

Simulation and Quantification of Tortuous Vessels– Cerebal AVM

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ABSTRACT

A Cerebral Arteriovenous Malformation is an abnormal connection between arteries and veins. In the healthy cases, the blood flows from arteries to veins through capillary bed. But patient having AVM, the capillary bed will be absent. The veins cannot handle the pressure of blood flowing from arteries to veins and it ruptures. The success of treatment by embolization in interventional Neuroradiolgy is highly dependent on the accuracy of the vessels visualization and quantification. In this paper, we have done simulation, quantification of tortuous vessels, which is a part of NIDUS using 3DRA for cerebral patients, so that the doctors can analyze and decide the mode of treatment.

Keywords

AVM, Tortuous, 3DRA, Segmentation, Quantification



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1. INTRODUCTION

Intracranial Arteriovenous malformations (AVM) constitute usually congenital vascular anomalies of the brain. AVMs are composed of complex connections between the arteries and veins that lack an intervening capillary bed. A Cerebral AVM (CAVM) is a set of abnormal vessels comprising feeding arteries; draining veins and a collection of arterialized veins called the Nidus.

AVM's consist of networks of large caliber vessels yielding an alternate pathway for blood flow with high flow/low pressure characteristics. As a result AVM's steals the blood supply away from capillary beds juxtaposed in parallel with the AVM. In the brain, such shunts reduce the blood supply to normal neuronal tissue perfused by the parent vessel which is shared with the AVM, resulting in varying degrees of cerebral ischemia and central nervous system breakdown. The arteries have a deficient muscularis layer. The draining veins often are dilated and tortuous due to the high velocity of blood flow through the fistulae.

The AVM"nidus", without feeders or veins, is measured in currently used predictive models and grading systems . The Spetzler-Martin AVM classification

stratifies the malformation according to its largest diameter, i.e. <3, 3–6 and >6 cm. The middle group may for example contain lesions with diameters of in between 1x 1x3 cm up to 6x6x6 cm, corresponding to volumes between 0, 5 cm3 and 113 cm3. In a radio surgical outcome model or for volume comparison a more exact measurement is necessary. The Gamma Knife® radio surgery outcome model defines AVM volume as being within the prescription isodose line, not the same as the "true" Nidus, but a reasonable approximation [1].





The literature shows the various models using Mechanical, Electro-Mechanical, and few Electrical Models for AVM "NIDUS". AVM Nidus volume is a predictor of the outcome of AVM surgery [2] and radio surgery. The impact of AVM size upon the results of endovascular treatment seems to be less clear. A recently published study [3] based on a material of 2262 AVM patients showed that the annual risk for presenting with hemorrhage increases with the volume of the malformation. Thus volume data should influence therapeutic decisions in the individual patient, be used in the assessment of the efficacy of any therapy and when comparing patient outcome. It is essential to precisely locate the position of vessels and also to track the vessels entering and leaving the malformation, as well as their radii

and bending angles before treatment. The problem statement is very complex as the NIDUS is very complex structure which varies from every patient and imaging modalities also used for this purpose. Many imaging techniques have been developed for this purpose. Conventional catheter angiography (CCA) is used at the end of follow-up to confirm complete occlusion [1], while for intermediate controls Magnetic resonance angiography (MRA) with time of flight (TOF) or phase contrast techniques or computed tomography angiography (CTA) are usually used. Digital subtraction angiography (DSA) with 3-D rotational angiography (3DRA) remains the standard technique, providing substantial additional information on CAVM angioarchitectural . In this paper, the tortuous vessel segmentation are performed with unique methodology , which is implemented in MATLAB.

2.0 METHODOLOGY

2.1 Segmentation & Modeling:

The following flowchart shows the methodology to segment and Model the 3DRA/DSA image acquisition, which is obtained from Philips Allure Unit. The methodology and flowchart to implement tortous vessel modeling is as follows:





Fig 2.Segmentation and Modeling Flowchart

- The input 3DRA image is used as the input volume of the Cerebral AVM (CAVM).
- 3D ROI is drawn for the NIDUS Portion, automatically propagated to all the slices, by applying interpolation technique.
- Preprocessing techniques are applied to the ROI by performing enhanced contrast, smoothing algorithm and edge detection algorithm based on intensity.
- The filtering is applied to remove the noise, we have used various filtering techniques –Mean, Median, Convolve and Gaussian Blur, FFT.
- Reconstruction of the filtered output and rendering of output in volume and surface rendering.
- The segmentation using OTSU is applied to the reconstructed image and segment the vessels.
- The path of the segmentation is tracked using centerline, which is the existing feature of the Philips Software for the DSA/3DRA analysis package. Using centerline, we able to find the various path. Hence path navigation software helps to find the various path inside the tortous vessel. For the segmented the tortous path, we have defined the model to simulate tortous structures [4-5].

2.2 Modeling:

The modeling for the specific segmented tortuous path is based on windkessel model analysis using electrical networks-R,L,C. The below image 3.0 shows the complex tortous path structure used for the modeling [6-8]:



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Figure 3 Tortuous Vessels indicating path

From the above Tortuous vessel, we derive the path 1-2-3-4-5, which is the initial vessel tortuous loop for analysis. Each part of the path is modeled using RLC as shown in figure 4.0



2.3 Experimental Results and Statistics:

The experimental analysis using RLC for each part of the path using windkessel model and simulated using electrical signals and pressure , flow rate is calculated at various nodes of the network. The below mentioned some of the quantification for the tortuous vessel is calculated using following parameters [9-10]:

Pearson's correlation coefficient- The population correlation coefficient ρX , Y between two random variables X and Y with expected values μX and μY and standard deviations σX and σY is defined as:

$$\rho_{X,Y} = \operatorname{corr}(X,Y) = \frac{\operatorname{cov}(X,Y)}{\sigma_X \sigma_Y} = \frac{E[(X - \mu_X)(Y - \mu_Y)]}{\sigma_X \sigma_Y}$$

where *E* is the expected value, *cov* means covariance and, *corr* a widely used alternative notation for the correlation coefficient.

t-test - The *p*-value is the probability, under the null hypothesis, of observing a value as extreme or more extreme of the test statistics. Data are expressed as the mean ± SD. Paired groups of data were compared for significance by an analysis of variance for a one-factor experiment with repeated measurements. p < .05 was considered indicative of a significant difference.



INPUT Pressure (interms of Voltages)	node 1 voltage diameter 1cm R L + R R=2.314 ohms L=2.098 H	node 2 voltage diameter 0.5cm RL series R=23 Kohms L = 1mH	node 3 voltage diameter 1cm R L + R R=2.314 ohms L=2.098 H	node 4 voltage diameter 1cm R L + R R=2.314 ohms L=2.098 H
0.65	0.57	0.51	0.275	0.24
0.75	0.482	0.429	0.235	0.215
0.85	0.7	0.384	0.38	0.325
0.95	0.75	0.39	0.38	0.34
1.1	0.735	0.408	0.407	0.356
1.2	0.9	0.507	0.5	0.39
1.3	1	0.55	0.56	0.5

The detailed quantification analysis for various node is as follows:

Statistical Analysis	Node 1	Node 2	Node3	Node4
Count	6	6	6	6
Minimum	3.14	3.14	3.1409	3.1400
Maximum	3.18	3.18	3.2000	3.1900
Sum	18.8843	18.8843	18.9358	18.9014
Mean	3.147383333	3.147383333	3.1560	3.1502
Range	0.04	0.04	0.0591	0.0500
Interquartile Range	0.0016	0.0016	0.0171	0.0062
Std Dev (Sample)	0.015994176	0.015994176	0.0225	0.0196
Std Dev (Population)	0.014600618	0.014600618	0.0206	0.0179
Variance (Sample)	0.000255814	0.000255814	0.0005	0.0004
Variance (Population)	0.000213178	0.000213178	0.0004	0.0003
Sum of Squares	59.43741015	59.43741015	59.7633	59.5457
Mean Squared Error	9.906235025	9.906235025	9.9605	9.9243
Root Mean	3.147417199	3.147417199	3.1560	3.1503



Squared Error				
Mean Absolute Deviation	0.010872222	0.010872222	0.0157	0.0133
Skewness	2.43891518	2.43891518	2.0296	2.3673
Std Error of Skewness	0.845154255	0.845154255	0.8452	0.8452
Excess Kurtosis	5.959851964	5.959851964	4.2244	5.6628
Std Error of Excess Kurtosis	1.74077656	1.74077656	1.7408	1.7408
Jacque- Bera Test Stat	14.82826611	14.82826611	8.5808	13.6209

3.0 RESULTS & DISCUSSION

The results shows the analysis of tortuous vessels for any organ can be modelled using Windkessel model with the help of RLC networks. The segmentation and path formation from complexity of tortuous vessel is still a challenging work and our results shows that tortuous vessels complex are split down in to simpler path for analysis in Cerebral AVM cases. The network analysis can be made more robust by considering various tortuous vessel analysis using bending angle, bifurcation. The analysis for more accurate results are in progress, which will be published in near future.

4.0 CONCLUSION

The simulation and quantification of tortuous vessels in cerebral AVM shows the base analysis for the model of NIDUS using Windkessel Model . The tortuous analysis is applicable for any vessel complex structure in the human body.

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REFERENCES

[1] Avman N, Bering EA, Jr (1961) A plastic model for the study of pressure changes in the circle of Willis and major cerebral arteries following arterial occlusion. J Neurosurg 21:361-365.

[2] Barnett GH, Little JR, Ebrahim ZY, Jones SC, Friel HT (1 987) Cerebral circulation during arteriovenous malformation operation. Neurosurgery 20:836-842.

[3] Bevan JA (1991) Pressure and f1ow: are these the true vascular neuroeffectors? [Review]. Blood Vessels 28:1 64-172.

[4] Bevan JA. Laher I (1991) Pressure and t1ow-dependent vascular tone [Review]. FASEB J 5; 2267-2273.

[5] Brown AP, Spetzler RF (1 997) Intracranial arteriovenous malformations: cerebrovascular hemodynamics. In: Cerebrovascular Disease.

[6] Cas sot F. Vergeur V, Bossuet P, Hillen B, Zagzoule M, Marc-Vergnes JP (1995) Effects of anterior communicating artery diameter on cerebral hemodynamics in internal carotid artery disease: a model study. Circulation 92: 3 1 22-3 1 31.

[7] Duckwiler G, Dion J, Vinuela F, Jabour B, Martin N, Bentson J (1 990) Intravascular microcatheter pressure monitoring: experimental results and early clinical evaluation. Am J Neuroradiol 1 1 : 1 69- 1 75.

[8] Fogarty-Mack P. Pile-Spellman J, Hacein-Bey L, Osipov A, DeMeritt J, Jackson EC, Young WL (1996a) The effect of Arteriovenous malformations on the distribution of intracerebral arterial pressures. Am J Neuroradiol 1 7 : 1 443- 1 449.

[9] Fogarty-Mack p. Pile-Spellman J, Hacein-Bey L, Ostapkovich N, Joshi S, Vulliemoz Y, Young WL (1996b) Superselective intraarterial papaverine administration: effect on regional cerebral blood flow in patients with arteriovenous malformations. J Neurosurg 8 5 : 395-





[10] . Hacein-Bey L, Nour R, Pile-Spellman J, Van Heertum R, Esser PD, Young WL (1 995) Adaptive changes in autoregulation to chronic cerebral hypotension with arteriovenous malformations: An acetazolamide- enhanced single-photon emission CT study. Am J Neuroradiol 1 6: 1 865- 1 874.

[11] Hademenos GJ, Massoud TF (1 996) Risk of intracranial Arteriovenous malformation rupture due to venous drainage impairment. A theoretical analysis. Stroke 27: 1 072- 1 08 3

[12] Hademenos G J, Massoud TF, Vinuela F (1 996) A biomathematical model of intracranial arteriovenous malformations based on electrical network analysis: theory and hemodynamics. Neurosurgery 38: 1 005-1 0 1 5.

Author' biography with Photo

Y.Kiran Kumar:- is currently working in Philips Electronics india ltd and doing is research in Manipal University on Cerebral Arteriovenous Malformation. His area of interest are simulation and modeling, image processing.

