Cerebral Arterial Compliance in Traumatic Brain Injury

Michael Dobrzeniecki, Alex Trofimov, and Denis E. Bragin

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 (IHs).

Materials and Methods: We examined 80 patients with TBI (mean age, 35.7 ± 12.8 years; 42 men, 38 women). Group 1 included 41 patients without IH and group 2 included 39 polytraumatized patients with brain compression by IH. Dynamic electrocardiography (ECG)-gated computed tomography angiography (DHCTA) was performed 1–14 days after trauma in group 1 and 2–8 days after surgical evacuation of the hematoma in group 2. Amplitude of arterial blood pressure (ABP), as well as systole and diastole duration were measured noninvasively. Transcranial Doppler was 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 groups 1 and 2 compared with normal data. The cAC in group 2 was significantly decreased compared with group 1, both on the side of the former hematoma ($p = 0.017$).

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

Keywords Brain injury · Intracranial hematoma · Cerebral arterial compliance

Introduction

The secondary insults to patients with traumatic brain injury (TBI) are 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. Because 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 the cAC, the greater the compliance of a vascular wall and, respectively, the better is the capacity of a vessel to change its lumen (i.e., the 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 aspects of the cAC reaction to the intracranial hematoma (IH) development and the disturbed cerebral blood flow (CBF) in the case of TBI 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.

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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 two groups. Group 1 included 41 patients with TBI without the development of IH; group 2 included 39 patients with TBI and IH.

Dynamic Helical Computed Tomography Angiography

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

DHCTA was performed on 16 volumes of data, 160 mm in thickness, within 60 s of administration of contrast agent (Ultravist 370; Schering, Berlin, Germany) [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 min [8].

Amplitude of arterial blood pressure (ABPamp) and electrocardiography (ECG)-gated duration of the systole (T_{sys}) and the diastole (T_{dia}) were measured noninvasively (IntelliView MP5; Philips Medizin Systeme, Hamburg, Germany). The system appearance is shown in Fig. 1.

The data volume was transferred to the workstation [Philips Extended Brilliance Workspace (Philips HealthCare, Best, The Netherlands) and MATLAB 2013b (The MathWorks, Natick, MA, USA)].

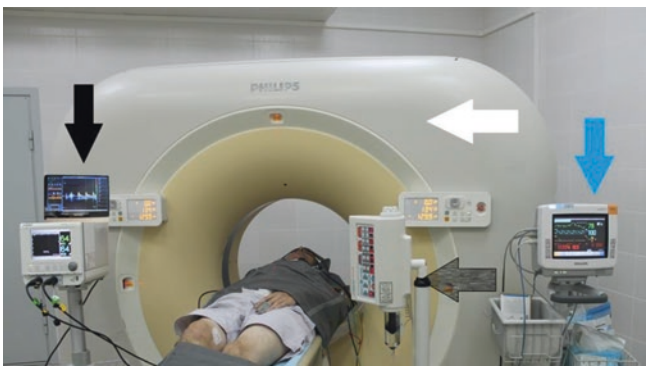

Fig. 1 The investigation system appearance. A *white arrow* indicates a computer tomograph, a *black arrow* shows a TCD, a *blue arrow* shows ECG-ABP monitor and a *gray arrow* marks a syringe-injector

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 middle cerebral artery (MCA) diameters (D_{sys} and D_{dia}) were determined in CTA series in the proximal part of the M1 of both MCAs.

The amplitude of regional CBV oscillation (ΔCBV) was calculated as the difference between CBVs which flowed through the MCA in systole (CBV_{sys}), and diastole (CBV_{dia}). We used the formulas (1, 2 and 3) proposed by de Jong, Alexandrov and Avezaat [8–10].

$$\Delta CBV = CBV_{sys} - CBV_{dia} \tag{1}$$

$$\Delta CBV = \frac{\pi}{4} \times {D_{sys}}^2 \times CBFV_{sys} \times T_{sys} - \frac{\pi}{4} \times D_{dia}^2 \times CBFV_{dia} \times T_{dia} \tag{2}$$

$$cAC = \Delta CBV \div ABP_{amp} \tag{3}$$

Reference range cAC was chosen according to Ikdip [11] as 0.105 ± 0.043 cm^3/mmHg.

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, Tulsa, OK, USA) was used for the analysis. Data are presented as mean ± SEM. A significance level was preset to $p < 0.05$.

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 group 1 and 10.1 ± 2.5 in group 2.

The acquired and analyzed data are summarized in Table 1.

The cAC was significantly decreased ($p < 0.001$) in both groups 1 and 2 (TBIs without or with IH, respectively) in comparison with normal data ($p < 0.001$).

The cAC in group 2 was significantly decreased compared with group 1, both on the side of the former hematoma ($p = 0.017$).

Table 1 Comparison of the analyzed parameters

		Amplitude ABP (mmHg)	ΔCBV_{MCA} (cm^3)	cAC (cm^3/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[a]
	P (1–3)	0.427	0.351	0.172
	P (2–3)	0.166	0.62	0.116

[a]Significant difference ($p < 0.01$)

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

Discussion

It is currently shown that the disturbed microcirculation plays 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, 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 compared with the norm.

In our opinion, there may be several reasons for such cAC dynamics, but all of them are associated, more or less, 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 the perifocal zone remained significantly lower compared to TBI without IH development.

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

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].

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

Conclusion

The cAC in TBI gets significantly lower compared with the normal condition ($p < 0.001$). After removal of the intracranial hematomas, the compliance in the perifocal zone remains much lower ($p = 0.017$) compared with compliance of the other brain hemisphere.

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Conflicts of interest statement We declare that we have no conflict of interest.

References

1. Nichols W, O'Rourke M. McDonald's blood flow in arteries: theoretical, experimental and clinical principles. Oxford: University Press; 2005.
2. Esther A, Warnert H. Noninvasive assessment of arterial compliance of human cerebral arteries with short inversion time arterial spin labeling. J Cereb Blood Flow Metab. 2015;35:461–8.
3. O'Rourke MF. Pulse wave form analysis and arterial stiffness: realism can replace evangelism and scepticism. J Hypertens. 2004;22:1633–4.
4. Berne R, Levy M. Physiology. 3rd ed. St Louis: Mosby; 1999.
5. Carrera E, Kim DJ, et al. Effect of hyper- and hypocapnia on cerebral arterial compliance in normal subjects. J Neuroimaging. 2011;21:121–5.

6. Pannier B. Methods and devices for measuring arterial compliance in humans. Am J Hypertens. 2002;15:743–53.
7. Pekkola J. Imaging of blood flow in cerebral arteries with dynamic helical computed tomography angiography (DHCTA) using a 64-row CT scanner. Acta Radiol. 2009;50(7):798–805.
8. Alexandrov AV. Neurovascular examination: the rapid evaluation of stroke patients using ultrasound waveform interpretation. Oxford: Blackwell Publishing Ltd.; 2013.
9. Jong de S. Quantifying cerebral blood flow of both the micro- and macrovascular system using perfusion computed tomography. Nijmegen: Twente; 2015.
10. Avezaat CJJ. Cerebrospinal fluid pulse pressure and craniospatial dynamics. A theoretical, clinical and experimental study. Rotterdam: Erasmus University; 1984.
11. Ikdip K. Exploring differences in vascular aging and cerebrovascular hemodynamics between older adults of White Caucasian and South Asian origin. Ontario: Waterloo; 2014.
12. Itoh T, et al. Rate of successful recording of blood flow signals in the middle cerebral artery using transcranial Doppler sonography. Stroke. 1993;24:1192–5.
13. Marmarou A. A review of progress in understanding the pathophysiology and treatment of brain edema. Neurosurg Focus 2007;22:1–12.
14. Behzadi Y. An arteriolar compliance model of the cerebral blood flow response to neural stimulus. NeuroImage. 2005;25:1100–11.