The Developmental Anatomy and Fluid Dynamics of Cerebral AVMs

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Abstract
The anatomy of the Arterio-venous malformations is described generally with reference to adult anatomy. These malformations occur due to developmental anomalies in the embryological stage. It is possible to describe the anatomy with reference to embryonic segmental origins.

The flow pattern in an AVM is unique because two fluid systems of different pressures and flows are interconnected. There are numerous published reports on the flow characteristics of AVMs based on the assumption that the equations for flow in a linear tube are also applicable to AVMs. We have demonstrated by experimental evidence and computational fluid dynamic studies that the flow is extremely complex. We hypothesize that AVMs in the brain are abnormal communications between definite embryonic arteries and veins.

ABBREVIATIONS
- AVM: Arterio-Venous Malformations
- CFD: Computational Fluid Dynamics

INTRODUCTION
It is a widespread practice to describe the anatomy of the arterio-venous malformations (AVM) with reference to adult anatomy. The arterial feeders and the venous sinuses into which the arterialized blood drains are easily identifiable. However, the arterialized venous channels extending between the arteries and the Dural sinuses are simply described as “abnormal venous channels”. These AVMs occur due to developmental anomalies in the embryological stage. It is possible to identify the normal embryonic veins in the early stages of development. These vessels normally disappear during development, as they become atretic due to diversion of blood away from them. In the presence of an AVM, these vessels persist because of the abnormal flow created by the fistula. (Figure 1). This paper attempts to demonstrate the method of identification of these segmental vessels in the adult. It is possible to deduce the anatomy of the persistent embryonic veins by following them through the various stages of the development of the embryo [1].

The flow pattern in an AVM is unique because two fluid systems of different pressures and flows are interconnected. There are numerous published reports on the flow characteristics of AVMs based on the assumption that the equations for flow in a linear tube are also applicable to AVMs. We have demonstrated by experimental evidence and computational fluid dynamic studies that the flow is extremely complex [2].

We hypothesize that AVMs in the brain are abnormal communications between definite embryonic arteries and veins. This study aims to demonstrate that accidental rupture occurs between two definite embryonic segmental pial vessels due to defects in the vessel walls (Figure 2).
METHODOLOGY

The process of identification of the main arterial component of the malformation

A preliminary review of all the angiograms of the patients diagnosed with AVMs was conducted. Of the 54 cases reviewed, 44 cases were selected for a more detailed study. The remaining 10 cases were excluded as the clarity of the radiographs was unsatisfactory for detailed examination. Each of the cases studied contained approximately twenty films.

To begin with, a series of tracings of all the films were made to define the source of the main arterial component feeding the vascular malformation and the course the feeder had taken to reach the site of the lesion. These cases were then grouped according to the main arterial feeder (see Table 1).

Identification of the angiomatous elements of the malformation

Each of the radiographs was traced to determine the size and the extent of the arterio-capillary tangle intervening between the feeding artery and the draining vein. The variation in the appearance and site of these angiomatous elements are summarized in Table (2).

The identification of the venous elements of the malformation

Attention was directed to the venous channels leaving the site of the malformation. Tracings of the x-rays were made to define the course taken by these vessels through the brain to reach the dural venous sinuses. The dural sinus into which each of these vessels drained was also recorded.

At this stage, it became apparent that many of these vessels could not be labelled in normal adult anatomical terms, although there seemed to be a repetitive pattern in many of them.

Regrouping of cases according to the similarity of venous pattern

Cases found to have similar venous patterns were grouped together. Most of them could be put into one of four groups, but about eight cases defied this categorization so they were set aside. The main arterial feeders of the malformation in each of these venous groupings were defined.

Identification and matching of the abnormal vascular channels with the normal embryonic vascular channels

The classical work on the development of the blood vessels in the embryo by Padget [3-7] was thoroughly examined. In our study, it appeared that many of the early embryonic channels identified by Padget, which normally disappear in the process of development, resembled some of the abnormal vascular channels seen in the tracings of the radiographs. It was possible to match these embryonic channels with the abnormal vessels in the angiograms in a considerable proportion of cases (see Table 1).

Table 1: Classification of the Arterio-Venous Malformations based on the pattern of the Venous drainage [C4].

<table>
<thead>
<tr>
<th>Arterial Feeder</th>
<th>Course and Mode of Termination of the Abnormal Veins</th>
<th>Case Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Backwards and Medially</td>
<td>Int. Cr. V.</td>
<td></td>
</tr>
<tr>
<td>Upwards, Medially and Backwards</td>
<td>S.S.S.</td>
<td></td>
</tr>
<tr>
<td>Upwards, Medially and Backwards</td>
<td>Gr. C. V.</td>
<td></td>
</tr>
<tr>
<td>Downward, Medially and Backwards</td>
<td>Trans. S.</td>
<td></td>
</tr>
<tr>
<td>Medially and Downwards</td>
<td>Gr. C. V.</td>
<td></td>
</tr>
<tr>
<td>Laterally and Downwards</td>
<td>Trans. S.</td>
<td></td>
</tr>
<tr>
<td>Medially and Upwards</td>
<td>Gr. C. V.</td>
<td></td>
</tr>
<tr>
<td>Laterally and Backwards</td>
<td>Trans. S.</td>
<td></td>
</tr>
<tr>
<td>Medially and Backwards</td>
<td>Trans. S.</td>
<td></td>
</tr>
<tr>
<td>Not Known</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Grouping the Cerebral Arteries according to their situation in the embryo [C5].

<table>
<thead>
<tr>
<th>The Artery</th>
<th>Embryonic Segment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior Cerebral Artery</td>
<td>Telencephalon</td>
</tr>
<tr>
<td>Middle Cerebral Artery</td>
<td></td>
</tr>
<tr>
<td>Anterior Choroidal Artery</td>
<td>Diencephalon</td>
</tr>
<tr>
<td>Posterior Choroidal Artery</td>
<td></td>
</tr>
<tr>
<td>Posterior Cerebral Artery</td>
<td></td>
</tr>
<tr>
<td>Mesencephalic Artery</td>
<td>Mesencephalon</td>
</tr>
<tr>
<td>Superior Cerebellar Artery</td>
<td>Metencephalon</td>
</tr>
<tr>
<td>Anterior Inferior</td>
<td></td>
</tr>
<tr>
<td>Cerebellar Artery</td>
<td>Myelencephalon</td>
</tr>
<tr>
<td>Posterior Inferior</td>
<td></td>
</tr>
<tr>
<td>Cerebellar Artery</td>
<td></td>
</tr>
</tbody>
</table>
Regrouping of cases of arteriovenous malformation according to the arterial feeder but with reference to the sources of these arteries during embryonic development

1. The developmental process of the arterial system was again studied regarding the embryonic division of the developing brain. The embryonic sources and segments of the adult cerebral arteries were categorized as follows:
   - Telencephalon
   - Diencephalon
   - Mesencephalon
   - Metencephalon
   - Myelencephalon

Table (2) shows that grouping of the cases according to the arteries in each of the embryonic regions corresponded to the grouping of the embryonic venous channels. Compare Table (1) and (2).

Preparation of the new classification of the arteriovenous malformation based on the embryological site of their formation

Padget’s classical embryological studies were reviewed and interpreted with the aid of new diagrams. A series of carefully selected tracings of the radiographs were prepared, and a set color code was used to draw the vessels as below:

- Artery: Red
- Adult vein: Blue
- Embryonic vein: Green
- Arterio-capillary tangle: Yellow

Based on the above findings, a hypothesis that these abnormal vessels seen in arteriovenous malformations represent normal embryonic channels that have failed to regress was suggested. To support this hypothesis, a careful study and tracings of the plates of Padget [3-7] were drawn. Special attention was given to the course and relationship of the embryonic arteries and veins at 20mm, 40mm, 80mm, and the final adult stages of the development.

The actual tracings of the vascular channels that were drawn from the radiographs were matched to the hypothetical lesions drawn from the interpretation of the embryological study by Padget [3-7].

Many matched sets of the hypothetical and the radiological vascular anatomy were found in our cases. A search for the previously published radiographs was made to collect examples to complete the validation of the hypothesis.

The illustrations were grouped according to the embryonic segment in which it was hypothesized that the malformation started. The simpler patterns in the caudal segment were dealt with first before proceeding to the more intricate cranial patterns.

The hypothetical points at which the embryonic (cerebral) arteries could rupture into the contiguous veins, producing arteriovenous communications, were established. The possible ways in which such embryonic vascular ruptures could prevent the normal process of atresia by continuous distension of the primitive veins were depicted in a series of hypothetical diagrams.

The effects of continuing growth and unfolding of the developing brain upon the ultimate anatomy of the cerebral arteriovenous malformation were also included in these diagrams.

FINDINGS

The arterial component of the malformations

In all the cases studied, the main arterial component of the malformation was easily identifiable in terms of adult anatomy. The caliber and the tortuosity of these feeding vessels varied considerably. In some cases, the main arterial trunk itself was the feeder. The development of the arterial tree was essentially normal in each case. The formation of the rest of the arterial system was entirely normal in every case. The development of the arterial system appeared to be unaffected by the fistula.

The angiomatous elements of the malformations

There were considerable variations in the size and situation of these arterio-capillary tangles.

Anterior cerebral artery malformations (12 cases): In the twelve cases studied, an angioma was present in seven and was absent in the remaining five cases. The location of the angioma varied considerably. The usual sites were sub frontal, frontal, temporal, parietal, and occipital. The sizes of the angiomas showed considerable variation.

Anterior choroidal artery (3 cases): There were three cases in this category, and an angioma was present in all of them. They were all situated in the temporal area. The size of the angiomas again varied considerably. Although these arterio-capillary tangles were situated close to the choroid plexus, there was no evidence of the involvement of the latter in any of these.

Middle cerebral artery lesions (7 cases): In all the cases studied in this group, the arterio-capillary tangles were situated either in the Sylvian territory or over the convexity of the cerebral hemisphere. In the Sylvian territory they were situated deep, sometimes extending to the surface. The angioma situated on the cerebral convexity again varied in its location and was generally distributed between the parietal and occipital lobes.

Posterior cerebral artery (8 cases): The angioma was absent in only one case. Of the remaining seven, the angioma was infratentorial in four and supra tentorial in three cases. Despite these variations in the location of the angioma, the other features of the malformation were identical in all these cases.

Superior cerebellar artery (3 cases): The angioma was supra tentorial in two of the cases and was infratentorial in one case. The infratentorial angioma had a supra tentorial extension also. There were no examples of posterior choroidal, anterior inferior or posterior inferior cerebellar artery malformations in the series of cases studied.

The venous elements of the malformations

The venous elements could be divided into two types:

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The dural venous sinuses: The ultimate venous drainage in all the cases studied was into one of the three major venous sinuses of the brain, namely, the superior sagittal, transverse, and the internal cerebral vein. The location and the development of these dural sinuses were normal in all. In some cases, more than one sinus was involved.

The abnormal venous channels: The course and direction of the venous channels draining from the angioma to the dural sinuses varied. In most cases, they could not be identified in terms of normal adult anatomy. On closer examination, in many instances these abnormal channels were repeated. Their course and direction resembled certain embryonic channels (the venous channels which normally undergo atresia in the process of development).

Anterior cerebral artery

i. The venous channels were of two typeA superficial group: These channels were situated close to the medial surface of the cerebral hemisphere, and their direction was always backwards and superiorly into the superior sagittal sinus.

ii. There was another set of venous channels situated deep in the substance of the hemisphere. These were directed backwards and medially, and they drained into either the great cerebral vein or the internal cerebral vein. In many of the cases the dilatation of the internal cerebral vein was enormous and could be interpreted as dilatations of the vein of Galen.

a. Anterior Choroidal Artery

The venous channels in this group either drained medially into the internal cerebral vein or laterally into the transverse sinus. Their course and direction were, therefore, either backwards, upwards, and medially, or backwards, upwards, and laterally.

b. The Middle Cerebral Artery

The pattern of the venous drainage for the middle cerebral artery lesions was more difficult to define. In some, it coursed downwards and laterally into the transverse sinus, and in others, medially into the great cerebral vein or superficially into the superior sagittal sinus. Various combinations of the above types were also seen. In one case, the drainage occurred by independent channels in each of the three sinuses.

c. Posterior Cerebral Artery Lesions

The venous drainage in this group was always medially, upwards, and backwards into the great cerebral vein.

d. Superior Cerebellar Artery Lesions

The venous channels drained into the transverse sinus in all the cases. The point of drainage into the transverse sinus varied. In one case, it drained near the torcular, and in the others, the drainage was near the sigmoid end.

It thus became clear at this point that the development of the arterial feeders and the dural venous sinuses were normal in every case. The variations related mainly to the site of the angioma and the course and direction of the abnormal venous channels extending between the angioma and the dural venous sinuses.

Regrouping of cases according to the similarity of venous pattern

1. When an attempt was made to regroup the lesions of similar-looking venous channels together, the following classification emerged: The anterior cerebral artery and middle cerebral artery lesions had comparable abnormal venous channels.

2. The anterior choroidal artery and posterior cerebral artery lesions had similar looking venous channels.

3. The venous drainage of the superior cerebellar artery had a pattern of its own.

Identification and matching of the abnormal venous channels with the normal embryonic venous channels

1. Some of the venous channels in the above groups were found to resemble the embryonic venous channels, as described by Padget [3-7].

Anterior Cerebral and Middle Cerebral Arteries

a. The superficial venous channels of both anterior cerebral and middle cerebral artery lesions could be compared with the venous tributaries of the marginal sinus (primitive superior sagittal sinus). Some of its tributaries, which were situated in the frontal regions of the embryo around the 20-mm stage, were found to be shifted to the parietal and occipital areas at 80 mm, following the backward swing of the superior sagittal sinus. It seemed that a similar process could account for the variations seen in this group.

b. The venous channels arising from the malformations situated deep in the cerebral hemisphere and draining medially had features comparable to that of the basal veins and anterior terminal vein in the embryo. The venous channels draining laterally could be compared with the primitive tentorial sinus.

2. Posterior Cerebral Artery

The resemblance between the embryonic venous channels was more easily seen in this group. The venous channels related to the posterior cerebral artery resembled the configuration of the dorsal diencephalic vein between 20- and 60-mm stage. Similarly, the ventral diencephalic vein and the vascular channels in relation to the anterior choroidal artery lesions were comparable.

Classification of the cases when arterial feeders are grouped with relation to their situation in the embryonic segment

The principal cerebral arteries were grouped in relation to the segment of the developing brain. It was then possible to place them in five categories:

1. Telencephalic vessels Anterior cerebral, middle cerebral

2. Diencephalic

3. Mesencephalic artery A branch of the posterior cerebral

4. Metencephalic artery Superior cerebellar arteries

5. Myelencephalon Inferior cerebellar arteries

HYPOTHESIS

1. The state of development of the arterial system is quite
advanced by the time an arteriovenous fistula occurs. Therefore, the arterial component of an arteriovenous fistula is not greatly affected.

2. Arteriovenous shunts do not occur normally in the human brain, and therefore, arteriovenous malformations found in adults must be the result of pathological rupture between embryonic arteries and veins.

4. In the embryo, the development of the cerebral venous system follows that of the arterial system. The anatomical abnormalities are more severe in the venous channels because they have not developed fully at the time when the artery ruptures into the venous system and the fistula is formed. Each embryonic segment has its own pattern of veins, and these patterns are specific for each of the segments.

5. The occurrence of a fistula in an embryonic segment will prevent the normal process of atresia in the venous channels of that segment. The persistence of these normally atritic vessels in the extruterine period are responsible for the complex anatomical configuration of arteriovenous malformations.

6. When an arteriovenous rupture occurs in an area of the brain that undergoes a very complicated developmental process, the anatomy of the arteriovenous anomaly will be correspondingly complex. The site and size of the arteriovenous rupture and the period of embryonic life at which it occurs will condition the final anatomical picture of the vascular lesion in adult life.

7. All the arteriovenous fistulae can be grouped in terms of the embryological site of formation.

8. The basic anatomical features of the malformation are specific to each segment. The pattern is easily conceived in the spinal cord. This is attributable to the simplicity of structure. The complex mode of development of the brain obscures the fundamental segmental layout.

9. The anatomical and radiological aspects of the malformation in each segment can be predicted.

The methodology used in various segments of the brain have been published by the author [7-12].

The following paper indicates how a simple pattern in the segment of Myelencephalon gets more complex in Telencephalon: Despite the complexity, it is possible to identify the persistent embryonic veins in the adult vascular malformations.

Myelencephalon: The Myelencephalon forms the junction between the spinal cord and the brain. It is represented by the medulla oblongata in the adult. This segment of the brain is only slightly more specialized than the spinal cord, so the basic pattern of the vascular system is very similar to that of the spinal cord [13].

The arterial components of the myelencephalon

There are two arteries in this segment of the brain, and these are represented in the adult as anterior and posterior inferior cerebellar arteries [7-12, 14, 15].

The arteriovenous fistula

Like all pia-arachnoidal vessels, the myelencephalic arteries cross the ventral myelencephalic veins at approximately right angles. The arteriovenous fistula in this location is possible (Figure 3) [3-7].

The venous elements of the myelencephalon

The developmental changes in the myelencephalon consist mainly of the consolidation of the notochordal segments. The ventral nerve roots are therefore brought closer. The alterations in the venous system match this process. The segmental veins are reduced in number, and the single ventral mesencephalic vein (the vagal vein) represents this change. As in the spinal cord, it makes longitudinal connections with the adjoining segmental veins cranially and the anterior spinal vein caudally. Since the afferent elements (sensory elements) of the vagal and hypoglossal nerves are not prominent in man, the dorsal nerve roots and their veins tend to disappear (Figure 4).

The pattern of venous drainage becomes more complex in the Telencephalon

The development of the telencephalon is the most complex
of all segments of the primitive brain. The segmental pattern is completely overshadowed by the developmental process. Correspondingly, the vascular system has no features easily attributable to the segmental pattern. On closer examination it is possible to appreciate the primary scheme, and the anatomical aspects of the malformations are predictable.

1. For the purpose of clarity, these lesions are dealt with separately in three sections as below: (Figure Surface of Telencephalon

2. Sylvian Malformations

3. Deep Telencephalic Malformations

**Surface of telencephalon**

The **arterial component of the malformation**: This section deals with the arteriovenous malformations situated on the cortical surface of the brain. This consists of the frontal, temporal, parietal, and occipital cortices of the adult. The anterior cerebral and middle cerebral arteries supply principally this part of the brain. The medial surface is primarily the territory of the anterior cerebral artery, and the lateral surface is supplied by the middle cerebral artery.

The **angiomatous elements of the malformation**: The terminal branches of the anterior middle cerebral arteries are closely associated with the tributaries of the superior sagittal sinus. In a situation like this, an arteriovenous fistula between these vessels is easily possible. The fistula so produced could be situated anywhere between the frontal and occipital poles. Padget, however, contended that a primary arteriovenous fistula in such a shifting and plexiform marginal sinus is not likely. It is difficult to accept this concept entirely until a more satisfactory explanation is available to account for the frequent occurrence of the arteriovenous lesions in this area.

**The venous elements**

The superior sagittal sinus constitutes the principal outlet for the veins over the surface. The development of the sinus and its tributaries begins around 40-mm stage. This period corresponds to the time when the pial arteries and veins are crossing each other at right angles in other areas of the brain also. Because of the numerous vessels involved in these plexuses, the possibilities of a fistula are very high.

**Regrouping of cases according to similarity of venous pattern**

The abnormal venous channels are directed backwards and superiorly towards the superior sagittal sinus. They are situated either on the lateral or on the medial surface of the cerebral cortex, depending upon the source of the arterial feeder.

**Identification and matching of the abnormal venous channels with the normal embryonic venous channels**

The venous channels extending between the fistula and the superior sagittal sinus are generally directed backwards and medially. They, as a rule, end in the superior sagittal sinus.

Occasionally, venous channels could be seen extending to the transverse sinus by way of the anastomotic veins of Labe and Trollard.

**Regrouping of the arteries according to their site of origin in the embryo**

Both anterior and middle cerebral arteries belong to the telencephalon, and the arterio- venous malformations arising from them have similar features. The entire cerebral cortex is formed by the telencephalon, and the anatomy of the arteriovenous malformations is identical (Figure 6).

**Vein of Galen AVMs**: Arteriovenous fistulae between the embryonic vessels situated in the midline are often associated with aneurismatic dilatation of the great cerebral vein. These fistulae occur in the telencephalic, diencephalic, and mesencephalic segments of the primitive brain. The evolution of these malformations is described (Figure 7).

**Dural AVMs**: The anatomy of Dural arteriovenous malformations is often difficult to understand. The Dural lesions have features common with those of internal carotid arterial malformations. The meningeal arteries arise from the internal...
carotid artery during the initial stages of development. They are shifted to the external carotid artery at a later stage in the development. Therefore, the Dural malformations have features similar to the internal carotid artery malformations [14].

**CFD studies**  Cerebral Arteriovenous malformations: a computational study, University of Sydney, unpublished.

**Introduction**

The unknown etiology and pathophysiology behind the progression of cerebral (AVM) has enhanced the need for the development of concise computational model to increase the overall understanding of the disorder. The difficulty however lies in the generation of a realistic model that is applicable for the study of AVMs in general. Many factors pertaining to AVM development are patient specific variables, such as arterial blood pressure, vessel geometry and AVM nidus location. As such there is a further difficulty to model via computational methods, pulsatile flow and 3D geometries for blood vessels.

**Methodology**

A set of 6 trials were conducted, each utilizing 10 different models. These models were regenerated using prior models utilizing simple pipe flow simplifications to generate a simple ‘H’ shaped AVM. Though the model geometry is three dimensional, this analysis followed a two-dimensional study to formulate a better understanding of the flow in the system. One element studied in this computational examination was the steal phenomenon, apparent as cerebrovascular steal, occurring at the arterial outlet to the nidus of an AVM [16,17].

Researchers have found a strong relationship between the onset of cerebrovascular steal and the rupture of an AVM [17]. Literature also documents that the onset of cerebrovascular steal correlates to the presentation of neurological deficits and symptoms.

These models were generated using SolidWorks (2015) and then exported to ANSYS Workbench (v. 16.2). Through ANSYS Workbench (v. 16.2) the meshing module and CFX module were used to analyze the flow characteristics.
Various assumptions were applied to each model, to simplify the problem suitable for a concise computational study. Firstly, physiological responses to stimuli in the body have been ignored in this examination, including cell renewal and auto-regulation of vessels. These factors are difficult to model and the inclusion of these in such an analysis requires significant research. Therefore, these factors were ignored in the examination, increasing the inaccuracy of the models employed. Additionally, vessel thickness was assumed constant equal to 1 millimeter, which is not the case present in the human body. To maintain validity of the model, and keep it simplified, this geometry was assumed constant. Figure (10) shows the base model applied to all the testing conditions. The influence of velocity, pressure and fistula diameters from 0.0014 to 0.0028 is shown in Figures (11-13).

SUMMARY OF FINDINGS

The figures obtained show that AVM fistula diameter increase, with all other variables constant, the pressure and velocity across the entire system shows significant changes. The computational evidence suggests that the critical change for both pressure and velocity are directly related steal phenomenon in the system. This further solidifies the various hypotheses relating to the critical nature of both diameter and the effect of the onset of steal phenomenon. The sudden alteration in pressure and velocity of blood in the AVM after during and after the onset of cerebrovascular steal may present further explanation for the dormant nature of the symptoms of the disorder where the symptoms are found to largely present only after cerebrovascular steal occurs [17,18].

In addition, it was found that the AVM system normalises with extensive fistula dilation. This occurrence is hindered in the body as a result of low wall shear stresses existing in the fistula as the vessel diameter increases. High wall shear stresses, however, resulting from increased volume through the fistula, should encourage remodelling to dilate the fistula.

Blood swirling was another important factor identified,
causing the rapid decrease in blood velocity through the fistula. This combined with low wall shear stress would likely lead to blood stagnation and increase cell adhesion in the vessel, causing additional stress on the AVM system overall [16].

The results obtained in this study highlight relationship between mass flow rate, fistula diameter, and amount of flow shunting upon the overall progression of an AVM. The increasing blood volume entering and exiting the fistula, then through the outlet of the draining vein results in a significant alteration in the pressure in this region. The venous outlet was found to be of most significant alteration as a result of the influx of fluid flowing in the area. This is contrary to traditional research conducted on the arterial outlet, as the critical region of examination [19].

We postulate that the combination of vessel dilation and diminished capacity of auto-regulation of the vessels may render the AVM incapable of approaching a point of equilibrium. This study has found evidence to explain the onset of symptomology of AVMs because of steal phenomenon, by which the system progression can be considered highly volatile.

CONCLUSION

The AVMs in adult present with very complex and often baffling anatomy. This article describes the how deductive embryological anatomy gives a clearer understanding of the evolution of the vascular structures. The segmental anatomy of the primitive brain is maintained in the adult despite developmental changes. This paper is only a brief introduction to this interesting subject.

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REFERENCES


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