

Original Articles

VARIATIONS IN THE CIRCULUS ARTERIOSUS OF WILLIS: AUTOPSY FINDINGS IN 101 HUMAN CADAVERS

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ABSTRACT

In order to evaluate the microvascular anatomy and the interconnections, 101 fixed brains from human cadavers with a mean age of 41.3 years were subjected to objective scrutiny. Symmetry was visualized with an unaided eye and measurements made under microscopic magnification technique using a micrometer.

Classic symmetry was observed in only 19 of the specimens, whereas the rest of the 82 specimens revealed considerable variations in the form of diverse anomalies. These anomalies consisted of hypoplasia (45 cases), duplication (40 cases), triplication (6 cases), fenestration (6 cases), aplasia (5 cases), aneurysm (2 cases), common trunk (1 case) and a rare presentation where the internal carotid artery was found to have anastomosis with the posterior cerebral artery.

The frequency of anatomic variations and the presence of prevalent anomalies at the circle of Willis underscore the difficulty in understanding various cerebral vascular diseases; and their anatomical knowledge and information serve to adopt effective strategies in tackling such microvascular diseases.

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INTRODUCTION

Since 1664 when Thomas Willis first introduced the popularly known circle of Willis, considerable investigations and researches have taken place in autopsy findings of arteries, angiography and microscopic surgery and advances made in this specter. Symmetry by means of changing variables such as diameter and length of the communicating vessels has been reported to occur between 21-52%.^{1,5,7,9,13,15,17,18}

Hypoplasia of the A₁ segment of the anterior cerebral artery (ACA), unilateral or bilateral in 4-30% of speci-

mens,^{12,15} association of this hypoplasia with aneurysms of the anterior communicating artery (ACoA) in 68% of Kwak's and 85% of Wilson's study,⁸ hypoplasia of the posterior communicating artery (PCoA) in up to 32%, and a fetal type posterior cerebral artery (PCA) in 22% of Saeki & Rhoton's cases,¹⁴ emphasize the extremely prevalent anatomical variations of the circle of Willis.

This study attempts to explore the variations of the circle of Willis and their implications in an understanding of cerebrovascular disease and conducting microvascular surgery.

Circle of Willis Variations



Fig. 1. Symmetric or classical form of circle of Willis.

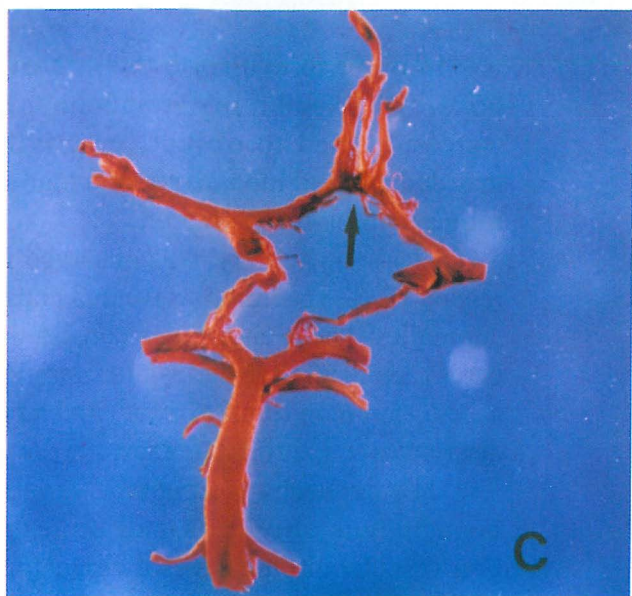
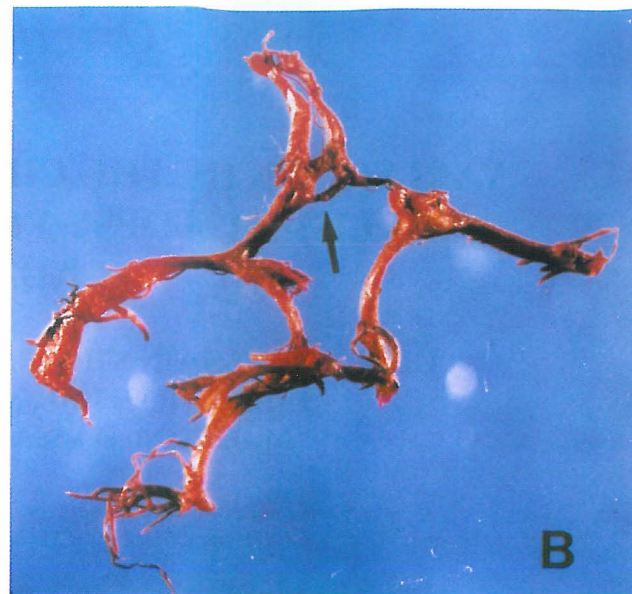
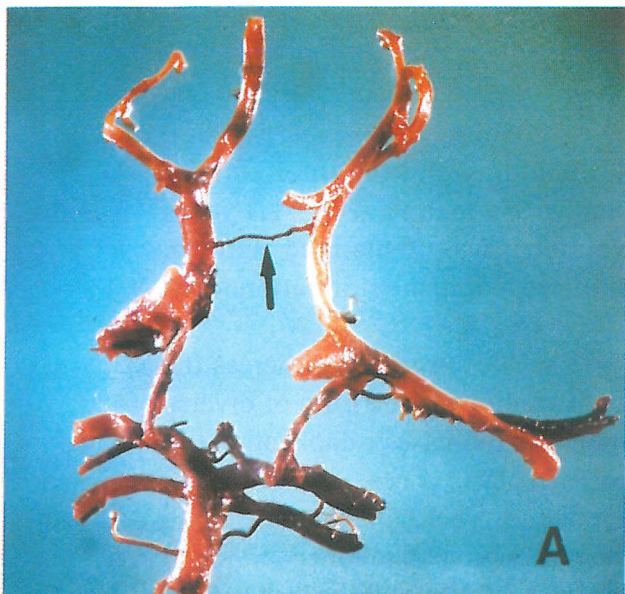


Fig. 2. Variations at the anterior part of the circle of Willis: ACoA hypoplasia (A), ACoA duplication (B) and A₂ trifurcation (C).

MATERIAL AND METHODS

Brains of 101 adult cadavers, 41 females and 60 males with an age ranging from 30 to 60 years (mean = 41.3)

Table I. Anatomic variations of the circle of Willis in 101 specimens.

Anomalies	Numbers	Arterial variations
Hypoplasia	43	31 PCoA, 6 A ₁ , 6 ACoA
Duplication	40	18 ACoA, 17 SCA, 4 A ₂ , 1 PCA
Triplication	6	5 A ₂ , 1 SCA
Fenestration	6	6 ACoA
Aplasia	5	3 PCoA, 2 A ₂
Common trunk	1	1 SCA-PCA
Total	101	

were autopsied to find any anatomical variations in the circle of Willis at the Department of Forensic Medicine in Tehran. The cadavers belonged to persons who had no primary lesion of the brain and had suffered a fatal noncerebral pathology.

Following craniotomy and dural opening, the brain was classically removed from the skull^{2,4} with cranial nerves and basal brain vessels fully preserved and kept intact. Blood clots were evacuated from the arteries and then fixed in 10% formalin for 3 weeks. The fixed specimens were studied by direct observation with the naked eye to visualize the shape and symmetry using a micrometer. The perforating vessels

were measured under microscopic magnification.

The arteries studied were A₁ & A₂ (proximal 4mm) segments, ACoA, the recurrent artery of Heubner (RAH), PCoA, P₁ segment of PCA, the anterior choroidal artery (AChA) and the superior cerebellar artery (SCA).

An artery was labelled as hypoplastic when its diameter was less than 1mm¹⁶ and asymmetry declared when the difference in diameter of two identical & synonymous arteries was 1 mm or more.⁹

RESULTS

Of 101 brain specimens examined, 19 were found to be symmetric (Fig.1) and 82 were found to have anatomical variations (Table I), of which 79 specimens showed variations in the anterior (Fig. 2) and posterior portion of the circle of Willis (Fig. 3); and a rare finding showing anastomosis between ICA & PCA (Fig. 4). Two ACoA aneurysms, one being in a symmetric circle and the other associated with hypoplastic ACA were also found.

ACA

A₁ segment: Out of 17 specimens, 11 were asymmetric and 6 were hypoplastic (4 on the left and 2 on the right side).

A₂ segment: Out of 13 specimens, 5 had arterial triplication, 4 duplication, 2 asymmetry and 2 aplasia (azygous artery).

ACoA: Of 30 specimens, 18 showed arterial duplication, 6 fenestration and 6 hypoplasia.

RAH

Found in all 101 specimens, arising on the right side from the ACA-ACoA junction (70%), A₁ (15%), and A₂

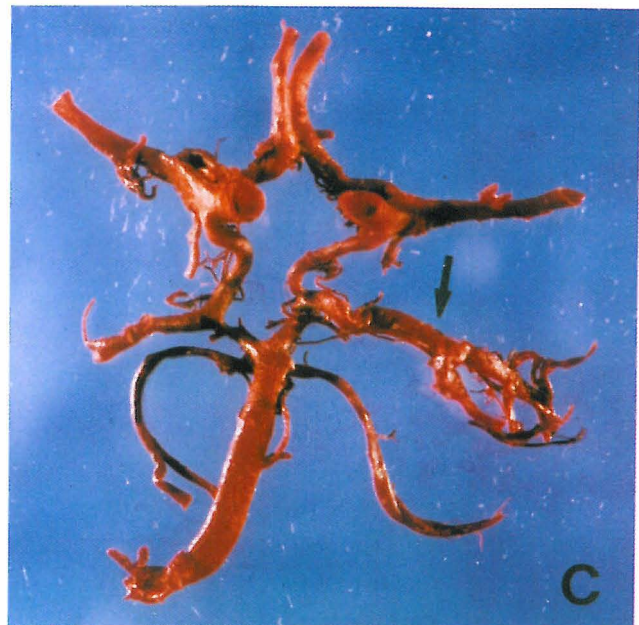
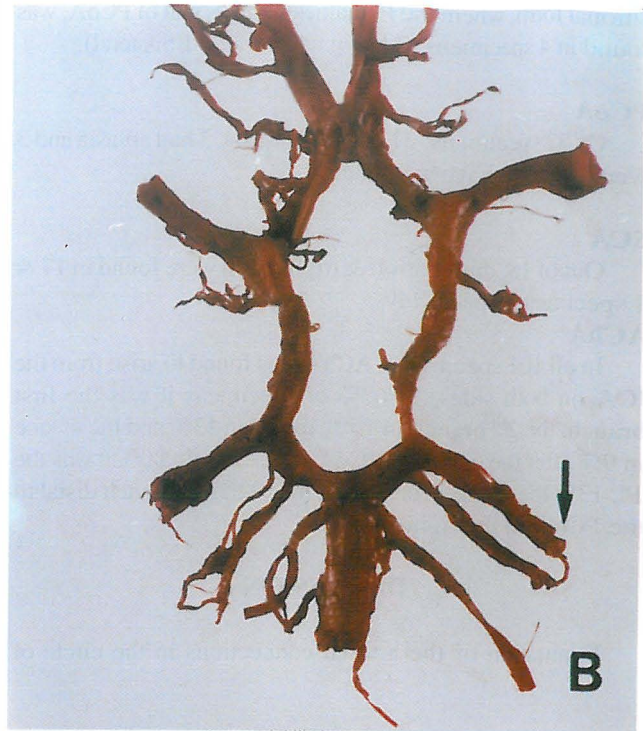


Fig. 3. Posterior Willis circle variations. PCoA hypoplasia (A), PCA duplication (B) and fetal type PCA (C).

(15%) and on the left side from the ACA-ACoA junction (75%), A₁ (20%) and A₂ (5%).

PCA

An adult form of PCA, where the P₁ segment is larger in diameter than PCoA, was seen in 50 specimens. The fetal type, where the P₁ is smaller in diameter than PCoA was found in 14 specimens (13 being bilateral and 1 unilateral) and the tran-

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sitional form, where the P_1 diameter equals that of PCoA, was found in 4 specimens (3 being unilateral & 1 bilateral).

PCoA

Of 37 specimens, 31 had hypoplasia, 3 had aplasia and 3 were found to have asymmetry.

SCA

Out of 18, duplication & triplication were found in 17 & 1 specimen respectively.

AChA

In all 101 specimens, AChA was found to arise from the ICA on both sides. In 63% of specimens it was the first branch, the 2nd branch in 15%, the 3rd in 13% and the 4th one in 9% after the origin of PCoA on the left. In 80% it was the 1st, 13% the 2nd, 5% the 3rd and in 2% the 4th branch distal to the PCoA on the right side.

DISCUSSION

Evaluation of the arterial connections in the circle of

Willis revealed that enormous variations in the form of hypoplasia, aplasia, duplication and triplication were apparent and the classical symmetry could only be found in nearly 20% of the specimens (Table II).

A deviation in the normal course of ACA in 25%,³ A_1 segment hypoplasia in association with ACoA aneurysms in up to 85%,⁸ hypoplasia of ACoA in 20%,^{6,9} fetal PCA in 22%,^{14,17} hypoplasia of PCoA in more than 50%^{13,14,16} and an aplastic PCoA in 6%^{6,14,19} have been reported. These variations are based on the evolution of fetal circulatory changes at the base of the brain,^{9,15} and also the rapid development of the telencephalon at 20-30 weeks of gestation¹⁵ are advocated as the mechanisms for presence of the adult type of PCA at birth, however in a Japanese research it has been said that hereditary factors may influence the fetal type of PCA.¹⁷

A larger specimen size would certainly increase the incidence of very rare presentations (Fig. 4).

An understanding and knowledge of such anatomical variations has an enormous impact on their clinical application in arteriovenous malformations, aneurysms, arterial

Table II. Reported various anomalies at the circle of Willis.

Anomaly	This study (%)	Author	(ref)	%
A_1 asymmetry	11	Riggs & Rupp	(8)	7
		Yasargil	(6,8)	4
		Perlmutter	(11)	10
A_1 hypoplasia	6	Wolschaeffer	(6)	8 (r.) 4.1(l.) 3.2 bilat
A_1 hypoplasia & ACoA aneurysm	1	Kwak	(8)	68
		Wilson	(8)	85
		Ogava	(10)	21.4
ACoA hypoplasia	6	Other studies	(6)	3- 20
ACoA duplication	17	Alpers	(9)	9- 40
ACoA fenestration	6			
PCoA hypoplasia	31	Alpers	(5,6,14)	20
		Riggs & Rupp	(13)	53
PCoA aplasia	3	Fetherman	(13)	5.5
		Stopfords	(13)	6
PCA adult type	50	Padget	(6,13,17)	<50
		Riggs & Rupp	(17)	76
PCA fetal type	14	McCormick	(17)	5.8
		Kleis	(17)	17
		Pedrosa	(17)	22
PCA transitional type	4	Kaseke	(17)	5.8
		Mitterwalner	(17)	8



Fig. 4. A rare form of anomaly: PCoA hypoplasia & ICA-PCA anastomosis (arrow) on the left side.

bypass and microsurgical skill,³ subarachnoid hemorrhage of nonaneurysmal arterial dilatations⁷ and diverse neurological variations in patients with such anomalies.¹⁰

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