"OVIDIUS" UNIVERSITY CONSTANȚA
MEDECINE FACULTY
PRECLINICAL DEPARTAMENT

CEREBRAL ARTERIAL CIRCLE MORPHOLOGY

- ABSTRACT -

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INTRODUCTION

Anatomy is a science based on observation that has a rich material of facts which requires processing by reasoning and experiment.

A very complex cerebral pathology developed in our century, is imposing an accurate and precise knowledge of brain vascularization. A leading place in the knowledge of the morphology and dynamics of cerebral circulation it belongs to cerebral arterial circle.

After a brief introduction we briefly theme I developed a history of theories STARE cerebral circulation, with details of description cerebral arterial circle over time and those that contributed to the development of knowledge regarding cerebral circulation.

After a brief introduction in which a have shortly presented the thesis subject, I have made a history of theories regarding cerebral circulation, with details of description cerebral arterial circle over time and those that had studied it.

We have considered a chapter to show ontogenetic stages of development and formation of cerebral arterial polygon, which may explain the anatomical variations of the participating segments.

In the general knowledge chapter my study has been conducted in relation to the Guy Lazorthes research, whom I considered the most representative from a morphological point of view, although disputed by several authors, provides detailed descriptions of the arterial segments and the cerebral arterial circle as a whole.

Method and material enrolled a total of 263 own cases, of which: 79 formalin treated and fresh brains that were collected from the necropsy rooms Constanta County Hospital of which 53 cases processed by dissection and 21 cases by plastic injection, 28 cases of digital subtraction angiography, 40 CT angiography, 120 RM angiography. CT and MRI examinations were performed in the clinic Medimar Imaging Services Constanta.

Also in the general study we introduced some concepts of physiology and pathophysiology involving cerebral circulation.

In describing the morphology of the cerebral arterial circle we have assessed participating branches and general aspect of cerebral arterial circle and the data obtained was analyzed in comparison with data already published in the literature. We also do appraisals of regional cerebral flow

in relation to morphological features of cerebral arterial circle. In this study we included morphological description of some abnormal basilar anastomosis and functional anatomy problems. I mention that some personal results were used by scientific or publishing articles in professional journals, such as: the National Society of Anatomy congresses (2010, 2012), the French Society of Anatomy congresses (Limoges 2007 and Rouen 2011, with abstracts published in the proceedings of conferences and "Morphologie" magazine published by Elsevier) Congress of the European Association of Clinical Anatomy (Istanbul 2009) summary published in society magazine ("Surgical Radiology, Anatomy", Springer publisher) and also in Ars Medica Tomitana, journal indexed Scopus (1 article) and Romanian Journal of macro and microscopic Functional and Clinical Anatomy, and Anthropology (2 articles).

We also studied the cerebral arterial polygon in pathological situation such as ischemic disease, cerebral artery aneurysms or arteriovenous malformations and regional cerebral flow studied in different cerebral arterial circle anatomical variants and in case of obstruction of main arterial trunks.

In the conclusion chapter we summaries the study results marking its importance for both anatomists, and especially for neurologists, neurosurgeons and radiologists.

General bibliography comprise over 200 titles that I had the opportunity to consult, which demonstrates that the morphology of the cerebral arterial circle is an issue for discussion and interest to specialist. Mention that at the end of each chapter I introduced a selective bibliography organized in order of citation and a general bibliography at the end of the paper, organized alphabetically.

I thank colleagues in the discipline, doctors Marius Popa, Constantin Ionescu, that helped me solve some problems in the realization of this work. I thank Dr. Mariana Bărdaş imaging center coordinator Medimar Imaging Services Constaţa were I had effectuated the CT and MRI exams.

Finally, thank prof. Dr. Bordei Petru, scientific leader of the thesis, to whose guidance I received in the making of this thesis during eight years.

PERSONAL RESEARCH

!

MATERIAL ŞI METODE DE LUCRU

To establish the morphological characteristics of arteries forming arterial Willis polygon, our study was performed on a total of 263 own cases, of which: 53 cases by dissection, 22 cases of plastic injection, 28 cases by digital subtraction angiography , 40 CT angiography AND 120 MRI angiography.

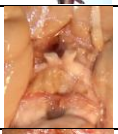
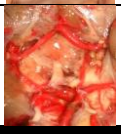
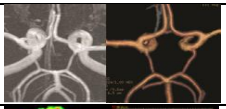
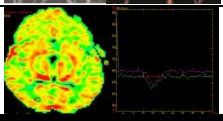
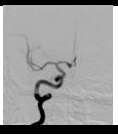
Dissections were performed in the Anatomy laboratory dissection rooms, Faculty of Medicine Constanţa, on brains extracted in morgue service. Dissection was carried out both on fresh brains and formalin treated. Of 22 brains injected with plastic, only 4 cases were completely corroded, others were corroded partially, in order to preserve relationships to other brain structures and others were dissected.

In 53 cases we used classical dissection on formalin preserved brains from which in 15 cases I have extracted the arterial branches that form the polygon to be better evaluated in two-dimensional plane.

Cerebral angiography was obtained courtesy of radiology departments that operate in the Hospital for Neurosurgeons Science and Emergency Hospital "Bagdasar - Arseni", Bucharest.

CT angiography was performed within Medimar Imaging Services Ltd on a General Electric CT Brightspeed Select 16 slice, on a total of 40 patients.

Magnetic resonance imaging provides not only an anatomical image of the vascular tree and information about cerebral microcirculation physiology or cerebral suffering, with special sequences (DWI, PWI). MRA provides morphological information similar to conventional angiography.

No.	METHOD	NR. CAZURI	PHOTO
1.	Dissection	79	
1a.	Formalin treated brain dissections	53	
1b.	Isolated polygons	15	
1c.	Fresh brain dissection	26	
1d.	Plastic injection	21	
2.	MRI	120	
2a	MRA	120	
2b	MRI perfusion	10	
3.	CTA	44	
4.	Angiography	20	
	Total	256	

TABEL2 – METODELE DE LUCRU FOLOSITE

PERSONAL RESULTS OF ARTERIAL CIRCLE OF BRAIN MORPHOLOGY

We have successively studied various segments of the cerebral arterial polygon before to study as a whole, I have followed the morphological variability of each branch participating in the formation of arterial anastomosis and polygon general aspect, because it forms a functional unit.

ANTERIOR CEREBRAL ARTERY

Anterior cerebral artery, medial division branch of the internal carotid artery has its size is less than that of the middle cerebral artery.

ORIGIN

In all cases we have found the origin of anterior cerebral artery to be always from ipsilateral internal carotid artery

TRAJECT

The anterior cerebral artery becomes pericallosal artery after front orbital and frontopolar

Only the anterior cerebral artery proximal segment take part at WILLIS arterial circle formation. This segment lies between anterior cerebral origin from internal carotid artery till anterior communicating artery junction, noted as A1.

A1 SEGMENT COLLATERAL BRANCHES

1. HEUBNER RECURENT ARTERY:

I have studied its origin, distribution territory and data obtained was compared with previous studies.

3. CENTRAL BRANCHES

3. CORTICAL BRANCHES:

I have also studied the frontoorbital , frontopolar and even pericallosal arteries originating from A1 segment (Fig. 58)

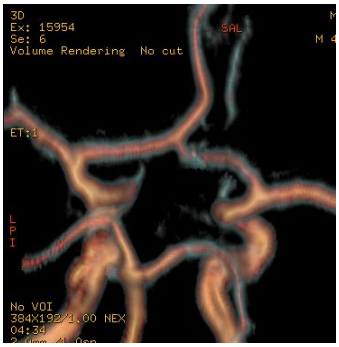


Fig.58 Common trunk formed by pericallosal and frontopolar arteries on the left side and right A2 segment hypoplasia

Rarely the two A2 segments are merging forming an anterior cerebral azygos artery (Fig.59).

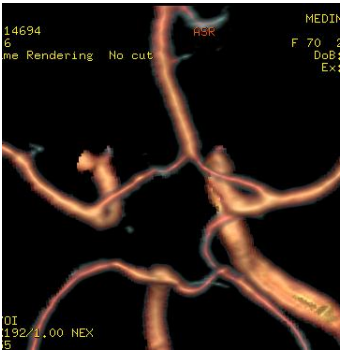


Fig. 59 Azygos anterior cerebral artery .

LENGTH

The A1 segment length varied between 8.7mm and 21.7mm. On the right side this segment length was between 9,1mm and 21.7mm, with a medium value of 14.52mm. On the left side this segment length was between 8.7mm and 18.6mm, with a medium value of 13.78mm. As can be seen from the data presented earlier cerebral artery length is slightly larger on the right side compared to the left.

CALIBER

Is variable, with a value between 0.3 mm and 3.3 mm, on both sides, the right side with values between 0.3 mm and 3.2 mm with a mean of 1.83 mm, on the left side with values ranging between 0.2 mm and 3.3 mm with a mean of 1.91 mm.

Not all the cases included a A1 segment right/left dimensional symmetry , being larger on the left side in 43 cases, 16.3% and larger on the right side in 28 cases 10.6%,.

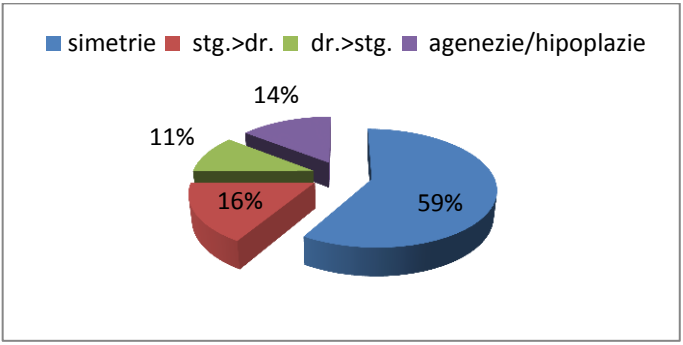


Chart.8 A1 segment aspect, according with its caliber.

In this study we identified 34 cases of hypoplasia of the A1 segment, 12.9%, of which 2 cases were bilateral hypoplasia (Fig.66), 0.8%, 8 cases of hypoplasia of left A1 segment, 3 , 1% and 24 cases of right A1 segment hypoplasia (Fig. 70), 9.1%.

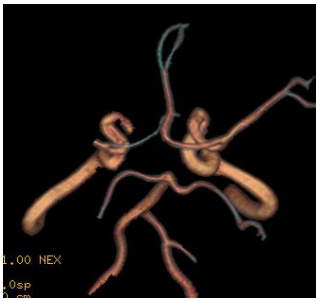


Fig.68 Left A1 segment hypoplasia.

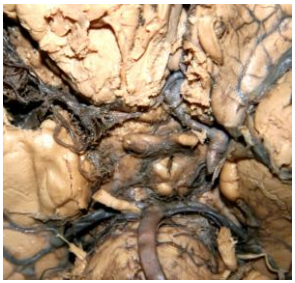


Fig.70 Right A1 segment hypoplasia

In 2 cases 0.8%, we have not found a left A1 segment, agenesis (Fig 71) and in two cases 0,8%, I observed a right A1 segment agenesis.

The results obtained were compared with other studies, published:

RESEARCH	NO.	A1 AGENESIS	A1 HYPOPLASIA
Piganiol (14)		1.46	4.21
Alpers (16)	350	0	2%
Fawcett (17)	700	-	-
Riggs & Rupp (15)	994		16%
Windle (18)	200	-	-
Fisher (12)	414		13%
Lazorthes & co. (13)	200		7.5%
Puchades (19)	62	0.8%	6.4%
This study	**263**	**0.8%**	**12,9% (9,1% dr., 3,1% stg., 0,8% bilateral)**

Table. 5 A1 segment variability compared with data published in the literature

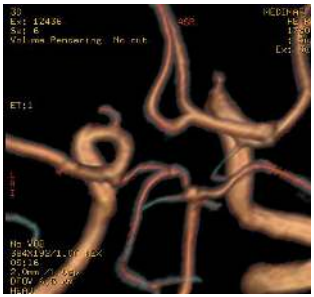


Fig.71 Left A1 segment agenesis.

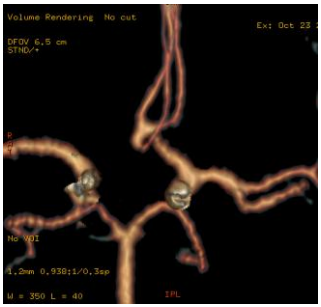


Fig.72 Right A1 segment agenesis.

ANTERIOR COMMUNICATING ARTERY

LENGTH

I have found the anterior communicating artery length to be with a value between 1.1 and 7.1 mm, with a medium of 2.75mm.

CALIBER

The caliber was with a value between 0.2 – 2.8 mm, with a medium of 1.33mm. In only 56 cases I've found its caliber under 1mm, hypoplasia (Fig.74, 75) (21,3%).

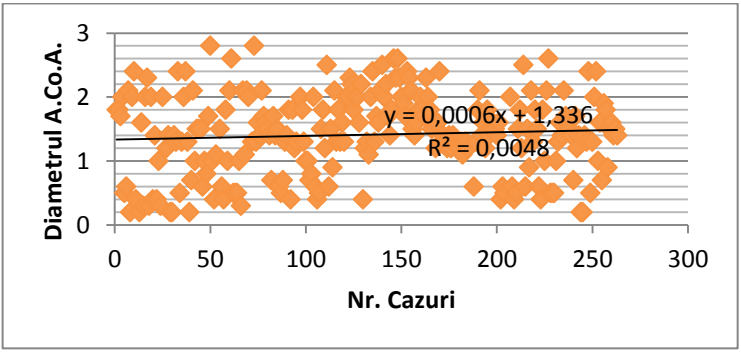


Chart.10 Distribution values of anterior communicating artery caliber

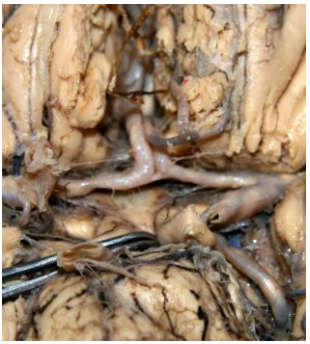


Fig.74 Anterior communicating artery hypoplasia

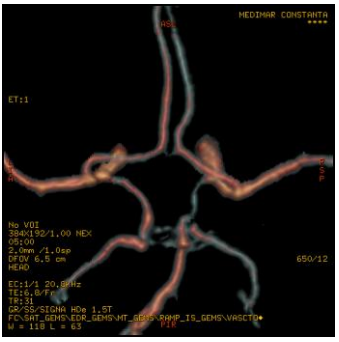


Fig. 75 Anterior communicating artery hypoplasia

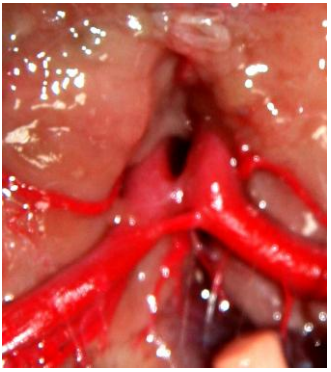


Fig. 77 Double anterior communicating artery

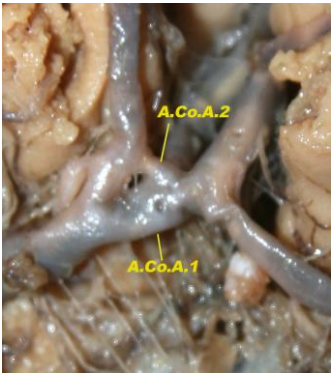


Fig.79 Double anterior communicating artery

Of the 263 polygons studied, we found only 27 cases of double anterior communicating artery (10.3%). In other 7 cases, 2.6%, the anterior communicating artery bifurcates, with a common root abuse anterior cerebral artery and the branches anastomosing with the opposite side anterior cerebral artery (Fig. 80, 82).

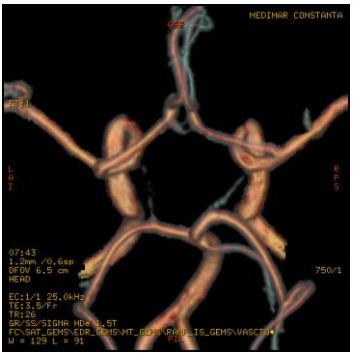


Fig.80 anterior communicating artery branching

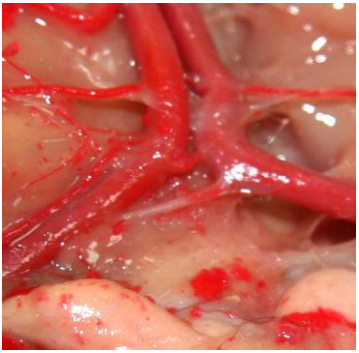


Fig.82 Triple anterior communicating artery

In 5 cases was threefold (Fig. 82), 1.9% with a scale like settlement, in this case the third communicating had length of 1.5-2.5 mm, a diameter of ~ 1 mm prior to being placed in front of the firs communicating artery at 15 to 19 mm and 3-5 mm, from the second communicating artery.

In 3 cases had plexiform appearance, 1.1% with multiple small filiform tracks that intersect in an uneven mesh network.

In 8 cases was absent: a latero/lateral anastomosis (Fig. 85), in 4 cases, 1,5%, or forming a common pericallosal artery, in 2 cases (Fig. 87) or simple the lack of this artery with a gap in between the two cerebral arteries (Fig. 88, 89).

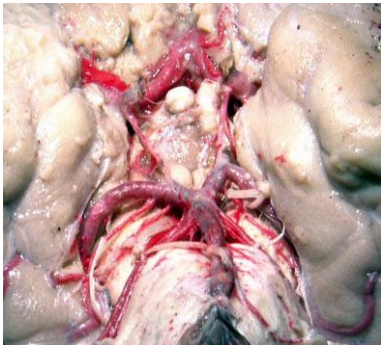


Fig.85 Latero-lateral anastomosis ACA

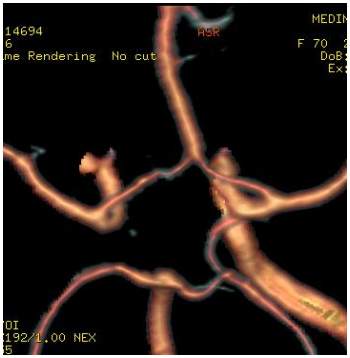


Fig.87 Joining anterior cerebral arteries in a single pericallosal artery, a azygos.

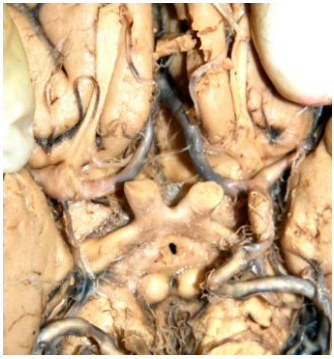


Fig.88 Lack of anterior communicating artery.

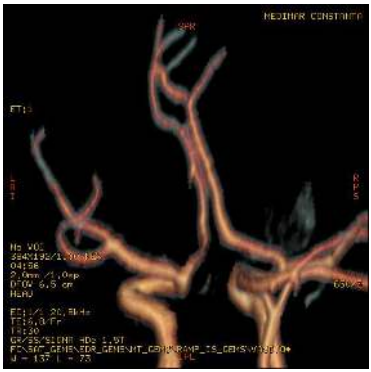


Fig.89 Lack of anterior communicating artery..

Case distribution of anterior communicating artery anatomical variants, we have delivered it in chart 12.

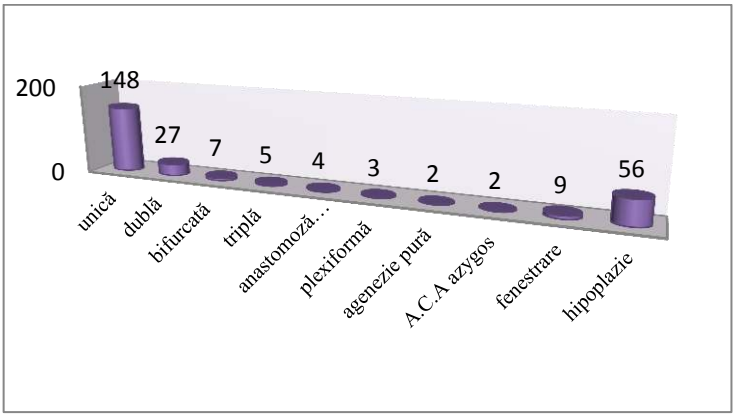


Chart.12 Distribution of anatomical variants of A.Co.A. number of cases

The results have been compared with other researches published.

STUDY	NO. CASES	AGENESIS	HYPOPLASIA
Alpers (16)	350	2%	3%
Fawcett (17)	700	0.14%	-
Riggs & Rupp (15)	994		27%
Windle (18)	200	1.5%	-
Fisher (12)	414		29%
Lazorthes & co. (13)	200		29.5%
Puchades (19)	62	3.2%	6.4%
This study	**263**	**3%**	**21,3%**

Table 6 Anterior communicating artery variability in relation to its size compared to studies in the literature.

POSTERIOR COMMUNICATING ARTERY

Has been described three main posterior communicating artery configurations (20): fetal, transitional and adult. If fetal pattern diameter of posterior cerebral artery precomunicant segment (P1) on the same side is smaller than the diameter of the posterior communicating artery.

ORIGIN

The origin of this arterial branch in from the internal carotid artery intracavernous segment near the origin of anterior choroidal artery.

TRAJECT

No relevant variations were found in the path of posterior communicating artery and morphology, there was a relationship between the length of the arterial segment, its size and trajectory so as the caliber of the artery is less the traject is more tortuous, making it difficult to assess the exact length.

LENGTH

Posterior communicating artery in my study had a length between 1.8 mm - 18mm. Right posterior communicating artery had a length between 1.8 mm and 26 mm, with a mean of 9.9 mm left posterior communicating artery had a length between 2.2 mm and 18 mm, with an average of 9mm.

I have compared the length of the two posterior communicating arteries and found that the two segments have lengths close in value to a slight dominance of the right side.

CALIBER

Right posterior communicating artery caliber has an average of 1 mm, ranging from 0.1 to 3.6 mm. In 9 cases was higher than 2.7 mm, with a maximum of 3.6 mm from the right side when associated with aneurysm (Fig. 95).

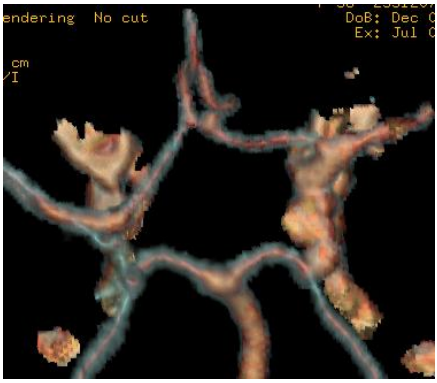


Fig.95 Right posterior communicating artery aneurysm

On the right side the caliber ranged between 0.2 and 3.6 mm with an average of 1mm, excluding cases of hypoplasia we have a mean diameter of 1.5 mm. On the left side ranged between 0.1 and 2.9 mm with an average of 1mm, excluding cases of hypoplasia have a mean diameter of 1.6 mm of posterior communicating artery

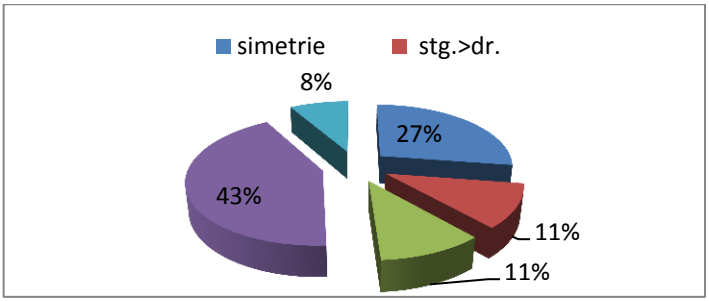


Chart.21 Posterior communicating artery variability in relation to its size

Size less than 1 mm, hypoplasia, we met in 112 cases, 42.6%. Of these 45 cases were bilateral hypoplasia (Fig. 97), 17.1%.

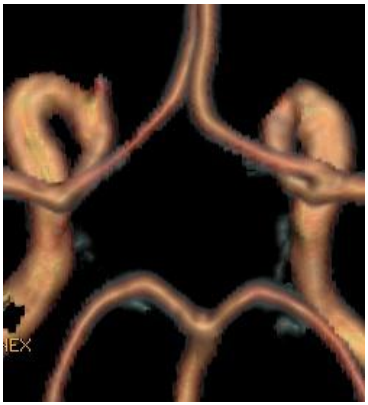


Fig.97 bilateral posterior communicating artery hypoplasia

Fig.98 Right posterior communicating artery hypoplasia

In 67 cases, 25.5% I found unilateral hypoplasia on the right side (Fig. 98), 35 cases, 13.3% and on the left 32 cases, 12.2%. Regarding the absence of posterior communicating artery I met it in 22 cases, 8.3%, of which 5 cases were bilateral agenesis (Fig. 102), 1.9%, 8 cases were left communicating artery agenesis (Fig.103), 3% and 9 cases were agenesis of the right (Fig.105), 3.4%.

Fig.103 Absence of left posterior communicating artery

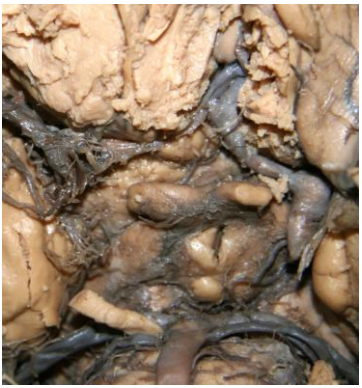


Fig.105 Absence of right posterior communicating artery

In one of the cases, unilateral agenesis, was associated with trigeminal artery, this situation corresponds to type II described by Saltzman (22) (Fig. 107, 108)

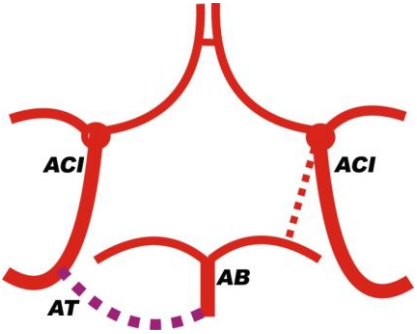


Fig.107 Schematically represented Saltzaman type II trigeminal artery

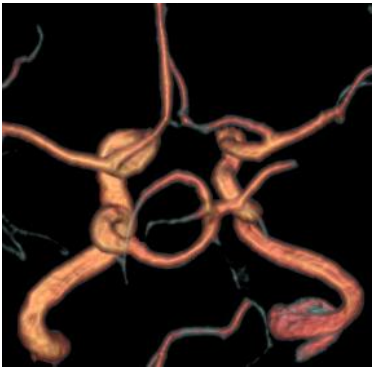


Fig.108 Saltzaman type II trigeminal artery

In other cases: 49, 18.6%, I met arterial segments with similar diameter and in 46% of cases of adult-type device, posterior communicating artery with a caliber smaller than the P1 segments.

I compared the results with other studies published in the literature.

STUDY	UNILATERAL HYPOPLASIA (%)	BILATERAL HYPOPLASIA	UNILATERAL AGENESIS (%)	BILATERAL AGENESIS (%)
Piganiol (14)			3.69	0.45
Riggs & Rupp (15)	21	30		
Lazorthes & co(13)	29.5	40		
Alpers (16)	13.5	9	0.6	
Fawcett (17)	21.5	0.7	3.2	0.4
Windle (18)	21.5	3.5	11	1.5
El Khamlichi & co. (24)	25	35		
Fisher (12)	32	49		
Puchades (19)	51		2.4	
This study	**13.3 (dr.)** **12.2 (stg.)** **T: 25.5**	**17.1**	**3.4 (dr.)** **3 (stg.)** **T: 6.4**	**1.9**

Table 7. A.Co.P variants frequency compared with other studies.

POSTERIOR CEREBRAL ARTERY

ORIGIN

Posterior cerebral arteries are terminal branches of the basilar artery. Although in 15% of the posterior cerebral artery of one side or both sides may form in the carotid artery.

TRAJECT

Posterior cerebral artery is located medial to the medial tentorial notch edge and follow this border.

LENGTH

Posterior cerebral artery segment, taking part in the formation of arterial polygon is located proximal to posterior communicating artery; P1 segment has a length of between 2 mm and 18.4 mm, with a mean of 7.1 mm.

We found that the right posterior cerebral artery has an average length between 2 to 18.4 mm, with a mean of 7.1 mm (Figure 22). Left posterior cerebral artery in its proximal segment has a length that we found between 2mm - 17mm, with a mean of 7.1 mm

CALIBER

P1 size is variable, ranging between 0.3 mm and 3.8 mm. This proximal segment may be hypoplastic, with a caliber of less than 1 mm with a posterior communicating artery with higher caliber, reaching 2.5 and even 3 mm

The proximal segment of right posterior cerebral artery size we found it between 0.3 mm and 3.7 mm, with a mean of 1.7 mm in 6 cases this arterial segment was 3mm or above this value. Caliber left P1 segment I found it in range from 0.3 to 3.8 mm, with a mean of 1.8 mm.

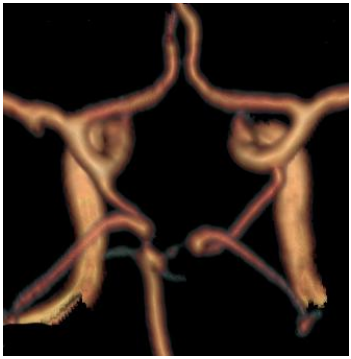


Fig.117 Bilateral P1 hypoplasia

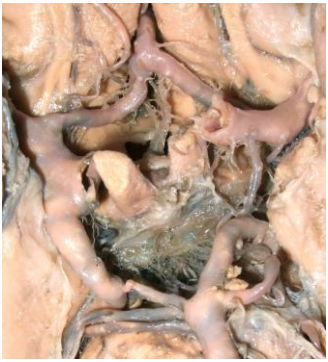


Fig.119 Right P1 hypoplasia

I met posterior cerebral artery P1 segment hypoplasia in 42 cases, 15.9%, of which 4 cases were bilateral hypoplasia, 1.5%, in some cases can

even speak of a terminal basilar artery segment hypoplasia (Fig. 118). In 38 cases we encountered unilateral hypoplasia, 14.5%, 18 cases of right side (Fig. 119), 6.9% and 20 7.6% cases left.

We also identified a case of left P1 segment hypoplasia that associated fenestration with the formation of two tracks united at their ends (Fig. 123).

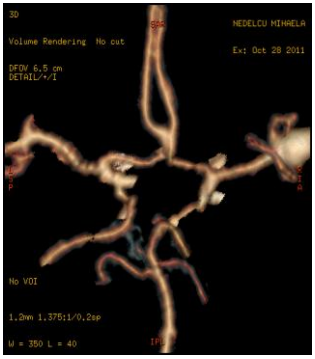


Fig.123 P1 segment fenestration.

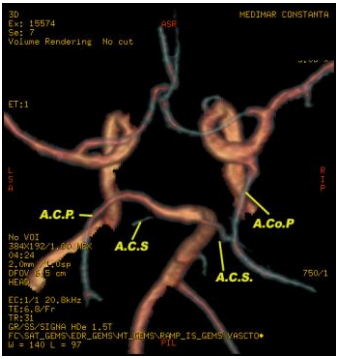


Fig. 124.Right P1 segment absence.

I noticed the absence of a P1 segment in 7 cases, 2.6%, in 4 cases the right side (Fig. 124), 1.5%.

In one case the absence of P1 segment was associated with Saltzamn type I trigeminal artery (Fig. 126, 127).

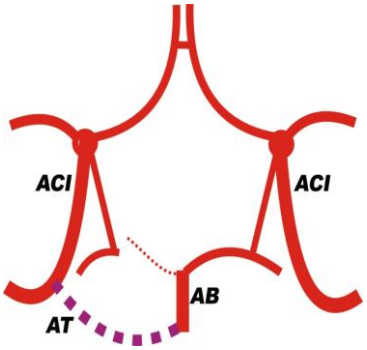


Fig.126 Saltzaman type I artery

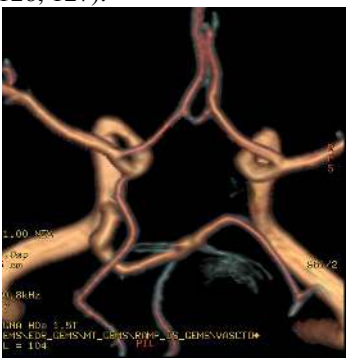


Fig.127 Saltzaman type I artery with left P1 agenesis.

In one case I encountered bilateral agenesis P1 segments, basilar artery ends with the emergence of superior cerebellar artery (Fig. 128)

Fig.128 Bilateral P1 agenesis.

The results obtained were compared with other studies on the same arterial segment (Table.9)

Study	**Unilateral hypoplasia (%)**	**Bilateral hypoplasia (%)**	**Unilateral agenesis (%)**	**Bilateral agenesis (%)**
Piganiol (14)	16,1	5,83	-	
Riggs & Rupp (15)	16	6	-	
Lazorthes & co (13)	7.5	4.5	1,5	
Alpers (16)	11	3.71		
Fawcett (17)	42	0.14		
Windle (18)	10	2		
El Khamlichi & co. (24)	14	1		
Fisher (12)	22	7		
Puchades (19)	11.3	11.3		
This study	6.9 (right.) 7.6 (left.) T: 14.5	1.5	1.5 (right.) 0.8 (left.) T: 2.3	0.4

Table 9. Frequency anomalies P1 segment size compared to other studies.

P1 POSTERIOR ARTERY SEGMENT COLLATERAL BRANCHES

Generally are central branches that can be separated into 3 groups: postero-medial, posterior-lateral, posterior choroidal arteries. Percheron have described normal neuro-vascular variations of thalamus and midbrain. One central branches variant included the formation of a common trunk of bilateral thalamic perforating arteries, as a single artery, named Percheron artery, that can rise from right or left P1 segment.(Fig. 130)

Fig.130 Percheron artery rising from left P1 segment .

In one case I met the right superior cerebellar artery originating from the posterior cerebral artery of the opposite side (Fig. 133).

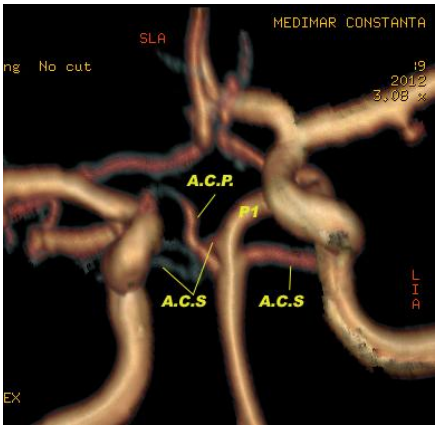


Fig.133 Superior cerebellar artery originating from the posterior cerebral artery of the opposite side.

Posterior cerebral artery is not always anastomosing with posterior communicating artery (Fig. 134), most of the times; one of them is hypoplastic, so you cannot individualize posterior cerebral artery segments, and also stops communication between vertebro-basilar pillar and carotid pillar.

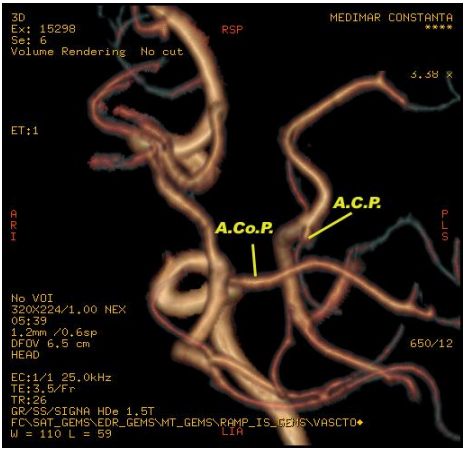
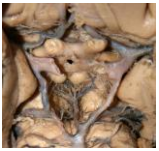


Fig.134 Lack of anastomosis between P1 segment and posterior communicating artery

WILLIS ARTERIAL CIRCLE

I found almost all classical cerebral arterial polygon as described by Guy Lazorthes except type 13.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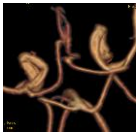
2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6


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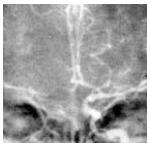
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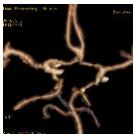
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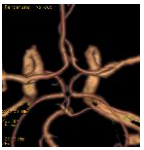
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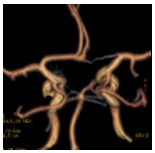
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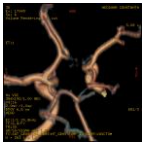
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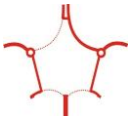
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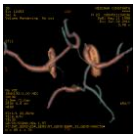
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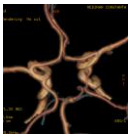
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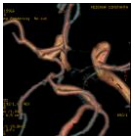
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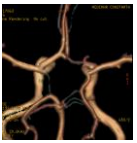
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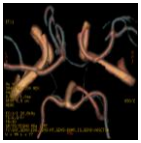
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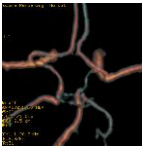
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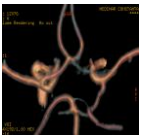
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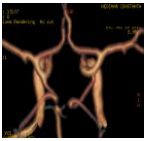
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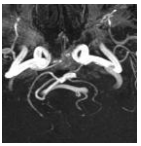
22

As can be seen from the data obtained, ideal type 1, with an arterial segment equal in size, is not a common variant, the most frequent variant being the asymmetric one, with unequal participating branches. (Chart 31)

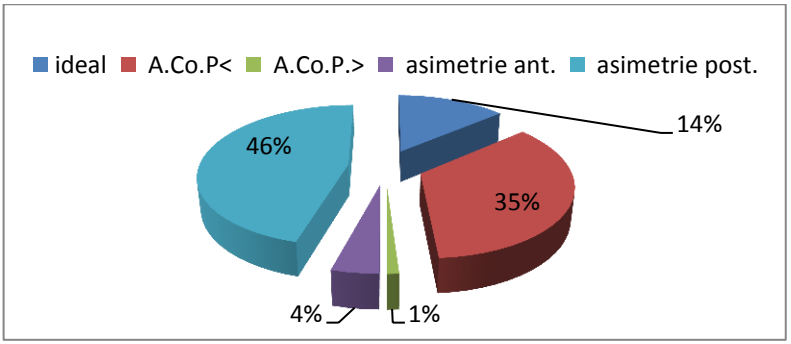

Chart.31 Type 1 variants

Outside the types described in Guy Lazorthes study, which I took as a starting point, we found a few private options, these include variations in the number of sides of the cerebral arterial polygon, incomplete variants, which would be excluded definition of "circle" that included agenesis of the participating arterial segments or lack of anastomosis between them.

In two other cases, 0.8%, arterial polygon with 6 sides noted the union of the two anterior cerebral arteries in the A2 segment, making one azygos artery (Fig. 194, 195), variant I wrote down with letter "b".

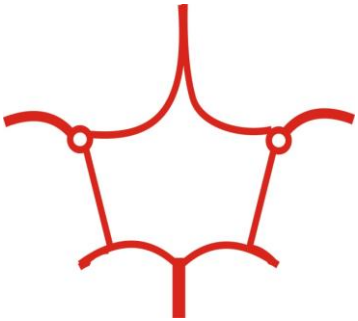

Fig.194 Graphical representation of the polygon type "b"

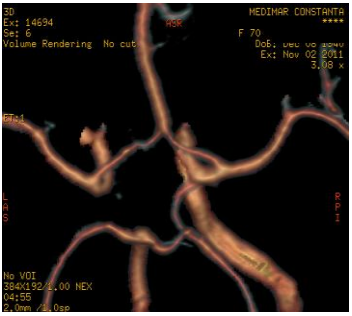

Fig.195 Arterial polygon with 6 sides with anterior cerebral artery azygos which replace type 4.

Other versions of this wide anastomosis located at the base of the brain, including incomplete circle variants which no longer correspond to the general definition. This situation we encountered in 39 cases, 14.8%. In most of the cases, 29 of them, 11%, this was due to the absence of one arterial segment participating in the formation of cerebral arterial circle. We

found no anterior communicating artery in 2 cases, 0.8%, arterial polygon version that I wrote with letter "c". In 4 cases, 1.5%, absence of one of the segments A1 (2 right and 2 left) arterial circle version I wrote a letter "d" (Fig. 198, 199).

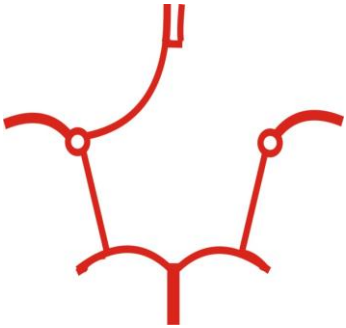

Fig.198 Graphical representation of the polygon type "d"

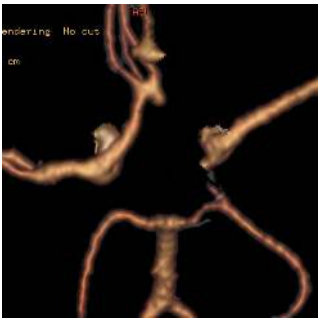

Fig.199 cerebral arterial circle with A1 segment absence

Another variant of incomplete arterial polygon is identified by the absence of posterior communicating artery (Fig. 200, 201), which I wrote down the letter "e" situation we encountered in 14 cases, 5.3% (7 right and 7 left).

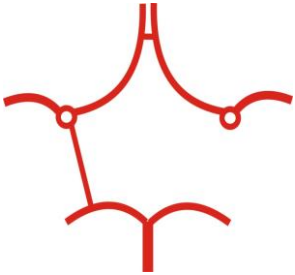

Fig.200 Graphical representation of the polygon type "e"

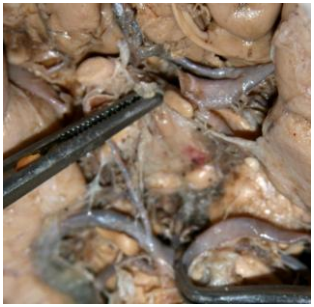

Fig.201 Arterial polygon with posterior communicating artery absence.

In 6 cases we met incomplete variants by the absence of arterial P1 posterior cerebral artery segment (Fig. 204, 205), 2.28%, variation I noted with letter "f", of which 4 of the left and two to the right.

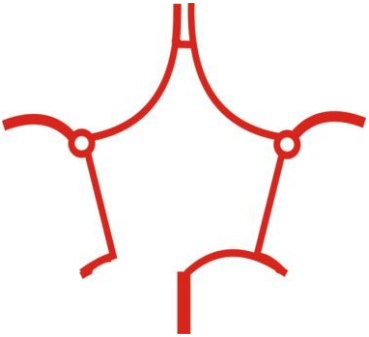

Fig.204 Graphical representation of the polygon type "f″

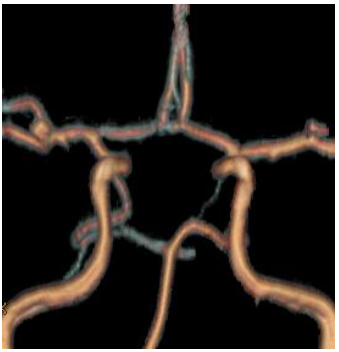

Fig.205 Arterial polygon with posterior cerebral artery absence.

Another cerebral artery variant I encountered in the absence of two arterial segments, in 6 cases, representing 2.3%. Of these, bilateral absence of posterior communicating arteries, variant I wrote a letter "g", in 5 cases, 1,9% (Fig. 208, 209).

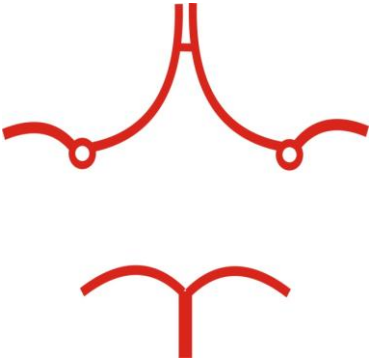

Fig.208 Graphical representation of the polygon type "g″

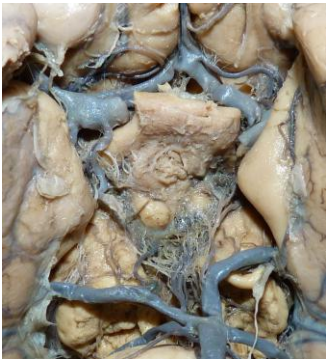

Fig.209 Arterial polygon with posterior communicating artery bilateral absence.

In one case, 0.4%, we encountered no bilateral P1 segments, posterior cerebral artery cortical territories supplied by internal carotid arteries. This version I wrote with letter "h". (Fig. 210, 211)

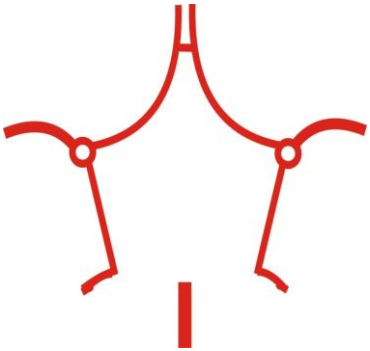

Fig.210 Graphical representation of the polygon type "h"

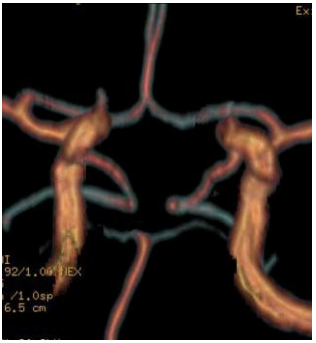

Fig.211 Arterial polygon with P1 segment bilateral absence.

In 2 cases, 0.8%, we found an absence of a P1 segment of the posterior cerebral artery associated with absence of the opposite side posterior communicating artery (Fig. 212, 213), variant I wrote down with letter "i"

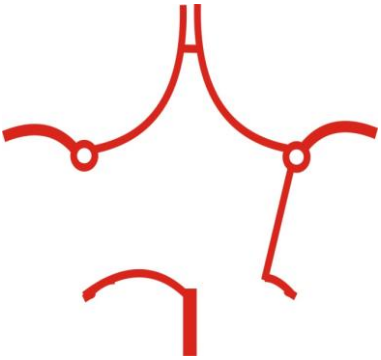

Fig.212 Graphical representation of the polygon type "i"

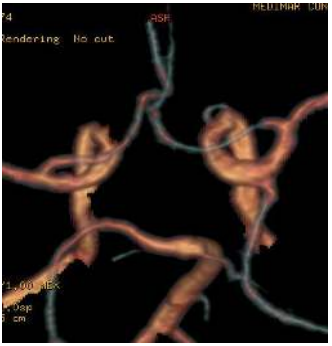

Fig.213 Type „i" arterial polygon

Another variant of incomplete arterial circle have been formed by no anastomosis between P1 segment of the posterior cerebral artery with posterior communicating artery, which I wrote down with letter "j". In this situation one participant is a hypoplastic segments.

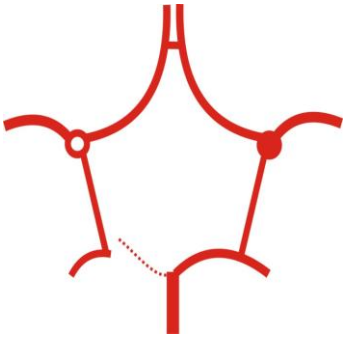

Fig.214 Graphical representation of the polygon type "j"

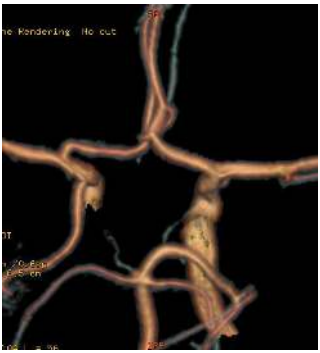

Fig.215 Type „j"polygon with P1 hypoplasia

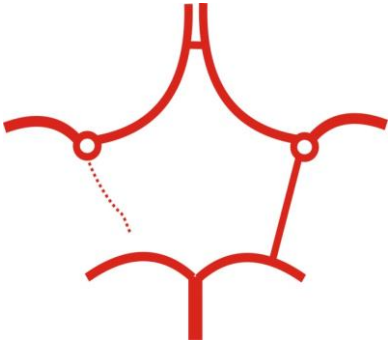

Fig.216 Graphical representation of the polygon type "j"

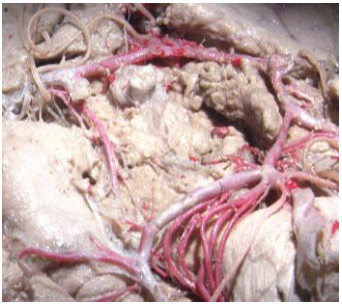

Fig.217 Type „j"polygon ACoP hypoplasia

Additional variants of incomplete arterial polygon, we analyzed by number of cases in comparison in Chart 33.

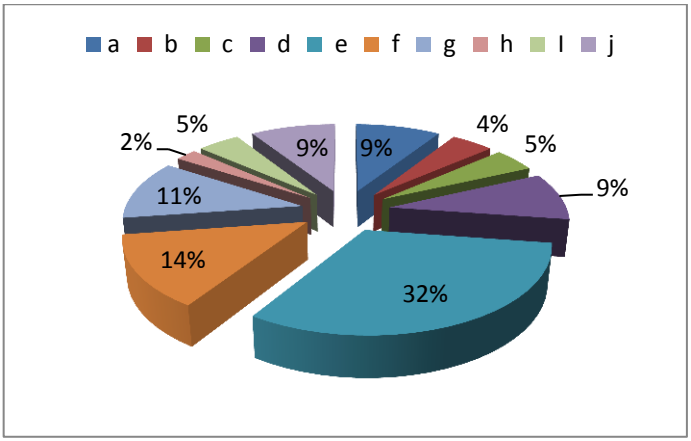

Chart 33 Cerebral arterial circle incomplete variants

The results obtained were compared with those already published in the literature (Table 12, Chart 34), from is observed to have some differences. This is due to methods of study, the time period in which they were made and the fact that most ruled incomplete arterial polygons variants.

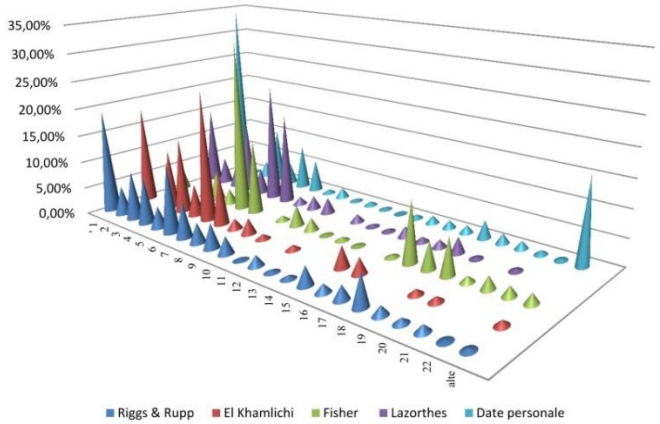

Chart.34 Comparative graphical representation of the types of cerebral arterial polygons

Types	Riggs & Rupp [21]		El Khamlichi & co. [23]		Fisher [22]		Lazorthes & co. [13]		Present study	
1	192	19.3%	18	18%	20	4.8%	29	14.5%	90	34,2%
2	54	5.4%	0		0		10	5%	1	0,4%
3	91	9.1%	11	11%	6	1.4%	9	4.5%	8	3%
4	88	8.8%	14	14%	24	5.8%	28	14%	28	10,6%
5	41	4.1%	6	6%	12	2.9%	10	5%	11	4,2%
6	126	12.7%	24	24%	131	31.6%	44	22%	22	8,4%
7	67	6.7%	10	10%	58	14%	34	17%	16	6,1%
8	38	3.8%	2	2%	0		3	1.5%	2	0,8%
9	47	4.7%	3	3%	4	1%	5	2.5%	5	1,9%
10	33	3.3%	1	1%	16	3.9%	6	3%	1	0,4%
11	2	0.2%	0		10	2.4%	0		2	0,8%
12	20	2%	1	1%	1	0.2%	3	1.5%	1	0,4%
13	5	0.5%	0		3	0.7%	1	0.5%	0	0%
14	7	0.7%	0		1	0.2%	1	0.5%	2	0,8%
15	35	3.5%	4	4%	0		4	2%	4	1,5%
16	16	1.6%	3	3%	2	0.5%	2	1%	4	1,5%
17	26	2.6%	0		46	11.1%	3	1.5%	4	1,5%
18	58	5.8%	0		21	5.1%	6	3%	9	3,4%
19	17	1.7%	1	1%	28	6.8%	1	0.5%	5	1,9%
20	10	1%	1	1%	5	1.2%	0		4	1,5%
21	13	1.3%	0		10	2.4%	1	0.5%	2	0,8%
22	3	0.3%	0		8	1.9%	0		1	0,4%
others	5	0.5%	1	1%	8	1.9%	0		41	15,6%

Table 12 Comparative evaluation of personal data regarding cerebral arterial circle typology compared with other published studies.

ARTERIAL CIRCLE MORPHOLOGICAL PECULIARITIES OF THE BRAIN IN PATHOLOGICAL CONDITIONS

ISCHEMIC CEREBRAL DISEASE

Stroke is the leading disease involving the cerebral vascular system. It is an acute neurological disorder, serious, is due to a blockage of blood irrigation of brain areas or brain hemorrhage.

If progressive obstruction, as in atheromatous disease where plaque are formed over time by successive deposits, vascular system is available to adapt to new conditions for a longer period, by creating ways to distribute

collateral flow bloodstream. This plasticity of the vascular system would explain the large number of anatomic variants described in the arterial circle at the base of the brain. I tried to do a statistical analysis of anatomical variants correlated with patient age and I divided them in a young study group, aged 10 years till 20 years or adults aged over 20 years. There was a predominance of type 1 majority arterial circle described by Guy Lazorthes to young people.

Type	**Young**		**Mature**	
1	16	64%	74	31,9%
2	0		1	0,4%
3	0		8	3,4%
4	2	8%	26	11%
5			11	4,6%
6	1	4%	21	8,9%
7			16	6,7%
8			2	0,8%
9	1	4%	4	1,7%
10			1	0,4%
11			2	0,8%
12			1	0,4%
13			0	
14			2	0,8%
15			4	1,7%
16			4	1,7%
17	1	4%	3	1,3%
18	1	4%	8	3,4%
19			5	2,1%
20	1	4%	3	1,3%
21			2	0,8%
22			1	0,4%
Others	2	8%	39	16,4%

Table 13 Distribution arterial circle variations with age

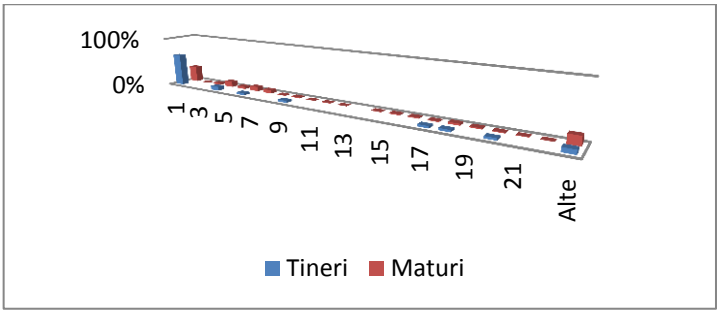

Chart. 35 The age distribution of cerebral arterial circle variants.

Polygon morphology influences served nervous tissue arterial perfusion, in the case anterior or posterior cerebral artery hypoplasia. There is a redistribution of flow through interconnected segments that are more developed than that of the opposite side; there is a nervous tissue asymmetry of regional cerebral flow. I took as an example for analyze type 19arterial polygon. We have assessed the absolute values of regional cerebral flow compared right / left in posterior cerebral artery territories (Fig. 219). We found a slight decrease of it.

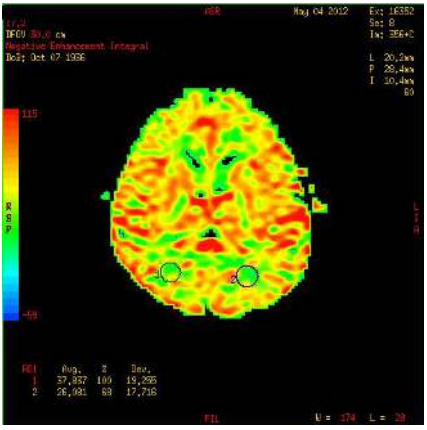

Fig. 219 Regional cerebral flow distribution map produced by MRI perfusion sequence

Comparing the paramagnetic curve tracer dynamics in brain tissue (Fig. 220), we found a broadening and flattening on left side in the left posterior cerebral artery hypoplastic territory.

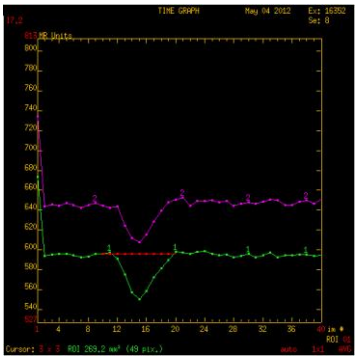

Fig. 220 Paramagnetic curve tracer dynamics compared right / left sequence obtained in perfusion MRI.

On the same case we analyzed relative regional cerebral flow in white matter and gray matter compared to right / left posterior cerebral artery territory (Fig. 221, 222).

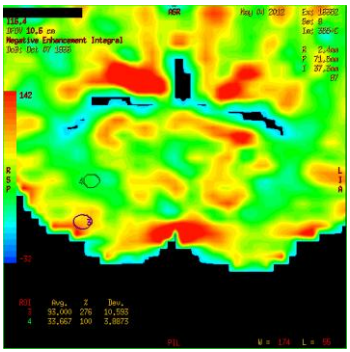

Fig. 221 Distribution map of regional cerebral flow with its absolute values on the right

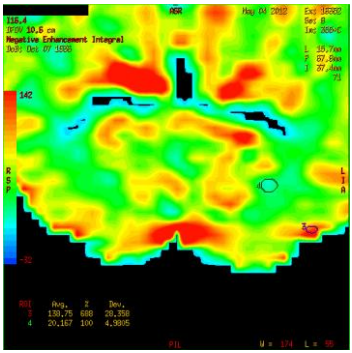

Fig. 222 Distribution map of regional cerebral flow with its absolute values on the left

There is a difference between regional cerebral flow in the white matter with a decrease of the left that has an absolute value of 20 ml/100g/min and right side 34 ml/100g/min. Gray substance have different brain regional flows, an increase in value of 139 ml/100g/min left, to the right of 93 ml/100g/min. Studying type 7 cerebral polygon, the three arterial pillars are isolated from an anatomical point of view, because of hypoplastic communicating arteries, there is a relatively symmetrical distribution of regional cerebral flow with average values of 30 ml/100g/min (Fig. 226)

and also curve tracer dynamics in nervous tissue symmetrical have appearance (Fig. 227)

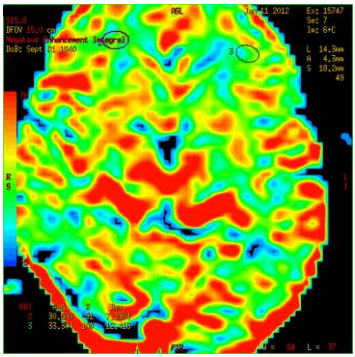

Fig. 226 Distribution map of regional cerebral flow

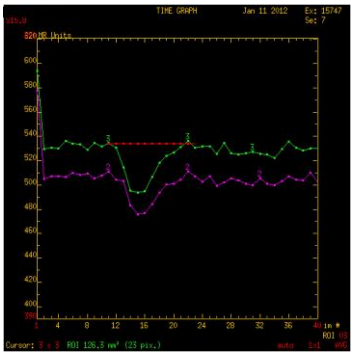

Fig. 226 Paramagnetic curve tracer dynamics compared right / left

Also we studied the redistribution of cerebral flow and pathological conditions. In this case we assessed an acute ischemic lesion in the left posterior cerebral artery territory, with an arterial polygon type 18. There is a low value of regional cerebral flow below the critical threshold, with 8.8 ml/100g/min. and also an increase in flow in the surrounding area with an aspect of hyperemia.

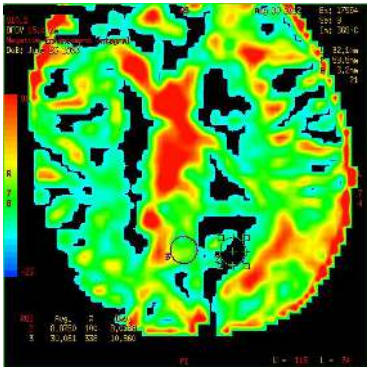

Fig. 228 Distribution map of regional cerebral flow

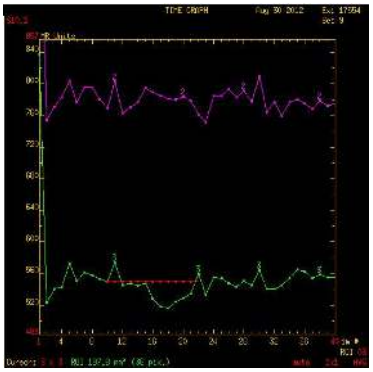

Fig. 229 Paramagnetic curve tracer dynamics compared right / left

Analyzing the paramagnetic tracer curves crossing the nervous tissue, we find that there are changes of form in both territories, their

flattening and widening of the left ischemia cannot speak of a curve tracer with a linear transition (Fig. 229).

I have compared ischemic patients cerebral arterial circles types with the ones included in the control group without ischemic disease. Pathological situation were met in 67 cases and included acute, sub acute and chronic brain ischemia, without a gender division. (Table 14, Graphic 36).

Types	**Ischemia**		**Non –ischemic cases**	
1	13	19.4%	77	39.3%
2	1	1.4%	0	0%
3	3	4.5%	5	2.5%
4	7	10.4%	21	10.7%
5	4	6%	7	3.5%
6	9	13.4%	13	6.6%
7	4	6%	12	6.1%
8	1	1.4%	1	0.5%
9	2	3%	3	1.5%
10	1	1.4%	0	0%
11	1	1.4%	1	0.5%
12	1	1.4%	0	0%
13			0	0%
14			2	1%
15	1	1.4%	3	1.5%
16	2	3%	2	1%
17	1	1.4%	3	1.5%
18	5	7.5%	4	2%
19			5	2.5%
20			4	2%
21	2	3%	0	0%
22			1	0.5%
other	9	13.4%	32	16.3%

Table 14 Percentage an case number distribution of arterial circle types in stoke patients and compared with patients without ischemia.

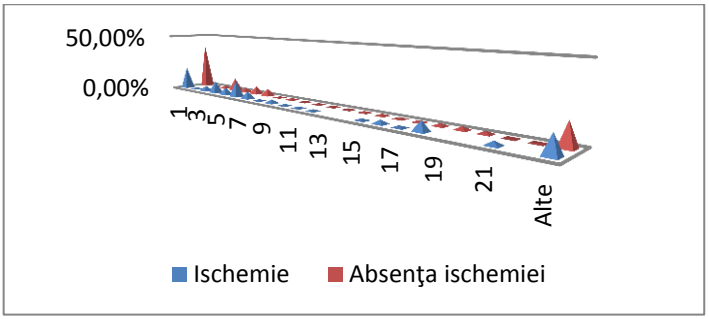

Chart 36 Distribution of arterial circle types in stoke patients and compared with patients without ischemia.

ARTERIO-VENOUS MALFORMATIONS

Arteriovenous malformations contain a ball pathological vascular network that creates high-speed shunts. A large nidus produces a turbulent arterio-venous shunt that forces the auto regulatory ability of Willis arterial circle combined with complex hemodynamic changes (29). Willis arterial circle segments, except the arterial segment that feeds the arterio-venous malformation, shrinks to reduce "stealing" phenomenon and acts as a valve to maintain satisfactory perfusion in neighborhood territory.

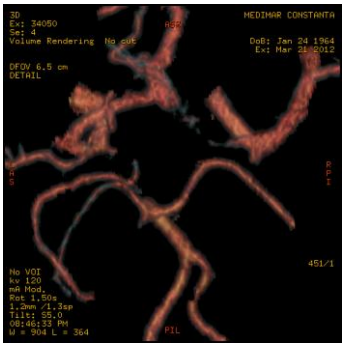

Fig. 230 Willis polygon with ACA and ACM stg. that feeds one MAV

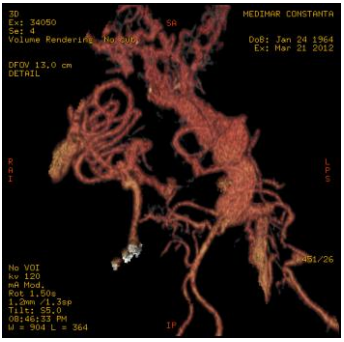

Fig. 231 MAV fed by ACA and left ACM

Artery that feeds the vascular malformation is increased in size, compared to the adjacent neighborhood branches that look hypoplastic but there are "closed", to maintain adequate perfusion in the periphery.

Arteriovenous malformation nidus obliteration may subsequently lead to the reopening of collateral segments, contracted and interpreted as hypoplastic

ANEURYSM

Long ago was launched aneurysm genesis hypothesis in relation to anatomical variations of Willis circle. (31, 32, 33).

Of 27 studied cases, 11 were anterior communicating artery aneurysms, some developed at pericallosal artery origin from the communicating artery (Fig. 235), 8 were middle cerebral artery aneurysms, 5 were internal carotid artery aneurysm, 2 anterior cerebral artery aneurysms, one of them was an distal A3 segment aneurysm associated with the presence of azygos anterior cerebral artery and one case of posterior communicating artery aneurysm (Fig. 236).

Fig. 235 Anterior communicating artery aneurysm at pericallosal artery root

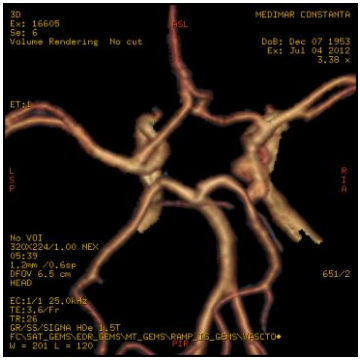

Fig. 236 Right posterior communicating artery aneurysm.

Case distribution of aneurysm in relation with cerebral arterial circle types was represented in table 15. Some type 1 related aneurysm, associated an cerebral arterial circle asymmetry.

TIP	1	4	5	6	7	8	9	14	16	17	ALTE
No. cases	5	3	2	4	1	4	1	1	2	1	5

Table 15 Case distribution of aneurysm in relation with cerebral arterial circle types.

To easily assess the impact of flow distribution in cerebral arterial circle in the development aneurysms course, we split polygons into 2 types asymmetric and symmetric. Symmetric type polygons included type 1 and 6, and the rest were asymmetrical polygons. Thus we find the presence of aneurysms in 7 cases of symmetrical polygons, 26%, excluding one type of A1 segments asymmetry and in 20 cases we found the asymmetry of Willis polygon, 74% (Chart 37).

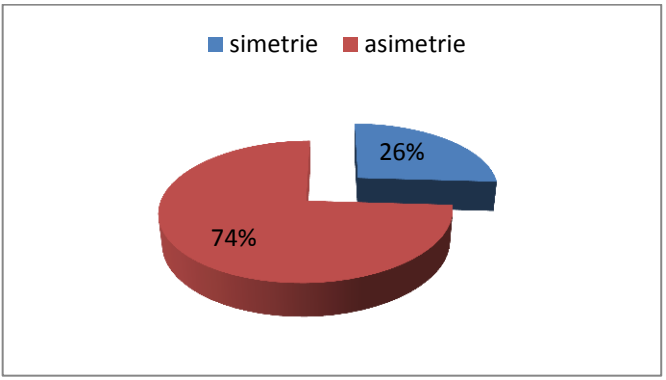

Chart. 37 Distribution of cases of aneurysms after Willis polygon asymmetry.

There is a prevalence of cerebral artery aneurysm in Willis circle asymmetry. The cerebral arterial circle arterial segments caliber asymmetries induce changes in the linear blood flow. These turbulence produce a blood vessel impingement that in time will have as a result the development of an aneurysm.

CONCLUSIONS

Arterial pillars are, according to interconnected segments size: dependent, independent and autonomous. In ideal polygon where all segments are of the same caliber, pillars are dependent. In poor polygon where the three communicating segments are weak, pillars are autonomous.

It is generally believed that anastomotic systems (normal or abnormal) are the primary means of ensuring blood supply, in case of reduction or interruption of circulatory flow in a major artery branch. On proper functioning of these alternate are depending the rapid coverage of deficits and will provide arterial blood flow to dependent territories.

The cerebral arterial polygon, is considered a unitary and indivisible morpho-functional element, it has an ideal functioning when it has a perfect shape, with all segments of equal caliber, but becomes unpredictable in relation to its anatomical variants.

Could also establish that polygon efficiency decreases with age, which is largely due to an interconnected segments hypotrophy by no need of vascular bypass on them.

I found a great variability of arterial segments constituting arterial Willis polygon, regarding the size, length and appearance. So we met more often posterior communicating artery hypoplasia. In terms of appearance the largest variability we encountered in anterior communicating artery.

Variability of arterial segments constituting cerebral polygon determines its holistic variability. The polygon with all branches with over 1mm gauge, I only met in 34.2%, of which only 4.9% we can consider "ideal", with almost equal participating branches.

Willis arterial circle participating segment hypoplasia induces a perfusion impairment in the distal depending cerebral territories. If one of the cerebral arteries is hypoplasic we can observe a hemodynamic impairment in the distal territories. Communicating segments hypoplasia doesn't have an effect on tissular perfusion, in physiological condition but becomes of major importance in the occlusion of one of the supplying pillars, as the main collateral pathway.

Cerebral arterial circle morphology, is also influencing the circulation linear flow. Its asymmetry induces disturbances in blood flow that produce vascular wall impingement, favoring aneurysm formation.

Thus we can simplify the cerebral arterial circle typology in ischemic risk polygons that stands for one or more communicating branches hypoplasia. One or more cerebral artery hypoplasia, induce an symmetrical distribution of blood flow that could induce aneurysm genesis and also an impairment of regional blood flow.

There is a remarkable plasticity of arterial circle at the base of the brain that operates in accordance with local cerebral flow and the metabolic needs from periphery. Thus it acts as a local flow regulator, with a tendency to equalize pressure perfusion in cerebral arteries and also has a protective role reacting to the nervous tissue needs both in basal conditions, increasing the flow to cortical areas metabolic and electrical active and also under conditions of ischemia to find new ways of distributing the flow in the suffering area. It can be said that not only the cerebral arterial circle morphology affects brain perfusion and also regional blood flow will influence the development of participating branches in this large anastomotic anatomical structure.

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