

MODELING OF SUBARACHNOID SPACE WIDTH CHANGE CAUSED BY BLOOD CIRCULATION IN BRAIN VESSELS

Kamila Mazur¹ and Renata Kalicka²

^{1,2}Faculty of Electronics, Telecommunications and Informatics, Gdańsk University of Technology, Narutowicza 11/12, 80-233 Gdańsk, ¹kammazur@pg.gda.pl, ²renata.kalicka@biomed.eti.pg.gda.pl

ABSTRACT

The subarachnoid space is an anatomic space, lying in the central nervous system, between the arachnoid membrane and the pia mater. It is filled with cerebrospinal fluid, which protects and nourishes the brain. The pathological disorder of the subarachnoid space (i.e. increase or decrease of its width) affects the nervous system. Therefore, it is important to diagnose the width changes of the subarachnoid space. One way to support the diagnostics of subarachnoid space is to model proper and improper work of human body. This article presents a model describing the width changes of the natural subarachnoid space due to blood flow velocity (heartbeat).

INTRODUCTION

The subarachnoid space (SAS) is an anatomic space between the pia mater and the arachnoid layers, compare Fig. 1. SAS is located in the skull and surrounds the brain [10]. The width of SAS is constant for each person and varies only slightly due to the blood circulation through the brain vessels. Large changes are pathology, and might be caused by a disease or an injury. Any deviation from normal physiological state causes damage of neurons. Damage of many neurons causes damage to the whole brain. Therefore, that is important to detect early enough the subarachnoid space decrease. This allows for counteracting negative changes in the brain by surgical intervention or pharmacological treatment. Currently, changes of the subarachnoid space are diagnosed on the basis of external symptoms, such as vomiting or headache, and by tests: Magnetic Resonance Imaging (MRI) or Computer Tomography (CT). These two methods are accurate and very important in the brain diagnosis. MRI and CT allow to judge the state of the brain, however do not allow for doing it in a real-time interval. Continuous measurements allows to observe changes of SAS width in time. There is still need for others, continuous and non-invasive diagnostic methods. One of idea for supporting diagnostic methods is modeling. Mathematical modeling can bring some new ideas regarding it. Modeling SAS width can use parameters from non-invasive methods, like a transcranial Doppler ultrasonography (TCD) or blood pressure. This paper presents the idea of the method supports diagnostics of the subarachnoid space disorders based on modeling and measurements. Blood flow velocity stands for an excitation signal in the model. This signal is described by a sine function [5, 6].



Figure 1. Construction of the skull interior for healthy person and examples of pathologies causing subarachnoid space width changes [11].

MATHEMATICAL MODEL OF SUBARACHNOID SPACE WIDTH

Skull is formed as a bony cavity which it is filled by brain, blood (in veins and arteries) and cerebrospinal fluid (CSF). Cerebrospinal fluid is situated in SAS. The sum of brain, blood and cerebrospinal fluid volumes, based on the Monro-Kellie hypothesis [3], is constant, and therefore can be by the following equation:

$$V_{brain} + V_{CSF} + V_{blood} = V_{skull} = const,$$
(1)

where:

 V_{blood} — volume of blood [m³]; V_{CSF} — volume of cerebrospinal fluid [m³];

 V_{brain} – volume of brain [m³];

 V_{skull} — volume of skull [m³].

Blood, like other liquids, has its rheological and mechanical properties. Some of these properties are measured by non-invasive methods. One of them is the value of blood velocity, while the other is the blood pressure. Values of these physical quantities are expressed using simple equations. The blood velocity changes according to the heartbeat.

The pressure is the ratio of force to the area over which that force is distributed. According to the Newton's second law and the definition of density, the pressure can be calculated using the following formula:

$$p = \frac{\rho V dv}{A dt},\tag{2}$$

where:

p — pressure [Pa];

A — area of the surface on a contact [m²];

V — a volume [m³];

- v velocity of blood [m/s];
- ρ density of blood [kg/m³];

t — time [s].

If it considers the pressure as a human mean arterial pressure (MAP) in formula (2), the blood's volume can be expressed as:

$$V_{blood} = \frac{MAP \cdot A \cdot dt}{\rho \cdot d\nu}.$$
(3)

For simplicity, it is assumed that the skull is hemisphere, while the brain with blood is smaller hemisphere inside the bigger one. The volume of CSF, as well as SAS, is defined as a difference between bigger hemisphere (skull volume) and smaller hemisphere (sum of blood volume in cerebral vessels and brain):

$$V_{CSF} = V_{skull} - V_{BB},$$

$$V_{CSF} = \frac{1}{2} \cdot \frac{4}{3} \pi R^3 - \frac{1}{2} \cdot \frac{4}{3} \pi r^3 = \frac{2}{3} \pi \left(R^3 - r^3 \right),$$
(4)

where:

 V_{BB} — volume of the sum of brain and blood in cerebral vessels [m³];

R — radius of the bigger hemisphere [m];

r — radius of the smaller hemisphere (the sum of brain and cerebral blood vessels) [m]. Eqs. (1), (3) and (4) yield:

$$V_{brain} + \frac{A \cdot MAP}{\rho} \left(\frac{dv}{dt}\right)^{-1} + \frac{2}{3}\pi \left(R^3 - r^3\right) = V_{skull}.$$
(5)

The main aim of this paper is to determine the value of subarachnoid space width w_{SAS} . The SAS width is equal to the difference between bigger and smaller radius (R - r). The radius of the smaller hemisphere can be calculated from Eq. (5) as:

$$r = \sqrt[3]{\frac{3}{2\pi}} \left(V_{brain} + \frac{A \cdot MAP}{\rho} \left(\frac{dv}{dt}\right)^{-1} \right).$$
(6)

The radius of the bigger one comes out from the formula for the hemisphere. Finally, the width of SAS can be expresses as:

$$w_{SAS} = R - r = \sqrt[3]{\frac{3}{2\pi}V_{skull}} - \sqrt[3]{\frac{3}{2\pi}\left(V_{brain} + \frac{A \cdot MAP}{\rho}\left(\frac{dv}{dt}\right)^{-1}\right)}.$$
(7)

The Eq. (7) was implemented as a model of SAS width changes.

MODEL OF SUBARACHNOID SPACE WIDTH CHANGES

Eq. (7) has many variables. For this model, some of them, are assumed to be constants. It is assumed that the standard value of the systolic pressure is 120 mm Hg and the standard value of the diastolic pressure equals to 70 mm Hg. The value of mean arterial blood pressure, which for the healthy humans, is in the range from 60 up to 150 mm Hg reads [2, 4]:

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

$$MAP = \frac{120 + 2 \cdot 70}{3} \approx 90mmHg,$$
(8)

where:

MAP — mean arterial pressure [mmHg];

SP — systolic pressure (maximum) [mmHg];

DP — diastolic pressure (minimum) [mmHg].

Following [2, 4] we assume that the other constants for human body are:

- the average density of blood $\rho = 1.055 \text{g/cm}^3$;
- the middle cerebral artery (MCA) diameter has, usually, about 3 mm, so the area of the surface on a contact, calculated per 1 mm of length, is A=9.425*10⁻⁶ m²;
- mean volume of human skull 1 700 ml;
- mean volume of blood in skull for healthy human 150 ml;
- mean volume of cerebrospinal fluid for healthy human 150 ml;
- mean volume of brain for healthy human 1 400 ml.

In Fig. 2 the exemplary Doppler velocity waveform is presented. Doppler echocardiography measures changes in blood flow velocity in time. The velocity of a blood changes over time is acceleration (dv/dt).



Figure 2. Doppler velocity waveforms obtained from the middle cerebral arteries [9].

The change in blood flow velocity in time can be described by a sine function:

$$u(t) = u_0 + u_{ampli} \cdot \sin(\omega t), \qquad (9)$$

where:

 u_0 — constant value;

 u_{ampli} — value of signal amplitude;

 ω — angular frequency (constant value) equal 1.

Moreover, the range of blood velocity in the middle cerebral artery is 0.54/1.02 m/s. while the constant value of u_0 in excitation signal is equal 0.55 m/s and the value of signal amplitude is given by:

$$u_{ampli} = \frac{v_{\text{max}} - v_{\text{min}}}{2} = 0.24 m/_{s}.$$
 (10)

Velocity signal is:

$$u(t) = 0.76 + 0.24 \cdot \sin(\omega t), \qquad (11)$$

and an excitation signal for this model:

$$\frac{dv}{dt} = \omega \cdot u_{ampli} \cdot \cos\left(\omega t\right). \tag{12}$$

Eq. (7) with all the assumptions described above was implemented in Simulink. The schema from Simulink is presented in Fig. 3.

RESULTS AND DISCUSSION

The model describes the SAS width of healthy people, without chronic disease. In Fig. 4 (red line) is presented the excitation signal described by Eq. (12) is presented. In the middle cerebral artery (MCA) the blood velocity is about 0.71 m/s (in the range of 0.54 m/s to 1.02 m/s).

In Fig. 4 is presented two variables. Red line is an input signal u(t) blood flow velocity. Blue line is output signal w_{SAS} , which is related with physiological changes of blood velocity. Corresponding SAS width changes consist with the changes of blood velocity in the case where there are no pathological lesions within the skull. The proposed model properly mimics functioning of the modeled system.

CONCLUSIONS

The cerebrospinal volume changes are colligated with excitation signal u(t). These changes are consistent with the physiology of healthy human. According to [12] the subarachnoid space width is in the range from 1 mm to 4 mm. The width of subarachnoid space was measured on coronal views during ultrasonography in subsequent 5 days on group of children [12].



Figure 3. Simulink schema of SAS width model.



Figure 4. Model of blood flow velocity v [m/s] and subarachnoid space width changes w_{sas} [mm] versus time.

The subarachnoid space width depends on the rheological properties of the blood. That is the reason it is comprised into the model description. The model parameters are obtained on the basis of non-invasive diagnostic methods such transcranial Doppler ultrasound and sphygmomanometer with stethoscope, which is the great advantage of this model.

In the next step of our research we intend to extend the model and verify it on the actual Doppler measurements. Extension of the model will include pathological changes, into the model, for

instance cerebral edema, blood pressure variations or hypertension will be considered. Modeling of subarachnoid space width changes is a first step for modeling of cerebral autoregulation process.

REFERENCES

- Y-C. Tzeng and P. N. Ainslie: Blood pressure regulation IX: cerebral autoregulation under blood pressure challenges, European Journal of Applied Physiology 114 (2014), 545–559.
- [2] R. AleksandrowiczWydawnictwo Lekarskie PZWL (ed.): Anatomia kliniczna głowy i szyi, Warszawa, 2007.
- [3] A. Marmarou, K. Shulman, and J. LaMorges: Compartmental analysis of compliance and outflow resistance of the cerebrospinal fluid system, Journal of Neurosurgery 43 (1975), 523–534.
- [4] W. Mizerski: Tablice biologiczne, Wydawnictwo Adamantan, Warszawa, 2004, Wyd. IV.
- [5] M. Latka, M. Turalska, M. Glaubic-Latka, W Kolodziej, D. Latka, and B. J. West: *Phase Dynamics In cerebral autoregulation*, American Journal of Physiology, Heart and Circulation Physiology 289 (2005), H2272–H2279.
- [6] N. A. Lassen: Cerebral blood flow and oxygen consumption in man, Physiological Reviews 39 (1959), 183–238.
- [7] F. J. H. Gijsen and F. N. van de Vosse: The influence of the non-Newtonian properties of blood on the flow in large arteries: steady flow in a carotid bifurcation model, Journal of Biomechanics 32 (1999), 601–608.
- [8] N. Masoumi, D. Bastani, S. Najarian, F. Ganji, F. Farmanzad, and A. S. Seddighi: *Mathematical Modeling of CSF Pulsatile Hydrodynamics Based on Fluid-Solid Interaction*, IEEE Transactions on Biomedical Engineering 57 (2010), 1255-1263.
- [9] J. Aranyosi, T. Deli, P. Bettembuk, B. Komza, T. Kovács, O. Táörök, and Z. Tóth: *Fetal Aortic-cerebral Doppler Resistance Index Ratio: An Indicator of Physiologic Blood Flow Distribution*, Donald School Journal of Ultrasound in Obstetrics and Gynecology 3 (2009), 91–95.
- [10] B. Young, J. S. Lowe, A. Stevens, and J. W. HeathChurchill Livingstone Elsevier (ed.): Wheater's Functional Histology. A text and Colour Atlas, Chine, 2006.
- [11] P. D. McNeely, J. D. Atkinson, G. Saigal, A. M. O'Gorman, and J. P. Farmer: Subdural Hematomas In Infants With Beningn Enlargement Of The Subarachnoid Spaces Are Not Pathognomonic For Child Abuse, American Journal of Neuroradilogy 27 (2006), 1725–1728.
- [12] D. L. Armstrong, C. Bagnall, J. E. Harding, and R. L. Teele: *Measurement of the subarachnoid space by ultrasound in preterm infants*, Archives of Disease in Childhood: Fetal and Neonatal 86 (2002), F124–F126.