ORIGINAL RESEARCH PAPER

Medicine

KEY WORDS: Circle of Willis, Ischemic Stroke, Cerebral Collateral Circulation, Cerebrovascular Anatomy.

ANATOMICAL VARIATIONS OF THE CIRCLE OF WILLIS AND THEIR CLINICAL CORRELATION IN ISCHEMIC STROKE

Dr. Abu Talha Dakhani Resident Doctor, Dept Of Anatomy, Al-Ameen Medical College, Vijayapura.

Dr. Ameer Khusru Kazi HOD & Professor, Dept Of Anatomy, Al-ameen Medical College, Vijayapura.

Dr. Sayed Abdul Lateef Kazi Professor, Dept Of Anatomy, Al-Ameen Medical College, Vijayapura.

ABSTRACT

The Circle of Willis (CoW) forms the pivotal arterial network at the brain's base, ensuring collateral circulation between the anterior and posterior cerebral territories. Despite its classical description, the CoW exhibits remarkable anatomical variability among individuals. These variations influence cerebral hemodynamics and play a decisive role in the extent, pattern, and prognosis of ischemic stroke. Understanding these deviations is crucial not only for clinicians managing cerebrovascular disorders but also for anatomists interpreting vascular morphometry and embryological development.

INTRODUCTION:

The human brain, though constituting merely two percent of total body weight, demands nearly twenty percent of the cardiac output, reflecting its dependence on uninterrupted blood flow.[1] The Circle of Willis (CoW), an arterial polygon located at the base of the brain, serves as the principal anastomotic channel between the internal carotid and vertebrobasilar systems. This vascular ring safeguards the brain against ischemic insults by providing collateral pathways in the event of arterial obstruction.[2] Classically, the CoW comprises the anterior cerebral arteries connected by the anterior communicating artery, the internal carotid arteries, the posterior cerebral arteries, and the posterior communicating arteries. However, anatomical investigations consistently reveal that a "complete" configuration where all components are patent and symmetrical is found in less than half of the population. Variations include hypoplasia, absence, duplication, or asymmetry of arterial segments, each with potential implications for cerebral perfusion.[3] These structural differences assume profound clinical importance in the context of ischemic stroke, one of the leading causes of morbidity and mortality worldwide. The compensatory capacity of the CoW determines the severity of neurological deficits following arterial occlusion. Incomplete configurations may predispose individuals to larger infarcts and diminished response to reperfusion therapy. Conversely, robust collaterals may protect against significant ischemic injury, even in the presence of major vessel obstruction.[4] Given the increasing availability of advanced imaging techniques, such as CT angiography and MR angiography, the anatomical and functional evaluation of the CoW has gained renewed attention. Yet, despite extensive anatomical studies, a comprehensive synthesis correlating these variations with stroke patterns remains limited.

This review aims to explore the spectrum of anatomical variations of the Circle of Willis and their clinical implications in ischemic cerebrovascular disease. By integrating anatomical, radiological, and clinical perspectives, it seeks to underscore the enduring relevance of vascular anatomy in contemporary medicine.

NORMAL ANATOMY OF THE CIRCLE OF WILLIS:

The Circle of Willis represents a vital arterial anastomosis situated within the interpeduncular fossa at the brain's base, encircling the optic chiasma and infundibulum. It forms a polygonal ring linking the carotid and vertebrobasilar systems, thereby ensuring redundancy of cerebral blood supply.

Classical Configuration:

In its idealized form, the CoW comprises the Anterior circulation, formed by the right and left anterior cerebral arteries (ACAs) connected via the anterior communicating artery (ACoA) and Posterior circulation, where the right and left posterior cerebral arteries (PCAs) arise from the basilar artery and connect to the internal carotid artery via the posterior communicating arteries (PCoAs). The internal carotid arteries (ICAs) serve as the main feeders, joining the anterior and posterior systems to complete the ring. When intact, this configuration equalizes perfusion pressure across hemispheres and compensates for unilateral arterial obstruction(Figure 1). The CoW, thus, plays a central role in maintaining hemodynamic stability within the cerebral circulation.[5]

Embryological Basis:

Developmentally, the CoW arises from a series of transient embryonic channels that remodel to form the mature arterial ring. Variations such as hypoplastic PCoA or fetal-type PCA often reflect persistence of embryonic patterns where posterior circulation remains dependent on the carotid system. Recognition of this developmental foundation is essential to understanding adult morphologic diversity.[6]

Functional Significance:

The physiological role of the CoW extends beyond simple arterial interconnection. It functions as a pressure-equalizing shunt, ensuring continued perfusion during transient or permanent occlusion of major vessels. The efficiency of this collateral mechanism depends on the patency, symmetry, and caliber of its component arteries. A well-formed CoW can sustain adequate cerebral flow during carotid or vertebral compromise, while an incomplete one may fail to compensate, precipitating ischemia.[6]

Morphometric Features:

Average diameters of the CoW arteries, as reported in radiological studies, typically range between 1-3 mm for communicating arteries and 2.5-4 mm for cerebral arteries. Symmetry is rare; minor caliber differences exist between right and left segments in most individuals. Morphometric parameters significantly influence flow dynamics. Arteries under 1 mm diameter are often termed hypoplastic and may not significantly contribute to collateral circulation.[7]

Radiological Identification:

Modern non-invasive imaging modalities, notably CT angiography (CTA) and MR angiography (MRA), have

revolutionized the in vivo assessment of the CoW. These techniques delineate vessel morphology, detect hypoplastic or absent segments, and quantify collateral pathways. CTA offers superior spatial resolution, whereas MRA provides functional flow evaluation without ionizing radiation. Radiological morphometry serves as the cornerstone for correlating anatomical variations with clinical cerebrovascular events.[7]

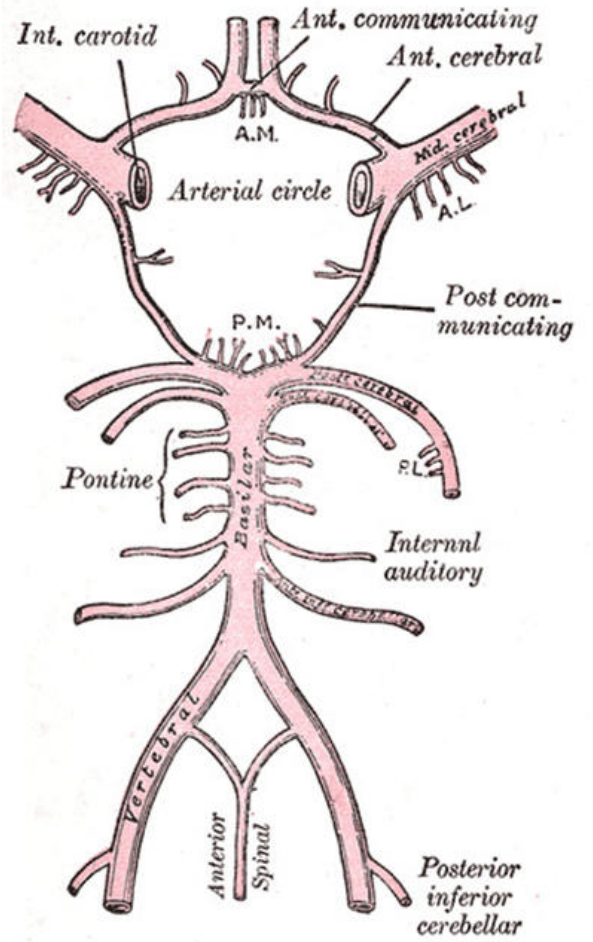

Figure 1: Normal Anatomy of Circle Of Willis

Anatomical Variations Of The Circle Of Willis:

Anatomical variations of the Circle of Willis (CoW) are among the most extensively documented yet variably interpreted phenomena in neurovascular anatomy. Though classically represented as a complete arterial polygon, true anatomical perfection is rare. Numerous studies both cadaveric and angiographic reveal that less than 40–50% of individuals possess a fully complete CoW. The remainder exhibit one or more structural deviations, ranging from subtle asymmetries to complete absence of communicating segments. These variations profoundly influence cerebral hemodynamics and, consequently, the brain's ability to maintain adequate collateral flow during ischemic episodes.[3]

Classification Of Variations:

Variations of the CoW are typically classified into:

- **Segmental absence or hypoplasia:** Complete absence or significantly reduced caliber (<1 mm) of an arterial segment.
- **Duplication or fenestration:** Presence of dual arterial channels or partial splitting within a segment.
- **Asymmetry:** Unequal diameters between right and left arteries, altering flow distribution.
- **Fetal-type configurations:** Persistence of embryonic patterns where posterior circulation remains primarily supplied by the internal carotid system.
- **Accessory or anomalous vessels:** Rare additions such as a median artery of the corpus callosum or persistent trigeminal artery.

These Patterns Can Occur In Isolation Or Combination, Yielding A Wide Morphological Spectrum.[3,8]

Variations In The Anterior Circulation:

The anterior segment of the CoW comprises the anterior cerebral arteries (ACAs) and the anterior communicating artery (ACoA). The Anterior Communicating Artery (ACoA) is the most frequent site of variation. Reported anomalies include duplication (in up to 15% of cases), absence (10-12%), and fenestration (5-7%). Absence or hypoplasia of the ACoA compromises interhemispheric compensation, leading to greater vulnerability during unilateral internal carotid occlusion. Conversely, a well-developed ACoA is often associated with better cross-flow and reduced infarct size. In addition anomalies involving Anterior Cerebral Artery (ACA), particularly A1segment influences the efficiency of anterior collateral circulation. Asymmetry of the A1 segment of the ACA is another common variation, observed in up to 30% of individuals. Hypoplasia or aplasia of one A1 segment shifts perfusion burden to the contralateral artery via the ACoA. Clinically, such asymmetry may predispose to hemodynamic insufficiency in the dependent hemisphere, particularly during carotid stenosis or embolic events.[9]

Variations In The Posterior Circulation:

The posterior segment involves the posterior cerebral arteries (PCAs) and the posterior communicating arteries (PCoAs), both of which display frequent deviations. The Posterior Communicating Artery (PCoA) is among the most variable arteries of the CoW. Hypoplasia is observed in approximately 25-35%, and complete absence in up to 20% of cases. The presence of a robust PCoA facilitates effective carotid-vertebrobasilar collateralization; its absence severely limits compensation during basilar or vertebral occlusion. The Fetal-type Posterior Cerebral Artery (fPCA), where the PCA arises predominantly from the internal carotid rather than the basilar artery, is one of the most clinically significant variants. Found in 10–30% of populations (unilateral more common than bilateral), it represents persistence of an embryonic pattern. This configuration results in carotid dominance of posterior circulation and is associated with altered perfusion dynamics during carotid or basilar artery compromise. Patients with fPCA may demonstrate atypical stroke patterns involving posterior territories secondary to carotid pathology.[10,11]

Complete vs. Incomplete Circle:

A complete Circle of Willis implies all components are present, patent, and symmetrical. However, angiographic and cadaveric studies consistently demonstrate that a complete CoW occurs in only about 40–50% of individuals. The incomplete type, characterized by absence or hypoplasia of one or more segments, predominates. Incomplete configurations are subdivided into Anterior incomplete type, characterised by defects in ACoA or A1 segment, Posterior incomplete type defined by hypoplastic or absent PCoA or PCA & Mixed type involving both anterior and posterior segments. Hemodynamically, an incomplete CoW offers limited compensatory flow and is associated with greater infarct size and poor prognosis following arterial occlusion.[12,13]

Morphological Patterns and Prevalence:

Meta-analyses of CTA and MRA studies reveal wide geographic and ethnic variation in CoW morphology, with hypoplastic PCoA reported in 25-35%, absent PCoA in 10-20%, fetal-type PCA in 10-30%, hypoplastic A1 (ACA) in 10-15%, absent ACoA in 10-12%, and complete CoW in 40-50%. Such variability suggests a genetic and developmental basis,

influenced by intrauterine hemodynamic conditions. Understanding the prevalence of these variants aids clinicians in interpreting angiographic findings and assessing individual cerebrovascular risk.[7]

Hemodynamic Implications:

The anatomical integrity of the CoW determines the direction, velocity, and adaptability of cerebral blood flow during vascular compromise. Computational flow dynamics (CFD) and Doppler studies reveal that hypoplastic segments contribute minimally to collateral circulation. In contrast, a complete and symmetrical CoW maintains effective physiological flow patterns even under occlusive stress. Thus, morphological completeness translates into greater functional resilience against ischemic events.[1]

Clinical Correlation In Ischemic Stroke:

Ischemic stroke represents one of the most catastrophic manifestations of cerebrovascular disease, resulting from the abrupt interruption of cerebral blood flow. The clinical spectrum from transient ischemic attacks to massive infarctions largely depends on the efficacy of collateral circulation. The Circle of Willis (CoW), as the principal intracranial collateral pathway, plays a critical role in maintaining perfusion to affected territories during arterial occlusion. Its anatomical completeness, symmetry, and vessel calibers dictate the brain's resilience to ischemia. In physiological conditions, the pressure across the CoW remains equalized, and flow through the communicating arteries is minimal. However, when one major artery (such as the internal carotid or vertebral) becomes narrowed or occluded, a pressure gradient develops, prompting compensatory flow through the collateral channels of the CoW. If the CoW is complete, this collateral redistribution occurs efficiently, maintaining perfusion until recanalization or therapeutic intervention. Conversely, in incomplete configurations with hypoplastic or absent communicating segments collateral flow becomes inadequate, resulting in hemodynamic insufficiency, increased infarct volume, and worse neurological outcomes.[4,13]

Influence of Specific Anatomical Variations on Stroke Patterns:

(a): Anterior Circulation Variations:

Hypoplastic or absent A1 segment (ACA) limits interhemispheric flow via the ACoA, predisposing to unilateral frontal or callosal infarcts even with contralateral carotid patency, and the clinical implication is that patients with significant carotid stenosis on one side and a hypoplastic contralateral A1 are at markedly higher risk of ischemia in the ipsilateral ACA territory. Absent or fenestrated ACoA interrupts anterior cross-circulation, and during carotid occlusion, such patients may lack compensatory flow to the opposite hemisphere, leading to more extensive cortical infarction.[9,14]

(b): Posterior Circulation Variations:

Hypoplastic or absent PCoA diminishes carotid-basilar communication and reduces collateral supply to the posterior cerebral territory, thereby predisposing individuals to occipital and thalamic infarcts, particularly in the setting of vertebrobasilar insufficiency. Radiologically, CT angiography often demonstrates poor retrograde filling of posterior territories during carotid occlusion in such cases. Conversely, the presence of a fetal-type PCA (fPCA) alters intracranial flow dynamics by making posterior territories dependent on the carotid circulation, such that carotid stenosis may lead to posterior infarcts that can mimic basilar pathology. Clinically, fPCA has been associated with a higher incidence of posterior cerebral artery strokes and paradoxical embolic patterns.

(c): Mixed and Incomplete Configurations:

When both anterior and posterior segments are compromised, collateral potential becomes minimal. Such patients often experience larger infarct volumes, early neurological deterioration, and reduced response to reperfusion therapy.[14,15,16]

Correlation Between CoW Completeness and Stroke Severity:

Several imaging-based clinical studies have demonstrated a clear association between CoW configuration and stroke severity. Complete CoW is associated with smaller infarct size, lower NIHSS (National Institutes of Health Stroke Scale) scores, and better outcomes post-thrombolysis. Whereas Incomplete CoW correlates with extensive ischemia, delayed recanalization, and higher risk of malignant middle cerebral artery (MCA) infarction. Hemodynamic studies (using Transcranial Doppler) show reduced compensatory flow in incomplete CoW during carotid compression tests. Meta-analyses suggest that patients with incomplete CoW have up to 2.5 times higher odds of poor clinical outcome following ischemic stroke compared with those having a complete configuration. While another study reported the presence of an incomplete Circle of Willis decreases odds of having good outcomes by 47%.[17,18,19]

Role In Collateral Circulation And Prognosis:

Collateral circulation via the Circle of Willis acts as the primary compensatory mechanism during acute ischemic events, enabling redistribution of blood from unaffected territories to preserve penumbral tissue and limit infarct expansion. Neuroimaging studies show that patients with complete CoW frequently exhibit smaller diffusion-perfusion mismatch and reduced infarct core expansion on MRI. Therapeutically, the effectiveness of interventions such as thrombolysis and mechanical thrombectomy is partly influenced by the robustness collateral pathways, a well-developed CoW enhances recanalization efficiency and improves reperfusion outcomes.[18,20,21]

Summary of Clinical Correlations:

Anatomical Variation	Pathophysiological Impact	Clinical Manifestation
Hypoplastic/absent A1 (ACA)	Poor anterior cross-flow	Unilateral ACA territory infarcts
Absent/fenestrated ACoA	Impaired hemispheric compensation	Larger cortical infarcts
Hypoplastic/absent PCoA	Reduced carotid–basilar collateralization	Increased vulnerability to Occipital/thalamic strokes
Fetal-type PCA	Carotid dominance of posterior flow	Posterior infarcts in carotid stenosis
Incomplete CoW	Global collateral insufficiency	Extensive infarction, poor recovery

CONCLUSION:

The Circle of Willis stands as a remarkable testament to the brain's adaptive vascular design, yet its variability defines the boundary between protection and vulnerability. A complete and symmetrical CoW ensures robust collateralization and resistance to ischemia, whereas incomplete configurations predispose to extensive infarction, poor neurological recovery, and adverse therapeutic outcomes. With the advent of advanced imaging, clinicians now possess the tools to evaluate CoW morphology non-invasively, transforming classical anatomy into a practical diagnostic instrument. Ultimately, the integration of anatomical insight with clinical acumen enriches our capacity to predict, prevent, and mitigate the burden of cerebrovascular disease.

REFERENCES:

1. Claassen, J. A. H. R., Thijssen, D. H. J., Panerai, R. B., & Faraci, F. M. (2021).

Regulation of cerebral blood flow in humans: physiology and clinical implications of autoregulation. *Physiological reviews*, *101*(4), 1487–1559. https://doi.org/10.1152/physrev.00022.2020

2. Singh, R., Kannabathula, A. B., Sunam, H., & Deka, D. (2017). Anatomical variations of circle of Willis - a cadaveric study. *International Surgery Journal*, *4*(4), 1249–1258. https://doi.org/10.18203/2349-2902.isj20171016
3. Ayre, J. R., Bazira, P. J., Abumattar, M., Makwana, H. N., & Sanders, K. A. (2022). A new classification system for the anatomical variations of the human circle of Willis: A systematic review. *Journal of anatomy*, *240*(6), 1187–1204. https://doi.org/10.1111/joa.13616
4. Oumer, M., Alemayehu, M., & Muche, A. (2021). Association between circle of Willis and ischemic stroke: a systematic review and meta-analysis. *BMC neuroscience*, *22*(1), 3. https://doi.org/10.1186/s12868-021-00609-4
5. Rosner, J., Reddy, V., & Lui, F. (2023). Neuroanatomy, circle of Willis. In StatPearls. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK534861/
6. Takakuwa, T., Koike, T., Muranaka, T., Uwabe, C., & Yamada, S. (2016). Formation of the circle of Willis during human embryonic development. *Congenital anomalies*, *56*(5), 233–236. https://doi.org/10.1111/cga.12165
7. Kızılgöz, V., Kantarcı, M., & Kahraman, □. (2022). Evaluation of Circle of Willis variants using magnetic resonance angiography. *Scientific reports*, *12*(1), 17611. https://doi.org/10.1038/s41598-022-21833-w
8. Iqbal S. (2013). A comprehensive study of the anatomical variations of the circle of willis in adult human brains. *Journal of clinical and diagnostic research : JCDR*, *7*(11), 2423–2427. https://doi.org/10.7860/JCDR/2013/6580.3563
9. Rajan, M. L. (2021). Study of variations in the anterior half of the circle of Willis using magnetic resonance angiography. Indian Journal of Clinical Anatomy and Physiology, 8(1), 36–41. https://doi.org/10.18231/j.ijcap.2021.008
10. Rajan, M. L. (2021). Study of variations in the posterior part of the circle of Willis using magnetic resonance angiography. Indian Journal of Clinical Anatomy and Physiology, 8(1), 11–14. https://doi.org/10.18231/j.ijcap.2021.003
11. Jones, J. D., Castanho, P., Bazira, P., & Sanders, K. (2021). Anatomical variations of the circle of Willis and their prevalence, with a focus on the posterior communicating artery: A literature review and meta-analysis. *Clinical anatomy (New York, N.Y.)*, *34*(7), 978–990. https://doi.org/10.1002/ca.23662
12. Nenna, A., Corrado, D., Loreni, F., Ferrisi, C., Sorrentino, G., Giacinto, O., Chello, M. (2024). Anatomic Completeness, Variations, Patency, and Functional Assessment of Circle of Willis: Implications for Chronic Aortic Dissection and Non-Emergent Arch Surgery. IntechOpen. https://doi.org/10.5772/intechopen.1005756
13. Al Saffar, R. A. M. (2026). A Systematic Review of Anatomical Variations of the Circle of Willis and Their Clinical Significance. *Journal of Medicinal and Chemical Sciences*, *9*(1), 137-152. https://doi.org/10.48309/jmcs.2026.229719
14. Negatie, H. M., Kebede, M. A., Abate, A. D., Admassie, S. H., Worku, A. B., Ahmed, H. T., Mesfine, Y. Y., & Melak, M. M. (2025). Association of circle of willis variants with stroke and aneurysm: insights from a tertiary hospital in Ethiopia. *BMC neurology*, *25*(1), 73. https://doi.org/10.1186/s12883-025-04082-y
15. Feng, L., Zhai, F. F., Li, M. L., Zhou, L. X., Ni, J., Yao, M., Jin, Z. Y., Cui, L. Y., Zhang, S. Y., Han, F., & Zhu, Y. C. (2023). Association between anatomical variations of the circle of Willis and covert vascular brain injury in the general population. Cerebrovascular Diseases, 52(4), 480–486. https://doi.org/10.1159/000527432
16. Brohi, A. H., Hameed, U., Iqbal, S., Haq, A. A., Mughal, A., & Hassan, N. (2024). Radiological imaging of anatomical variations of arteries of the circle of Willis using magnetic resonance angiography in a subset of the Pakistani population. Rawal Medical Journal, 49(4), 855–858.
17. Lin, E., Kamel, H., Gupta, A., RoyChoudhury, A., Girgis, P., & Glodzik, L. (2022). Incomplete circle of Willis variants and stroke outcome. *European journal of radiology*, *153*, 110383. https://doi.org/10.1016/j.ejrad.2022.110383
18. Zhou, H., Sun, J., Ji, X., Lin, J., Tang, S., Zeng, J., & Fan, Y. H. (2016). Correlation Between the Integrity of the Circle of Willis and the Severity of Initial Noncardiac Cerebral Infarction and Clinical Prognosis. *Medicine*, *95*(10), e2892. https://doi.org/10.1097/MD.0000000000002892
19. Chuang, Y. M., Chan, L., Lai, Y. J., Kuo, K. H., Chiou, Y. H., Huang, L. W., Kwok, Y. T., Lai, T. H., Lee, S. P., Wu, H. M., & Yeh, Y. C. (2013). Configuration of the circle of Willis is associated with less symptomatic intracerebral hemorrhage in ischemic stroke patients treated with intravenous thrombolysis. *Journal of critical care*, *28*(2), 166–172. https://doi.org/10.1016/j.jcrc.2012.08.018
20. Maguida, G., & Shuaib, A. (2023). Collateral Circulation in Ischemic Stroke: An Updated Review. *Journal of stroke*, *25*(2), 179–198. https://doi.org/10.5853/jos.2022.02936
21. Brohi, A. H., Borges, K. J. J., Yar, G. H., Shah, S. N. N., & Hassan, N. (2018). Configuration of circle of Willis and its clinical significance. Journal of Bahria University Medical and Dental College, 8(4). https://doi.org/10.51985/jbumdc2018078