MODEL OF DEPENDENCE BETWEEN ARTERIAL BLOOD PRESSURE AND CEREBRAL BLOOD FLOW. STATE OF THE ART AND THE NEW PROPOSAL

MODEL ZALEŻNOŚCI POMIĘDZY TĘTNCZYM CIŚNIENIEM KRWI A MÓZGOWYM PRZEPŁYWEM KRWI. STAN WIEDZY I NOWA PROPOZYCJA

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ABSTRACT

Cerebral autoregulation is a very important and complicated process to maintain adequate and stable cerebral blood flow. In current medical practice cerebral autoregulation is diagnosed using two measurements: cerebral blood flow (by Doppler Ultrasonography, Magnetic Resonance Imaging or Computed Tomography) and arterial blood pressure.

The aim of our study is to determine the dependence of the cerebral blood flow (CBF) on the mean arterial pressure (MAP) by measuring blood pressure and cerebral blood flow using modelling and data processing. The dependence will be presented as a curve based on the Strandgaard's observation of cerebral blood flow changes in response to changes in the mean arterial pressure. These observations we have described using polynomials of degree n = 3, 4, 5 and 6. The four curves were identified using least-squares fitting to Strandgaard's measurements. As the best of the considered curves of degree n = 3, 4, 5 and 6 we choose the one for which the Akaike Information Criterion AIC reaches a minimum. The criterion in the minimum finds compromise between the goodness of fit of the curve to the data and the curve complexity. Minimum of AIC was obtained for n = 4 (Kalicka&Mazur curve). The modelled dependence of CBF on MAP was used in CBF(MAP) simulations for important and typical pressure values: normotensive, hypertensive and patients under unexpected stress.

The model selected by the AIC informative criterion, degree n = 4, proved to be good candidate for describing CBF(MAP). This model (Kalicka&Mazur) gives CBF values which are comparable to those the measured for successive MAP values. The measured and the modelled Kalicka&Mazur curve CBF values differed less than the measured and the modelled CBF values of the Dirnagl&Pulsinelli curve and the Olsen curve.

Keywords: model, cerebral blood flow, mean arterial pressure, Lassen's curve

STRESZCZENIE

Autoregulacja przepływu mózgowego jest niezwykle ważnym i skomplikowanym procesem umożliwiającym utrzymanie stałych i stabilnych wartości przepływu mózgowego. Obecnie, zaburzenia autoregulacji są diagnozowane dwoma metodami: ocena przepływu mózgowego krwi na tętnicy szyjnej (przy użyciu USG Dopplera) oraz w tkankach mózgowych (przy użyciu rezonansu magnetycznego).

Celem tej pracy jest określenie zależności pomiędzy mózgowym przepływem krwi (CBF) a średnim ciśnieniem tętniczym (MAP). Utworzona i wykorzystana zależność CBF(MAP) bazuje na pomiarach Strandgaard'a. Na podstawie tych pomiarów zostały utworzone wielomiany 3, 4, 5 i 6 stopnia poprzez dopasowanie ich, metodą najmniejszych kwadratów, do pomiarów Strandgaard'a. Informacyjne kryterium Akaike (AIC) pozwoliło na wybranie wielomianu stopnia 4 jako modelu osiągającego najlepszy kompromis pomiędzy dobrocią dopasowania krzywej do danych a złożonością krzywej (najmniejsza wartość AIC). Wielomian ten został nazwany krzywa Kalicka&Mazur. Tak przygotowana zależność CBF(MAP) posłużyła do symulacji wartości CBF dla najbardziej typowych wartości ciśnienia: dla pacjentów ze standardowym ciśnieniem tętniczym, dla pacjentów ze stale podwyższonym ciśnieniem tętniczym, a także dla pacjentów z nagłym skokiem ciśnienia tętniczego (pacjenci poddani nagłemu stresowi).

Wybrany i zamodelowany wielomian 4 stopnia, krzywa Kalicka&Mazur, wydaje się być dobrym pomysłem na opisanie zależności zmiany CBF(MAP). Wartości zamodelowane dzięki krzywej Kalicka&Mazur w porównaniu z istniejącymi krzywymi Dirnagl&Pulsinelli czy Olsena daje bardziej obiecujące efekty przy porównaniu na danych rzeczywistych.

Słowa kluczowe: model, mózgowy przepływ krwi, średnie ciśnienie tętnicze, krzywa Lassena

1. Introduction

Cerebral autoregulation (*CA*) is a process of maintaining constant cerebral perfusion and brain tissue oxygenation with regard to changes in arterial blood pressure. It plays a dominant role in cerebral blood flow (*CBF*) homeostasis. *CBF* is the blood supply to the brain at given time. The average value of CBF is 50 to 54 ml of blood per 100 g of brain tissue per minute. It is evaluated also as 15% of the cardiac output. *CBF* depends on cerebral perfusion pressure (*CPP*) and vascular resistance (*SVR*), [1]:

$$CBF = \frac{CPP}{SVR} \tag{1}$$

where $CPP = MAP - ICP$, and *ICP* is intracranial pressure.

CBF is controlled to maintain its constant value. The regulation mechanisms are: metabolic (sometimes named chemical), myogenic and neurogenic control. The task of metabolic control is to adjust *CBF* values in relation to varying concentrations of metabolites. Myogenic control is the response of vascular smooth muscle cells to changes in transmural blood pressure [2]. Neutral control [3] is the action of vascular smooth muscle actuators in the resistance arterioles, which are controlled via sympathetic innervation.

When mean arterial pressure (*MAP*) stays within the range of 60 mmHg to 150 mmHg, then the value of *CBF* is constant. *MAP* is a weighted average blood pressure [1]:

$$MAP = \frac{2 \cdot DP + SP}{3} \tag{2}$$

where the SP is systolic arterial pressure and DP is diastolic arterial pressure.

MAP is defined also as:

$$MAP = (CO \cdot SVR) + CVP \tag{3}$$

where the *CO* is cardiac output, and *CVP* is central venous pressure, usually small enough to be neglected in this formula.

Changes of *CBF* correspond to the physiological state of the brain. Cerebral blood flow is related to arterial blood pressure according to the Lassen's curve. The curve, with a wide plateau, [4] is a dependence between mean arterial blood pressure and cerebral blood flow. Originally, Lassen's curve was plotted using the mean values of 11 category groups reported in 7 studies. The curve was made by

joining the *CBF* values observed in each group. Other researchers [5, 6] have reanalyzed Lassen's curve by dividing it into three parts: lower limit of autoregulation (*LLA*), upper limit of autoregulation (*ULA*) and plateau. Below *LLA* there is a decrease in *CBF* from 0.9% to 2.6% for every 1 mmHg decrease in *MAP*. Above the *ULA*, *CBF* increases from 2.2% to even 5% (for rats) for every 1 mmHg increase in *MAP*.

2. Objectives

The aim of the study presented in this paper is to analyze mathematical dependencies known from literature and search for new mathematical dependencies between arterial blood pressure and cerebral blood flow. The most popular is Lassen's classic cerebral blood flow curve *CBF*(*MAP*) [2, 7] (Fig. 1). The shape and range of the curve plateau depend on the taken measurements and the author's interpretation. Traditional physiology literature [2, 8] shows that *CA* is efficient and *CBF* is constant when *MAP* is in the range of 80 mmHg to 180 mmHg. Another approach is presented by Czosnyka [9]. He has modified Lassen's curve to depend on exclusively on *CPP*, and not on *MAP*: *CA* is efficient and *CBF* is constant when the range of *CPP* is from 60 mmHg to 100 mmHg. *CPP* is the difference between *MAP* and intracranial pressure (*ICP*):

$$CPP = MAP - ICP \tag{4}$$

ICP is normally equal to 7–15 mmHg. Czosnyka observations suggest a narrower control range than the Lassen range of *CA*.

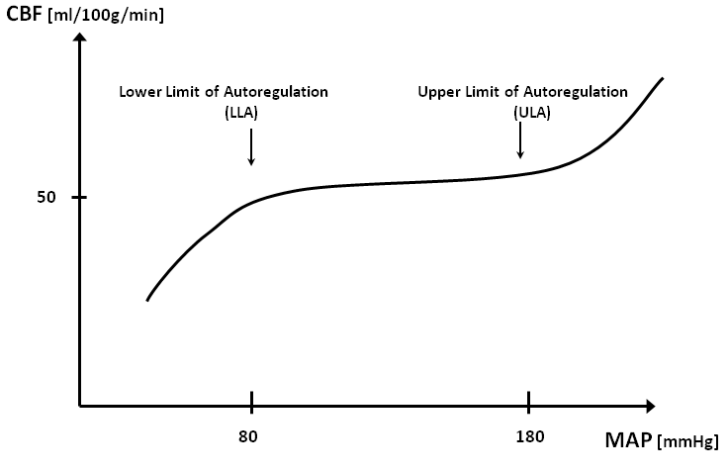

Fig. 1. Classic Lassen's curve based on [2] with the cerebral autoregulation range between 80 and 180 mmHg of mean arterial pressure (*MAP*)

The most important models of dependence between *MAP* and *CBF* are presented below.

The M. Ursino et al. [10] mathematical model of intracranial hemodynamics included data from transcranial Doppler velocity in the middle cerebral artery (*MCA*), intracranial pressure (*ICP*), mean systemic arterial pressure (*SAP*) and CO_2 arterial pressure (pCO_2).

Paper [11] presents a mathematical model of cerebrospinal fluid pressure (*CSF*) volume compensation (originally designed by A. Marmarou, 1973) with modifications. The model includes *CSF* production, circulation, absorption and storage. It also shows the hydrodynamic consequence of shunting.

CA is a complex process and is still not fully understood. Some models of *CA* are based on parameters measured on a normotensive patient. Experiments relate to parameters such as blood pressure, velocity of blood in the middle cerebral artery (Doppler ultrasound), blood oxygenation level and the subarachnoid space width (NIRT, MRI, CT). Attempts are being made to find interdependences between the signals. For instance, paper [12] presents measurements of fluctuation of arterial blood pressure (*ABP*) and cerebral blood flow velocity (*CBFV*) in a middle cerebral artery using transcranial Doppler ultrasonography and photoplethysmography. Analysis of these measurements, e.g. by using the complex

Morlet wavelet, showed the phase synchronization between fluctuations of arterial blood pressure and blood flow velocity for a normotensive subject.

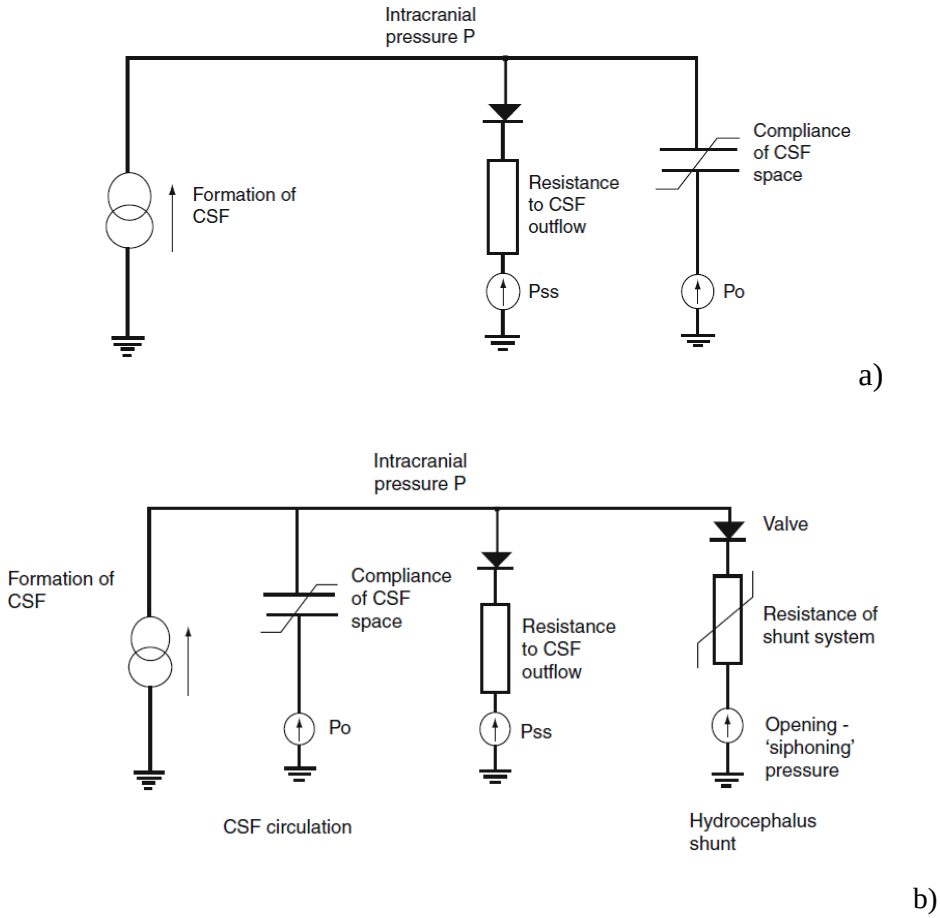

Fig. 2. Model of cerebrospinal fluid (*CSF*) dynamics according to Marmarou. a): source represents formation of *CSF*, resistor and diode – unilateral absorption to sagittal sinus, P_{SS} represents sagittal sinus pressure, capacitor – nonlinear compliance of the *CSF* space. b): extended model analyzed by Czosnyka [11].

Y.C. Tzeng and P.N. Ainslie [7] present a comprehensive description of cerebral autoregulation. Described are aspects of *CA*, such as: the relationship between average blood pressure and average blood flow under steady-state conditions; the dynamic relationship between blood pressure and blood flow; factors which can potentially influence interpretation of pressure-flow relationships and physiological mechanisms which are responsible for the *CA* response (nitric oxide, neurogenic factors, myogenic factors).

S.K. Piechnik et al. [13], present a theoretical model of cerebrospinal fluid circulation and cerebral venous blood outflow using laboratory instruments such as a pump, bubble trap, *ICP* level control, drop counter, etc. This laboratory set enabled analysis of cerebrospinal fluid circulation.

The above described models include parameters, such as arterial blood pressure, cerebral blood flow, cerebrovascular resistance and cerebral perfusion pressure, which are important in the *CA* process. However, none of the above *CA* models encompasses all these parameters.

3. Methods

Dirnagl and Pulsinelli [14] used measured *CBF* and *MAP* values to produce the following *CBF*(*MAP*) curve:

$$CBF_{\text{Dimagl\&Pulsinelli}} = 6.11 \cdot 10^{-5} \cdot MAP^3 - 2.37 \cdot 10^{-2} \cdot MAP^2 + 3.00 \cdot MAP - 75.00 \qquad (5)$$

CBF(MAP) curve obtained by Olsen et al. is as follows:

$$CBF_{Olsen} = 4.79 \cdot 10^{-5} \cdot MAP^3 - 1.74 \cdot 10^{-2} \cdot MAP^2 + 2.51 \cdot MAP - 38.80 \quad (6)$$

The above autoregulation curves were prepared by different researchers as a result of different experiments. Dirnagl&Pulsinelli curve (Fig. 4, solid line) is based on the study of 15 spontaneously hypertensive rats anesthetized with halothane. Olsen's curve (Fig. 4, dotted line) is based on the study of eight healthy volunteers whose mean arterial blood pressure was increased by the infusion of angiotensin and decreased by a combination of lower-body negative pressure and labetalol.

In the Lassen's curve, two values are indicated: the lower limit of autoregulation (*LLA*) and the upper limit of autoregulation (*ULA*). The average values of *LLA* and *ULA*, for human are 70 mmHg and 161 mmHg, respectively. Beyond this range there are slopes: the lower slope and the upper slope. The lower slope is a 1.27 percent change in *CBF* per 1 mmHg, and the upper slope is a 3.7 percent change in *CBF* per 1 mmHg [6, 15]. The normal value of *CBF* is assumed to be 50 ÷ 54 ml / 100 g / min. Based on the above data, we considered the following relationships between *MAP* and *CBF* to fulfill the given *ULA* and *LLA* slopes and the plateau range (Fig. 3):

$$CBF_{\text{degree3}} = 1.262 \cdot 10^{-4} \cdot MAP^3 - 3.606 \cdot 10^{-2} \cdot MAP^2 + 3.2067 \cdot MAP - 36.570 \quad (7)$$

$$CBF_{\text{degree4}} = 1.589 \cdot 10^{-6} \cdot MAP^4 - 5.411 \cdot 10^{-4} \cdot MAP^3 + 5.898 \cdot 10^{-2} \cdot MAP^2 - \\ - 2.0369 \cdot MAP + 53.019 \quad (8)$$

$$CBF_{degree5} = 1.053 \cdot 10^{-8} \cdot MAP^5 - 3.940 \cdot 10^{-6} \cdot MAP^4 + 5.335 \cdot 10^{-4} \cdot MAP^3 - \\ - 3.570 \cdot 10^{-2} \cdot MAP^2 + 1.641 \cdot MAP + 4.328 \quad (9)$$

$$CBF_{degree6} = 1.005 \cdot 10^{-10} \cdot MAP^6 - 5.276 \cdot 10^{-8} \cdot MAP^5 + 1.166 \cdot 10^{-5} \cdot MAP^4 - \\ - 1.367 \cdot 10^{-3} \cdot MAP^3 + 8.302 \cdot 10^{-2} \cdot MAP^2 - 1.890 \cdot MAP + 42{,}552 \quad (10)$$

For hypertensive emergency, systolic pressure higher than 180 mmHg and diastolic pressure higher 120 mmHg [16], the *MAP* is 140 mmHg. We considered a *MAP* range from 20 mmHg to 190 mmHg. These are limit values for people.

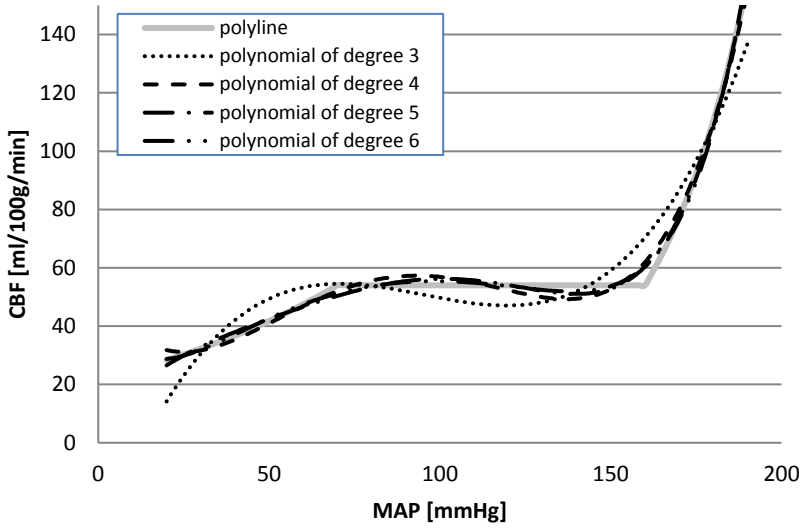

Fig. 3. Polyline (grey line) is based on the Strandgaard's observations [16]: lower slope 1.27% *CBF* per 1 mmHg *MAP*, upper slope 3.7% *CBF* per 1 mmHg of *MAP*, range of plateau from 70 to 160 mmHg. Regression functions of the polyline, in the form of polynomials of degree 3 to 6, were obtained by fitting procedure

In accordance with the Akaike Information Criterion (AIC criterion) the best potential model is one which provides a good fit to the date and, at the same time, has a small number of parameters. This criterion is [17]:

$$AIC = OF(\mathbf{p}) + 2 \cdot n = min \tag{11}$$

where n is number of model parameters $\mathbf{p} = [p_i], i = 3,..,n$, and OF is the objective function, defined as follows [17]:

$$OF = \sqrt{\frac{\sum_{i=1}^{N} \left(CBF_{\text{degree(n-1)}}(\mathbf{p}, MAP_i) - CBF_{\text{polyline}}(MAP_i) \right)^2}{N - n}} \tag{12}$$

$$\mathbf{p} = \arg\left(OF(\mathbf{p}) = \min \right) \tag{13}$$

The second element is a penalty function for too many model parameters. The *AIC* values for regression functions in the form of polynomials of degree 3 to 6 are presented in Table 1.

Table 1. Comparison of the quality of polynomial regression functions using the Akaike criterion

The compared polynomial regression functions	Akaike Criterion $AIC = OF + 2 \cdot n = \min$
Degree 3	$7.309 + 2 \cdot 4 = 15.309$
Degree 4	$2.776 + 2 \cdot 5 = 12.776$
Degree 5	$1.990 + 2 \cdot 6 = 13.990$
Degree 6	$1.942 + 2 \cdot 7 = 15.942$

Table 1 shows that the polynomial of degree 4 ensures an *AIC* minimum, therefore it was selected as our relationship between *CBF* and *MAP* and called the Kalicka&Mazur curve. Dirnagl&Pulsinelli, Olsen and Kalicka&Mazur curves are shown in Fig. 4. The Kalicka&Mazur curve is situated between the other two curves and it is consistent with both the form and the scope of the classic Lassen curve [4].

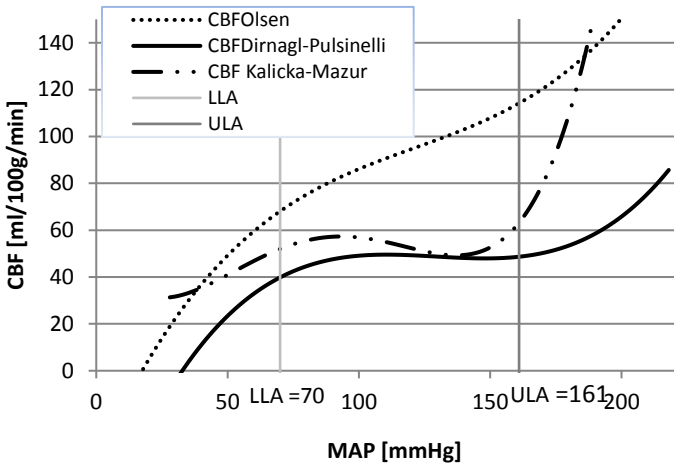

Fig. 4. Comparison of regression functions: Dirnagl&Pulsinelli curve (solid line), Olsen curve (dotted line) and Kalicka&Mazur curve (dash dot line). The vertical lines show lower and upper limits of autoregulation: *LLA* = 70 mmHg and *ULA* = 161 mmHg.

The input signal for the model is arterial blood pressure (*ABP*). It changes between the highest value (systolic blood pressure SP) and the lowest value (diastolic blood pressure *DP*). According to formula (2), the value of *MAP* for normotensive patients is:

$$MAP = \frac{2 \cdot 70 + 120}{3} = 86.67[\text{mmHg}] \tag{14}$$

and for hypertensive patients:

$$MAP = \frac{2 \cdot 90 + 150}{3} = 110.00[\text{mmHg}] \tag{15}$$

MAP may also increase as a consequence of momentary stress or a taken medicine. To represent such an increase in *MAP*, we used the Gaussian curve on account of its smooth, physiological shape:

$$MAP(t) = a \cdot \exp \frac{-(t-b)^2}{2 \cdot c^2} + d \tag{16}$$

with the following parameters: $a = 60$, $b = 40$, $c = 10$, $d = 80$. The resulting curve is shown in Fig. 6: it has a shape similar to the physiological MAP response to stress.

Equations (5) (6) (8) present three dependences of *CBF*(*MAP*). Each equation was used in a different model, and each model included normotensive patients, hypertensive patients and patients under stress (Gaussian input signal). These models are placed in the Table 2.

Table 2. Input (*MAP*) and output (*CBF*) signals for Dirnagl&Pulsinelli, Olsen and Kalicka&Mazur curves

Input signal: *MAP*	Type of relationship *CBF* versus *MAP*	Output signal: *CBF*(*MAP*)
MAP_norm (normotensive patients)	Dirnagl&Pulsinelli's curve	CBF_norm_DP
	Olsen's curve	CBF_norm_O
	Kalicka&Mazur's curve	CBF_norm_KM
MAP_hyper (hypertensive patients)	Dirnagl&Pulsinelli's curve	CBF_hyper_DP
	Olsen's curve	CBF_hyper_O
	Kalicka&Mazur's curve	CBF_hyper_KM
MAP_stress (patients under stress)	Dirnagl&Pulsinelli's curve	CBF_stress_DP
	Olsen's curve	CBF_stress_O
	Kalicka&Mazur's curve	CBF_stress_KM

For all the models, the dependence *CBF(MAP)* was simulated using the Simulink program (Matlab). The Simulink scheme for modelling and simulating the Dirnagl&Pulsinelli *CBF*(*MAP*) curve is presented in Fig. 5.

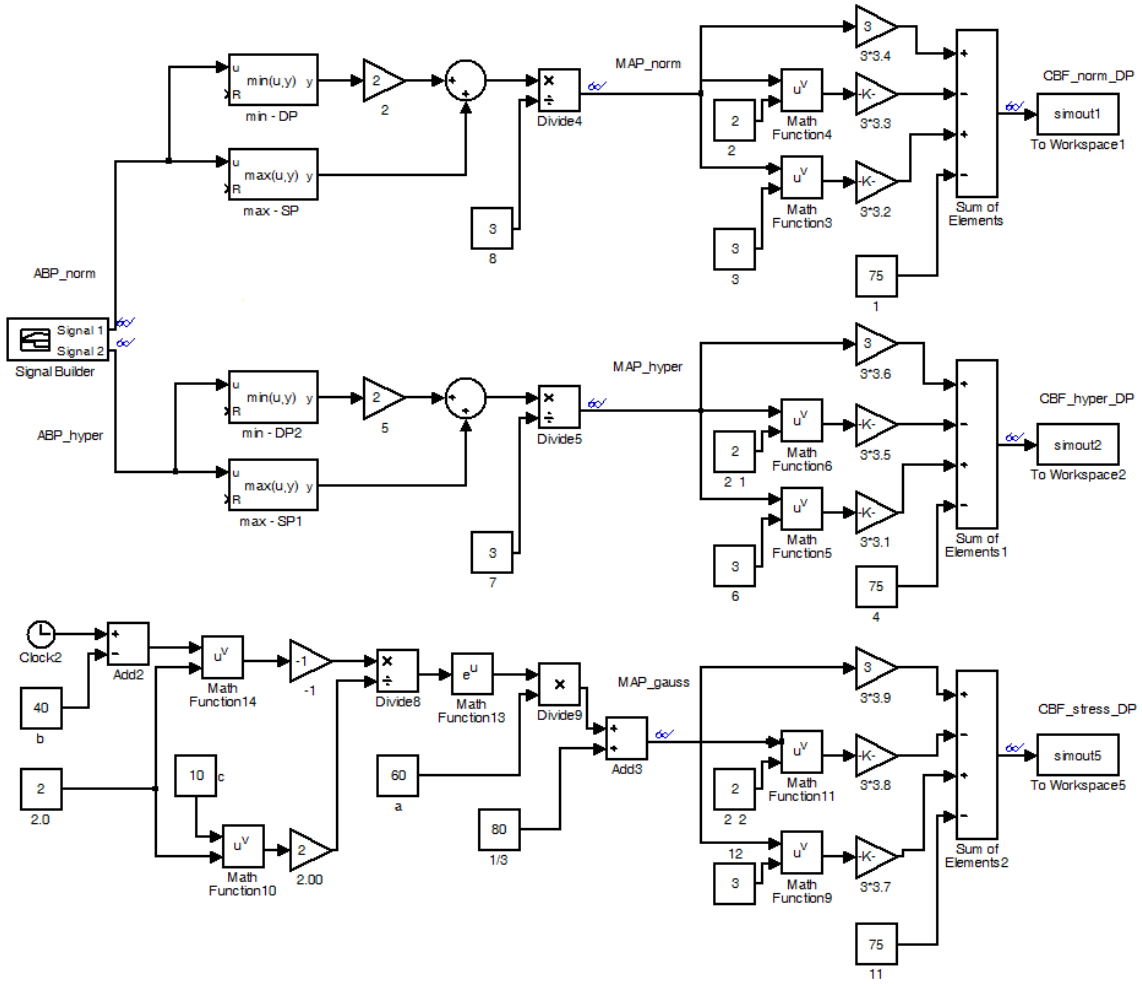

Fig. 5. Simulink *CBF*(*MAP*) scheme for the Dirnagl&Pulsinelli curve, including patients who were normotensive, hypertensive or under momentary stress

Figure 5 presents the Simulink scheme of *CBF*(*MAP*) for Dirnagl&Pulsinelli curve. Simulink schemes were created also for the Olsen and Kalicka&Mazur curves.

4. Results

The main purpose of cerebral autoregulation (*CA*) is to maintain a constant value of cerebral blood flow *CBF* versus *MAP*. We considered three types of *MAP* which are characteristic for normotensive patients, hypertensive patients and patients under momentary stress (see Fig. 6).

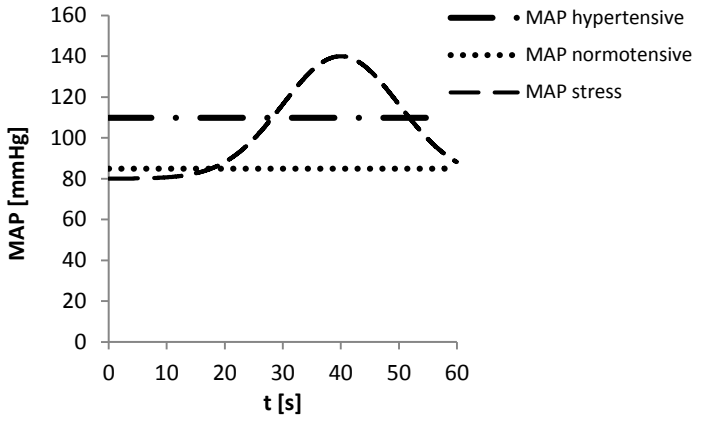

Fig. 6. Mean arterial pressure (*MAP*) for normotensive patients (dotted line), hypertensive patients (dash dot line) and patients under momentary stress (dash line)

Next, using Simulink software, *CBF*(*MAP*) was simulated for normotensive, hypertensive, and momentary stress patients. The results are presented in Figure 7.

Figure 7 shows that in Dirnagl&Pulsinelli and Kalicka&Mazur curves for normotensive, hypertensive and momentary stress patients the *CBF* is constant despite an MAP increase. On the other hand, in Olsen's curve *CBF* values are not constant, which means that *CA* is not fully successful.

Models were tested for measurements [18]of $MAP_{measured}$ and $CBF_{measured}$. The measurements were taken from 13 patients suffering from severe atheromatous occlusive disease and undergoing internal carotid endarterectomy (treated patients) and also 11 patients suffering from cerebral vascular disease but not undergoing surgery (control patients) [18]. The *MAP* measurements, $MAP_{measured}$ were used in the Olsen, Dirnagl&Pulsinelli and Kalicka&Mazur models to obtain the modeled cerebral blood flow, $CBF_{modelled}$. A comparison of the $CBF_{measured}$ and the $CBF_{modelled}$ is shown in Table 3.

Table 3. Comparison of $CBF_{measured}$ [18] and $CBF_{modelled}$ (calculated from $MAP_{measured}$ [18] and the Olsen, Dirnagl&Pulsinelli and Kalicka&Mazur curves). Shaded cells refer to the results obtained from laboratory rats

	$MAP_{measured}$ [mmHg] ± *std dev*	$CBF_{measured}$ [ml/100g/min] ± *std dev*	$CBF_{modelled}$ [ml/100g/min]	$\left\|\frac{CBF_{modelled} - CBF_{measured}}{CBF_{meassured}}\right\|$ [%]
treated with endarterectomy, first measurement				
Olsen	106 ± 15	46.9 ± 9.6	88.80	89.35
Dirnagl&Pulsinelli	106 ± 15	46.9 ± 9.6	49.48	5.50
Kalicka&Mazur	106 ± 15	46.9 ± 9.6	56.89	21.30
treated with endarterectomy, second measurement				
Olsen	96 ± 15	52.5 ± 7.3	84.18	60.34
Dirnagl&Pulsinelli	96 ± 15	52.5 ± 7.3	48.64	7.36
Kalicka&Mazur	96 ± 15	52.5 ± 7.3	57.23	9.02
control, first measurement				
Olsen	110 ± 14	38.5 ± 6.6	90.51	135.10
Dirnagl&Pulsinelli	110 ± 14	38.5 ± 6.6	49.55	28.71
Kalicka&Mazur	110 ± 14	38.5 ± 6.6	55.01	42.89
control, second measurement				
Olsen	118 ± 23	35.9 ± 7.3	93.80	161.29
Dirnagl&Pulsinelli	118 ± 23	35.9 ± 7.3	49.39	37.58
Kalicka&Mazur	118 ± 23	35.9 ± 7.3	52.87	47.27

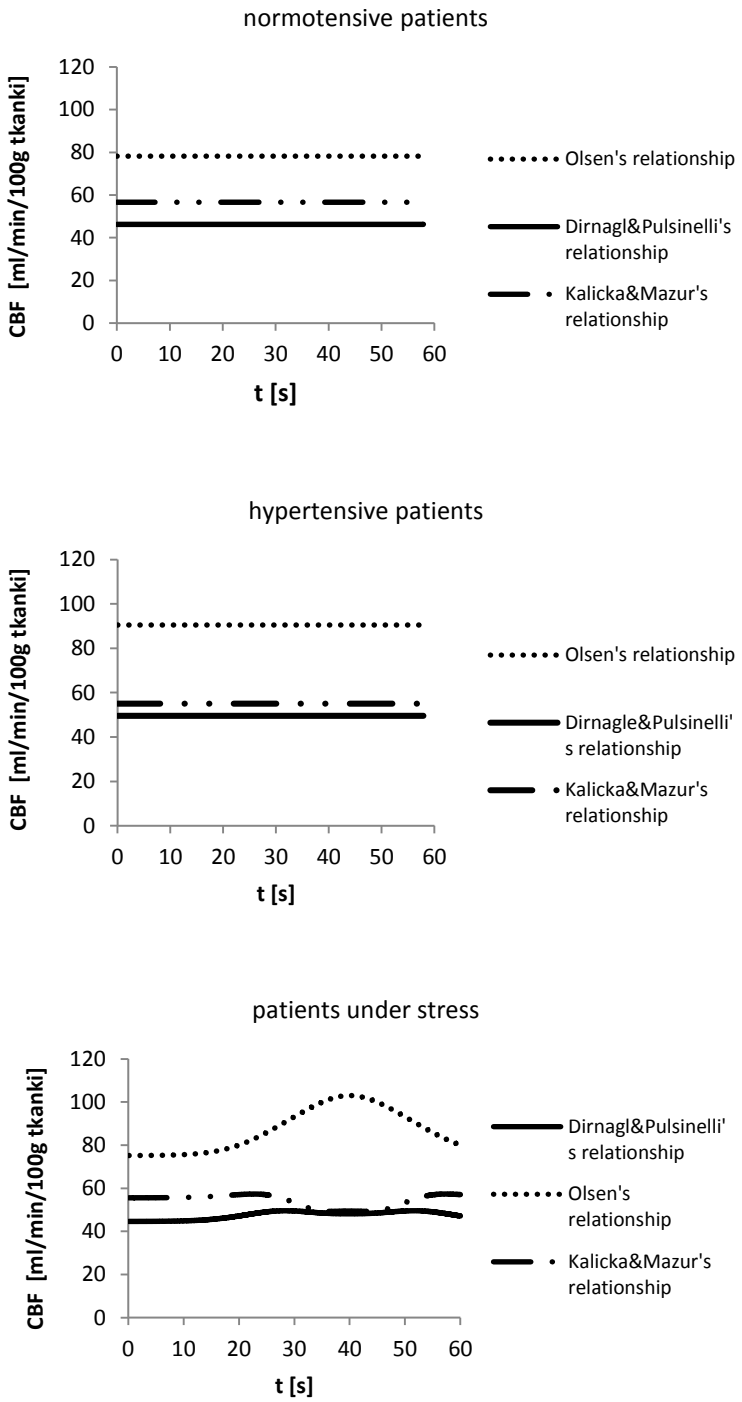

Fig. 7. *CBF* values of in Dirnagl&Pulsinelli curve (straight line), Olsen curve (dotted line) and Kalicka&Mazur curve (dash dot line) for normotensive patients, hypertensive patients and patients under momentary stress

Table 3 presents a comparison of the $CBF_{measured}$ and the $CBF_{modelled}$. The shaded cells in the Table 3 concern results obtained from 15 hypertensive laboratory rats [14]. The remaining results were obtained from human patients.

5. Discussion

The Kalicka&Mazur *CBF(MAP)* curve was obtained by fitting polynomials to Strandgaard's observations [15]. Next, the Akaike Information Criterion was used to choose a polynomial that provides a minimum *AIC*. The polynomial of degree 4 was chosen and called the Kalicka&Mazur curve.

The $CBF_{measured}$ and $MAP_{measured}$ were obtained from 13 treated patients and 11 control patients [18]. We compared this measured data with the modeled data obtained from the Dirnagl&Pulsinelli, Olsen and Kalicka&Mazur curves. The relative difference between $CBF_{measured}$ and $CBF_{modelled}$ is smaller in the Kalicka&Mazur curve than in the Olsen curve, but larger than in the Dirnagl&Pulsinelli curve. However, it should be noticed that Dirnagl&Pulsinelli curve was obtained from measurements of laboratory animals (rats). The results for people and for laboratory animals are difficult to compare.

6. Conclusions

Cerebral autoregulation is a very important process that largely depends on mean arterial pressure. The aim of this article was to find a mathematical dependence between cerebral blood flow, *CBF*, and mean arterial blood pressure, *MAP*, (with the use of modelling and data processing) and to compare it with previously used models. We choose Strandgaard's observation as a basis for the calculations. We applied the polynomials of degree $n = 3, 4, 5$ and 6 to the *CBF*(*MAP*) dependence using the least-squares fitting method. Using the Akaike Information Criterion, *AIC*. we choose polynomial of degree $n = 4$ (the minimum of *AIC*) and called it the Kalicka&Mazur curve. The Kalicka&Mazur's dependence between *CBF* and *MAP* was used for simulations *CBF*(*MAP*) of important and typical pressure values: normotensive, hypertensive and momentary stress. We compared the Kalicka&Mazur $CBF_{modelled}$ values with those of Dirnagl&Pulsinelli and Olsen (as typical autoregulation curves) and with $CBF_{measured}$. The relative difference between $CBF_{measured}$ and $CBF_{modelled}$ in the Kalicka&Mazur curve is smaller than in the Olsen curve.

Three important conclusions may be drawn from the above analyses and calculations. First, literature and our calculations have shown that while every person has an individual *CBF*(*MAP*) curve, all of them fall within a certain range (Lasen's curve). Second, the Kalicka&Mazur curve compares well with other *CBF*(*MAP*) curves. Third, the Kalicka&Mazur curve serves as a first step to modelling the cerebral autoregulation process.

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