
**ORIGINAL ARTICLE**

**AN INSIGHT INTO THE CIRCLE OF WILLIS, ITS VARIATION IN THE FORMATION AND COMPONENT VESSELS IN THE ADULT HUMAN CADAVERS**

Prasanna LC, MD¹, Andrade LS, PhD¹, Ravichandra P, MD¹, Hosapatna M, MD¹

**ABSTRACT**

**Introduction:** Circle of Willis, is an arterial circle, furnishing arterial supply to most of the brain and meninges. It is formed by the branches of anterior cerebral, middle cerebral and posterior cerebral arteries. However, its formation and a complete arterial circle, differs among individuals.

**Objectives:** In the present study, an attempt has been made to identify those variations and to segregate them into anterior and posterior circulation.

**Methods:** Thirty-two cadaveric brains were used for the study. The circle of Willis was identified and painted for better visualisation and then photographed.

**Results:** The results show that the variations of the anterior circulation exceeded those observed in the posterior circulation.

**Conclusions:** These variations would add on to the previous findings and thus help the neurosurgeons while operating this region.

**Key words:** Anterior cerebral artery; basilar artery; brain, cadaver; circle of Willis; communicating artery; internal carotid; posterior cerebral

**INTRODUCTION**

The optimum functioning of an individual at any given point of time is highly dependent on constant neural activity going on in the brain, which reflects the high metabolic activity and the amount of energy requirement. This enormous demand is met with through a profuse network of blood vessels called the circle of Willis/circulus arteriosus cerebri which serve to convey an uninterrupted supply of nourishment and oxygen to the brain (1).

The circle is formed by the terminal branches of internal carotid and vertebro-basilar arterial systems in the interpeduncular fossa at the base of the brain. Although a complete circular channel almost always exists, one vessel is usually sufficiently narrowed to reduce its role as a collateral route rendering the circle functionally incomplete (2,3). The cerebral and communicating arteries may be absent, hypoplastic, double or even triple (4,5).

Considering the complexity of the developmental pattern in arterial circle, arterial variations occur mostly because of absence of a vessel normally present or abnormal persistence of an embryonic stage of a vessel (6).

Keeping in view the surgical importance of this region, the present study was undertaken to observe the formation of the Circle of Willis, in terms of origin and branching pattern of the component vessels, to evaluate any variations in the component vessels, and to note the frequency of occurrence of different patterns of variant circles (in terms of anterior and posterior circulations). The study not only adds clinically significant unreported variations to the literature but also finds out the prevalence of these variations in the study population.

**MATERIAL AND METHODS**

Thirty-two cadaveric specimens were procured from the routine dissection done for the undergraduate students in the Department of Anatomy, Kasturba Medical College, MAHE, Karnataka, India. After cleaning and carefully removing the overlying meninges without damaging the vessels, the pattern of formation and the existing anatomical variations of the constituent vessels of the circulus arteriosus cerebri was noted. Each specimen was photographed using a digital camera with the variations noticed. The observations were carried out by a single person. Ethical clearance was obtained from the institutional ethical committee (IEC). The number of the ethical clearance is IEC 331/2012.

¹ Department of Anatomy, Kasturba Medical College, Manipal, MAHE, Manipal, Karnataka, India.
*Corresponding Author E-mail: lidibudy@gmail.com
RESULTS
Among the 32 cadaveric specimens observed for the formation of circle of Willis, 15 (46.9%) specimens show the normal description of symmetric polygon architecture without hypoplasia or aplasia. The remaining 17 (53.1%) showed variant formative patterns which ranged from complete absence of vessels to presence of extra vessels.

Among the variant circle, three (9.4%) circles were found to be incomplete of which two were found to be open in the posterior part (Figures 1a and 1b) and one in the anterior part of the circle (Figure 1c).

Twelve specimens (37.5%) were found to have a variant Anterior Communicating Artery (AComA). Absence of the vessel constituted the most common anomaly. Among the six (18.7%) circles with absent AComA (Figure 1c), one was open with no communication between the two anterior cerebral arteries and no clear-cut demarcation between A1 and A2 segments of the anterior cerebral artery (ACA) of each side (Figure 1c).

The remaining five showed fused ACAs of both sides which united at the site of AComA and then redivide to continue their course as A2 segments (Figure 2a).

Next common variation seen was duplication of the AComA, of which three (9.4%) were observed (Figure 2b). The other variations seen of the AComA were network type of arterial vessels in the region of AComA as seen in one case. Y-shaped anterior circulation (Figure 1b) was seen in two cases one on each side.

In this type of anterior circulation, the AComA and the A2 segments of both sides seem to be fed by A1 segment of any one side either left or right. Occurrence of aneurysm was seen in one specimen (3.125%) in the region of junction between ACA and AComA. None of the observed specimens showed hypoplasia (<0.5mm diameter).

Among the cadaveric specimens, five (15.6%) variant types of ACA were found. Unilateral hypoplastic (Figure 1b and 3a), fenestrated (Figure 3b) and duplicated (Figure 3c) types were seen in one (3.1%) specimen each. Two specimens showed asymmetry of the ACAs in terms of diameter on the left and right sides. Anterior cerebral arteries arising from the internal carotid arteries of the respective sides are divided into a precommunicating part (designated as A1) and a post communicating part (designated A2) by the presence of an AComA. The A1 segments of the two sides along with the AComA constitute the anterior circulation.
**Figure 2:** Circle of Willis showing different types of AComA

Fig 2a: Absent AComA with fused ACAs in its place indicated by white arrow

Fig 2b: White arrows indicate duplicated AComA

**Figure 3:** Variations observed in ACA

Fig. 3a: White arrow indicates hypoplastic A1-R segment

Fig. 3b: Fenestrated A1-L segment indicated by the white arrow

Fig. 3c: Duplicated A1-R segment indicated by white arrow

Only eight (25%) specimens showed variations of the PComAs. Among the variant PComAs, four (12.5%) were found to be unilateral (Fig. 1a) and the remaining four bilateral (Figure 1b). Among the unilateral variations, one was completely absent (Figure 1a) and one hypoplastic (Figure 4a), two were found to be network type (Figure 4b). Among the bilateral variations, one was completely absent (Figure 1b) with an open circle of Willis.

**Figure 4:** Types of PComAs observed

Fig. 4a: White arrows indicate hypoplastic PComA

Fig. 4b: Network type PCom-R indicated by white arrow

Fig. 4c: Bilateral fetal type of PComA indicated by white arrows
Bilateral absence of PComAs was seen in two cases. Only 2 (6.3%) out of the 32 studied cadaveric specimens showed variations. Both were unilateral and presented with complete absence (Figure 1a) of the vessel either in the right or left side. One of the two variations were the incomplete circle of Willis, awhile the other circle was completed by direct communication of the PComA with the basilar artery in place of absent PCA.

DISCUSSION

The cerebral arterial circle of Willis has shown to have a wide range of variations from 4.6% to 72.2% (4,5,7,8). It could be due to the sheer number of vessels involved in its formation or different methods of assessing its formation, and disparate nomenclature given to component vessels by different authors and hence an attempt has been made to describe the variations in terms of anterior and posterior circulations.

Previous authors have found that the anomalies of the circle are more common in the posterior part as observed in Table 1.

Table 1: comparison of Posterior part of circle of willis between the previous authors and present study

<table>
<thead>
<tr>
<th>Author</th>
<th>Variations - anterior part</th>
<th>Variations - posterior part</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saeki et al (8)</td>
<td>-</td>
<td>49%</td>
</tr>
<tr>
<td>Jain et al (9)</td>
<td>29.6%</td>
<td>51.4%</td>
</tr>
<tr>
<td>Van (10)</td>
<td>29%</td>
<td>81%</td>
</tr>
<tr>
<td>Gunal (5)</td>
<td>40%</td>
<td>50%</td>
</tr>
<tr>
<td>Present study-cadaveric</td>
<td>25.0%</td>
<td>15.6%</td>
</tr>
</tbody>
</table>

Circulus Arteriosus: Anterior Part
AComA-ACA Complex:

1. Anterior Communicating Artery:
In the present study, among the total AComA-ACA complexes, AComA was present and single in 62.5% of the cadaveric specimens. Double and fenestrated vessel was seen in 9.3% and 3.3% cases. The present study also showed an equal proportion of network type (3.3%) and Y-shaped (6.6%) AComA. Windle reported AComA absent in 3% and double in 3% of the cases (6).

Fawcett & Blackford reported that the artery was single in 92.1%, absent in 0.14% and double in 7.2% of the cases (3) Vare & Bansal studies showed that AComA was absent in 1.1%; double in 10.28% and AComA-ACA complex was H-shaped in 1.1% (11). Luzsa illustrated that the artery was absent in 0.3% of the cases and the occurrence of a median trunk in 1.2% of the cases (12). Osborn also reported the presence of more common azygous ACA which ranges from 0.2-4% (13).

In the present study, the pre-communicating anterior cerebral artery (A1 segment) was present in all the cadaveric specimens and in 96.7% of the specimens. It was found to be hypoplastic in one specimen (3.1%), fenestrated and with an asymmetric A1 segment in two specimens (6.3%) each. Windle reports the absence of the artery in 1% (6). Luzsa report states the absence of the artery in 0.7 - 11% and hypoplasia in 8 - 15% (12). Osborn states the absence of the A1 segment in 1 - 2% (13). Macchi and Stephen & John also reports 2% hypoplasia of the A1 segment (14,7). Kane reports 9.61% each of aplasia & hypoplasia of A1 segment (15).

3. Posterior communicating artery (PComA):
In the present study, the PComA was found to be absent in approximately 6% of the cases seen both in dissected specimens and radiological images studied. Hypoplastic PComA was seen in one (3.1%) of the specimens. Two (6.3%) specimens showed network type PComA.
PCoMA arising from basilar artery was seen in 3.3%. Windle reported the vessel to be absent in 15% of the cases; both PCoMA were absent in 1.5%; 4.5% involving the right and 6.5% on left side and the vessel was hypoplastic in 3.5% of the cases. Fawcett & Blackford reported that the vessel was absent in 0.4% on both sides; absent on the right side in 1.8% and on the left side in 1.4% of the cases.

Riggs mentions bilateral hypoplasia of PCoMA in 11% and unilateral hypoplasia of PCoMA in 6% of the cases. Jain reports indicated that the artery presented maximum anomalies in 50% of the cases. Stephen & John (1991) mentions the absence of posterior communicating artery on one side in 23% cases. In Macchi et al., hypoplasia of the PCoMAs was noted in 21% of the cases. Osborn mentions hypoplasia or absent PCoMA in 25-33% of the cases. Merkola et al., reported absence of PCoMA in 46% of cases. Eftekhar et al., reports the absence of PCoMA on both sides in 3%; right side in 4% and left side in 3% of the cases. Bilateral hypoplasia of PCoMA was seen in 33%; unilateral hypoplasia of PCoMA was observed in 27% out which, right side was 16% and left side was in 11% of the cases.

5. Posterior Cerebral Artery (P1 Segment):

In the present study, the pre-communicating artery (P1 Segment) was present in 30 out of the 32 specimens (93.8%) and absent in two cases (6.3%). It was hypoplastic in two cases (6.3%), one on each side. The artery originated from the terminal bifurcation of the basilar artery in 26 (86.7%) cases. In four cases (13.3%) the artery originated from Internal carotid artery (ICA), two cases on each side accounting for 6.7% each.

Windle study shows an anomalous P1 segment in 13.5%; PCA originating from ICA on the right in 5.5% of the cases and left in 2% of the cases. Fawcett & Blackford study indicates that the artery had an abnormal origin, arising from ICA in 0.14%; 0.9% on the right and 0.57% on the left. Riggs' study showed that the unilateral hypoplasia of P1 present was present in 16% of the cases. Vare & Bansal studies show 25% P1 segment anomalies having an abnormal origin from ICA and 5.7% of the cases had both P1 segments arising from the ICA; 13.7% on the right and 5.7% on the left. Stephen & John illustrated 14% of the posterior cerebral arteries arising from Internal carotid with P1 being hypoplastic on the same side. Macchi showed 2% of the cases had P1 segment arising from the ICA. Osborn illustrations show 15-22% hypoplastic P1 segment.

Conclusion:
It is noteworthy that most of the above described variations of the component arteries do not occur in isolation but are seen in various permutations and combinations attributing to a highly versatile range of types circles of Willis. The communicating arteries remained the most important vessels which helped maintain collateral circulation.

Competing interest:
The authors declare that this manuscript was approved by all authors in its current form and that no competing interest exists.

REFERENCES

2. DeSilva KRD; Silva R; Amaratunga D; Gunasekera WSL; Jayasekera RW. Types of the cerebral arterial circle (circle of Willis) in a Sri Lankan Population. BMC Neurology 2011; 11:5.


15. Kane AG; Dillon W; Barkovich AJ; Norman D; Dowd CF; Kane TT. Reduced caliber of the internal carotid artery: a normal finding with ipsilateral absence or hypoplasia of the A1 segment. American Journal of Neuroradiology 1996; 17(7): 1295-1301.


18. Eftekhar B; Dadmehr M; Ansari S; Ghodsi M; Nazparvar B; Ketabchi E. 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.