Archives of Academic Emergency Medicine. 2026; 14(1): e43

REVIEW ARTICLE

Quantitative EEG and EEG-Derived Markers of Severity, Prognosis, and Recovery After Ischemic Stroke: A Systematic Review

Shirwan Hamasalih Omer[1*]

1. Department of Basic Medical Sciences, College of Medicine, University of Sulaimani, Sulaymaniyah, Iraq.

Received: June 2026; Accepted: July 2026; Published online: 24 August 2026

Abstract: **Introduction:** Electroencephalography (EEG) and quantitative electroencephalography (QEEG) may provide noninvasive functional biomarkers that complement clinical and imaging predictors when evaluating stroke outcomes. This systematic review evaluated QEEG, selected routine EEG, processed EEG, and advanced neurophysiological biomarkers of severity, lesion burden, longitudinal prognosis, cognitive/language outcome, rehabilitation response, and recovery after ischemic stroke. **Methods:** PubMed, Embase, Scopus, and Web of Science were searched on 30 March 2026 using ischemic stroke or cerebral infarction terms, QEEG or EEG-derived biomarker terms, and prognosis, outcome, severity, infarct-volume, mortality, rehabilitation, motor-recovery, and language-recovery terms. Selected structured routine EEG, visual EEG grading, processed EEG/Bispectral Index (BIS), and EEG-derived neurovascular-coupling studies were included when they provided predefined severity, prognostic, monitoring, cognitive/language, rehabilitation, or recovery-related evidence. The search identified 2,564 records; 851 duplicates were removed, leaving 1,713 unique records for screening. Evidence was classified as cross-sectional severity or lesion-burden association, longitudinal functional prognosis, mortality or clinical deterioration, rehabilitation or motor recovery, cognitive/language outcome, selected routine or processed EEG evidence, or advanced QEEG/connectivity/nonlinear/neurovascular-coupling evidence. Risk of bias was assessed using Quality in Prognostic Studies (QUIPS)-oriented criteria, and certainty was summarized using domain-level Grading of Recommendations Assessment, Development and Evaluation (GRADE)-informed judgments. **Results:** Thirty-three study records were included, representing 1,696 stroke or suspected-stroke participants, 1,598 EEG/QEEG/BIS/neurovascular coupling (NVC)-analyzable participants, and 317 controls or comparators. The most consistent prognostic pattern was that greater EEG/QEEG abnormality, particularly increased slow-wave activity and higher delta/alpha ratio (DAR) or delta-theta/alpha-beta ratio (DTABR), was associated with greater neurological severity, larger infarct or lesion burden, and poorer functional outcomes. Measures of interhemispheric imbalance, including a higher brain symmetry index (BSI), pairwise-derived brain symmetry index (pdBSI), and greater hemispheric asymmetry, were also generally associated with unfavorable outcomes, including greater disability, clinical deterioration, mortality, or less favorable recovery. Reduced alpha or beta activity was similarly associated with poorer neurological or functional outcomes in several studies. Post-endovascular treatment (EVT)/mechanical thrombectomy (MT) studies suggested potential monitoring value for detecting or predicting cortical dysfunction, early neurological deterioration, cerebral edema, nonconvulsive seizures/status epilepticus, infarct growth, and unfavorable 90-day outcome. Rehabilitation and advanced QEEG studies suggested that motor/language recovery may relate to interhemispheric balance, phase synchrony, weighted node degree, nonlinear complexity, and neurovascular coupling. However, the evidence was heterogeneous, mostly exploratory, and generally low to very-low certainty; no universal prognostic threshold or pooled prognostic estimate was supported. **Conclusions:** EEG/QEEG abnormalities may provide candidate severity association, monitoring, prognostic, and recovery biomarkers after ischemic stroke. This review distinguishes cross-sectional severity or lesion burden associations from longitudinal prognostic, monitoring, and recovery outcomes. However, current evidence remains heterogeneous and mostly low to very low certainty and does not support a universal prognostic threshold or global pooled prognostic estimate.

Keywords: Ischemic Stroke; Electroencephalography; Prognosis; Recovery of Function; Rehabilitation; Neurovascular Coupling

Cite this article as: Omer SH. Quantitative EEG and EEG-Derived Markers of Severity, Prognosis, and Recovery After Ischemic Stroke: A Systematic Review. Arch Acad Emerg Med. 2026;14(1):e43. https://doi.org/10.22037/aaem.v14i1.3043.

1. Introduction

Stroke remains a major cause of death and long-term disability worldwide, with its burden sustained by population growth, aging, and continued exposure to vascular risk factors (1). After ischemic stroke, clinicians must assess neurological severity, estimate functional prognosis, identify patients at risk of deterioration, and plan rehabilitation. These decisions cannot rely on anatomical lesion detection alone because patients with similar infarct volumes, vascular findings, or baseline severity may follow different recovery trajectories. Differences in cortical function, network integrity, comorbidities, treatment response, and early post-stroke physiology may contribute to this variability. Electroencephalography (EEG) and quantitative electroencephalography (QEEG) may therefore complement clinical examination and neuroimaging as functional biomarkers of stroke severity, prognosis, and recovery.

***Corresponding Author:** Shirwan Hamasalih Omer; Department of Basic Medical Sciences, College of Medicine, University of Sulaimani, Sulaymaniyah, Iraq. Phone: (+964) – 07701554828; Email: shirwan.omer@univsul.edu.iq; ORCID: https://orcid.org/0009-0004-7022-5157.

Early prognostic stratification influences monitoring, rehabilitation planning, complication prevention, and discharge decisions (2-6). The National Institutes of Health Stroke Scale (NIHSS), modified Rankin Scale (mRS), infarct volume, vascular imaging, and treatment variables provide essential clinical and structural information, but they do not directly measure the physiological state of surviving cortical networks. A repeatable bedside measure of brain function may be particularly useful when examination is limited by impaired consciousness, aphasia, fluctuating deficits, fatigue, or sedation.

EEG records cortical electrical activity with high temporal resolution, while QEEG converts these signals into numerical features, including band power, slow-to-fast ratios, hemispheric asymmetry indices, connectivity measures, nonlinear complexity metrics, and neurovascular-coupling indices (4-6). Cerebral hypoperfusion and metabolic stress are generally associated with increased slow-frequency activity and attenuation of faster rhythms (4-6). This pattern is consistent with ischemic penumbra physiology, in which neuronal electrical dysfunction may occur before irreversible structural injury (7, 8).

Among conventional QEEG markers, the delta/alpha ratio (DAR), delta-theta/alpha-beta ratio (DTABR), Brain Symmetry Index (BSI), pairwise-derived BSI, revised BSI, and laterality coefficients have been most frequently studied. These measures have been associated with NIHSS, mRS, infarct volume, Barthel Index, mortality, disability, motor impairment, and recovery outcomes (9-25). Studies performed after endovascular treatment or mechanical thrombectomy were included only when EEG/QEEG was related to neurological severity, infarct evolution, complications, functional outcome, or physiological recovery. Such studies were interpreted as prognostic or outcome-monitoring evidence rather than as evidence for primary treatment triage (15, 16, 26-31).

EEG-based markers have also been examined during rehabilitation. BSI, laterality measures, phase synchrony, weighted node degree, and fractal dimension have been evaluated in relation to motor impairment, upper-limb recovery, language improvement, brain–computer interface rehabilitation, and neural complexity (32-38). Coherence, phase synchronization, nonlinear metrics, and EEG–cerebral blood-flow velocity coupling may provide information about network integrity, interhemispheric communication, and neurovascular regulation (39-44). However, these approaches remain heterogeneous in acquisition methods, preprocessing, mathematical definition, timing, and outcome measurement.

Accordingly, this review distinguished concurrent associations with NIHSS or infarct volume from longitudinal associations with disability, mortality, cognitive or language outcomes, rehabilitation response, and recovery. The term EEG-derived biomarkers was used broadly to include conventional QEEG, selected routine or processed EEG measures, visual EEG grading, and neurovascular-coupling indices when they provided extractable evidence on severity, lesion burden, prognosis, post-treatment monitoring, rehabilitation, or functional recovery after ischemic stroke.

2. Methods

2.1. *Design and reporting framework*

This systematic review evaluated EEG and QEEG markers of severity and outcomes (lesion burden, longitudinal prognosis, mortality or deterioration, motor and language recovery, rehabilitation response, cognitive outcomes, and advanced neurophysiological recovery mechanisms) after ischemic stroke. The review was designed as a prognostic and recovery-focused evidence synthesis rather than a diagnostic accuracy review. The review followed Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 principles (45) and was informed by guidance for synthesis without meta-analysis when broad pooling is inappropriate (46, 47).

2.2. *Eligibility criteria*

Eligible studies included human participants with suspected or confirmed ischemic stroke, cerebral infarction, stroke-related neurological impairment, ischemic stroke rehabilitation needs, post-EVT/MT status, or ischemic stroke recovery outcomes. Studies from hyperacute, acute, subacute, and chronic phases were eligible when EEG/QEEG was evaluated in relation to severity, lesion burden, functional outcome, mortality, deterioration, post-EVT/MT monitoring, motor recovery, language recovery, cognitive recovery, rehabilitation response, or advanced neurophysiological recovery mechanisms. Eligible index measures included conventional EEG, QEEG spectral power, relative band powers, DAR, DTABR, theta-alpha ratio when interpreted as a slow-to-fast abnormality, BSI, pdBSI, revised BSI, laterality coefficients, visual EEG abnormalities, processed EEG indices, single-channel EEG features, phase synchrony, coherence, weighted phase lag index, weighted node degree, functional connectivity, fractal dimension, nonlinear complexity measures, and electroencephalography–cerebral blood flow velocity (EEG-CBFV) neurovascular-coupling metrics. These non-conventional QEEG modalities were included because the review focused on EEG-derived functional markers rather than spectral QEEG alone. Therefore, selected structured routine EEG, visual EEG grading, processed EEG indices such as BIS, and EEG-CBFV neurovascular-coupling measures were eligible when they were linked to predefined severity, lesion-burden, prognostic, monitoring, cognitive/language, rehabilitation, or recovery outcomes. Unstructured routine EEG reports without extractable markers, monitoring, prognostic, or recovery-related information were not included.

Study records with mixed ischemic and hemorrhagic stroke populations were included only when ischemic stroke participants predominated or when ischemic-specific data were extractable; such records were interpreted cautiously and were not used to support ischemic-specific pooled inference when ischemic-only estimates were unavailable.

2.3. *Information sources and search strategy*

A prognosis/recovery-focused search was performed on 30 March 2026 in PubMed, Embase, Scopus, and Web of Science. The search combined ischemic stroke or cerebral infarction terms, QEEG biomarker terms, and prognosis, outcome, and recovery terms. The strategy was biomarker-oriented. Structured routine EEG, visual EEG, processed EEG, and Bispectral Index (BIS) studies were retained when they were retrieved by the database search or identified through reference checking/manual searching and met the predefined EEG-derived biomarker and outcome criteria. This review should not be interpreted as an exhaustive review of all unstructured routine EEG or anesthesia-depth monitoring literature. The full database-specific search strategies are provided in Supplementary Appendix 1.

2.4. *Study selection process*

Records were screened for relevance to ischemic stroke or cerebral infarction and to EEG/QEEG severity, prognosis, monitoring, cognitive/language, rehabilitation, or recovery outcomes. A study record was retained for this article if its primary extractable contribution involved severity association, lesion burden, longitudinal functional outcome, mortality, deterioration, post-EVT/MT monitoring, motor recovery, language recovery, cognitive recovery, rehabilitation response, routine EEG prognostic value, processed EEG prognostic evidence, or advanced mechanistic neurophysiology. Study records whose only extractable contribution was acute diagnostic triage, large-vessel occlusion (LVO)/medium-vessel occlusion (MeVO) detection, or stroke/transient ischemic attack (TIA) classification without severity, monitoring, cognitive, lesion-burden, or prognostic content were not analyzed as primary evidence in this article.

Title/abstract screening, full-text assessment, data extraction, and risk-of-bias assessment were performed independently by two reviewers using the predefined eligibility criteria and structured forms. Disagreements were resolved by discussion, with adjudication by a third reviewer when required. Reasons for full-text exclusion were recorded in the screening log and are summarized in the PRISMA flow diagram (Figure 1).

2.5. *Data extraction*

Data extraction used a structured prognostic and recovery extraction form. Extracted variables included first author, year, country, design, stroke population, sample size, control group, stroke subtype, lesion location, stroke phase, time from onset or treatment to EEG, EEG recording state, montage, device, number of electrodes, preprocessing approach where available, feature family, exact EEG/QEEG marker, outcome measure, outcome time point, effect metric, adjustment status, precision estimates, threshold selection, and clinical interpretation category. When multiple feature-level estimates were reported from the same cohort, estimates were summarized at the study-feature-family-outcome-domain level to avoid overrepresenting highly correlated results. When studies reported both unadjusted and adjusted estimates, adjusted estimates were prioritized for interpretation. When only correlations, area under the curves (AUCs), thresholds, or model-based results were available, these were retained descriptively.

2.6. *Evidence classification and handling of shared evidence*

Clinical interpretation categories were defined before synthesis as cross-sectional neurological severity association, lesion-burden or infarct-volume association, post-EVT/MT monitoring, longitudinal functional prognosis, mortality or clinical deterioration, motor recovery or rehabilitation response, language or cognitive outcome, routine or visual EEG prognostic evidence, processed EEG prognostic evidence, and advanced connectivity, nonlinear, or neurovascular-coupling evidence. Studies that also reported diagnostic performance were not excluded if they contained relevant prognostic or monitoring information. In such studies, diagnostic accuracy metrics were described only when needed to contextualize the study design, while the article-level synthesis emphasized severity, monitoring, cognitive, imaging, or prognostic outcomes.

Before synthesis, each outcome was classified as cross-sectional, short-term longitudinal, long-term longitudinal, rehabilitation/recovery, monitoring, or mechanistic. Concurrent NIHSS, baseline functional status, and infarct volume measured near the EEG recording were classified as severity or lesion-burden associations. Follow-up NIHSS, mRS, Barthel Index, mortality, clinical deterioration, early neurological deterioration (END) after EVT, cerebral edema, Fugl-Meyer Assessment, recovery gain, language improvement, cognitive impairment, or functional status measured after the EEG recording were classified as monitoring, prognostic, or recovery outcomes. This distinction was used to avoid conflating disease severity with prediction of future outcome.

2.7. *Narrative synthesis strategy*

The narrative synthesis was organized by clinical outcome domain and EEG/QEEG feature family. First, acute and subacute severity or lesion-burden associations were summarized, with emphasis on slow-wave activity, DAR, DTABR, alpha/beta attenuation, BSI/pdBSI, and infarct-volume relationships. Second, longitudinal functional prognosis was summarized across mRS, NIHSS change, Barthel Index, mortality, and clinical deterioration. Third, post-treatment studies were summarized as outcome-monitoring evidence when EEG/QEEG was acquired during or after EVT/MT and evaluated against complications, infarct evolution, physiological recovery, or later clinical outcome. Fourth, rehabilitation and recovery studies were synthesized by motor recovery, language recovery, BCI/transcranial direct current stimulation (tDCS) response, phase synchrony, connectivity, and nonlinear complexity. Fifth, routine or visual EEG and processed EEG studies were summarized separately. Sixth, advanced QEEG studies were synthesized as mechanistic evidence involving connectivity, coherence, phase synchrony, fractal dimension, and neurovascular coupling.

2.8. *Quantitative display and criteria for pooling*

Evidence unsuitable for pooling was not excluded from the review. Instead, it was retained for structured narrative synthesis, study-level tabulation, and exploratory quantitative display when appropriate. The synthesis focused on the clinical question, interpretation category, EEG/QEEG feature family, outcome domain, effect direction, study independence, and consistency of findings, rather than vote counting based only on statistical significance. Potential cohort overlap or shared parent-study structures were considered when interpreting the independence of estimates.

Broad prognostic pooling was not planned as the primary synthesis method. Quantitative pooling was considered only within strict feature-outcome-setting-time-effect clusters defined by the EEG/QEEG feature construct, clinical or imaging outcome construct, stroke phase or clinical setting, outcome time window, and effect metric. At least three comparable estimates from at least three independent study records were required before a pooled estimate was considered. Clusters with fewer estimates, fewer independent study records, or clinically non-equivalent constructs were retained for narrative synthesis or descriptive visual display without a pooled summary.

For correlation estimates, effect directions were harmonized only to aid interpretation within clinically comparable evidence clusters. Higher slow-wave burden, DAR, DTABR, theta/alpha ratio (TAR) when interpreted as a slow-to-fast abnormality, BSI, pdBSI, hemispheric asymmetry, and visual abnormality scores were treated as adverse EEG/QEEG

directions. Lower alpha, beta, or other faster-frequency activity was treated as adverse when reduced faster activity represented loss of normal cortical background organization. When higher clinical scores represented better function or recovery, such as Barthel Index or Fugl-Meyer Assessment, correlation directions were reversed when required. Direction harmonization was not used to justify broad pooling across different physiological or clinical constructs.

Restricted exploratory quantitative displays are provided as supplementary analysis. These analyses include a restricted exploratory correlation forest display, leave-one-study and leave-one-collapsed-unit influence displays, descriptive feature-family and outcome-domain displays, a descriptive band-power feature-level display, and a funnel display. These findings were interpreted as supportive visual displays only and were not used as the primary basis for certainty grading or clinical conclusions. The funnel display was retained for transparency and should not be interpreted as a formal publication-bias assessment.

2.9. *Risk of bias and certainty assessment*

Risk of bias was assessed using Quality in Prognostic Studies (QUIPS)-oriented criteria for prognostic and monitoring evidence. Domains included study participation, attrition, prognostic factor measurement, outcome measurement, confounding, and statistical analysis/reporting. Particular attention was given to small samples, heterogeneous EEG timing, incomplete confounder adjustment, selective reporting of multiple EEG features, internally derived thresholds, lack of external validation, and incomplete reporting of precision. Prediction-model or threshold-based studies were summarized descriptively; formal Prediction model Risk of Bias ASsessment Tool (PROBAST) or PROBAST + artificial intelligence (AI) assessment was not performed because the review focused on biomarker evidence rather than complete prediction-model validation (48-50). Certainty was judged at the evidence-domain level using Grading of Recommendations Assessment, Development and Evaluation (GRADE)-informed criteria. Ratings were interpreted as domain-level certainty judgments rather than fully outcome-specific GRADE ratings for each feature-outcome pair.

3. Results

3.1. *Study selection and characteristics of included evidence*

The database search identified 2,564 records: PubMed 407, Embase 747, Scopus 759, and Web of Science 651. After removal of 851 duplicates, 1,713 unique records were screened. Of these, 1,654 records were excluded at title/abstract screening, and 59 full-text reports were assessed for eligibility. Twenty-six full-text reports were excluded with reasons, and 33 study records met the eligibility criteria for this prognostic, monitoring, severity association, recovery, cognitive/language, routine EEG, processed EEG, and advanced QEEG synthesis (Figure 1) (9-38, 41, 51-53). The included records were retained because each provided extractable severity association, monitoring, prognostic, recovery, cognitive/language, routine EEG, processed EEG, or advanced QEEG evidence.

The 33 included study records represented 1,696 stroke or suspected-stroke participants, of whom 1,598 contributed EEG, QEEG, BIS, EEG-transcranial Doppler (TCD), or related neurophysiological data. In addition, 317 healthy or clinical control participants were included across comparative studies. Sample sizes ranged from 10 to 151 participants in most non-EVT studies, with larger post-EVT or processed EEG cohorts also represented. The clinical populations included hyperacute anterior-circulation ischemic stroke, acute cortical or middle cerebral artery (MCA)-territory stroke, broader anterior-circulation ischemic stroke cohorts, lacunar and posterior circulation syndromes, post-EVT/MT cohorts, subacute and chronic rehabilitation cohorts, routine EEG cohorts, processed EEG mortality studies, post-stroke cognitive impairment cohorts, and advanced QEEG or EEG-TCD studies.

EEG timing ranged from within hours of symptom onset to subacute recordings several days after stroke, post-EVT/MT recordings within the first 24-72 hours or serially during the first week, and chronic rehabilitation-phase assessments months after stroke. EEG modalities included conventional multichannel EEG, QEEG spectral analysis, visual EEG classification, processed EEG indices such as BIS, single-channel EEG, high-density EEG, EEG source imaging, functional connectivity, phase synchrony, fractal dimension, and EEG-CBFV coupling. Because studies differed in stroke phase, EEG timing, feature definition, outcome domain, and statistical metric, evidence was synthesized by clinical domain and feature family rather than as a single pooled prognostic dataset.

3.2. *Acute and subacute severity or lesion-burden associations*

Several acute and subacute studies evaluated associations between EEG/QEEG abnormalities and neurological severity or lesion burden (9-14, 17-19, 25). Across these studies, the most consistent pattern was that greater slow-wave activity, higher slow-to-fast ratios, and greater interhemispheric asymmetry were associated with more severe neurological impairment or greater imaging burden. Ajcevic et al. reported a strong positive correlation between DAR and admission NIHSS and a strong inverse correlation between relative alpha power and admission NIHSS in hyperacute anterior-circulation ischemic stroke (14). Finnigan et al. showed that acute delta change index correlated with 30-day NIHSS and diffusion-weighted imaging (DWI) lesion volume, and a later subacute cohort showed strong correlation between DAR and 30-day NIHSS (9, 10).

Sheorajpanday et al. contributed two relevant reports. In lacunar and posterior circulation syndromes, pdBSI and DTABR were evaluated for lesion confirmation and were also associated with imaging burden or early functional status (11). In a broader ischemic-stroke cohort, DTABR correlated with 6-month mRS and pdBSI correlated with infarct volume; adjusted models suggested associations with later disability or dependency (12). The study by Wang et al. (51) was included because it provided cross-sectional cognitive and infarct-burden information after cerebral infarction. Although it was originally framed as a post-stroke cognitive impairment classification study, the study also reported infarct number and largest infarct size and therefore, supports the cognitive/lesion-burden domain rather than acute vascular triage.

Assenza et al. found that low-frequency powers were associated with acute NIHSS and that several contralesional frequency-band powers were associated with effective recovery (17). Cuspineda et al. reported QEEG absolute energy Z-score variables with predictive value for short- and long-term mRS outcomes after acute MCA stroke (18). Xin et al. reported associations between BSI or revised BSI and short-term mortality,

Barthel Index, or mRS outcomes (19, 25). Overall, severity and lesion-burden evidence suggested a directionally coherent electrophysiological pattern: higher DAR/DTABR, higher BSI/pdBSI or r-BSI, increased delta or low-frequency activity, and reduced alpha/beta activity were generally associated with worse neurological severity, larger lesion burden, or poorer early clinical status.

3.3. *Longitudinal functional prognosis*

Several studies evaluated longitudinal functional outcomes, including mRS, Barthel Index, NIHSS change, mortality, and disability (9, 10, 12, 13, 15, 16, 18, 19, 21-26, 28, 30). The most consistent prognostic evidence came from ratio and asymmetry markers, particularly DAR, DTABR, BSI/pdBSI, and related measures of slow-wave burden or hemispheric imbalance. Bentes et al. provided one of the larger acute ischemic stroke cohorts and reported that higher relative delta power and DTABR and lower alpha/beta activity were associated with unfavorable functional outcome; adjusted DTABR models showed odds ratios around 1.67-1.70 and cross-validated AUCs around 0.83-0.86 (13).

Cillessen et al. showed that visual EEG poor-prognosis or major-ischemia classification predicted poor 1-year functional outcome (21). Akgol Gur et al. reported high within-study discrimination for in-hospital mortality using the processed EEG-derived BIS index, although this threshold-based evidence is distinct from conventional QEEG and requires external validation (23). Rogers et al. found that relative theta power from acute single-channel EEG correlated with 30-day modified Barthel Index and mRS (24). Wolf et al. reported that abnormal EEG and generalized slowing, but not focal slowing, were associated with clinical deterioration in acute ischemic stroke without seizures (22). Across longitudinal prognostic studies, greater EEG/QEEG abnormality tended to indicate worse functional outcome, higher disability, clinical deterioration, or mortality, but outcomes, thresholds, and model structures were heterogeneous.

3.4. *Post-EVT/MT monitoring and post-treatment prognosis*

Eight studies contributed post-EVT/MT monitoring or post-treatment prognostic evidence (15, 16, 26-31). These study records were included because post-treatment QEEG, routine EEG, processed EEG, and EEG-TCD measures provided prognostic or monitoring information after reperfusion therapy. Wang et al. reported that post-EVT DAR showed high within-study discrimination for poor 90-day functional outcome in a preliminary cohort; however, the cutoff was internally derived and requires external validation (15). Zhang et al. found that delta power at 24 hours after MT had the strongest receiver operating characteristic (ROC) performance for poor 3-month outcome, while later DAR and DTABR values correlated with NIHSS or mRS outcomes (16). Shen et al. reported that global and frontal DAR were independently associated with cerebral edema, END, and 90-day functional dependence after MT (26).

Routine and visual EEG evidence also supported post-treatment monitoring relevance. Prandin et al. identified early post-EVT status epilepticus, all nonconvulsive status epilepticus (NCSE), and found that it was associated with reduced short-term clinical improvement (27). Yang et al. reported that serial visual EEG grading after EVT was associated with 3-month prognosis, with increasing AUCs across 24 hours, day 3, and day 7 (28). Yan et al. contributed to this review because, in addition to END detection, the study reported associations between QEEG features and NIHSS or infarct volume after EVT; affected-hemisphere relative delta power correlated positively with infarct volume, whereas relative beta power correlated negatively with infarct volume (29). Batra et al. evaluated BIS and near-infrared spectroscopy (NIRS) during EVT as physiological monitoring measures, and Zhang et al. provided post-EVT neurovascular-coupling evidence using simultaneous EEG-TCD monitoring (30, 31). Taken together, post-EVT/MT evidence supported EEG/QEEG as a candidate monitoring approach for reperfusion-related cortical dysfunction, edema, END, seizure-related complications, infarct growth, and neurovascular-coupling physiology.

3.5. *Rehabilitation and motor recovery*

Eight studies evaluated rehabilitation or recovery-related EEG/QEEG evidence (20, 32-38). These studies differed substantially in stroke chronicity, rehabilitation setting, task state, EEG acquisition, and outcome scale. Agius Anastasi et al. found strong correlations between BSI and motor scores in a small longitudinal cohort (32). Saes et al. found that early theta-band BSI contributed to prediction of 26-week upper-limb motor impairment (36). Sebastian-Romagosa et al. reported associations between BSI or alpha/beta laterality coefficients and upper-extremity motor function (37). Mane et al. reported intervention-specific associations between BSI or pairwise ratio index (PRI) and Fugl-Meyer Assessment (FMA) gains after BCI or tDCS-BCI rehabilitation (34).

Network-level rehabilitation studies suggested that connectivity measures may carry recovery-related information. Kawano et al. reported that interhemispheric alpha phase synchrony was associated with baseline Fugl-Meyer Assessment of the Upper Extremity (FM-UE) and that contralesional theta phase synchrony was associated with FM-UE gain (35). Nicolo et al. reported that ipsilesional beta-band and contralesional theta-band weighted node degree predicted future motor and language improvement and showed partial validation in an independent cohort (33). Zappasodi et al. found that fractal dimension and hemispheric complexity measures were associated with neurological impairment and recovery (38). Capon provided early rehabilitation-phase quantitative EEG and brain-mapping evidence and did not find significant correlations between EEG changes and clinical improvement (20). Overall, rehabilitation and recovery evidence suggested that asymmetry, laterality, phase synchrony, weighted node degree, and nonlinear complexity may reflect motor or language recovery potential, but the evidence remains heterogeneous.

3.6. *Cognitive/language outcomes*

Cognitive and language outcomes were less frequently studied. Wang et al. evaluated beta-power abnormality in post-stroke cognitive impairment after cerebral infarction and contributes to the cognitive/lesion-burden domain (51). Nicolo et al. contributed language-recovery evidence using connectivity-based weighted node degree (33). Capon and Sebastian-Romagosa et al. contributed additional cognitive, aphasia, or functional-state evidence, but the outcomes, time windows, and EEG features were too heterogeneous for quantitative synthesis (20, 37).

Routine or visual EEG evidence remained clinically relevant because conventional EEG is more widely available than ad-

vanced QEEG pipelines. Cillessen et al. and Bentes et al. supported the prognostic value of visual EEG abnormalities or visual asymmetry models (13, 21). Wolf et al. linked abnormal EEG and generalized slowing with clinical deterioration in acute ischemic stroke without seizures (22). Prandin et al. provided post-EVT NCSE evidence (27). Processed EEG evidence was represented by BIS studies in the emergency-department mortality and intra-procedural EVT monitoring settings (23, 31).

Advanced QEEG evidence included connectivity, coherence, weighted phase lag index (WPLI), phase synchrony, weighted node degree, fractal dimension, small-world metrics, and EEG-CBFV neurovascular coupling (29, 30, 33, 35, 38, 41, 53). Hofmeijer et al. reported acute hemispheric ischemic stroke evidence involving DAR, magnitude-squared coherence, and WPLI (53). Liu et al. evaluated cross-frequency coupling between cerebral blood-flow velocity and EEG in acute ischemic stroke with LVO (41). Yan et al. contributed coherence and small-world metrics after EVT, while Zhang et al. contributed post-EVT neurovascular-coupling evidence (29, 30). These studies broadened the biological interpretation of EEG/QEEG beyond spectral slowing and asymmetry, but most feature families were represented by one or two studies only.

3.7. *Quantitative synthesis decision*

No global prognostic pooled estimate was calculated. This decision reflected the predefined synthesis framework and the heterogeneity of the evidence rather than absence of a prognostic signal. The included studies evaluated different physiological constructs, including DAR, DTABR, BSI, pdBSI, relative band powers, visual EEG grades, BIS, single-channel theta power, phase synchrony, weighted node degree, fractal dimension, small-world metrics, and EEG-CBFV coupling. Outcomes also differed substantially and included concurrent NIHSS, follow-up NIHSS, infarct volume, mRS, Barthel Index, mortality, clinical deterioration, END, cerebral edema, Fugl-Meyer Assessment, motor recovery, language recovery, cognitive impairment, and rehabilitation response. Most feature-outcome-setting-time clusters did not contain at least three comparable estimates from at least three independent studies; therefore, broad pooling was considered inappropriate.

Restricted exploratory quantitative displays are provided in Supplementary Figures S1-S7. These figures include a restricted exploratory correlation forest display (Figure S1), leave-one-study-out and leave-one-collapsed-unit influence displays (Figures S2-S3), descriptive feature-family and outcome-domain displays (Figures S4-S5), a descriptive band-power feature-level display (Figure S6), and a funnel display (Figure S7). These figures were interpreted as supportive visual displays only and were not used as the primary basis for certainty grading or clinical conclusions; the funnel display should not be interpreted as a formal publication-bias assessment.

The supplementary figures were prepared to provide a transparent visual summary of the extracted quantitative evidence across feature families and outcome domains. These displays were exploratory and were not interpreted as formal meta-analyses. They were used to show the direction, magnitude, and heterogeneity of reported EEG/QEEG associations, rather than to generate pooled prognostic estimates.

Across the supplementary displays, the most coherent direction of association was observed for slow-to-fast spectral ratios and hemispheric asymmetry indices. Higher DAR, DTABR, BSI, pdBSI, or revised BSI generally aligned with worse neurological severity, greater infarct or lesion burden, poorer functional outcome, or less favorable recovery. Conversely, preserved alpha and beta activity tended to align with better clinical status or outcome, whereas reduced faster-frequency activity was more often associated with greater impairment. However, effect sizes varied substantially across studies because of differences in stroke phase, EEG timing, montage, preprocessing, outcome definition, and statistical reporting.

The post-EVT/MT supplementary displays highlighted a clinically coherent but methodologically heterogeneous monitoring domain. Post-treatment EEG/QEEG abnormalities, including increased DAR or frontal DAR, reduced relative alpha activity, abnormal visual EEG grades, NCSE/status epilepticus detection, and impaired neurovascular-coupling metrics, were associated with outcomes such as early neurological deterioration, cerebral edema, infarct growth, poor 3-month functional outcome, or reduced short-term neurological improvement. These findings support the concept of EEG/QEEG as a candidate post-reperfusion monitoring framework, but the evidence remains insufficient to define a single monitoring cutoff or standardized post-EVT/MT EEG protocol.

The rehabilitation and advanced-neurophysiology supplementary displays showed broader mechanistic heterogeneity. Recovery-related studies reported associations involving BSI, laterality coefficients, phase synchrony index, weighted node degree, fractal dimension, coherence, WPLI, and EEG-CBFV coupling. These markers suggest that post-stroke recovery may depend not only on lesion burden or acute severity but also on interhemispheric balance, residual network integrity, nonlinear cortical dynamics, and neurovascular coupling. Nevertheless, these domains were represented by small studies, varied rehabilitation contexts, and different mathematical feature definitions; therefore, they should be interpreted as hypothesis-generating rather than clinically validated prognostic tools.

Because most feature-outcome-setting-time clusters were represented by fewer than three sufficiently comparable independent study records, the supplementary figures were not used for formal pooling. Funnel or precision-oriented displays, where provided, were included only for transparency and should not be interpreted as reliable tests for publication bias, given the small number of studies, dependent feature-level estimates, and substantial clinical heterogeneity.

3.8. *Risk of bias and certainty of evidence*

Risk of bias was frequently concerning. Common limitations included small sample sizes, heterogeneous EEG timing, multiple dependent feature-level estimates, incomplete adjustment for confounding, internally derived thresholds, incomplete reporting of precision, and limited external validation. Rehabilitation and advanced QEEG studies had additional concerns related to task-state heterogeneity, intervention context, feature multiplicity, and small numbers of independent cohorts. Domain-level certainty was low for general prognostic ratio/asymmetry evidence and post-EVT/MT monitoring evidence. Certainty was very low for band-power prognostic features, rehabilitation and motor recovery, cognitive/language outcomes, routine or qualitative EEG and processed EEG indices, and advanced

QEEG/connectivity/nonlinear/neurovascular-coupling evidence.

4. Discussion

This systematic review synthesized QEEG, routine EEG, processed EEG, post-EVT/MT monitoring, and advanced neurophysiological evidence for severity association, longitudinal prognosis, and recovery after ischemic stroke. In contrast to diagnostic-triage applications, this article focused on what EEG/QEEG reveals about stroke severity, lesion burden, post-treatment deterioration, later functional outcome, rehabilitation potential, cognitive/language outcomes, and network-level recovery biology. The search strategy was specifically aligned with prognosis, functional outcome, severity, infarct volume, mortality, rehabilitation, motor recovery, and language recovery.

The most consistent finding was that greater EEG/QEEG abnormality was associated with worse neurological severity, imaging burden, functional outcome, post-treatment course, or recovery. Across acute, subacute, and post-treatment studies, higher DAR or DTABR, greater BSI/pdBSI or revised BSI, increased delta or low-frequency activity, and reduced alpha or beta activity were generally associated with worse NIHSS, mRS, infarct volume, disability, mortality, END, cerebral edema, or recovery outcomes (9-16, 19, 25, 26, 28, 29). This pattern is coherent with established ischemic neurophysiology. Cerebral ischemia reduces perfusion and metabolic substrate delivery, which can impair synaptic activity and organized cortical rhythms before irreversible infarction is complete (4-8) (Figure 2).

From a clinical prognostic standpoint, these findings matter because early outcome after ischemic stroke is often uncertain. NIHSS, age, prestroke disability, infarct volume, occlusion site, collateral status, reperfusion status, and comorbidities are established clinical and imaging predictors, but they do not directly measure cortical function. QEEG could theoretically add bedside functional information when estimating severity, recovery potential, or risk of deterioration. However, the present evidence does not define a ready-to-use prognostic tool. Most included studies were small, thresholds varied, EEG timing differed substantially, and adjustment for established prognostic factors was inconsistent. Therefore, QEEG should be considered a candidate risk-stratification biomarker rather than a validated clinical prediction model.

The strongest conventional QEEG signal involved slow-to-fast ratios and hemispheric asymmetry. DAR and DTABR are physiologically attractive because they combine two complementary consequences of ischemic dysfunction: increased slow activity and loss of faster alpha/beta organization. Finnigan et al. showed that acute and subacute QEEG indices correlated with 30-day NIHSS (9, 10). Sheorajpanday et al. reported associations of DTABR and pdBSI with infarct volume and later functional status (11, 12). Bentes et al. found that higher delta and DTABR and lower alpha/beta activity were associated with unfavorable functional outcome (13). Wang et al. and Zhang et al. extended this pattern to post-EVT/MT outcome, showing associations between DAR/DTABR, delta power, alpha activity, and later clinical outcomes (15, 16).

Post-EVT/MT evidence should be interpreted through an outcome and prognosis lens. Recanalization does not guarantee recovery of normal cortical function, and post-treatment outcomes may be influenced by edema, early neurological deterioration, seizures, infarct growth, and neurovascular-coupling physiology. Shen et al., Yan et al., Yang et al., Prandin et al., and Zhang et al. support the concept that EEG/QEEG, visual EEG, routine EEG, and EEG-TCD coupling can provide information about the post-treatment physiological state (26-30). In this review, these studies were not treated as a single diagnostic accuracy domain. Instead, they were considered post-treatment prognostic or outcome-monitoring evidence because their endpoints included complications, infarct evolution, neurological status, or later functional outcome.

BSI, pdBSI, revised BSI, and laterality coefficients also have a strong physiological rationale. Focal ischemic stroke can produce asymmetric slowing, reduced functional output from the affected hemisphere, and altered interhemispheric balance. In the included studies, asymmetry markers were associated with lesion confirmation, infarct volume, functional status, motor impairment, and recovery. This fits current concepts of stroke as both a focal lesion and a network disorder. Structural injury can alter activity locally and remotely through diaschisis, interhemispheric imbalance, and reorganization of surviving networks (43, 44).

The study by Wang et al. (51) contributes to the cognitive domain. Its beta-power classification results should not be interpreted as validated longitudinal prognosis, because the study was cross-sectional and data-driven. However, it is relevant to the present article because post-stroke cognitive impairment and infarct-burden context fall within the eligibility criteria. This study should therefore be discussed as cognitive/lesion-burden evidence and not as acute diagnostic triage or LVO detection evidence.

The rehabilitation and recovery evidence broaden the interpretation of EEG/QEEG beyond acute lesion severity. Several studies suggested that recovery depends not only on how abnormal the ischemic hemisphere is but also on the integrity and reorganization of distributed networks. Agius Anastasi et al., Saes et al., Sebastian-Romagosa et al., and Mane et al. linked BSI, laterality, or intervention-specific QEEG biomarkers with upper-limb impairment or recovery (32, 34, 36, 37). Nicolo et al., Kawano et al., and Zappasodi et al. linked weighted node degree, phase synchrony, and fractal dimension with motor or language recovery (33, 35, 38). These findings support a mechanistic view of recovery as network reorganization, but the evidence remains small and methodologically diverse.

Routine and processed EEG evidence is clinically relevant because these methods are more accessible than advanced QEEG pipelines. Visual EEG abnormalities, generalized slowing, and qualitative EEG grading were associated with poor outcome or deterioration in selected studies (13, 21, 22, 27, 28). BIS also showed potential as a processed EEG index for mortality prediction or intra-procedural physiological monitoring in ischemic stroke (23, 31). However, qualitative EEG is operator-dependent, grading systems vary, and processed EEG indices were not developed specifically for stroke prognosis. These findings support further study rather than immediate implementation.

A key methodological conclusion is that severity association, longitudinal prognosis, post-treatment outcome monitoring, and recovery should not be collapsed into one pooled effect. Concurrent NIHSS or infarct-volume correlations answer a different question from 3-month mRS, 6-month motor recovery,

mortality, END, cerebral edema, cognitive impairment, or language improvement. Similarly, DAR, DTABR, BSI, alpha power, beta power, visual EEG grades, BIS, phase synchrony, weighted node degree, fractal dimension, and neurovascular coupling represent different physiological constructs. Pooling them would risk creating a numerically precise but clinically uninterpretable estimate. This is consistent with guidance that synthesis methods should reflect clinical and methodological comparability when meta-analysis is not appropriate (45-47).

4.1. *Limitations*

The present evidence has important limitations. Many studies were small, exploratory, single-center, and internally thresholded. EEG timing, montage, preprocessing, feature definitions, outcomes, and statistical reporting varied substantially. Several studies reported multiple dependent feature-level estimates from the same cohort. Confounding adjustment was inconsistent, and external validation was uncommon. Prediction model reporting was incomplete in several model-based or threshold-based studies, and formal PROBAST or PROBAST+AI assessment was not performed; therefore, prediction-model certainty remains limited. Although the search strategy was designed specifically for ischemic stroke prognosis, recovery, severity, outcome, infarct volume, mortality, rehabilitation, motor recovery, and language recovery, studies using nonstandard terminology for EEG features or outcomes may still have been missed. Risk-of-bias judgments were domain-level and biomarker-focused. Finally, two included records used mixed-stroke populations or validation samples that were not purely ischemic: Rogers et al. (24) included 12 ischemic and 4 hemorrhagic first-stroke participants, and the validation cohort in Nicolo et al. (33) included ischemic and hemorrhagic stroke. These records were interpreted cautiously and were not used as ischemic-specific pooled evidence.

Future research should move from exploratory association to prespecified, externally validated prognostic and recovery models. Acute and stroke-unit studies should test whether EEG/QEEG adds incremental prognostic value beyond NIHSS, imaging, age, prestroke disability, reperfusion status, infarct volume, and collateral metrics. Post-EVT/MT studies should standardize monitoring windows and distinguish early complications from later functional outcomes. Rehabilitation studies should define timing, task state, motor and language outcomes, and clinically meaningful recovery thresholds in advance. Neurophysiology studies should standardize frequency-band definitions, reference montages, artifact handling, connectivity metrics, and source versus scalp-level interpretation.

5. Conclusions

In summary, EEG/QEEG abnormalities provide a biologically plausible framework for assessing severity, prognosis, and recovery after ischemic stroke. The consistent adverse pattern of increased slow activity, reduced faster rhythms, and greater hemispheric asymmetry reflects cortical dysfunction, energy failure, network disruption, and altered interhemispheric balance. Connectivity, complexity, and neurovascular-coupling studies further suggest that recovery depends on distributed network physiology, not lesion anatomy alone. However, current evidence remains heterogeneous and mostly low to very-low certainty. EEG/QEEG should therefore be viewed as a candidate functional biomarker framework rather than an established prognostic or rehabilitation-selection tool.

6. Declarations

6.1. *Acknowledgments*

Many thanks for Dr. Mohammed Ibrahim Mohialdeen Gubari (Department of Clinical Science, College of Medicine, University of Sulaimani, Sulaymaniyah, Kurdistan Region, Iraq) for his valuable help in the data gathering process.

6.2. *Conflict of interests*

The authors declare no competing interests.

6.3. *Funding and supports*

None.

6.4. *Authors' contributions*

Shirwan Hamasalih Omer participated in study design, data gathering, analysis and drafting of the manuscript.

6.5. *Ethical statement*

Not applicable.

6.6. *Informed consent*

Not applicable.

6.7. *Availability of supporting data*

All the data are presented in the paper.

6.8. *Using artificial intelligence chatbots statement*

Artificial intelligence-assisted tools were used only for English-language editing and generating graphical abstract. The author reviewed and verified all content and takes full responsibility for the integrity and accuracy of the manuscript.

7. References

1. GBD 2021 Stroke Risk Factor Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990-2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurol. 2024;23(10):973–1003.

2. Goyal M, Menon BK, van Zwam WH, Dippel DWJ, Mitchell PJ, Demchuk AM. Endovascular thrombectomy after large-vessel ischaemic stroke: a meta-analysis of individual patient data from five randomised trials. Lancet. 2016;387(10029):1723–31.

3. Saver JL, Goyal M, van der Lugt A, Menon BK, Majoie C, Dippel DWJ. Time to treatment with endovascular thrombectomy and outcomes from ischemic stroke: a meta-analysis. JAMA. 2016;316(12):1279–88.

4. Foreman B, Claassen J. Quantitative EEG for the detection of brain ischemia. Crit Care. 2012;16:216.

5. Finnigan S, van Putten M. EEG in ischaemic stroke: quantitative EEG can uniquely inform (sub-)acute prognoses and clinical management. Clin Neurophysiol. 2013;124(1):10–9.

6. van Putten M, Hofmeijer J. EEG monitoring in cerebral ischemia: basic concepts and clinical applications. J Clin Neurophysiol. 2016;33(3):203–10.

7. Astrup J, Siesjö BK, Symon L. Thresholds in cerebral ischemia - the ischemic penumbra. Stroke. 1981;12(6):723–5.

8. Hossmann KA. Viability thresholds and the penumbra of focal ischemia. Ann Neurol. 1994;36(4):557–65.

9. Finnigan SP, Rose SE, Walsh M, Griffin M, Janke AL, McMahon KL. Correlation of quantitative EEG in acute ischemic stroke

with 30-day NIHSS score: comparison with diffusion and perfusion MRI. Stroke. 2004;35(4):899–903.

10. Finnigan SP, Walsh M, Rose SE, Chalk JB. Quantitative EEG indices of sub-acute ischaemic stroke correlate with clinical outcomes. Clin Neurophysiol. 2007;118(11):2525–32.

11. Sheorajpanday RVA, Nagels G, Weeren A, De Deyn PP. Quantitative EEG in ischemic stroke: correlation with infarct volume and functional status in posterior circulation and lacunar syndromes. Clin Neurophysiol. 2011;122(5):884–90.

12. Sheorajpanday RVA, Nagels G, Weeren A, van Putten M, De Deyn PP. Quantitative EEG in ischemic stroke: correlation with functional status after 6 months. Clin Neurophysiol. 2011;122(5):874–83.

13. Bentes C, Peralta AR, Viana P, Martins H, Morgado C, Casimiro C. Quantitative EEG and functional outcome following acute ischemic stroke. Clin Neurophysiol. 2018;129(8):1680–7.

14. Ajčević M, Furlanis G, Naccarato M, Miladinović A, Buoite Stella A, Caruso P. Hyper-acute EEG alterations predict functional and morphological outcomes in thrombolysis-treated ischemic stroke: a wireless EEG study. Med Biol Eng Comput. 2021;59(1):121–9.

15. Wang Y, Liu D, Liu J, Kong C, Zhang Z, Duan W. Quantitative EEG provides early prediction of poor outcome in acute ischemic stroke after endovascular treatment: a preliminary study. Neurol Res. 2021;43(10):831–7.

16. Zhang N, Chen F, Xie X, Xie Z, Hong D, Li J. Application of quantitative EEG in acute ischemic stroke patients who underwent thrombectomy: a comparison with CT perfusion. Clin Neurophysiol. 2022;141:24–33.

17. Assenza G, Zappasodi F, Pasqualetti P, Vernieri F, Tecchio F. A contralesional EEG power increase mediated by interhemispheric disconnection provides negative prognosis in acute stroke. Restor Neurol Neurosci. 2013;31(2):177–88.

18. Cuspineda E, Machado C, Galan L, Aubert E. QEEG prognostic value in acute stroke. Clin EEG Neurosci. 2007;38(3):155–60.

19. Xin X, Gao Y, Zhang H, Cao K, Shi Y. Correlation of continuous electroencephalogram with clinical assessment scores in acute stroke patients. Neurosci Bull. 2012;28(5):611–7.

20. Capon AP. Quantitative EEG with brain mapping in strokes: Is it useful for prognosis? Brain Topogr. 1996;9(2):77–82.

21. Cillessen J, van Huffelen AC, Kappelle LJ, Algra A, van Gijn J. Electroencephalography improves the prediction of functional outcome in the acute stage of cerebral ischemia. Stroke. 1994;25(10):1968–72.

22. Wolf ME, Ebert AD, Chatzikonstantinou A. The use of routine EEG in acute ischemic stroke patients without seizures: generalized but not focal EEG pathology is associated with clinical deterioration. Int J Neurosci. 2017;127(5):421–6.

23. Akgol Gur ST, Akbas I, Kose MZ, Kocak AO, Eren A, Cakir Z. Bispectral Index in predicting in-hospital mortality in patients with ischemic stroke: A methodological study. Hong Kong J Emerg Med. 2022;29(3):144–50.

24. Rogers J, Middleton S, Wilson PH, Johnstone SJ. Predicting functional outcomes after stroke: an observational study of acute single-channel EEG. Top Stroke Rehabil. 2020;27(3):161–72.

25. Xin X, Chang J, Gao Y, Shi Y. Correlation between the revised brain symmetry index, an EEG feature index, and short-term prognosis in acute ischemic stroke. J Clin Neurophysiol. 2017;34(2):162–7.

26. Shen Y, You H, Yang Y, Tang R, Ji Z, Liu H. Predicting brain edema and outcomes after thrombectomy in stroke: frontal delta/alpha ratio as an optimal quantitative EEG index. Clin Neurophysiol. 2024;164:149–60.

27. Prandin G, Furlanis G, Scali I, Palacino F, Mancinelli L, Vincis E. Status epilepticus after mechanical thrombectomy: the role of early EEG assessment in Stroke Unit, clinical and radiological prognostication. Epilepsy Res. 2024;202:107343.

28. Yang Y, Peng L, Li Y, Wang M, Zhou W, Zhou Z. The use of EEG in predicting the prognosis of patients undergoing endovascular treatment for acute anterior circulation infarction. J Clin Neurosci. 2025;137:111252.

29. Yan Y, An X, Ma Y, Jiang Z, Di Y, Li T. Detection of early neurological deterioration using a quantitative electroencephalography system in patients with large vessel occlusion stroke after endovascular treatment. J Neurointerv Surg. 2025;17(8):883–9.

30. Zhang Z, Hasan S, Sadan O, Rosenthal ES, Pu Y, Wen Z. Contralateral neurovascular coupling in patients with ischemic stroke after endovascular thrombectomy. Neurocrit Care. 2025;42:996–1006.

31. Batra D, Chen M, Meis J, Möhlenbruch MA, Klose C, Ringleb P. Feasibility of noninvasive neuromonitoring using BIS and NIRS during endovascular treatment of acute ischemic stroke. Neurol Res Pract. 2025;7:60.

32. Agius Anastasi A, Falzon O, Camilleri K, Vella M, Muscat R. Brain symmetry index in healthy and stroke patients for assessment and prognosis. Stroke Res Treat. 2017;2017:8276136.

33. Nicolo P, Rizk S, Magnin C, Di Pietro M, Schnider A, Guggisberg AG. Coherent neural oscillations predict future motor and language improvement after stroke. Brain. 2015;138(10):3048–60.

34. Mane R, Chew E, Phua KS, Ang KK, Robinson N, Vinod AP. Prognostic and monitory EEG-biomarkers for BCI upper-limb stroke rehabilitation. IEEE Trans Neural Syst Rehabil Eng. 2019;27(8):1654–64.

35. Kawano T, Hattori N, Uno Y, Hatakenaka M, Yagura H, Fujimoto H. Electroencephalographic phase synchrony index as a biomarker of poststroke motor impairment and recovery. Neurorehabil Neural Repair. 2020;34(8):711–22.

36. Saes M, Meskers CGM, Daffertshofer A, van Wegen EEH, Kwakkel G. Are early measured resting-state EEG parameters predictive for upper limb motor impairment six months poststroke? Clin Neurophysiol. 2021;132(1):56–62.

37. Sebastián-Romagosa M, Udina E, Ortner R, Dinarès-Ferran J, Cho W, Murovec N. EEG biomarkers related with the functional state of stroke patients. Front Neurosci. 2020;14:582.

38. Zappasodi F, Olejarczyk E, Marzetti L, Assenza G, Pizzella V, Tecchio F. Fractal dimension of EEG activity senses neuronal impairment in acute stroke. PLoS One. 2014;9(6):e100199.

39. Buzsáki G, Draguhn A. Neuronal oscillations in cortical networks. Sci. 2004;304(5679):1926–9.

40. Fries P. A mechanism for cognitive dynamics: neuronal communication through neuronal coherence. Trends Cogn Sci. 2005;9(10):474–80.

41. Liu X, Pu Y, Wu D, Zhang Z, Hu X, Liu L. Cross-frequency coupling between cerebral blood flow velocity and EEG in ischemic stroke patients with large vessel occlusion. Front Neurol. 2019;10:194.

42. Iadecola C. The neurovascular unit coming of age: a journey through neurovascular coupling in health and disease. Neuron. 2017;96(1):17–42.

43. Grefkes C, Fink GR. Reorganization of cerebral networks after stroke: new insights from neuroimaging with connectivity approaches. Brain. 2011;134(5):1264–76.

44. Silasi G, Murphy TH. Stroke and the connectome: how connectivity guides therapeutic intervention. Neuron. 2014;83(6):1354–68.

45. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372: n71.

46. McKenzie JE, Brennan SE. Chapter 12: Synthesizing and presenting findings using other methods. In: Higgins JPT TJ, Chandler J, Cumpston M, Li T, Page MJ, Welch VA, editor. Cochrane Handbook for Systematic Reviews of Interventions: Cochrane 2023.

47. Campbell M, McKenzie JE, Sowden A, Katikireddi SV, Brennan SE, Ellis S. Synthesis without meta-analysis (SWiM) in systematic reviews: reporting guideline. BMJ. 2020;368: l6890.

48. Moons KGM, de Groot JAH, Bouwmeester W, Vergouwe Y, Mallett S, Altman DG. Critical appraisal and data extraction for systematic reviews of prediction modelling studies: the CHARMS checklist. PLoS Med. 2014;11(10):e1001744.

49. Collins GS, Moons KGM, Dhiman P, Riley RD, Beam AL, Van Calster B. TRIPOD+AI statement: updated guidance for reporting clinical prediction models that use regression or machine learning methods. BMJ. 2024;385:e078378.

50. Wolff RF, Moons KGM, Riley RD, Whiting PF, Westwood M, Collins GS. PROBAST: a tool to assess the risk of bias and applicability of prediction model studies. Ann Intern Med. 2019;170(1):51–8.

51. Wang Y, Zhang X, Huang J, Zhu M, Guan Q, Liu C. Associations between EEG beta power abnormality and diagnosis in cognitive impairment post cerebral infarcts. J Mol Neurosci. 2013;49(3):632–8.

52. Wilkinson CM, Burrell JI, Kuziek JWP, Thirunavukkarasu S, Buck BH, Mathewson KE. Predicting stroke severity with a 3-min recording from the Muse portable EEG system for rapid diagnosis of stroke. Sci Rep. 2020;10(1):18465.

53. Hofmeijer J, van Kaam R, Vermeer SE, van Putten MJ. Contralesional brain activity in acute ischemic stroke. Clin Neurophysiol. 2018;129:e93–e4.

Table 1: Characteristics of included studies

Ref.	Study; country	Design	Population and sample	Age and sex	Baseline severity	Timing and method	Target or outcome domain
32	Agius Anastasi 2017 Malta	Pilot longitudinal case-control study	First-ever stroke; 4 cortical and 6 subcortical strokes Stroke/suspected: N= 10; controls: N= 10; EEG/QEEG: N= 10	Stroke patients mean: 59.4 years; controls mean: 60.3 years 10/0 (100.0% male)	No NIHSS reported. Session 1 motor function: MI mean: 56.5, median: 62.0, range: 9-100; FMA-UE mean: 40.3, median: 43.0, range: 5-66 (calculated from Table 1).	Monthly sessions; first assessment 5-60 days after stroke across patients 32-channel EEG; BSI during rest and hand grasping	Motor recovery / upper-limb function
14	Ajčević 2021 Italy	Prospective observational wireless EEG study	Hyperacute anterior IS before thrombolysis Stroke/suspected: N= 20; controls: N= 0; EEG/QEEG: N= 20	Mean: 75.8 ± 12.1 years 8/12 (40.0% male)	Admission NIHSS median: 10 (range: 3-23); ASPECTS median: 9.5 (range: 7-10).	Within 4.5 h from symptom onset before thrombolysis; median onset-to-EEG: 196 min (range: 81-267) Wireless 19-electrode QEEG; relative band powers, DAR, DTABR	Neurological severity; functional outcome; final infarct volume
23	Akgol Gur 2022 Turkey	Prospective methodological diagnostic/prognostic accuracy study	Ischemic stroke; hemorrhagic stroke and microvascular ischemic lesions excluded Stroke/suspected: N= 80; controls: N= 0; EEG/QEEG: N= 80	Median: 74.0 years (IQR: 65.0-82.0) 40/40 (50.0% male)	NIHSS not reported. GCS median: 13.5 (IQR: 10.0-15.0); BIS median: 85.5 (IQR: 68.5-91.0).	BIS monitored for 10 min after emergency-department admission; 10th-min value recorded Bispectral Index / processed EEG index	In-hospital mortality
17	Assenza 2013 Italy	Prospective observational case-control prognostic study	Unilateral MCA IS; cortical, subcortical, or cortico-subcortical lesions Stroke/suspected: N= 42; controls: N= 20; EEG/QEEG: N= 42	Patients: 74 ± 9 years; controls: 71 ± 6 years 27/15 (64.3% male)	NIHSS range: 1-17, median: 5.5, mean: 6.5, SD: 4.6. Lesion side: left 25 (60%), right 17 (40%).	4-10 days after stroke onset 19-channel resting EEG; spectral band powers and interhemispheric coherence	Neurological severity and 6-month recovery
31	Batra 2025 Germany	Post-hoc analysis of SIESTA randomized clinical trial monitoring data	AIS due to anterior-circulation LVO undergoing EVT Stroke/suspected: N= 150; controls: N= 0; EEG/QEEG: N= 58	BIS-monitoring subgroup median: 77 years (IQR: 66-84); included monitoring group median: 76.5 years (IQR: 66-84) 31/27 (53.4% male)	BIS-monitoring subgroup baseline NIHSS median: 15 (IQR: 14-19); baseline ASPECTS ≥8 in 45/58 (78%)	Continuous BIS monitoring during EVT from angiography-suite arrival until 5 min after procedure BIS / processed frontal EEG; NIRS ancillary monitoring	Intra-procedural recanalization physiology
13	Bentes 2018 Portugal	Prospective longitudinal cohort	Anterior circulation IS; previously independent; NIHSS ≥4 at admission Stroke/suspected: N= 151; controls: N= 0; EEG/QEEG: N= 151	Mean: 67.4 ± 11.9 years 112/39 (74.2% male)	Admission NIHSS summary not reported in accessible main text; inclusion required NIHSS ≥4. Unfavorable outcome at discharge: 99/151 had mRS ≥3; at 12 months: 77/150 had mRS ≥3.	First EEG within 72 h; second EEG at discharge or day 7 after stroke 64-channel video-EEG; global band powers, DTABR/DAR symmetry, and change indices	Functional outcome at discharge and 12 months
20	Capon 1996 Belgium	Prospective rehabilitation follow-up study	Hemispheric ischemic infarct; 18 cortical and 14 subcortical lesions; 15 left and 17 right hemiplegia Stroke/suspected: N= 32; controls: N= 0; EEG/QEEG: N= 32	Mean: 64.8 ± 12.7 years; range: 37-85 18/14 (56.3% male)	No NIHSS reported. All had hemiplegia; clinical impairment measured using motricity index, aphasia index, and Barthel index at 2/4/8/12 weeks.	Three QEEG recordings during approximately 3 months; first recording mean: 31.3 ± 18.1 days after stroke Quantified EEG and brain mapping; spectral amplitude and delta mapping	Rehabilitation evolution: motor ability, ADL, speech
21	Cillessen 1994 Netherlands	Prospective consecutive cohort prognostic classification study	First-ever supratentorial IS; lacunar syndrome: 17, cortical syndrome: 38. Stroke/suspected: N= 55; controls: N= 0; EEG/QEEG: N= 55	Median: 64 years; range: 38-85 NR	Admission Rankin grade 1-3 in 30 patients and Rankin grade 4-5 in 25 patients. No NIHSS reported.	EEG days 1-10 after onset; median: day 3 Visual EEG classification plus spectral analysis	Poor 1-year functional outcome

Ref.	Study; country	Design	Population and sample	Age and sex	Baseline severity	Timing and method	Target or outcome domain
18	Cuspineda 2007 Cuba	Prospective prognostic QEEG study	Acute MCA territory IS within 72 h; major ischemia >33% of MCA territory excluded Stroke/suspected: N= 28; controls: N= 0; EEG/QEEG: N= 28	Mean: 65 years; range: 48-85 17/11 (60.7% male)	No NIHSS reported. Hachinski scale score mean: 5.8, median: 6.0, range: 3.0-8.0 calculated from Table 1. Premorbid mRankin 0 or 1 by inclusion criteria.	Within first 72 h: 13 recordings <24 h, 9 at 24-48 h, 15 at 48-72 h 19-channel QEEG; absolute energy Z values by frequency band	Short-term and 3-month Rankin outcome prediction
9	Finnigan 2004 Australia	Prospective multimodal observational/prognostic pilot study	Acute ischemic cortical stroke; MCA/PCA/ICA territories; qEEG prognosis vs MRI Stroke/suspected: N= 11; controls: N= 6; EEG/QEEG: N= 11	Stroke patients: mean: 74.5 years, range: 55-87; controls: age range: 58-82 5/6 (45.5% male)	Admission NIHSS from Table 1: median: 18, mean: 16.3, range: 4-33; two patients died before 30-day follow-up	Initial EEG mean: 6.6 h after onset, range: 5-8 h; second acute EEG approximately 6-11 h later; EEG 8 and 15 h after onset 64-channel scalp EEG; acute delta change index (aDCI), average scalp delta power	Neurological severity/prognosis; 30-day NIHSS; MRI DWI/PWI lesion volumes
10	Finnigan 2007 Australia	Prospective observational subAIS prognostic study	Subacute ischemic cortical stroke; MCA/PCA/ICA territories; QEEG prognostic indices Stroke/suspected: N= 13; controls: N= 13; EEG/QEEG: N= 13	Stroke patients: median: 66 years, range: 54-86; mean (from Table 1): 70.5 ± 11.9; controls age range: 58-82 6/7 (46.2% male)	NIHSS at 49 ± 3 h: median: 13, range: 2-23; one patient died within 12 days	Resting EEG acquired 49 ± 3 h after symptom onset 62-channel EEG plus 19-channel subset; DAR, PRI, relative band powers	Neurological severity/prognosis; 30-day NIHSS
53	Hofmeijer 2018 Netherlands	Conference abstract; observational AIS EEG study	Acute hemispheric IS; contralesional brain activity and functional connectivity Stroke/suspected: N= 18; controls: N= 18; EEG/QEEG: N= 18	Age not reported in accessible abstract; controls described as age-matched NR	Baseline NIHSS or other admission severity not reported in accessible abstract	Acute EEG timing not numerically specified in abstract; two-minute epochs from 21-electrode EEG 21-electrode conventional EEG; DAR, magnitude-squared coherence, WPLI	Functional outcome/contralesional connectivity; 3-month mRS category
35	Kawano 2020 Japan	Observational poststroke rehabilitation biomarker study	Poststroke hemiparesis after cortical IS; upper-limb motor impairment/recovery Stroke/suspected: N= 40; controls: N= 22; EEG/QEEG: N= 40	Patients: 69.8 ± 13.8 years; controls: 66.9 ± 6.5 years 24/16 (60.0% male)	NIHSS median: 5 (IQR: 3-10.5); FIM median: 79.5 (IQR: 51.5-101); FM-UE1 median: 44.5 (IQR: 9-60), mean: 36.8 ± 24.2	EEG recording 36.9 ± 11.8 days after stroke; FM-UE1 assessment 32.6 ± 12.3 days; EEG-FM interval 4.3 ± 4.2 days 19-channel resting EEG; Phase Synchrony Index (PSI)	Motor impairment and rehabilitation recovery; FM-UE and FM-UE gain
41	Liu 2019 China	Retrospective multimodal neurovascular monitoring study	Acute IS with LVO; ICA/MCA/basilar artery occlusions Stroke/suspected: N= 16; controls: N= 0; EEG/QEEG: N= 16	Reported mean age: 60.9 ± 7.9 years; patient-level table range: approximately 45-85 12/4 (75.0% male)	Admission NIHSS available for 15/16 patients: median: 15, mean: 19.1, range: 10-40	Continuous ICU monitoring of 8-lead EEG and CBFV during acute admission; exact onset-to-EEG timing not extractable from main text 8-lead EEG with bilateral CBFV monitoring; phase-amplitude cross-frequency coupling	Neurovascular coupling; mortality/unfavorable outcome and stenosis physiology
34	Mane 2019 Singapore	Double-blind randomized controlled rehabilitation trial with QEEG biomarker analysis	Chronic stroke rehabilitation; unilateral upper-limb motor impairment; BCI/tDCS-BCI response prediction Stroke/suspected: N= 19; controls: N= 0; EEG/QEEG: N= 19	Combined cohort calculated from Table I: mean: 54.1 ± 10.6 years, median: 58, range: 29-70; tDCS-BCI 52.1 ± 11.6, BCI 56.3 ± 9.5 14/5 (73.7% male)	NIHSS not reported; baseline upper-limb FMA T0 mean: 34.0 ± 7.9, median: 33, range: 20-51 from Table I	Pre-intervention resting/task EEG before 2-week BCI/tDCS-BCI training; chronic stage ≥9 months after stroke EEG during BCI motor-imagery rehabilitation; QEEG band powers, BSI, PRI, DAR/TAR/DTABR-type markers	Upper-limb rehabilitation response; FMA gains at 2 and 4 weeks

Ref.	Study; country	Design	Population and sample	Age and sex	Baseline severity	Timing and method	Target or outcome domain
33	Nicolo 2015 Switzerland	Prospective observational network-plasticity study with independent validation cohort	First-ever territorial ischemic MCA stroke in main cohort; validation cohort included ischemic and hemorrhagic stroke; motor/language recovery Stroke/suspected: N= 42; controls: N= 26; EEG/QEEG: N= 42. Interpreted cautiously because one validation sample was mixed ischemic/hemorrhagic stroke.	Population 1: mean: 60.7 years, range: 37-81; Population 2: mean: 67 years, range: 32-85; approximate pooled mean: 63.4; controls mean: 62.4, range: 32-88 25/17 (59.5% male)	Population 1: NIHSS mean: 13, range: 3-27; Population 2: NIHSS mean: 13.8, range: 3-22; at least mild motor/language impairment	High-density EEG at 2-3 weeks/3 weeks after stroke and follow-up at 3 months depending on cohort High-density EEG/EEG source imaging; functional connectivity/weighted node degree	Motor and language recovery from subacute to chronic phase
27	Prandin 2024 Italy	Retrospective single-center cohort	Large vessel occlusion/large stroke treated with EVT; status epilepticus monitoring Stroke/suspected: N= 138; controls: N= 0; EEG/QEEG: N= 138	Non-SE median: 77 years (IQR: 68-83); SE median: 79 years (IQR: 74-81); aggregate age not reported 57/81 (41.3% male)	Admission NIHSS by SE group; no-SE median: 13 (IQR: 8-20), SE median: 17 (IQR: 12-18)	Conventional EEG within 72 h from symptom onset/admission; median EEG recording time 1 day (IQR: 0-2) Routine 21-electrode conventional EEG; SE/NCSE assessment	Post-EVT status epilepticus and outcome
24	Rogers 2020 Australia	Observational cohort study	First-ever stroke; 12 ischemic and 4 hemorrhagic; left-sided lesion: 7, right-sided lesion: 9 Stroke/suspected: N= 16; controls: N= 0; EEG/QEEG: N= 16	Mean: 65.75 ± 16.98 years; range: 33-93 9/7 (56.2% male)	NIHSS mean: 6.56 ± 6.45, range: 0-18; 7 minor, 6 moderate, 3 moderately severe	EEG within 20-72 h after stroke; mean: 46.63 ± 20.63 h Single-channel wireless EEG at FP1; relative band powers and ratios	Functional outcome/disability and activities of daily living
36	Saes 2021 Netherlands	Prospective longitudinal observational cohort (4D-EEG)	First-ever ischemic hemispheric stroke; LACI/PACI/TACI 20/14/5; affected hemisphere left/right 18/21 Stroke/suspected: N= 39; controls: N= 0; EEG/QEEG: N= 39	Mean: 67.3 ± 11.4 years 23/16 (59.0% male)	Baseline NIHSS median: 5 (IQR: 4-8); FM-UE median: 21 (IQR: 7-42)	EEG recording 12.3 ± 5.8 days after stroke; clinical baseline 11.6 ± 5.3 days; 26-week follow-up 185.2 ± 20.0 days 62-channel resting-state EEG; DAR, BSI, directional BSI	Upper-limb motor impairment/recovery at 26 weeks
37	Sebastián-Romagosa 2020 Spain	Prospective rehabilitation/BCI cohort with controls	Stroke with upper-limb hemiparesis; cortical: N=5, subcortical: N=17, cortical+subcortical: N=12 among analyzed patients Stroke/suspected: N= 36; controls: N= 32; EEG/QEEG: N= 34	Analyzed stroke patients mean: 65.3 ± 14.4 years; controls mean: 42.3 ± 15.4 years 22/12 (64.7% male)	NIHSS not reported; motor impairment measured by FMA, BBT, FTRS, and other rehabilitation scales	Four assessment sessions and 25 BCI rehabilitation sessions; EEG during resting state and motor imagery Resting-state BSI and motor-imagery laterality coefficients in alpha/beta bands	Rehabilitation and motor recovery
26	Shen 2024 China	Prospective cohort	Anterior circulation AIS after MT Stroke/suspected: N= 105; controls: N= 0; EEG/QEEG: N= 105	Median: 70 years (IQR: 59-78) 66/39 (63.0% male)	Baseline NIHSS median: 14 (IQR: 11-17); NIHSS 24 h after MT median: 10 (IQR: 5-23)	EEG 24 ± 6 h after MT 19-electrode EEG; global and frontal DAR, DTABR, relative delta power	Post-MT cerebral edema, END, hemorrhagic transformation, and 90-day functional outcome
11	Sheorajpanday 2011 Belgium	Prospective diagnostic/prognostic cohort from MISS-EEG project	Lacunar syndrome (N=24) or posterior circulation syndrome (N=36) of presumed ischemic origin Stroke/suspected: N= 60; controls: N= 0; EEG/QEEG: N= 60	POCS median: 71 years (range: 33-86); LACS median: 66.5 years (range: 40-87) NR	Median NIHSS: POCS 3 (IQR: 0-5), LACS 2 (IQR: 0-3)	Majority underwent EEG within 72 h of symptom onset (reported 81% POCS and 92% LACS) Quantitative EEG; pdBSI and DTABR	Diagnostic lesion confirmation and short-term outcome in LACS/POCS

Ref.	Study; country	Design	Population and sample	Age and sex	Baseline severity	Timing and method	Target or outcome domain
12	Sheorajpanday 2011 Belgium	Prospective prognostic cohort from MISS-EEG project	Acute IS with persistent neurological deficit Stroke/suspected: N= 110; controls: N= 0; EEG/QEEG: N= 110	Median: 75 years (range: 24.5-89) 55/55 (50.0% male)	Median NIHSS: 6 (IQR: 2-14)	Median delay of EEG after stroke onset: 3.3 days (range: 0.4-11.4) reported in Results Quantitative EEG; pdBSI and DTABR	Six-month functional outcome
48	Wang 2013 China	Case-control diagnostic/cognitive impairment study	Cerebral infarction with MoCA-defined cognitive impairment vs cognitive normality Stroke/suspected: N= 110; controls: N= 110; EEG/QEEG: N= 110	CI-CI: 71 ± 11.9 years; CI-NC: 72 ± 12.8 years; controls: 71 ± 13.5 years 60/50 (54.5% male)	NIHSS not reported; infarct number and largest infarct size reported	EEG within 1 day of hospitalization 16-channel EEG; relative beta power and K-means classification	Post-stroke cognitive impairment classification and infarct burden
15	Wang 2021 China	Preliminary prospective cohort	Acute IS after EVT Stroke/suspected: N= 31; controls: N= 0; EEG/QEEG: N= 31	Mean: 67.7 ± 9.0 years 21/10 (67.7% male)	Baseline NIHSS median: 16 (IQR: 13-19); discharge NIHSS median: 17 (IQR: 8-20)	24-hour EEG monitoring within 48 h after EVT Continuous EEG; global relative powers, DAR and DTABR	Post-EVT 90-day functional outcome
22	Wolf 2017 Austria	Prospective/observational routine EEG cohort	Acute IS without seizures Stroke/suspected: N= 69; controls: N= 0; EEG/QEEG: N= 69	Mean: 69 ± 18 years; range: 19-93 43/26 (62.3% male)	Admission NIHSS median: 4 (IQR: 3-16), range: 0-25; discharge NIHSS median: 3 (IQR: 1-13), range: 0-26	EEG performed mean: 3.6 ± 2.8 days after stroke Routine 21-electrode visual EEG; abnormal EEG, generalized slowing, focal slowing	Routine EEG prognostic evidence
25	Xin 2017 China	Prospective observational prognostic study	Acute ischemic anterior-circulation infarction; left/right/bilateral lesions 14/8/7 Stroke/suspected: N= 29; controls: N= 0; EEG/QEEG: N= 29	Approximate weighted mean: 62.5 years from lesion-site subgroups; inclusion age 35-80 years 24/5 (82.8% male)	NIHSS within 72 h mean: 11.759 ± 6.957; NIHSS at 21 days mean: 8.069 ± 6.088	EEG monitoring began within 72 h of onset Continuous/scalp EEG with revised BSI	Short-term prognosis: BI and mRS at 21 days
19	Xin 2012 China	Prospective observational prognostic study	Acute IS; 10 posterior circulation, 12 anterior-circulation infarctions Stroke/suspected: N= 22; controls: N= 7; EEG/QEEG: N= 22	Survived group mean: 73.06 ± 4.06 years (N=16); died group mean: 65.5 ± 14.90 years (N=6); approximate weighted mean: 71.0 15/7 (68.2% male)	NIHSS: survived mean: 14.875 ± 4.856; died mean: 21.500 ± 3.331	EEG at admission / acute phase; within 48 h in extraction context Continuous EEG; BSI	Short-term mortality/28-day mRS prognosis
29	Yan 2025 China	Prospective cohort with nested case-control END diagnostic analysis	Anterior circulation LVO stroke treated with EVT Stroke/suspected: N= 47; controls: N= 34; EEG/QEEG: N= 47	Stroke patients median: 66.0 years (IQR: 60.0-72.0); controls median: 62.5 (IQR: 46.5-66.3) 30/17 (63.8% male)	Baseline NIHSS median: 12.0 (IQR: 8.0-16.0); after EVT median: 9.5 (IQR: 6.0-14.0)	Baseline EEG on admission and after EVT/recanalization; END EEG within 20 min of END onset if applicable Quantitative EEG system; relative powers, DAR/TAR/DTABR, coherence, small-world metrics	Post-EVT END detection/monitoring
28	Yang 2025 China	Retrospective EVT prognostic EEG grading study	Acute anterior-circulation LVO treated with EVT; ICA infarction: 15, MCA infarction: 25 Stroke/suspected: N= 40; controls: N= 0; EEG/QEEG: N= 40	Age reported categorically: <70 years: N=28 (70%), ≥70 years: N=12 (30%); no mean/median reported 23/17 (57.5% male)	Preoperative NIHSS categories: <6 N=2 (5%), 6-16 N=28 (70%), >16 N=10 (25%)	EEG at 24 h, day 3, and day 7 after EVT Visual/routine EEG grading: normal, mild, moderate, severe abnormality	Post-EVT qualitative EEG prognosis

Ref.	Study; country	Design	Population and sample	Age and sex	Baseline severity	Timing and method	Target or outcome domain
38	Zappasodi 2014 Italy	Case-control prognostic complexity study	First-ever unilateral IS in MCA territory; left lesion N=22, right lesion N=14 Stroke/suspected: N= 36; controls: N= 19; EEG/QEEG: N= 36	Stroke patients mean: 73.0 ± 8.6 years; controls mean: 71.1 ± 6.3 years 24/12 (66.7% male)	Acute NIHSS range: 1-17; median: 6.0; 6-month NIHSS range: 1-12, median: 3.5	EEG collected 4-10 days after stroke; mean: 5.4 ± 1.2 days 19-channel resting EEG; Higuchi fractal dimension, FD asymmetry, spectral powers	Nonlinear complexity, NIHSS, and recovery
16	Zhang 2022 China	Prospective/observational post-thrombectomy QEEG study	Anterior circulation AIS after MT; good 3-month mRS 0-2: N=13, poor mRS 3-6: N=16 Stroke/suspected: N= 29; controls: N= 0; EEG/QEEG: N= 29	Good outcome group 64.9 ± 7.3 years (N=13); poor outcome group 67.9 ± 12.3 years (N=16); approximate weighted mean: 66.6 21/8 (72.4% male)	NIHSS before MT: good outcome 13.5 ± 5.5, poor outcome 17 ± 3.9; approximate weighted mean: 15.4	EEG before MT (N=20), 12 h after MT (N=29), 24 h after MT (N=27), and 7 d after MT (N=22) 16-lead QEEG; delta power, DAR, DTABR; CTP comparators	Post-MT functional outcome and NIHSS
30	Zhang 2025 China	Single-center retrospective observational analysis of a prospectively collected EVT registry	Large-vessel occlusive ischemic stroke treated with EVT; 52 enrolled; contralateral NVC computed in 48 participants Stroke/suspected: N= 52; controls: N= 0; EEG/QEEG/NVC: N= 48	Mean: 61.5 ± 10.4 years 47/52 (90.4% male)	Median preprocedural NIHSS: 14 (IQR: 10-17)	Synchronous EEG-TCD monitoring within 48 h after EVT; 10-20 EEG with selected bipolar MCA-region channels and bilateral MCA CBFV monitoring EEG-CBFV contralateral neurovascular coupling using phase-amplitude cross-frequency coupling	Post-EVT neurovascular coupling; 7-day NIHSS change, infarct volume progression, and 90-day mRS outcome

4D-EEG: four-dimensional electroencephalography; aDCI: acute delta change index; ADL: activities of daily living; AIS: acute ischemic stroke; ASPECTS: Alberta Stroke Program Early CT Score; BBT: Box and Block Test; BCI: brain–computer interface; BI: Barthel Index; BIS: Bispectral Index; BSI: Brain Symmetry Index; CBFV: cerebral blood flow velocity; CI-CI: cerebral infarction with cognitive impairment; CI-NC: cerebral infarction with normal cognition; CTP: computed tomography perfusion; DAR: delta/alpha ratio; DTABR: delta-theta/alpha-beta ratio; DWI: diffusion-weighted imaging; EEG: electroencephalography; EEG-CBFV: electroencephalography–cerebral blood flow velocity; END: early neurological deterioration; EVT: endovascular treatment; FD: fractal dimension; FIM: Functional Independence Measure; FMA: Fugl-Meyer Assessment; FM-UE: Fugl-Meyer Assessment of the Upper Extremity; FP1: left frontopolar EEG electrode (Fp1) in the International 10–20 system; FTRS: Functional Test for the Hemiplegic Upper Extremity; GCS: Glasgow Coma Scale; h: hours; ICA: internal carotid artery; ICU: intensive care unit; IQR: interquartile range; IS: ischemic stroke; LACI: lacunar infarct; LACS: lacunar syndrome; LVO: large-vessel occlusion; MCA: middle cerebral artery; MI: Motricity Index; min: minutes; MISS-EEG: electroencephalography cohort of the Middelheim's Interdisciplinary Stroke Study; MoCA: Montreal Cognitive Assessment; MRI: magnetic resonance imaging; mRS: modified Rankin Scale; MT: mechanical thrombectomy; N: number; NCSE: nonconvulsive status epilepticus; NIHSS: National Institutes of Health Stroke Scale; NIRS: near-infrared spectroscopy; NR: not reported; NVC: neurovascular coupling; PACI: partial anterior circulation infarct; PCA: posterior cerebral artery; pdBSI: pairwise-derived Brain Symmetry Index; POCS: posterior circulation syndrome; PRI: pairwise ratio index; PSI: phase synchrony index; PWI: perfusion-weighted imaging; QEEG: quantitative electroencephalography; SD: standard deviation; SE: status epilepticus; SIESTA: Sedation versus Intubation for Endovascular Stroke TreAtment; T0: baseline time point (time zero); TACI: total anterior circulation infarct; TAR: theta/alpha ratio; TCD: transcranial Doppler; tDCS: transcranial direct current stimulation; WPLI: weighted phase lag index.

Table 2. Main findings of prognostic, monitoring, severity association, rehabilitation/recovery, cognitive/language, routine EEG, processed EEG, and advanced QEEG studies

Study	Clinical question / outcome	EEG/QEEG marker(s)	Main findings for Results narrative
Agius Anastasi 2017 [32]	Motor impairment and recovery after first-ever stroke	Brain Symmetry Index / revised BSI	BSI showed strong negative correlations with motor scores in a small cohort, including Motricity Index at session 1 (r=-0.829) and Fugl-Meyer score at session 2 (r=-0.812). Supportive but not suitable for row-level pooling.
Ajčević 2021 [14]	Hyperacute anterior-circulation ischemic stroke before reperfusion treatment; neurological severity and imaging/functional outcomes	Relative alpha power, DAR, and other spectral features	Relative alpha power was strongly inversely correlated with admission NIHSS (rho=-0.87), while DAR was strongly positively correlated with admission NIHSS (rho=0.86). Supports spectral slowing and loss of faster activity as acute severity markers.
Akgol Gur 2022 [23]	In-hospital mortality prediction in ED ischemic stroke	Bispectral Index (BIS)	BIS ≤74 showed very high discrimination for in-hospital mortality (AUC: 0.984; 95% CI 0.926-0.999; sensitivity: 93.6%; specificity: 95.9%). Processed EEG evidence, separate from conventional QEEG biomarkers.
Assenza 2013 [17]	Acute MCA stroke; NIHSS and effective recovery	Spectral power over ipsilesional and contralesional MCA territories	Low-frequency powers were associated with acute NIHSS, and several contralesional frequency-band powers were associated with effective recovery. Strongest extracted recovery association: contralesional beta-band power with effective recovery adjusted for baseline NIHSS (r=-0.626).
Batra 2025 [31]	Intra-procedural monitoring during EVT and recanalization physiology	BIS plus NIRS monitoring	BIS changes were retained as paired pre/post recanalization monitoring measures and did not show a clear prognostic association with clinical outcome. NIRS-derived measures changed more consistently with successful recanalization. Physiological monitoring evidence, not QEEG outcome-prediction evidence.
Bentes 2018 [13]	Acute ischemic stroke; unfavorable 12-month functional outcome (mRS ≥3)	Relative delta, alpha, beta power, DTABR, and visual EEG models	Higher delta and DTABR and lower alpha/beta activity were associated with unfavorable outcome. Adjusted DTABR models showed ORs around 1.67-1.70 and cross-validated AUCs around 0.83-0.86. Best extracted visual asymmetry model AUC: 0.89.
Capon 1996 [20]	Rehabilitation-phase hemispheric infarct followed for approximately 3 months	Serial quantitative EEG and brain mapping; delta and band-power measures	The study concluded that neither clinical improvement nor clinical outcome correlated significantly with modifications of EEG or initial EEG data. Use as early descriptive rehabilitation evidence rather than robust supportive prognostic evidence.
Cillessen 1994 [21]	Acute first-ever supratentorial cerebral ischemia; poor 1-year functional outcome	Visual EEG classification with quantitative spectral confirmation	EEG poor-prognosis/major-ischemia classification predicted poor 1-year outcome with TP 11, FP 3, FN 2, TN 39 (sensitivity: 84.6%; specificity: 92.9%; crude OR: 71.5). Severe-baseline-handicap cortical-syndrome subgroup: sensitivity: 91.7%, specificity: 75.0%.
Cuspineda 2007 [18]	Acute MCA ischemic stroke; short- and long-term mRS prediction	QEEG absolute energy Z-score variables in delta, theta, alpha, beta, and total power	QEEG within 72 hours showed predictive value for mRS outcomes. Alpha and theta absolute energy were best predictors for short-term outcome, while delta absolute energy was emphasized for long-term outcome. Reporting was not directly comparable with standard correlation meta-analysis.
Finnigan 2004 [9]	Acute ischemic cortical stroke; 30-day NIHSS and MRI lesion measures	Acute delta change index (aDCI)	aDCI correlated strongly with 30-day NIHSS (rho=0.80, P<0.01), comparable to initial MTT abnormality volume (rho=0.79). aDCI also correlated with 15-hour DWI lesion volume (rho=0.62).
Finnigan 2007 [10]	Subacute ischemic cortical stroke; 30-day NIHSS	DAR, relative alpha power, and related spectral indices	DAR was highly correlated with 30-day NIHSS (rho=0.91, P<0.001), and relative alpha power was inversely correlated with 30-day NIHSS (rho=-0.82, P<0.01). Similar findings were retained using a 19-channel subset.
Hofmeijer 2018 [53]	Acute hemispheric ischemic stroke; contralesional activity and 3-month outcome	DAR, magnitude-squared coherence, and WPLI	DAR was higher in patients than controls in both ipsilateral and contralateral hemispheres. Alpha- and beta-band MSC and WPLI were lower in patients than controls. Poor-outcome patients tended to have lower contralateral connectivity, but extractable quantitative outcome estimates were limited.

Study	Clinical question / outcome	EEG/QEEG marker(s)	Main findings for Results narrative
Kawano 2020 [35]	Post-stroke motor impairment and motor recovery during rehabilitation	Phase Synchrony Index (PSI)	Interhemispheric C3-C4 alpha PSI correlated with baseline FM-UE (rho about 0.56), and contralesional intrahemispheric theta PSI correlated with FM-UE gain (rho about 0.53 overall; rho 0.62 in the severe subgroup). Theta PSI threshold for clinically important recovery: AUC: 0.81, sensitivity: 90%, specificity: 65%.
Liu 2019 [41]	Acute ischemic stroke with large-vessel occlusion; mortality, unfavorable discharge outcome, stenosis physiology	EEG-CBFV phase-amplitude coupling	PAC between CBFV and EEG was strongest in beta and gamma bands and in occipital channels. Global PAC showed AUC: 0.81 for mortality and 0.65 for unfavorable outcome, without extractable thresholds or 2×2 data. PAC asymmetry correlated with degree of stenosis.
Mane 2019 [34]	Chronic stroke upper-limb BCI rehabilitation, with or without tDCS	BSI, PRI, DAR/DTABR, relative band powers during rest and motor imagery	Best prognostic biomarkers differed by intervention: BSI predicted gains after BCI rehabilitation (r=-0.80, P=0.02), whereas PRI predicted gains after tDCS-BCI (r=-0.96, P=0.004). Many dependent feature-level correlations from 19 participants; collapse or narrative synthesis only.
Nicolo 2015 [33]	Future motor and language improvement after stroke	Functional connectivity / weighted node degree	Ipsilesional M1 beta-band WND and contralesional M1 theta-band WND predicted future motor improvement (r=0.57 and r=0.52), reproduced in validation cohort (r=0.60 and r=0.50). Ipsilesional Broca beta and contralesional Broca theta WND predicted language improvement (r=0.69 and r=0.70).
Prandin 2024 [27]	Status epilepticus after EVT and post-EVT outcome	Early routine EEG within 72 hours; NCSE detection	Among 138 EVT patients with early EEG, 9 (6.5%) had status epilepticus, all NCSE. SE was associated with lower NIHSS improvement at discharge (2/9 versus 84/129 improved; p=0.025; unadjusted OR: 6.53 for no significant improvement).
Rogers 2020 [24]	Acute first stroke; 30- and 90-day mRS and modified Barthel Index	Single-channel FP1 EEG; relative theta power and other single-channel metrics	Relative theta power correlated with 30-day mBI (rho=0.60) and 30-day mRS (rho=-0.54). Relative theta cutoff ≥0.25 classified good 30-day mBI outcome with AUC: 0.90, sensitivity: 83.3%, and specificity: 90.0%.
Saes 2021 [36]	Upper-limb motor impairment at 26 weeks after first-ever ischemic stroke	DAR, BSI, directional BSI; particularly BSItheta	BSItheta was the strongest EEG predictor of 26-week FM-UE (beta=0.40; P=0.013), with higher BSItheta predicting lower FM-UE. Adding BSItheta to baseline FM-UE increased explained variance from 61.5% to 68.1% (P=0.006).
Sebastián-Romagosa 2020 [37]	Upper-extremity BCI rehabilitation and motor function	BSI and alpha/beta laterality coefficients during motor imagery	BSI correlated with FMA upper extremity (rho=-0.430, P=0.046). Several alpha-band laterality coefficients were associated with motor and functional scales; one extracted association was laterality coefficient versus FMA upper extremity (rho=-0.706). Pre-post therapy improvement was not treated as a QEEG prognostic effect.
Shen 2024 [26]	Post-mechanical-thrombectomy monitoring; cerebral edema, END, and 90-day dependence	Global/frontal DAR, DTABR, relative delta power	Global and frontal DAR were independently associated with cerebral edema severity, END, and 90-day functional dependence. Frontal DAR had AUC: 0.81 for edema, AUC: 0.81 for END, and AUC: 0.78 for functional dependence; threshold ≥3.3 yielded adjusted ORs 7.3, 8.8, and 6.3.
Sheorajpanday 2011 [11]	Lacunar/posterior circulation syndromes; imaging burden and early functional outcome	pdBSI and DTABR	pdBSI and DTABR correlated with infarct volume (both r about 0.49). In lacunar syndrome, DTABR cutoff >2.4 predicted unfavorable outcome with AUC: 0.88, sensitivity: 100%, and specificity: 77.8%.
Sheorajpanday 2011b [12]	Ischemic stroke; 6-month functional outcome and infarct volume	DTABR and pdBSI	DTABR correlated with 6-month mRS (rho=0.47), and pdBSI correlated with infarct volume (rho=0.54). Adjusted ORs included pdBSI for 6-month disability (OR: 4.07, 95% CI: 1.32-12.58) and DTABR for 6-month dependency (OR: 2.25, 95% CI 1.16-4.37).
Wang 2013 [48]	Cerebral infarction; infarct burden and cognitive impairment context	Relative beta power	Relative beta power was strongly inversely associated with infarct size (r=-0.88881) and infarct number (r=-0.66498). Cognitive impairment classification was interpreted as cross-sectional cognitive/lesion-burden evidence rather than longitudinal prognosis.

Study	Clinical question / outcome	EEG/QEEG marker(s)	Main findings for Results narrative
Wang 2021 [15]	AIS after EVT; poor 90-day functional outcome (mRS 4-6)	DAR, DTABR, relative alpha power	Higher DAR and DTABR and lower relative alpha power characterized poor outcome. DAR ≥14.3 had AUC: 0.93, sensitivity: 90.9%, specificity: 90.0%, and reconstructed counts TP: 10, FP: 2, FN: 1, TN: 18. DTABR showed lower discrimination (AUC: 0.75).
Wolf 2017 [22]	Acute ischemic stroke without seizures; clinical deterioration and hemorrhagic transformation	Routine visual EEG abnormalities, generalized slowing, focal slowing	Abnormal EEG and generalized slowing were associated with clinical deterioration. Reconstructed crude ORs were 11.1 for abnormal EEG and 19.0 for generalized slowing; focal slowing was not predictive of deterioration. Abnormal EEG and focal slowing were also associated with hemorrhagic transformation.
Xin 2012 [19]	Acute stroke; day-28 functional outcome and mortality	Brain Symmetry Index	BSI at admission was higher in patients who died by day 28 than in survivors and correlated with day-28 mRS (r=0.441, P=0.040). BSI also differentiated acute stroke patients from healthy controls.
Xin 2017 [25]	Acute ischemic stroke within 72 hours; 21-day BI and mRS	Revised Brain Symmetry Index	r-BSI was higher in patients with poor BI-defined outcome (BI<60) than in those with BI≥60. Regression models retained r-BSI for both Barthel Index at 21 days and mRS at 21 days, supporting model-based narrative synthesis rather than correlation pooling.
Yan 2025 [29]	Anterior-circulation LVO stroke after EVT; NIHSS and infarct volume	Relative band powers, DAR/TAR/DTABR, coherence, small-world metrics	Relative delta power in the affected hemisphere correlated positively with infarct volume (r=0.73), while relative beta power correlated negatively with infarct volume (r=-0.62). Alpha/beta/gamma powers tended to be negatively related to NIHSS/infarct volume, whereas DAR/TAR/DTABR tended to be positively related.
Yang 2025 [28]	Visual EEG grading after EVT; 3-month prognosis	Serial visual EEG grading at 24 h, day 3, and day 7	EEG grade predicted poor 3-month outcome after EVT. AUCs increased across time points: 0.811 at 24 hours, 0.909 at day 3, and 0.955 at day 7. Day-3 and day-7 moderate/severe abnormality achieved sensitivity: 100% with specificity: 81.8% and 90.9%, respectively.
Zappasodi 2014 [38]	Acute MCA stroke; neurological impairment and 6-month recovery	Higuchi fractal dimension, FD asymmetry, and spectral powers	Global fractal dimension correlated inversely with acute NIHSS (rho=-0.426), and lesioned-hemisphere FD also correlated inversely with NIHSS (rho=-0.471). FD asymmetry correlated with effective recovery at 6 months (r=0.354).
Zhang 2022 [16]	AIS after mechanical thrombectomy; serial QEEG, NIHSS, and 3-month mRS	Delta power, DAR, DTABR at pre-MT, 12 h, 24 h, and 7 d	Delta power at 24 h after MT had the best ROC performance for poor 3-month outcome (AUC: 0.893; sensitivity: 92.9%; specificity: 84.6%). DTABR at 7 days correlated with 3-month mRS (r=0.71), and DAR at 7 days correlated with NIHSS at 7 days (r=0.718).
Zhang 2025 [30]	Stroke outcome, infarct growth, and collateral/coupling physiology	EEG-CBFV coupling indices, DAR, autoregulation metrics	Favorable outcome was associated with higher contralateral coupling in delta and theta bands and higher stroke-side DAR. Contralateral delta and theta coupling correlated inversely with infarct growth (r=-0.461 and r=-0.358), suggesting exploratory neurovascular-coupling evidence.

aDCI: acute delta change index; AIS: acute ischemic stroke; AUC: area under the receiver operating characteristic curve; BCI: brain–computer interface; BI: Barthel Index; BIS: Bispectral Index; BSI: Brain Symmetry Index; CBFV: cerebral blood flow velocity; CI: confidence interval; DAR: delta/alpha ratio; DTABR: delta-theta/alpha-beta ratio; DWI: diffusion-weighted imaging; ED: emergency department; EEG: electroencephalography; EEG-CBFV: electroencephalography–cerebral blood flow velocity; END: early neurological deterioration; EVT: endovascular treatment; FD: fractal dimension; FMA: Fugl-Meyer Assessment; FM-UE: Fugl-Meyer Assessment of the Upper Extremity; FN: false negative; FP: false positive; h: hours; LVO: large-vessel occlusion; mBI: modified Barthel Index; MCA: middle cerebral artery; MRI: magnetic resonance imaging; mRS: modified Rankin Scale; MSC: magnitude-squared coherence; MT: mechanical thrombectomy; MTT: mean transit time; NCSE: nonconvulsive status epilepticus; NIHSS: National Institutes of Health Stroke Scale; NIRS: near-infrared spectroscopy; OR: odds ratio; PAC: phase-amplitude coupling; pdBSI: pairwise-derived Brain Symmetry Index; PRI: pairwise ratio index; PSI: phase synchrony index; QEEG: quantitative electroencephalography; r-BSI: revised Brain Symmetry Index; ROC: receiver operating characteristic; SE: status epilepticus; TAR: theta/alpha ratio; tDCS: transcranial direct current stimulation; TN: true negative; TP: true positive; WND: weighted node degree; WPLI: weighted phase lag index.

Table 3: QUIPS-oriented risk of bias for prognostic, monitoring, recovery, and advanced EEG/QEEG evidence

Study	Overall ROB	Domain-level judgments	Main basis for judgment
Agius Anastasi 2017 [32]	High	Participation: High; Attrition: Unclear; Prognostic factor: Low/unclear; Outcome: Low; Confounding: High; Analysis/reporting: High	Pilot longitudinal case-control motor-recovery study with very small stroke sample, repeated sessions, and limited confounding adjustment.
Ajčević 2021 [14]	High	Participation: High/unclear; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: High	Small hyperacute wireless EEG cohort; exploratory QEEG measures and limited adjustment/external validation.
Akgol Gur 2022 [23]	High/unclear	Participation: Unclear; Attrition: Unclear; Prognostic factor: Low/unclear; Outcome: Low; Confounding: High; Analysis/reporting: High	BIS mortality prediction in ED ischemic stroke; threshold-based analysis without clear validation and limited detail on confounding control.
Assenza 2013 [17]	High	Participation: High; Attrition: Unclear; Prognostic factor: Low; Outcome: Low/unclear; Confounding: High; Analysis/reporting: High	Small exploratory QEEG prognostic study with multiple correlations and limited adjustment.
Batra 2025 [31]	High	Participation: High/unclear; Attrition: High/unclear; Prognostic factor: Low/unclear; Outcome: Low/unclear; Confounding: High; Analysis/reporting: High	Post-hoc EVT monitoring subgroup with BIS/NIRS physiological measures; selection into monitored subgroup and outcome-model reporting limit prognostic inference.
Bentes 2018 [13]	Moderate	Participation: Low/unclear; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: Moderate; Analysis/reporting: Moderate	Larger acute ischemic stroke cohort with standardized qEEG and functional outcome; observational design and residual confounding remain.
Capon 1996 [20]	High	Participation: High/unclear; Attrition: Low/unclear; Prognostic factor: Low/unclear; Outcome: Low; Confounding: High; Analysis/reporting: High	Small rehabilitation cohort with serial qEEG and clinical scales; older methods, limited adjustment, and no clear prognostic association.
Cillessen 1994 [21]	Moderate	Participation: Low/unclear; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: Moderate; Analysis/reporting: Moderate	Consecutive first-ever supratentorial ischemia cohort; EEG interpreted blinded and outcome assessed at 1 year, but sample was modest and classification-based.
Cuspineda 2007 [18]	High	Participation: High/unclear; Attrition: Unclear; Prognostic factor: Low/unclear; Outcome: Low; Confounding: High; Analysis/reporting: High	Small acute MCA stroke cohort with prediction models and leave-one-out validation but no external validation and limited confounding control.
Finnigan 2004 [9]	High	Participation: High; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: High	Very small acute cortical stroke MRI/qEEG cohort; correlation-based prognostic analysis with limited adjustment.
Finnigan 2007 [10]	High	Participation: High; Attrition: Low; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: High	Very small subacute ischemic cortical stroke cohort; strong correlations but limited sample, confounding control, and validation.
Hofmeijer 2018 [53]	High/unclear	Participation: High/unclear; Attrition: Unclear; Prognostic factor: Unclear; Outcome: Unclear; Confounding: High/unclear; Analysis/reporting: High	Conference abstract only with restricted methodological and numerical reporting.
Kawano 2020 [35]	Moderate	Participation: Low/unclear; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: Moderate; Analysis/reporting: Moderate	Observational rehabilitation cohort with standardized PSI and motor outcomes; adjusted analyses were reported, but sample size and exploratory frequency-band testing remain concerns.

Study	Overall ROB	Domain-level judgments	Main basis for judgment
Liu 2019 [41]	High	Participation: High; Attrition: High/unclear; Prognostic factor: Low/unclear; Outcome: Low/unclear; Confounding: High; Analysis/reporting: High	Retrospective neurocritical monitoring study with 16 patients, exploratory PAC metrics and descriptive ROC without complete predictive operating data.
Mane 2019 [34]	High	Participation: High; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: High	Small chronic-stroke RCT subgroup with many intervention-specific QEEG biomarkers and correlations; high within-study multiplicity and limited external validation.
Nicolo 2015 [33]	Moderate	Participation: Moderate; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: Moderate; Analysis/reporting: Moderate	Connectivity recovery study with an independent validation motor cohort and covariate adjustment, but modest samples and multiple network predictors.
Prandin 2024 [27]	High	Participation: High/unclear; Attrition: Unclear; Prognostic factor: Low/unclear; Outcome: Low; Confounding: High; Analysis/reporting: High	Retrospective single-center EVT cohort; EEG within 72 h only in a subset; small SE subgroup and no multivariable model due event count.
Rogers 2020 [24]	High	Participation: High; Attrition: Low; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: High	Pilot single-channel EEG cohort of 16 first-stroke patients; ROC thresholds derived internally and no external validation.
Saes 2021 [36]	Moderate	Participation: Low/unclear; Attrition: Moderate; Prognostic factor: Low; Outcome: Low; Confounding: Moderate; Analysis/reporting: Moderate	Prospective multicenter rehabilitation cohort with standardized 62-channel EEG and 26-week FM-UE; attrition from 55 to 39 and model selection limit certainty.
Sebastián-Romagosa 2020 [37]	High	Participation: High/unclear; Attrition: Unclear; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: High	BCI rehabilitation cohort with many EEG laterality/BSI features and multiple clinical outcomes; no prespecified prognostic model or validation.
Shen 2024 [26]	Moderate	Participation: Moderate; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: Moderate; Analysis/reporting: Moderate	Post-MT cohort with 105 patients, adjusted models for DAR and clinical endpoints; single-center design and data-derived thresholds remain concerns.
Sheorajpanday 2011 [11]	High/unclear	Participation: High/unclear; Attrition: Unclear; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: High	Selected lacunar/posterior circulation syndromes and exploratory qEEG prognostic analyses; small subgroups and ROC/correlation multiplicity.
Sheorajpanday 2011b [12]	Moderate/High	Participation: Low/unclear; Attrition: Unclear; Prognostic factor: Low; Outcome: Low; Confounding: Moderate; Analysis/reporting: Moderate	Larger ischemic-stroke functional-outcome qEEG study with several prognostic metrics; observational design and multiple feature testing.
Wang 2013 [48]	High	Participation: High; Attrition: Low/unclear; Prognostic factor: Low/unclear; Outcome: Low/unclear; Confounding: High; Analysis/reporting: High	Cross-sectional cognitive impairment and infarct-burden study rather than longitudinal prognosis; data-driven classification.
Wang 2021 [15]	High	Participation: High; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: High	Small post-EVT cohort (31 patients) with adjusted ORs and derived cutoffs; limited events and single-center threshold derivation.
Wolf 2017 [22]	Moderate/High	Participation: Moderate/High; Attrition: Low/unclear; Prognostic factor: Low/unclear; Outcome: Low; Confounding: High; Analysis/reporting: Moderate/High	Routine EEG study in AIS without seizures; outcome deterioration and hemorrhagic transformation analyses based on small event counts.

Study	Overall ROB	Domain-level judgments	Main basis for judgment
Xin 2012 [19]	High	Participation: High; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: High	Small acute stroke cohort (22 patients) with BSI correlations to 28-day mRS and mortality; limited adjustment.
Xin 2017 [25]	Moderate	Participation: Moderate; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: Moderate; Analysis/reporting: Moderate	r-BSI prognostic study with multivariable regression for BI/mRS; observational design and model reporting limit certainty.
Yan 2025 [29]	High	Participation: High/unclear; Attrition: Unclear; Prognostic factor: Low/unclear; Outcome: Low/unclear; Confounding: High; Analysis/reporting: High	Post-EVT qEEG/END monitoring study with multiple features, hemispheres, and electrode locations; modest sample and exploratory thresholds.
Yang 2025 [28]	High	Participation: High/unclear; Attrition: Unclear; Prognostic factor: Low/unclear; Outcome: Low; Confounding: High; Analysis/reporting: High	Small post-EVT visual EEG grading cohort; serial thresholds and prognostic 2×2 tables derived from the same sample.
Zappasodi 2014 [38]	Moderate/High	Participation: Moderate/High; Attrition: Low; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: Moderate/High	Nonlinear complexity study with 36 MCA stroke patients; useful mechanistic correlations but small sample and multiple exploratory measures.
Zhang 2022 [16]	High	Participation: High; Attrition: Low/unclear; Prognostic factor: Low; Outcome: Low; Confounding: High; Analysis/reporting: High	Small serial post-MT QEEG cohort with varying EEG availability across time points and multiple ROC/correlation analyses.
Zhang 2025 [30]	High	Participation: High; Attrition: Moderate/unclear; Prognostic factor: Low/unclear; Outcome: Low; Confounding: Moderate/High; Analysis/reporting: High	Single-center retrospective analysis of a prospectively collected EVT registry. The analyzed cohort was highly selected (52 enrolled from 265 EVT patients; exclusions for posterior circulation stroke, age, premorbid disability, severe comorbidity, poor acoustic window, intolerance/sedation, and consent). The selection criteria limit generalizability.

AIS: acute ischemic stroke; BCI: brain–computer interface; BI: Barthel Index; BIS: Bispectral Index; BSI: Brain Symmetry Index; DAR: delta/alpha ratio; ED: emergency department; EEG: electroencephalography; END: early neurological deterioration; EVT: endovascular treatment; FM-UE: Fugl-Meyer Assessment of the Upper Extremity; h: hours; MCA: middle cerebral artery; MRI: magnetic resonance imaging; mRS: modified Rankin Scale; MT: mechanical thrombectomy; NIRS: near-infrared spectroscopy; PAC: phase-amplitude coupling; PSI: phase synchrony index; QEEG: quantitative electroencephalography; r-BSI: revised Brain Symmetry Index; ROB: risk of bias; ROC: receiver operating characteristic; SE: status epilepticus; QUIPS: Quality in Prognostic Studies.

Table 4. GRADE-informed certainty profile for prognostic, monitoring, recovery, cognitive/language, routine EEG, processed EEG, and advanced QEEG domains

Evidence domain / outcome	Studies (participants)	Effect / finding	Risk of bias	Inconsistency	Indirectness	Imprecision	Publication bias	Overall certainty	Key explanation
General prognostic ratio/asymmetry narrative synthesis	10 studies before collapse (~364 EEG-analyzable participants)	No pooled estimate calculated; findings were directionally consistent but clinically heterogeneous.	Serious	Serious	Not serious to serious	Not serious to serious	Not formally assessable; possible concern	LOW	Association direction was generally consistent, but evidence remains exploratory with observational designs, heterogeneous outcomes/settings, and dependent feature-level estimates.
Band-power prognostic features	9 (327 EEG-analyzable plus 202 controls/comparators)	Reduced alpha/beta and increased delta were generally associated with worse outcomes; theta less precise.	Serious	Serious	Serious	Serious	Not formally assessable; possible concern	VERY LOW	Feature definitions, normalization, timing, and outcome domains differed; analyses were supplementary.
Post-EVT/MT monitoring	8 (496 EEG/QEEG/BIS/NVC-analyzable plus 34 controls)	DAR/DTABR/frontal DAR, relative alpha, visual grade, NCSE, and NVC were associated with post-treatment outcomes in separate studies.	Serious	Serious	Not serious to serious	Serious	Not formally assessable; possible concern	LOW	Clinically coherent monitoring domain, but index tests and outcome definitions were too diverse for pooling.
Rehabilitation and motor recovery	8 (252 EEG-analyzable plus 109 controls)	BSI, laterality, PSI, WND, and FD measures were associated with selected motor impairment/recovery outcomes.	Serious to very serious	Serious	Serious	Serious	Not formally assessable; possible concern	VERY LOW	Small studies, varied chronicity and interventions, multiple correlated outcomes/features, and limited replication.
Cognitive and language outcomes	4 (218 EEG-analyzable plus 168 controls)	Beta-power cognitive classification and connectivity-based language recovery evidence; no pooled analysis.	Serious	Serious	Very serious	Serious	Not formally assessable; possible concern	VERY LOW	Different constructs and endpoints; sparse evidence.
Routine/qualitative EEG and processed EEG indices	7 (591 EEG/BIS-analyzable)	Visual EEG abnormalities, EEG grading, NCSE, and BIS were associated with deterioration, poor outcome, or mortality in separate studies.	Serious	Serious	Serious	Serious	Not formally assessable; possible concern	VERY LOW	Clinically relevant but different from QEEG biomarkers; visual/processed indices were not standardized across studies.
Advanced QEEG: connectivity, nonlinear complexity, neurovascular coupling	8 (312 EEG-analyzable plus 119 controls)	Connectivity, WPLI/coherence, PSI, WND, FD, and EEG-CBFV coupling were associated with selected recovery, END, infarct growth, or outcome metrics.	Serious	Very serious	Serious	Serious	Not formally assessable; possible concern	VERY LOW	Metrics are mechanistically important but mathematically heterogeneous and mostly unreplicated.

BIS: Bispectral Index; BSI: Brain Symmetry Index; CBFV: cerebral blood flow velocity; DAR: delta/alpha ratio; DTABR: delta-theta/alpha-beta ratio; EEG: electroencephalography; EEG-CBFV: electroencephalography–cerebral blood flow velocity; END: early neurological deterioration; EVT: endovascular treatment; FD: fractal dimension; MT: mechanical thrombectomy; NCSE: nonconvulsive status epilepticus; NVC: neurovascular coupling; PSI: phase synchrony index; QEEG: quantitative electroencephalography; WND: weighted node degree; WPLI: weighted phase lag index; GRADE: Grading of Recommendations Assessment, Development and Evaluation.

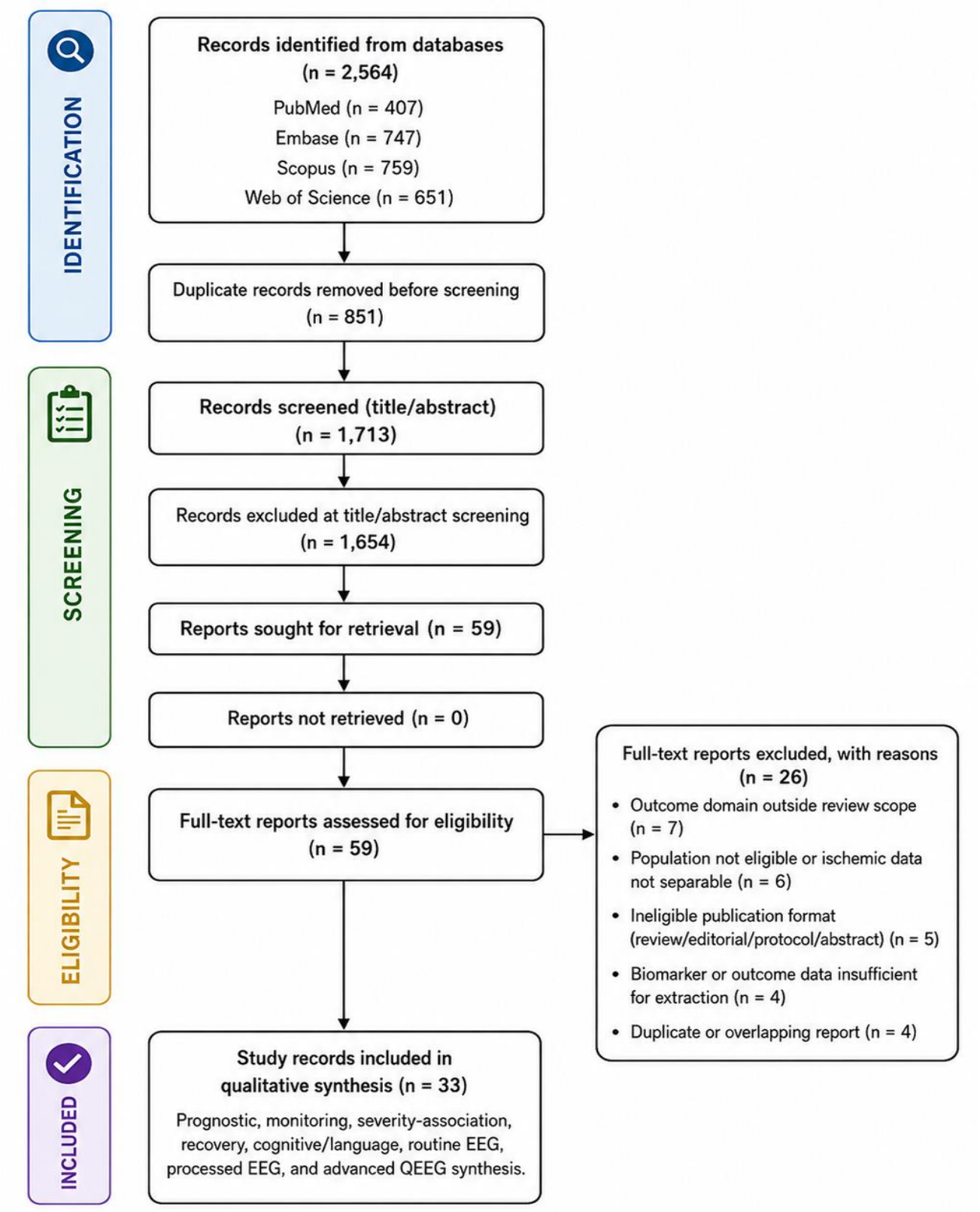


Figure 1: PRISMA 2020 flow diagram for the standalone prognosis, monitoring, severity, and recovery review. EEG: electroencephalography; PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses; QEEG: quantitative electroencephalography.

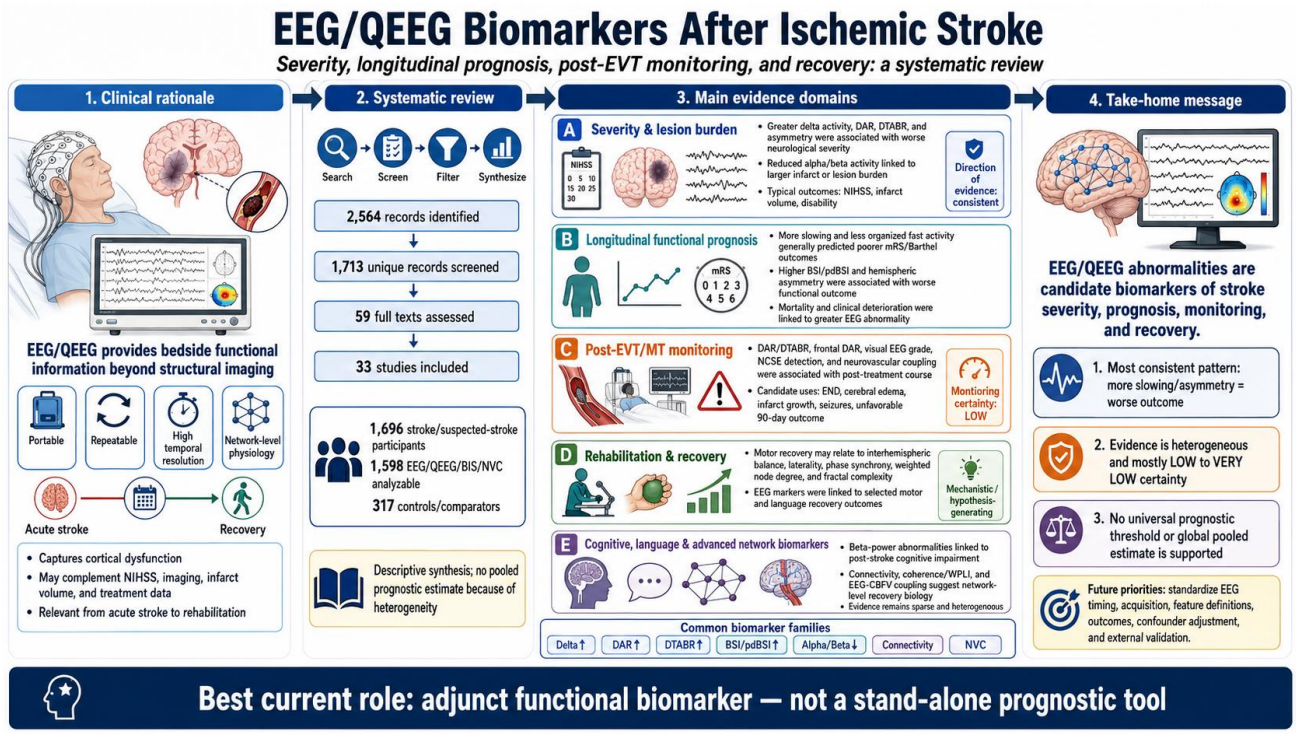


Figure 2: Conceptual overview of EEG/QEEG and EEG-derived biomarkers as candidate functional markers after ischemic stroke. The review synthesized evidence across severity association, lesion burden, longitudinal prognosis, post-EVT/MT monitoring, cognitive/language outcomes, rehabilitation response, and recovery. Candidate markers included slow-wave activity, DAR, DTABR, BSI/pdBSI, visual EEG abnormalities, processed EEG indices, connectivity measures, nonlinear complexity, and EEG-CBFV neurovascular coupling. Overall, greater EEG/QEEG abnormality generally aligned with worse severity, larger lesion burden, poorer functional outcome, deterioration, mortality, or less favorable recovery. However, the evidence remains heterogeneous and mostly low to very-low certainty, and no universal prognostic threshold, stand-alone clinical tool, or global pooled prognostic estimate is currently supported. EEG: electroencephalography; QEEG: quantitative electroencephalography; EVT: endovascular treatment; NIHSS: National Institutes of Health Stroke Scale; MT: mechanical thrombectomy; DAR: delta/alpha ratio; DTABR: delta-theta/alpha-beta ratio ; BSI: Brain Symmetry Index; pdBSI: pairwise-derived Brain Symmetry Index; CBFV: cerebral blood flow velocity.

Appendix

Supplementary Appendix 1: Database search strategies

PubMed

("ischemic stroke"[tiab] OR "ischaemic stroke"[tiab] OR "acute ischemic stroke"[tiab] OR "acute ischaemic stroke"[tiab] OR "cerebral infarct*"[tiab] OR "cerebral ischemia"[tiab] OR "cerebral ischaemia"[tiab] OR "brain ischemia"[tiab] OR "brain ischaemia"[tiab] OR "Brain Ischemia"[Mesh] OR "Cerebral Infarction"[Mesh])

AND

("quantitative EEG"[tiab] OR qEEG[tiab] OR "quantitative electroencephalograph*"[tiab] OR "spectral analys*"[tiab] OR "frequency domain"[tiab] OR "delta alpha ratio"[tiab] OR "delta/alpha ratio"[tiab] OR "alpha delta ratio"[tiab] OR DAR[tiab] OR DTABR[tiab] OR "delta theta alpha beta ratio"[tiab] OR "brain symmetry index"[tiab] OR BSI[tiab] OR pdBSI[tiab] OR "pairwise-derived brain symmetry index"[tiab] OR "revised brain symmetry index"[tiab] OR "laterality coefficient*"[tiab] OR "functional connectivity"[tiab] OR "phase synchron*"[tiab] OR "phase synchrony index"[tiab] OR "weighted node degree"[tiab] OR "fractal dimension"[tiab] OR "neurovascular coupling"[tiab] OR "EEG biomarker*"[tiab] OR "EEG marker*"[tiab])

AND

(prognos*[tiab] OR "functional outcome"[tiab] OR "clinical outcome"[tiab] OR "neurological outcome"[tiab] OR "motor recovery"[tiab] OR "language recovery"[tiab] OR rehabilitat*[tiab] OR "stroke recovery"[tiab] OR severity[tiab] OR NIHSS[tiab] OR "modified Rankin"[tiab] OR mRS[tiab] OR "Barthel Index"[tiab] OR "Fugl-Meyer"[tiab] OR "infarct volume"[tiab] OR "lesion volume"[tiab] OR mortality[tiab])

Embase

('brain ischemia'/exp OR 'cerebral infarction'/exp OR 'ischemic stroke':ti,ab,kw OR 'ischaemic stroke':ti,ab,kw OR 'acute ischemic stroke':ti,ab,kw OR 'acute ischaemic stroke':ti,ab,kw OR 'cerebral infarct*':ti,ab,kw OR 'cerebral ischemia':ti,ab,kw OR 'cerebral ischaemia':ti,ab,kw OR 'brain ischemia':ti,ab,kw OR 'brain ischaemia':ti,ab,kw)

AND

('quantitative eeg':ti,ab,kw OR qeeg:ti,ab,kw OR 'quantitative electroencephalograph*':ti,ab,kw OR 'spectral analys*':ti,ab,kw OR 'frequency domain':ti,ab,kw OR 'delta alpha ratio':ti,ab,kw OR 'delta/alpha ratio':ti,ab,kw OR 'alpha delta ratio':ti,ab,kw OR dar:ti,ab,kw OR dtabr:ti,ab,kw OR 'delta theta alpha beta ratio':ti,ab,kw OR 'brain symmetry index':ti,ab,kw OR bsi:ti,ab,kw OR pdbsi:ti,ab,kw OR 'pairwise-derived brain symmetry index':ti,ab,kw OR 'revised brain symmetry index':ti,ab,kw OR 'laterality coefficient*':ti,ab,kw OR 'functional connectivity':ti,ab,kw OR 'phase synchron*':ti,ab,kw OR 'phase synchrony index':ti,ab,kw OR 'weighted node degree':ti,ab,kw OR 'fractal dimension':ti,ab,kw OR 'neurovascular coupling':ti,ab,kw OR 'eeg biomarker*':ti,ab,kw OR 'eeg marker*':ti,ab,kw)

AND

(prognos*:ti,ab,kw OR 'functional outcome':ti,ab,kw OR 'clinical outcome':ti,ab,kw OR 'neurological outcome':ti,ab,kw OR 'motor recovery':ti,ab,kw OR 'language recovery':ti,ab,kw OR rehabilitat*:ti,ab,kw OR 'stroke recovery':ti,ab,kw OR severity:ti,ab,kw OR nihss:ti,ab,kw OR 'modified rankin':ti,ab,kw OR mrs:ti,ab,kw OR 'barthel index':ti,ab,kw OR 'fugl-meyer':ti,ab,kw OR 'infarct volume':ti,ab,kw OR 'lesion volume':ti,ab,kw OR mortality:ti,ab,kw)

Scopus

TITLE-ABS-KEY("ischemic stroke" OR "ischaemic stroke" OR "acute ischemic stroke" OR "acute ischaemic stroke" OR "cerebral infarct*" OR "cerebral ischemia" OR "cerebral ischaemia" OR "brain ischemia" OR "brain ischaemia")

AND

TITLE-ABS-KEY("quantitative EEG" OR qEEG OR "quantitative electroencephalograph*" OR "spectral analys*" OR "frequency domain" OR "delta alpha ratio" OR "delta/alpha ratio" OR "alpha delta ratio" OR DAR OR DTABR OR "delta theta alpha beta ratio" OR "brain symmetry index" OR BSI OR pdBSI OR "pairwise-derived brain symmetry index" OR "revised brain symmetry index" OR "laterality coefficient*" OR "functional connectivity" OR "phase synchron*" OR "phase synchrony index" OR "weighted node degree" OR "fractal dimension" OR "neurovascular coupling" OR "EEG biomarker*" OR "EEG marker*")

AND

TITLE-ABS-KEY(prognos* OR "functional outcome" OR "clinical outcome" OR "neurological outcome" OR "motor recovery" OR "language recovery" OR rehabilitat* OR "stroke recovery" OR severity OR NIHSS OR "modified Rankin" OR mRS OR "Barthel Index" OR "Fugl-Meyer" OR "infarct volume" OR "lesion volume" OR mortality)

Web of Science

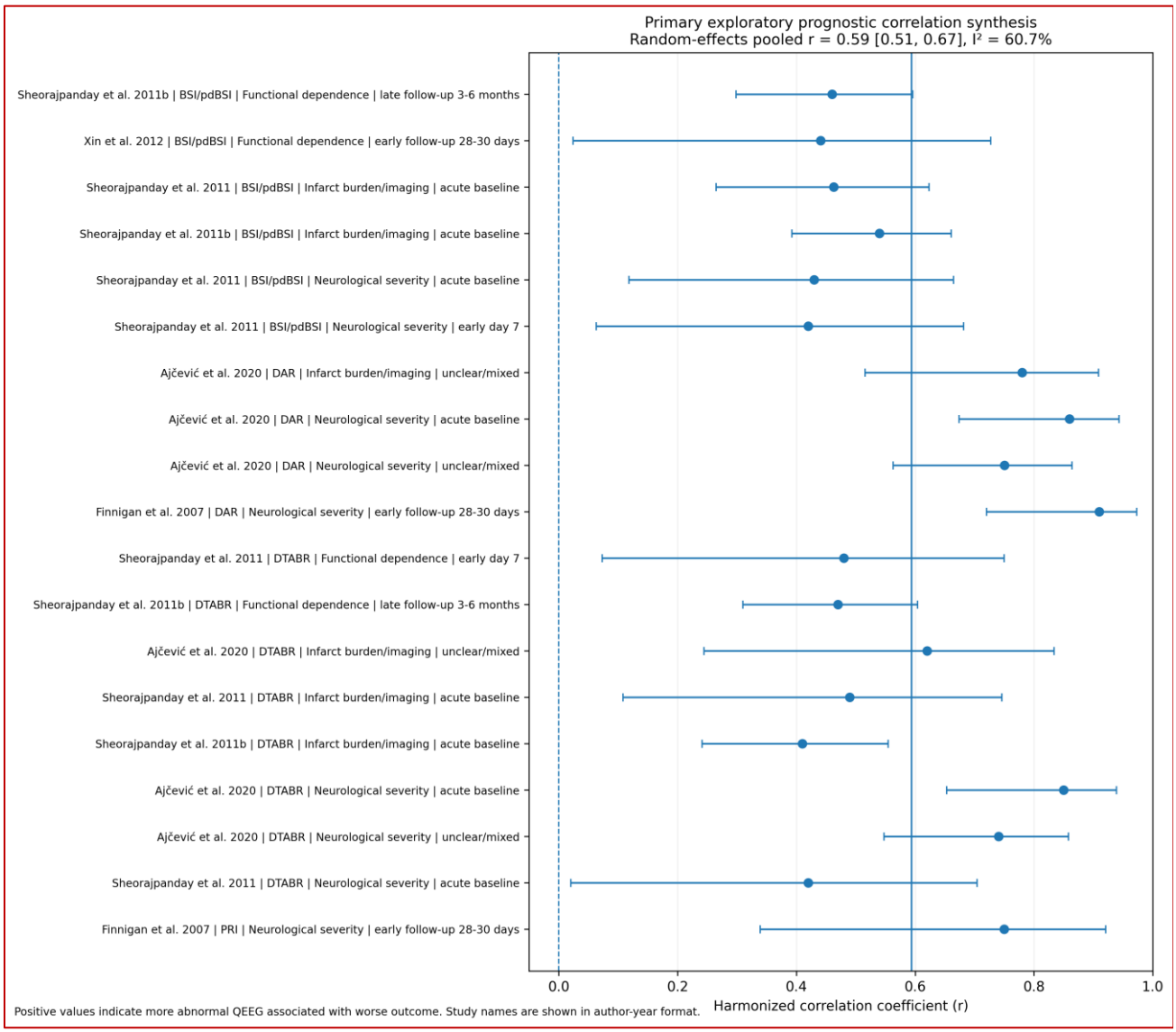
TS=("ischemic stroke" OR "ischaemic stroke" OR "acute ischemic stroke" OR "acute ischaemic stroke" OR "cerebral infarct*" OR "cerebral ischemia" OR "cerebral ischaemia" OR "brain ischemia" OR "brain ischaemia")

AND

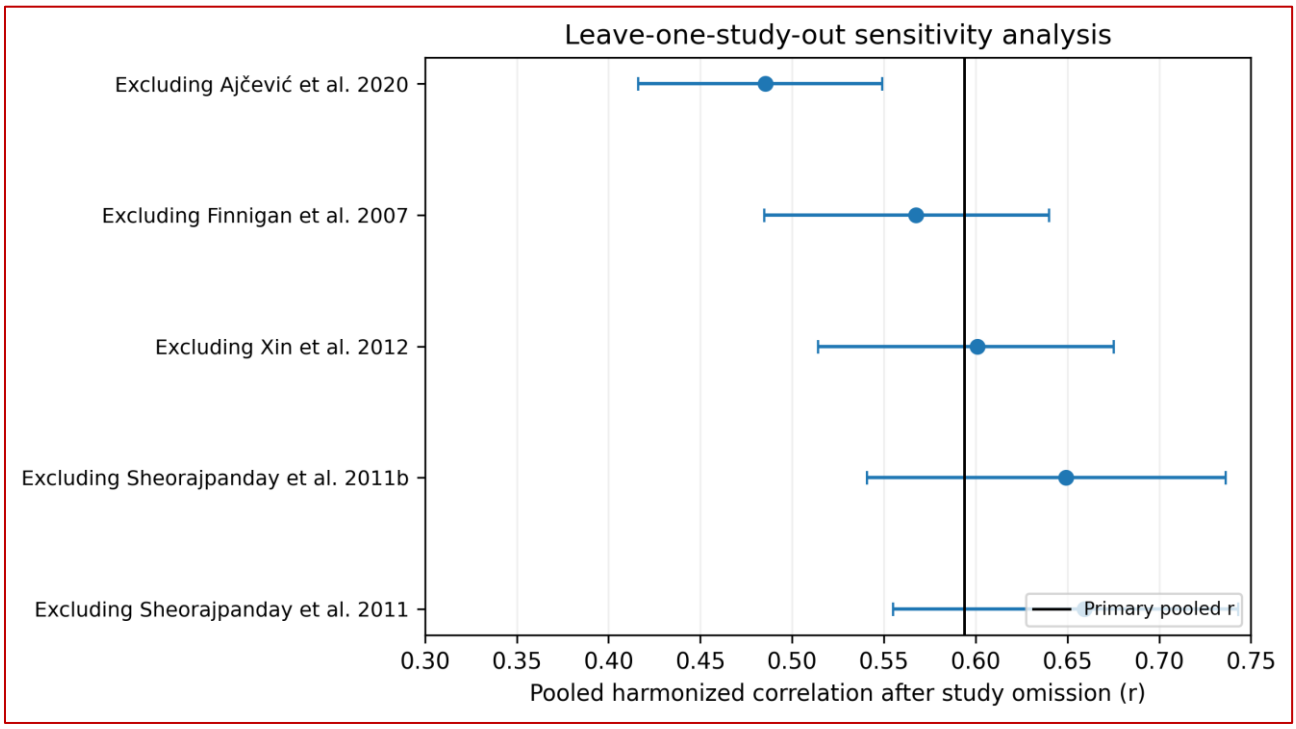
TS=("quantitative EEG" OR qEEG OR "quantitative electroencephalograph*" OR "spectral analys*" OR "frequency domain" OR "delta alpha ratio" OR "delta/alpha ratio" OR "alpha delta ratio" OR DAR OR DTABR OR "delta theta alpha beta ratio" OR "brain symmetry index" OR BSI OR pdBSI OR "pairwise-derived brain symmetry index" OR "revised brain symmetry index" OR "laterality coefficient*" OR "functional connectivity" OR "phase synchron*" OR "phase synchrony index" OR "weighted node degree" OR "fractal dimension" OR "neurovascular coupling" OR "EEG biomarker*" OR "EEG marker*")

AND

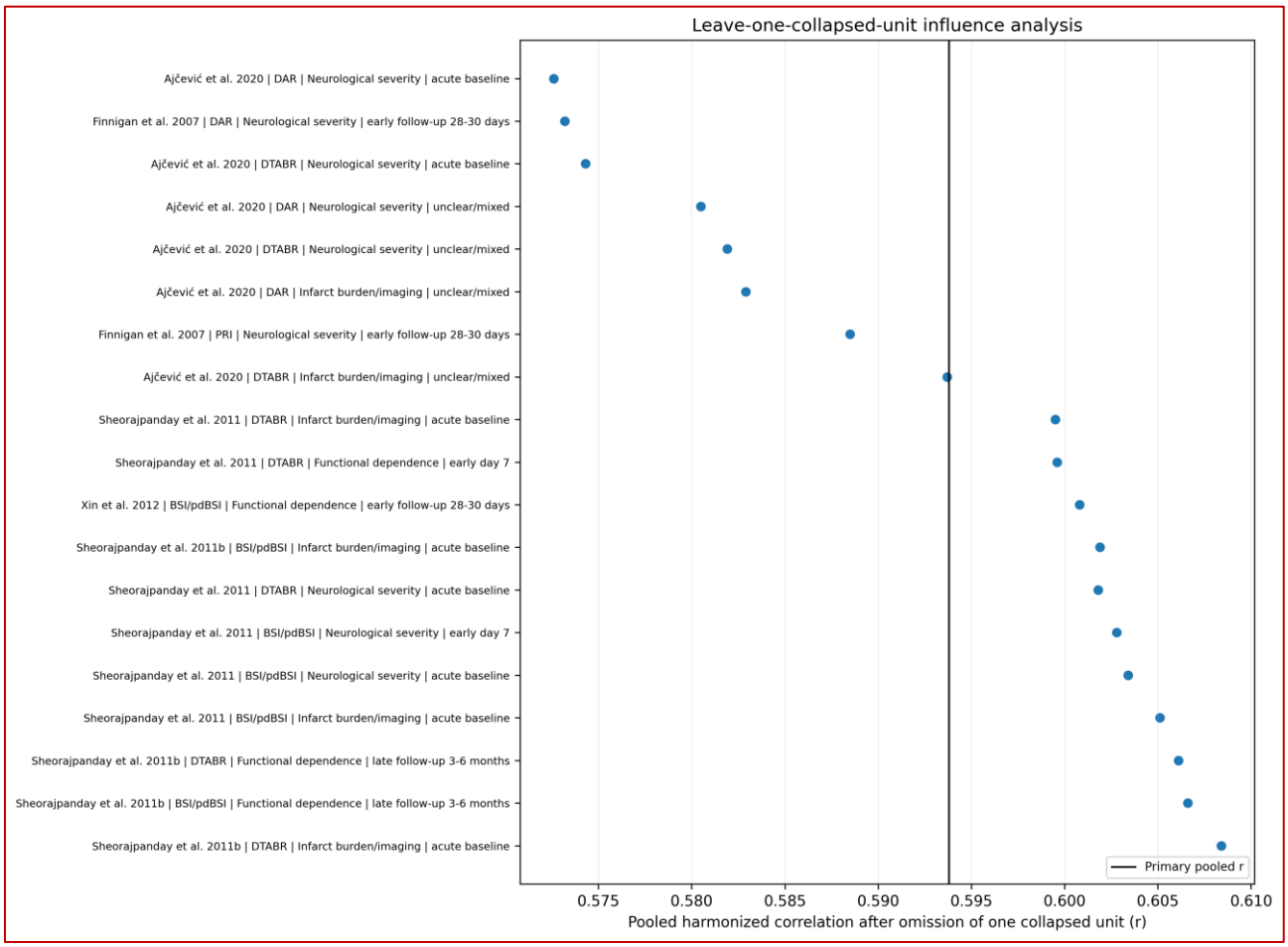
TS=(prognos* OR "functional outcome" OR "clinical outcome" OR "neurological outcome" OR "motor recovery" OR "language recovery" OR rehabilitat* OR "stroke recovery" OR severity OR NIHSS OR "modified Rankin" OR mRS OR "Barthel Index" OR "Fugl-Meyer" OR "infarct volume" OR "lesion volume" OR mortality)



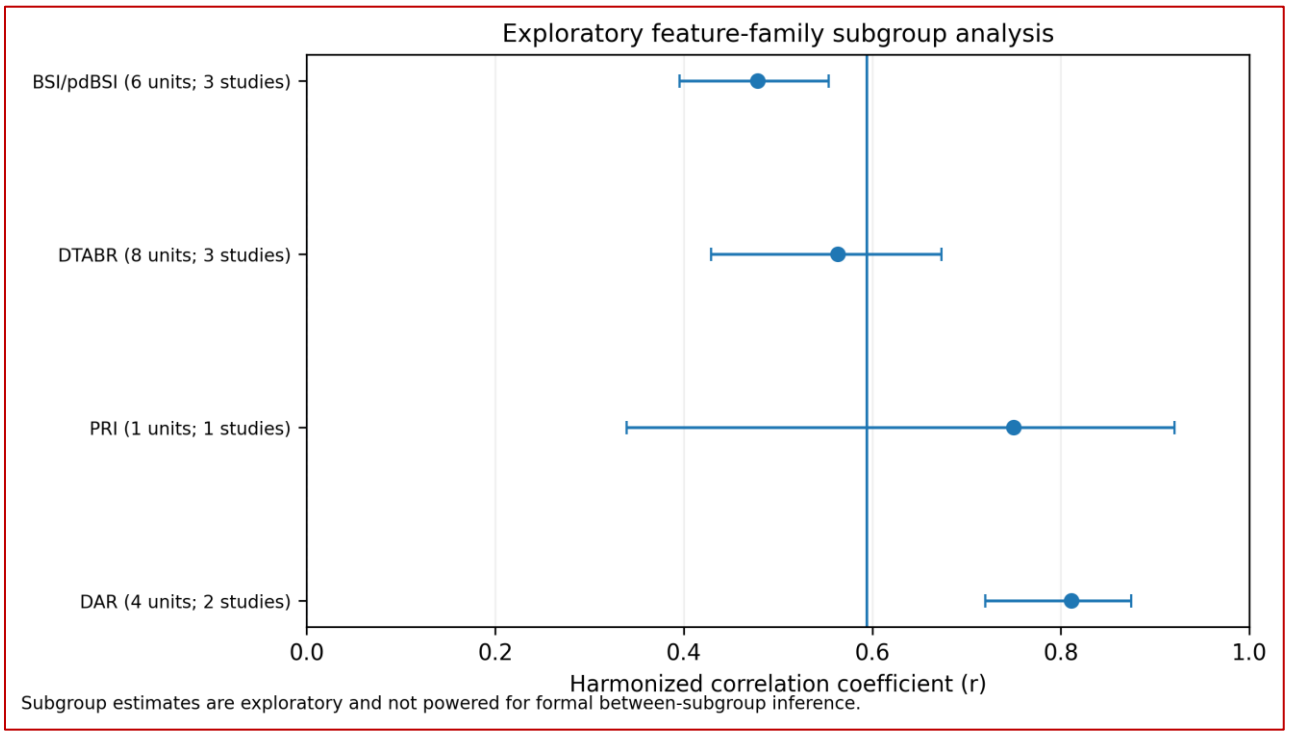
Supplementary Figure S1: Restricted exploratory correlation forest display for prognostic correlation estimates. BSI: Brain Symmetry Index; DAR: delta/alpha ratio; DTABR: delta-theta/alpha-beta ratio; pdBSI: pairwise-derived Brain Symmetry Index; PRI: pairwise ratio index; QEEG: quantitative electroencephalography.



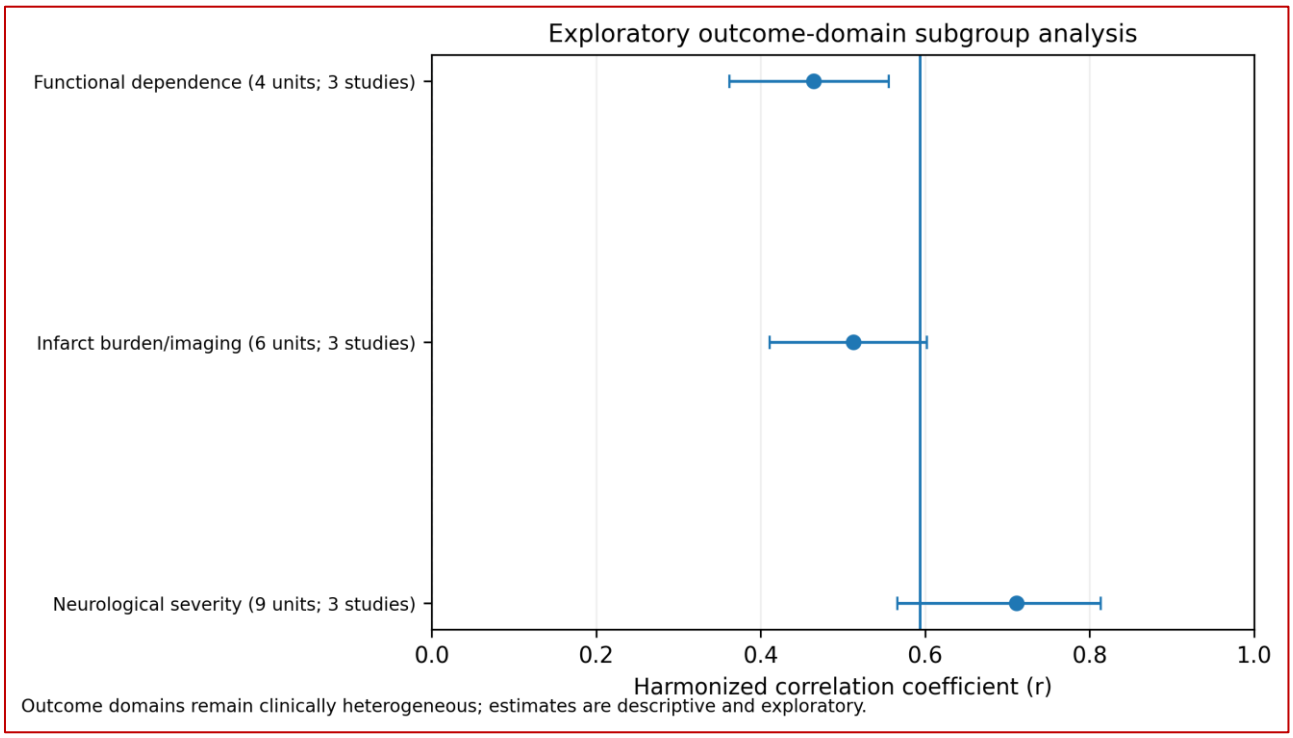
Supplementary Figure S2: Leave-one-study-out influence display.



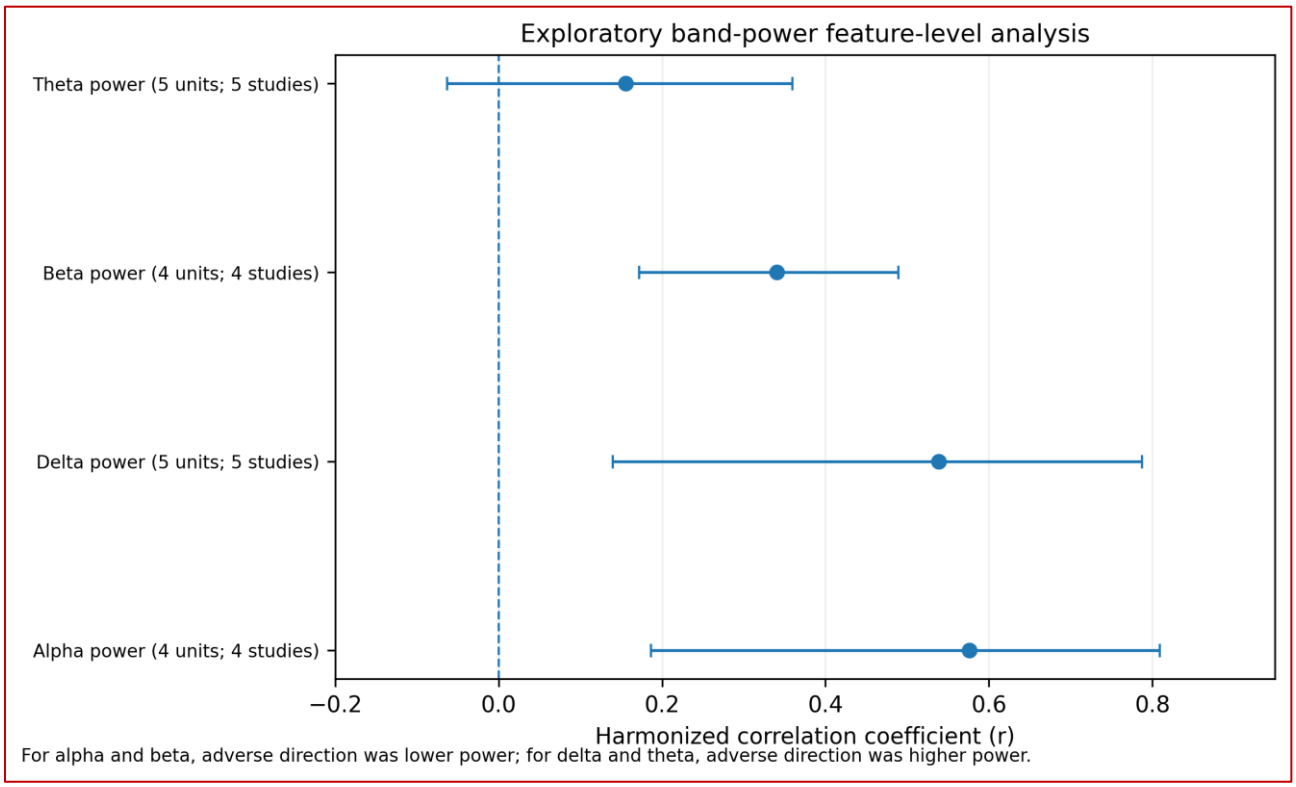
Supplementary Figure S3: Leave-one-collapsed-unit influence display. BSI: Brain Symmetry Index; DAR: delta/alpha ratio; DTABR: delta-theta/alpha-beta ratio; pdBSI: pairwise-derived Brain Symmetry Index; PRI: pairwise ratio index.



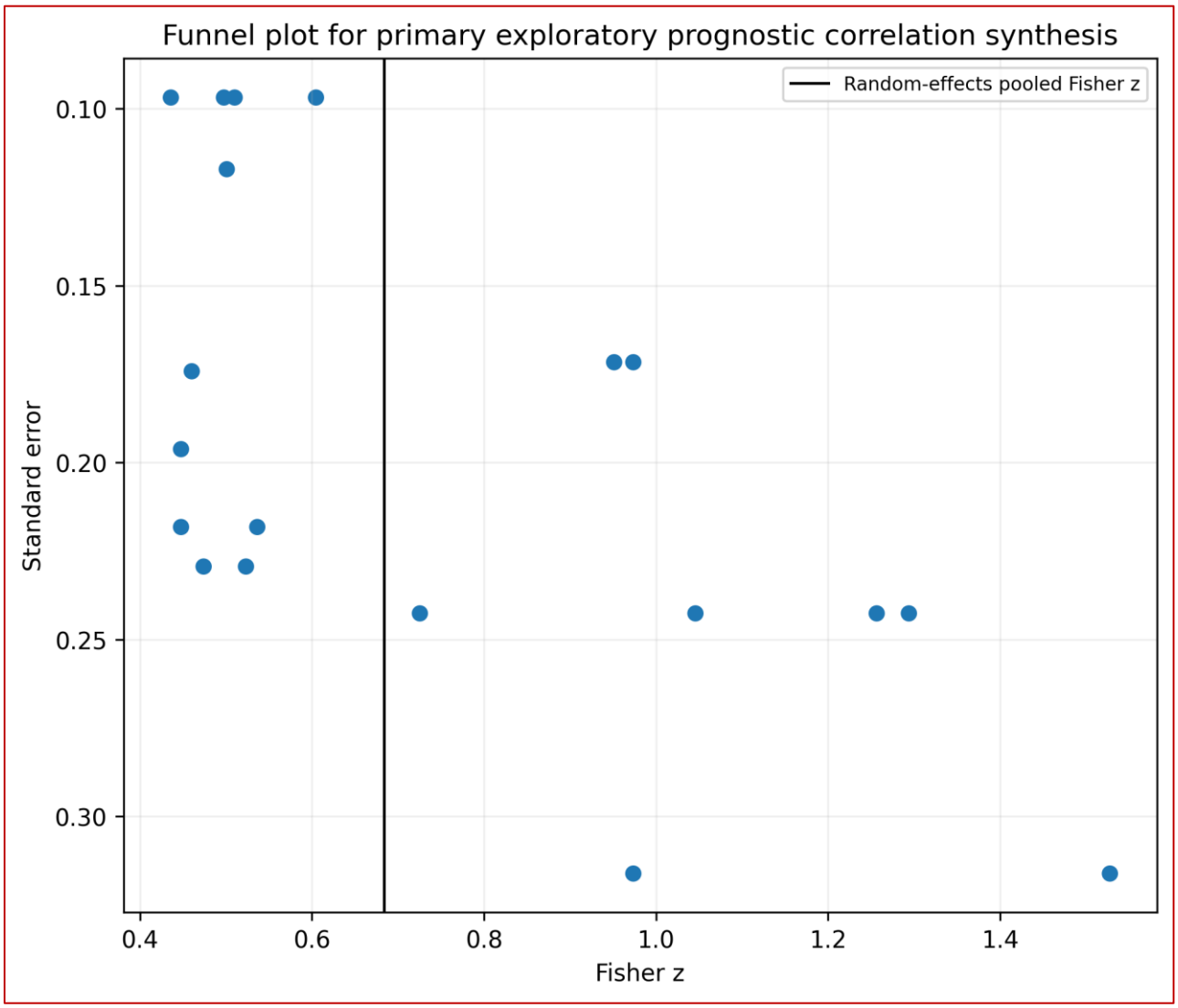
Supplementary Figure S4: Descriptive feature-family subgroup display. BSI: Brain Symmetry Index; DAR: delta/alpha ratio; DTABR: delta-theta/alpha-beta ratio; pdBSI: pairwise-derived Brain Symmetry Index; PRI: pairwise ratio index.



Supplementary Figure S5: Descriptive outcome-domain subgroup display.



Supplementary Figure S6: Descriptive band-power feature-level display.



Supplementary Figure S7: Funnel display for the restricted exploratory prognostic correlation synthesis. This display is not a formal publication-bias assessment.