ONLINE FIRST



**ISSN:** 0015-5659

**e-ISSN:** 1644-3284

# Hypoplastic arteries of the cerebral arterial ring in the blind spot of computed tomography angiography

**Authors**: Milena Trandafilović, Miroslav Milić, Aleksandra Antović, Ivan Stojanović, Voja Pavlović, Stefan Todorović, Gorazd Drevenšek, Ljiljana Vasović, Miljana Pavlović, Martina Drevenšek

**DOI:** 10.5603/fm.96767

**Article type:** Original article

**Submitted:** 2023-07-31

**Accepted:** 2023-09-26

**Published online:** 2023-10-06

**Hypoplastic arteries of the cerebral arterial ring in the blind spot of computed tomography angiography**

Milena Trandafilović et al., Hypoplastic cerebral arteries on CTA

Milena Trandafilović[1], Miroslav Milić[2, 3], Aleksandra Antović[2, 3], Ivan Stojanović[2, 3], Voja Pavlović[4], Stefan Todorović[5], Gorazd Drevenšek[6], Ljiljana Vasović[7], Miljana Pavlović[1], Martina Drevenšek[8, 9]

[1]Department of Anatomy, Faculty of Medicine, University of Niš, Serbia
[2]Department of Forensic Medicine, Faculty of Medicine, University of Niš, Serbia
[3]Department of Forensic Medicine, Niš, Serbia
[4]Department of Physiology, Faculty of Medicine, University of Niš, Serbia
[5]Clinic of Neurology, University Clinical Center Niš, Serbia
[6]Institute of Pharmacology and Experimental Toxicology, Faculty of Medicine, University of Ljubljana, Slovenia
[7]Faculty of Medicine, University of Niš, Serbia
[8]Department of Orthodontics, Faculty of Medicine, University of Ljubljana, Slovenia
[9]Department of Orthodontics, Ljubljana University Medical Centre, Ljubljana, Slovenia

Address for correspondence: Milena Trandafilović, MD, Assistant Professor, Faculty of Medicine, Dept. of Anatomy, 81 Dr. Zoran Đinđić Blvd., 18000 Niš, Serbia, tel: +381 18 4570 029, fax: +381 18 4238 770, e-mail: milena.trandafilovic@medfak.ni.ac.rs

## ABSTRACT

**Background:** Some variations of the cerebral arterial circle (CAC) are associated with an increased risk for the development of various pathological conditions. This paper aimed to determine the prevalence of hypoplastic arteries of CAC and to emphasize the limited possibility of their visualization by computed tomography angiography (CTA).

**Materials and methods:** The research was performed on 400 adult cadavers by macro- and microdissection of the cerebral arteries. Each case was photographed and the diameter of the arteries was measured digitally, by analyzing photographs of the bases of the brain in the ImageJ program.

**Results:** The largest prevalence of artery diameter <1mm (<0.6mm) in CAC had the posterior communicating artery (PCoA). PCoA on the left side was hypoplastic in 44.9% (11.4%) of cases, while the same artery on the right side was hypoplastic in 44.3% (6.6%) of cases. The posterior cerebral artery was hypoplastic on the left side in 3% (0.6%) and on the right side in 4.2% (0.6%) of cases. The anterior cerebral artery had a hypoplastic caliber only on the right side in 2.4% (0.6%) of the cases, while the internal carotid arteries did not have a diameter <1mm in any case. The anterior communicating artery showed the greatest variability in morphology. Studies on CTA describe the occurrence of aplasia in a statistically significantly higher percentage, and the occurrence of hypoplastic arteries in a statistically significantly lower percentage compared to studies on cadavers.

**Conclusions:** Due to significant differences between cadaveric and radiological studies, it is necessary to analyze their results regarding arterial hypoplasia and aplasia separately. A diameter of less than 1 mm has been suggested as a criterion for arterial hypoplasia.

**Key words: adult human cadaver, brain base, hypoplasia, computed tomography angiography, cerebral arterial circle**

## INTRODUCTION

The cerebral arterial circle (CAC) represents a complex arterial anastomotic chain between the carotid and vertebrobasilar system and consists of nine vascular components: paired precommunicating segment of the posterior cerebral artery (PCA - P1), paired posterior communicating artery (PCoA), paired cerebral segment of the internal carotid artery (ICA), paired precommunicating segment of the anterior cerebral artery (ACA - A1) and unpaired anterior communicating artery (ACoA) [40].

All arterial components of CAC and their (eventual) variations are developed during the prenatal life, at the stage of the embryonic length of 40 mm, that is ovulation age of 52 days, as

cited by Burbank and Morris [5]. After that, the arteries change in terms of their diameter, which is conditioned by numerous factors, primarily hemodynamic [11].

It is common for communicating arteries to have the smallest diameter and to play a role in the redistribution of blood between neighboring arteries of larger caliber [14]. Based on the ratio of the diameters of the arteries, the types of the posterior segment of CAC are described. A less complex typification refers to the adult, transitory and fetal type [34], while a more complex typification was given by Vasović et al. and 59 types of the posterior segment of the CAC were described [38].

The morphological types of CAC are very important, but the functionally critical feature of the CAC artery is its hypoplasia. There is no exact definition of CAC arterial hypoplasia in the available literature. Functional testing of arterial diameters emphasizes a diameter of 1 mm as a critical value below which the blood vessel is not competent to provide adequate collateral circulation if there is an interruption of blood flow in one of the main neighboring arteries [6, 26]. Papantchev et al. [27, 28] defined ischemic zones of the central nervous system in 6, that is 7 (sub)types of CAC that are not able to provide adequate blood flow during unilateral selective perfusion (cannulation of the brachiocephalic trunk) in surgical procedures on the aortic arch.

Cadaveric studies show morphologically small variations, but clinical practice always relies on radiological examination methods. Two large cadaveric studies showed the frequency of incomplete CAC 6.32% [39] and 10.66% [13], while in computed tomography angiography (CTA) studies these percentages were 29% [19], 62.8% [23], 69.2% [20], 77.5% [7], 89% [37]. These data indicate that a certain percentage of hypoplastic arteries in CTA studies are shown as aplastic. It is obvious that the technical capabilities of CTA are still not at the level to be able to visualize structures of sufficiently small dimensions.

The mentioned research on the morphology of CAC and the status of the arteries that make it up represent only a part of the published studies on this topic, but they indicate their great clinical importance. Although arterial hypoplasia as a term has been known for a long time, no consensus has been reached regarding its definition, which contributes to additional differences in the incidence of its occurrence in the available literature.

Due to the differences in the definition of the term hypoplasia and the large differences in the literature data on the frequency of hypoplastic (and aplastic) CAC arteries, this paper aims to suggest criterion for hypoplasia based on the physiological contribution and anatomical

presentation, determine the frequency of hypoplastic blood vessels in a cadaveric study and critically examine the possibility of their visualization by CTA.

## MATERIALS AND METHODS

The research was performed on 400 human cadavers of both genders and different ages (from neonates to 95). Cadavers were routinely autopsied due to different causes of death at the Department of Forensic Medicine.

Each single brain base with blood vessels of all cadaveric cases was recorded on the photo film and schematically presented in the workbook. The angioarchitecture of the CAC was inspected macroscopically and under the magnifying glass, and after that, the outer diameters of the cerebral arteries were calculated by the digital images processing in the ImageJ program (http://rsb.info.nih.gov/ij/index.html). The determination of the arteries and their variation was performed according to the recommendations of Yasargil [40].

The comparison of the obtained values was interpreted regarding the literature data on the criteria for hypoplasia, which were presented previously. After that, consideration was given to the visibility of hypoplastic arteries regarding the limit value of CTA sensitivity, for which a cut-off value of 0.6 mm was taken as an approximate value of the slice thickness (0.625 mm) in CTA [7, 37].

### Ethical considerations

The authors state that every effort was made to follow all local and international ethical guidelines and laws that pertain to the use of human cadaveric donors in anatomical research [15] while conducting this study.

The approval for research is obtained from the Research Ethics Committee (No. 01-9068-4) of the Faculty of Medicine University of Niš.

### Statistical analysis

Statistical analysis was performed in SPSS ver 23.0 (IBM Corp. Released 2015. IBM SPSS Statistics for Windows, Version 23.0. Armonk, NY: IBM Corp.). Comparison between groups tested by using Student`s t-test or Mann-Whitney U test (depends on the normality of

values in groups) with the level of significance at $p < 0.05$. The Shapiro–Wilk test was a test of normality.

## RESULTS

Four neonates and a 5-year-old child were excluded from the study because their cerebral arteries were small in diameter, so they were considered an exception compared to the statistical average. The youngest of the remaining cases was a 16-year-old girl. Further analysis included 395 cases (171 female and 224 male) with an average age of 65.7 ± 17.5 years.

The average values and standard deviations of the measured diameters of the arteries of CAC, as well as their maximum and minimum values, are shown in Table 1. No statistically significant difference was found between the diameter values of the arteries on opposite sides. The criterion for hypoplasia chosen for this study is a diameter value of less than 1 mm. Among the hypoplastic arteries, those that had a diameter of less than 0.6 mm were noted, assuming that such arteries would not be visible using the CTA examination method. The percentage of hypoplastic CAC arteries and arteries with a diameter of less than 0.6 mm is given in Table 1.

The highest percentage of hypoplastic diameters was recorded on the PCoA - 44.3% on the right and 44.9% on the left side. In 28.14% of cases, both PCoAs had a hypoplastic diameter (Figure 1).

The hypoplastic diameter was also recorded in 14.4% of ACoA, but it should be emphasized that only cases with unique ACoA trunks were analyzed. This artery is extremely variable and in a third of the cases on the examined material, it is shown in the form of partial or total duplication, triplication, quadruplication, fenestration, and rete form. In variable forms, almost always one of the segments of the variation is hypoplastic. Communicating arteries also had the highest frequency of diameters less than 0.6 mm. Internal carotid arteries were not hypoplastic in any case, but in 1.8% of cases on the right side and 1.2% of cases on the left side, they had a diameter of less than 2 mm.

The absence of one or two CAC components was noted in 5.1% of cases. In 12.4% of cases, there was an excess artery of the posterior segment of the CAC, while only in 3 cases (0.75%) there was simultaneously the absence of PCoA on the right side and the presence of an excess artery of the posterior segment on the left side (Figure 2). This excess artery had a

diameter of less than 1 mm in 89.8% of cases, and a diameter of less than 0.6% in 44.9% of cases.

In the examined series, CAC with all arteries larger than 1 mm in diameter was noted in only 15.2% of cases and these CACs will be visible as complete CAC at CTA scans, while cases with an arterial diameter of less than 0.6 mm and cases with truly aplastic CAC segments were 30.6% of all cases.

The various criteria for arterial hypoplasia in CAC from the available literature, as well as the major results presented in that studies, are shown in Table 2. After summing up cases with aplasia and hypoplasia of arteries and calculating the frequency of cases with hypoplasia and aplasia in CAC in the available literature, it was determined that studies on cadaveric material recorded the occurrence of hypoplasia statistically significantly more often ($p<0.05$), while studies in which CTA was used statistically significantly more often noted the appearance of aplasia ($p < 0.001$).

## DISCUSSION

A complete and functional CAC that allows collateral blood flow has all nine vascular components in continuity and that has all vascular structures greater than 1 mm in diameter [19, 39]. It has been clinically proven that only collaterals with a diameter of at least 1 mm are effective in preventing watershed infarction [32, 35]. Arterial hypoplasia in CAC is associated with an increased risk of aneurysm formation [4, 22, 31], but also has a higher frequency in patients who have had brain ischemia and other neurological problems [19, 23, 24, 29, 36, 37].

Almost all authors who have been interested in the morphology of CAC have shown the absolute values of the diameters of CAC arteries or CAC types made concerning the diameters of its vascular components. The analysis of published studies leads to large discrepancies in the results that may depend on the size of the sample, the analyzed population [19, 23, 39], the studied pathology [7, 22, 23, 36, 37], but also on the research methodology [20]. Angiographic studies describe a lower prevalence of duplications and fenestrations [23].

Computed tomography (CT) is still a more accessible screening method than magnetic resonance imaging, especially in small hospitals or the majority of hospitals in developing countries. Although there are several generations of CT scanners, the slice thickness is usually 0.625 mm. Some CT scanners have a slice thickness of 0.6 mm or 0.5 mm. Visualization of

small-sized structures on a CT image depends on the thickness of the slice, i.e. pixel characteristics, pitch factor, radiation dose, and reconstruction quality which is significantly affected by artifacts caused by surrounding structures. For all the above reasons, as well as due to contrast media injections, postprocessing techniques, and maximum intensity projection, that affect the possibilities of visualization, CTA will not be able to visualize some small diameter cerebral arteries [12, 33]. Statistical analysis of the frequency of CAC with hypoplastic and aplastic arteries in studies performed on CTA and studies performed on cadaveric material proved that CTA studies with high statistical significance more often recorded aplasia and less often hypoplasia of cerebral arteries. Precisely in this difference compared to cadaveric studies, some percentage of hypoplastic arteries remained unvisualized. Although CAC arteries less than 1 mm in diameter are considered incompetent to provide collateral blood flow in the event of obstruction of a larger supplying blood vessel, cerebral arteries can change postnatally in terms of diameter changes affected by local hemodynamics [24]. Papantchev et al. consider this fact but also emphasize that blood will follow the lowest resistance and pass through the arteries avoiding the hypoplastic ones [27, 28]. Manninen et al. analyzed CAC using CTA and cerebral cast angiography and discussed the possibility that arteries in CAC may enlarge in diameter due to hemodynamic shear stress in the case of ICA occlusion [24], while Varga et al. concluded that ICA stenosis was an independent predictor of CAC morphology [37]. In the available literature, we did not find any information about how long and to what extent a change in the caliber of arteries could be achieved, but in blood vessels of small diameter, even a small change in absolute values represents a large percentage of diameter change. This is especially important when considering the Poiseulles-Hagen law and the consequent changes in flow rate [27]. For these reasons, it is important to make a distinction between the aplasia of an artery and the impossibility of its visualization. This difference should be emphasized in publications as well as in radiological reports. Some authors consider an artery with a diameter of less than 0.8 mm as "hypoplastic/absent" [23], while others marked as hypoplastic those that are less than 1 mm in diameter or invisible on CTA and MRA [4]. Klimek-Piotrowska et al. considered that CAC is complete if all its segments are despicable on CTA [20]. Some authors analyzed CAC morphology including hypoplasia, in large study series, without any defined criterion for hypoplasia [9, 13, 20, 21]. Additional misinterpretations arise when investigators try to interpret the results of cadaveric studies with data obtained from CTA studies and vice versa, without

knowledge of these facts and differences. In the recent study, by trying to use a cut-off value of 0.6 mm to divide possibly invisible as a part of hypoplastic cadaveric arteries, percentages of the frequency of such blood vessels were obtained, and these percentages are very similar to the results obtained in CTA studies from the literature. Small deviations can be caused by population differences or by the fact that comparison was not done with results obtained on the same material. There is a possibility that exactly 0.6 mm is the value of the artery diameter below which the blood vessel would not be visible using CTA, but it would be interesting to perform a study on the same material using both methods and determine the cut-off value more precisely.

Karatas et al. analyzed CAC morphology on 100 CTA images of patients without cerebrovascular malformations and stroke, and before that, they had performed a cadaveric study. They concluded that in the CTA study complete CAC without hypoplastic arteries was recorded in 28% of cases, and including hypoplastic arteries, that percentage was 71%. In their cadaveric study, incomplete CAC was found in 9% of cases. These authors also criticized the technical limitations of the CTA method and stated that the CTA method had limitations in detecting arteries with a diameter of less than 1 mm and that CTA-detected arterial hypoplasia and aplasia should be critically analyzed [19].

The great clinical importance of hypoplastic CAC arteries, which is described in the literature, leads first to the need for a clear definition of the term hypoplasia, and then for a precise determination of the technical limitations of visualization radiological methods used in daily clinical practice.

Several authors emphasize that it is necessary to radiologically observe and especially describe the morphology of CAC not only when examining the brain, but also during other examinations such as imaging the blood vessels of the neck [7] or when preparing for interventions on the arch of the aorta [27, 28].

**Limitations of the study**

Studies performed using CTA show the diameter of the arterial lumen because they visualize the contrast agent within the lumen, while studies on cadaveric material show the outer diameter of the blood vessel that can be more or less collapsed because of the lack of circulation. In elderly people, atherosclerotic plaques can contribute to the reduction of blood vessel diameter values on CTA and their increase in cadaveric studies. There is still no data on possible post-

mortem changes (during the first 24 hours) in the wall of brain arteries, which could lead to false measurement changes in blood vessel diameter during autopsies, also. The authors believe that the stated reasons are the cause of the differences in absolute arterial measurement values, but there is a possibility that they should not be the cause of statistically significant differences, especially in studies of a large number of cases.

## CONCLUSIONS

Arteries larger than 1 mm in diameter existed in 15.2% of the examined CACs, and among that communicating arteries had the highest frequency of hypoplastic form. If a value of 0.6 mm were used as a cut-off, below which CTA could not visualize the artery, aplasia of one of the vascular components would most likely be recorded by CTA in 30.6%, while it existed in 5.1% of cases. These results approximate data from CTA studies in the literature. Comparison of literature data and the finding of statistically significant differences in the prevalence of hypoplastic and aplastic arteries in CTA and cadaveric studies indicate significant technical limitations of CTA in the visualization of arteries of small diameter.

We suggest that hypoplastic arteries should be with a diameter of less than 1 mm because, below that value, an artery is not capable to provide collateral circulation in CAC. There is a high probability that arteries with a diameter of less than 0.6 mm are invisible on CTA.

## Acknowledgements

This study was funded by the Ministry of Science, Technological Development and Innovation of Republic of Serbia (Grant No: 451-03-47/2023-01/200113), Internal project of Faculty of Medicine, University of Niš (No. 38/20), and the Slovenian Research Agency (P3-0293 research program).

Autopsied donors and their families deserve our highest gratitude.

**Conflict of interest:** None declared

## REFERENCES

1. Al-Hussain SM, Shoter AM, Bataina ZM. Circle of Willis in adults. Saudi Med J. 2001; 22(10):895-898, indexed in Pubmed: 11744950.

2. Ansari S, Dadmehr M, Eftekhar B, et al. A simple technique for morphological measurement of cerebral arterial circle variations using public domain software (Osiris). Anat Cell Biol. 2011; 44(4):324-330, doi: 10.5115/acb.2011.44.4.324.
3. Battacharji SK, Hutchinson EC, McCall AJ. The Circle of Willis - the incidence of developmental abnormalities in normal and infarcted brains. Brain. 1967; 90(4):747-758, doi: 10.1093/brain/90.4.747.
4. Bor AS, Velthuis BK, Majoie CB, Rinkel GJ. Configuration of intracranial arteries and development of aneurysms: a follow-up study. Neurology. 2008; 70(9):700-705, doi: 10.1212/01.wnl.0000302176.03551.35.
5. Burbank NS, Morris PP. Unique anomalous origin of the left anterior cerebral artery. AJNR Am J Neuroradiol. 2005; 26(10):2533-2535, indexed in Pubmed: 16286397
6. Chuang YM, Liu CY, Pan PJ, Lin CP. Posterior communicating artery hypoplasia as a risk factor for acute ischemic stroke in the absence of carotid artery occlusion. J Clin Neurosci. 2008; 15(12):1376-1381, doi: 10.1016/j.jocn.2008.02.002
7. Coulier B. Morphologic variants of the Cerebral Arterial Circle on computed tomographic angiography (CTA): a large retrospective study. Surg Radiol Anat. 2021; 43(3):417-426, doi: 10.1007/s00276-020-02661-x.
8. De Silva KR, Silva R, Amaratunga D, et al. Types of the cerebral arterial circle (circle of Willis) in a Sri Lankan population. BMC Neurol. 2011; 11:5, doi: 10.1186/1471-2377-11-5.
9. Dimmick SJ, Faulder KC. Normal variants of the cerebral circulation at multidetector CT angiography. Radiographics. 2009; 29(4):1027-1043, doi: 10.1148/rg.294085730.
10. Eftekhar B, Dadmehr M, Ansari S, et al. Are the distributions of variations of circle of Willis different in different populations? - Results of an anatomical study and review of literature. BMC Neurol. 2006; 6:22, doi: 10.1186/1471-2377-6-22.
11. Fujimoto K. "Medial defects" in the prenatal human cerebral arteries: an electron microscopic study. Stroke. 1996; 27(4):706-708, doi: 10.1161/01.str.27.4.706.
12. Goldman LW. Principles of CT: multislice CT. J Nucl Med Technol. 2008; 36(2):57-68. doi: 10.2967/jnmt.107.044826.
13. Gunnal SA, Farooqui MS, Wabale RN. Anatomical variations of the circulus arteriosus in cadaveric human brains. Neurol Res Int. 2014; 2014:687281. doi: 10.1155/2014/687281.

14. Hartkamp MJ, van Der Grond J, van Everdingen KJ, et al. Circle of Willis collateral flow investigated by magnetic resonance angiography. Stroke. 1999; 30(12):2671-2678. doi: 10.1161/01.str.30.12.2671.
15. Iwanaga J, Singh V, Takeda S, et al. Standardized statement for the ethical use of human cadaveric tissues in anatomy research papers: Recommendations from Anatomical Journal Editors-in-Chief. Clin Anat. 2022; 35(4):526-528. doi: 10.1002/ca.23849.
16. Jiménez-Sosa MS, Cantu-Gonzalez JR, Morales-Avalos R, et al. Anatomical variants of anterior cerebral arterial circle. A study by multidetector computerized 3D tomographic angiography. Int J Morphol. 2017; 35(3):1121-1128. available at: https://www.scielo.cl/pdf/ijmorphol/v35n3/art49.pdf
17. Kamath S. Observations on the length and diameter of vessels forming the circle of Willis. J Anat. 1981; 133(Pt 3):419-423, indexed in Pubmed: 7328048.
18. Kapoor K, Singh B, Dewan LI. Variations in the configuration of the circle of Willis. Anat Sci Int. 2008; 83(2):96-106, doi: 10.1111/j.1447-073X.2007.00216.x.
19. Karatas A, Coban G, Cinar C, et al. Assessment of the Circle of Willis with cranial tomography angiography. Med Sci Monit. 2015; 21:2647-2652. doi: 10.12659/MSM.894322.
20. Klimek-Piotrowska W, Kopeć M, Kochana M, et al. Configurations of the circle of Willis: a computed tomography angiography based study on a Polish population. Folia Morphol (Warsz). 2013; 72(4):293-299. doi: 10.5603/fm.2013.0049.
21. Kovač JD, Stanković A, Stanković D, et al. Intracranial arterial variations: a comprehensive evaluation using CT angiography. Med Sci Monit. 2014; 20:420-427. doi: 10.12659/MSM.890265.
22. Lazzaro MA, Ouyang B, Chen M. The role of circle of Willis anomalies in cerebral aneurysm rupture. J Neurointerv Surg. 2012; 4(1):22-26, doi: 10.1136/jnis.2010.004358.
23. Machasio RM, Nyabanda R, Mutala TM. Proportion of variant anatomy of the circle of Willis and association with vascular anomalies on cerebral CT angiography. Radiol Res Pract. 2019; 2019:6380801, doi: 10.1155/2019/6380801.
24. Manninen H, Mäkinen K, Vanninen R, et al. How often does an incomplete circle of Willis predispose to cerebral ischemia during closure of carotid artery? Postmortem and clinical imaging studies. Acta Neurochir (Wien). 2009; 151(9):1099-1105. doi: 10.1007/s00701-009-0468-1.

25. Merkkola P, Tulla H, Ronkainen A, et al. Incomplete circle of Willis and right axillary artery perfusion. Ann Thorac Surg. 2006; 82(1):74-79, doi: 10.1016/j.athoracsur.2006.02.034.
26. Osborn AG. Diagnostic cerebral angiography. Lippincott Williams Wilkins, Philadelphia 1999.
27. Papantchev V, Hristov S, Todorova D, et al. Some variations of the circle of Willis, important for cerebral protection in aortic surgery - a study in Eastern Europeans. Eur J Cardiothorac Surg. 2007; 31(6):982-989, doi: 10.1016/j.ejcts.2007.03.020.
28. Papantchev V, Stoinova V, Aleksandrov A, et al. The role of Willis circle variations during unilateral selective cerebral perfusion: a study of 500 circles. Eur J Cardiothorac Surg. 2013; 44(4):743-753, doi: 10.1093/ejcts/ezt103.
29. Pennekamp CW, van Laar PJ, Hendrikse J, et al. Incompleteness of the circle of Willis is related to EEG-based shunting during carotid endarterectomy. Eur J Vasc Endovasc Surg. 2013; 46(6):631-637, doi: 10.1016/j.ejvs.2013.09.007.
30. Perlmutter D, Rhoton AL. Microsurgical anatomy of the anterior cerebral-anterior communicating-recurrent artery complex. J Neurosurg. 1976; 45(3):259-272, doi:10.3171/jns.1976.45.3.0259
31. Rinaldo L, McCutcheon BA, Murphy ME, et al. Relationship of A1 segment hypoplasia to anterior communicating artery aneurysm morphology and risk factors for aneurysm formation. J Neurosurg. 2017; 127(1):89-95, doi: 10.3171/2016.7.JNS16736
32. Schomer DF, Marks MP, Steinberg GK, et al. The anatomy of the posterior communicating artery as a risk factor for ischemic cerebral infarction. N Engl J Med. 1994; 330(22):1565-1570, doi: 10.1056/NEJM199406023302204.
33. Seeram E. Computed Tomography: Physical Principles, Patient Care, Clinical Applications, and Quality Control. Elsevier Inc, Philadelphia. 2022.
34. Silver JM, Wilkins RH. Arterial Embryology. In: Persistent Embryonic Intracranial and Extracranial Vessels. In: Wilkins RH, Rengachary SS (eds.) Neurosurgery Update II: Vascular, Spinal, Pediatric, and Functional Neurosurgery. McGraw-Hill, Health Professions Divisions, New York 1991: 50-51.
35. Stock KW, Wetzel S, Kirsch E, et al. Anatomic evaluation of the circle of Willis: MR angiography versus intraarterial digital subtraction angiography. AJNR Am J Neuroradiol. 1996; 17(8):1495-1499, indexed in Pubmed: 8883648.

36. van Raamt AF, Mali WP, van Laar PJ, van der Graaf Y. The fetal variant of the circle of Willis and its influence on the cerebral collateral circulation. Cerebrovasc Dis. 2006; 22(4):217-224, doi: 10.1159/000094007.
37. Varga A, Di Leo G, Banga PV, et al. Multidetector CT angiography of the Circle of Willis: association of its variants with carotid artery disease and brain ischemia. Eur Radiol. 2019; 29(1):46-56, doi: 10.1007/s00330-018-5577-x.
38. Vasović LP, Jovanović ID, Ugrenović SZ, Andjelković ZP. Normal subtypes of the posterior part of the cerebral arterial circle in human fetuses. Surg Neurol. 2008; 70(3):287-294, doi: 10.1016/j.surneu.2007.06.087.
39. Vasović L, Trandafilović M, Jovanović I, et al. Morphology of the cerebral arterial circle in the prenatal and postnatal period of Serbian population. Childs Nerv Syst. 2013; 29(12):2249-2261, doi: 10.1007/s00381-013-2151-3.
40. Yasargil MG. Intracranial Arteries. In: Operative Anatomy. In: Yasargil MG (Ed.) Microneurosurgery. Thieme, Sttutgart-New York 1984: 54-158.

**Table 1.** The average values (mean) and standard deviations (SD) of the measured diameters of the arteries of CAC, their maximum (max.) and minimum (min.) values in millimeters (mm), as well as percentage of arteries with diameter less than 1 mm and 0.6 mm

| **CAC components** | **PCA–P1 dex.** | **PCA–P1 sin.** | **PCoA dex.** | **PCoA sin.** | **ICA dex.** | **ICA sin.** | **ACA–A1 dex.** | **ACA–A1 sin.** | **ACoA** |
|---|---|---|---|---|---|---|---|---|---|
| Mean | 2.066 | 2.066 | 1.255 | 1.314 | 3.715 | 3.75 | 2.087 | 2.136 | 1.657 |
| SD | 0.617 | 0.604 | 0.701 | 0.71 | 0.863 | 0.802 | 0.476 | 0.477 | 0.66 |
| p | n.s. | | n.s | | n.s. | | n.s. | | |
| Max. | 4.048 | 3.459 | 4.196 | 3.527 | 7.128 | 6.625 | 3.31 | 3.408 | 3.985 |
| Min. | 0.47 | 0.335 | 0.316 | 0.361 | 1.264 | 1.527 | 0.525 | 1.114 | 0.452 |
| < 1 mm (%) | 4.2 | 3.0 | 44.3 | 44.9 | 0.0 | 0.0 | 2.4 | 0.0 | 14.4 |
| < 0.6 mm (%) | 0.6 | 0.6 | 6.6 | 11.4 | 0.0 | 0.0 | 0.6 | 0.0 | 1.2 |

*PCA-P1 — precommunicating segment of the posterior cerebral artery; PCoA — posterior communicating artery; ICA — cerebral segment of the internal carotid artery; ACA-A1 — precommunicating segment of the anterior cerebral artery, ACoA — anterior communicating artery; n.s. (non significant): $p > 0.05$

**Table 2.** The various criteria for arterial hypoplasia in CAC and incidence of arterial hypoplasia and/or aplasia in CAC

| The criteria for hypoplasia | Number of subjects / methodology of the study | Incidence of hypoplastic arteries and/or aplastic arteries (incomplete CAC) | Year of the study |
|---|---|---|---|
| < 1.5 mm ACA–A1 and ACoA | 50 / cadavers | 10% hypoplastic ACA–A1; 44% hypoplastic ACoA; 0.5% ACA–A1 < 1 mm; 16% ACoA < 1 mm | 1976 [30] |
| < 1 mm PCoA and ACoA | 137 / cadavers | 46% hypoplastic PCoA; 3.6% hypoplastic ACoA | 1967 [3] |
| ≤ 1 mm | 143 / cadavers | 22.37% complete CACs without hypoplastic arteries; 25.17% bilateral hypoplastic PCoA; 32.16% unilateral hypoplastic PCoA; 1.39% hypoplastic ACoA; 6.29% unilateral hypoplastic PCA–P1; 4.19% bilateral hypoplastic PCoA and hypoplastic ACoA | 2013 [39] |
| < 1 mm | 102 / cadavers | 7% unilateral aplastic PCoA; bilateral aplastic PCoA 3%; 27% unilateral hypoplastic PCoA; 33% bilateral hypoplastic PCoA; 7% hypoplastic PCA–P1 | 2006 [10] |
| | 112 / cadavers | Defined 6 types of CAC with specific hypoplastic arteries in 42.4% of cases | 2007 [27] |
| | 1000 / cadavers | 1.8% aplastic ACoA; 2.1% hypoplastic ACoA; 1% aplastic PCoA; 16.7% hypoplastic PCoA; 0.4% aplastic ACA–A1; 1.7% hypoplastic ACA–A1 | 2008 [18] |
| | 102 / cadavers | 10.8% incomplete CAC; 10.8% hypoplastic ACoA; 1% hypoplastic ACA–A1; 6.9% hypopastic PCA–P1; 26.5% | 2011 [2] |

| | | | |
|---|---|---|---|
| | | unilatelal hypoplastic PCoA; 33.3% biletaral hypoplastic PCoA | |
| | 225 / cadavers | 14% complete CAC without hypoplastic arteries; 23% bilateral hypoplastic PCoA; 11.5% unilateral hypoplastic PCoA; 14% hypoplastic ACoA; 16% bilateral hypoplastic PCoA and hypoplastic ACoA | 2011 [8] |
| | 100 / CTA | 29% incomplete CAC; 43% CAC with hypoplastic artery (38% hypoplastic PCoA) | 2015 [19] |
| | 283 / CTA | 14.13% aplastic ACoA; 5.65% aplastic ACA–A1; 5.3% hypoplastic ACA–A1 | 2017 [16] |
| | 511 / CTA | 6.1% complete CAC without hypoplastic arteries; 77.5% incomplete CAC | 2021 [7] |
| < 0.8 mm | 740 (544 carotid endarterectomy patients + 196 controls) / CTA | 85.5% incomplete CAC; hypoplastic artery in the anterior part of CAC in 24.5% in study group and in 41% in control group; hypoplastic artery in the posterior part of CAC in 39.3% in study group and in 32.2% in control group | 2019 [37] |
| | 94 / CTA | 62.8% incomplete CAC | 2019 [23] |
| | 431 / CTA and MRA | 11% invisible ACoA; 7% ACA–A1 invisible, 24% hypoplastic ACA–A1; 92% invisible PCoA; 15% invisible PCA–P1 | 2013 [29] |
| < 0.5 mm PCoA and ACoA | 87 / CCA | 22% aplastic ACoA; 46% aplastic PCoA sin; 41% CAC without hypoplastic arteries | 2006 [25] |
| | 92 / CCA | 79.3% incomplete CAC; 22.8% invisible ACoA; 1.1% hypoplastic ACoA; 43.5% invisible ACoP dex. and 5.4% hypoplastic ACoP dex.; 43.5% invisible ACoP sin. | 2009 [24] |

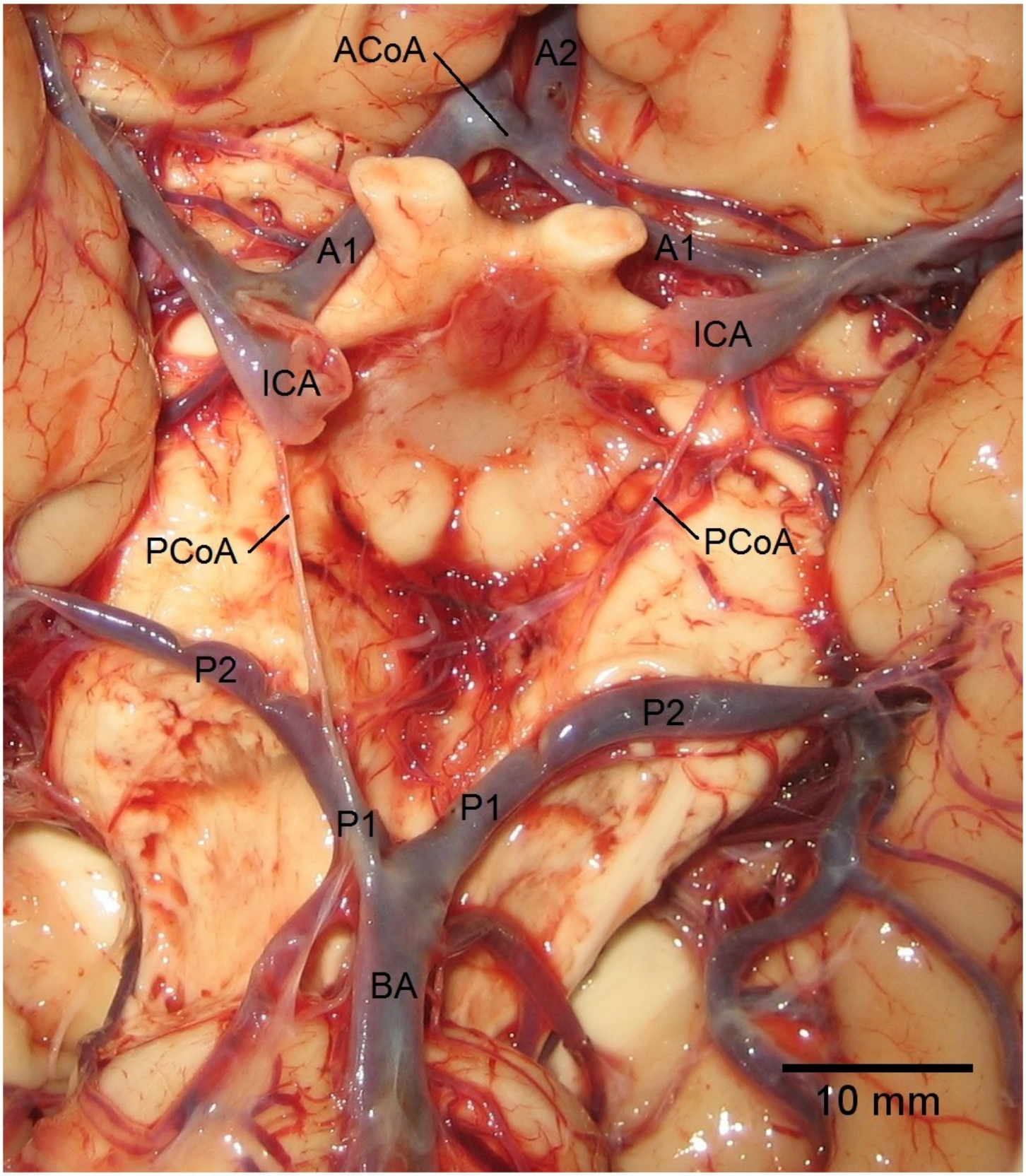
| | | | |
|---|---|---|---|
| | | and 3.7% hypoplastic ACoP sin. | |
| < 0.5 mm PCoA and ACoA;<br><br>< 1 mm other arteries | 100 / cadavers | 25% CAC with hypoplastic arteries (11% PCA–P1, 10% PCoA; 2% ACA–A1; 2% ACoA) | 1981 [17] |
| Diameter less than a half in comparison with the adjacent artery diameter | 50 / cadavers | 4.5% CAC with aplastic PCoA (2% CAC without PCoAs bilaterally); 33/100 hypoplastic PCoA; 15/100 hypoplastic PCA–P1; 4/100 hypoplastic ACA–A1 | 2001 [1] |
| Diameter of ACA–A1 is less than 50% of the diameter of the same contralateral artery | 204 / DSA | 16.7% hypoplastic ACA–A1 in patients with ACoA aneurysm | 2017 [31] |
| Frequency of hypoplasia (cadaveric study vs. CTA) 49.26 ± 7.28 vs. 23.69 ± 4.06; $p < 0.05$ | | | |
| Frequency of aplasia (cadaveric study vs. CTA) 4.07 ± 1.41 vs. 42.07 ± 9.15; $p < 0.001$ | | | |

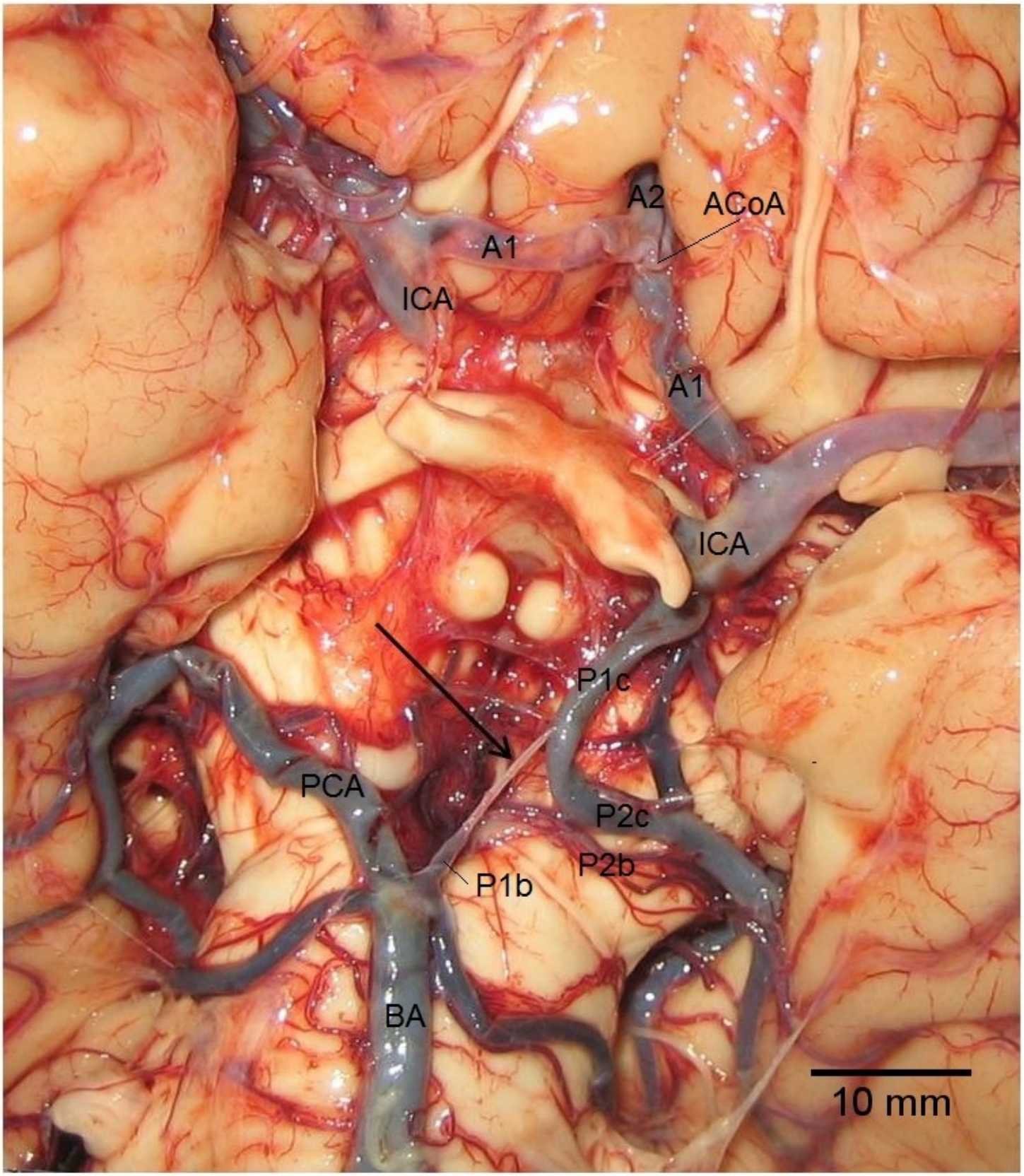
*CTA — computed tomography angiography; MRA — magnetic resonance angiography; CCA — cerebral cast angiography; DSA — digital subtraction angiography; Results for frequency of hypoplasia / aplasia finding in the percentage are presented as mean ± standard error

**Figure 1.** Case with bilateral hypoplastic posterior communicating arteries (diameter less than 0.6 mm). *ACoA — anterior communicating artery (ACoA); A1 — precommunicating segment of the anterior cerebral artery (ACA–A1); A2 — postcommunicating segment of the anterior cerebral artery (ACA–A2); ICA — cerebral segment of the internal carotid artery; PCoA — posterior communicating artery; P1 — precommunicating segment of the posterior cerebral artery (PCA–P1); P2 — postcommunicating segment of the posterior cerebral artery (PCA–P2); BA — basilar artery.

**Figure 2.** Case with aplastic right posterior communicating artery and with the presence of the hypoplastic excess artery in the posterior segment of the cerebral arterial circle on the left side (arrow). *ACoA — anterior communicating artery (ACoA); A1 — precommunicating segment of the anterior cerebral artery (ACA–A1); A2 — postcommunicating segment of the anterior

cerebral artery (ACA–A2); ICA — cerebral segment of the internal carotid artery; PCoA — posterior communicating artery; PCA — posterior cerebral artery; P1c — precommunicating segment of the posterior cerebral artery (PCA–P1) with carotid origin; P2c — postcommunicating segment of the posterior cerebral artery (PCA–P2) with carotid origin; P1b — precommunicating segment of the posterior cerebral artery (PCA–P1) with basilar origin; P2b — postcommunicating segment of the posterior cerebral artery (PCA–P2) with basilar origin BA — basilar artery.


ACoA
A2
A1
A1
ICA
ICA
PCoA
PCoA
P2
P2
P1
P1
BA
10 mm


A2
ACoA
A1
ICA
A1
ICA
P1c
PCA
P2c
P2b
P1b
BA
10 mm