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ANATOMICAL VARIATIONS OF CEREBRAL BLOOD VESSELS AND THEIR CLINICAL SIGNIFICANCE IN CEREBROVASCULAR DISEASES: A NARRATIVE REVIEW

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ABSTRACT: Cerebral arterial anatomy exhibits substantial interindividual variation, particularly within the circle of Willis and the branching patterns of the anterior, middle, and posterior cerebral arteries. Such differences are relevant to collateral circulation, interpretation of vascular imaging, planning of endovascular or microsurgical procedures, and assessment of selected cerebrovascular disorders. This narrative review synthesizes anatomical and clinical literature on developmental mechanisms, common configurations, diagnostic visualization, and potential implications of cerebral arterial variants. Particular attention is given to hypoplasia or absence of communicating segments, fetal-type posterior cerebral artery, anterior cerebral artery variants, and middle cerebral artery branching patterns. Available studies demonstrate marked heterogeneity in the reported frequency of these configurations, partly because definitions, imaging techniques, and sampled populations differ. An incomplete circle of Willis may limit certain collateral pathways under hemodynamic stress, but the presence of an anatomical variant does not, by itself, establish an individual risk of stroke or aneurysm. A clinically meaningful assessment therefore requires integration of vascular morphology with vessel caliber, collateral function, clinical presentation, and imaging findings. The review highlights the need for standardized anatomical terminology and cautious interpretation of observational associations.

Keywords: cerebral arterial anatomy; circle of Willis; anatomical variation; collateral circulation; ischemic stroke; intracranial aneurysm; CT angiography; MR angiography.

INTRODUCTION

The cerebral arterial system is organized to provide a continuous supply of oxygen and metabolic substrates to the brain. Blood reaches the intracranial compartment predominantly through the internal carotid and vertebrobasilar systems, which communicate at the base of the brain through the arterial polygon traditionally termed the circle of Willis. Although textbook illustrations commonly present a symmetric and complete arterial ring, this arrangement represents only one of numerous configurations encountered in clinical and anatomical practice [1,2]. Variability may involve vessel presence, diameter, course, origin, duplication, fenestration, and patterns of branching. These features are especially important when vascular reserve is challenged by stenosis, occlusion, vasospasm, or changes in perfusion pressure.

Recent advances in computed tomography angiography (CTA), magnetic resonance angiography (MRA), and digital subtraction angiography (DSA) have increased recognition of arterial variants in living patients. The growing use of neurovascular imaging has also made precise anatomical interpretation essential for neurologists, radiologists, neurosurgeons, and interventional specialists. Nevertheless, an anatomical difference should not automatically be classified as pathological. Many variants represent stable developmental configurations that remain clinically

silent throughout life, and their significance depends on accompanying disease and functional circulation [3,4].

The relationship between cerebral arterial morphology and cerebrovascular disease is complex. In ischemic conditions, communicating arteries may provide alternative pathways for blood flow, yet the effectiveness of these routes depends on their caliber and on pressure gradients. In aneurysmal disease, local branching geometry and flow dynamics may contribute to lesion formation, but age, hypertension, smoking, and other vascular factors also influence risk. A balanced review should therefore distinguish anatomical observations from causal conclusions about disease [5,6].

AIM OF THE REVIEW

The aim of this article is to summarize major anatomical variations of the cerebral arterial circulation, examine their developmental and imaging characteristics, and critically discuss their potential clinical relevance to ischemic stroke, intracranial aneurysms, and neurovascular interventions.

MATERIALS AND METHODS

This work is a narrative literature review rather than an original patient-based investigation or a systematic review. Foundational anatomical studies, peer-reviewed reviews, and selected clinical imaging publications were considered to synthesize established descriptions of the circle of Willis and its major branches. The literature was evaluated qualitatively with attention to anatomical definitions, imaging modality, study population, and limitations in inferring clinical outcomes from vessel morphology. Sources addressing the prevalence of circle-of-Willis configurations, collateral flow, stroke, aneurysm development, and diagnostic imaging informed the discussion [1–10]. No new participants were enrolled, no images were independently interpreted, and no statistical meta-analysis was performed.

RESULTS AND DISCUSSION

Normal organization and embryological basis of cerebral arterial circulation

The circle of Willis connects the paired internal carotid systems with the posterior circulation through the anterior communicating artery and paired posterior communicating arteries. Its conventional components include the proximal anterior cerebral artery segments, anterior communicating artery, terminal internal carotid arteries, posterior communicating arteries, and proximal posterior cerebral artery segments. The middle cerebral arteries arise from the internal carotid arteries but are not structural components of the arterial ring itself. This distinction is important because imprecise labeling can obscure descriptions of complete and incomplete circles [1,2].

During embryogenesis, the developing cerebral circulation undergoes sequential formation, enlargement, regression, and remodeling of arterial channels. The posterior cerebral territory is initially supplied substantially through the carotid system; later maturation of the vertebrobasilar system alters the relative contribution of the posterior communicating and proximal posterior cerebral arteries. Persistence of an early arrangement can produce a fetal-type posterior cerebral artery. Similarly, asymmetrical development or regression of communicating segments contributes to hypoplasia, aplasia, or unequal caliber of vessels in the mature circle [3,7]. Developmental terminology explains the origin of these variants but should not be interpreted as indicating a congenital disease in every patient.

Variations of the anterior communicating complex

The anterior communicating artery complex exhibits considerable diversity, including a short or long communicating segment, duplication, fenestration, and asymmetry of the proximal anterior

cerebral arteries. Hypoplasia of one A1 segment may cause the contralateral internal carotid artery to supply substantial portions of both anterior cerebral territories through the anterior communicating connection. This arrangement changes the direction and distribution of flow compared with a symmetric configuration and may be relevant when assessing unilateral carotid stenosis or planning vascular procedures [1,4].

Additional variants include an azygos anterior cerebral artery, in which a single distal trunk supplies both medial hemispheric territories, and accessory or bihemispheric anterior cerebral arteries. These configurations have practical importance for surgical orientation and infarct localization. For example, compromise of a single dominant distal anterior cerebral trunk can potentially affect a broader bilateral territory than would be expected from conventional paired anatomy. Their presence should be documented accurately on vascular imaging, especially before interventions near the anterior communicating region [3,7].

Posterior communicating arteries and fetal-type posterior cerebral artery

The posterior communicating arteries are highly variable in caliber and may be small or not demonstrable on a particular imaging examination. A fetal-type posterior cerebral artery refers to a configuration in which the posterior cerebral territory receives a predominant contribution from the internal carotid artery through a relatively large posterior communicating artery, with a small or absent P1 segment. Definitions differ across publications: some distinguish complete fetal-type configurations from partial variants based on whether the P1 segment is absent or merely smaller than the posterior communicating artery [3,8]. Consequently, prevalence estimates should not be compared without checking classification criteria.

The fetal-type configuration can alter the vascular territory at risk during internal carotid artery disease. An embolus originating in the carotid circulation may reach a posterior cerebral artery territory when that territory depends on the carotid pathway. Conversely, collateral options between the anterior and posterior systems may differ according to the completeness of the proximal posterior circulation. These are plausible hemodynamic implications rather than evidence that the variant invariably causes posterior circulation ischemia [4,8].

Middle cerebral artery and vertebrobasilar branching variants

The middle cerebral artery commonly divides into major superior and inferior trunks, but early bifurcation, trifurcation, accessory branches, and duplicated vessels are recognized anatomical patterns. These variants may affect catheter navigation, the appearance of vessel occlusion, and the planning of aneurysm surgery near the bifurcation. A duplicated middle cerebral artery arising from the internal carotid artery must be distinguished from an accessory middle cerebral artery arising from the anterior cerebral artery, because the origin determines the vascular route and possible collateral contribution [3,7].

The vertebrobasilar circulation also displays variation in vertebral artery dominance, basilar artery curvature, branching patterns, and cerebellar artery origins. Although not all such configurations are part of the circle of Willis, they influence the overall geometry of intracranial blood flow and may complicate interpretation of angiographic studies. Clinically relevant descriptions should specify the involved segment and its relationship to adjacent arteries rather than using the nonspecific term “vascular anomaly” [7,9].

Anatomical completeness versus functional collateral circulation

One of the most important clinical functions attributed to the circle of Willis is its capacity to redistribute blood when a major supplying artery becomes narrowed or occluded. Theoretically, a complete arterial ring offers alternative pathways between hemispheres and between carotid and vertebrobasilar circulations. However, the presence of a vessel on an anatomical image does not

guarantee that it can carry sufficient flow under stress. Functional collateral capacity depends on luminal diameter, vascular resistance, autoregulatory responses, pressure gradients, and the status of leptomeningeal collaterals outside the circle itself [4,5].

The classic work of Alpers and colleagues and subsequent population-based imaging investigations demonstrated that configurations of the circle of Willis vary widely [1,2]. In clinical cohorts, associations have been reported between particular incomplete configurations and patterns of ischemic injury, but findings are not uniformly consistent across populations. Differences in imaging sensitivity, stroke mechanism, arterial stenosis, and patient selection can influence the observed relationship. Therefore, vascular variants should be treated as one component of a broader hemodynamic assessment rather than as stand-alone predictors of cerebrovascular events [4,5].

Relevance to ischemic stroke and carotid artery disease

In acute ischemic stroke, anatomical knowledge assists with identification of the likely vascular territory, interpretation of collateral routes, and selection of an endovascular approach. Hypoplasia of an A1 segment can influence cross-flow through the anterior communicating artery, while a small posterior communicating artery may reduce one potential route of compensation during carotid compromise. A fetal-type posterior cerebral artery may extend the distribution of carotid-sourced embolic ischemia to the occipital or inferomedial temporal regions. Nevertheless, infarct patterns also depend on embolic source, occlusion site, duration of ischemia, and distal collateral networks [4,5,8].

In patients with carotid stenosis, the functional importance of a communicating artery is best understood alongside perfusion assessment and the status of the contralateral carotid and vertebrobasilar systems. A morphologically complete circle may coexist with poor collateral performance, whereas patients with an incomplete ring may retain adequate perfusion through alternative extracranial or leptomeningeal routes. Clinical decisions about revascularization must therefore follow established disease-specific evidence and guidelines rather than anatomical completeness alone [5,9].

Anatomical variants and intracranial aneurysms

Intracranial aneurysms frequently arise near arterial bifurcations and communicating junctions, where local flow patterns and wall stresses may be complex. Anatomical asymmetry within the anterior communicating artery complex, including unequal A1 segments, can modify inflow distribution and local hemodynamic conditions. Observational studies have explored associations between these configurations and anterior communicating artery aneurysms; however, anatomical association does not establish that a particular variant is sufficient to cause aneurysm formation [6,10].

Aneurysm development is multifactorial and involves vascular wall biology, hemodynamics, inherited predisposition in some patients, and acquired risk factors. Clinical interpretation must also separate aneurysm presence from the risk of rupture. Morphological variants may affect treatment planning by changing the position of parent vessels, the direction of inflow, or access for clipping and coiling, but decisions require evaluation of aneurysm size, location, morphology, patient characteristics, and treatment risks [6,10].

Imaging methods and sources of measurement variability

CTA provides rapid, high-resolution depiction of many intracranial arterial structures and is widely used in the evaluation of acute vascular conditions. Time-of-flight MRA can demonstrate intracranial vessels without ionizing radiation or intravenous contrast, although slow or complex flow may reduce signal and make a patent but diminutive artery appear absent. Contrast-enhanced techniques and DSA may clarify uncertain findings in selected clinical situations. DSA offers

dynamic vascular information and high spatial resolution but is invasive and therefore is not indicated solely to catalog incidental variants [3,9].

Differences in modality and vessel-diameter thresholds contribute to variation in published prevalence figures. Studies may label a segment "absent" when it is below the technique's detection limit, while others classify the same segment as hypoplastic. In addition, two-dimensional projections can obscure overlapping vessels and may not distinguish fenestration from duplication. Reporting should therefore state the imaging method, relevant vessel caliber, and the operational definition of completeness or hypoplasia whenever possible [2,3].

Implications for neurosurgery, endovascular therapy, and anatomy education

Precise identification of arterial variants supports safer procedural planning. During aneurysm surgery, an unexpected accessory artery or fenestration may alter the dissection plane and increase the importance of preserving small perforators. In thrombectomy or diagnostic angiography, unusual branch origins may affect catheter selection and interpretation of angiographic roadmaps. Before interventions involving the carotid circulation, recognition of a fetal-type posterior cerebral artery can help clinicians anticipate the territory potentially dependent on the treated vessel [7–9].

For anatomy education, teaching should move beyond the single idealized diagram of the circle of Willis. Students benefit from comparing conventional schematics with real CTA and MRA examples and from learning to describe variation using precise segmental terminology. A useful pedagogical distinction is between normal variation, developmental anomaly with possible clinical implications, and established vascular pathology. This distinction discourages overdiagnosis and strengthens communication between anatomists and clinicians.

Limitations of existing evidence and priorities for future research

The literature is limited by heterogeneous definitions, differing population characteristics, selection bias in hospital-based cohorts, and incomplete measurement of functional collateral flow. Cadaveric studies provide detailed morphology but cannot directly characterize dynamic perfusion in living individuals. Imaging studies offer clinical context but may miss small communicating arteries because of resolution or flow-dependent signal. Cross-sectional associations with stroke or aneurysm therefore require cautious interpretation, particularly when potential confounding factors have not been adequately controlled [2,4,5].

Future research would benefit from standardized definitions of hypoplasia, aplasia, fetal-type posterior cerebral artery, and circle-of-Willis completeness; consistent reporting of imaging techniques; and integration of anatomy with perfusion measurements and longitudinal outcomes. Multicenter studies could help distinguish variants that are merely common from those that provide clinically useful prognostic information in clearly defined patient groups. Such investigations should account for traditional vascular risk factors and avoid treating morphology as a substitute for functional assessment.

CONCLUSION

Cerebral arterial variations are common features of human vascular anatomy and are especially prominent within the circle of Willis. Differences in the caliber and continuity of communicating arteries, fetal-type posterior cerebral artery configurations, and variations in anterior and middle cerebral artery branching may influence collateral pathways, vascular territories, and procedural anatomy. Their clinical relevance is context-dependent: a variant may be important during arterial occlusion or intervention while remaining entirely asymptomatic in other circumstances.

The available literature supports careful description of arterial morphology and its integration with clinical findings, vascular imaging, and hemodynamic information. At the same time,

observational associations should not be interpreted as proof that a specific variant independently causes stroke or aneurysm. Standardized terminology, improved imaging interpretation, and prospective studies linking anatomy with functional outcomes are essential for advancing clinically useful understanding of cerebrovascular variation.

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