Local Model of Arteriovenous Malformation of the Human Brain

Ms Nadezhda Telegina, Mr Aleksandr Chupakhin and Mr Aleksandr Cherevko
Lavrentyev Institute of Hydrodynamics Siberian Branch of Russian Academy of Science, Novosibirsk, Russia
e-mail: nadezhda.telegina@gmail.com, chupakhin@hydro.nsc.ru, cherevko1@ngs.ru

Abstract. Vascular diseases of the human brain are one of the reasons of deaths and people’s incapacitation not only in Russia, but also in the world. The danger of an arteriovenous malformation (AVM) is in premature rupture of pathological vessels of an AVM which may cause haemorrhage. Long-term prognosis without surgical treatment is unfavorable. The reduced impact method of AVM treatment is embolization of a malformation which often results in complete obliteration of an AVM. Pre-surgical mathematical modeling of an arteriovenous malformation can help surgeons with an optimal sequence of the operation. During investigations, the simple mathematical model of arteriovenous malformation is developed and calculated, and stationary and non-stationary processes of its embolization are considered. Various sequences of embolization of a malformation are also considered. Calculations were done with approximate steady flow on the basis of balanced equations derived from conservation laws. Depending on pressure difference, a fistula-type AVM should be embolized at first, and then small racemose AVMs are embolized. Obtained results are in good correspondence with neurosurgical AVM practice.

1. Introduction
The human brain is the unique organ which has an expanded complex system of blood supply. Its hemodynamics is of great importance for medicine [1]. Human brain hemodynamics under normal and pathological conditions is difficult to investigate. It requires description of general physiological features of blood circulation for the human brain, special experimental data and further mathematical modeling using these data.

Outstanding complexity of the cardio-vascular system of the human brain, difficult mechanisms of brain circulation regulation and the lack of full experimental data on blood flow explain the absence of the uniform model which could describe blood flowing in brain vessels. Different geometrical and mechanical properties of vessels (i.e. arteries and veins), taking account of the influence of blood corpuscles, and the necessity of local and global blood flow modeling are main difficulties at the development of adequate hemodynamic models [2 – 6].

In recent decades there appeared new high-technology medicine equipment (e.g. various tomography equipment, intravascular surgery equipment) that leads neurosurgery to higher
level of development. This equipment allows obtaining and using information on blood flow in real time during surgical operations.

2. Abnormalities of the Vascular System of the Human Brain. Arteriovenous malformations

Vascular diseases of the human brain are one of the reasons of deaths and people’s incapacitation not only in Russia, but also in the world. An arteriovenous malformation (AVM) is a congenital pathology which occurs with the interconnection between arterial and venous bed and with the absence of the capillary area in the circulatory system. An AVM is a ‘bundle’ of small twisted vessels (up to 6 cm in diameter). These bundles do not have a capillary network, so blood is directly shunted from arterial network to the system of superficial and deep veins. Therefore, some parts of the human brain are not fed with oxygen and nutrients which are distributed with blood. The most common malformation type is a racemose AVM which is shown in Figure 1.

![Figure 1. Arteriovenous malformation](image)

1, 2 – supplying arteries; 3 – ‘bundle’ of modified vessels (‘core’ of an AVM); 4 – draining vein

The presence of abnormalities in the vascular system causes the change of both hemodynamic (i.e. flow velocity and flow pressure) and strength properties of this vascular system. The danger of an AVM is in premature rupture of pathological vessels of an AVM which causes haemorrhage. Long-term prognosis without surgical treatment is unfavorable.

At present, the process of AVM treatment is a complex staged procedure (e.g. open operation, embolization, and radiosurgery), an AVM should be completely removed without any negative neurological consequences for a patient. The reduced impact method of AVM treatment is embolization of a malformation which often results in complete obliteration of an AVM. Embolization is also used before an open operation for minimizing an existing malformation. Nowadays, information on the procedure of embolization is based on practical experience of surgeons (in other words, professional skills of a surgeon).

Pre-surgical mathematical modeling of an AVM can help surgeons with an optimal sequence of the operation (i.e. optimal sequence of the procedure of embolization), evaluation of the degree of possible risks and possible aftereffects for a patient (e.g. possible repeated haemorrhages).

3. Local Model of Arteriovenous Malformation

The local model of malformation (see Figure 2) is developed as follows: input artery (1) is divided into two vessels (2, 3) with a racemose AVM which is modeled as a ‘bundle’ of many pipes (4, 5). Then vessels gather into common vein (6). There is also a fistula-type AVM between arterial and venous areas. A fistula-type AVM is a pathologically modified great vessel shunting the most part of flowing blood. Vessels are modeled as pipes with rigid clean walls where water with density of 998 kg/m³ and viscosity of 4.2 cP flows. Pressure at the input of the AVM is set to 100 mmHg while at the output of the AVM pressure is slightly overrated to 19 mmHg which corresponds to real practice. Flow rate through the AVM is
slightly overrated and supposed to be constant. It is set to 747 cm$^3$/min. Calculations are done with the approximation of one-dimensional steady-state flow on the basis of balance equations derived from conservation laws.

At the first stage embolization of one or two racemose AVMs is performed. As a result, decrease of blood flow rate in afferent vessels (see ‘operation 1+2’ in Figure 3) is observed.

Using embolization combination for a fistula-type AVM and one smaller racemose AVM, venous blood pressure is normalized to the value of 7 mmHg (see ‘operation 1+f’ in Figure 3).

Embolization of a fistula-type AVM with one bigger racemose AVM leads to the decrease of flow rate and final normalization of venous blood pressure (see ‘operation 2+f’, ‘operation 1+2+f’ in Figure 3).

Figure 2. Local model of malformation

Figure 3 demonstrates a pressure change curve along a racemose AVM No.1 with various methods of embolization. Depending on pressure difference, a fistula-type AVM should be embolized at first, and then small racemose AVMs are embolized. Obtained results are in good correspondence with neurosurgical AVM practice.

4. Modeling for Patients with AVM

The paper presents the results of modeling of vessel blood circulation for two patients with AVM (the first patient is with AVM located in end lobe, the second patient is with AVM in temporal lobe).

For calculations, data obtained by neurosurgeons of Meshalkin State Research Institute of Circulation Pathology in Novosibirsk during surgical operations is used. Special device provides a surgeon with additional information at any moment of operation. It measures pressure and velocity inside a vessel simultaneously with endovascular operation. This device is equipped with a sensor of 0.36mm diameter which is inserted into a vessel through a catheter, while on the screen display surgeons can observe graphs of velocity and pressure changing in real time. Further, this data can be digitally processed.

Measurements are conducted in various points of a vessel located with a different distance from abnormality. The most important measurements for surgeons are those which are conducted in the same point before and after an AVM embolization.

Output data for pressure and velocity is digitally processed and used for mathematical modeling. In each case with a help of patient angiograms a graph of significant vessels is plotted. Malformation modeling is performed according to methods described in Section 3. Required values for pressure and velocity are achieved by preliminary choice of malformation parameters (i.e. parameters of ‘pipes’ in an AVM and their quantity).
Figures 4a and 4b show local graphs for significant vessels near AVM for patient 1 (end lobe AVM) and patient 2 (temporal lobe AVM). Sensor positions before embolization are marked with green circles, after embolization – red circles.

![Graphs showing local vessels near AVM for patients 1 and 2.](image)

(a) (b)

**Figure 4.** Local graph for patient 1 (end lobe AVM, figure a) and patient 2 (temporal lobe AVM, figure b)

Calculations confirm experimental data for pressure and velocity before and after an embolization. Comparison of calculated and experimentally obtained parameters is shown in Table 1.

| Diameter of vessel | p, mmHg experimentally obtained | p, mmHg calculated | v, m/s experimentally obtained | v, m/s calculated |
|--------------------|---------------------------------|--------------------|--------------------------------|-------------------|
| patient 1: before embolization, sensor – in a vessel with the following diameter, mm: |
| d=2,3mm            | 71                              | 70.91              | 0.27                           | 0.2631            |
| d=2,5mm            | 74                              | 71.56              | 0.24                           | 0.2227            |
| d=3,6mm            | 73                              | 72.91              | 0.17                           | 0.1512            |
| patient 1: after embolization, sensor – in a vessel with the following diameter, mm: |
| d=3,6mm            | 79                              | 79.95              | 0.11                           | 0.1112            |
| patient 2: before embolization, sensor – in a vessel with the following diameter, mm: |
| d=3,3mm            | 50                              | 50.33              | 0.61                           | 0.5952            |
| d=2,2mm            | –                               | 51.99              | 0.45                           | 0.4716            |
| patient 2: after embolization, sensor – in a vessel with the following diameter, mm: |
| d=3,3mm            | 50                              | 48.72              | 0.45                           | 0.4531            |
| d=2,2mm            | –                               | 48.58              | 0.57                           | 0.5588            |
| d=3,3mm            | 48                              | 51.16              | 0.72                           | 0.7015            |

**5. Conclusions**

1. AVM modeling is performed, different types of embolization are studied.
2. Results of AVM modeling for real patients are presented.
3. Obtained results are in good correspondence with neurosurgical AVM practice. This means that even such simple models help us in correct evaluation of risks for surgical intervention. For detailed prognosis, more complex models are required; spatial relationship between vessels, structure of vessel walls and blood rheology should be also taken into account.
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