

An EEG-informed fNIRS framework uncovers stage-associated cortical dynamics during unassisted upper-limb movement after stroke

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Abstract

Background

Upper-limb impairment after stroke is primarily characterized by the emergence and resolution of spastic synergies. Brunnstrom staging captures these recovery phases and provides a clinically grounded framework for studying cortical reorganization. Applying functional near-infrared spectroscopy (fNIRS) during ecological gross-motor tasks introduces task-related systemic hemodynamic artifacts that obscure focal cortical signals. This study aims to determine whether an EEG-informed multimodal framework can effectively mitigate these artifacts and extract stage-sensitive patterns of cortical reorganization.

Methods

After multimodal quality control, 32 participants with stroke (Brunnstrom stages III–VI) and 15 healthy controls were enrolled. EEG and fNIRS were recorded simultaneously during an auditory-cued unilateral arm-raising task with concomitant fist clenching, comprising movement initiation, sustained elevation, and rest. Our framework combined Filter Bank Common Spatial Pattern (FBCSP) with an EEG-informed General Linear Model (GLM). FBCSP extracted focal sensorimotor features, while EEG-derived motor-intention regressors improved GLM modeling of task-related hemodynamic responses. Model performance was evaluated using R^2 and ΔAIC , and feature discriminability using AUC from a linear discriminant analysis (LDA) classifier.

Results

The proposed FBCSP-GLM framework demonstrated improved model fit and spatial discriminability compared with conventional analytical approaches. Participant-level FBCSP estimates revealed a distinct non-linear, stage-associated trajectory of cortical reorganization. Specifically, the FBCSP-derived EW-LI significantly decreased from 0.46 at Stage III to -0.55 at Stage IV, and rebounded to 0.13 at Stage V. The target-M1 response t -score similarly dropped from -0.06 to -1.05 before increasing to 0.85 at Stage V (all FDR-corrected $q < 0.01$). The standard GLM did not capture these stage-associated patterns, as its corresponding comparisons did not survive FDR correction.

Conclusions

By improving hemodynamic model fit during ecological gross-motor execution, the EEG-informed multimodal framework reveals cortical reorganization patterns associated with post-stroke synergistic recovery stages. Its artifact-resilient EW-LI and target-M1 response t -score provide a robust computational assessment of neural state transitions.

Background

Upper limb motor impairment after stroke severely limits functional independence, often characterized by the loss of independent joint control and the emergence of involuntary spastic synergies [1–4]. Tracking the transition through these pathological phases is essential for personalized neurorehabilitation [5]. In clinical practice, continuous scales such as the Fugl-Meyer Assessment (FMA) serve as the gold standard for finely quantifying sensorimotor impairment severity. However, post-stroke motor recovery is not a strictly linear process; it involves the emergence and subsequent resolution of abnormal muscle synergies. To systematically map this nonlinear pathological evolution, the Brunnstrom staging system provides a crucial complementary clinical reference. By delineating the physiological thresholds between obligatory synergies and isolated motor control, it captures the onset, progression, and dissolution of spasticity [6, 7]. Aligning multimodal neuroimaging responses with ordered Brunnstrom stages provides a mechanistic framework to objectively investigate the discrete neural state transitions that occur as the brain overcomes maladaptive plasticity [8–10].

To decode the neural mechanisms underlying these stage-specific transitions, non-invasive neuroimaging modalities, such as electroencephalography (EEG) and functional near-infrared spectroscopy (fNIRS), are increasingly employed [11, 12]. Task-evoked brain responses during ecological gross-motor tasks, such as robot-assisted training, are particularly valuable as they engage motor execution circuits more directly than resting-state paradigms [13–15]. However, acquiring high-fidelity neural signals during dynamic interventions presents significant methodological challenges. Naturalistic upper limb movements inevitably induce task-correlated systemic hemodynamic fluctuations, Valsalva-like physiological noise, and trunk-compensatory motion artifacts [16–18]. Conventional analytical approaches, including standard general linear models (GLM) or isolated time-frequency decompositions, struggle to robustly separate focal motor-execution intents from diffuse systemic noise [19]. Consequently, relying on single-modality or single-parameter connectivity measures is often biased by these spatially diffuse artifacts, rendering longitudinal functional assessments unreliable [20].

Mapping the neural dynamics of synergistic recovery requires quantifying continuous physiological variance to track the metabolic demands of overcoming spasticity. Therefore, optimal neural feature extraction must be evaluated by its goodness-of-fit (R^2) to physiological dynamics and balanced against model parsimony (Akaike information criterion, AIC) to penalize noise-driven overfitting [23]. Fusing multimodal data with advanced spatial filtering algorithms optimized for these criteria is critical to mitigating the pseudo-connectivity issues prevalent in conventional optical neuroimaging [20, 24].

To address this methodological gap, this study proposes a multimodal framework combining EEG-informed filter bank common spatial pattern (FBCSP) feature extraction with a trial-concatenated GLM for fNIRS analysis. High-temporal-resolution EEG priors were used to construct spatially filtered motor-intention regressors, while participant-level GLM statistics were summarized across prespecified sensorimotor (ROIs). By aligning these artifact-resilient statistical summaries with ordered Brunnstrom

stages, this framework aims to systematically map the non-linear cortical dynamics associated with the emergence, exacerbation, and resolution of spastic synergies

In this work, we aim to determine whether, supported by an EEG-informed FBCSP-GLM multimodal framework, we can effectively mitigate severe systemic hemodynamic artifacts to extract stage-sensitive patterns of cortical reorganization in individuals with stroke. This approach has the potential to address a critical methodological limitation in current non-invasive neuroimaging and BCI-based rehabilitation strategies: achieving robust decoding of endogenous sensorimotor intentions while preserving the ecological validity of unassisted gross-motor execution. By yielding artifact-resilient, non-linear statistical features across progressive recovery phases, this framework lays the computational groundwork for developing future state-aware, assist-as-needed robotic neurorehabilitation systems.

Methods

Participants and Clinical Assessment

Following multimodal data quality control, 32 male individuals with subacute stroke and 15 healthy male controls (HC; mean age: 25.7 ± 2.5 years; all right-handed) were included in the final analysis. The clinical cohort was restricted to males because proximal upper-limb and torso sensor placement requires partial upper-body exposure. The inclusion criteria for the stroke cohort were: (1) first-ever unilateral ischemic or hemorrhagic stroke confirmed by neuroimaging; (2) sufficient cognitive capacity to follow experimental instructions (Mini-Mental State Examination ≥ 24); and (3) absence of severe aphasia or other severe concurrent neurological disorders. Individuals in Brunnstrom stages I (flaccid paralysis) and II (minimal voluntary synergistic activation) were excluded because they lack the ability to autonomously initiate voluntary movements, rendering them unable to complete the active upper-limb motor task required for the multimodal (EEG-fNIRS) acquisition in this study. Healthy controls were required to have no history of neurological or psychiatric disorders to establish a normative neurovascular baseline. This cross-sectional study was conducted at the Department of Rehabilitation Medicine, the First Hospital of Jilin University, Changchun, China. The study protocol was approved by the Institutional Ethics Committee of the First Hospital of Jilin University (Approval No. 25K019-001) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment.

To evaluate motor recovery, the participants with stroke were stratified according to their Brunnstrom motor recovery stages (Stages III–VI). Brunnstrom staging was selected as the primary clinical metric because it defines discrete phases of post-stroke motor recovery, accurately capturing the emergence, exacerbation, and dissolution of obligate synergistic patterns [6, 25]. This stage-based grouping provides a mechanistic framework to examine cortical state transitions during recovery from maladaptive synergy, complementing continuous kinematic assessments. Detailed demographic and clinical characteristics, including the stage distribution, are summarized in Table 1.

Table 1
Demographic and clinical characteristics of participants with stroke by Brunnstrom stage.

Characteristics	Stroke Cohort (<i>n</i> = 32)	Healthy Controls (<i>n</i> = 15)
Age (years), mean $\pm$ SD	54.4 $\pm$ 10.9	25.7 $\pm$ 2.5
Sex (Male/Female), <i>n</i>	32 / 0	15 / 0
Pre-stroke Handedness (Right/Left), <i>n</i>	32 / 0	15 / 0
Time since stroke (months), mean $\pm$ SD	4.5 $\pm$ 1.8 (range: 1.0–8.5)	N/A
Hemiplegic side (Left/Right), <i>n</i>	16 / 16	N/A
Brunnstrom Stage (III/IV/V/VI), <i>n</i>	8 / 8 / 8 / 8	N/A
Stroke Type (Ischemic/Hemorrhagic), <i>n</i>	32 / 0	N/A
MMSE Score, mean $\pm$ SD	26.6 $\pm$ 1.6	N/A

SD = Standard Deviation; N/A = Not Applicable.

Experimental Paradigm and Multimodal Acquisition

As shown in Fig. 1, an auditory-cued, unilateral arm-raising task was utilized as an ecologically valid gross-motor paradigm. This unassisted, multi-joint dynamic task challenges the compromised corticospinal integrity of stroke individuals, thereby naturally evoking pathological spastic synergies and pronounced task-correlated systemic hemodynamic fluctuations [26]. Consequently, it provides a robust clinical scenario to evaluate the artifact resilience of the proposed multimodal framework, avoiding the confounding somatosensory feedback introduced by robotic assistance [11].

Each 20-s trial was initiated by an auditory cue, followed by approximately 2s of movement initiation, 6 s of sustained arm elevation with concomitant fist clenching, and subsequent arm lowering into a resting phase. Cortical signals were recorded using a synchronized multimodal EEG-fNIRS system. Continuous EEG data were acquired at 1000 Hz via a wireless system (NeuSen W, Neuracle, China), with spatial filtering restricted to 8 motor-related channels to optimize computational efficiency. Concurrently, hemodynamic responses (at 730 and 850 nm wavelengths) were monitored at 11 Hz using a 20-channel portable fNIRS system (NirSmart, China) covering the bilateral primary motor (M1), premotor cortex (PMC), and primary somatosensory (S1) cortices. The two modalities were aligned using a hardware synchronization box. After quality control, 87 valid motor-execution runs were retained. Each run comprised ten 20-s trials. For algorithmic benchmarking (R^2 and AIC), evaluation was performed at the run level.

Multimodal Signal Preprocessing

All offline signal preprocessing and algorithm implementation were performed using custom scripts in the MATLAB R2021a (MathWorks, USA) environment, supplemented by the EEGLAB (v2025.1.0) and Homer3 (v2.8.0, patch 2.1) toolboxes.

For the fNIRS data, raw optical densities were converted into oxygenated (HbO) and deoxygenated hemoglobin (HbR) concentration changes using the modified Beer-Lambert law, followed by zero-phase bandpass filtering (0.01–0.1 Hz). Subsequent analyses exclusively utilized HbO due to its superior signal-to-noise ratio.

For the EEG data, continuous signals were down-sampled to 500 Hz, bandpass-filtered at 1–80 Hz, notch-filtered at 50 Hz and re-referenced to the common average. Corrupted channels were reconstructed via spherical spline interpolation. Independent component analysis (ICA) with ICLabel was then applied to identify and remove ocular and myogenic artifacts. Finally, trials with peak-to-peak EEG amplitudes exceeding an absolute Z-score of 2.5 were excluded. The retained artifact-free trials were temporally concatenated for continuous GLM analysis.

Spatial Filtering and Feature Extraction

An EEG-informed spatial filtering framework was implemented to mitigate the volume conduction effects of EEG and the limited spatial resolution of fNIRS [27]. Specifically, the FBCSP algorithm, evaluated via a leave-one-out cross-validation (LOOCV) approach, was employed to extract focal motor-intention features [28].

EEG signals from the eight ROI channels were decomposed into narrow frequency bands covering the μ (8–12 Hz) and β (13–30 Hz) ranges to capture distinct sensorimotor rhythms. For EEG-informed regression, motor-related oscillatory features were extracted from the peri-movement window spanning –1 to 2s relative to movement-command onset, capturing cue-induced motor preparation and early movement initiation preceding the delayed hemodynamic response.

In each LOOCV iteration, the spatial projection matrix W was optimized on the training trials by maximizing the Rayleigh quotient:

$$J(W) = \frac{W^T \Sigma_{task} W}{W^T \Sigma_{rest} W} \quad \text{[1]}$$

where Σ_{task} and Σ_{rest} represent the spatial covariance matrices of the motor execution and resting phases, respectively. The optimized matrix W was then applied to the unseen hold-out test trials. Instantaneous power envelopes of the extracted components were computed via the Hilbert transform to serve as neural precursors. These EEG-derived features effectively captured the early sensorimotor neural dynamics associated with motor intent generation, providing a precise temporal prior for the subsequent hemodynamic modeling.

For algorithmic benchmarking, three alternative frameworks were evaluated under the same LOOCV protocols: Task-related component analysis (TRCA) as a spatial filtering reference, Complete ensemble empirical mode decomposition with adaptive noise (CEEMDAN) as a temporal decomposition reference, and the standard GLM as a baseline.

EEG-Informed GLM Integration

A concatenated-trial, EEG-informed GLM framework was employed to model HbO responses across repeated motor executions [28]. The augmented design matrix contained an intercept, a global-signal nuisance regressor, the canonical task hemodynamic response function (HRF) and its temporal derivative, left- and right-hemisphere EEG-informed regressors, their interaction, and discrete cosine transform (DCT) drift terms. The standard GLM contained the intercept, global-signal nuisance regressor, canonical task HRF and derivative, and the same DCT terms.

The canonical task regressor modeled the instructed 8-s physical motor-execution period. The cross-validated EEG power envelopes captured early motor-intention-related activity, were resampled to 11 Hz, and were convolved with the canonical HRF before inclusion as left- and right-hemisphere EEG-informed regressors. The global signal and DCT basis functions (128-s cutoff) were included as nuisance terms. Model-level R^2 and AIC were calculated from the complete fitted models.

$$Y_{HbO} = \beta_0 + \beta_1 X_{global} + \beta_2 X_{DCT} + \beta_3 R_{std} + \beta_4 R'_{std} + \beta_5 R_{EEG,L} + \beta_6 R_{EEG,R} + \beta_7 (R_{EEG,L} \times R_{EEG,R}) + \epsilon \quad (2)$$

For the standard GLM, the channel statistic stored as $t_{Standard}$ corresponded to the canonical task-HRF coefficient. For the EEG-informed models, the stored channel statistic (e.g., t_{FBCSP}) corresponded to the hemisphere-matched EEG-informed regressor: the right EEG regressor for right-hemisphere fNIRS channels and the left EEG regressor for left-hemisphere channels. These two statistics represent distinct model predictors-canonical task timing versus endogenous neural dynamics. They were evaluated in parallel as competing analytical pipelines to directly demonstrate the methodological difference between integrating EEG neural priors and conventional task-locked modeling.

Model estimation utilized generalized least squares (GLS) with a first-order autoregressive [AR (1)] iterative fitting procedure to correct for serial correlations [28]. Statistical inferences were derived using Heteroskedasticity and autocorrelation consistent (HAC) robust covariance estimators. Temporal signal artifacts were excluded using a $1.5 \times \text{IQR}$ criterion before downstream clinical analysis.

Evaluation Metrics and Clinical Reorganization Biomarkers

The proposed multimodal framework was evaluated through a tiered progression from model fidelity to neurophysiological interpretation:

1. Modeling Efficacy (R^2 and ΔAIC): The coefficient of determination (R^2) was used to assess goodness-of-fit to physiological variance, and ΔAIC was used to evaluate model parsimony relative to the standard GLM baseline.
2. Spatial Discriminability (AUC): AUC was calculated within the neurotypical cohort to quantify feature separability while avoiding confounding by stroke-related pathological lateralization.
3. Clinical Reorganization Summaries: EW-LI and the target-M1 response t -score were used as dimensionless summaries of model-specific channel-wise GLM t -statistics across Brunnstrom groups.

Conventional dynamic thresholding methods (e.g., the Wilke method) were developed for high-density fMRI data and can be skewed by isolated outlier channels when applied to sparse fNIRS arrays [19]. To account for the spatial sparsity of the fNIRS montage, we used an ROI-based positive-statistic formulation to calculate the Extent-weighted laterality index (EW-LI). This study-specific measure emphasizes both the magnitude and spatial extent of positive-valued channel statistics.

For each hemisphere H , the regional statistical weight was defined as $W_H = n_{H+} \times \sum (t_i | t_i > 0)$, where n_{H+} is the number of ROI channels with positive model-specific GLM t -statistics. Equivalently, $W_H = (n_{H+})^2 \times \text{mean}(t_i | t_i > 0)$; the formulation therefore intentionally applies additional weighting to spatial extent. EW-LI was then calculated from the target- and non-target-hemisphere weights as shown below.

$$EW - LI = \frac{W_{\text{target}} - W_{\text{nontarget}}}{W_{\text{target}} + W_{\text{nontarget}}} (3)$$

The criterion $t > 0$ selected the positive direction of the statistic and was not a statistical-significance threshold. If both regional weights were zero, EW-LI was recorded as missing. Otherwise, EW-LI ranged from -1 to $+1$: positive values indicated greater target-hemisphere weighting, negative values indicated greater non-target-hemisphere weighting, and zero indicated equal weighting. The target hemisphere was defined as the hemisphere contralateral to the moving hand.

The target-M1 response t -score was calculated as the arithmetic mean of the unthresholded, channel-wise model-specific GLM t -statistics in the predefined target-hemisphere M1 ROI. Positive and negative values were retained. This standardized ROI summary quantifies the robustness of the focal neurovascular response associated with the selected motor-intention regressor.

Statistical Analysis

Participant-level analyses included 32 individuals with stroke (8 per Brunnstrom stage, III–VI) and 15 healthy controls ($N = 47$ total). Stage groups were treated as independent, while FBCSP and standard GLM estimates from the same individual were paired. Group means are reported with 95% confidence intervals (CIs). Between-group comparisons (III–IV, IV–V, V–VI, and VI–HC) were evaluated using two-sided Mann–Whitney U tests for the EW-LI and Welch's t -tests for the target-M1 response t -score. To control for multiple testing, the Benjamini–Hochberg false discovery rate (FDR) correction was applied

separately within each metric and analytical method. Furthermore, Stage V-minus-IV mean contrasts were quantified using Welch–Satterthwaite 95% CIs. Direct between-method performance across this transition was assessed by comparing participant-level FBCSP-minus-Standard differences via Welch's t -tests, with FDR correction applied across the two metrics.

Results

Algorithmic Validation and Signal Fidelity

The FBCSP-GLM framework was first evaluated at the algorithmic level using 87 valid motor-execution runs retained after artifact rejection and quality-control procedures. Its performance was compared with the standard GLM baseline, the spatial filtering method (TRCA) and the temporal decomposition method (CEEMDAN).

Goodness-of-Fit and Model Parsimony

The FBCSP-GLM framework was evaluated at the algorithmic level using the 87 valid motor-execution runs retained after artifact rejection and quality control. Signal fidelity and model complexity were quantified using R^2 and AIC (Fig. 2). Across the 87 runs, the EEG-informed FBCSP-GLM framework demonstrated improved model fidelity compared to the standard GLM baseline, evidenced by higher goodness-of-fit (mean $\Delta R^2 = +0.014$) and enhanced parsimony (mean $\Delta \text{AIC} = -122.58$). This indicates that the FBCSP spatial filters effectively extracted true neurovascular variance.

A bi-metric quadrant analysis was subsequently employed to directly compare FBCSP with TRCA. FBCSP descriptively outperformed TRCA, simultaneously achieving higher R^2 and lower AIC in 56.3% (49/87) of the runs. An additional 5.7% (5/87) showed higher R^2 without improved AIC, yielding a total of 62.1% (54/87) runs with higher R^2 . Conversely, lower AIC without improved R^2 was observed in 4.6% (4/87) of the runs. TRCA achieved both higher R^2 and lower AIC in 33.3% (29/87) of the runs. While CEEMDAN reduced temporal drift, it did not optimize spatial variance, resulting in lower R^2 gains than spatial filtering methods.

Spatial Discriminability of Motor Intent

We next examined whether the improved signal fidelity translated into spatial separation of motor-related activity. Area under the curve (AUC) was utilized to quantify the discriminability between the target M1 and contralateral non-target regions within the neurotypical cohort.

The standard GLM yielded a baseline spatial discriminability with a mean AUC of 0.66 ± 0.10 (Fig. 3). Application of CEEMDAN decreased this discriminability (mean AUC = 0.62 ± 0.09), likely reflecting the over-smoothing of task-evoked hemodynamic peaks. In contrast, spatial filtering approaches enhanced topographical separability: TRCA increased the mean AUC to 0.71 ± 0.12 , while FBCSP achieved the highest classification performance (mean AUC = 0.73 ± 0.12). These results align with established BCI

performance benchmarks [29], demonstrating the efficacy of FBCSP in isolating focal motor-execution features from diffuse systemic artifacts.

Stage-Associated EW-LI and Target-M1 Response t-Score Patterns

Following algorithmic validation, participant-level EW-LI and target-M1 response *t*-scores were evaluated across Brunnstrom groups III–VI and healthy controls (Fig. 4). FBCSP and Standard results are presented in separate panels to delineate the specific response patterns extracted by the EEG-informed versus canonical task regressors.

Stage-Associated Pattern of Interhemispheric Lateralization

For the FBCSP EEG-informed regressor, the EW-LI exhibited a distinct Stage IV-centered decrease-and-rebound pattern. Mean EW-LI significantly decreased from 0.46 at Stage III to -0.55 at Stage IV (BH-FDR $q = 0.001$), indicating a pronounced shift toward contralesional hemisphere weighting. Subsequently, it significantly increased to 0.13 at Stage V ($q = 0.004$). Mean EW-LI was 0.26 at Stage VI and 0.43 in healthy controls; neither the Stage V–VI comparison nor the separate Stage VI–HC comparison was significant (both $q = 0.328$) (Fig. 4A).

The Standard canonical task regressor did not capture this dynamic lateralization shift. Mean EW-LI values were 0.49 , -0.17 , 0.05 , 0.25 , and -0.05 at Stages III, IV, V, VI, and in healthy controls, respectively (Fig. 4B). None of the four comparisons survived FDR correction (III–IV $q = 0.152$; IV–V, V–VI, and VI–HC $q = 0.599$).

Stage-Associated Pattern in the Target-M1 Response t-Score

Consistent with the EW-LI findings, the FBCSP-derived target-M1 response *t*-score showed a corresponding Stage IV-centered decrease-and-rebound pattern. The mean significantly decreased from -0.06 at Stage III to -1.05 at Stage IV ($q < 0.001$), reflecting the nadir of ipsilesional localized activation. This was followed by a significant neuroplastic rebound, increasing to 0.85 at Stage V (Stage V-minus-IV mean contrast = 1.90 , 95% CI 1.18 to 2.62 ; $q < 0.001$) (Fig. 4C). The mean was 1.07 at Stage VI and 0.81 in healthy controls; neither the Stage V–VI comparison ($q = 0.534$) nor the separate Stage VI–HC comparison ($q = 0.529$) was significant.

In contrast, estimates derived from the Standard task regressor lacked the sensitivity to map these early-to-mid recovery dynamics. Mean target-M1 response *t*-scores were 0.81 , -0.89 , -0.54 , 1.40 , and 1.26 at Stages III, IV, V, VI, and in healthy controls, respectively (Fig. 4D). The Stage III–IV and Stage IV–V comparisons did not survive FDR correction ($q = 0.242$ and $q = 0.833$, respectively). While the Stage V–VI comparison was significant ($q = 0.026$), the separate Stage VI–HC comparison was not ($q = 0.833$).

Topographical Mapping of Model-Specific Response Statistics

To facilitate visualization and group-level comparisons, all topographical maps were spatially flipped such that the executing limb corresponds to the left hand. Consequently, the right hemisphere uniformly represents the target motor cortex (i.e., contralateral to the movement for healthy controls, and the ipsilesional hemisphere for individuals with stroke). 3D topographical brain maps were generated for individuals whose EW-LI most closely approximated the group median for each Brunnstrom stage and the healthy control cohort, as shown in Fig. 5.

The FBCSP-derived topographical maps visually paralleled the stage-associated statistical patterns. Healthy controls exhibited typical focal activation in the target (right) sensorimotor areas. At Stage III, the FBCSP maps showed overall faint activation, with slight engagement localized primarily to the target PMC, consistent with severely impaired descending motor drive. At Stage IV, cortical activation was predominantly observed in the non-target (contralesional, left) hemisphere, alongside only weak activation in the target hemisphere. This visually captures the compensatory reliance on uncrossed pathways and aligns with the nadir of the Stage IV-centered decrease-and-rebound pattern. At Stage V, the activation pattern became bilateral, but with more pronounced engagement shifting back to the target (ipsilesional) hemisphere, reflecting the neuroplastic effort associated with overcoming pathological synergies. At Stage VI, the maps demonstrated a return to target-hemisphere dominance, closely resembling the focal pattern observed in healthy controls [30].

Conversely, topographical maps derived from the standard GLM exhibited more diffuse and variable spatial distributions across these stages, which did not clearly delineate this specific stage-associated neuroplastic trajectory. Taken together, the visual and statistical evidence suggests that the FBCSP-GLM framework provides a more distinct mapping of non-linear cortical reorganization during post-stroke motor recovery.

Discussion

This study proposed and validated an EEG-informed FBCSP-GLM framework for decoding discrete, stage-specific neurophysiological trajectories of upper-limb motor restitution after stroke. By accounting for the temporal dissociation between neuronal activity and hemodynamic responses, our approach successfully extracted artifact-resilient neural features during unassisted gross-motor tasks. Leveraging this refined multimodal analytical strategy, we identified a non-linear EW-LI pattern and a pronounced Stage IV-centered decrease-and-rebound pattern in the target-M1 response t -score spanning Stages III to V. These findings underscore the critical value of anchoring neurovascular assessments to mechanistically grounded, stage-specific clinical metrics to decode the emergence and recovery of spastic synergies.

Overcoming Systemic Artifacts via Spatial Deconvolution

A persistent challenge in neurorehabilitation imaging is that active, unassisted gross-motor tasks induce systemic hemodynamic fluctuations (including Valsalva-like responses) along with compensatory trunk movements [19, 26, 31]. Our algorithmic benchmarking demonstrated that the proposed FBCSP-GLM framework yielded superior signal fidelity and spatial discriminability compared to the standard GLM and temporal decomposition techniques (e.g., CEEMDAN). By integrating high-temporal-resolution EEG priors, the spatial filtering approach effectively mitigates diffuse systemic artifacts, offering a more robust and accurate representation of true focal neurovascular coupling. This capability addresses a critical methodological challenge in ecological neuroimaging, allowing for a more sensitive evaluation of endogenous cortical activity during high-effort dynamic tasks.

Interpreting the Stage-Associated EW-LI and Target-M1 Response Patterns

Clinically, Stage IV marks the initial decline of spasticity and the critical turning point where patients first attempt to deviate from obligatory synergies. Our analysis captured a pronounced V-shaped nadir in network lateralization (negative EW-LI) and a negative target-M1 offset precisely at this stage. This substantial contralesional activation reflects the immense neural transmission resistance and metabolic load encountered when the motor system attempts to break rigid synergies to recruit isolated movements. Because the recovering ipsilesional corticospinal tract (CST) remains insufficient to independently drive this nascent isolated control, the system heavily recruits the contralesional hemisphere and uncrossed descending pathways as a phase-specific compensation. This intense, effort-driven transcallosal network shift directly drives the observed EW-LI nadir. Subsequently, the transition to Stage V demonstrates a positive ipsilesional target-M1 rebound, reflecting the successful re-engagement of the ipsilesional network to solidify isolated joint control [8, 33]. Overcoming obligatory synergies requires the recovering ipsilesional hemisphere to actively suppress contralesional reticulospinal interference while recruiting surviving CST fibers [32]. Our EEG-informed framework successfully captured this heightened neuroplastic demand, demonstrating a positive ipsilesional target-M1 rebound at Stage V. In contrast, the standard GLM did not detect this discrete phase transition, highlighting the enhanced sensitivity of our multimodal methodology to localized target-M1 responses. This positions the FBCSP-derived t -score as a highly promising neuroimaging biomarker for stage-specific motor restitution.

Clinical Implications for Neurorehabilitation and BCI

The ability to accurately decode these discrete neuroplastic phase transitions holds substantial implications for the optimization of next-generation brain-computer interface (BCI) systems. Current BCI paradigms frequently extract neural features during robot-assisted movements; however, continuous mechanical torque and peripheral proprioceptive feedback can inherently obscure endogenous cortical motor preparation [11, 34].

By employing an unassisted, anti-gravity gross-motor paradigm, our framework establishes a rigorous, artifact-resilient baseline for decoding endogenous motor intents. This state-aware capability is a

prerequisite for developing adaptive, assist-as-needed (AAN) interventions. For instance, rehabilitation strategies could incorporate non-invasive brain stimulation (NIBS) for stroke survivors identified at Stage IV to downregulate the contralesional hemisphere, actively counteracting maladaptive reticulospinal synergies [35]. Conversely, as individuals progress to Stage V, BCI-driven upper-limb exoskeletons could utilize the target-M1 response *t*-score as a personalized, offline calibration threshold to trigger timed mechanical assistance, thereby reinforcing true CST-driven motor execution [36].

Limitations and Future Directions

The male-only cohort restricts the generalization of findings across sexes, as it may not fully capture sex-related differences in neuroplasticity or muscular activation patterns. Expanding to larger, multi-center trials will further enhance generalizability across age-related vascular changes and stroke-specific individual lesion profiles. Future studies will explore modified sensor configurations or alternative wearable designs to facilitate the inclusion of female participants. Furthermore, while the current cohort provides robust initial validation across discrete Brunnstrom stages, expanding to larger, multi-center trials will further enhance generalizability. Factors such as age-related vascular changes and stroke-specific individual lesion profiles introduce variance in neurovascular coupling [38, 39]. To translate these cross-sectional statistical patterns into clinical BCI applications, ongoing and future efforts are incorporating blinded, stratified pilot validation protocols and longitudinal tracking to verify online reliability and durability during dynamic interventions [40]. Finally, integrating high-density electromyography (sEMG) will help elucidate the underlying cortico-muscular coupling mechanisms governing synergistic recovery [41, 42]

Conclusions

The proposed FBCSP-GLM framework demonstrated improved model fit and spatial discriminability during unassisted gross-motor execution compared with conventional analytical approaches. Participant-level FBCSP estimates showed a non-linear, stage-associated trajectory of cortical reorganization, specifically revealing a pronounced V-shaped pattern spanning Brunnstrom Stages III to V. By extracting artifact-resilient features—the EW-LI and target-M1 response *t*-score—this approach computationally captured both the exacerbation of spastic synergies and the subsequent restitution of localized motor control. Ultimately, these stage-sensitive statistical summaries provide a robust neuroimaging baseline for evaluating neural state transitions.

Abbreviations

fNIRS

Functional near-infrared spectroscopy

EEG

Electroencephalography

FBCSP

Filter bank common spatial pattern

GLM
General linear model
LI
Laterality index
BCI
Brain-computer interface
M1
Primary motor cortex
PMC
Premotor cortex
S1
Primary somatosensory cortex
CST
Corticospinal tract
RST
Reticulospinal tract
CEEMDAN
Complete ensemble empirical mode decomposition with adaptive noise
TRCA
Task-related component analysis
AUC
Area under the curve
AIC
Akaike information criterion
HRF
Hemodynamic response function
DCT
Discrete cosine transform
ROI
Region of interest
NIBS
Non-invasive brain stimulation
AAN
Assist-as-needed
BH-FDR
Benjamini–Hochberg false discovery rate
EW-LI
Extent-weighted laterality index
CI
Confidence interval

HbO
Oxygenated hemoglobin
HbR
Deoxygenated hemoglobin
ICA
Independent component analysis
LOOCV
Leave-one-out cross-validation
LDA
Linear discriminant analysis
GLS
Generalized least squares
HAC
Heteroskedasticity and autocorrelation consistent
IQR
Interquartile range
HC
Healthy control
FDR
False discovery rate
EMG
Electromyography
AR (1)
First-order autoregressive model

Declarations

Ethics approval and consent to participate

The study was approved by the Institutional Ethics Committee of the First Hospital of Jilin University (Approval No. 25K019-001) and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to their enrollment.

Consent for publication

Consent for publication was obtained from all participants included in this study. Written informed consent was acquired prior to the commencement of the study, and participants were informed that the results may be published in peer-reviewed journals and presented at scientific conferences. All participants provided their consent voluntarily, and no incentives were offered for their agreement. Data from participants who did not provide consent for publication were excluded from the analysis and subsequent publication.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

Conception and design: X.C. and M.W. Data analysis: M.W. Data collection: Z.L. Writing manuscript: M.W. and X.C. Recruitment of participants: Z.L. Interpretation of the results and statistics: M.W. and Y.G.

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Data Availability

The datasets and custom code supporting this study, including scripts for signal extraction and analysis, are available from the corresponding author upon reasonable request.

References

1. Feigin VL, Stark BA, Johnson CO, Roth GA, Bisignano C, Abady GG, et al. Global, regional, and national burden of stroke and its risk factors, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet Neurol.* 2021;20(10):795–820.
2. Lang CE, MacDonald JR, Reisman DS, Boyd L, Jacobson Kimberley T, Schindler-Ivens SM, et al. Observation of amounts of movement practice provided during stroke rehabilitation. *Arch Phys Med Rehabil.* 2009;90(10):1692–8.
3. Dewald JPA, Pope PS, Given JD, Buchanan TS, Rymer WZ. Abnormal muscle coactivation patterns during isometric torque generation at the elbow and shoulder in hemiparetic subjects. *Brain.* 1995;118(Pt 2):495–510. 10.1093/brain/118.2.495.
4. Twitchell TE. The restoration of motor function following hemiplegia in man. *Brain.* 1951;74(4):443–80.

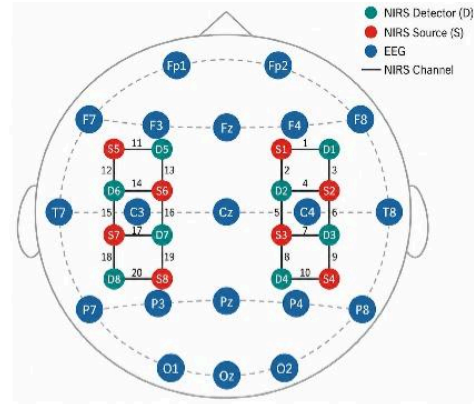
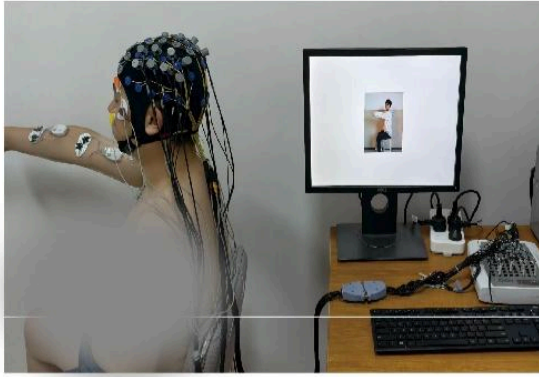
5. Krakauer JW. Motor learning: its relevance to stroke recovery and neurorehabilitation. *Curr Opin Neurol.* 2006;19(1):84–90.
6. Brunnstrom S. Motor testing procedures in hemiplegia: based on sequential recovery stages. *Phys Ther.* 1966;46(4):357–75.
7. Fugl-Meyer AR, Jaasko L, Leyman I, Olsson S, Steglind S. The post-stroke hemiplegic patient. 1. a method for evaluation of physical performance. *Scand J Rehabil Med.* 1975;7(1):13–31.
8. Ward NS, Brown MM, Thompson AJ, Frackowiak RSJ. Neural correlates of motor recovery after stroke: a longitudinal fMRI study. *Brain.* 2003;126(11):2476–96.
9. Grefkes C, Fink GR. Reorganization of cerebral networks after stroke: new insights from neuroimaging with connectivity approaches. *Brain.* 2011;134(5):1264–76.
10. Belda-Lois JM, Mena-del Horno S, Bermejo-Bosch I, Moreno JC, Pons JL, Farina D, et al. Rehabilitation of gait after stroke: a review towards a top-down approach. *J Neuroeng Rehabil.* 2011;8(1):66.
11. Vidaurre C, Irastorza-Landa N, Sarasola-Sanz A, Insausti-Delgado A, Ray AM, Bibián C, et al. Challenges of neural interfaces for stroke motor rehabilitation. *Front Hum Neurosci.* 2023;17:1070404.
12. Silasi G, Murphy TH. Stroke and the connectome: how connectivity guides therapeutic intervention. *Neuron.* 2014;83(6):1354–68.
13. Cirstea MC, Levin MF. Compensatory strategies for reaching in stroke. *Brain.* 2000;123(5):940–53.
14. Schwarz A, Kanzler CM, Lamercy O, Luft AR, Veerbeek JM. Systematic review on kinematic assessments of upper limb movements after stroke. *Stroke.* 2019;50(3):718–27.
15. Blank AA, French JA, Pehlivan AU, O'Malley MK. Current trends in robot-assisted upper-limb stroke rehabilitation: promoting patient engagement in therapy. *Curr Phys Med Rehabil Rep.* 2014;2(3):184–95.
16. Scholkmann F, Tachtsidis I, Wolf M, Wolf U. Systemic physiology augmented functional near-infrared spectroscopy: a powerful approach to study the embodied human brain. *Neurophotonics.* 2022;9(3):030801.
17. Scholkmann F, Kleiser S, Metz AJ, Zimmermann R, Mata Pavia J, Wolf U, et al. A review on continuous wave functional near-infrared spectroscopy and imaging instrumentation and methodology. *NeuroImage.* 2014;85(Pt 1):6–27.
18. Alawieh A, Zhao J, Feng W. Factors affecting post-stroke motor recovery: Implications on neurotherapy after brain injury. *Behav Brain Res.* 2018;340:94–101.
19. Tachtsidis I, Scholkmann F. False positives and false negatives in functional near-infrared spectroscopy: issues, challenges, and the way forward. *Neurophotonics.* 2016;3(3):031405.
20. Damoiseaux JS, Rombouts SARB, Barkhof F, Scheltens P, Stam CJ, Smith SM, et al. Consistent resting-state networks across healthy subjects. *Proc Natl Acad Sci U S A.* 2006;103(37):13848–53.

21. Lotte F, Bougrain L, Cichocki A, Clerc M, Congedo M, Rakotomamonjy A, et al. A review of classification algorithms for EEG-based brain-computer interfaces: a 10 year update. *J Neural Eng.* 2018;15(3):031005. 10.1088/1741-2552/aab2f2.
22. Strangman G, Culver JP, Thompson JH, Boas DA. A quantitative comparison of simultaneous BOLD fMRI and NIRS recordings during functional brain activation. *NeuroImage.* 2002;17(2):719–31.
23. Plichta MM, Herrmann MJ, Baehne CG, Ehlis AC, Richter MM, Pauli P, et al. Event-related functional near-infrared spectroscopy (fNIRS): are the measurements reliable? *NeuroImage.* 2006;31(1):116–24. 10.1016/j.neuroimage.2005.12.008.
24. Fazli S, Mehnert J, Steinbrink J, Curio G, Villringer A, Muller KR, et al. Enhanced performance by a hybrid NIRS-EEG brain computer interface. *NeuroImage.* 2012;59(1):519–29.
25. Li S, Francisco GE, Rymer WZ. A new definition of poststroke spasticity and the interference of spasticity with motor recovery from acute to chronic stages. *Neurorehabil Neural Repair.* 2021;35(7):601–10.
26. Yücel MA, Lüthmann AV, Scholkmann F, Gervain J, Dan I, Ayaz H, et al. Best practices for fNIRS publications. *Neurophotonics.* 2021;8(1):012101.
27. von Lüthmann A, Ortega-Martinez A, Boas DA, Yücel MA. Using the general linear model to improve performance in fNIRS single trial analysis and classification: a perspective. *Front Hum Neurosci.* 2020;14:30. 10.3389/fnhum.2020.00030.
28. Ang KK, Chin ZY, Zhang H, Guan C. Filter bank common spatial pattern (FBCSP) in brain-computer interface. In: *Proceedings of the 2008 IEEE International Joint Conference on Neural Networks; 2008 Jun 1–6; Hong Kong, China.* Piscataway (NJ): IEEE; 2008. pp. 2390–2397.
29. Ang KK, Chin ZY, Wang C, Guan C, Zhang H. Filter bank common spatial pattern algorithm on BCI Competition IV datasets 2a and 2b. *Front Neurosci.* 2012;6:39.
30. Takeda K, Gomi Y, Imai I, Shimoda N, Hiwatari M, Kato H. Shift of motor activation areas during recovery from hemiparesis after cerebral infarction: a longitudinal study with near-infrared spectroscopy. *Neurosci Res.* 2007;59(2):136–44. 10.1016/j.neures.2007.06.1466.
31. Scholkmann F, Spichtig S, Muehlmann T, Wolf M. How to detect and reduce movement artifacts in near-infrared imaging using moving standard deviation and spline interpolation. *Physiol Meas.* 2010;31(5):649–62.
32. McPherson JG, Chen A, Ellis MD, Yao J, Heckman CJ, Dewald JPA. Progressive recruitment of contralesional cortico-reticulospinal pathways drives motor impairment post-stroke. *J Physiol.* 2018;596(7):1211–25.
33. Jung SH, Kim YK, Kim SE, Paik NJ. Prediction of motor function recovery after subcortical stroke: case series of activation PET and TMS studies. *Ann Rehabil Med.* 2012;36(4):501–11.
34. Cantillo-Negrete J, Carino-Escobar RI, Carrillo-Mora P, Rodriguez-Barragan MA, Hernandez-Arenas C, Quinzaños-Fresnedo J, et al. Brain-computer interface coupled to a robotic hand orthosis for stroke patients' neurorehabilitation: a crossover feasibility study. *Front Hum Neurosci.* 2021;15:656975. 10.3389/fnhum.2021.656975.

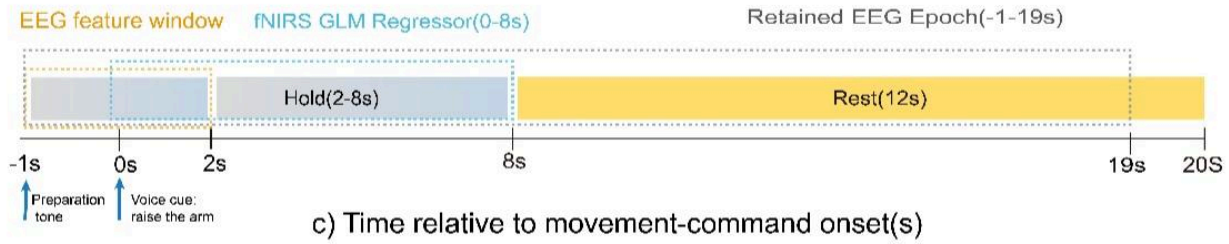
35. Biasiucci A, Leeb R, Iturrate I, Perdakis S, Al-Khodairy A, Corbet T, et al. Brain-actuated functional electrical stimulation elicits lasting arm motor recovery after stroke. *Nat Commun*. 2018;9(1):2421.
36. Pehlivan AU, Losey DP, O'Malley MK. Minimal assist-as-needed controller for upper limb robotic rehabilitation. *IEEE Trans Robot*. 2016;32(1):113–24.
37. Reeve EH, Barnes JN, Moir ME, Walker AE. Impact of arterial stiffness on cerebrovascular function: a review of evidence from humans and preclinical models. *Am J Physiol Heart Circ Physiol*. 2024;326(3):H689–704.
38. Sunil S, Jiang J, Shah S, Kura S, Kilic K, Erdener SE, et al. Neurovascular coupling is preserved in chronic stroke recovery after targeted photothrombosis. *Neuroimage Clin*. 2023;38:103377.
39. Grefkes C, Fink GR. Connectivity-based approaches in stroke and recovery of function. *Lancet Neurol*. 2014;13(2):206–16.
40. Cheung VCK, Turolla A, Agostini M, Silvoni S, Bennis C, Kasi P, et al. Muscle synergy patterns as physiological markers of motor cortical damage. *Proc Natl Acad Sci U S A*. 2012;109(36):14652–6. 10.1073/pnas.1212056109.
41. Zandvoort CS, van Dieën JH, Dominici N, Daffertshofer A. The human sensorimotor cortex fosters muscle synergies through cortico-synergy coherence. *NeuroImage*. 2019;199:30–7.

Figures

A



a) Participant, EEG/fNIRS cap, and movement b) Diagram of EEG-NIRS Co-recording Locations



c) Time relative to movement-command onset(s)

B

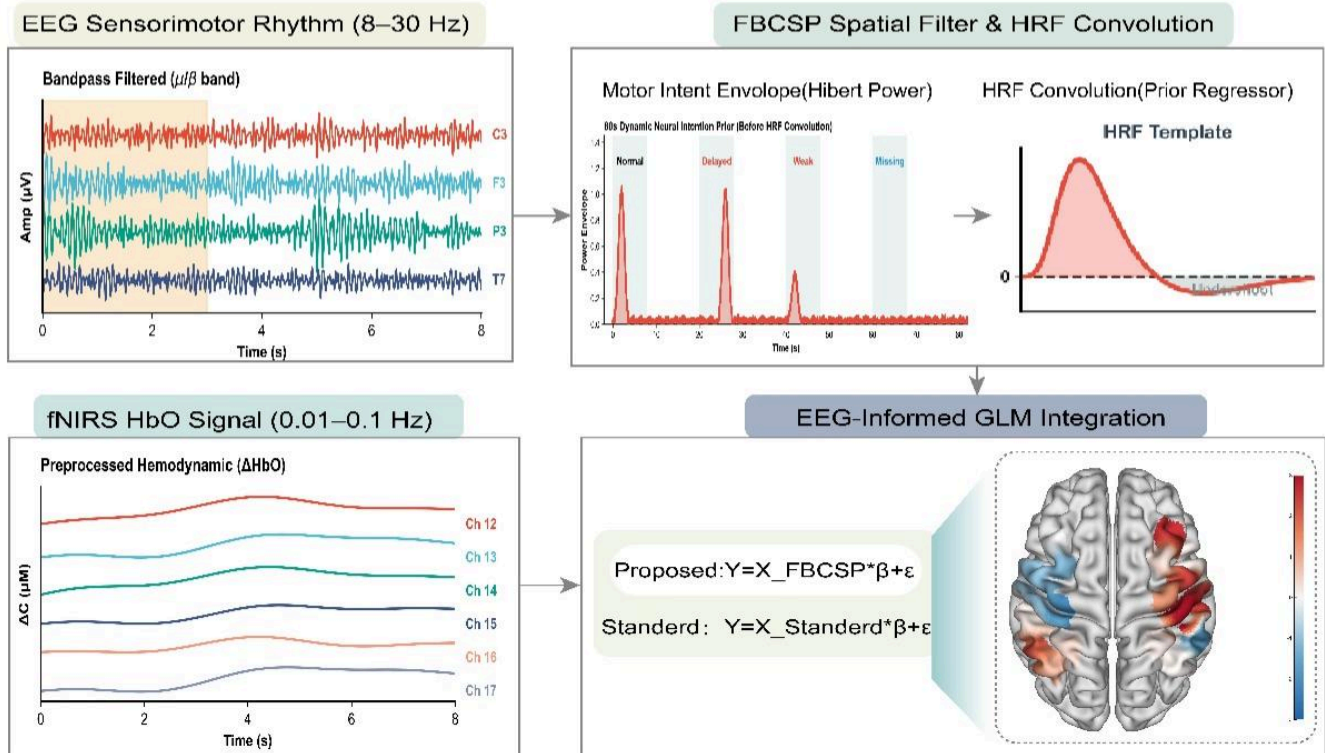


Figure 1

Experimental paradigm and EEG-informed fNIRS analysis framework. **(A)** Multimodal acquisition: (a) simultaneous EEG-fNIRS recording during unilateral arm raising; (b) EEG-fNIRS sensor montage; and (c) trial timing and analysis windows. **(B)** Signal-processing workflow. EEG sensorimotor rhythms (8–30 Hz) were extracted using filter-bank common spatial pattern analysis and convolved with the hemodynamic response function. The resulting motor-intention regressors were integrated with preprocessed

oxygenated hemoglobin signals in a general linear model to estimate cortical activation. Written informed consent for publication of the identifiable photograph was obtained from the participant.

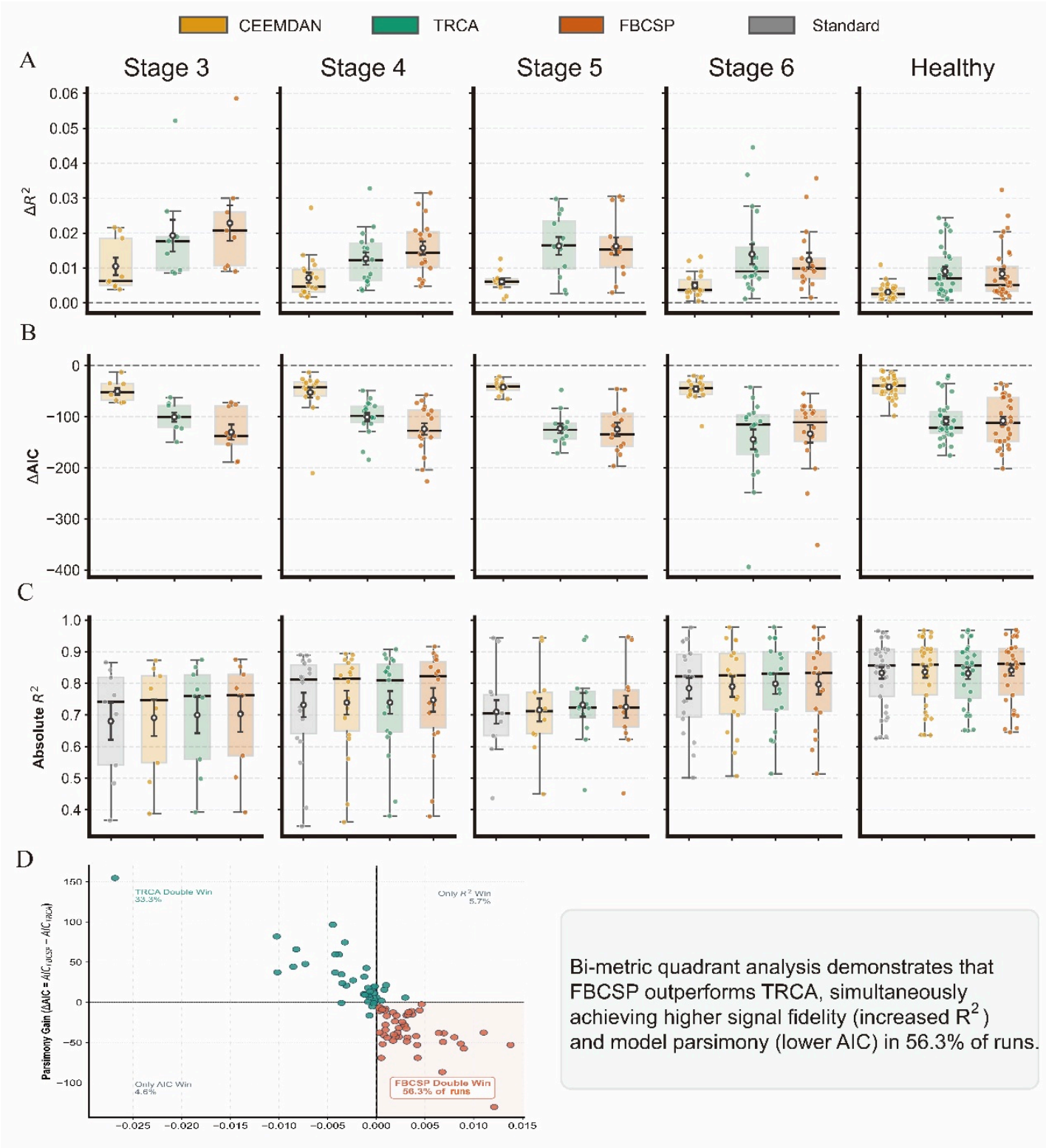


Figure 2

Comparison of model fitting performance across Brunnstrom stages. **(A)** Fit improvement (ΔR^2) and **(B)** parsimony improvement (ΔAIC) relative to the baseline standard GLM. **(C)** Absolute performance

comparison utilizing raw R^2 . **(D)** Bi-metric quadrant analysis comparing FBCSP with TRCA across 87 valid motor-execution runs. Each point represents an individual run. The axes denote the performance gain of FBCSP over TRCA ($\Delta R^2 = R^2_{\text{FBCSP}} - R^2_{\text{TRCA}}$; $\Delta \text{AIC} = \text{AIC}_{\text{FBCSP}} - \text{AIC}_{\text{TRCA}}$). The red quadrant indicates runs where FBCSP achieved both higher goodness-of-fit and improved model parsimony.

Spatial Discriminability Comparison (HbO)

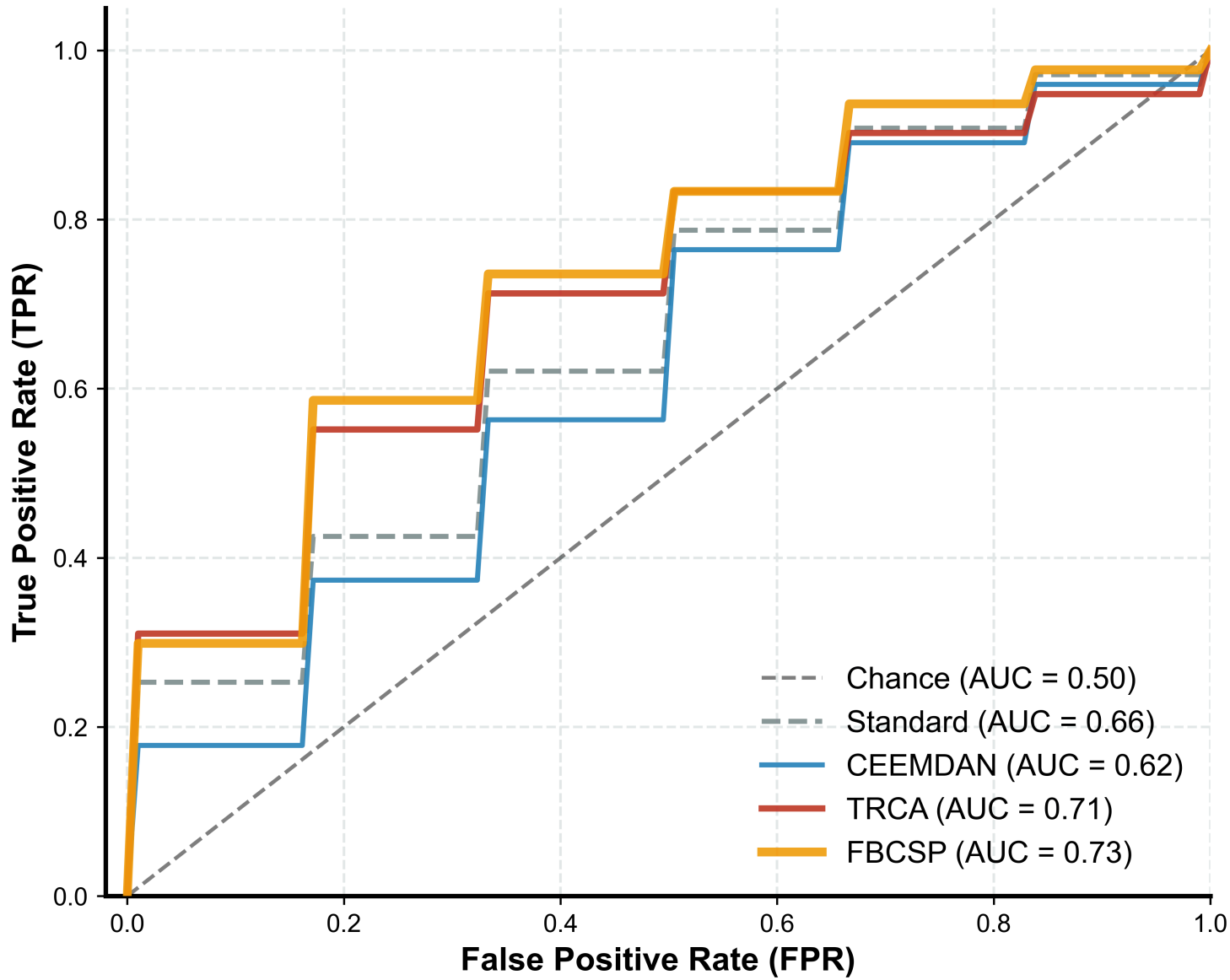


Figure 3

Spatial discriminability of HbO signals. Receiver operating characteristic curves compare the standard GLM, CEEMDAN, TRCA, and FBCSP. The diagonal grey dashed line represents chance-level performance (AUC = 0.50).

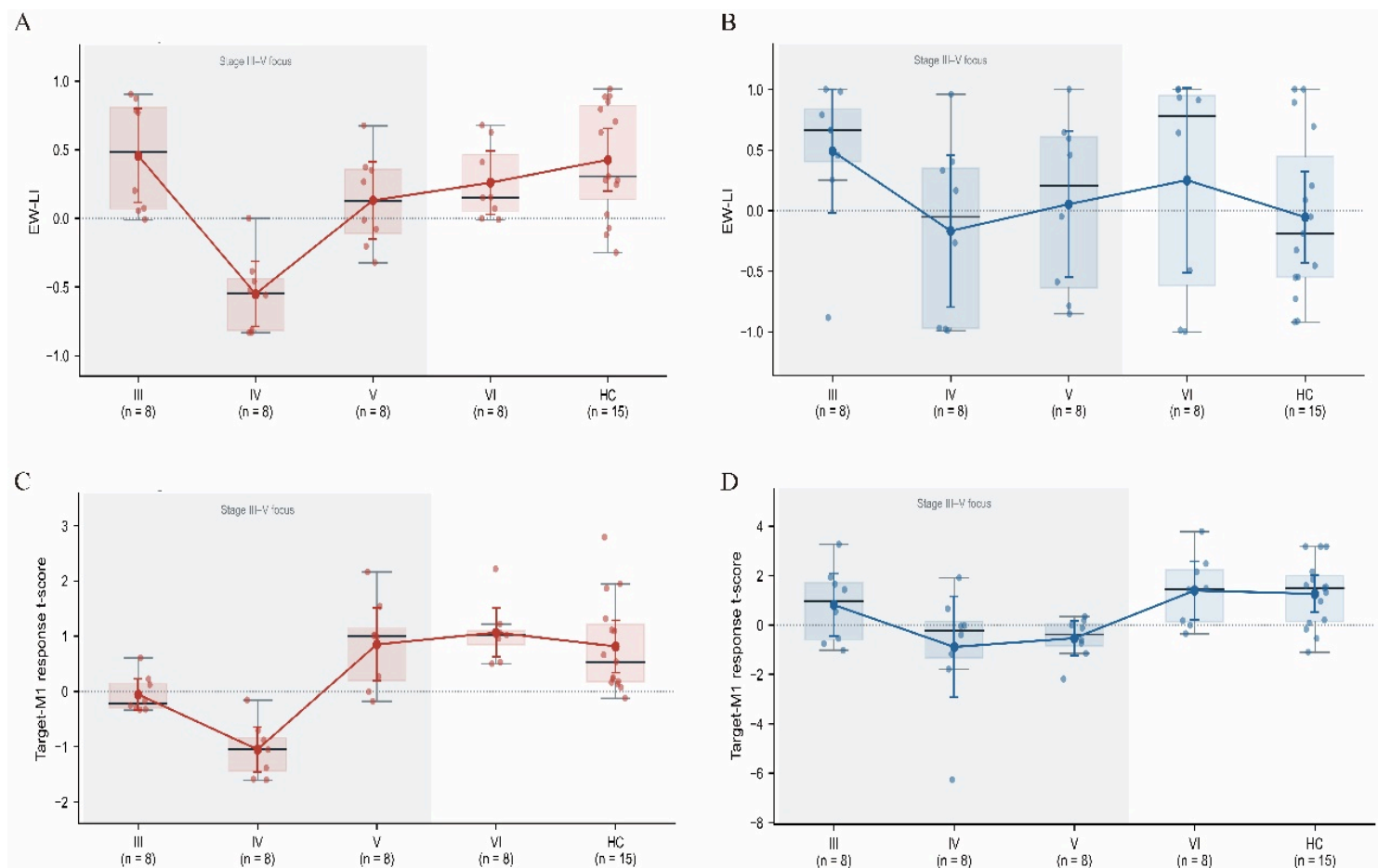


Figure 4

Stage-associated EW-LI and target-M1 response *t*-score patterns. **(A)** EW-LI derived from the FBCSP hemisphere-matched EEG-informed regressor. **(B)** EW-LI derived from the Standard canonical task regressor. **(C)** Target-M1 response *t*-score derived from the FBCSP hemisphere-matched EEG-informed regressor. **(D)** Target-M1 response *t*-score derived from the Standard canonical task regressor. Each point represents one participant; boxes show medians and interquartile ranges, with whiskers extending to observations within $1.5 \times \text{IQR}$. Circles and error bars indicate means and pointwise 95% *t*-based confidence intervals. Lines connect independent group means for visualization and do not represent longitudinal observations. Shading highlights Stages III–V, encompassing the Stage IV-centered decrease-and-rebound pattern; Stage VI and healthy controls are retained for context. Each stroke-stage group contains eight participants, and the healthy-control group contains 15 participants. BH-FDR correction was applied across three adjacent stroke-stage comparisons (III–IV, IV–V, and V–VI) and the separate VI–HC comparison within each metric and model.

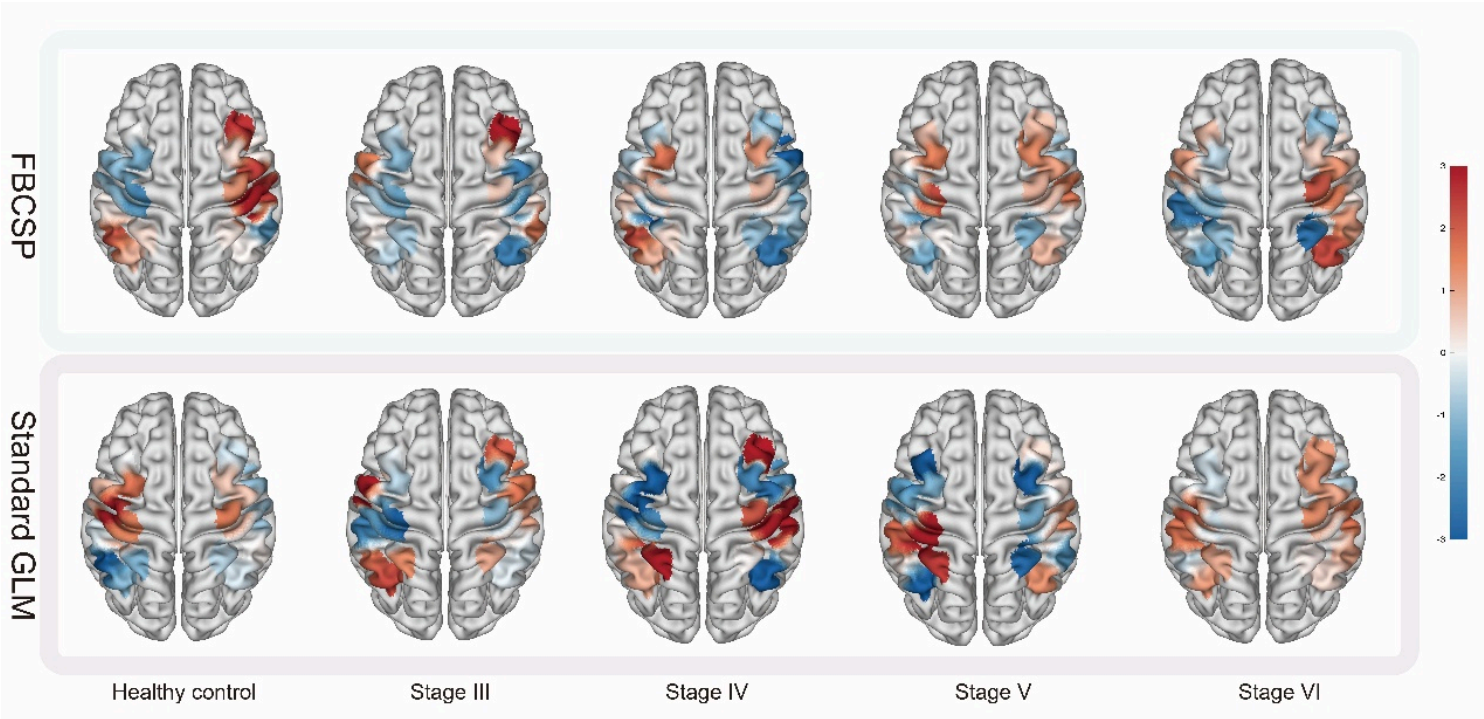


Figure 5

Topographical maps of model-specific channel-wise t -statistics across Brunnstrom groups. To standardize visualization, all anatomical mappings were spatially flipped to represent left-hand execution, rendering the right hemisphere as the target (ipsilesional/contralateral) side. Spatial distributions are shown for the FBCSP hemisphere-matched EEG-informed regressor (top row) and the Standard canonical task regressor (bottom row). Columns show representative observations from the healthy-control group and Brunnstrom groups III–VI. The common color scale ranges from -3 to $+3$.