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The Circle of Willis as a Functional Unit: Myth or Reality

Valchkevich Dzmitry[1]* and Tokina Ina[2]

PhD, Associate Professor, Department of Normal Anatomy, Department of Grodno State Medical University, Belarus

ABSTRACT

Background: The Circle of Willis (COW) has historically been regarded as a crucial collateral pathway ensuring cerebral perfusion during arterial obstruction. However, its true role as a functional unit remains debated, given the frequent anatomical variations and inconsistent clinical protection against ischemic events.

Methods: This review integrates historical accounts, anatomical and morphological studies, hemodynamic analyses, computational modeling, and clinical observations. Evidence was synthesized to evaluate the structural variability of the COW, its hemodynamic behavior, and its clinical relevance in cerebrovascular disease.

Results: Anatomical investigations show that a complete and symmetric COW is relatively rare, with hypoplasia and absence of key segments being common. Hemodynamic studies and computational simulations demonstrate that collateral efficiency depends on vessel diameter, branching geometry, pressure gradients, and vascular compliance, rather than anatomical presence alone. Clinical findings reveal paradoxes: patients with a morphologically intact COW may still experience severe ischemia, whereas incomplete variants may maintain adequate perfusion through alternative collateral routes.

Conclusion: The COW should be viewed as a conditional, context-dependent reserve system rather than a continuously active circulatory safeguard. Its collateral role emerges predominantly under pathological stress and is constrained by individual vascular geometry and hemodynamic conditions. Advances in imaging and patient-specific computational modeling hold promise for bridging the gap between morphology and functional competence, with potential to improve risk stratification and therapeutic decision-making in cerebrovascular care.

***Corresponding author**

Valchkevich Dzmitry, PhD, Associate Professor of Normal Anatomy Department of Grodno State Medical University, Belarus.

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Introduction

The arterial polygon known as the Circle Of Willis (COW) has long been regarded as a pivotal collateral network at the base of the brain, postulated to afford a degree of protection against focal cerebral ischemia. First elaborated in Thomas Willis's «Cerebri Anatome» in 1664, the concept of an arterial «circle» connecting the anterior and posterior circulations inaugurated a paradigm of cerebral perfusion resilience [1]. Over ensuing centuries, anatomical and physiological studies have entrenched the notion that this ring functions as a safety valve: in the event of occlusion or narrowing in one vessel, blood is presumed to divert through communicating branches to maintain downstream perfusion [2].

However, a more nuanced view has gradually gained traction. Anatomical surveys and imaging studies show that a "classical" complete COW configuration is present in only a minority of individuals, and many variants exhibit hypoplastic or absent segments [3]. The mere presence of all communicating arteries does not guarantee functional effectiveness of the network. Clinical observations have revealed that even when the anatomical structure appears favorable, compensatory flow during vascular occlusion may be insufficient, especially in the face of comorbid vascular disease, age-related changes, or hemodynamic constraints. These findings cast a degree of doubt on the canonical idea of the COW as a fully reliable, self-regulating collateral system [4].

The central question thus arises: should we conceive the Circle of Willis primarily as a functional, dynamic unit, continuously participating in cerebral hemodynamics under variable conditions, or rather as an anatomical scaffold with limited or conditional functional relevance – activated only under pathologic stress? In other words, to what extent is the COW truly a "functional unit," and to what extent is that an idealized construct?

This article seeks to explore this question by integrating historical perspectives, morphologic variability, experimental and in vivo hemodynamic data, and clinical correlates. We will critically examine supporting and opposing evidence for the functional paradigm, analyze contexts in which the COW performs (or fails to perform) as a collateral network, and propose criteria by which the functional integrity of the COW might be appraised in individual patients. Through this synthesis, we aim to delineate when the Circle of Willis is truly a functional safeguard, and when its role remains more theoretical than operative.

Anatomical and Morphological Basis

The classical description of the Circle of Willis portrays it as a polygonal arterial anastomosis at the skull base, linking the anterior and posterior circulations. In its canonical form, it involves the following components (from anterior to posterior): both anterior cerebral arteries (A1 segments), connected by the Anterior Communicating Artery (AComA); bilateral internal carotid arteries (ICAs), which give off Posterior Communicating Arteries (PComAs) connecting to the posterior cerebral arteries (P1 segments); and the basilar artery bifurcating into bilateral posterior cerebral arteries (P2 territories). This circular network is thought to facilitate redistribution of blood in the event of flow compromise in one branch, by providing alternative routes via communicating arteries [5].

Yet, the textbook representation functions more as an idealized template than as a faithful account of human variation. In vivo, the COW seldom conforms perfectly to that symmetric, closed-loop model. The classical architecture is best viewed as a referential standard against which variants can be contrasted [6].

A key foundation for assessing functional potential is the frequency with which an "intact" COW is observed. Numerous autopsy and imaging studies demonstrate that the textbook complete circle – defined as all major communicating and primary segments present and of sufficient caliber – is relatively uncommon [7,8]. For example, a meta-analysis by estimated that about 68.22% ± 14.32% of examined specimens exhibit some form of variation, meaning that perfectly symmetric COW configurations are in the minority [9]. Variants commonly involve hypoplasia or absence of communicating arteries, especially the PComAs.

Large population imaging studies corroborate this. In the Tromsø Study, reported a wide diversity of COW morphologies, with many participants exhibiting one or more missing or hypoplastic segments [10]. In a Magnetic Resonance Angiography (MRA) cohort of 867 patients, identified a broad spectrum of anatomical variants, confirming that deviations from the canonical model are frequent in a normal population [6]. recently proposed a refined classification of COW variants, underscoring the ubiquity of asymmetry, segmental hypoplasia, and nonstandard branching patterns [4].

Some more recent cadaveric work suggests that the proportion of "complete" COWs may be higher in specific populations: for instance, report ~62.7 % of specimens in their series had a full circle, while 37.3 % had partial variants (with posterior communicating artery hypoplasia being the most frequent deviation) [11]. However, the definition of "complete" varies between studies (e.g. diameter thresholds, inclusion/exclusion criteria), so cross-study comparisons warrant caution.

Because variation is the norm rather than the exception, taxonomies have been developed to systematize COW morphologies. proposed a hierarchical classification in which variants are grouped according to which communication or interlinking segments deviate from the standard pattern (e.g., absence, hypoplasia, duplication) [4]. Key variant categories include:

- Hypoplasia or aplasia of PComA(s) – the posterior communicating arteries are among the most variable; one or both may be underdeveloped or wholly absent.
- Hypoplastic or missing AComA / A1 segments – absence or reduced caliber of the anterior interconnection diminishes the ability to cross-flow between anterior circulations.
- Fetal-type PCA – the P1 segment is diminished or absent, making the PCA perfused predominantly via the PComA, effectively converting that side's flow dependency to the internal carotid system.
- Asymmetry in segment size or dominance – one side's vessel may be larger (dominant) and better able to supply cross-flow, while the other side is diminutive.
- Duplications, fenestrations, or accessory branches – morphological variants that alter the topology but may or may not contribute functionally.
- Unilateral or bilateral absence of segments – leading to open or incomplete ring configurations (i.e. "C-shaped" circulations).

Because many studies define hypoplasia by arbitrary diameter or proportional thresholds (e.g. less than 1 mm or < 50 % of the contralateral vessel), consistency across literature is limited, and classification schemes sometimes diverge.

Having a connecting vessel in the COW does not by itself guarantee functional efficiency. The morphological details – diameter, length, curvature, branching angles – strongly influence whether a communicating conduit can carry meaningful flow under stress. Some of the relevant morphometric and topological considerations are:

- Lumen diameter: flow resistance scales roughly with the inverse fourth power of radius (by Poiseuille's law), so even small reductions in diameter sharply diminish potential collateral flow.
- Length and tortuosity: a longer or more tortuous pathway imposes greater resistance, making it less favorable in hemodynamic redistribution.
- Branching angles and junction geometry: acute take-off or sharp angles at junctions may produce unfavorable flow separation or pressure losses, reducing the effective contribution of a collateral route.
- Pressure gradients and pulsatility: effective collateral flow depends on favorable transsegmental pressure differentials; in some cases, pulsatile pressure mismatches can hinder rather than help flow redistribution.
- Wall compliance, vessel stiffness, and vascular remodeling: with age or vascular disease, vessel walls stiffen or remodel, which may further constrain flow capacity through previously patent communicating arteries.

These geometric and mechanical constraints mean that many anatomical "connections" in the COW may lie functionally silent under normal conditions, only recruited when pressure conditions shift sufficiently [12,13].

Another complicating factor is that the COW does not operate in isolation. The brain's collateral circulation relies on multiple overlapping systems (e.g. leptomeningeal (pial) collaterals, external–internal carotid anastomoses). Thus, in many individuals, the COW may be only one element of a distributed collateral network [14].

Moreover, because anatomical variants are frequent, the effective "structural reserve" of the COW may lie dormant under normal perfusion states; it may be invoked only when primary pathways fail. In those cases, only those communicating vessels whose morphometry and topology are favorable may contribute meaningfully. That is, not all anatomical continuity equates to functional continuity.

Understanding this architecture and its limitations sets the stage for interpreting hemodynamic studies and clinical data about when

(and in whom) the Circle of Willis truly behaves as a functional unit rather than a vestigial scaffold.

Hemodynamic Concept

The traditional interpretation of the Circle of Willis posits it as a collateral hub capable of redistributing Cerebral Blood Flow (CBF) when a primary vessel is compromised. In a fully patent COW, flow diverted from a stenosed or occluded artery is thought to follow alternative low-resistance paths via communicating branches, thereby mitigating ischemia in distal territories. Under idealized assumptions (Rigid Vessels, Laminar Flow, Negligible Pulsatility), one may represent the COW as a network of resistors in parallel and series; in such a simplified circuit, flow follows paths of least hydraulic resistance, proportional to pressure gradients and inversely proportional to resistance (Per Poiseuille's Principle). However, real physiology complicates this picture: vessels are compliant, flow is pulsatile, and flow separation or nonlinear effects may occur. The capacity of a communicating artery to serve as a collateral depends not only on its presence, but on the magnitude of allowable pressure gradients, the resistance of alternative paths, and dynamic changes during stress.

From a hemodynamic perspective, the key determinants of the COW's collateral function include:

- Pressure gradients across communicating segments. Any flow diversion requires a driving pressure differential; if upstream pressures decline symmetrically, little driving force remains.
- Hydraulic resistance of communicating vessels: governed by lumen diameter, length, tortuosity, and branching geometry.
- Relative resistances in competing pathways. If alternative collateral routes (e.g. leptomeningeal collaterals) or native vascular beds offer lower resistance, flow may prefer those paths over COW channels.
- Vessel wall compliance and dynamic changes – arterial compliance, vasomotion, and autoregulatory mechanisms can modulate resistance in situ.
- Temporal and pulsatile elements: inertial, inertance, viscous and unsteady components can affect transient flow redistribution, especially during acute changes.

In short, the COW behaves as a dynamic vascular network rather than a static anatomic bypass. Its functional capacity is contingent on instantaneous hemodynamic states, including systemic blood pressure, collateral tone, and interregional gradients.

Because in vivo direct measurement of flow redistribution across communicating arteries is challenging, Computational Fluid Dynamics (CFD) and in vitro models have become indispensable tools to test hemodynamic hypotheses.

Constructed physical models of the COW and examined flow partitioning under simulated occlusions, revealing that the efficiency of collateral transfer is highly sensitive to the diameters and connectivity of communicating arteries [15]. Their results suggest that collaterals do not always suffice to restore symmetric flow: some communicating vessels effectively lie "silent" unless pressure drops are steep.

More recently, used idealized CFD models to simulate combined Internal Carotid Artery (ICA) stenosis and absence of specific communicating segments. They observed that elimination of certain vessels (e.g. LP1, RA1) in the COW could mimic the effect of significant ICA stenosis, reducing total CBF and altering shear indices downstream [16]. The study also documented how increased stenotic severity shifted regions of low Time-Averaged Wall Shear Stress (TAWSS) and elevated Oscillatory Shear Index (OSI) into larger vascular territories.

Using mathematical network models, explored whether collateral flow through communicating arteries is necessary for normal perfusion. Interestingly, they concluded that in healthy subjects, the COW's communicating branches may not be substantially "active" in routine conditions – the system's native arterial pathways are often sufficient to satisfy baseline demand [17]. This view tempers the notion of a constantly active collateral network, positing that COW segments lie dormant until challenged.

In the context of hypoplastic or stenotic segments, computational studies of have shown that reduced caliber in connecting arteries substantially increases resistance, limiting effective collateral throughput [18]. In such cases, flow redistribution is constrained or even negligible under moderate pressure differentials.

Further emphasized the sensitivity of modeled flow patterns to the choice of CoW geometry. Their comparisons between full-circle, half-circle, and asymmetric models in aneurysm contexts reveal that omitting segments alters pressure and flow fields significantly, underscoring that small structural differences may yield large hemodynamic consequences [19]. Finally, documented that not only is COW flow adaptable acutely, but the network may exhibit plastic remodeling in response to altered flow distribution (e.g. after device placement) collateral patterns evolve dynamically over time, not purely instantaneously [20].

An important dimension of the hemodynamic concept is that communicating arteries must surpass a functional threshold to contribute meaningfully. studied thresholds for cross-flow in human COW specimens and proposed that effective collateral flow requires communicating artery diameters between approximately 0.4 and 0.6 mm [21]. Below this caliber, resistance is so high that diverted flow is negligible under physiologic gradients. This threshold concept helps explain why many anatomical "connections" fail to function in practice.

Another limitation arises from pressure decay along collateral conduits. If pressure losses across a communicating branch exceed the pressure gradient to distal beds, flow cannot be maintained. In the case of severe stenosis or near-occlusion, the driving force may collapse, and even an anatomically intact COW may not suffice to sustain perfusion.

CFD and network models highlight nonlinearity: marginal changes in stenosis severity or vessel diameter can disproportionately degrade collateral capacity. For instance, flow may decline precipitously rather than linearly once resistance passes a tipping point, owing to cascading pressure drops. Moreover, interactions between ipsilateral and contralateral flow, steal phenomena, and dynamic autoregulation complicate straightforward predictions.

Collateral recruitment via the COW is not necessarily instantaneous or static. Several factors modulate its activation over time:

- Autoregulatory adjustments, when distal arterioles vasodilate or vasoconstrict in response to perfusion pressure changes, altering local resistances downstream and influencing driving gradients through communicating arteries.
- Vasoactive responses, when local production of nitric oxide, metabolic byproducts, and neurovascular coupling may change vessel tone and effective resistance dynamically.
- Temporal delay or latency, when activation of collateral flow may require seconds to minutes, especially in scenarios of

gradual vascular compromise.
- Pulsatile fluctuations and inertial effects, when instantaneous flow reversal, oscillations, or unsteady wave reflections may transiently oppose or augment collateral recruitment.

Thus, the COW must be understood not only in a static snapshot but as a flow system evolving over time, responding to hemodynamic perturbations.

Clinical Observations and Paradoxes

One of the most cited clinical associations is the relationship between anatomical variants of the Circle of Willis and the incidence or severity of ischemic stroke. Several large imaging and angiographic studies have demonstrated that patients who suffer ischemic stroke more often present with one or more noncanonical COW variants compared to control populations. For example, reported that 62.1 % of acute ischemic stroke patients had at least one vascular variant in the COW, versus 54.8 % of age- and sex-matched controls ($p < 0.01$) [22]. Similarly, found that the presence of a variant, incompleteness, or hypoplasia anywhere in the COW was associated with 1.4-fold increased odds of ischemic stroke [23].

Such epidemiologic findings suggest that a less robust COW may predispose to cerebral ischemia, particularly when additional vascular risk factors or stenoses are present. However, the strength of association is modest and often confounded by age, atherosclerosis burden, hypertension, and other cerebrovascular risk factors.

Beyond risk predisposition, several studies have probed whether the morphological integrity of the COW affects clinical outcomes, infarct size, and collateral compensation during acute ischemia. The results, however, are not uniformly consistent, hence the term "paradoxes" in this context.

In a systematic review of 11 studies (n = 4,643), determined that COW integrity plays a significant role in collateral flow, especially in the context of distal Internal Carotid Artery (ICA) T-occlusion; the presence of a fully developed contralateral A1 segment and an intact anterior communicating artery (AComA) were associated with more favorable outcomes [24]. Yet, in occlusions at the M1 segment (middle cerebral artery), COW configuration appeared less impactful on outcomes, likely because the COW lies upstream and is less directly engaged in distal MCA occlusion dynamics.

On the other hand, reported that COW variants were not significantly associated with functional outcome in patients with ischemic strokes in some settings, raising the possibility that COW morphology is not always a decisive factor [25]. In a similar vein, found that while incomplete COW variants were more frequent in ischemic stroke cases, the link to worse prognosis was not consistent across all anatomical variant types and stroke subtypes [26].

A more recent study by attempted to stratify COW into subtypes and examine 90-day outcomes in acute ischemic stroke, reinforcing that COW configuration has prognostic relevance but again highlighting nuance and dependence on occlusion location and collateral context [27].

Thus, clinical observations present a dual picture: in some scenarios, COW integrity is a significant modifier of stroke severity and recovery; in others, its influence is attenuated or masked by other collateral pathways, vascular reserve, or the particular pattern of occlusion.

Several paradoxical observations challenge the simple narrative of "better COW → better collateral protection."

Paradox A: Fully intact COW but insufficient compensation. There are documented cases where patients with a morphologically complete COW (by angiographic criteria) nevertheless suffer large infarcts or poor outcomes after proximal vessel occlusion. This suggests that anatomical presence does not guarantee functional adequacy, due to limitations in vessel caliber, pressure gradients, or dynamic resistance.

Paradox B: Incomplete COW with preserved perfusion. Conversely, some individuals with apparently incomplete or hypoplastic COW configurations remain asymptomatic despite high-grade stenoses or occlusions of major cerebral arteries, presumably because alternative collateral systems (e.g. leptomeningeal arteries) compensate sufficiently, or because hemodynamic stress is not reached.

Paradox C: Thrombus migration and "paradoxical" infarction patterns. In some acute stroke cases, thrombus migration leads to unexpected infarct distribution that is not congruent with angiographic anatomy. A paradox discussed in the literature is that an initial proximal occlusion may seem well compensated by COW collaterals, but subsequent distal embolization leads to infarcts even in territories that "should" have been protected. This phenomenon complicates interpretation of imaging and weakens simple morphological predictions.

Earlier classic work by established that nonfunctional or "collateral deficient" COWs are more common in ischemic stroke patients than in controls. Using transcranial Doppler with carotid compression tests, the authors found that a nonfunctional anterior collateral pathway was present in 33 % of stroke cases versus 6 % of controls ($p < 0.001$), and nonfunctional posterior pathways were more prevalent in cases than controls (57 % vs 43 %) [28]. In patients with severe ICA stenosis or occlusion, absence of functional anterior CoW collaterals conferred an odds ratio of 7.33 for ischemic stroke. This underscores the notion that functional (not merely anatomical) deficiency can represent an independent risk for cerebral ischemia when major vessels are compromised.

The constellation of findings and paradoxes militates for caution in interpreting COW morphology in clinical practice. Some considerations are:
- Anatomy is necessary but not sufficient. A favorable anatomical COW does not guarantee effective functional collateral recruitment when challenged.
- Context matters. The impact of COW variants depends heavily on occlusion site, collateral reserve elsewhere (leptomeningeal, extracranial/intracranial anastomoses), systemic perfusion pressure, and vascular disease burden.
- Dynamic compensations may confound static imaging. A snapshot angiogram may not reflect latent collateral activation or deferred recruitment.
- Heterogeneity in definitions and measurement: differences in how hypoplasia, completeness, or "functional sufficiency" are defined hamper comparisons across studies.
- Selection bias and reverse causality, i.e patients with more severe vascular disease and age often have more COW variants, so attributing causality is complex.

In sum, clinical observations support that COW morphology has prognostic and risk-modifying relevance in many, but not all, cases. The observed paradoxes emphasize that the Circle of Willis functions as a dynamic system whose effectiveness is constrained by hemodynamics, alternative collaterals, and individual vascular status. The next section will explore contemporary debates and theoretical arguments for and against considering the COW a true functional unit.

Modern Debates

A central dialectic in contemporary discussion revolves around whether the Circle of Willis should be regarded as a constantly active component in cerebral hemodynamics, or as a reserve, "on-demand" system that becomes relevant only under pathological stress. Some authors argue for a continuous role: that even in non-occlusive conditions, the COW contributes to balancing pressure fluctuations, smoothing pulsatile flow, and maintaining uniform perfusion. proposed that the COW may act not merely as a collateral backup but also as a "pressure dissipating" network, transmitting pressure waves rather than substantial flow under normal conditions [29]. In contrast, others suggest that in the healthy brain, the communicating arteries are largely dormant, and only activate meaningfully when major flow imbalances arise.

This debate has practical implications. If the COW is functionally engaged even in baseline states, then morphological variation may always be hemodynamically consequential; by contrast, if its function is largely latent, one may tolerate greater anatomical deviation without undue risk unless challenged by disease.

A continuing tension lies between anatomical classification and functional (hemodynamic) significance. Many recent studies caution against equating morphological "completeness" with functional sufficiency. For instance, emphasize that variant classification is descriptive and does not necessarily reflect flow competence [4]. Computational works further demonstrate that absence or hypoplasia of certain segments may mimic the hemodynamic effect of significant arterial stenosis even if the rest of the COW appears structurally intact [16].

In practical terms, a radiologist or clinician may observe a "complete" COW in imaging, but that does not guarantee that collateral flow will be sufficient in an occlusive event, especially if communicating branches are small in diameter, tortuous, or subject to stiffening. Conversely, modest anatomical deviations may be functionally benign in many patients, buffered by other collateral networks.

In modern discourse, imaging modalities (CTA, MRA, 4D flow MRI) and computational modeling are increasingly central. The ability to simulate individual patient vascular geometry with hemodynamic flow prediction challenges simplistic morphology-based interpretations.

observed that many hemodynamic studies adopt full or half models of COW indiscriminately when building computational meshes, a practice they critique for overestimating or misrepresenting collateral flow in variant anatomies [19]. They recommend tailoring the model structure according to actual anatomical data to avoid bias.

Moreover, advances in 4D flow MRI (not yet widely adopted) may allow in vivo assessment of flow through communicating arteries under various physiological and stress conditions, potentially bridging the gap between morphology and function. These techniques may help predict which anatomical variants are at risk for failure during occlusion events.

Another strand of contemporary debate frames the COW not merely as a collateral structure but as an evolutionary remnant or developmental artifact. Some argue that the COW's original biological role may have less to do with stroke protection and more with embryonic vascular development, pulsation dampening, or pressure equalization across hemispheric circulations (especially in nonhuman vertebrates) [30]. In this view, the collateral function is secondary, co-opted in pathological states rather than designed as its primary role.

Furthermore, the embryologic origin of COW segments influences their morphologic variability and capacity to remodel. discuss how developmental vascular pathways and remodeling, under interplay of hemodynamic forces and genetic programming, shape adult variation, and may predispose certain configurations to vulnerability [31].

From a clinical standpoint, the debate centers on whether COW morphology should be integrated into prognostic and therapeutic algorithms. Proponents argue that understanding a patient's COW architecture (and ideally functional capacity) could refine risk assessment for stroke, guide revascularization decisions, or tailor therapeutic thresholds for carotid or intracranial stenosis.
Critics caution that until functional validation is routine (e.g. flow measurements in vivo), reliance on morphology alone may mislead. COW variation is common in healthy populations, and many individuals with "incomplete" circles never manifest symptomatic ischemia. The sensitivity and specificity of COW morphology as a prognostic biomarker remain under investigation.

The modern debates underscore that the Circle of Willis sits at the intersection of anatomy, hemodynamics, and clinical neurology. Some emerging principles and open debates include:

- Functional latentism, likely, the COW is not fully active in baseline states but acts as a reserve that is variably recruited depending on hemodynamic stress.
- Morphology is a necessary but insufficient predictor. Structural integrity must be considered in the context of vessel caliber, compliance, and competing collateral pathways.
- Modeling and advanced imaging hold promise: integrating patient-specific computational models and in vivo flow data may bridge the gap between morphology and functional prediction.
- Evolutionary and developmental context matters. The COW's variable architecture may stem from developmental constraints and evolutionary tradeoffs, not purely optimized collateral design.
- Clinical utility demands rigorous validation: before COW morphology becomes a standard prognostic tool, more prospective studies correlating structural, functional, and outcome data are needed.

Conclusion

The question at the heart of this article "Circle of Willis: myth or functional unit?" cannot be settled by a simple yes or no. Rather, the evidence suggests a more nuanced view: the Circle of Willis is a conditional, context-dependent functional reserve rather than a universally operative, self-regulating arterial loop.

Anatomically, the COW is highly variable. A "perfect" ring is relatively uncommon, and many individuals harbor hypoplastic, asymmetric, or absent communicating segments. These morphological deviations impose real constraints on collateral capability: even when a connecting vessel is macroscopically present, its geometry, lumen size, tortuosity, branching angle, and stiffness may render it effectively nonfunctional in stress situations.

Hemodynamic theory and computational modeling support this conditional collateral role. Under baseline conditions, communicating arteries may play a marginal role; only when perfusion pressure gradients become significant does collateral flow become recruited, and only along those paths whose hydraulic resistance is sufficiently low. The COW thus acts as a latent network – silent in health, engaged only when needed.

Clinically, empirical observations and paradoxes reinforce the conditional paradigm. Some patients with structurally "ideal" COWs suffer large infarcts despite that anatomy, while others with incomplete or variant COWs remain asymptomatic under vascular challenge. COW integrity exerts measurable influence in many¸ but not all, stroke scenarios, especially in proximal vessel occlusions, but its predictive power is moderated by alternative collateral networks, systemic perfusion pressure, and microvascular reserve.

Modern debates emphasize that morphology and function must be bridged: a patent communicating artery is not synonymous with functional competence. Advances in imaging (particularly 4D-flow MRI) and patient-specific computational modeling hold promise for more reliably predicting which COW architectures are likely to function as effective collaterals in an individual patient. Integrating structural and functional data might allow clinicians to stratify risk and guide therapeutic decision making (e.g. carotid revascularization timing, endovascular planning).

In practice, the COW is best conceptualized as a contingent safeguard, not an ever-active circulatory lifeline. Under favorable geometry and in the face of vascular stress, it can meaningfully redistribute flow but only within the bounds imposed by vessel design, hemodynamics, and collateral redundancy beyond the COW itself.

In closing, the Circle of Willis is neither pure myth nor perpetual guardian. It is a vascular reserve system whose efficacy emerges under stress, not as a constant highway. Future research, especially longitudinal clinical studies correlating structure, flow, and outcomes, is essential to refine when and for whom the COW truly functions as the collateral backbone of cerebral circulation.

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