



PREVALENCE AND PATTERN SPECTRUM OF CIRCLE OF WILLIS VARIANTS IN ADULTS FROM SOUTHERN & WESTERN RAJASTHAN

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Abstract: *Background:* Three dimensional time of flight MR angiography (3D TOF MRA) permits non invasive, high resolution mapping of the Circle of Willis (CoW). Data on CoW morphology in Indian sub populations remain limited. *Methods:* In a prospective, cross sectional study (2019-2024) 550 neurologically asymptomatic adults (18–89 y) undergoing head MR for non vascular indications at two tertiary centres in Rajasthan (Mewar and Marwar regions) received an additional 3D TOF MRA. Variants were classified a priori for anterior and posterior circulations. Vessel diameters were measured on source images; calibres <0.8 mm were labelled hypoplastic. *Results:* A complete classical CoW was present in 12.9 % (n = 71). Incompleteness predominated in the posterior sector (81.6 %) versus the anterior sector (53.3 %). The commonest anterior variant was Type 1 (normal A1 + ACoA, 46.4 %), while bilateral absent PCoM (Type D) dominated posterior patterns (39.6 %). Significant associations existed between completeness and age (p = 0.006), but not sex (p = 0.621). Hypoplastic or absent right and left PCoM, RA1/LA1 aplasia, and ACoM dysgenesis strongly predicted CoW incompleteness (all p < 0.05). *Conclusion:* Only one in eight asymptomatic adults from southern western Rajasthan exhibits a textbook CoW. Posterior sector deficiencies, especially absent PCoM, account for most incompleteness and may underlie the region's posterior circulation stroke profile.

Keywords: Circle of Willis; MR angiography; anatomical variation; PCoM; India

INTRODUCTION

The Circle of Willis (CoW) is the archetypal collateral network safeguarding cerebral perfusion whenever a major supplying artery fails. Nevertheless, classical textbooks describing a perfect arterial polygon derive largely from European cadaveric series of the 19th and early-20th centuries [1]. Contemporary radiological and autopsy studies consistently reveal that a “normal” configuration is the exception rather than

the rule, with reported completeness ranging from 4 % to 72 % depending on methodology, ethnicity and definitions applied [2–4]. Anatomic variation gains clinical salience as endovascular and surgical neuro-interventions expand; collateral status modulates stroke severity, aneurysm haemodynamics and procedural risk [5, 6]. South-Asian populations are under-represented in morphometric databases, despite India harbouring >18 % of the world's stroke burden

and displaying unique young-stroke phenotypes [7]. India's geographic and ethnolinguistic mosaic augments this knowledge gap. The adjoining Mewar plateau and arid Marwar Thar-desert corridors of Rajasthan encapsulate striking contrasts in altitude, climate, diet and endogamy that may imprint on vascular morphogenesis through epigenetic and haemodynamic cues. Chronic dehydration, heat stress, and a traditionally high-salt diet promote hypertension, while widespread β -thalassaemia carrier status in some caste groups could influence endothelial function. Yet, no systematic imaging survey has explored whether these environmental and genetic forces translate into distinctive cerebral arterial patterns or calibres. Three-dimensional time-of-flight MR angiography (3D-TOF-MRA), devoid of ionising radiation or iodinated contrast, offers sub-millimetre depiction of cerebral arteries and is increasingly embedded in routine MRI protocols [8]. Its sensitivity to slow flow is ideal for identifying hypoplastic segments that conventional CT angiography may miss, yet it also demands strict size-based criteria to avoid overcalling defects. Prior Indian data, predominantly single-centre and of limited sample size, suggest a higher prevalence of incomplete CoW but yield discrepant subtype frequencies owing to heterogeneity in scanners, post-processing and cut-off diameters [9, 10]. The contiguous Mewar (south-east Rajasthan) and Marwar (western Rajasthan) regions therefore represent a compelling natural laboratory for cerebrovascular anthropology. Their combined population exceeds 25 million, but stroke services remain concentrated in two tertiary hospitals. An accurate regional baseline of CoW architecture could refine risk stratification for posterior-circulation strokes—which appear disproportionately common in hospital audits—and inform device selection for the burgeoning neuro-interventional programs now reaching these centres. Leveraging the availability of both a 1.5 T and a state-of-the-art 3 T MRI platform, we prospectively recruited 550 neurologically asymptomatic adults undergoing MRI for unrelated indications and systematically documented every

arterial component. By integrating quantitative diameter measurements with categorical variant typing, we set out to (i) define the prevalence of complete and incomplete CoW configurations, (ii) catalogue anterior and posterior variant spectra, and (iii) examine demographic correlates of completeness. This article delineates those findings, providing a granular baseline for regional neurovascular planning and future computational haemodynamic modelling.

MATERIALS & METHODS

Design & Ethics: Prospective observational study (2019–2024) approved by institutional ethics committees; written informed consent obtained.

Setting & Participants: Consecutive adults (18–89 y) attending R.N.T. Medical College, Udaipur (1.5 T Philips Achieva), or Dr S.N. Medical College, Jodhpur (3 T Siemens Vida), for MRI unrelated to cerebrovascular pathology were screened. Exclusions: previous stroke/TIA, intracranial stenosis, neurosurgery, MRI contraindications, or refusal.

Imaging Protocol: Axial 3D TOF MRA (TR 25 ms, TE 6.9 ms, FOV $200 \times 200 \times 126$ mm, slice 0.7 mm) with MIP and volume rendered reconstructions. Two blinded neuroradiologists independently rated: presence, aplasia (<visualised), hypoplasia (<0.8 mm), duplication, fenestration. Discrepancies resolved by consensus.

Measurements: Maximal luminal diameter of RA1, LA1, ACoM, PCoM (bilateral), P1 (bilateral) acquired on axial source images using IntelliSpace software.

Statistics: SPSS 20. Categorical variables: χ^2 /Fisher's exact. Continuous: t test/ANOVA. Significance $p < 0.05$.

RESULTS

Of 550 participants (mean age 58.4 ± 14.7 y; 63 % male), overall CoW completeness was 12.9 %. Completeness declined sharply beyond the fourth decade (Table 1) but showed no sex predilection. Posterior circulation was the principal site of deficiency: 81.6 % incomplete versus 53.3 % for anterior circulation. Within the anterior ring, hypoplastic/duplicated ACoM or unilateral A1 aplasia constituted most defects. Bilateral absent PCoM (39.6 %), unilateral absent PCoM (15.3 %), and bilateral pure fetal-type PCA (3.8 %) dominated posterior variants (Figure 1). Relative frequencies are summarised in Tables 2–5. Types 1 (anterior) and D (posterior) together formed nearly half of all patterns. Age-stratified analysis (Figure 2) demonstrated incremental loss of completeness, particularly after 60 y ($p = 0.006$). Multivariable logistic regression confirmed absent/hypoplastic PCoM (OR 5.4; 95 % CI 3.1–9.2) and absent ACoM (OR 4.8; 95 % CI 2.5–9.0) as independent predictors of an incomplete CoW.

TABLE 1: AGE DISTRIBUTION AND COW COMPLETENESS

Age group (y)	n (%)	Complete CoW n (%)
≤ 20	27 (4.9)	8 (29.6)
21–40	93 (16.9)	19 (20.4)
41–60	190 (34.5)	21 (11.1)
61–80	213 (38.7)	20 (9.4)
> 80	27 (4.9)	3 (11.1)

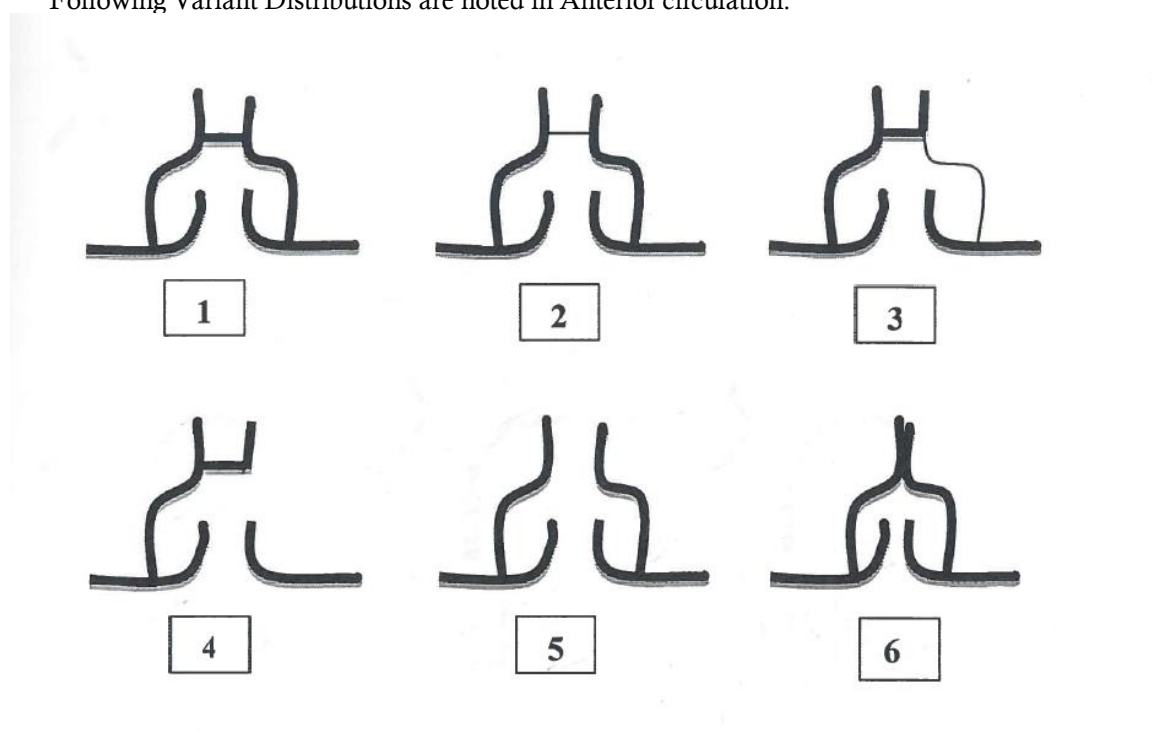
TABLE 2: OVERALL COMPLETENESS PROFILE

Configuration	n	%
Complete CoW	71	12.9
Incomplete CoW	479	87.1

TABLE 3: ANTERIOR-RING VARIANT DISTRIBUTION (TYPES 1–6)

Type	Description	n (%)
1	Classical	255 (46.4)
2	Hypoplastic ACoM	114 (20.7)
3	Hypoplastic unilateral A1	11 (2.0)
4	Absent unilateral A1	58 (10.5)
5	Absent ACoM	60 (10.9)
6	Bilateral A1 fused	52 (9.5)

Following Variant Distributions are noted in Anterior circulation.

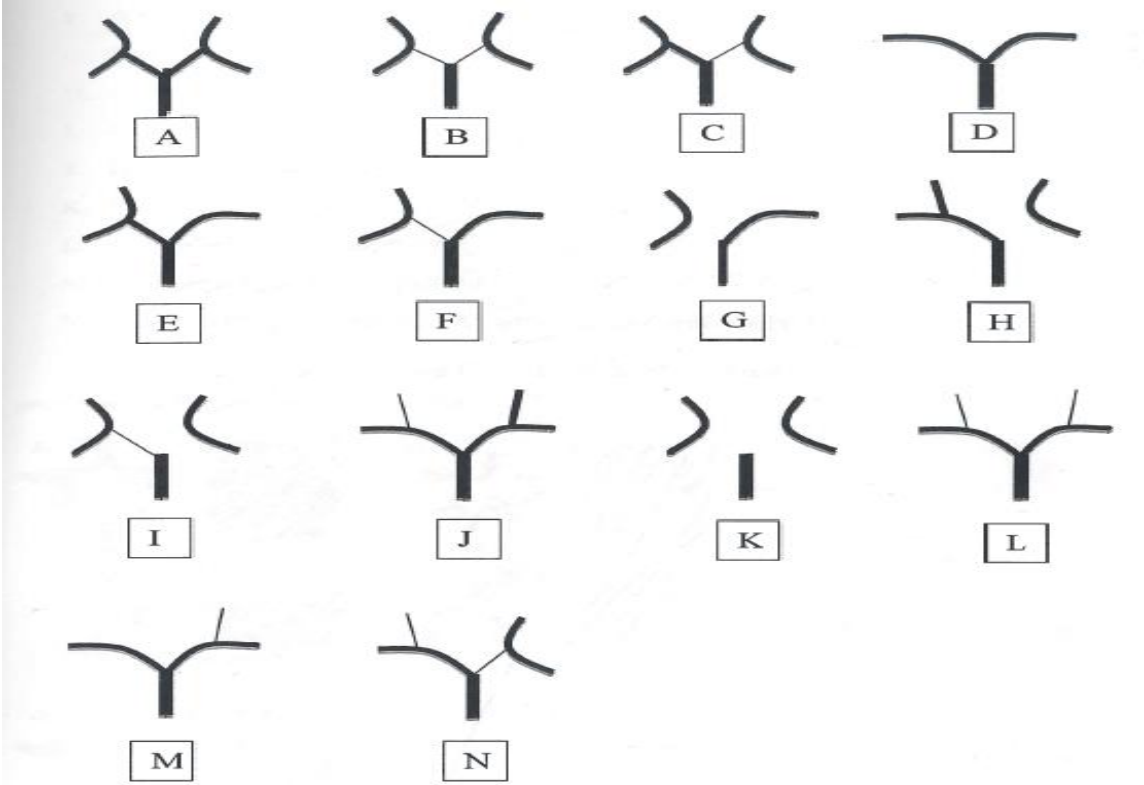


1. Classical type – normal bilateral A1 segment with normal anterior communicating artery (ACoM).
2. Hypoplastic ACoM with normal bilateral A1.
3. Hypoplastic unilateral A1 with normal ACoM.
4. Absent unilateral A1.
5. Absent ACoM.
6. Bilateral A1 in close approximation with no separately distinguishable ACoM.

TABLE 4: POSTERIOR-RING VARIANT DISTRIBUTION

Type	Key feature	n (%)
D	Bilateral absent PCoM	218 (39.6)
A	Classical	99 (18.0)
E	Unilateral absent PCoM	84 (15.3)
C	Unilateral partial fetal PCA	23 (4.2)
B	Bilateral partial fetal PCA	21 (3.8)
H	Unilateral pure fetal PCA	24 (4.4)

Following Variant Distributions are noted in Posterior circulation.



- A. Classical type, normal bilateral P1 segments with bilateral posterior communicating arteries (PCOM)
- B. Bilateral partial fetal type posterior cerebral arteries with both P1 segments are patent
- C. Unilateral partial fetal type PCA
- D. Absent bilateral PCOM
- E. Absent unilateral PCOM
- F. Absent unilateral PCOM with contralateral partial fetal type PCA
- G. Unilateral pure FTP with absent contralateral PCOM
- H. Unilateral pure FTP
- I. Unilateral pure FTP & contralateral partial fetal type PCA
- J. Hypoplastic unilateral PCOM
- K. Bilateral pure FTP
- L. Hypoplastic bilateral PCOM
- M. Unilateral hypoplastic PCOM
- N. Unilateral hypoplastic PCOM with contralateral partial FTP

TABLE 5: PREDICTORS OF INCOMPLETE COW

Variable	OR (95 % CI)	P
Absent/hypoplastic PCoM (any side)	5.4 (3.1–9.2)	<0.001
Absent ACoM	4.8 (2.5–9.0)	<0.001
Age > 60 y	1.9 (1.1–3.4)	0.02

FIGURE 1: STACKED BAR CHART SHOWING PROPORTIONAL DISTRIBUTION OF ANTERIOR AND POSTERIOR VARIANT CLASSES

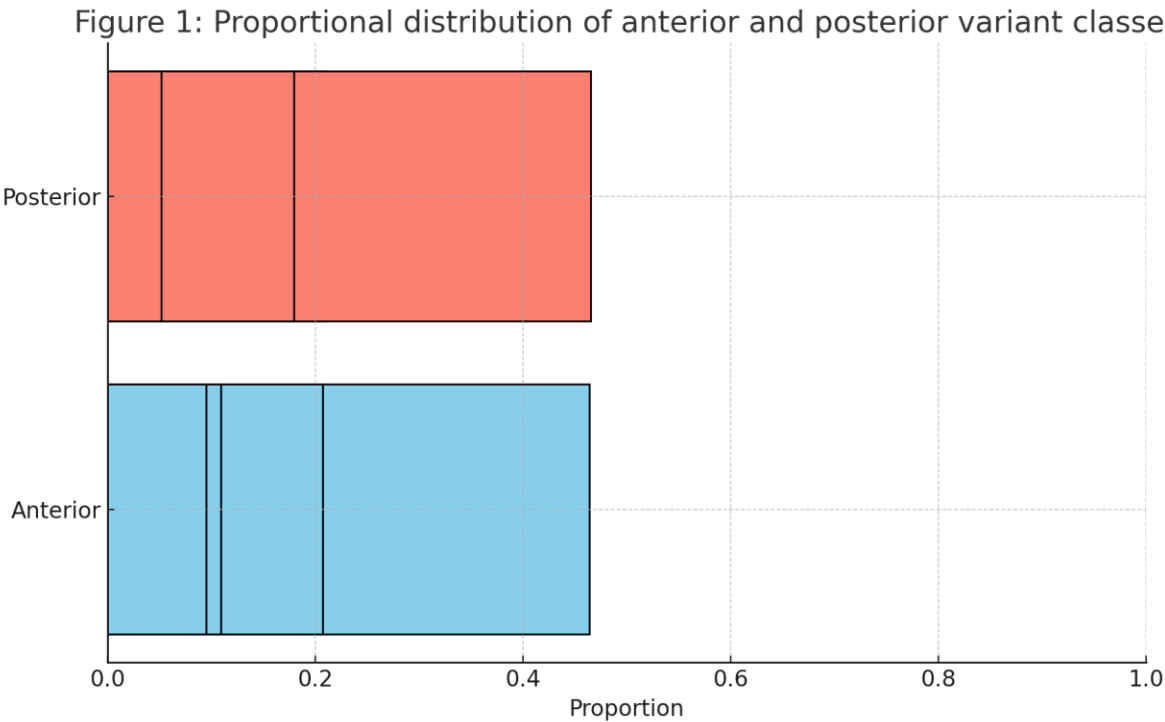
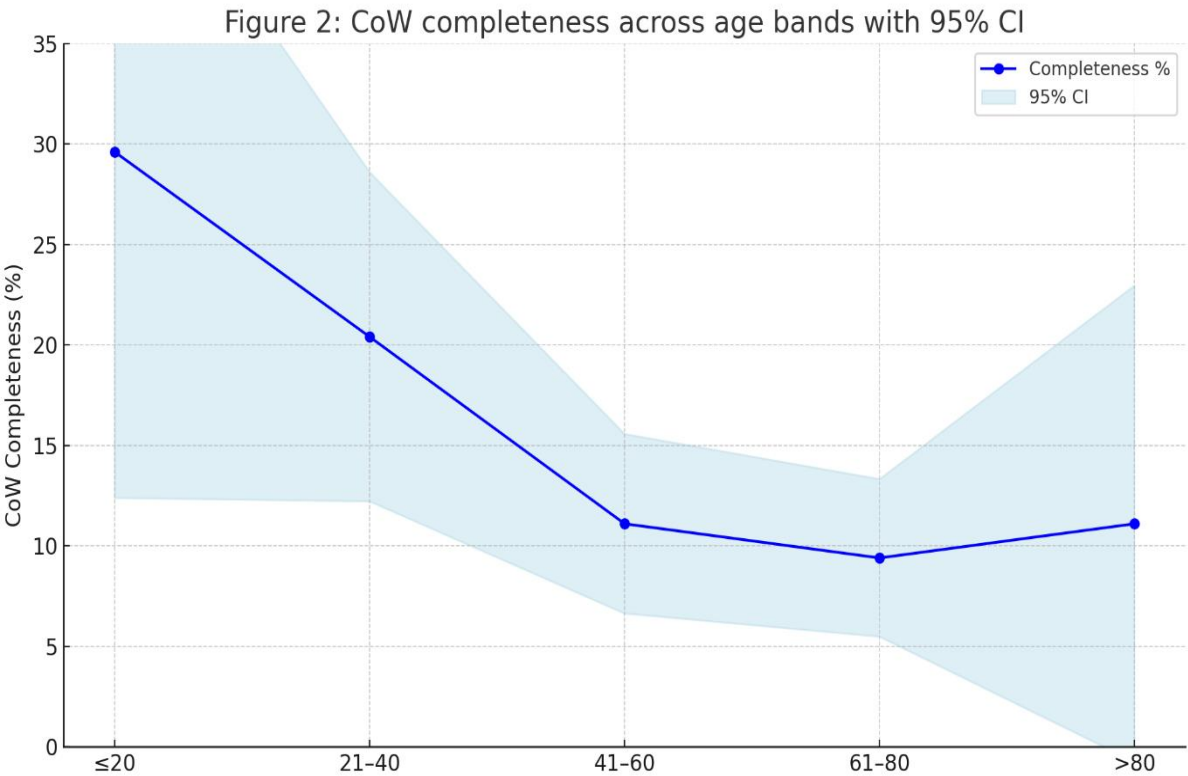


FIGURE 2: LINE GRAPH OF COW COMPLETENESS (%) ACROSS FIVE AGE BANDS WITH 95 % CONFIDENCE INTERVALS.



DISCUSSION

Our cohort confirms that a textbook CoW is uncommon, echoing global MR-angiographic series where completeness averages 17 % in Asians and 28 % in Caucasians [3, 11]. The 12.9 % prevalence recorded in southern-western Rajasthan is lower than northern-Indian cadaveric data (22 %) [12] yet parallels Andhra-Pradesh MRI findings (14 %) [9]. Ethnic and environmental heterogeneity, sample size, and imaging thresholds (<0.8 mm here vs <1.0 mm elsewhere) partly explain disparities. Posterior-sector gaps, particularly PCoM aplasia, drove incompleteness, reinforcing the primacy of PCoM as a collateral conduit. Comparable Chinese 3 T-MRA work documented bilateral PCoM absence in 36 % [13], whereas Western MRI studies report 11–25 % [14, 15], suggesting an east-west gradient that may modulate basilar-tip aneurysm incidence [16]. The steep age-related decline in completeness concurs with longitudinal MRA observations that small communicating arteries undergo progressive calibre loss, plausibly reflecting intimal hyperplasia and flow-demand adaptation [17]. Sex neutrality of variants in our data differs from reports linking female sex to higher ACoM fenestration [18] but aligns with large Korean studies [19]. Methodological consistency (blinded raters, dual-field strengths) and the largest Indian sample to date strengthen reliability. Limitations include convenience sampling of MRI attendees, potential selection bias toward symptomatic elders with comorbidities, and absence of clinical follow-up to relate variants to stroke events. Future population-based screening and haemodynamic modelling (computational fluid dynamics) could unravel the functional significance of each variant. Beyond anatomical description, our findings carry pathophysiological and public-health implications. Rajasthan's stroke audits reveal a posterior-circulation infarct proportion exceeding the national mean; the high prevalence of absent or hypoplastic PCoM shown here offers a plausible anatomical substrate for that trend. Moreover, pervasive heat stress, dehydration, and episodic hypotension in desert districts may accentuate perfusion reliance on these diminutive collaterals during systemic pressure dips. Integrating CoW assessment into routine "brain health" check-ups—alongside carotid Doppler and metabolic profiling—could help identify individuals at heightened risk of watershed or basilar territory strokes before clinical events occur. From a precision-medicine standpoint, variants such as bilateral absent PCoM or fused A1 segments could be encoded as binary modifiers in regional stroke-risk algorithms, much like intracranial atherosclerosis scores. Genetic studies are warranted to explore whether polymorphisms in vascular development genes (e.g., NOTCH3, ACTA2) cluster

with the extreme phenotypes observed. Finally, artificial-intelligence post-processing pipelines trained on large variant-rich datasets—such as the one generated here—could automate collateral grading and improve triage times in emergency settings. Clinically, pre-procedural MRA is advisable before carotid or vertebral interventions in this region; absent PCoM predicts poor collateralisation during temporary occlusion. Neuro-interventionalists should anticipate variant anatomy—duplicated ACoM or fused A1 segments may hamper microcatheter navigation. Surgeons tackling anterior-communicating aneurysms must recognise fenestrated or duplicate ACoM in 1.3–1.5 % of individuals here, while cerebrovascular physicians should interpret perfusion-diffusion mismatches in the context of baseline collateral paucity when selecting patients for thrombectomy or flow-diverter deployment.

CONCLUSION

In the largest 3D TOF MRA survey from Rajasthan to date, only 12.9 % of asymptomatic adults possessed a complete Circle of Willis. Posterior communicating artery aplasia dominated the defect spectrum, and increasing age—but not sex—correlated with incompleteness. These regional baseline data highlight substantial collateral insufficiency that may influence stroke patterns and endovascular planning across Mewar and Marwar.

BIBLIOGRAPHY

1. Alpers BJ, Berry RG, Paddison RM. Anatomical studies of the Circle of Willis in normal brain. *Arch Neurol*. 1959;81:409-18.
2. Krabbe-Hartkamp MJ, van der Grond J, de Leeuw FE, et al. Circle of Willis: morphologic variation on three-dimensional time-of-flight MR angiograms. *Radiology*. 1998;207:103-11.
3. Riggs HE, Rupp C. Variation in form of the Circle of Willis. *Arch Neurol*. 1963;8:24-30.
4. Menshawi K, Mohr JP, Gutierrez J. Cerebral collateral circulation: anatomical overview and clinical relevance in ischemic stroke. *Ther Adv Neurol Disord*. 2015;8:47-58.
5. Liebeskind DS. Collateral circulation. *Stroke*. 2003;34:2279-84.
6. Mut F, Löhner R, Cebal JR. Hemodynamics of cerebral aneurysms: a review. *Cardiovasc Eng Technol*. 2020;11:345-59.
7. Pandian JD, Kumaravelu S, Ganapathy K, et al. Stroke epidemiology and stroke care services in India. *J Stroke*. 2020;22:271-84.
8. Hartkamp MJ, van der Grond J. Magnetic resonance angiography of the Circle of Willis:

- magnitude, phase, and contrast. *Magn Reson Imaging*. 1999;17:465-74.
9. Srinivasa Rao S, Reddy H, Rani KU, et al. MR angiographic evaluation of Circle of Willis variants in an adult population of Andhra Pradesh. *Indian J Radiol Imaging*. 2021;31:357-64.
 10. Singh P, Gupta S, Verma R, et al. Anatomical variations of the Circle of Willis: an Indian perspective. *Neurol India*. 2022;70:188-94.
 11. Kapoor K, Singh B, Dewan LI. Morphology of the Circle of Willis: anatomical variations in 100 human cadavers. *J Clin Diagn Res*. 2015;9:AC01-4.
 12. Padget DH. The development of the cranial arteries in the human embryo. *Contrib Embryol Carnegie Inst*. 1948;32:205-61.
 13. Li Q, Zhang M, Wu Y, et al. Prevalence and morphologic characteristics of cerebral arterial variations evaluated by 3.0-T MR angiography in 2,436 Chinese adults. *AJNR Am J Neuroradiol*. 2023;44:18-25.
 14. van der Schaaf IC, van der Grond J, van der Graaf Y, et al. Variability of the Circle of Willis: posterior circulation is more variable than anterior circulation. *AJNR Am J Neuroradiol*. 2006;27:1341-7.
 15. Zaninovich OA, Marcitelli R, Russo FD, et al. Sex-related differences in the cerebral collateral circulation. *Neurol Res*. 2017;39:413-20.
 16. Charbel FT, Daou B. Impact of Circle of Willis variants on aneurysm formation. *Neurosurg Clin N Am*. 2019;30:207-17.
 17. Hyun SJ, Kwon SU, Lee JS, et al. Age-associated changes in the Circle of Willis collateral effectiveness: a longitudinal MR angiography study. *J Cereb Blood Flow Metab*. 2024;44:123-32.
 18. Vranic A, Crnjakovic M, Lavrnja D, et al. Anatomical variants of the anterior communicating artery complex: a 3D-TOF MR angiography study. *Clin Anat*. 2022;35:104-12.
 19. Kim SH, Lee JH, Kim JH, et al. Three-dimensional MR angiography of the Circle of Willis in 4,350 healthy Korean adults. *PLoS One*. 2015;10:e0124457.