Acta Neurochir (2005) [Suppl] 94: 53–58

Printed in Austria

The intracranial B-waves' amplitude as prognostication criterion of neurological complications in neuroendovascular interventions

V. B. Semenyutin, V. A. Aliev, P. I. Nikitin, and **A. V. Kozlov**

Russian Polenov Neurosurgical Institute, Saint-Petersburg, Russia

Summary

The purpose of this study was to evaluate dynamics of B-waves' amplitudes (BWA) of blood flow velocity (BFV) in patients with cerebrovascular diseases during endovascular operations. We examined 12 patients with neurovascular pathology during neuroendovascular interventions. Patients were divided into two groups: 1st group (6 cases) – without intraoperative neurological complications, 2nd group (6 cases) – with complications. Bilateral monitoring of BFV in middle cerebral arteries was carried out applying Multi Dop X. To estimate BWA Fourier analysis was used. In the 1st group preoperative BWA on the affected side was 3.9 ± 0.6 cm/s. Intraoperative (during an access to pathologic formation and its embolisation) BWA increased up to 7.7 ± 1.1 cm/s ($p < 0.05$). Postoperative BWA decreased to 4.2 ± 0.8 cm/s. In the 2nd group the preoperative BWA on the affected side was 9.6 ± 1.1 cm/s ($p < 0.05$), thus higher than in the 1st group. Intraoperatively we observed further increase of BWA up to 12.1 ± 2.6 cm/s, accompanied by occurrence or increase of neurological symptoms. Postoperative BWA decreased to 10.4 ± 2.9 cm/s, whereas we didn't observe regression of neurological symptoms.

Keywords: Cerebral blood flow; cerebral autoregulation; transcranial Doppler; spectral analysis; intracranial B-waves; neuroendovascular intervention.

Introduction

Efficient treatment of neurovascular disorders is strongly dependent on the state of cerebral circulation during endovascular intervention [3, 4, 10, 13]. Quick and timely estimation of mechanisms regulating cerebral blood flow (CBF) would allow to reveal impaired function of cerebral circulation system at a stage that precedes development of neurological complications, and, thus, prevent them by taking necessary treatment-and-prophylaxis measures.

Existing methods of regulation assessment are based on use of transcranial Doppler to monitor the linear blood flow velocity (BFV) in basilar arteries at different stages of treatment of neurosurgical cases [3, 4, 10]. Special attention is paid to studying cerebral blood flow autoregulation (CA) as one of the main properties of a system of cerebral circulation supporting constancy of CBF in case of changes of cerebral perfusion pressure (CPP) within certain limits [1, 4, 19].

From the 90ies on, conventional methods to study CA (in time domain) [1, 4, 6] were supplemented by applying spectral analysis (in frequency domain) [2, 5, 7, 9, 12, 16, 22]. Estimation of BFV and CPP in time domain enabled obtaining information on static (by changing CPP by pharmacological means) and dynamic (by using cuff or compression tests, as well as Valsava's maneuver) CA in case of normocapnia, hypocapnia and hypercapnia [1, 11, 15, 17, 20]. Studying CA on the basis of spectral analysis resulted in forming a concept according to which CA was a complex multi-component mechanism ensuring constancy of CBF not only in quick changes of CPP, but also in its slow spontaneous oscillations within MF (middle frequency) (0.1 Hz) and LF (low frequency) (0.02–0.07 Hz) ranges without any considerable effect on systemic and cerebral hemodynamics as a whole [2, 6, 9, 12]. From this point of view CA can be regarded as a high-pass filter transmitting HF oscillations of BFV (>0.2 Hz) which are characterized by high coherence and a smaller phase shift in comparison with analogous oscillations of the systemic blood pressure (BP). At the same time, this high-pass filter damps down MF and LF oscillations of BFV (Mayer's waves, etc.). It manifests itself in low coherence and a bigger phase shift between BFV and BP within the above mentioned range. These data demonstrate that CA depends on frequency and is more effective within MF and LF ranges, as compared to HF range. Disorders of CA lead to an increase of the filter's transmitting capacity and, as a result, to higher coherence and a smaller phase shift between BP and BFV within MF and LF ranges of spontaneous oscillations.

Spectral analysis is an informative, simple and absolutely safe method of CA estimation. Thanks to this fact, it is used in clinical practice for diagnosis of CA disorders in cases with intracranial aneurysms, arteriovenous malformations and occluding lesions of major cervical vessels [6, 9, 15, 18]. However, its wider use is limited by the – technically often not feasible – necessity of continuous recording of BP.

Though acknowledging that CA is a frequency-dependent phenomenon, many researchers pay more attention to studying the spontaneous oscillations within the range of Mayer's systemic waves and respiratory waves instead of focusing on the B-waves (0.008–0.03 Hz). For the first time these waves were detected in the spectrum of intracranial pressure [8] its rise causing an increase of B-waves' amplitude. Later on it was discovered that they originated within the spectrum of BFV [14]. However, the exact cause for their appearance is not entirely clear [14, 22]. B-waves are likely to reflect a state of regulatory mechanisms of CBF mediated via smooth-muscle cells of cerebral vessels or stem pace-makers which change CBF within certain periodicity by effecting the activity of vasomotor neurons.

Our investigation is based on a supposition, according to which disorders of CA not only cause changes of coherence and a phase shift between BFV and BP within the MF range, but also changes of B-waves' parameters. The purpose of the study is to evaluate an amplitude of intracranial B-waves of BFV and its dynamics in endovascular interventions performed in cases with neurovascular pathology.

Materials and methods

Description of patients

There were 12 cases (7 males and 5 females, aged 25–60) with different neurovascular pathology, including malformations (7), aneurysms (4) and carotid-cavernous fistula (1). Supratentorial and subtentorial localization of malformations were observed in 6 and 1 patients respectively; all aneurysms were localized in the region of the internal carotid artery.

Malformations and aneurysms manifested themselves in a single episode of subarachnoid hemorrhage or a seizure; carotid-cavernous fistula developed after head injury of moderate severity.

Accompanying somatic diseases were represented by coronary heart disease, observed in elderly patients (50–60 years old); though there was no need to prescribe additional cardiac drugs during the period of treatment.

The patients were admitted to a hospital a month after disease onset. As for admission, subsequent examination and intervention, they were characterized either by absence of any neurological symptoms or presence of oculomotor or minor motor disorders.

Interventions

Due to localization of malformations in eloquent cerebral areas, big and giant aneurysms or their localization at the level of an ophthalmic segment of the internal carotid artery, endovascular operation was considered to be preferable to open surgical intervention.

Operations were executed according to a standard method with an approach through the right femoral artery and use of catheters for endovascular interventions (Balt). Superselective embolization of malformations was performed through afferent vessels, using histoacryl as an embolizing substance; aneurysms and carotid-cavernous fistula were occluded with mechanically detachable coils and latex balloon. Anesthesiologic support consisted in sedation-analgesia (a standard-dose combination of propofol and fentanyl).

Monitoring

Perioperative bilateral monitoring of BFV in the middle cerebral artery was carried out applying Multi Dop X (DWL). BP was monitored invasively through the 20–22 GA catheter, introduced into the radial artery; the M-34 mingograph (Siemens) was used. Recorded indices were monitored every 5 min during operation; a patient was in a horizontal position with his head elevated up to 30°; monitoring was carried out at rest against a background of preserved spontaneous breathing.

A protocol of perioperative monitoring during neuroendovascular interventions was approved of by the Ethic Committee of the Polenov Neurosurgical Institute. Monitoring started after receiving the patient's written consent.

Data analysis

Spectral analysis of B-waves of BFV in the middle cerebral artery was carried out according to a standard algorithm with the help of Statistica 6.0 for Windows (Time Series/Forecasting module). A time series within an interval of 240–300 sec was chosen based on the Kotelnikov-Shannon's theorem stating the estimation of the spectrum of LF oscillations requires analysis of a time series for a period, which is at least twice as long as a maximum period of LF oscillations (120 sec for B-waves). To ensure stability of this series, spectral analysis was preceded by subtraction of the mean from values of a time series. Smoothing of values, characteristic of one period, was performed with the purpose of incidental noise suppression, reducing dispersion of a time series and revealing frequencies with high spectral densities, which contributed significantly to a periodic behavior of the whole time series. Smoothing was achieved by transformation of the running weighted mean in Hemming's window. Then the spectral density of all oscillations in a chosen time interval, including the range of intracranial B-waves (0.0083–0.033 Hz), was calculated. Calculation of B-waves' amplitude (BWA) was carried out in accordance with the following formula:

$$\mathrm{BWA} = \sqrt{\mathrm{SD} \times \mathrm{F}}\ (\mathrm{cm/s})$$

(SD – spectral density, $(\mathrm{cm/s})^2/\mathrm{Hz}$; F – frequency, Hz)

Statistics

Statistical processing of data was carried out with the help of standard methods (Statistica 6.0 for Windows). Criteria used were parametric (Student's t-criterion) and non-parametric (Kholmogorov-Smirnov's criterion, Pirson's χ^2 criterion). Difference was considered to be statistically reliable with $p < 0.05$.

Results

BWA was estimated in succession at three stages of intravascular intervention. The first stage preceded with the introduction of a catheter guide into major cervical vessels. The second stage corresponded to the period of microcatheter's delivery to the pathologic formation and the actual surgical intervention. The third stage corresponded to completion of the operation and removal of catheters.

The patients were subdivided into two groups, depending on the course of the intraoperative period. The 1st group included cases (6) without neurological complications during intraoperative period; the 2nd group (6) comprised patients with development of intraoperative neurological complications (headache, suppressed consciousness, nausea, vomiting, gross motor and speech disturbances). The causes of complications were as follows: thrombosis of the internal carotid artery – 2 cases with aneurysm and 1 case with carotid-cavernous fistula; cerebral vasospasm and intracranial hypertension – 1 female patient with aneurysm and 1 male patient with malformation; parenchymal-subarachnoid hemorrhage into cerebellar hemispheres – 1 female patient with malformation of subtentorial localization. As for the last female, development of marked bulbar symptoms resulted in a fatal outcome during the first postoperative day.

Patients without intraoperative complications

Mean values of BP, BFV and BWA, recorded on the side of operation and on the opposite side in patients of the 1st group at different stages of intravascular intervention, are given in Table 1.

Due to anesthesiologic support (sedation-analgesia) causing no considerable suppression of breathing and BP reduction and transnasal insufflation of an oxygen-air mixture, spontaneous breathing of patients corresponded to normoventilation conditions. It was confirmed by capnography data and measurements of pCO_2 in arterial blood. At the main stage of operation there was a reliable increase of BWA ($p < 0.05$) (Table 1) on the side of intervention and on the opposite side. On both sides no considerable changes of BP and BFV were observed during intervention (Table 1).

Table 1. *BP, BFV and BWA of patients of the first group at different stages of endovascular intervention*

Stages of operation	BP [mm Hg]	BF parameters [cm/s]			
		On the side of operation		On the opposite side	
		BFV	BWA	BFV	BWA
Preoperative	79 ± 9	77 ± 11	3.9 ± 0.6	82 ± 12	3.8 ± 0.7
Intraoperative	84 ± 10	85 ± 12	7.7 ± 1.1	75 ± 12	7.5 ± 1.3
Postoperative	84 ± 11	87 ± 14	4.2 ± 0.8	83 ± 15	4.0 ± 0.6

Figure 1 demonstrates monitoring of BFV and BWA on the side of operation at different stages of intravascular intervention performed in a 25 years old patient with giant aneurysm of the left internal carotid artery. Complete exclusion of the aneurysm from blood circulation was achieved by means of its occlusion by a latex balloon. There were no intraoperative and early postoperative neurological complications.

Patients with intraoperative complications

Mean values of BP, BFV and BWA, recorded on the side of operation and on the opposite side in patients of the 2nd group at different stages of intravascular intervention, are given in Table 2.

Perioperative changes of BFV and BP in patients of the 2nd group, as well as in cases of the 1st group, were insignificant. Their breathing was spontaneous and corresponded to normoventilation parameters. BWA was considerably higher already before operation ($p < 0.05$), i.e. before performing manipulations on major vessels (a change of an angiographic catheter for a catheter guide, introduction of a microcatheter into intracranial vessels), as compared to patients without complications (Table 2). However, there were no objective signs of augmentation of neurological symptoms. Further increase of BWA, observed on the side of operation and opposite side at the main stage of intervention, was accompanied by occurrence or increase of neurological symptoms (appearance of headache, nausea, vomiting, motor and speech disorders, sharp suppression of consciousness up to deep stupor, sopor). Reduction of BWA after operation (detachment of a spiral, balloon, injection of a glue composition, removal of catheters) was insignificant. There was no regression of neurological symptoms.

Figure 2 demonstrates monitoring of BFV and BWA on an operated side in a 42 years old female

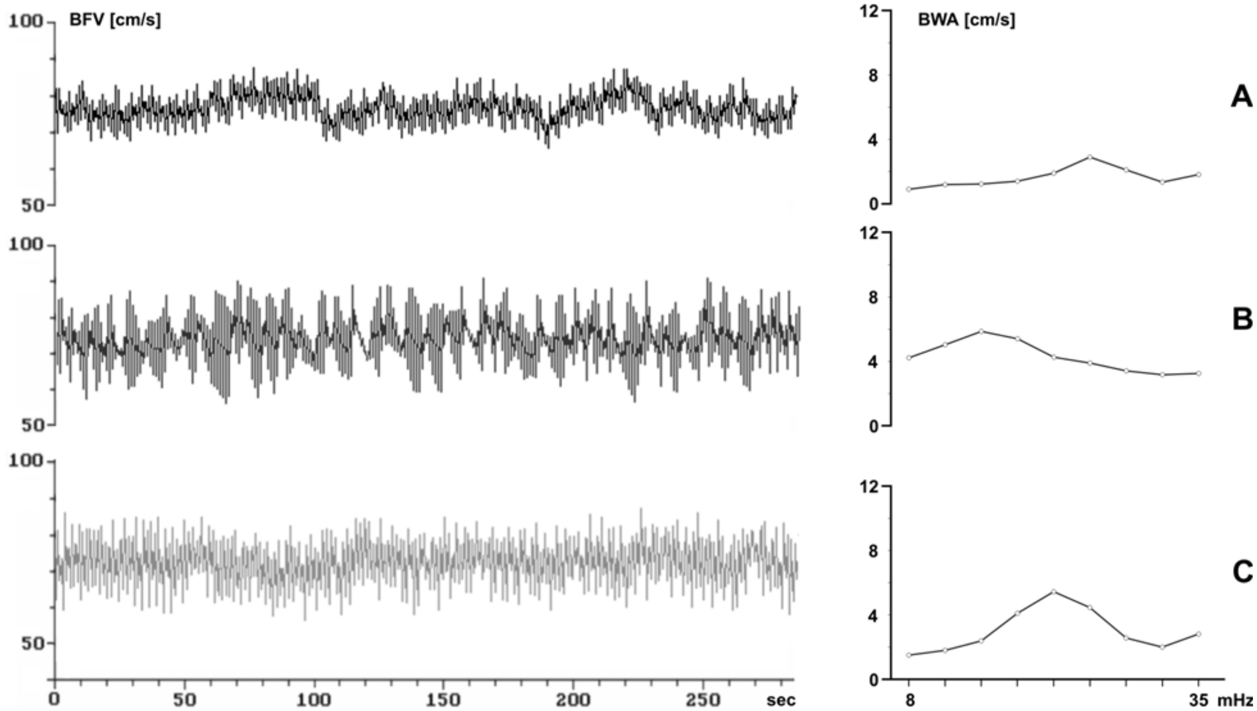

Fig. 1. Monitoring of BFV and BWA on the side of operation before (A), during (B) and after (C) the operation, performed in 25 years old patient with giant aneurysm of an ophthalmic segment of the left internal carotid artery

patient with a large saccular aneurysm of the supraclinoid segment of the left internal carotid artery.

Before operation BWA on the side of the aneurysm was considerably higher than in patients without complications; it was watched against a background of relatively normal parameters of BFV in the left middle cerebral artery. The patient developed nausea, vomiting, psychomotor excitement, dysphasic disorders at the stage of an approach to aneurysm (introduction of a coil into aneurysm and its subsequent detachment). Monitoring of BFV, carried out just before the described aggravation, did not reveal its considerable changes, though there was a further increase of BWA. After a coil detachment and catheters removal BWA reduced but did not reach the initial means before the operation. After operation the female patient was admitted to the Intensive Care Unit, where she spent a day and was subjected to intensive infusion therapy. Neurological symptoms regressed during a week.

Table 2. *BP, BFV and BWA of patients of the second group at different stages of endovascular intervention*

Stages of operation	BP [mm Hg]	BF parameters [cm/s]			
		On the side of operation		On the opposite side	
		BFV	BWA	BFV	BWA
Preoperative	85 ± 11	71 ± 13	9.6 ± 1.1	79 ± 15	8.4 ± 1.4
Intraoperative	96 ± 9	89 ± 18	12.1 ± 2.6	83 ± 12	9.9 ± 2.3
Postoperative	90 ± 7	66 ± 12	10.4 ± 2.9	64 ± 13	7.9 ± 2.9

Discussion

Volby *et al.*, in 1982 found in patients with acute subarachnoid hemorrhage, that correlation of B-waves amplitude increase during recording of intracranial pressure (ICP) with the severity of the bleeding according to the Hunt and Hess scale, as well as with the severity of intracranial hypertension and cerebral vasospasm [21]. Carrying out spectral analysis of BFV and ICP in patients with severe craniocerebral trauma, Newell *et al.* [14] concluded that B-waves of ICP were derivatives of B-waves of BFV, which, most likely, characterized a state of myogenic or neurogenic mechanisms of CBF regulation. In our opinion, intracranial

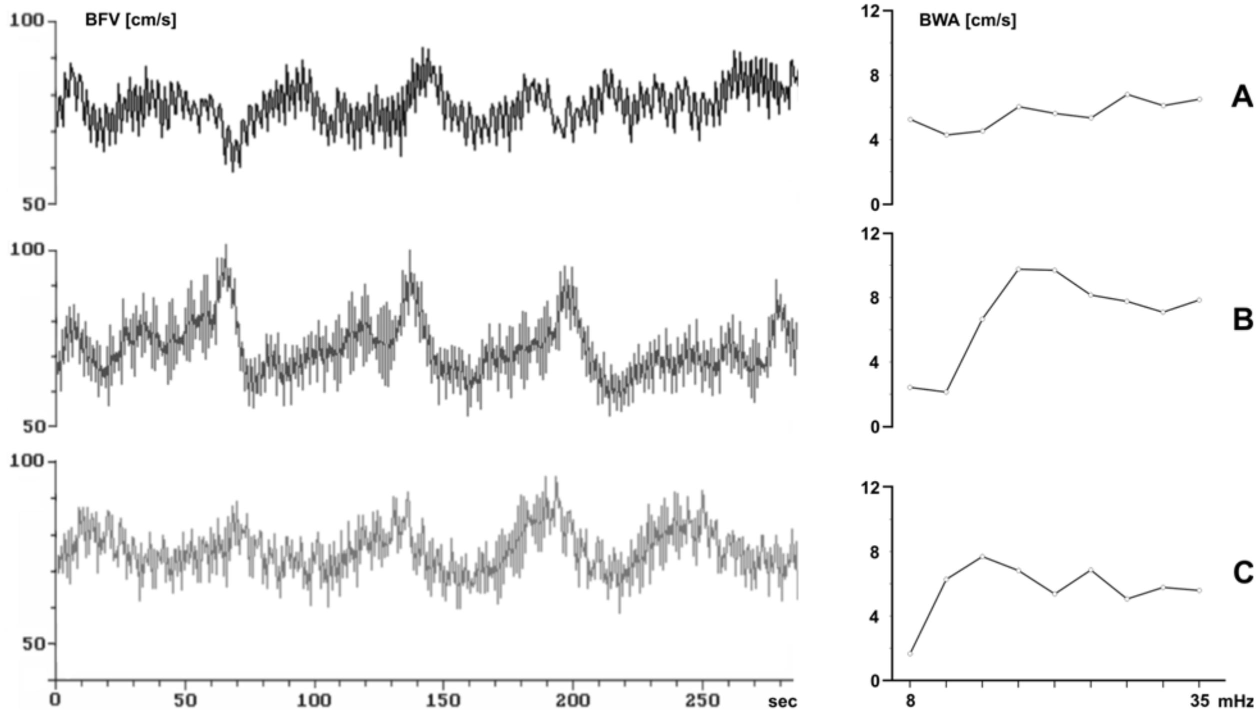

Fig. 2. Monitoring of BFV and BWA on the side of operation before (A), during (B) and after (C) the operation, performed in 42 years old patient with large aneurysm of a supraclinoid segment of the left internal carotid artery

B-waves reflect a state of regulatory mechanisms of CBF as a whole. It appears, that different pathologic states (intracranial hypertension, cerebral vasospasm, hemorrhage, brain compression) are accompanied by dysfunction of stem pace-makers, which control CBF, analogous with a effect of the brain stem on respiration rate, heart rate and other physiologic parameters. At the same time, these very states, causing changes of CPP, result in impairment of vasodilatation and vasoconstriction processes in cerebral vessels, which is revealed with a cuff test, cross-spectral analysis within the range of MF waves. One cannot answer exactly whether a neurogenic or myogenic mechanism is the first to be affected; thus, this problem requires further examination.

It is known, that low coherence and a big phase shift between BP and BFV within the range of MF waves, observed in healthy individuals with normal CA, are combined with a low BWA [4, 22]. Our examination of neurosurgical patients with preserved CA and a compensated function of the high-pass filter showed BWA indices close to normal values. As for cases with impaired CA and a decompensated function of the high-pass filter, BWA indices were far from normal. Determining limits of normal values of BWA is a subject for further research.

Mean values of BWA were compared with mean indices of a phase shift within the range of MF waves. This was done in both groups of patients at rest and before intervention. As for the 1st group, the preoperative mean values of a phase shift between BFV and BP within the range of MF waves were 79.8 ± 11.4 on the side of operation and 83.4 ± 13.4 – on the opposite side. In the 2nd group mean values of phase shift within the same range were much smaller: 17.2 ± 8.5 and 24.6 ± 8.5 respectively ($p < 0.05$). These data are similar to the ones previously presented by other authors [5, 6, 16].

The data obtained demonstrate possible use of BWA of BFV to estimate the state of a cerebral circulation system and prognosticating intra- and postoperative neurological complications in endovascular interventions performed in neurosurgical patients.

It is doubtless that simultaneous recording of BFV and other functionally important parameters (BP, ICP) gives more extensive information about the state of regulatory mechanisms of cerebral circulation system than studying BFV only. However, prolonged non-invasive recording of BP for some 4–5 minutes, carried out with the help of the Finapress apparatus, is accompanied by transient disorders of microcirculation in a finger and, often causes measurement errors and necessity to discontinue investigation and to place a cuff on another finger. Using this simple (and available) method – that does not demand additional continuous measurement of BP and ICP – to estimate BWA of BFV, would permit to reduce time and volume of intraoperative investigation without effecting diagnostic and prognostic significance of the obtained results which is extremely important as to ensure choosing an adequate algorithm of therapeutic and preventive measures.

References

1. Aaslid R, Lindegaard K, Sorteberg W, Nornes H (1989) Cerebral autoregulation dynamics in humans. Stroke 20: 45–52
2. Birch AA, Dirnhuber MJ, Hartley-Davies R, Iannotti F, Neil-Dwyer G (1995) Assessment of autoregulation by means of periodic changes in blood pressure. Stroke 26: 834–837
3. Chioffi F, Pasqualin A, Beltramello A, Da Pian R (1992) Hemodynamic effects of preoperative embolization in cerebral arteriovenous malformations: evaluation with transcranial Doppler sonography. Neurosurgery 31: 877–885
4. Diehl RR, Henkes H, Nahser HC, Kuhne D, Berlit P (1994) Blood flow velocity and vasomotor reactivity in patients with arteriovenous malformations. A transcranial Doppler study. Stroke 25: 1574–1580
5. Diehl RR, Linden D, Lucke D, Berlit P (1995) Phase relationship between cerebral blood flow velocity and blood pressure. A clinical test of autoregulation. Stroke 26: 1801–1804
6. Diehl RR (2002) Cerebral autoregulation studies in clinical practice. Review. Eur J Ultrasound 16: 31–36
7. Giller CA (1990) The frequency-dependent behavior of cerebral autoregulation. Neurosurgery 27: 362–368
8. Kjaellquist Ä, Lundberg N, Pont'en U (1964) Respiratory and cardiovascular changes during rapid spontaneous variations of ventricular fluid pressure in patients with intracranial hypertension. Acta Neurol Scand 40: 291–317
9. Kuo TB, Chern CM, Sheng WY, Wong WJ, Hu HH (1998) Frequency domain analysis of cerebral blood flow velocity and its correlation with arterial blood pressure. J Cereb Blood Flow Metab 18: 311–318
10. Lagalla G, Ceravolo MG, Provinciali L, Recchioni MA, Ducati A, Pasquini U, Piana C, Salvolini U (1998) Transcranial Doppler sonographic monitoring during cerebral aneurysm embolization: a preliminary report. AJNR Am J Neuroradiol 19: 1549–1553
11. Lam JM, Smielewski P, Czosnyka M, Pickard JD, Kirkpatrick PJ (2000) Predicting delayed ischemic deficits after aneurysmal subarachnoid hemorrhage using a transient hyperemic response test of cerebral autoregulation. Neurosurgery 47: 819–826
12. Lang EW, Diehl RR, Mehdorn HM (2001) Cerebral autoregulation testing after aneurysmal subarachnoid hemorrhage: the phase relationship between arterial blood pressure and cerebral blood flow velocity. Crit Care Med 29: 158–163
13. Laumer R, Steinmeier R, Gonner F, Vogtmann T, Priem R, Fahlbusch R (1993) Cerebral hemodynamics in subarachnoid hemorrhage evaluated by transcranial Doppler sonography: Part 1. Reliability of flow velocities in clinical management. Neurosurgery 33: 1–9
14. Newell DW, Aaslid R, Stooss R, Reulen HJ (1992) The relationship of blood flow velocity fluctuations to intracranial pressure B waves. J Neurosurg 76: 415–421
15. Newell DW, Weber JP, Watson R, Aaslid R, Winn HR (1996) Effect of transient moderate hyperventilation on dynamic cerebral autoregulation after severe head injury. Neurosurgery 39: 35–44
16. Panerai RB, Rennie JM, Kelsall AW, Evans DH (1998) Frequency-domain analysis of cerebral autoregulation from spontaneous fluctuations in arterial blood pressure. Med Biol Eng Comput 36: 315–322
17. Ratsep T, Asser T (2001) Cerebral hemodynamic impairment after aneurysmal subarachnoid hemorrhage as evaluated using transcranial Doppler ultrasonography: relationship to delayed cerebral ischemia and clinical outcome. J Neurosurg 95: 393–401
18. Reinhard M, Roth M, Müller T, Czosnyka M, Timmer J, Hetzel A (2003) Cerebral autoregulation in carotid artery occlusive disease assessed from spontaneous blood pressure fluctuations by the correlation coefficient index. Stroke 34: 2138–2144
19. Strandgaard S, Paulson OB (1984) Cerebral autoregulation. Review. Stroke 15: 413–416
20. Tiecks FP, Douville C, Byrd S, Lam AM, Newell DW (1996) Evaluation of impaired cerebral autoregulation by the Valsalva maneuver. Stroke 27: 1177–1182
21. Voldby B, Enevoldsen EM (1982) Intracranial pressure changes following aneurysm rupture. Part 1: clinical and angiographic correlations. J Neurosurg 56: 186–196
22. Zhang R, Zuckerman JH, Giller CA, Levine BD (1998) Transfer function analysis of dynamic cerebral autoregulation in humans. Am J Physiol 274: H233–H241

Correspondence: Semenyutin Vladimir Borisovich, Russian Polenov Neurosurgical Institute, 12 Mayakovsky St., 191104 Saint-Petersburg, Russia. e-mail: lbcp@rnsi.hop.stu.neva.ru