and microvascular vasospasm [14].

To our knowledge, it is impossible to carry out the dynamic assessment of the cAC without a repeated DHCTA. In our study we failed to eliminate a mathematical error associated with the measurement of the MCA diameters [8, 15].

Thus, our results enable us to conclude that in the early period of TBI some pronounced changes in the cAC and cerebral microcirculation are observed, which are exacerbated by the development of enveloped hematomas.

Our findings may have the certain practical significance for optimizing the brain edema therapy, which would prevent the development of cerebral perfusion disorders in patients with TBI.

5 Conclusion

The cAC in TBI gets significantly lower as compared to the normal condition ($p < 0,001$). After removal of the ICH the compliance in the perifocal zone remains much lower ($p = 0,017$) as compared to compliance of the other brain hemisphere.

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Fig. 1.
The Investigation System Appearance. A white arrow indicates a computer tomograph, a black arrow shows a TCD, a blue arrow shows ECG-ABP monitor and a gray arrow marks a syringe-injector.

Table 1

Comparison of the analyzed parameters.

		Amplitude ABP (mmHg)	CBV_{MCA} (cm³)	cAC (cm³/mmHg)
1	Group 1	63.9±11.5	2.7±0.9	0.049±0.035
2	Group 2 (ipsilateral sides)	65.3±12.2	2.6±1.8	0.026±0.017
3	Group 2 (contralateral sides)	65.3±12.2	2.9±1.4	0.037±0.03
	P (1-2)	0.539	0.756	0.017 [*]
	P (1-3)	0.427	0.351	0.172
	P (2-3)	0.166	0.62	0.116

^{*} Significant difference (p<0.01).