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The influence of propofol on middle cerebral artery flow velocity (V_{MCA}) in patients with unruptured intracranial aneurysms during induction of general anaesthesia

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Abstract

Background: The estimated prevalence of unruptured intracranial aneurysms is 3%. Standard monitoring does not enable one to assess the influence of anaesthetics on the factors determining intracranial homeostasis. Thanks to transcranial Doppler ultrasonography, middle cerebral artery flow velocity ($V_{MCA)}$, reflecting cerebral blood flow, can be measured. The aim of the study was to assess the effects of propofol on intracranial homeostasis in patients with unruptured intracranial aneurysms during the induction of anaesthesia based on V_{MCA} changes.

Methods: The study encompassed 21 patients (group II) anaesthetised for elective craniotomy due to unruptured intracranial aneurysms. The control group (group I) included 21 patients who underwent discoidectomy. V_{MCA}, as well as HR, MAP, $etCO_2$, and SpO_2 were monitored at the following time points: T_0 — onset of study; T_1 — after 1 minute; T_2 — onset of preoxygenation; T_3 — after 1 minute of preoxygenation; T_4 — administration of fentanyl; T_5 — 1 minute after fentanyl; T_6 — administration of propofol; T_7 — 1 minute after propofol; T_8 — intubation; T_9 — 1 minute after intubation; T_{10} — 2 minutes after intubation.

Results: In both groups, no changes in mean HR, $etCO_2$ and SpO_2 were observed at the successive time points of observation. In groups I and II, an MAP decrease between T_6 and T_7 and an MAP increase between T_7 and T_9 were noted. There were no intergroup differences in mean values of MAP at the times of observation. In both groups and bilaterally, a V_{MCA} decrease was recorded between T_6 and T_7 and an increase between T_7 and T_8. There were no intergroup differences in mean values of V_{MCA} at the times of observation. In both groups, a weak correlation between V_{MCA} and MAP changes was found bilaterally.

Conclusions: Propofol depresses the cerebral circulation during the induction of anaesthesia. The presence of an unruptured aneurysm does not affect the reactivity of the cerebral vessels during the induction of anaesthesia with propofol.

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Key words: propofol, unruptured intracranial aneurysm, mean arterial blood pressure, middle cerebral artery flow velocity

Despite the impressive advances in anaesthetic and surgical techniques observed over the last three decades which have resulted in reduced mortality and improved safety of anaesthetised patients, perioperative central nervous system (CNS) injury remains a serious clinical problem. Induction is a key element of general anaesthesia. Extremely strong agents administered at short intervals can lead to depression of the circulatory system and disorders of intracranial homeostasis [1]. During the induction, patients undergo direct laryngoscopy and endotracheal intubation protecting against aspiration of gastric contents to the lungs and enabling the maintenance of airway patency and ventilation ensuring proper CO_2 and O_2 pressure in arterial blood. Laryngoscopy and intubation, both very potent stressogenic stimuli, can induce strong sympathetic stimulation expressed as increased arterial pressure and an accelerated heart rate [2–5]. To prevent such adverse responses, a suitable depth of anaesthesia should be provided using anaesthetics.

The effects of propofol, an intravenous hypnotic agent, on the CNS are multifaceted. Propofol reduces metabolism [6–8], which is accompanied by a decrease in cerebral blood flow (CBF) [9–11]. In patients without CNS pathologies, propofol does not affect autoregulation and reactivity of the cerebral circulation to changes in arterial CO_2 pressure [12, 13]. Moreover, propofol reduces the increase in intracranial pressure (ICP) induced by the pathology [14, 15]. Many authors have emphasised the potential neuroprotective effects of propofol [8, 16, 17].

The induction of anaesthesia with propofol is connected with a decrease in arterial pressure accompanied by compensative response from baroreceptors [18–20]. Arterial pressure decreases mainly via dilation of the peripheral vascular bed and reduction in cardiac output [21, 22]. Haemodynamic stability of the systemic circulation is of particular importance, as it is the *sine qua non* for protection of cerebral perfusion pressure (CPP), both under physiological conditions and in cases of CNS pathologies.

The aim of the study was to assess the effects of the induction of anaesthesia using propofol on intracranial homeostasis in patients with unruptured intracranial aneurysms (UIAs) based on middle cerebral artery flow velocity (V_{MCA}), selected parameters of systemic circulation and their correlations.

METHODS

The study was conducted on 42 patients undergoing surgical procedures in the Department of Neurosurgery of the Medical University of Gdansk. The study design was approved by the Independent Bioethical Committee for Scientific Research at the Medical University of Gdansk. The study included ASA I patients after obtaining their informed consent. The exclusion criteria were focal CNS deficits, cardiovascular diseases, diabetes mellitus, impaired lipid and hormonal metabolism, tobacco smoking and alcohol abuse. The study population was divided into two groups. Group I (control) included patients operated on due to lumbar spine discopathy. Group II consisted of patients undergoing surgical clipping of unruptured intracranial aneurysms accidentally detected during the diagnostic neurological procedures for non-specific symptoms, predominantly headaches.

The patients in both groups were orally premedicated 45 minutes before the onset of anaesthesia using midazolam at a dose of 0.2 mg kg^{-1}. In the operating room, the basilic vein was cannulated (Ø 1.3 mm) and 500 mL of Ringer`s solution was transfused within 15 minutes.

The anaesthesia was induced using propofol at a dose of 1.5 mg kg^{-1} in both groups. Moreover, all patients were administered fentanyl 2 μg kg^{-1} and vecuronium 0.15 mg kg^{-1}. After administering the muscle relaxant, mechanical ventilation was started with 100% oxygen in the half-closed system maintaining $ETCO_2$ within the range of 37–40 mm Hg.

The following parameters were monitored: heart rate (the electrocardiographic curve); arterial blood saturation (SpO_2); mean arterial pressure (MAP); neuromuscular blockade using TOF-Watch (Organon, Oss, Holland); and body temperature in the external auditory meatus.

The middle cerebral artery velocity ($V_{MCA}{}^{L}$ — left, $V_{MCA}{}^{P}$ — right) was measured using a Multi-Dop®T2 device (DWL Elekronische Systeme GmbH, Singen, Germany). The measurements were continuous and bilateral; the 2-MHz Doppler probes were fixed over the temporal bone windows with a dedicated metal frame, which ensured unchanged measurement conditions (the angle of insonation) throughout the study.

During the 10-minute study, attention was paid to the values of continuously recorded parameters in the final 10 seconds of each observation minute. The anaesthesia was induced according to the following scheme: minute 2 — passive oxygenation was started; minute 4 — fentanyl was administered; minute 6 — the muscle relaxant and anaesthetic were administered; and minute 8 — intubation was performed.

The data were statistically analysed using STATISICA for WINDOWS 7.1 PL (StatSoft Inc. Tulsa, USA). The distribution of data was assessed with the Shapiro-Wilk test. For non-normally distributed data, intra- and intergroup comparisons were analysed using the Friedman and Kruskal-Wallis tests, respectively. For normally distributed data, intra- and intergroup comparisons were performed using the two-way analysis of variance (Fisher's test) after checking the homogeneity of variances with Levene's test.

The strength of MAP and V_{MCA} associations was assessed by determining the Pearson's correlation coefficient. Numerical data were expressed as a mean ± standard variation. Statistically significant differences were expressed as a mean difference (MD), level of significance (*P*) and –95% +95% confidence intervals (CI). $P < 0.05$ was considered as statistically significant.

RESULTS

The aneurysms were located in the middle cerebral artery (MCA) in 9 patients and in the internal cerebral artery (ICA) in 12 patients. The characteristics of the study groups are presented in Table 1. There were no intergroup differences in age, weight, serum haemoglobin concentration, haematocrit, and body temperature.

During the study period, no intra- and intergroup differences in mean $EtCO_2$ values were found (Table 2).

In group I, the mean values of SpO_2 increased between observation minute 3 and 4 (MD = –1.15; $P = 0.004684$; –95% CI = –1.93; +95% CI = –0.35) (Table 3). Likewise, in group II, the mean values of SpO_2 increased between observation minute 3 and 4 (MD = –1.24; $P = 0.002144$; –95% CI = –2.0; +95% CI = –0.44) (Table 3). There were no intergroup

Table 1. Patient charakteristics *

Parameter	Group I	Group II
Age (years)	48.9 ± 7.6	53.3 ± 11.6
Body mass (kg)	76.9 ± 9.9	68.6 ± 7.4
Hematocrit (%)	44.8 ± 4.3	45.7 ± 5.4
Hemoglobin (g L^{-1})	145.2 ± 10.9	135.5 ± 12.3
Temperature (ºC)	36.7 ± 0.2	36.9 ± 0.4

*mean ± standard deviation

Table 2. Mean values of $ETCO_2$ (mm Hg) in investigated groups*

Group	Period of investigation (minutes)				
	6	7	8	9	10
I	38.0	38.1	38.2	38.3	38.0
	(1.1)	(1.0)	(1.0)	(0.9)	(0.8)
II	38.0	38.0	37.9	38.4	38.3
	(1.1)	(1.3)	(0.9)	(1.2)	(1.3)

*mean (standard deviation)

Table 3. Mean values of SpO_2(%) in investgated groups*

Group	Period of investgation (minutes)										
	0	1	2	3	4	5	6	7	8	9	10
I	97.8	97.8	98.0	98.0	99.2**	99.5	99.4	99.1	99.0	98.9	98.9
	(1.7)	(1.8)	(1.8)	(1.7)	(1.2)	(0.8)	(1.0)	(1.2)	(1.3)	(0.9)	(1.0)
II	96.9	96.9	97.3	97.9	99.0**	99.2	99.3	99.1	99.0	99.0	99.3
	(1.4)	(1.3)	(1.4)	(1.3)	(1.1)	(1.1)	(0.9)	(1.1)	(0.8)	(0.9)	(0.7)

*mean (standard deviation)
**$P < 0.05$ compared to previous value

differences in mean SpO_2 values at the respective times of observation.

In groups I and II, there were no statistically significant changes in heart rate observed during the successive stages of induction of anaesthesia, or intergroup differences at the respective times of observation (Fig. 1).

In group I, no statistically significant changes in MAP were observed during the first 6 minutes of the study. MAP decreased between minute 6 and 7 (MD = 12.57; $P = 0.0012$; –95% CI = 4.92; +95% CI = 20.19) and increased between minute 7 and 8 (MD = –23.57; $P = 0.000001$; –95% CI = –31.19; +95% CI = –15.95) (Fig. 2).

Likewise, in group II, there were no statistically significant changes in MAP during the first 6 minutes of observation. MAP decreased between minute 6 and 7 and increased between minute 7 and 8 (MD = 9.06; P = 0.018; –95% CI = 1.47; +95% CI = 17.71; MD = –15.0; $P = 0.000124$; –95% CI = –22.51; +95% CI = –7.38, respectively) (Fig. 2). No intergroup differences in mean MAP values were found at the respective times of observation.

In group I, no statistically significant changes in $V_{MCA}{}^L$ were observed during the first 6 minutes of observation. $V_{MCA}{}^L$ decreased between minute 6 and 7 (MD = 30.34; $P = 0.000001$; –95% CI = 20.64; +95% CI = 4.39) and increased between minute 7 and 8 (MD = –34.85; $P = 0.000001$; –95% CI = –45.08; +95% CI = –24.82). Likewise, in group II, there were no statistically significant differences in $V_{MCA}{}^L$ observed during the first 6 minutes of observation. A decrease was found between minute 6 and 7 (MD = 25.62; $P = 0.000001$; –95% CI = 15.74; +95% CI = 35.49) while an increase was noted between minute 7 and 8 (MD = –21.09; $P = 0.000031$; –95% CI = –30.97; +95% CI = –11.22) (Fig. 3).

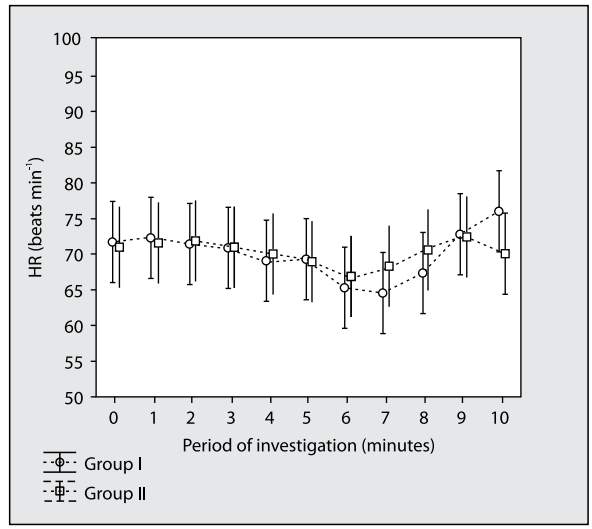

Figure 1. Mean values of heart rate (HR) during investigation. (mean ± 0,95 confidence interval)

In group I, there were no statistically significant changes in $V_{MCA}{}^P$ during the first 6 minutes of observation. The mean value of $V_{MCA}{}^P$ decreased between minute 6 and 7 (MD = 30.43; $P = 0.0000001$; –95% CI =20.36; +95% CI =40.50) and increased between minute 7 and 8 (MD = –32.32; $P = 0.0000001$; –95% CI = –42.39; +95% CI = –22.25). Likewise, in group II, there were no statistically significant changes in $V_{MCA}{}^P$ observed during the first 6 minutes of observation. The mean value of $V_{MCA}{}^P$ decreased between minute and 7 (MD = 26.03; $P = 0.0000001$; –95% CI =15.96; +95%, CI =36.10) and increased between minute 7 and 8 (MD = –21.55; $P = 0.00003$; –95% CI = –31.62; =95% CI = –11.48) (Fig. 4).

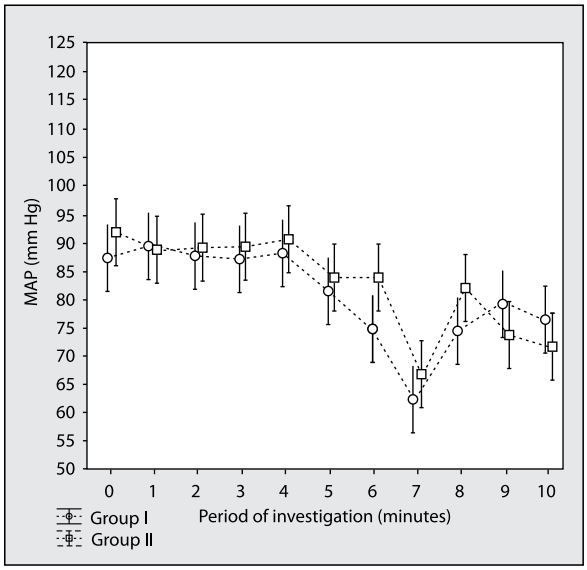

Figure 2. Mean values of mean arterial blood pressure (MAP) during ivestigation. (mean ± 0,95 confidence interval); *$P < 0.05$ compared to previous value in both groups

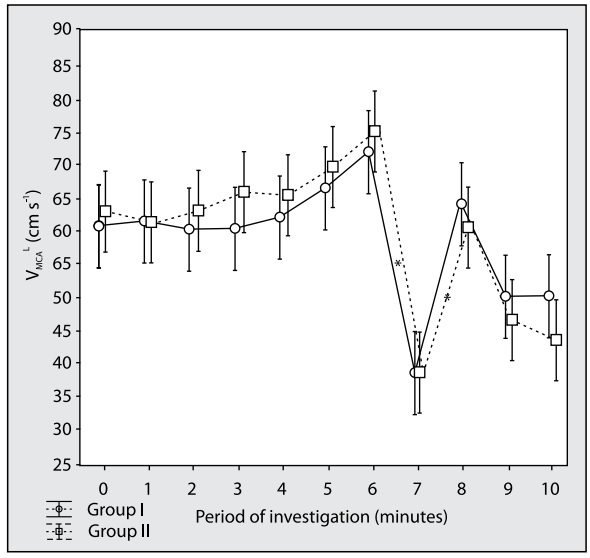

Figure 4. Mean values of right middle cerebral arterial flow velocity (V_{MCA}^{R}) in investigated group. (mean ± 0,95 confidence interval); *$P <$ 0,05 compared to previous value in both groups

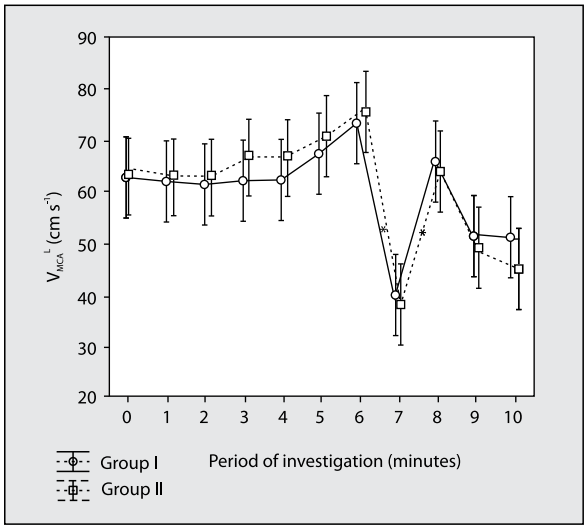

Figure 3. Mean values of left middle cerebral arterial flow velocity (V_{MCA}^{L}) in investigated group (mean ± 0,95 confidence interval); *$P <$ 0.05 compared to previous value in both groups

In group I, there was a weak correlation between mean MAP versus mean V_{MCA}^{L} (r = 0.20808) and mean V_{MCA}^{P} (r = 0.16158) (Figs 5, 6).

Likewise, in group II, there was a weak correlation between mean MAP versus mean V_{MCA}^{L} (r = 0.16392) and mean V_{MCA}^{P} (r = 0.18980) (Figs 7, 8).

DISCUSSION

The estimated prevalence of UIAs in the general population is 3% [23]. Our observations demonstrate that UIAs are more commonly located in the ICA and MCA [24]. UIAs were found to have no impact on autoregulation of cerebral circulation [25]. Endovascular treatment of UIAs leads to ipsilateral impairment of carbon dioxide reactivity of the cerebral vessels [26]. Moreover, our observations reveal that the presence of UIA does not affect bilateral V_{MCA}, which is consistent with the findings reported in the literature [25, 27].

Irrespective of the treatment method applied, patients undergoing surgeries require general anaesthesia. In our previous study, the usefulness of totally intravenous anaesthesia using propofol and a laryngeal mask was demonstrated [28].

As far as the maintenance of systemic homeostasis is concerned, the induction of anaesthesia is its key element as the anaesthetics administered depress both the CNS and the cardiovascular system. Standard monitoring in the operating room does not enable one to evaluate the effects of anaesthetics on the parameters of intracranial homeostasis.

Transcranial Doppler ultrasonography allows for non-invasive measurements of blood flow in large cerebral arteries, which indirectly reflects CBF. The use of a dose of propofol for the induction of anaesthesia has not fully ensured the stability of systemic circulation. Haemodynamic imbalance often accompanies the induction of anaesthesia, which is confirmed by the results of experimental [18] and clinical studies [20, 29].

There were no statistically significant changes in heart rate observed at the successive stages of induction. However, the tendency towards heart rate increases associated with direct laryngoscopy and endotracheal intubation is visible, which is consistent with the findings of many stud-

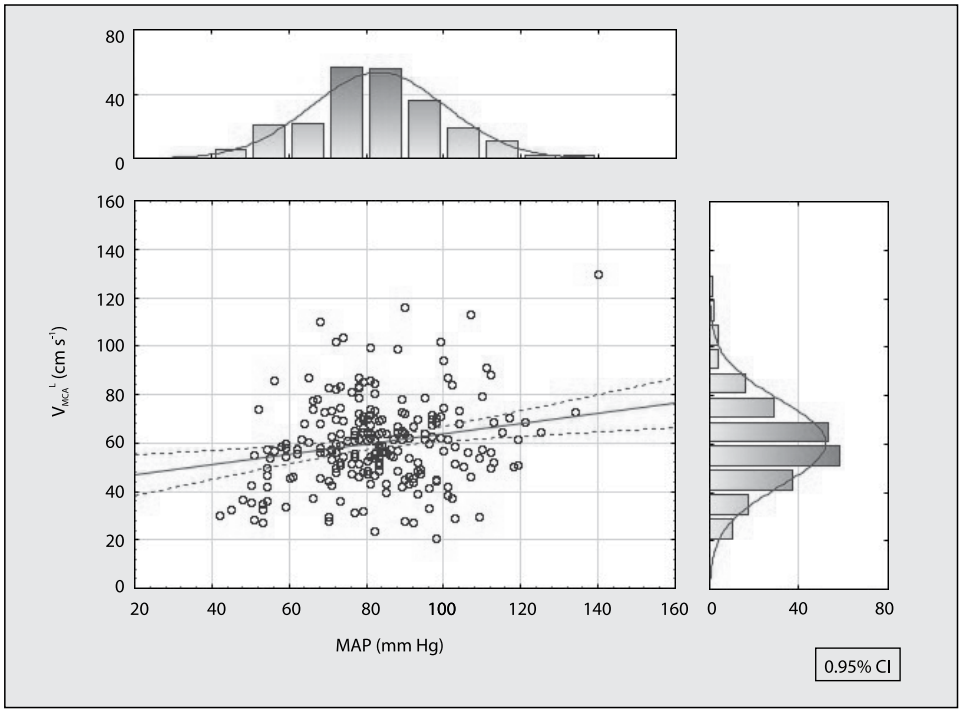

Figure 5. Correlation of mean arterial blood pressure (MAP) and mean values of left middle cerebral arterial flow velocity (V_{MCA}^{L}) in group I

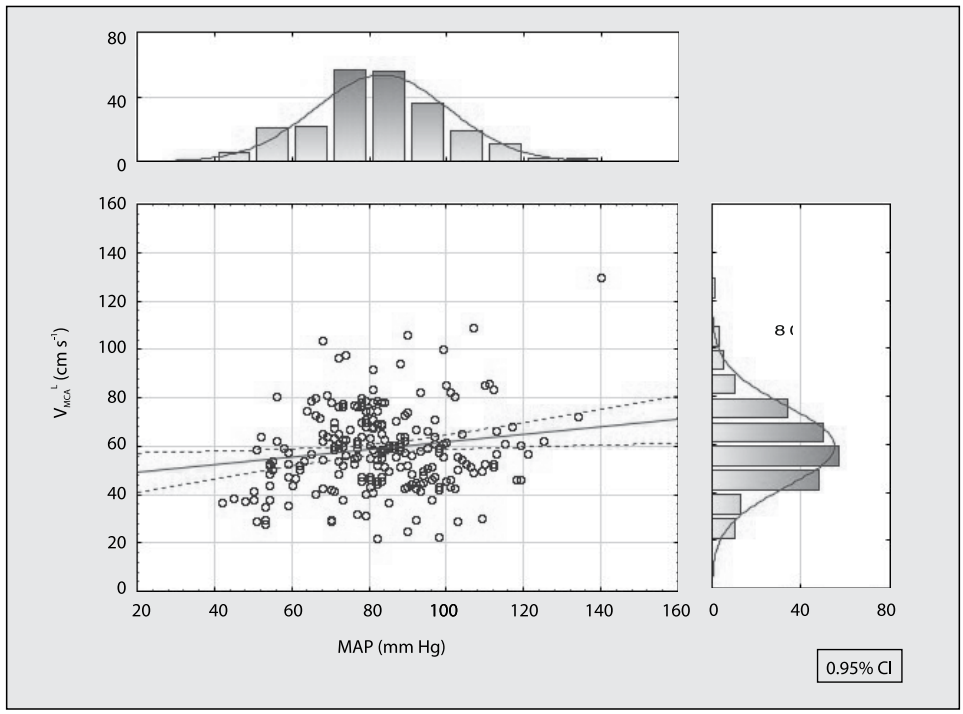

Figure 6. Correlation of mean arterial blood pressure (MAP) and mean values of right middle cerebral arterial flow velocity (V_{MCA}^{R}) in group I

ies [20, 30] and results from stimulation of the sympathetic nervous system. Interestingly, during anaesthesia with propofol, the induction causes increases in the concentration of norepinephrine [4]. Acting centrally sympatholytically and inhibiting the reactivity of baroreceptors, propofol leads to vagotomy, which results in the loss of reflex acceleration of heart rate in response to a decrease in arterial pressure [18, 20].

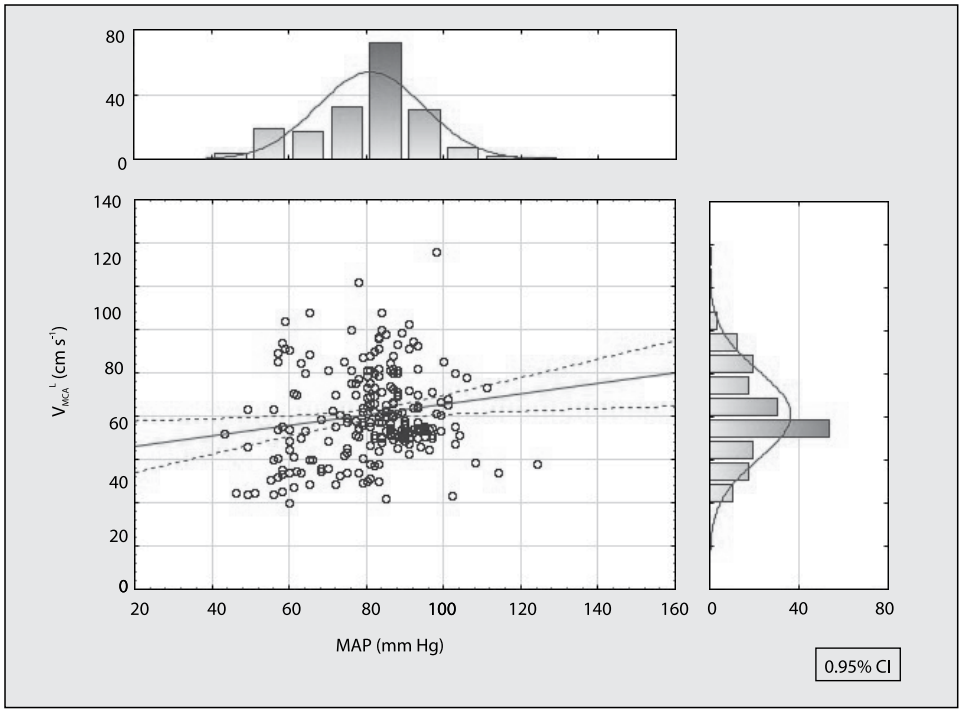

Figure 7. Correlation of mean arterial blood pressure (MAP) and mean values of left middle cerebral arterial flow velocity (V_{MCA}^{L}) in group II

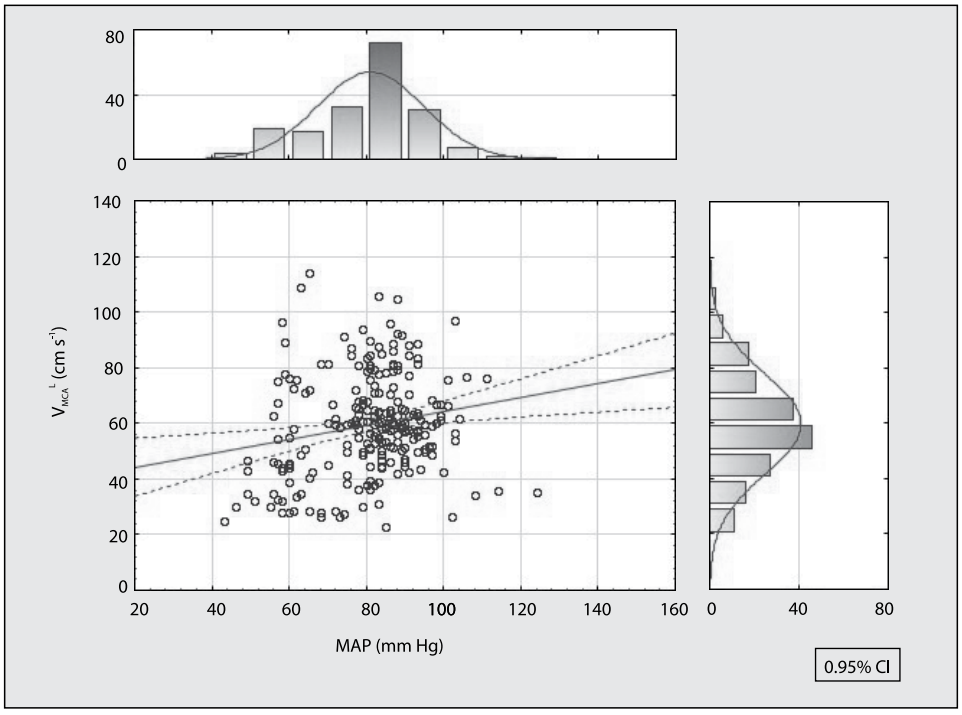

Figure 8. Correlation of mean arterial blood pressure (MAP) and mean values of right middle cerebral arterial flow velocity (V_{MCA}^{R}) in group II

A decrease in arterial pressure after intravenous induction of anaesthesia is commonly observed [4, 19, 20]. In their study analysing the induction with propofol, etomidate, thiopental and midazolam, Reich *et al.* [31] observed hypotension during the first 10 minutes of anaesthesia, particularly after the administration of propofol.

MAP is the product of cardiac output and peripheral vascular bed resistance [32]; its reduction induced by gen-

eral anaesthetics results from negative inotropic effects leading to reduced cardiac output and from relaxing effects on the vascular bed accompanied by decreased pre- and afterload [32]. Of note is the fact that the inhibitory effects of anaesthetics on the sympathetic nervous system seem essential [18, 22].

The findings of numerous studies have confirmed markedly larger decreases in MAP caused by propofol, as compared with equivalent doses of thiopental and etomidate [4, 20, 29, 31, 32]. Price *et al.* [29], who used transcranial Doppler (TCD) ultrasound, have demonstrated a 23% reduction in peripheral resistance during the induction of anaesthesia with propofol, as compared with baseline values; no such effects have been observed in the case of thiopental or etomidate. Goodchild *et al.* [21] have suggested a prophylactic transfusion of crystalloids in order to avoid sudden decreases in MAP after the administration of propofol. However, our observations did not confirm the expected effects. Using echocardiography, Bilotta *et al.* [33] observed reduced pre-load and afterload, as well as decreased myocardial contractility without reflex tachycardia in patients undergoing neurosurgical procedures and anaesthetised with propofol.

During laryngoscopy and endotracheal intubation, the mean values of MAP increased. Direct laryngoscopy and endotracheal intubation are one of the most stressogenic elements of general anaesthesia. Potent nociceptive stimulation causes sudden haemodynamic changes in the systemic circulation induced by the stimulation of the sympathetic nervous system [4, 34]. Kayhan *et al.* [35], using thiopental, fentanyl and vecuronium for the induction of anaesthesia, have observed intubation-induced increases in serum concentrations of epinephrine and norepinephrine, accompanied by increased MAP and an accelerated heart rate. The concentrations of norepinephrine and epinephrine normalised during the fifth minute after intubation. Increased concentrations of catecholamines in the venous blood mixture have also been demonstrated by Lindgren *et al.* [4]. The fact that the haemodynamic changes observed can be attenuated with β-blockers or α-2 agonists proves that the stimulation of the sympathetic nervous system exerts the most decisive effects on them [34].

Propofol used in the study, in the dose considered sufficient to induce anaesthesia, did not fully protect one against MAP increases in response to laryngoscopy and endotracheal intubation, which corresponds with the observations reported by other authors [36–38]. In a study comparing induction using propofol, thiopental and etomidate, El-Orbany *et al.* [36] observed increased MAP associated with endotracheal intubation. The values of MAP found in patients anaesthetised with propofol were significantly lower and persisted throughout the study. Erhan *et al.* [39], who analysed the effect of propofol combined with remifentanil, noted no increases in MAP after intubation. The discrepancies between the above study and our observations are likely to result from the effects of remifentanil, which causes bradycardia and hypotension [2].

Clinical practice has confirmed the effectiveness of opioids in limiting the circulatory effects of laryngoscopy and endotracheal intubation [2, 3, 30]. Fentanyl prevents tachycardia and arterial pressure increases during manipulations in the upper respiratory tract both in children [4] and adults [3, 30]. Moreover, fentanyl has been demonstrated to ensure higher haemodynamic stability of the systemic and cerebral circulation in response to intubation, as compared with remifentanil. [19]. Harris *et al.* [3] and Sakai *et al.* [30] have found that fentanyl at a dose of 2 μg kg^{-1} combined with etomidate, thiopental or propofol, sufficiently inhibits the response of the circulatory system to endotracheal intubation. Complete abolition of reflex reactions of the circulatory system to laryngoscopy and intubation requires much higher doses of drugs, as compared with those used in everyday clinical practice [40 41], which leads not only to their prolonged action and delayed recovery from anaesthesia but is also associated with the risk of more severe hypotension, especially in elderly patients [2].

Sudden changes in haemodynamic parameters during the induction of anaesthesia are particularly important in patients with additional cardiovascular and CNS diseases. In patients with the latter, impaired autoregulation of the cerebral circulation is often observed, and CPP is found to passively "follow" the changes to MAP [42, 43]. To prevent such adverse effects of laryngoscopy and intubation, many authors suggest local anaesthesia of the larynx with lignocaine and used the additional dose of a hypnotic immediately before endotracheal intubation or to apply of esmolol [30, 34, 37].

According to the results of many studies, when autoregulation is preserved, there is no direct correlation between MAP and V_{MCA} during anaesthesia [9, 44, 45, 46].

The findings of experimental and clinical studies confirm the thesis that propofol does not affect the mechanism of autoregulation [47] and CO_2 reactivity of the cerebral vessels [13]. Therefore, it can be assumed that in our patients autoregulation was presserved while MAP, as well as V_{MCA} changes were caused by various unrelated mechanisms, particularly that no correlation was found between the changes in their mean values.

TCD ultrasound does not directly measure CBF in the large vessels of the base of the brain. However, once a relatively constant cross-section of the vessel is maintained and the angle of insonation remains unchanged, the values of V_{MCA} determined using this method correlate well with the values of CBF, which has been demonstrated in experimental [48] and clinical studies [12, 49]. According to current

knowledge, the MCA cross-section does not change due to changes in MAP and $PaCO_2$ or during the use of anaesthetics or vasoactive drugs [12, 15]. The large arteries of the circle of Willis are conducting arteries, as opposed to the resistance vessels changing their lumina due to the above factors.

According to Giller *et al.* [50], changes in the diameters of the MCA, ICA and vertebral artery caused by changes in MAP or pCO_2 do not exceed 4%. Thus, if the MCA cross-section remains unchanged, V_{MCA} will reflect CBF.

Opioids have slight or no effects on V_{MCA} [19, 51]. Likewise, non-depolarising muscle relaxants have no such effects [52]. In our study, the patients were transfused with 500 ml of Ringer's solution during the 30 minutes preceding the study. Whenever this affected haematocrit, the effects were comparable in all patients. After the administration of the anaesthetic and relaxation, mechanical ventilation was initiated maintaining the level of $etCO_2$ within normal limits. SpO_2 was also within normal limits during the study, and thus had no effect on V_{MCA}. The temperature in the external auditory meatus monitored during the study is closest to the intracranial temperature. Its values were also within normal limits and no intergroup differences were observed.

Our study findings demonstrated that propofol reduced V_{MCA}. Similar results have been reported by many authors analysing V_{MCA} during the use of anaesthetics [53, 54]. A reduction in V_{MCA} results from propofol-induced CBF depression [54]. The studies using the classical Kety-Schmidt method and transcranial Doppler ultrasound [54], as well as relatively new positron emission tomography [6], have revealed that propofol depresses CBF in a dose-dependent manner. The above confirms the markedly reduced velocity of blood flow in the MCA induced by propofol used for induction that was found in our study. A reduction in CBF after the administration of propofol is observed in healthy patients [7, 10] as well as those with craniocerebral traumatic injuries [46] or brain tumours [55]. In our study, laryngoscopy- and intubation-induced increases in V_{MCA} were observed, which is consistent with the findings of numerous reports [19, 43, 53]. A strong intubation-induced nociceptive stimulation, conducted thorough the reticular formation of the brain stem, hypothalamus to the cerebral cortex [56], triggers a characteristic pattern of excitation in EEG recordings, even in deeply anaesthetised patients [30]. Enhanced activity of the cerebral cortex is associated with increased oxygen and glucose requirements, which results in increased CBF and accelerated V_{MCA}.

After the intubation-induced transient increase in V_{MCA}, its values decreased and were maintained at this level. The direct, strong vasoconstrictive effects of propofol on the cerebral vessels, described by some authors [11, 14], may at least partly explain our findings.

The lack of correlation between changes in V_{MCA} and MAP may partly evidence the efficient mechanism of autoregulation, which, in all likelihood, allows us to eliminate any possible effects of changes in haemodynamic parameters of the systemic circulation on the changes in V_{MCA} observed in our study.

Eng *et al.* [57], Karsli *et al.* [11] and Doyle *et al.* [58] found a propofol-induced reduction in V_{MCA} without a simultaneous decrease in MAP. According to Vandesteene *et al.* [45], propofol reduces CBF and increases the resistance of the cerebral circulation also in cases when MAP is maintained at a constant level using the infusion of phenylephrine. The authors have suggested that the above results from a decrease in cerebral metabolism caused by propofol and its direct vasoconstrictive effects on the cerebral vessels, which at least partly explains the lack of correlation between changes in MAP and V_{MCA} observed in our study.

When the feedback mechanism is preserved, the reduced cerebral metabolic rate (CMR) decreases glucose and oxygen requirements, simultaneously reducing CBF. The majority of authors have emphasised that propofol does not impair flow-metabolism coupling [54, 58].

In conclusion, propofol exerts depressive effects on the systems of systemic and cerebral circulation. From the point of view of everyday neuroanaesthesiological practice, the action of anaesthetics in cases of CNS pathologies is of key importance. Research regarding such issues broadens our knowledge about the effects of anaesthetics, thus improving the safety of surgical patients.

CONCLUSIONS

Propofol depresses the cerebral circulation during the induction of anaesthesia. The presence of unruptured intracranial aneurysm has no effect on the propofol-induced reactivity of the cerebral vessels.

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2. Conflict of interest: none

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