International Journal of Anatomy and Research,
Int J Anat Res 2014, Vol 2(2):344-53. ISSN 2321- 4287

Original Article

CIRCLE OF WILLIS: VARIANT FORMS AND THEIR EMBRYOLOGY USING GROSS DISSECTION AND MAGNETIC RESONANCE ANGIOGRAPHY

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ABSTRACT

Background: The circle of Willis is a large arterial anastomotic ring present at the base of the brain uniting the internal carotid and the vertebrobasilar systems. Branches from the internal carotid and vertebral arteries anastomose to form an arterial circle in the basal cisterns and then distribute to supply the brain. The anatomy of the circle is known to vary significantly; the vessels may be absent or sufficiently narrowed altering the hemodynamics of the circle of Willis and affecting its role as a collateral route. These variant forms can be correlated to their phylogeny and embryology. Prior knowledge of these variant forms is important in pathologies and treatment (e.g. parent artery occlusion for carotid aneurysms) resulting occlusion of carotid and vertebral arteries.

Context and purpose: Our study was undertaken to observe and compare the morphology of circle of Willis using two entirely different methods; gross dissection (GD) and Magnetic resonance angiography (MRA) and to correlate the variant patterns encountered with the possible underlying developmental events. Gross dissection was carried out in 70 human cadavers and equal numbers of MRA's of healthy individuals were studied retrospectively.

Results: Only 31 cases (22.14%) presented with a complete circle of Willis, out of which 14 (20%) were cadaveric specimen and 17 (24.18%) were in MRA group. Unilateral hypoplastic posterior communicating artery was the most common variation observed in our study (19.28%).

Conclusions: The wide variation in completeness of the circle of Willis in general population is similar to earlier observations. Review of phylogeny and embryology makes us familiar with variant forms which would be otherwise difficult to recognize and may be misinterpreted. MRA and gross dissection findings despite certain variations are comparable.

KEYWORDS: Internal carotid artery, vertebral artery, Collateral flow.

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Access this Article online

Quick Response code

Web site: International Journal of Anatomy and Research
ISSN 2321-4287
www.ijmhr.org/ijar.htm

Received: 10 April 2014
Peer Review: 10 April 2014 Published (O):31 May 2014
Accepted: 02 May 2014 Published (P):30 June 2014

BACKGROUND

Cerebrovascular accidents are one of the leading causes of death and disability throughout the world. The clinical manifestation of these cerebrovascular accidents reflects the area perfused by the cerebral vessels affected. However, the situation becomes more complicated with the presence or absence of adequate collateral blood flow provided by, an arterial ring of anastomosis present at the base of brain

popularly known as the circle of Willis. The circle of Willis, unites the internal carotid and vertebrobasilar systems and is formed by anastomosis between the internal carotid, precommunicating part (A1) of anterior cerebral, anterior communicating, precommunicating part (P1) of posterior cerebral and posterior communicating arteries (fig. 1). The collateral flow thus depends on the pattern and caliber of these branches forming the circle, which is known to vary. Some of these variations arise early during vasculogenesis by alteration of vascular construction programme, due to some triggering factors whereas; some of them represent the mainstream of evolution [1].

Variations can be described as patterns which represent the mainstream of evolution of a system whereas, an anomaly represents increased rigidity of the system at its edges which requires a minimal constrain to reveal its limited flexibility [1]. Absence of a consistent relationship between the size of the vessels and its territory, encountered in the variant forms of the circle can be interpreted as to be an abnormal one only during an occluding episode of the feeders, whereas in normal circumstances embryological events like territorial transfer and sharing of territories between the adjacent developing vessels should results in normal circulation [1]. The acquaintance of these variations further becomes important as without it diagnostic procedures may be misinterpreted or a neurosurgical procedure may become complicated. The variations determine the angiographic filling pattern and their knowledge enables distinction between hypoplasia and spasm [2]. During this study gross cadaveric dissection was carried out to observe the variations and later on for a further detail study Magnetic Resonance Angiography was studied retrospectively in living population.

Magnetic Resonance angiography (MRA) enables evaluation of the intracranial vessels without the need for invasive procedures like catheter angiography, avoiding small but definite risk of embolism, complications like psuedoaneurysms, contrast associated reactions and vascular dissections [3]. Although MRA can not reveal any arteries invisible in conventional angiography, it provides a specificity of 100%, sensitivity of 89.2% for the anterior and 81.3% for the posterior communicating arteries and 100% for the anterior, middle and posterior cerebral arteries [4]. In view of such high specificity and sensitivity we undertake this retrospective study on the various morphological variations of the circle of Willis. Three methods for performing MRA are available which includes Time of flight (TOF), phase contrast (PC), and Black blood Imaging. We used TOF in the present study.

Relevant Phylogeny and Embryology

The interpretation of common variations in the circle of Willis may be linked with their development as most of the branches are derived from the internal carotid, formed progressively from the third arch artery [5]. The large arteries supplying the brain are phylogenetically more recent and are still in a continuous process of evolution in accordance with its developing territory. New variants can be expected anytime and can only be recognized if we are familiar with the prior disposition of the vascular tree in lower animals. Similarly an embryonic vessel never disappears completely unless its territory does so [1].

In lower group of animals the internal carotid artery (ICA) or its equivalent branches into two main intracranial trunks; caudal and rostral (figure 2). The caudal division travels dorsally and branches into tectal and cerbellar arteries to constitute the basilar trunk and supply the posterior cranial fossa. The rostral division divides into medial and lateral branches. The medial branch supplies the olfactory nerve and rhinencephalic structures which can be considered as the primitive expression of anterior cerebral artery (ACA) complex in man, which in case of fishes still remain separate branches. Gillilan mentioned anastomosis of the medial olfactory arteries (future ACA) seen in case of reptiles as representation of anterior communicating artery complex in man [6]. The lateral branch follows the lateral root of olfactory nerve and can be considered as representative of future artery of Heubner and anterior choroidal artery.

The middle cerebral artery (MCA) is not an individual large vessel but it represents a series

of small anastomotic vessels from the lateral striate group. It develops after the "recurrent artery of Heubner" another branch from the ACA complex. They both share territories and variations, and are in haemodynamic balance with respect to their deep territories. The anterior choroidal artery comes up as a branch from the ACA and most of its territories will be transformed to the tectal artery of the caudal division of the ICA, later to become the Posterior cerebral artery (PCA) [1]. The PCA prolongs the caudal division of the ICA. It supplies only the tectum; it becomes the true PCA when it annexes most of the cortical territories from the anterior choroidal artery. The basilar artery results from a cranio-caudal fusion of the posterior division of the ICA, so that the posterior communicating artery (PCoA), the P1 segment of PCA and upper basilar system, distal to the origin of trigeminal artery, should be all considered as a single system; the caudal division of ICA [1].

MATERIALS AND METHODS

In the present study the circle of Willis (COW) was studied by gross dissection in 70 cadaveric specimens and by Magnetic resonance angiography on equal number of living individuals. The study includes subjects of both sexes which belong to different communities in the Northeast India. In both studies the anterior cerebral artery (ACA) were studied under; A1 segment, defined as a part of ACA from its origin at the internal carotid artery bifurcation till its junction with the anterior communicating artery and A2 segment was defined as the course distal to it till the origin of pericallosal and callosomarginal arteries. Similarly in the posterior cerebral arteries (PCA), P1 segment originates at basilar bifurcation up to the junction with the posterior communicating artery and the P2 segment as the portion of PCA beyond it within the perimesencephalic cistern.

A. Gross dissection:

Gross dissection was carried out in the department of Anatomy, Gauhati Medical College, Assam, from July 2009 to September 2010. A total number of 70 brain specimens without obvious pathological changes and decomposition were collected from cadavers, for a period from October 2009- September 2010 in the department of Anatomy and unclaimed dead bodies in the department of Forensic Medicine, Gauhati Medical College, Assam, India.

Dissection and processing

The brains from the cadavers with their arteries intact were gently taken out by detaching the falx cerebri and the ventral surfaces were cleared. The COW and its branches were cleared off from the overlying meningeal coverings and any adhesions wherever present, to expose them distinctly. The specimens were preserved in 10% formalin and dissected further at a convenient time later. Wherever needed red fabric colour was used to enhance contrast and photographs were taken. Relevant data were recorded and compared.

B. Magnetic Resonance Angiography:

The Magnetic resonance angiographies (MRA) of 70 living individuals were studied retrospectively in the Department of Radiology, NEIGRIHMS, Shillong, Meghalaya, India, for a period from 10th April 2012 to 22nd November 2012. All patients underwent 3D time of flight MR angiography (3D TOF MRA) imaging using 1.5 tesla MRI scanner (Avanto, Siemens, Germany). Following imaging parameters were used repetition time/ echo time 23/7.0, flip angle 25 degrees, slice thickness 0.7 mm, number of slice 44/slab, number of slabs 4, slice overlap 25%, flow direction feet to head with 40 mm saturation at the head end, field of view 180 x 158 and 256 matrix size. Reconstructions were done in Syngo MR Workplace using 3D maximum intensity projection or MIP and Volume rendering technique or VRT. The data collected from either method was compared. The individual variant forms were correlated with the possible underlying embryological event and to compare it with earlier established findings.

RESULTS

In the present study the circle of Willis were studied by two different methods in two different groups; in 70 human cadaveric specimens by gross dissection and in 70 living individuals by Magnetic resonance angiography (MRA). In order to be consistent with the previous works, hypoplastic vessels were defined to be those with external diameters less than one millime-

ter (1mm). Out of the total 140 cases, only 31 cases (22.14%) presented with a complete (classic) circle of Willis (fig.3). A classic circle of Willis was found in 14 (20%) of 70 cadaveric specimens and in 17 (24.18%) 70 MRA's studied. (fig.1). For a comprehensive understanding of the different patterns of the variations observed during our study, a schematic representation for the variant forms of circle of circle of Willis in anterior and posterior part is shown in Fig. 3 and Fig. 4 respectively.

Table 3: Incidence of Normal Pattern of Circle of Willis.

Study	Technique	Brains examined	Normal pattern (%)
Windle [12]	Gross dissection	200	38%
Blackburn [13]	Gross dissection	220	29.50%
Von Mitterwallner [14]	Gross dissection	360	27.20%
Riggs& Rupp [10]	Gross dissection	994	21%
A. Puchades-Orts, et al. [15]	Gross dissection	62	13%
P.N Jain et al. [11]	Gross dissection	144	19.45%
Krabbe- Hartkamp et al. [16]	MRI	150	42%
Parthapratim P. et.al. [9]	Gross Specimen	76	7.89%
Haripriya & Melani [17]	MRI	50	32%
Our study	**Gross Specimen + MRI**	**140**	**22.14%**

Type of Variation (in percentage)													
Group	a	b	c	d	e	f	g	h	I	j	k	l	Total
GD	0	7.1	2.8	8.6	0	1.4	0	1.4	8.6	5.7	5.7	5.7	**47.14%**
MRA	4.3	11.4	1.4	4.3	1.4	1.4	1.4	8.6	7.1	4.3	0	2.8	**48.47%**
GD+MRA	2.1	9.2	2.1	6.4	0.7	1.4	0.7	5	7.8	5	2.8	4.3	**47.80%**

Table1: Frequency of different variations in anterior part of the "circle of Willis" according to the type (classification) shown in Fig. 4.

These labeled representations of the variant forms were used for classifying and numbering the variations in table 1 and table 2.

Table 2: Frequency of different variations in posterior part of the "circle of Willis" according to the type (classification) shown in Fig.5.

Type of Variation (in percentage)									
Group	a	B	c	d	e	f	g	h	Total
GD	11.4	1.4	5.7	2.8	18.6	14.3	1.4	0	**55.7**
MRA	12.8	7.1	7.1	8.6	20	11.4	1.4	7.1	**75.7**
GD+MRA	12.1	4.3	6.4	5.7	19.3	12.8	1.4	3.6	**65.7**

Fig. 1

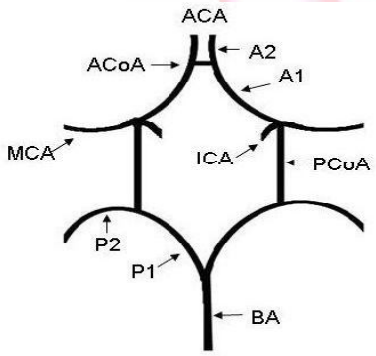

Fig. 1: Schematic representation (vessels forming the circle of Willis) of the anterior part of the circle formed by the pre-communicating segments (A1) of the right and left anterior cerebral arteries (ACA) and an anterior communicating artery (ACoA) between them. The posterior part of the circle formed by the pre-communicating segments (P1) of the right and left posterior cerebral arteries (PCA), together with the right and left posterior communicating arteries (PCoA). The right and left PCoAs originate from the right and left internal carotid arteries (ICAs) The A2 and P2 segments are the post - communicating portions of the anterior and posterior cerebral arteries respectively. BA: basilar artery, MCA: middle cerebral artery.

Fig. 2

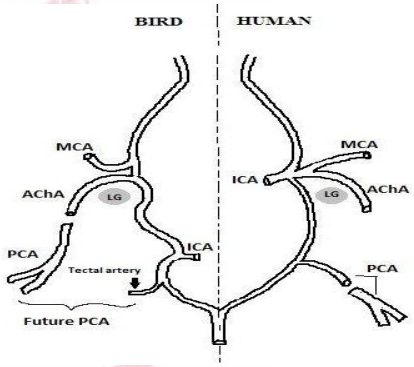

Fig. 2: The position of anterior choroidal artery (AChA) in man shows its complete homology with the representation of posterior artery in bird. Both the arteries can be compared with each other and with their position in respect to the lateral geniculate body (LGB) confirming their identity. The pseudo-posterior cerebral artery and the tectal artery (in birds) together constitute the components of posterior cerebral artery in man.

Fig. 3

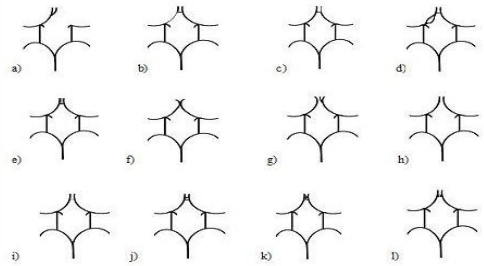

Fig. 3: Schematic diagram to show the variations in the circle of Willis in the anterior circulation, i.e. anterior cerebral artery (ACA) & anterior communicating artery (ACoA): a) absent A1 segment with both ACA arising from the opposite ICA. b) Hypoplastic A1, c) Hypoplastic A2. d) Fenestration of A1. e) Median artery of Corpus callosum. f) Fused ACA with absent ACoA. g) Absent ACoA with early origin of the Callosomarginal branch. h) Absent ACoA. i) Hypoplastic ACoA. j) Double ACoA. k) Plexiform ACoA. l) Fenestrated ACoA. ICA: Internal carotid artery.

Fig. 4

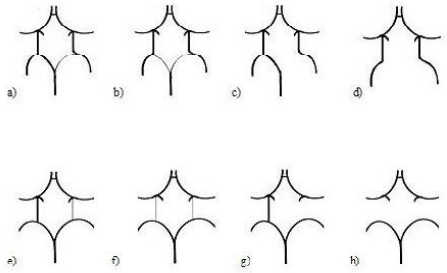

Fig. 4: Schematic diagram to show the variation in the posterior portion of the "circle of Willis" i.e., Posterior cerebral artery (PCA) & Posterior communicating artery (PCoA): a) Hypoplastic P1 (U/L) with partial Fetal PCoA b) Hypoplastic P1 (B/L) with partial Fetal PCoA. c) Absent P1 (U/L) with complete Fetal PCoA d) Absent P1 (B/L) with complete Fetal PCoA. e) Hypoplastic PCoA (U/L) f) Hypoplastic PCoA (B/L) g) Aplastic PCoA (U/L) h) Aplastic PCoA (B/L).

Fig. 5

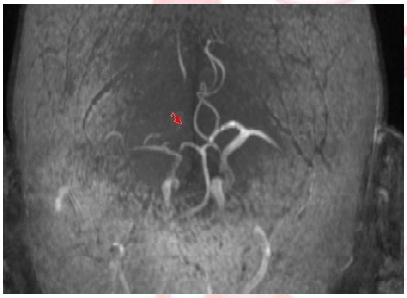

Fig. 5:. Photograph of MRA of circle of Willis showing absence of A-1 segment of anterior cerebral artery of left side (arrow) with both the anterior cerebral artery arising from the right internal carotid artery.

Fig. 6

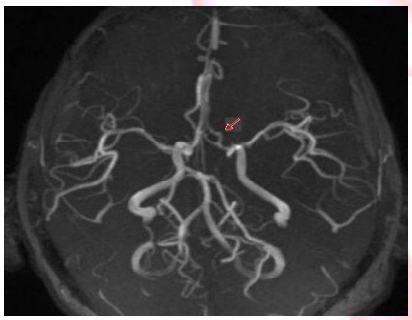

Fig. 6: Photograph of MRA of circle of Willis showing fenestrated A-1 segment of anterior cerebral artery (red arrow) with aplastic posterior communicating artery over right side.

Fig. 7

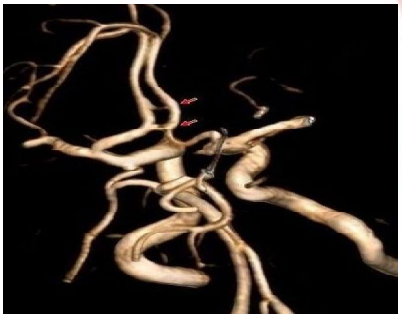

Fig. 7: Photograph of MRA (reconstructed) of circle of Willis showing double anterior communicating artery (red arrows) with hypoplastic right A-1 segment of anteriorcommunicating artery.

Fig. 8

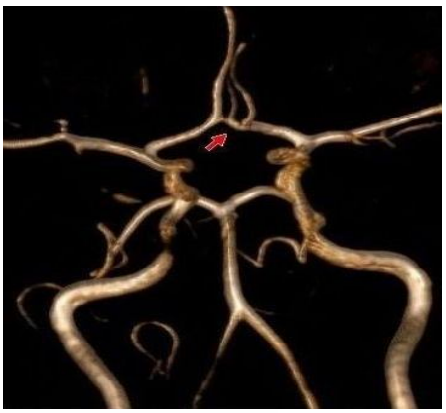

Fig. 8: Photograph of MRA (reconstructed) of circle of Willis showing median artery of corpus callosum arising from the anterior communicating artery (arrow).

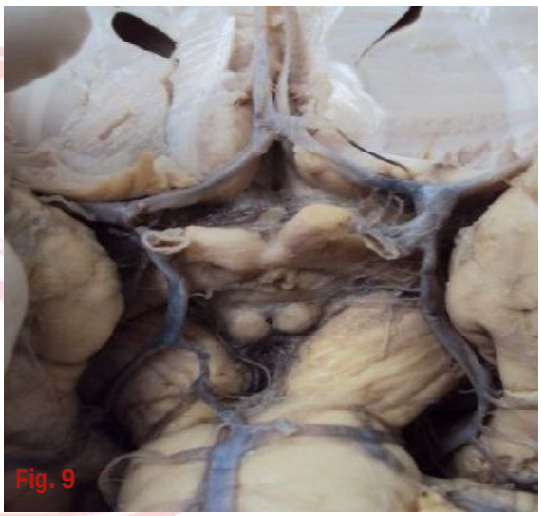

Fig. 9

Fig. 9: Photograph of gross dissection of "circle of Willis" showing fusion of both the anterior cerebral artery with absence of anterior communicating artery and complete fetal posterior cerebral artery on the left side.

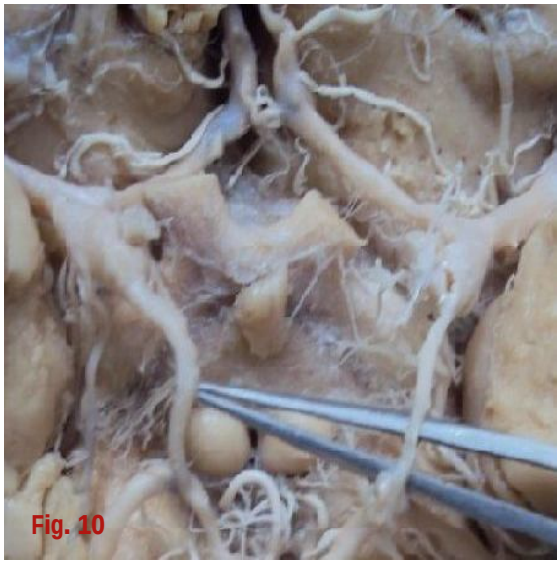

Fig. 10

Fig. 10: Photograph of gross dissection of "circle of Willis" showing plexiform anterior communicating artery.

Variations in the anterior part of circle of Willis:

The variant forms of vessels in the anterior part of the circle of Willis (anterior cerebral artery & anterior communicating artery) observed by gross dissection and MRA are 33 (47.14%) and 34 (48.57%) respectively (Table 1). Variation in the anterior cerebral artery (ACA) was found in 20% of the cadaveric specimens and in 25.71% MRA's whereas, anterior communicating artery (ACoA) presented with variant forms in 27.14% of cadaveric specimens and 22.85% cases in MRA. The most common variant found in anterior circulation was hypoplastic A1 segment (9.28%).

Variations in the posterior part of circle of Willis:

15 cadaveric specimens (21.42%) and 20 cases (28.57%) in MRA presented with variant forms of Posterior cerebral artery (PCA). 12.85% cadaveric specimens and 20% MRA cases, presented with fetal posterior communicating artery (PCoA) with hypoplastic P1 whereas, 8.57% cadaveric specimens and15.71% MRA cases presented with aplastic P1 with complete Fetal PCoA. The variations in the PCoA were found in 24 (34.28%) cadaveric specimens and 28 (40%) cases in MRA. Hypoplastic PCoA was found in 32.85% cases, bilateral in 18.57% and unilateral in 14.28% cadaveric specimens and in 31.42% cases, with unilateral 20% and bilateral 11.42% cases in MRA. Absent PCoA was seen in 1.42% cadaveric specimen and 8.57% cases in MRA. Unilateral hypoplastic PCoA was found to be the most common variant, in 19.28 % observations out of 140 (gross- 18.57%, & MRA-20%) as shown in table 2.

DISCUSSION

In the present study the anatomical variants of the circle of Willis (COW) were studied both by gross dissection (GD) and MRA technique. The possible underlying development factors for each variant forms observed were identified. Observations made by both the procedure were compared with each other and also with the established findings of the other authors. These variations encountered in the COW may be a result of triggers. Triggers are phenomena that may alter the vascular construction programmed at any stage without necessarily producing an immediately detectable morphological abnormality. The trigger alters the programme or its execution either in a transient or in a permanent way. If it doesn't stimulate repair or apoptosis; then the programme alteration can be transmitted to the next cell generation [1]. The congenital disposition of these vessels represents the earlier natural adaptation of the territories to their arterial supply. Territories and arteries grow together in harmony because of constant interaction. The effects of a given vascular insult vary according to the pre-existing congenital arrangement. An acquired constrain factor upsetting hemodynamic balance of a given area will lead to a hemodynamically unstable situation leading to a clinically expressed disorder. Fusion of vessels may be a result of an event (or trigger) which will testify to the partial or transient interruption of the maturation phase during vasculogenesis [1] or it may be correlated phylogenetically, as similar pattern of a united A2 segment is normal in monkeys (Macaca mulatta) [7]. This type of incomplete fusion or fenestration may be result of an incomplete maturation process of arterial wall rather than fortuitous. The lack of cell selection that such patterns imply may preserve weaker endothelial cells which may later reveal arterial aneurysms. This area of COW represents an area of hemodynamic stress and immature situations which are vulnerable. Absent, multiple or plexiform vessels suggest incomplete cell selection and apoptosis during vasculogenesis [1]. If the trigger that lead to altered vasculogenesis is not corrected to stimulate repair or apoptosis, then the programme alteration can be transmitted which may appear to be a racial or regional pattern as described by previous authors. Congenital hypoplastic vessels are a common finding which should be differentiated from an acquired one. When narrowing is segmental, congenital hypoplasia is unlikely. Congenital situations are usually harmonius and one should expect a congenital hypoplastic vessel to follow its ontogenetic branching [1].

The normal classical pattern of COW as observed by different authors has wide range of variations from as high as 42% [8] to as low as 7.89 % [9] (Table 3). Our study is the first to compare both GD and MRA to study the variations in the COW.

In our study of 140 subjects, only 22.14% were of normal pattern (20% in GD and 24.18% in MRA) which is similar to the observations made by Riggs & Rupp et al. and P.N. Jain et al. [10, 11].

A functional vessel may prevent a neurological damage during an occlusive cerebrovascular episode [18, 19, 20]. The role of these arteries as a collateral route in classic COW or as a risk factor in case of variant forms cannot be established by the present study and remains a mere speculation. However, vascular aplasia remains a definite risk factor to develop a neurological insult. Complete fusion of both anterior cerebral arteries (ACA) somewhat distal to the commencement of median longitudinal fissure has been described earlier [12]. In the present study complete fusion of the ACA was not observed, however, a focal fusion of both ACA's forming a single arterial trunk which re-divides into right and left branches was observed, one case each in MRA and GD. This type of focal fusion of the ACA's has been previously reported [9]. The Anterior communicating artery (ACoA) is recognized as the most important collateral channel during severe internal carotid artery (ICA) stenosis [21, 22, 23]. The impairment of the perforating branches of the AcoA is reported to cause memory disturbances similar to that seen in Korsakoff's syndrome [24]. Aplasia of the A1 was seen earlier in many cases [10, 12, 14, 15]. It was observed earlier that if both ACA's fill only from one side, it is usually from the left one [25]. This observation was later confirmed by the arteriographic study which also showed that the left carotid was eight times more likely to be the site of origin of both ACA's [26]. In our study, aplasia of the A1 was found in 3 cases of MRA, 2 on the right and one on the left side and none in the cadaveric specimens. String like or hypoplastic A1 segment as reported earlier [15, 16, 17], were seen in 5 cadaveric specimens (3 right & 2 left) and 8 MRA's (3 right & 5 left). Presence of the median ACA was first reported by Windle, P.N. Jain et al and Krabbe-Hartkamp in 4.5%, 3.47% and 1.33% cases respectively [11, 12, 16]. According to Padget [27] this artery appears to be formed from lower group arterial plexus, similar to the formation of ACA. In our study we did not find any median ACA. Buttonhole formation or fenestrated ACA is characteristics of rats, which was described by Brown in his study of adult albino rats [28]. In our study 9 cases (6 in GD and 3 in MRA) of buttonhole formation in ACA was observed. This can be explained as an evolutionary throw back. Experimental studies have shown that the ACoA does not allow any mingling of blood between the two ACA's, however it does acts as a bypass channel if one ICA is occluded [29]. The frequency of duplication and triplication of the ACoA appear to be racial [30, 31]. Windle [12] found duplication of the ACoA in 22 out of 200 cases. It was later reported by almost all the authors examining the COW. Triplication of the ACA's was reported in one case by P.N. Jain et al. [11]. Absence of the ACoA's was reported earlier by several authors [13, 14]. Hartkamp [8] reported absence of ACoA in 1.8% of cases. Plexiform ACoA was first reported by Windle [12] in 0.5% cases. Puchades-Orts et al. [15] found plexiform ACoA in 4.83% of his cases and explained it as persistence of the embryonic form. In our study duplication of ACoA were observed in a total of 7 (5%) cases (GD, MRA), plexiform type in 4 (5.71%) cadaveric specimens, fenestration in 6 cases (GD, MRA) and in 7 (5%) cases the ACoA was absent. The most frequent variation observed in posterior cerebral artery (PCA) was the PCA originating from the ICA as a result of persistent primitive embryological pattern. The name Fetal PCA came due to persistence of the fetal pattern [25]. As the PCA has its embryonic origin from the ICA and only during the late embryonic and early post-fetal life does the distal part of the PCA begin to gain much of the blood flow from the basilar artery (BA) system through its junction with the distal branches of the BA (the mesencephalic arteries) commonly called P1. Kaplan suggested that the proximal portion of the so called PCA's, which arises from the BA, should be designated the mesencephalic artery and only the distal portion beyond the junction with the posterior communicating artery (PCoA) should be referred to as the PCA [32]. This terminology however merits consideration since it should help to simplify clinical description of patterns of blood flow. Krabbe-Hartkamp [16] and P.N. Jain [11] observed fetal type of PCA in 32% and 16.66% of their specimens respectively.

It was observed that patients with fetal-type PCA could be more prone to develop vascular insufficiency [33]. In our study complete fetal PCA was seen in 16.42% of subjects whereas, incomplete fetal PCA was seen 12.14%. PCA hypoplasia could be a contributory risk factor for ischemic stroke, even in absence of ICA occlusion [34]. In our study 9 cadaveric specimens and 14 MRA's presented with hypoplastic P1 with partial fetal PCA whereas, 6 cadaveric specimens and 11 MRA's presented with absent P1 with complete PCA arising from ICA. Anomalies of the PCoA have a great significance since it forms a link between two major arterial systems – the internal carotid and the vertebrobasilar circuit. A hypoplastic PCoA may be a risk factor for developing neurological deficit in patients with ICA occlusion [19]. Windle [12] found the PCoA's small in 2.33% and absent in 1.5% of specimens. He observed anomalies in 49% and described the origin of the PCA's from one or both ICA's as the most common variant. Mitchell [35] described 16 patients with anomalies in the PCoA's, with 9 of them absent. P.N. Jain et al. [11] found 20 absent and 26 hypoplastic PCoA and Partopratim et al. [9] found 44 hypoplastic forms of PCoA's. In our study out of 140 cases, the most common variant observed was unilateral hypoplastic PCoA which was seen in a total of 27 (13 GD & 14 MRA) cases. This observation was similar with previous authors [8, 11, 15]. Bilateral hypoplastic PCoA was seen in a total of 18 (10 GD & 8 MRA) cases. The PCoA was unilaterally absent in 2 cases and bilaterally in 5 cases. The incidence of aneurysms of the circle of Willis reported in published literature is 0.25% to 4.9%. In our study a single aneurysm in the A1 segment of the right ACA (1.42%) was observed.

CONCLUSION

The present study is based on the analysis made from the gross dissection in 70 post mortem specimens of brain and in 70 MRA's. In a fetal-type posterior cerebral artery (PCA) there is an embryonic derivation of the PCA from the internal carotid artery (ICA). In complete fetal-type PCA, the possibility for collateral circulation to develop between the anterior and posterior part of the cerebral circulation by way of leptomeningeal vessels is impossible as the vascularization of the PCA flow territory is completely dependent on the ICA. Whether this is a risk factor for stroke should be subject of further investigation. Both MRA and cadaveric specimens show that morphological variations are quite frequent in the circle of Willis (COW) in human beings. The foundation of arterial COW lies in vessels which are formed in the early stages of fetal life some of these vessels succumb to involution while others are evolving to supply blood to the developing structures. However, functional changes as well as the anatomical factors may influence the effectiveness of anastomosis. The COW is unquestionably important as a potential source of anastomotic blood flow. Segments of the COW which are narrow or string-like, or even absent are a result of agenesis or involution during embryonic development. Because of this variability, the clinical manifestation of occlusion of the vertebral or carotid arteries may vary considerably from one individual to another, and the effectiveness of collateral circulation may be greatly influenced. Our study comprising gross dissection and MRA is first of its kind to study giving an indirect opportunity to compare the variations of the COW in patient who have decreased due to various causes and in living persons. Postmortem dissections have some disadvantages. There may be a minor change in the diameter of the vessels, the diameter of 'hypoplastic' vessels may increase with time and exceed the 1 mm criteria. Degree of preservation of each cadaver may vary leading to subtle differences. Since the results cannot be directly compared with the angiography, as in angiography we can measure only the internal diameter and that too in living subjects. Preparing acrylic cast of the blood vessels can be more accurate but is time consuming and costly. In TOF-MRA limitations include dependence on amount and direction of flow as well as the exact technique employed. Therefore smaller arteries with very little flow might not be detected and leading to interpretation as an aplastic artery instead of it being actually hypoplastic. Despite these limitations we were able to document several variations during this study, by both methods. Further studies based on quantitative measurements of the luminal diameters and flow of the

segments and so forth should be carried out for a more specific detail on the clinical manifestation of these variations.

LIST OF ABBREVIATIONS:

COW: Circle of Willis
ICA: Internal carotid artery
VA: Vertebral artery
BA: Basilar artery
ACA: Anterior cerebral artery
ACoA: Anterior communicating artery
PCoA: Posterior communicating artery
PCA: Posterior cerebral artery
MCA: Middle cerebral artery
MRA: Magnetic resonance angiography
TOF- MRA: Time of flight – Magnetic resonance angioigraphy
GD: Gross dissection

Conflicts of Interests: None

Acknowledgements:

1. Prof. (Dr.) M. M. Deka, Principal cum chief Superintendent, Gauhati Medical College and Hospital, Guwahati.
2. Dr. H. Bayan, Professor and Head, Deptt. Of Anatomy, G.M.C.H.
3. Dr. J. D. Sarma, Professor, Deptt. Of Anatomy, G.M.C.H.
4. Dr. R. Chaliha, Professor and Head of the Deptt. Of Forensic Medicine, G. M. C H.
5. Prof. A.G.Ahangar, Director, NEIGRIHMS, Shillong.
6. Prof V.Raphael, Dean, NEIGRIHMS, Shillong.

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How to cite this article:

Bishwajeet Saikia, Akash Handique, Pranjal Phukan, Donboklang Lynser, Amitav Sarma. CIRCLE OF WILLIS: VARIANT FORMS AND THEIR EMBRYOLOGY USING GROSS DISSECTION AND MAGNETIC RESONANCE ANGIOGRAPHY. Int J Anat Res 2014;2(2):344-53.