Electrical Models in the Analysis of Hemodynamic Characteristics of Arteriovenous Malformations

Part 1: Baseline Measurements

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Summary

This paper describes the creation of two electric arteriovenous malformation (AVM) models where electric resistors simulate AVM vessels. The flow of electrons simulates the flow of blood. Using the models, it was possible to analyze the pressure and flow patterns in the nidus of a small, low-flow AVM and in the nidus of a large, high-flow AVM. "Normal" hemodynamic "physiologic" conditions of the two AVMs were studied, including the autoregulation of normal brain surrounding the AVMs. It is the first time that the hemodynamics within the different parts of the nidus (arterious, arteriolar, venular, venous) can be determined. The models will be utilized to assess AVM hemodynamic changes when different parameters (systemic blood pressure, embolization of arterial feeders, embolization of nidus, embolization of draining veins, surgical by-pass of the AVM) are changed. This will be the subject of part 2 of this paper.

Introduction

In recent years the management of patients harboring brain arteriovenous malformations (AVMs) has undergone noteworthy improvements. Once treatable by open surgery only, AVMs may now be treated by surgery, radiosurgery and embolization, often in combination. Each of these techniques, however, pre-

sents limitations. Larger lesions, lesions located in or near vital areas of the brain, and deep-seated AVMs still constitute an important challenge for any form of treatment.

The surgical approach requires a craniotomy, brain tissue manipulation and retraction. The risk of profuse intraoperative hemorrhage and the risk of surgical damage of vessels or brain tissue are other factors that add to an already difficult operative act.

Radiosurgery seems more indicated for non hemorrhagic lesions, is less effective in larger lesions and carries the risk of normal tissue radionecrosis.

Endovascular embolization allows complete exclusion from the circulation in only 10%-20% of lesions. The remaining 80%-90% need additional (surgical or radiosurgical) treatment. In spite of this, the endovascular technique would seem to be the most logical in that it implies an approach of blood vessels only in a disease of the blood vessels only.

It is believed that the results attainable via the endovascular route have not significantly evolved in the last decade, due also to the lack of a reliable AVM model. The lack of data on what happens hemodynamically in the core or "nidus" of an AVM probably prevented the development of new concepts, modalities, and techniques in both surgical and endovascular settings. Attempts to reproduce in vivo the complex hemodynamics of AVMs have not

provided enough information on the hemodynamic stress acting upon the AVM core.

Intra-operative or endovascular measurement of the pressure in the feeding arteries and in the draining veins have helped us to understand the pressure gradients across the AVM. The core of a brain AVM, however, is composed of channels (50 to 500 microns in diameter)⁵ that are too small to be accessible for direct measurements.

Blood flow measurements in patients proved very useful in determining the blood flow through the AVM as compared to the normal blood flow through an intact brain hemisphere. These measurements also helped determine that in most cases the autoregulation of normal brain vessels is maintained, while there is no autoregulation of the vessels involved in the AVM⁶.

Due to the current limited information on the hemodynamics of the brain AVM nidus, an electrical model was created.

As to the rationale of this paper, one must derive from physics that

a) the flow of electrons (current) of an electric circuit can be compared to the flow of a liquid in a conduit, b) the voltage of an electric current can be compared to the pressure of a liquid, and c) the electrical resistance of a conductor to the flow of electrons can be compared to the resistance to advancement of a fluid in a conduit. Two electrical models were constructed to simulate flow, pressure, and resistance through brain AVMs, including feeding arteries, nidus, and draining veins.

Material and Methods

Blood pressure, blood flow, and other possible forms of mechanical energy (i.e. shear stress, pulsatile blood flow) are responsible for AVMs rupture. It is likely that these mechanical energies act upon the weak points of the AVM structure (i.e. the "venular" part of the nidus) determining rupture and hemorrhage⁶. The goal of this study is to determine the stress on the AVM nidus in various conditions of pressure and flow using an electrical model.

In vivo pressure and flow measurements from previous studies were first utilized to determine the values of voltage (pressure) and current (flow) to be applied across AVM electrical models. The pressure values, in mmHg, were converted to voltage values expressed in Volts. The flow

values (ml/min) were converted to electrical current values (milliamperes (mA)).

Electric resistors of the model correspond to the resistance that vascular conduits (arteries, nidus, and veins) oppose ("resist") to the blood flow.

Clinical pressure measurements

Nornes et Al³ measured the arterial and venous pressure in four non hemorrhagic AVMs. One of the major feeding arteries and a major draining vein were selected for intraoperative measurements. At the same time they also measured the systemic arterial pressure and the central venous pressure.

In one small AVM the mean systemic arterial pressure (SAP) was 100 mmHg, the mean feeder artery pressure (FAP) was 70 mmHg, and the mean draining vein pressure (DVP) was 18 mmHg. In one large AVM the mean SAP was 105 mmHg, the mean FAP was 40 mmHg, and the mean DVP was 15 mmHg. As expected, a progressive drop in pressure was observed throughout the AVM system.

Clinical flow measurements

Nornes et Al³ calculated the flow through the same four non hemorrhagic AVMs. These flow measurements ranged from 105 ml/min to 550 ml/min. The minimum (105 ml/min) and the maximum (550 ml/min) values were adopted to construct a low-flow AVM electrical model and a high-flow AVM electrical model, respectively.

Flow through the brain

It is estimated that the blood flow through a brain hemisphere is approximately 350 ml/min, on average⁶. The mechanism of autoregulation maintains this flow constant, within a certain range of the systemic blood pressure changes. This mechanism acts by changing the resistance of the arterioles to flow. The blood flow is autoregulated extremely well between the mean pressure limits of 60 and 140 mm/Hg.

The AVM feeding arteries and arterioles, on the contrary, do not respond to changes in systemic arterial pressure⁶. Therefore the AVM flow is not autoregulated, and varies together with the systemic blood pressure.

The AVM model

Nornes³, Miyasaka², Handa¹, and Ornstein⁴ pressure measurements, Nornes flow measure-

Figure 1 Electrical model of a small, low-flow AVM. The resistors simulate vessels (the resistance of vessels to the flow of blood). Their values are in ohms (Ω). Arrows point to the AVM nidus.

"Heart": battery simulating the human heart.

ICA: internal carotid artery.

N1: pressure at the arterial part of the nidus.

N2: pressure at the arteriolar part of the nidus.

N3: pressure at the venular part of the nidus.

N4: pressure at the venous part of the nidus.

SSS: superior sagittal sinus pressure.

TS: transverse sinus(es).

JV: jugular vein(s).

Feeders: arterial feeders of the AVM

mA: milliamperes (flow in ml/min)

V: volts (pressure in mm/Hg)

ments, and the value of the blood flow through a brain hemisphere were utilized to design and construct the electric model of two AVMs, one simulating a small, low-flow AVM and the other simulating a large, high-flow AVM. Both models are composed of a chain of resistors in series and in parallel (Figure 1,2). Each vascular segment is simulated by a resistor, having a certain voltage difference (pressure drop) and a certain current (flow) across it. The value of each resistor was calculated according to Ohm's law: knowing the values of current (flow) and voltage (pressure), it is possible to calculate the value of the resistance ($R=V/I$; where R is the resistance in Ohms, V is the voltage in Volts, I is the current in Amperes) (Figure 1,2).

In both models the brain hemisphere and the AVM are two systems in parallel that share the same inlet (internal carotid artery [ICA]) and the same outlet (jugular veins [JV]) (Figure 1,2).

In the models all electric measurements were obtained using the following software: Electronics Workbench (Electronics Workbench Canada, 111 Peter Street, Suite 801, Toronto, Ontario M5V 2H1, www.electronicworkbench.com), which allows a computer simulation of electronic circuits.

Definitions

Voltage (in Volts) corresponds to pressure (in mmHg). In the models, 100 Volts correspond to 100 mm Hg.

Current (in milliamperes (mA)) corresponds to flow (in ml/min). In the models, 100 mA correspond to 100 ml/min.

Whenever voltage or current are mentioned in this paper, they will correspond to pressure or flow, respectively.

Electrical resistance (in Ohms) corresponds to the resistance of a vessel to blood flow.

Figure 2 Electrical model of a large, high-flow AVM. The resistors simulate vessels (the resistance of vessels to the flow of blood). Their values are in ohms (Ω). Arrows point at the AVM nidus.

"Heart", ICA, N1, N4, SSS, TS, JV, Feeders, mA, V: same as fig.1.

N2f: pressure at the arteriolar part of the fistulous nidus.

N3f: pressure at the venular part of the fistulous nidus.

N2p: pressure at the arteriolar part of the plexiform nidus.

N3p: pressure at the venular part of the plexiform nidus.

Baseline settings

Low-flow, small AVM:

The model of the low-flow AVM (Figure 1) is powered with a 100 Volts direct electric current, i.e. the mean systemic pressure (100 mmHg) measured by Nornes et Al in the low-flow AVM. The total value of electrical resistance of the model (ICA + AVM and brain + JV) was first calculated to have an electrical current of 455 mA (455 ml/min) which is the flow calculated by Nornes³ in low-flow AVMs (105 ml/min) plus the flow through the brain hemisphere (350 ml/min) (Figure 1). The resistance of the ICA was set at 11 ohms to allow a pressure (volts) drop of 5 mmHg (5 volts) and therefore a pressure (voltage) of 95 mmHg (95 volts) at the circle of Willis (Figure 1).

The resistance of AVM and brain was calculated at 191 ohms, and the resistance of the

transverse sinuses (TS) plus JV at 18 ohms to have a voltage/pressure of 8 Volts (8 mmHg) at the superior sagittal sinus (SSS) (Figure 1). After these initial settings, it was necessary to simulate the complex anatomy of the AVM itself, including feeding arteries, core (or "nidus"), and draining vein.

It was arbitrarily decided that the low-flow AVM model would have two feeding arteries (resistors) of different caliber (resistance). The value of these two resistors is such to have the pressure (voltage) value of 70 mmHg (70 volts) reported by Nornes³ at the distal end of the feeders near the AVM nidus (Figure 1).

It was arbitrarily decided to compose the nidus of the low-flow AVM of four channels (resistors) in parallel. Each channel has an identical total resistance to simulate a plexiform AVM nidus, and is formed by three resistors in series (Figure 2). The overall value of these re-

sistors was such to have the pressure (voltage) value of 8 mmHg (8 volts) as reported by Miyasaka et Al² at the proximal end of the (single) vein near the AVM nidus (Figure 1).

High-flow, large AVM:

The model of the high-flow AVM (Figure 2) is powered with a 105 Volts direct current, which is the mean systemic pressure measured by Nornes³ in the high-flow AVM (105 mmHg). The total value of electrical resistance of the model (ICA + AVM and brain + JV) was calculated in order to have an electrical current of 900 mA which is the flow calculated by Nornes in the high-flow AVM (550 ml/min) plus the brain flow (350 ml/min) (Figure 2).

The resistance of ICA was set at 50 ohms to allow a pressure/voltage drop of 45 mmHg (45 volts) at the circle of Willis (Figure 2). The resistance of the AVM and the brain was calculated at 58 ohms, and the resistance of TS plus JV at 9 ohms to have a pressure/voltage of 8 mmHg (8 volts) at the SSS (Figure 2).

The high-flow AVM model has four arterial feeders (resistors), two of them having a smaller caliber (resistance) and two having a larger caliber (resistance). The overall value of these resistors in parallel was set at 36 ohms to have a pressure (voltage) value of 40 mmHg (40 volts) as reported by Nornes³ at the distal end of the feeders near the AVM nidus (Figure 2).

The nidus of the high-flow AVM model is composed of five channels (resistors) in parallel. Four of these channels (resistors) have identical resistance to simulate the plexiform part of a high-flow AVM nidus, and each channel is formed by three resistors in series. The remaining (fifth) channel has a lower resistance than the other four, to simulate the fistulous part of the high-flow nidus. The overall value of these resistors was set at 45 ohms to have the pressure (voltage) value of 8 mmHg (8 volts) as reported by Miyasaka² at the proximal end of the veins near the AVM nidus (Figure 2).

Veins

The number of draining veins was determined by Miyasaka², who assessed that a small AVM has an average of 1.2 draining veins, whereas a large AVM has 4.1 draining veins, on average. Therefore, the low-flow AVM model has been constructed with one draining vein

(resistor) (Figure 1), whereas the high-flow AVM has three draining veins (resistors) of two different calibers (resistance values) (Figure 2).

Features in common

Each channel of the nidus is composed of three resistors in series in both high and low-flow models to simulate the cascade of pressure throughout the AVM nidus from the arteriolar to the venular side (Figure 1, 2).

Both models were powered with direct electrical current to simulate the mean arterial pressure. The positive electrode was connected to the proximal "arterial" end, and the negative to the "venous" end. The "heart" of the model is a constant voltage power generator. The features of the generator make it similar to the real heart, which, within certain limits, continues to deliver the same pressure regardless of the flow demand.

Electrical/hemodynamic baseline values

As already mentioned (vide supra), the overall baseline parameters of both models were set up according to data available in the literature. The next step was to learn from the models data not attainable by in vivo measurements. This was accomplished by a) Measuring the baseline values of intranidal voltages (pressures) and currents (flows); and b) Assessing how induced changes in the "hemodynamics" of the AVM affect the intranidal voltages (pressures) and currents (flows). The latter assessments will be described in Part 2 of this paper.

Hemodynamic observations-results

It is possible to observe some "physiological," and hemodynamic features of these two baseline configurations.

1) The resistance of the ICA varies between low flow and high flow AVMs.

The higher resistance in high flow conditions is explained by the much higher drop in pressure across the artery. In fact, the flow through a blood vessel is determined by two factors: 1) The pressure difference between the two ends of the vessel, and 2) the impediment to blood flow through the vessel, which is the resistance. The blood flow, therefore, is directly proportional to the pressure gradient but inversely proportional to the resistance.

2) In the large AVM, the resistance of the

brain vessels is less than in the small AVM (149 ohms versus 249 ohms). This is explained by the “steal” effect of the large, high flow AVM that induces the brain arterioles to dilate in order to maintain adequate flow through the brain (350 ml/min).

3) The pressure regimen through the small AVM nidus is higher than in the large AVM. This is much more pronounced in the arterial side of the nidus (70 mm/Hg versus 40 mm/Hg). This higher pressure regimen is due to the higher resistance of the nidus of the small AVM. However, the distribution of the pressure regimen is such that the venous collector of the nidus is subject to almost the same pressure in large and small AVMs (15 mm/Hg versus 18 mm/Hg). This is noteworthy as the venous part of the nidus is thought to be the most fragile and prone to rupture⁶.

4) As to the fistulous part of the large AVM nidus, the blood flow is very high (365 ml/min). In the entire plexiform part of the nidus the total blood flow is 190 ml/min. No comparison can be made (and no conclusions can be drawn) between the blood pressure of the fistulous and plexiform parts of the large AVM. This is due to the arbitrary values given to the resistors (vide supra).

5) The resistance of the arterial feeders has to be calculated because the feeders are in parallel. In the small AVM the resistance is 240 ohms, while in the large AVM the resistance is 36 ohms. This huge difference is explained by the much larger cross-section area of the feeders in large AVMs.

6) The pressure in the “arteriolar” part of the nidus of the small AVM is much higher (44 mm/Hg) than in the large AVM (25.5 mm/Hg).

7) The “venular” part of the nidus in the small AVM is subject to a pressure of 24.5 mm/Hg and a flow of 26 ml/min, while in the large AVM the venular part is subject to a pressure of 18.5 mm/Hg and a flow of 48 ml/min. This is due to the lower resistance to the blood flow of the plexiform channels of the large AVM.

8) The resistance of the draining veins follows the same pattern as the arterial feeders: in the small AVM the total resistance is 95 ohms, while in the large AVM it is 13 ohms. This huge difference is explained by the larger cross-section area of the draining veins of the large AVM.

9) The blood pressure at the level of the torcula is identical in both large and small AVMs.

Discussion

Data on pressure and flow through arterial feeders and draining veins of human brain AVMs are available in the literature^{1,2,3,4}. These data have been utilized to design and construct electrical models of true AVMs. Arteriolar and venular (nidus) pressure and flow cannot be directly measured due to the smallness of these channels. This limiting factor has not permitted an accurate analysis of the hemodynamics of the nidus. The venular part of the nidus of brain AVMs is the weak site, responsible for rupture and hemorrhage. Animal models have been constructed but suffer from the same limitations as human AVMs: pressure and flow in the nidus cannot be measured. In both human cases and animal models, the mechanical forces that act in the various components of the nidus remain unknown.

These electrical models constitute an attempt to simulate the complex hemodynamics in the nidus of brain AVMs. These models represent, however, a simplified version of the real AVM components. In spite of this limitation, it seems reasonable to infer that the behavior of pressure and flow in real AVMs is similar to what is observed in the electrical models. In particular, the models allowed: 1) the quantification of the cascade of pressure in the arterial, nidal and venous components, and 2) the flow in the components of the nidus.

We must take into consideration that in order to obtain a more accurate electrical model, the following factors could have been introduced: blood viscosity, shear stress in the nidus, pulsatile blood flow, distensibility-compliance of the AVM vessels, possible multicompartmentalization of the nidus, normal arteries arising from the arterial feeders, and normal vein tributaries of the draining veins. It would have been technically possible to add all these parameters, but this would have implied a much greater complexity of the models. In fact, it was decided to obtain models that are readily understandable and immediately useful to researchers. Moreover, it would have been possible to increase the number of the channels of the nidus from 4-5 to 10-20. But, again, it was decided to keep the models as simple as possible.

Resistors were utilized to simulate the vessels of the brain and of the brain AVM. In an electric circuit, in fact, a resistor can be compared to a vessel in that 1) a resistor opposes

resistance to the flow of electrons much like a vessel opposes resistance to the flow of blood, and 2) it is the difference in pressure (voltage) between two ends of a vessel (resistor) that determines the rate of flow (current) and not the absolute pressure in the vessel.

In the nidus the resistance progressively diminishes because, going toward the venular part, there is a progressive increase in the diameter of the vessels. Yamada⁵ determined that the arterioles have a diameter of 50-200 microns, while the venules have a diameter of 0.5-2 mm. This has been simulated in the electrical models: in the nidus there is a progressive decrease of the value of the resistors.

The autoregulation of the normal brain surrounding and in parallel to the malformation was calculated and taken into account. Normal blood flow through a brain hemisphere is 350 ml/min (range 300-450 ml/min). This flow is autoregulated: pre-capillary arterioles dilate or constrict to maintain this flow. This is demonstrated in the models by the difference in the values of the resistor that simulates normal

brain between low-flow and high-flow AVM. Normal brain autoregulation is impaired when the arterioles are at their maximum dilatation. This occurs when a large brain AVM "steals" blood away from the brain and the arterioles dilate at their maximum (lower resistance) in order to maintain adequate flow through the brain, or when the systemic arterial mean pressure (SAP) is below 60 mm/Hg (mean SAP is calculated by adding 2/3 of the diastolic pressure to 1/3 of the systolic pressure).

Conclusions

These AVM electrical models were created to obtain more information on the "physiology" of the AVM nidus for the first time. The main purpose of this study, however, was to induce "hemodynamic" changes (systemic blood pressure, embolization of arterial feeders, embolization of nidus, embolization of draining veins, surgical by-pass of the AVM) and assess their effect in the nidus. This will be the subject of part 2 of this paper.

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