

Variations in the brain circulation – the circle of Willis

Syed Yaseen¹, Zuberi Hussain Riyaz^{2,*}, Azhar Ahmed Siddiqui³

¹Tutor, ²Associate Professor, ³Professor and Head, Dept. of Anatomy, JIIUs Indian Institute of Medical Science and Research, Maharashtra

***Corresponding Author:**

Zuberi Hussain Riyaz

Associate Professor, Dept. of Anatomy, JIIUs Indian Institute of Medical Science and Research, Maharashtra

Email: zuberihussain@gmail.com

Abstract

Background Knowledge of the variations of vascular anatomy of Circle of Willis will guide the neurosurgeons during intracranial surgery.

Objectives: The aim of our study is to analyze the variations regarding shape, symmetry, length and diameters of the component arteries. The study also includes other variations like absence, duplication and abnormal origin of component arteries.

Methods: In the present study 31 specimens of human brains are obtained from the Department of Anatomy IIMSR Warudi, Badnapur, Dist. Jalna. Careful observation & measurement of the component arteries are done after proper preservation and necessary dissection. The findings are then recorded and analyzed.

Results: There were 21 out of 31 specimens (67.74%) found to be classic form of 'Circle of Willis', that was, complete, symmetrical, normal caliber and heptagonal in shape. These 21 specimens have, therefore, been considered as 'Normal'. The rest 10 specimens (33.5%) of human brain were shows variations.

Conclusion: Out of 33.5% variations most of the variations are seen in posterior communicating artery 3.22%, followed by anterior cerebral artery (6.45%) and anterior communicating artery (6.45%). Most common type of variation is hypoplasia.

Keyword: 'Circle of Willis cerebral arteries, Arterial variation, Internal Carotid Artery, Vertebral Artery, Hypoplasia.

Introduction

The arterial 'Circle of Willis' is situated at the base of the brain occupying the interpeduncular fossa within which lies the infundibulum, tuber cinereum, mamillary bodies and posterior perforated substances from before backwards. The basilar artery which is formed at the junction of the pons and the medulla by fusion of the two vertebral arteries bifurcates at the rostral border of the pons into two symmetrical posterior cerebral arteries. The carotid system gives off right and left internal carotid arteries. The internal carotid artery of each side gives off an anterior cerebral artery which comes in close relationship with that of the opposite side and is joined by a short transverse trunk, the anterior communicating artery. Each internal carotid artery again gives rise to the posterior communicating artery which anastomoses with the proximal portion of the corresponding posterior cerebral artery⁽¹⁾.

Thomas Willis (1621-1675) in his book *Cerebri Anatome* in 1664 the history of arterial circle of Willis goes back to Hetrophilus, who discovered a structure which he called as 'rete mirabile'; later on, Galen mentioned that the carotid arteries run in the neck and enter the cranium forming 'rete mirabile' (wonderful net), giving two cerebral arteries to supply the brain. Fallopius (1523–62) gave the first reasonably correct description of basal arterial ramifications except for the posterior communicating artery which he thought to be indirectly connected with the internal carotid artery through a network of small arteries. Casserius (1561–1616) corrected this mistake unilaterally.^(2,3)

There exists complete symmetry, particular in respect of shape and configuration in the pattern of the arterial circle so formed. Normally, gross anomalies such as atresia, stenosis or aneurysm are absent in the 'Circle of Willis'. In our present study, we observed the shape, symmetry, completeness of the circle and also searched for variations in length & circumference of the component arteries, duplication or doubling of any artery and anomalous origin of posterior communicating arteries. Knowledge of the variations of 'Circle of Willis' will help us to find out causes of some cases of cerebrovascular accidents, to search for probable site of thromboembolism. Areas of insufficient communication will enable us to find out functional problems of the brain. Hence the objective of the study was to search information about variations in the pattern of 'Circle of Willis' in human brain to review other relevant previous studies.

Materials and Method

Materials used

1. One set of dissection instruments – forceps, scalpel, and scissors
2. Water, bowel.
3. Magnifying lens
4. Red enamel paint
5. Sliding vernier calipers
6. Filter paper

Method

The present study was carried out in anatomy department at IIMSR, Warudi, Jalna. On 31 brains from embalmed bodies the specimens were collected. In each specimen was cleaned and cerebral arterial circle of Willis was identified. The arachnoid mater was removed from the arteries and areas around it, to facilitate the accurate measurements. The specimens were duly numbered. The cerebral arterial circle was studied in detail in each specimen with reference to parameters. Completeness, pattern, variations and symmetry of circle of Willis; presence, origin, external diameter and number of component vessels of circle of Willis and the presence of any aneurysm. The external diameter of all the arteries forming the Circle of Willis was measured using sliding vernier calipers. Magnifying lens was used to observe the vessels closely. The arteries were wiped using filter paper followed by acetone. The arteries were coloured using red enamel paint. Each specimen was photographed using a digital camera with different magnifications for better clarity of the variations noticed. 'Hypoplasia' of component artery was defined as diameter less than 0.001M.

Results

- There were 21 out of 31 specimens (67.74%) found to be classic form of 'Circle of Willis', that was, complete, symmetrical, normal caliber and heptagonal in shape. These 21 specimens have, therefore, been considered as 'Normal'. The rest 10 specimens (33.5%) of human brain were shows variations.
- 26 specimens (83.87%) were found 'Heptagonal' in shape and complete; rest 5 specimens (16.12%) were incomplete and not heptagonal in shape.
- 22 specimens (70.96%) were found 'symmetric', 9 specimens (29.03%) were found to be 'asymmetric'.

- Comparison of length and diameter between right and left sided arteries of the circle of Willis are depicted with the help of Student's paired t test below: (Table 1) Variations of 'Circle of Willis' were found during study. They are as follows:
- Complete, heptagonal form of CW with Right hypoplastic anterior cerebral artery in one specimen.
- Complete form of CW with no anterior communicating artery, that is, anterior cerebral artery were fused two specimens were found.
- Incomplete form of circle of Willis CW with absence or aplasia of Left posterior communicating artery one such specimen was found.
- Incomplete form of circle of Willis CW with absence or aplasia of Right posterior communicating artery. One such specimen was found.
- Complete form of circle of Willis CW with hypoplasia of right posterior communicating artery one such specimens was found.
- Complete form of circle of Willis CW with hypoplasia of left posterior communicating artery. One such specimen was found.
- Complete form of circle of Willis CW with one (one proximal and one distal) anterior communicating artery two such specimens were found.
- Complete form of circle of Willis CW with hypoplasia of anterior Communicating artery two such specimen were found.
- Total variants were 10 out of 31 variants. Percentage has been shown in the table-2. Most of the variation was seen in Posterior communicating artery (10%). Number of specimens exhibiting variation of ACA, PCOM and ACOM; nature of abnormalities; their number and percentage with the total are depicted below through Table 3.

Table 1: Comparison of Length and diameter between Right and Left side of circle of Willis

Group	Mean	SD	Diff.	Std. Dev. Diff	t	P
AcaR_L	13.90	1.19	-0.0427	0.1529	-2.13	0.0176
AcaL_L	13.80	1.15				
AcaR_D	2.16	0.372	-0.0071	0.1686	-0.26	0.619
AcaL_D	2.14	0.270				
PcaR_L	5.94	1.015	2.8610	0.1854	-0.67	0.587
PcaL_L	5.96	1.112				
PcaR_D	2.12	0.236	0.0040	0.0326	2.20	0.321
PcaL_D	2.16	0.222				
AcomR_L	13.16	0.160	-0.0668	0.4151	-1.573	0.0852
AcomL_L	13.05	0.172				
PcomR_D	1.81	0.284	0.0220	0.0831	1.347	0.2882
PcomL_D	1.80	0.286				

ACaR- Right Anterior Cerebral Artery, AcaL-Left Anterior Cerebral Artery, PcaR-Posterior Cerebral Artery Right, PcaL-Posterior Cerebral Artery Left, AcomR-Right Anterior Communicating Artery, AcomL-Left Anterior

Communicating Artery PcomR-Right Posterior Communicating Artery, PcomL-Left Posterior Communicating Artery, L-Length-Diameter

Table 2: Percentage of variation with total specimens

Group	n	% of variant	% of total
ACA	2	20	6.4
PCA	3	30	9.6
ACOM	3	30	9.6
PCOM	2	20	6.4

ACA-Anterior cerebellar artery, PCA- Posterior Cerebral Artery ACOM Anterior Communicating Artery PCOM-

Table 3: Nature, Number and Percentage of variation with variants and total specimens

Name of artery	Nature	Number	% of total variants	% of total specimen
ACA	Hypoplastic, right	1	10	3.22
ACA	Fused left	2	20	6.45
PCOM	Aplastic, left	1	10	3.22
PCOM	Aplastic, right	1	10	3.22
PCOM	Hypoplastic, left	1	10	3.22
PCOM	Hypoplastic, right	1	10	3.22
ACOM	double	2	20	6.45
ACOM	Hypoplastic	1	10	3.22

Discussion

Circle of Willis maintenance of and stable, constant blood flow to the brain and any change in its morphology produces different syndromes of variable vascular insufficiency in adults.⁴ Knowledge of the variations in cerebral arterial circle has importance with the increasing number of procedures like aortic arch surgery, carotid end arterectomy and microsurgical clipping of anterior communicating artery aneurysms; its variations are common and the textbook picture of symmetrical, large, approximately equal sized vessels were present only in 30% subjects.^(5,6,7) In several studies the diameters of the vessels of 'circle', has not measured and has relied upon rough estimations of the vessel diameter in determining the anomalies of the Circle of Willis rather than actual measurements. Vessels have been described as 'thread-like', 'string-like', 'minute', and 'very small' without regards to measured diameter. In the present study, typical or classic configuration was found only in 67.74% and variant in the rest 33.5% of the brains. These observations similar to Windle (1888)⁽⁸⁾ Fawcett (1905)⁽⁹⁾ who observed normal pattern in 72.8% to 82.5% cases and variant pattern in 18% to 27.2%. But the present observation are at great variance with those of Alper's et al(1959)⁽¹⁰⁾, Baptista (1963)⁽¹¹⁾, who recorded typical or classic pattern in 30% to 90% and variant in 10% to 70% cases. As mentioned earlier, in our present study variations are seen in anterior cerebral artery 9.4% posterior cerebral artery 9.6% anterior communicating artery 9.6% posterior communicating artery 9.4% (Table 2). Common variation is seen in the posterior communicating artery (10%), common type of variation is hypoplasia. Other variations are double or

duplication of artery, fused artery, absent or aplastic artery. Statistically significant difference in length but not in diameter of both sides in anterior cerebral artery has been seen. In the present study, the usual posterior communicating artery has been found aplastic in 3.22% cases on right. hypoplastic 3.22% on left side, (Table 3). Incidence of absent arteries in the circle of Willis in normal brains leading to an incomplete circle range from 0.6% to 17%. Our observations correlate with those Gardener et al (1963)⁽¹²⁾, and Romanes(1964)⁽¹³⁾ regarding posterior communicating artery, who mentioned that this artery may be absent on one or both side. The above investigation also appear to be fully in accord with those of Windle (1888)⁽⁸⁾. Furthermore, Alper's et al (1959)⁽¹⁰⁾ mentioned that this artery is never absent on both side, which is consistent with our finding. Hypoplasia of posterior communicating artery. As a whole, we observed 20% variations in posterior communicating artery.(Table 2) No both sided hypoplasia and aplasia of PCOM were seen during the course of the study. The most frequent site of abnormal diameter was seen in the posterior half of the circle, and the proportion in the present study is similar to other reported series. This may be due to embryological development of the posterior half of the CW, where the basilar and the ICAs anastomose during development of the cerebral arteries. Posterior communicating artery was seen to exhibit maximum instances of abnormalities resulting in anomalous circle. These observations corroborate with those of Windle (1888)⁽⁸⁾, Romanes (1964)⁽¹³⁾ who emphasized that the majority of anomalies occur in the posterior portion of the CW, particularly in the posterior communicating artery. In our present study, the anterior cerebral artery, one of

the components of CW has been found to exhibit abnormalities by the way of fusion between the arteries of one side with that of the contra lateral side, forming fused ACA 3.22% of such specimens were found (Table 3). Fusion of the anterior cerebral artery may cause absence of ACOM artery. Absence of ACOM is also possible without fusion of anterior cerebral artery. The present observations largely corroborate with Windle (1888)⁽⁸⁾ and Alpers et al (1959)⁽¹⁰⁾ who recorded 3% and 2% cases of absence of the ACOM due to fusion of the two anterior cerebral arteries respectively. The present observations fail to demonstrate the complete absence of ACOM without fusion of anterior cerebral artery and so unable to compare the finding with those of Fawcett et al (1905)⁽⁹⁾ who found complete absence of anterior communicating artery in 0.14% cases. Another form of variation was found, that is, right sided hypoplastic anterior cerebral artery (3.22%, in Table 3). The diameter below which the segment of ACA that is part of CW could be called hypoplastic has not been well defined, but Perlmutter and Rhoton⁽¹⁴⁾ used 1.5 mm as the cut off value. They found 10% of the brains to have less than 1.5 mm in diameter in the aforesaid segment. Alpers et al (1959)⁽¹⁰⁾ found string like components of one of the vessels of the CW in 28% cases, with that part of ACA being the predominant site. Riggs and Rupp (1963)⁽¹⁵⁾ observed hypoplasia of that part of ACA in 7% cases. No other form of abnormalities has been found in ACA. During the course of the study, we had not observed any third anterior cerebral artery (artery of the corpus callosum). In our study, the anterior communicating artery, one of the components of CW was found to exhibit abnormalities by the way of doubling, that is, presence of two anterior communicating arteries; one proximal, another distal to connect ACA of both side 3.22% of specimens had such type of abnormalities. (Table 3). This finding is largely in accord with the observations of Fawcett et al (1905)⁽⁹⁾, Alpers et al (1959)⁽¹⁰⁾. It is to be noted further that Gardener et al (1963)⁽¹²⁾ and Romanes (1964)⁽¹³⁾ also mentioned that the anterior communicating artery may exist as double. Another form of variation was found, that is, hypoplastic anterior communicating artery (1.25%, Table 3). No other form of abnormalities has been found in Acom. Total variations found in our study in ACOM was 9.6% (Table 2). The state of the circle becomes important in determining the adequacy of the brain circulation. The possibility of by-passing or shunting effects in occlusion of one of the cerebral vessels and the adequacy of recovery or lack of recovery after vascular occlusions may be explained in part by variations in the anatomy of the CW⁽¹⁶⁾. A rapid, high reperfusion strongly increases survival in the ischemic penumbra, inhibiting the growth of the core region. In our study we could not detect any aneurysm or arteriovenous malformation. This is not in accord with findings by Martinez et al 2004⁽¹⁷⁾ where they recorded vascular

malformation in their study. The knowledge of these anatomical variants is of vital importance in surgery, the aim being to preserve the arteries in unusual locations, which if injured can determine invalidating sequel. So a detailed knowledge of various configurations of the CW is an important factor affecting the result of surgical interventions. Cerebrovascular diseases, internal carotid artery occlusion, unilateral flow restrictive extracranial carotid artery disease together with their signs and symptom grossly depend upon the variations of the anatomical pattern of CW Kluytmans et al 1999⁽¹⁸⁾; Emsley et al 2006⁽¹⁹⁾. The hemodynamic profile of cerebral artery in both symptomatic and asymptomatic patients with carotid artery stenosis is of clinical importance. In those cases, important role in maintaining collateral within the CW is played by anterior communicating artery, although in some patients middle cerebral artery may also be supplied by posterior communicating artery. In patients with symptomatic internal carotid occlusion blood supply of collateral flow, which is affected by the pattern of collateral pathways, may be important factors determining the severity of hemodynamic impairment Yamauchi et al 2004⁽²⁰⁾. Hendrikse et al (2005)⁽²¹⁾ suggested that large asymmetries in volume flow between the right and left internal carotid arteries or decreased volume flow in the basilar artery is not necessarily caused by vascular diseases but may be caused by variations in the anatomy of the CW. In Moyamoya disease which primarily occurs among oriented people (Japanese, Koreans, Caucasians), there is progressive intracranial vascular obliteration of the CW, resulting in successive ischaemic or haemorrhagic events Marcinkevicius et al 2006⁽²²⁾. Again, anterior communicating artery aneurysms are frequently associated with an asymmetrical CW. In such case the anterior communicating artery is probably exposed to high hemodynamic stress caused by a considerable shunt flow across the anterior communicating artery to the distal segment of the contralateral anterior cerebral artery (Ujiie et al 1996)⁽²³⁾.

Conclusion

The knowledge of these anatomical variants is of vital importance in surgery, the aim being to preserve the arteries in unusual locations, which if injured can determine invalidating sequel. So a detailed knowledge of various configurations of the CW is an important factor affecting the result of surgical interventions. Cerebrovascular diseases, internal carotid artery occlusion, unilateral flow restrictive extracranial carotid artery disease together with their signs and symptom grossly depend upon the variations of the anatomical pattern of CW Variations in cerebral arterial circle of Willis in producing hypoxia of brain in many clinical conditions and provides the relevant data on these variations for its possible implications. This may have

special utility to neurosurgeons as well who perform advance procedures while maintaining constant blood supply to the brain.

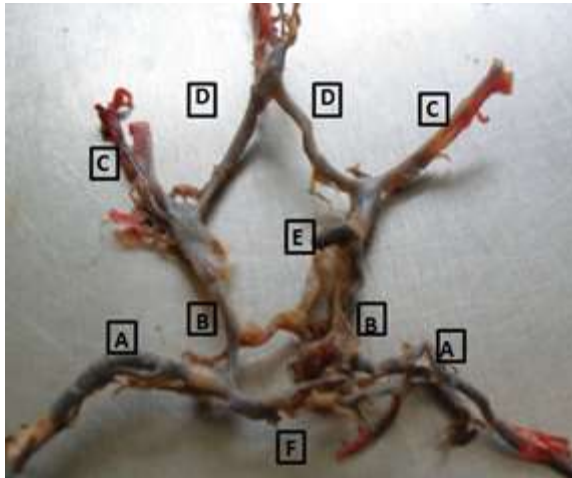


Fig. 1: Showing Dissected circle of Willis. A: PCA. B: PCoA. C: MCA. D: ACA. E: ICA. F: Vascular malformation connecting both posterior cerebral arteries

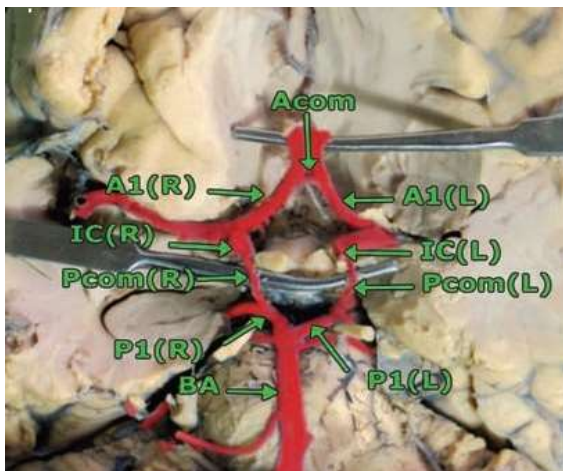


Fig. 2: Showing the complete cerebral arterial circle of Willis painted in red. A1 (R): Pre-communicating segment of right anterior cerebral artery, A1 (L): Pre-communicating segment of left anterior cerebral artery, Acom: Anterior communicating artery, IC (R): Right internal carotid artery, IC (L): Left internal carotid artery, Pcom (R): Right posterior communicating artery, Pcom (L): Left posterior communicating artery, P1 (R): Pre-communicating segment of right posterior cerebral artery, P1 (L): Precommunicating segment of left posterior cerebral artery, BA: Basilar artery

References

1. Standring S (chief editor): Gray's Anatomy, In Vascular supply of the brain, 40th Edn. Elsevier, Churchill Livingstone, 2008; 247-248.
2. William AN. Thomas Willis' understanding of cerebrovascular disorders. *J Stroke Cerebrovasc Dis* 2003;12:280-4.
3. Ustun C. Dr. Thomas Willis' famous eponym: the circle of Willis. *Turk J Med Sci* 2004;34:271-4.
4. Puchades-Orts A, Nombela-Gomez M, Ortuno-Pacheco G. Variations in form of circle of Willis: some anatomical and embryologic considerations. *Anat Rec* 1975; 185:119-23.
5. Fisher CM. The circle of Willis: anatomical variations. *Vasc Dis* 1965; 2:99-105.
6. Kim GE, Cho YP, Lim SM. The anatomy of the circle of Willis as predictive factor for intra-operative cerebral ischemia (shunt need) during carotid end arterectomy. *Neurol Res* 2002;24:237-40.
7. Tsutsumi M, Aikawa H, Onizuka M, Kodama T, Nii K, Matsubara S, et al. *Neuroradiology* 2008;50:509-15.
8. Windle BCA. On the arteries forming circle of willis. *J. Anat and Phy.* 1888; 22:289.
9. Fawcett E, Blachford JV. The circle of Willis. An examination of specimens. *J Anat and Physiol.* 1905;40:63-70
10. Alpers BJ, Berry RG. Circle of Willis in cerebral vascular disorders. *Arch Neurol.* 1963;8:398-402.
11. Baptista AG. Studies on arteries of the brain. The anterior cerebral artery. Some anatomic features and their clinical implication. *Neurology* 1963;13:825-835.
12. Gardner E, Gray DJ, O'Rahilly R. Anatomy a regional study of human structure. 3rd edition. W.B. Saunders Company; Philadelphia, Toronto, London: 1969;60-555.
13. Romanes GJ. Cunningham's Textbook of Anatomy. 10th edition. London, UK: Oxford University Press;1964.
14. Perlmutter D, Rhoton AL. Jr. Microsurgical anatomy of the anterior cerebral artery- anterior communicating artery-recurrent artery complex. *J Neurosurg* 1976;45(3):259-72.
15. Riggs HE, Rupp C. Variations in form of circle of Willis. *Arch Neurol.* 1963;8:8-14.
16. Miralles M, Dolz JL, Cotillas J, Aldoma J, Santiso MA, Gimenez A, et al. The role of the circle of Willis in carotid occlusion: Assessment with phase contrast MR angiography and transcranial duplex. *Eur J Vasc Endovasc Surg.* 1995;10:424-30.
17. Martinez F, Spaquuolo E, Calvo-Rubal A, Laza S Sqarbi N, Soria-Vargas VR et. al: Variants of the anterior circle of Willis. Anatomic and angiographic correlation and its implications in the surgery of intracranial aneurysms. *Neurocirugia (Astur).* 2004; 15(6):578-89.
18. Klutmans M, van der Grond J, van Everdingen KJ, Klijn CJ, Kappelle LJ, Vierqever MAB: Cerebral hemodynamics in relation to patterns of collateral flow. *Stroke.* 1999;30(7):1432-9.
19. Emsley HC, Young CA, and White RP: Circle of Willis variation in a complex stroke presentation: a case report. *BMC Neurol.* 2006;6(1):13.
20. Yamauchi H, Kudoh T, Sugimoto K, Takahashi M, Kishibe Y, Okazawa H. Pattern of collaterals, type of infarcts, and haemodynamic impairment in carotid artery occlusion. *J Neurol Neurosurg Psychiatry.* 2004;75:1697-1701.
21. Hendrikse J, van Raamt AF, van der Graaf Y, Mali WP, van der Grond J: Distribution of cerebral blood flow in the circle of willis. *Radiology* 2005;235(1): 184-9.
22. Marcinkevicius E, Liutkus D, Gvazdaitis A, Experience of treatment of moyamoya disease at the clinic of Neurosurgery of Kaunas University of Medicine. *Medicina (Kaunas)* 2006;42(2):130-6.

23. Ujiie H, Liepsch DW, Goetz M, Yamaguchi R, Yonetani H, Takakura K: Hemodynamic study of the anterior communicating artery. *Stroke*. 1996;27(11): 2086-94.