Perioperative Dynamics of Intracranial B-waves of Blood Flow Velocity in the Basal Cerebral Arteries in Patients with Brain Arteriovenous Malformation

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Introduction

B-waves were first observed by Lundberg [1] in intracranial pressure (ICP) monitoring as regular oscillations in ICP at frequencies of 0.5–2 waves/min (8–30 mHz). The exact cause of their occurrence is not entirely clear. There are different physiological and pathophysiological factors that are considered sources of this phenomenon: primary slow rhythmic changes in the partial pressure of arterial CO_2 (related to the respiration rhythm), changes in systemic blood pressure (BP), and cerebral perfusion pressure [2–4]. Various classifications and subgroups of B-waves in ICP based on their amplitude, types of waveform, and rate of occurrencehave been proposed for correct identification and interpretation of possible mechanisms that can generate these fluctuations. With the introduction of transcranial Doppler ultrasound (TCD) into clinical practice, similar patterns of slow waves have been found in the spectrum of linear blood flow velocity (BFV) in the basal cerebral arteries in both healthy individuals and patients with a severe head injury, hydrocephalus, a ruptured cerebral aneurysm, or a hypertensive intracerebral hemorrhage [5–8]. Nevertheless, it is still debatable which of the B-waves (of ICP or BFV) is the surrogate and which one is the original. Newell et al. [5] simultaneously recorded TCD and ICP signals in artificially ventilated patients with a severe head injury and demonstrated high coherence of both parameters within the range of B-waves. However, B-waves of BFV preceded B-waves of ICP and were independent of any change in arterial or cerebral perfusion pressure, as well as respiration and end-tidal CO_2 pressure. These findings, supported by those of other experimental and clinical studies, underpin the most acceptable vasogenic theory of the origin of intracranial B-waves [9–13]. According to this theory, intracranial B-waves are induced by periodic changes (dilation and constriction) of small regulating arteries in the cerebral microcirculation, with a frequency of 8–30 mHz. Hence, one can conclude that cerebral blood flow is a basic parameter where intracranial B-waves initially occur. From this point of view, it would be more correct to define B-waves in ICP as a surrogate, whereas B-waves in BFV in the basal cerebral arteries, for obvious reasons, are considered to be the first derivative of a similar fluctuation in cerebral blood flow.

It is still unknown whether slow vasogenic activity with such a periodicity reflects the functional state of either myogenic or neurogenic mechanisms (with an intrinsic brain stem pacemaker) of cerebral blood flow regulation. Moreover, there is no agreement about the prognostic and diagnostic value of intracranial B-waves. Some authors insist that TCD B-waves often occur in healthy individuals and that their disappearance highly correlates with impaired cerebral autoregulation (CA) and poor outcomes in various pathological conditions [2, 10]. Other investigators suggest that increased B-wave amplitude in ICP and BFV is the result of intracranial hypertension and low compliance, as well as cerebral hypoperfusion [5, 8, 9].

The cerebral circulation system in patients with brain arteriovenous malformation (AVM) is commonly characterized by impaired autoregulation on the affected side, which is due to pathological arteriovenous shunting [14, 15]. However, there are no data clarifying the relationship between B-waves of BFV in the basal arteries and this type of compromised cerebral hemodynamics without evident signs of intracranial hypertension.

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The aim of this study was to evaluate the amplitude of BFV within the range of B-waves (BWA) in patients with AVM before and after endovascular intervention.

Materials and Methods

The design of the study was approved by the local ethical standards committee of the Almazov National Medical Research Centre, in accordance with the 1975 Declaration of Helsinki (and as revised in 1983). The study procedures were performed after written informed consent was obtained from the patients or their legal representatives.

We retrospectively examined a cohort of 38 patients with brain AVM who were admitted to the neurovascular department of the Russian Polenov Neurosurgical Institute for endovascular management. The main demographic and clinical characteristics of the patients are presented in Table 1. The patients were studied and operated on beyond the stage of hemorrhagic and epileptic symptoms. The type of AVM located in one of the hemispheres of the brain was classified in accordance with the Spetzler–Martin Grading Scale [16]. AVMs were embolized with either an adhesive agent (n-butyl-2-cyanoacrylate; Hystoacryl) or a nonadhesive agent (Onyx) through afferent vessels originating from the middle cerebral artery (MCA) and/or the anterior cerebral artery (ACA) [17]. Clinical assessment was performed according to the Modified Rankin Scale (mRs) prior to and after the endovascular intervention [18]. All patients had standard perioperative computer tomography (CT) imaging of the brain, Doppler ultrasound of the precerebral and cerebral arteries, and evaluation of CA.

Table 1 Demographic and clinical characteristics of patients with a supratentorial arteriovenous malformation (AVM)

Characteristic	Value
Age (years)[a]	38 ± 11
Male/female ratio (*n*/*n*)	20/18
Type of AVM according to the Spetzler–Martin scale (*n*)	
I–II	9
III	18
IV–V	11
Manifestation of hemorrhage/epileptic seizure (*n*/*n*)	21/17
Radicality of AVM embolization (*n*)	
75–100%	10
50–75%	16
50%	12
Shunt flow index: preoperative/postoperative (ml/min)[a]	564 ± 115/346 ± 96*
Modified Rankin scale score: preoperative/postoperative[a]	1.3 ± 0.6/1.1 ± 0.4

[a]The values are expressed as mean ± standard deviation
*$p < 0.05$

The patients underwent ultrasound assessment of the precerebral arteries, using the Vivid E ultrasound system (GE Healthcare, Chicago, IL, USA) with a multifrequency linear transducer (4–12 MHz). The benefits of duplex scanning for assessment of the radicality of AVM embolization on the basis of shunting blood flow dynamics before and after the procedure have been demonstrated previously [15].

CA measurements were performed with both a cuff test [19, 20] and transfer function analysis (TFA) [21, 22]. The subjects were in a supine position with 30° elevation of the upper body while breathing at a rate of 6 breaths/min. The end-tidal partial pressure of CO_2, measured with a Novametrix Tidal Wave capnograph (Philips Medical Systems, Eindhoven, the Netherlands) corresponded to normocapnia (36–38 mmHg). BFV in the main basal cerebral arteries was insonated through the temporal bone window at a depth of 50–60 mm with a 2-MHz probe fixed on a headframe. BP was registered continuously via a servocontrolled finger photoplethysmograph (CNAP Monitor 500 HD, CNSystems Medizintechnik, Graz, Austria) with the subject's hand position at the heart level. BP recording was transmitted through the analogue input channel of a MultiDop X system block into the monitoring module of DWL software.

For TFA, the data segments were inspected visually and edited for artifacts and ectopy. Only steady state data were digitized and stored on a hard disk in universal ASC files in DWL software for further offline CA assessment. While TFA was performed, the spectral amplitudes of BP and BFV, the phase shift (PS), and the coherence coefficient were calculated in a specifically selected range of systemic Mayer waves (M-waves, 80–120 mHz). This frequency band commonly reflects periodic fluctuations in BP and is more informative for assessment of the myogenic component of the cerebrovascular response than high-frequency oscillations. According to the high-pass filter model, the pressure–flow relation between the input (BP) and output (BFV) signals is characterized by a positive PS (0.8–1.2 rad). In the case of impaired autoregulation, the passing capacity of the filter for slow waves increases; therefore, the PS between both parameters reduces, even to zero. Coherence between 0 and 1 reflects a linear relation between BP and BFV. To ensure the greatest reliability of PS values for further statistical analysis, they were estimated only at frequencies that exhibited coherence >0.6 and maximum M-wave amplitude in BP. Additionally, the spectral amplitudes of BP and BFV were also calculated within the range of slow B-waves (8–30 mHz). TFA was performed with the commercially available data acquisition software Statistica for Windows version 7 (StatSoft, Tulsa, OK, USA) in the "time series and forecasting" module.

Descriptive statistics were used to describe the patients' demographic characteristics and clinical, angiographic, ultrasound, and TFA data. A paired Student's *t* test was used to estimate the significance of CA changes after AVM embolization. Parametric data were expressed as mean ± standard deviation, and values of $p < 0.05$ were considered statistically significant.

Results

Among 38 patients with brain AVM, 21 manifested with intracranial hemorrhage and 17 with epileptic seizures. Nevertheless, none of them had any clinical or CT/MRI (magnetic resonance imaging) markers of intracranial hypertension at the time of admission, examination, and endovascular intervention. The mean mRs value prior to intervention was 1.3 ± 0.6 and indicated slight or insignificant disability (Table 1). AVMs located in one of the hemispheres and supplied with a feeding artery originated from the unilateral MCA and/or ACA.

In patients with a type I–II of AVM, according to the Spetzler–Martin scale ($n = 9$), embolization in one or two sessions led to elimination of up to 75–100% of the initial size of the pathological arteriovenous lesion from the circulation. In cases of AVM type III ($n = 18$) or type IV–V (9) on the Spetzler–Martin scale, because the AVM was located in a functionally important area or was bigger (more than 3 cm in size), the first session of endovascular intervention was significantly less effective. The original size of the AVM was reduced by 50–75% in 16 cases and by no more than 50% in 12 cases. No early postoperative neurological complications nor complications at the time of hospital discharge (7–10 days after the procedure) were observed in our study. The postoperative mean mRs value increased to 1.3 ± 0.6 but nonreliably (Table 1).

Ultrasound examinations were performed 1–3 days prior to intervention, as well as on the third day after it. The shunt flow index prior to intervention in all 38 patients was 564 ± 115 ml/min and reliably correlated ($p < 0.01$) with the type of AVM according to the Spetzler–Martin scale. After embolization, the shunt flow index decreased remarkably to 292 ± 76 ml/min but remained high mainly because the patients had an AVM of type IV–V gradation and it was only partly embolized. In patients with a type I–II of AVM, shunt flow index was zero postoperatively (Table 1).

Perioperative dynamics of BP, BFV, and autoregulation parameters before and after AVM embolization are presented in Table 2. Initial TCD patterns in the basal cerebral arteries on the affected side showed pathological arteriovenous shunting through the AVM. The mean BFV values were 142 ± 49 cm/s on the AVM side and 71 ± 14 cm/s contralaterally. The pulsatility indices were 0.49 ± 0.13 and 0.87 ± 0.14, respectively. Moreover, a reliable decrease in the CA rate on the AVM side was noted (autoregulation index (ARI) 1.2 ± 0.8, PS 0.2 ± 0.1 rad) in comparison with the contralateral side (ARI 5.8 ± 1.5, PS 0.9 ± 0.2 rad). As for BWA, it was significantly greater ($p < 0.01$) on the AVM side (4.5 ± 2.7 cm/c) than on the contralateral side (2.2 ± 1.4 cm/s). These asymmetrical changes in BFV within the B-wave range appeared under stable recordings of BP, end-tidal CO_2 pressure, and the respiration rhythm, without any slow fluctuations at the same frequency.

Figure 1 illustrates the results of a perioperative examination of a patient with a left frontal lobe AVM (type III according to the Spetzler–Martin scale), which manifested with a headache and epileptic seizure. There were no symptoms of intracranial hypertension in the patient's medical history or in the neurological examination at the time of admission. Preoperative BFV values in the left MCA showed the presence of pathological arteriovenous shunting. A cuff test and TFA indicated CA impairment on the affected side (ARI 1, PS 0.21 rad). BWA in the left MCA (3.8 cm/s) was considerably greater than that in the right MCA (2.2 cm/s). On the third day after safe and total AVM embolization, the BFV and pulsatility index on the side of the lesion normalized and the CA parameters reached

Table 2 Dynamics of blood pressure, blood flow velocity, and autoregulation parameters before/after embolization in 38 patients with a supratentorial arteriovenous malformation (AVM)

Variable	Ipsilateral side	Contralateral side
Blood flow velocity in the basal artery (cm/s)	142 ± 49/101 ± 23*	71 ± 14/68 ± 12
Pulsatility index	0.49 ± 0.13/0.69 ± 0.11*	0.87 ± 0.14/0.86 ± 0.14
Phase shift (rad)[a]	0.2 ± 0.1/0.6 ± 0.1*	0.9 ± 0.2/0.9 ± 0.1
Autoregulation index (cuff test)	1.2 ± 0.8/3.6 ± 1.6*	5.8 ± 1.5/5.7 ± 1.4
B-wave amplitude of blood flow velocity (cm/s)	4.5 ± 2.7/2.7 ± 1.8*	2.2 ± 1.4/2.0 ± 0.5
Blood pressure (mmHg)	89 ± 10/85 ± 12	

The values are expressed as mean ± standard deviation before/after embolization
[a]Phase shift between M-waves of blood pressure and blood flow velocity
*$p < 0.05$

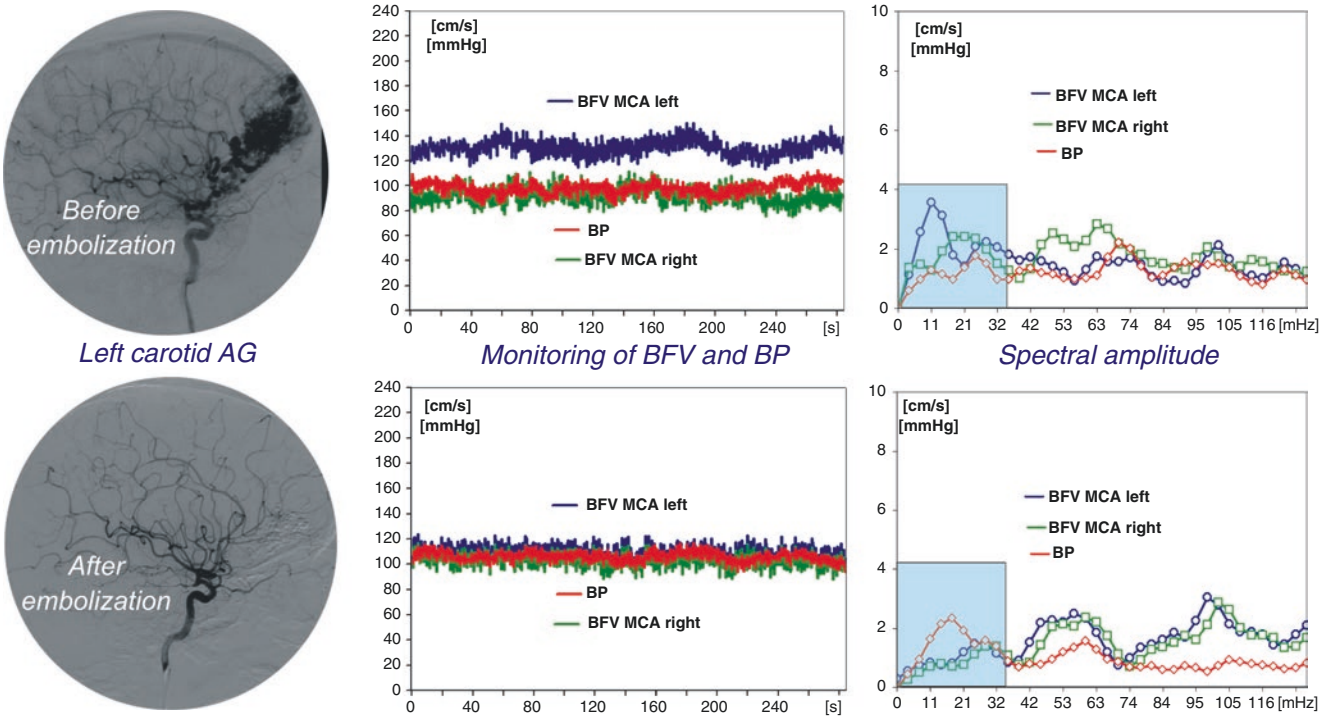

Fig. 1 Results of examination of a 41-year-old man with an arteriovenous malformation (AVM) in the left frontal lobe of the brain. Afferent vessels originate from the left middle cerebral artery (MCA). *From left to right:* left carotid angiogram, multichannel monitoring of blood pressure (BP) and blood flow velocity (BFV) in the MCA over a period of 280 s, and amplitude spectra within the low-frequency range. *From top to bottom:* before and 3 days after total (up to 100%) embolization. The *blue area* comprises the range of intracranial B-waves in an amplitude spectrum analysis

normal values (ARI 4, PS 0.8 rad). The postoperative BWA values in both MCAs were almost identical and less than 2 cm/s.

Moreover, 10 out of the 38 patients (six type IV–V and four type III according to the Spetzler–Martin scale) had greater BWA (exceeding 3 cm/s (4.7 ± 1.1 cm/s)) on the AVM side ($p < 0.01$) than the other 28 patients. There were no reliable differences between the two subgroups in terms of BFV, BP, ARI, and PS on the AVM side. After embolization, there were reliable increases ($p < 0.01$) in the CA rate (ARI 3.6 ± 1.9, PS 0.5 ± 0.2 rad) and decreases in BWA on the AVM side (2.7 ± 1.8 cm/s) in the whole cohort.

Figure 2 illustrates the results achieved in a patient with a left temporal lobe AVM (type IV according to the Spetzler–Martin scale) and no symptoms of intracranial hypertension. Along with TCD patterns of shunting and severe CA impairment, there was also an extremely large BWA (13 cm/s) with a period of more than 2 min. On the third day after the first session of partial embolization (of up to 30% of the lesion), BFV in the left MCA remained high and CA did not improve substantially. Of note, BWA decreased but was still greater than that on the contralateral side. There were no postoperative neurological complications.

Discussion

Intrinsic CA impairment in the basal cerebral arteries, which are involved in AVM feeding, can be due to cerebral hypoperfusion and "steal" in the perinidal area. Sato et al. [23] discovered the presence of abnormally enlarged capillaries (up to 7 mm in diameter) in the brain adjacent to the AVM nidus. The so-called perinidal dilated capillary network, with its inherent pathomorphological features and their functional disability, may potentially reflect the real state of autoregulation under conditions of cerebral hypoperfusion and inadequate collateral circulation. However, in practice, it is quite difficult to preoperatively differentiate true negative results of CA assessment from false negative ones, particularly in AVM type III–V, because of high shunting through AVM. In such cases, baseline low CA indices on the affected side reflect the inability for regulation in the afferent vessels of the AVM and its network as a result of their morphologically determined low resistance. Actually, CA assessment is most informative after total embolization of the AVM, when the perfusion pressure has normalized, and we can detect the real state of CA after intervention and predict the risk of possible hemorrhagic complications.

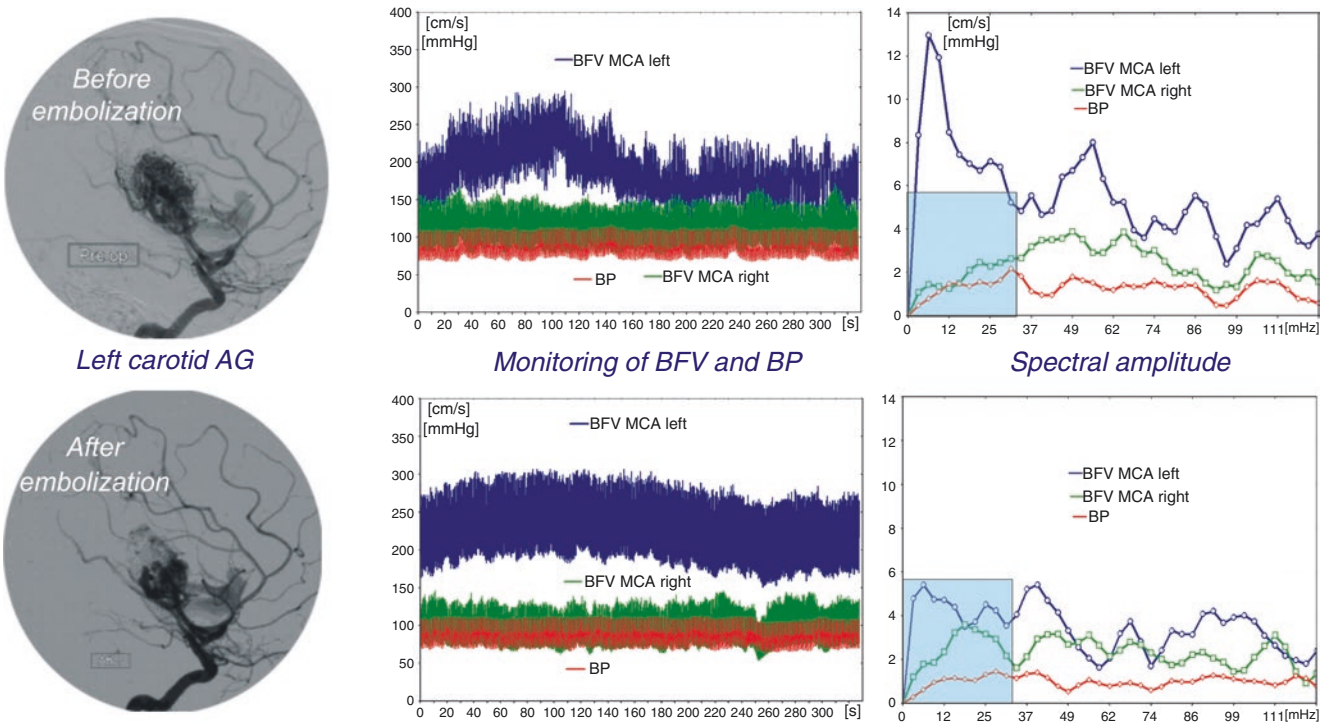

Fig. 2 Results of examination of a 31-year-old man with an arteriovenous malformation (AVM) in the left temporal lobe of the brain. Afferent vessels originate from the left middle cerebral artery (MCA). *From left to right:* left carotid angiogram, multichannel monitoring of blood pressure (BP) and blood flow velocity (BFV) in the MCA over a period of 280 s, and amplitude spectra within the low-frequency range. *From top to bottom:* before and 3 days after partial (up to 30%) embolization

A significantly increased BWA of BFV not induced by fluctuations in BP prior to endovascular treatment may be due to additional neurogenic vasodilation of pial arteries in the perinidal zone of the AVM when the perfusion pressure is quite low. This assumption is supported by a reduction in BWA in BFV in the basal cerebral arteries on the affected side even after partial AVM embolization. Further research is needed to establish the reasons for this phenomenon and the interrelation between myogenic and neurogenic components of regulation. Along with CA assessment in patients with an AVM in the brain, it might provide a useful tool for evaluation of the treatment strategy and prognostication of the risk of hemorrhage in the patient's natural course and after endovascular interventions.

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