

E-field modeling of transcranial magnetic stimulation differentiates parietal contributions for grasping after stroke

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ABSTRACT

Background: After a motor stroke, brain networks mediating reaching and grasping undergo functional reorganization, particularly in the parietal cortex. Online repetitive transcranial magnetic stimulation (rTMS) can probe the behavioral relevance of stimulated cortical territories. However, anatomical interpretation is limited because the induced electric field is spatially distributed and may not correspond precisely to the nominal stimulation target.

Objective: To investigate how individual rTMS-induced E-field distributions relate to interference effects of online rTMS over the anterior intraparietal sulcus (IPS) on grasping in stroke patients and healthy controls.

Methods: Eighteen chronic stroke patients and eighteen matched controls performed a reach-grasp-lift task during online rTMS of the IPS. Individual E-fields were modeled and correlated with rTMS-induced changes in 3D kinematic measures. The overlap between E-field maxima and a meta-analytically defined grasping network, as well as cytoarchitectonic parietal regions, was quantified.

Results: In both groups, spatial overlap between the induced E-field and the grasping-related network was linked to rTMS effects on movement smoothness. Changes in grip shaping and smoothness were associated with E-field overlap in the IPS in healthy controls. In patients, the analogous association was observed for the inferior parietal lobule (IPL) rather than the IPS, a pattern compatible with reorganization of parietal specialization after stroke.

Conclusions: E-field-based analyses may improve the interpretation of rTMS effects on grasping in healthy and reorganized neural circuits. This can provide a framework for neuromodulation strategies that focus on individually defined functional targets rather than standard landmarks.

1. Introduction

Grasping requires the dynamic integration of visual, proprioceptive, and somatosensory input as well as motor output within a distributed frontoparietal network [1–3]. Among the regions supporting this process, the anterior portion of the intraparietal sulcus (IPS) plays a central role in transforming visual information about object properties into hand shape during prehension [4–7]. In addition, the primary somatosensory cortex (S1) contributes to grasp stabilization by integrating

real-time feedback [8], while the inferior parietal lobule (IPL) and superior parietal lobule (SPL) contribute to higher-order aspects of sensorimotor control [9,10].

After a stroke, this system undergoes substantial reorganization, altering structure-function relationships. One prominent framework for understanding this process is the vicariation model. As the term, derived from the Latin word *vicarius* (en: “substitute” or “acting in place of”), suggests, this model proposes that intact brain regions may compensate for functions of damaged areas [11,12]. Empirical evidence supports

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this notion, as stroke patients often exhibit increased activity in adjacent areas to the intraparietal cortex and its contralesional homologue when moving their stroke-affected hand [13–15]. Yet, the causal contribution of grasping-related regions, specifically in the parietal cortex, for hand movement performance remain unclear.

Transcranial magnetic stimulation (TMS) has emerged as a valuable tool for non-invasively probing cortical function. When applied during task execution (online-rTMS), it can transiently interfere with neural processing in a targeted brain region, enabling causal inference about the role of this brain region for the behavior of interest [13,16–18]. However, TMS effects are often highly variable, in part due to inter-individual anatomical differences - such as cortical folding and scalp-to-cortex distance - which influence the spatial distribution and strength of the induced E-field [19–21]. These aspects are critical in stroke populations, where variability is further amplified by lesion heterogeneity, structural reorganization, and age-related changes in brain morphology [22–25].

To address these limitations, individualized modeling of TMS-induced E-fields based on high-resolution structural MRI allows the estimation of stimulation strength across cortical tissues [26–28]. By quantifying the effective stimulation volume, E-field modeling may improve the interpretability of TMS experiments and enables more accurate mapping of structure-function relationships, especially when TMS is applied in stroke-affected individuals.

In the present study, we analyzed data from an online-rTMS experiment targeting the aIPS during a reach-grasp-lift task in individuals with chronic stroke and age-matched healthy controls [29]. Using individualized E-field simulations, we tested whether functional task impairment induced by rTMS could be explained by the spatial distribution of the E-field across frontoparietal regions. Kinematic markers of grasping performance were recorded using high-resolution motion tracking and analyzed in relation to both functionally (meta-analytic) and structurally (cytoarchitectonic) defined grasp-related regions. This approach was chosen to move beyond the assumption of nominal stimulation targets, testing whether the functional consequences of rTMS depended on the degree of spatial overlap between the rTMS-induced E-field and the IPS or whether, consistent with the vicariation model, stroke patients show altered sensitivity toward distinct regions.

We formulated two hypotheses. First, in healthy participants, rTMS interference during grasping correlates with the degree of E-field overlap in the IPS, reflecting its well-investigated role in visuomotor integration [1,3,4,7,30]. Second, in stroke patients, this relationship was expected to be altered, such that online-rTMS effects become less specifically linked to IPS but more related to neighboring or distant grasping-related regions due to neural reorganization.

2. Materials and methods

2.1. Participants

We analyzed data from 18 participants with chronic ischemic stroke (mean age $\pm$ SD: 66.2 $\pm$ 13.0 years; 13 male) and 18 healthy, age-matched participants (66.5 $\pm$ 7.2 years; 12 male), who had previously taken part in a TMS study investigating frontoparietal contributions to hand motor control [29]. All stroke patients had a unilateral hand motor deficit due to a single ischemic stroke occurring more than six months earlier. Exclusion criteria were contraindications for TMS or MRI, history of cerebral hemorrhage, bihemispheric infarcts, and severe aphasia, apraxia, or neglect. Patients underwent substantial recovery, indicated by the upper limb subscore of the NIH Stroke Scale (NIHSS), which decreased from a median of 2.5 (IQR: 2.0–3.0) in the acute phase to 1.0 (IQR: 0.0–1.0) in the chronic phase. A score of 2–3 in the acute phase corresponds to moderate to severe arm weakness, i.e., patients were mostly unable to hold the stroke-affected arm against gravity. In contrast, a score of 0–1 indicates the ability to hold the arm against

gravity with no or only mild impairment in the chronic phase. To assess more complex hand motor functions, the Action Research Arm Test (ARAT) was used at study enrollment, indicating mild to moderate residual deficits in the chronic phase (median score: 55.5 out of 57.0; IQR: 45.3–57.0).

The present study focused on rTMS effects over the anterior intraparietal sulcus in the ipsilesional hemisphere. Accordingly, only sessions targeting the anterior IPS contralateral to the moving hand (ipsilesional for stroke patients) were entered in the current analysis. Stimulation targets were in the left hemisphere for 8 patients and in the right hemisphere for 10 patients; healthy controls were correspondingly balanced (9 left, 9 right). All participants had provided written informed consent. The study was approved by the local ethics committee at the University of Cologne (file no: 17-244) and conducted in accordance with the Declaration of Helsinki.

2.2. Online-rTMS

The experiment consisted of two sessions conducted within one week. In the first session, participants underwent structural MRI (T1- and T2-weighted). In the second session, participants performed a visually cued fMRI finger-tapping task to localize individual peak activation in the anterior IPS for neuronavigated rTMS [29]. Further, online-rTMS was applied to test the behavioral response to interfering with a brain region such as the anterior IPS. Therefore, participants performed three motor tasks—finger-tapping, pointing, and reach-grasp-lift—in pseudo-randomized order while receiving 10 Hz online-rTMS (16 pulses per trial) to one of four conditions: ipsilesional anterior IPS, contralesional anterior IPS, contralesional M1, or a sham control site. For sham stimulation, the coil was tilted over the parieto-occipital vertex to avoid effective cortical stimulation while preserving auditory and somatosensory input. For the current study, only trials involving stimulation of the ipsilesional anterior IPS (i.e., contralateral to the moving hand) and sham were analyzed.

rTMS was delivered at 90% of resting motor threshold using a Magstim Super Rapid2 stimulator and a 70 mm Double Air Film Coil. Coil positions were navigated with a frameless computerized stereotaxic system (Brainsight V.2.0.7, Rogue Research, Inc). Stimulation was time-locked to movement onset, cued by an audiovisual signal, and controlled via Presentation® software (Version 9.9, Neurobehavioral Systems, USA). Each task was performed within a fixed time window—1.6 s for finger-tapping and 2.0 s for both pointing and reach-grasp-lift—followed by a 5.5-s inter-trial interval. Stimulation sites were pseudo-randomized every seven trials to counterbalance order effects. Each participant completed 28 trials per task and condition: 28 reach-grasp lift trials during rTMS over anterior IPS and 28 during sham rTMS. Data analysis focused exclusively on the reach-grasp-lift task, which was expected to respond most sensitively to anterior IPS disruption [6, 31–34].

2.3. Behavioral assessment

Motor performance was recorded using a high-resolution motion capture system (Zebris, Isny, Germany). Markers were placed on the dorsum of the index finger and the thumb to capture movements in x (left-right), y (anterior-posterior), and z (vertical) directions. Raw kinematic data were preprocessed using Python 3.8 and the SciPy library (<https://www.scipy.org>). Trajectories were smoothed using a second-order, 12 Hz low-pass Butterworth filter applied in forward and reverse directions (resulting in a fourth-order filter). Grasping movements were assessed while participants grasped a wooden block. 3D kinematic data were segmented based on fingertip velocity: movement onset was defined as exceeding 5% of peak velocity, and offset at the local minimum of velocity at the position of the wooden block. We extracted three kinematic features closely linked to core functions of the anterior IPS [4,9,35]:

- (i) **Number of velocity peaks**, reflecting movement subunits as an indicator for online motor control [29];
- (ii) **Maximum grip aperture**, normalized to finger position during grasping, and
- (iii) **Time to maximum grip aperture**, normalized to the movement cycle, both capturing visuomotor integration essential for object-oriented hand shaping during reach-to-grasp actions.

To evaluate whether online TMS differentially affected patients and controls, we performed separate one-way ANOVAs on each kinematic parameter. Of note, maximum grip aperture and time to maximum grip aperture have not been examined previously in this dataset, and none of the extracted parameters have been reported in relation to E-field modeling.

2.4. Electric field (E-field) estimation

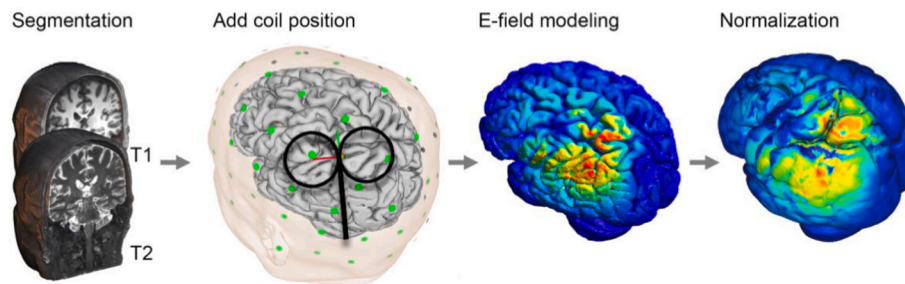
E-field simulations were performed for each participant using SimNIBS v4.0.0 (www.simnibs.org) [27]. To generate realistic head models, we applied the CHARM segmentation pipeline [36] which uses high-resolution T1- and T2-weighted MRI scans to create 15-tissue head-meshes optimized for E-field modeling. To account for the altered conductive properties of lesioned tissue in patients, each individual stroke lesion was delineated on the T2-weighted image using

MRICron [37] and incorporated into the corresponding head model as an additional tissue compartment with CSF-like conductivity via the `add_tissues_to_upsampled` function in SimNIBS, ensuring that enlarged CSF spaces and cystic transformations were represented with appropriate conductive properties during E-field estimation (simulated with the conductivity of 1.654 S/m; [38]). All segmentations were manually reviewed. All lesion masks are presented in [Supplementary Fig. S3](#).

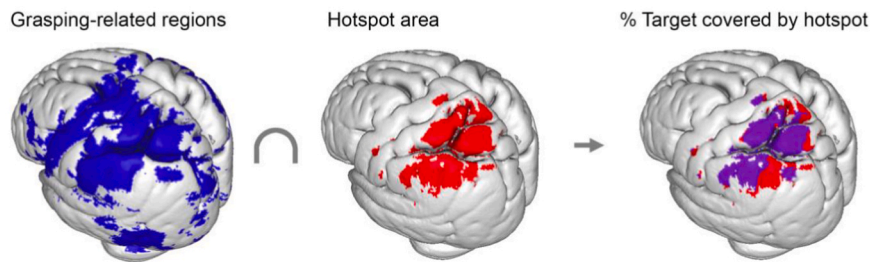
The TMS coil position and orientation were defined using individual neuronavigation data from the experimental session. Specifically, we used the recordedBrainsight coil log files to reconstruct the exact stimulation coordinates and coil orientation relative to anatomical landmarks (coil direction: CPz). Stimulation intensity was set at 90% of the resting motor threshold (rMT), consistent with the parameters used during the rTMS session. This yielded each participant's stimulator output, which was further converted to the rate of change of the coil current (dI/dt) using the coil-specific maximum determined for the Magstim D70 coil of $dI/dt_{max} = 114.7 \text{ A}/\mu\text{s}$ [39]. Global peak E-field magnitudes did not differ between healthy controls (mean $78.5 \pm 19.6 \text{ V/m}$; median 75.8 V/m) and patients (mean $74.8 \pm 13.2 \text{ V/m}$; median 75.9 V/m ; Welch's t -test: $t = 0.66$, $df = 29.8$, $p = 0.52$, [Fig. S2](#)).

The TMS hotspot was defined as the cluster of voxels within the cortical surface that received the strongest E-field exposure, determined through a stepwise percentile approach [40,41]. E-field percentiles were

Step 1: Estimating the electric field (E-field)



Step 2: Estimate overlap between E-field (hotspot area) and grasping network



Step 3: Test for subregion-specific effects

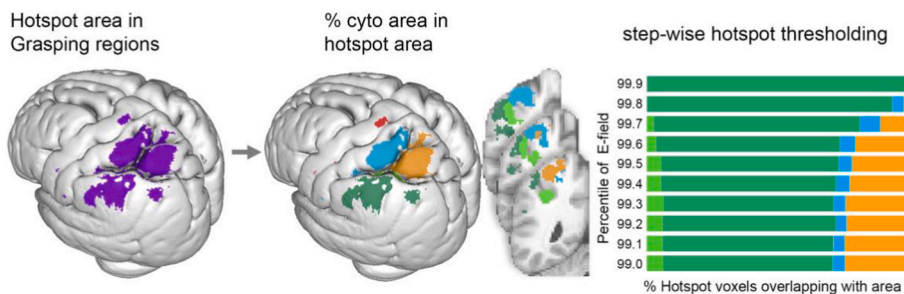


Fig. 1. Illustrates the analysis pipeline starting at (Step 1, top) E-field estimation, (Step 2) testing for overlap with functional networks associated with grasping and (Step 3) testing for region-specific overlap with the E-field.

computed from 99.0 to 99.9 in 0.1 percentile increments, generating a graded set of high-intensity E-field maps (Fig. 1). For each percentile step, we quantified the percentage of the hotspot overlapping with a region of interest (ROI). For each participant, the mean overlap across all percentile steps was used for subsequent analyses to estimate TMS-induced E-field overlap with the ROIs.

2.5. Overlap with regions of interest (ROIs)

Two approaches were used to quantify spatial overlap between the E-field hotspot and cortical targets: (i) a functional network-analysis, and (ii) a structure-based cytoarchitectonic analysis.

In the *function-based analysis*, the hotspot was tested for overlap with a grasping-related network derived from a Neurosynth meta-analysis of studies associated with the term “grasping” (<https://neurosynth.org/>) [42]. The resulting uniformity map (downloaded 11/2022, illustrated in Fig. 1, step 2) shows activation which has been most frequently reported in studies on grasping. This network included bilateral frontoparietal regions known to support visually guided hand movements [43,44].

The *structure-based analysis* aimed to increase anatomical interpretability by testing for overlap between the E-field hotspot and probabilistic cytoarchitectonic maps from the Julich Brain Atlas [45] within the grasping network derived from Neurosynth (Fig. 1, step 3). In the original study, coil positions were defined using fMRI to localize activation maxima in the anterior intraparietal sulcus [29]. Therefore, we tested for overlap of the E-field hotspot with all regions closest to the original target, namely the intraparietal sulcus (IPS) [33], primary sensorimotor area (S1) [46–48], the inferior parietal lobule (IPL) [49], and the superior parietal lobule (SPL) [50,51]. The accuracy of the subject-to-MNI mapping was visually quality-checked for each participant by inspecting the MNI-normalized outputs/overlays (major anatomical landmarks and perilesional cortex in patients) to rule out gross transformation errors. Heatmaps of normalized hotspot maps are depicted in the supplementary material (Fig. S1).

In addition to these population-derived maps, we performed a complementary analysis in which the E-field hotspot was tested for overlap with each participant's individual task-fMRI activation map, acquired to guide neuronavigation [29]. Details and results of this analysis are provided in the supplementary material.

2.6. Relating E-field to behavior

To assess how individual differences in E-field targeting predicted behavioral rTMS effects, two complementary linear modeling approaches were implemented for the function- and structure-based analyses.

For the function-based model, we examined whether the degree of overlap between the E-field hotspot and a functionally defined grasping network was associated with motor deterioration during online-rTMS compared with sham rTMS. A linear regression model was fitted with rTMS-induced change in motor performance as the dependent variable and functional overlap, Group (stroke vs. control), and their interaction as predictors.

$$\text{TMS}_{\text{performance}} = \beta_0 + \beta_1 \cdot \text{Group} + \beta_2 \cdot \text{Overlap} + \beta_3 \cdot (\text{Group} \times \text{Overlap}) + \epsilon$$

TMS_{performance}: TMS-induced change in motor performance.

Group: healthy control or stroke.

Overlap: percentage overlap between the E-field hotspot and the grasping network.

Group × Overlap: interaction term testing whether the effect of overlap differs by group.

ε: error term.

For the structure-based model, we analyzed how E-field magnitude in cytoarchitectonic regions within the grasping network related to

motor impairment. A multiple linear regression model was used entering the mean E-field intensity in each region as predictors, along with Group and their interaction terms. This model tested whether stimulation of specific anatomical subregions explained greater behavioral variance and whether these effects differed between patients and controls.

All models were implemented in Python using the statsmodels package. Interaction terms were explicitly modeled to test for group-dependent effects. For post-hoc correlations, false discovery rate (FDR) correction was applied to control for multiple comparisons across ROIs.

$$\text{TMS}_{\text{performance}} = \beta_0 + \beta_1 \cdot \text{Group} + \beta_2 \cdot \text{Region}_1 + \dots + \beta_n \cdot \text{Region}_n + \beta_{n+1} \cdot (\text{Group} \times \text{Region}_1) + \dots + \beta_{n+m} \cdot (\text{Group} \times \text{Overlap}_n) + \epsilon$$

TMS_{performance}: TMS-induced change in motor performance.

Group: healthy control or stroke.

Overlap_{R1} ... Overlap_{Rn}: percentage overlap between the E-field hotspot and each anatomical region (IPS, IPL, SPL, SI).

Group × Overlap_x: interaction effects between each regions E-field exposure and group

ε: error term.

We computed both models for three dependent variables reflecting different aspects of motor performance changes induced by online-rTMS, namely the number of velocity peaks, maximum grip aperture and time to maximum grip aperture.

2.7. Data availability

The functions used to model E-fields based on anatomical MRI and coil positions are publicly available in the SimNIBS toolbox (www.simnibs.org). The kinematic data from real and control TMS conditions, along with each participant's and demographic and clinical scores are available at https://github.com/LukasHensel/chronic_stroke_fc_tms.

3. Results

3.1. Grasping behavior

We assessed grasping behavior using three kinematic features: the number of velocity peaks, the maximum grip aperture, and the time to maximum grip aperture.

The number of velocity peaks differed significantly between groups. Healthy participants showed fewer velocity peaks (mean = 1.741, SD = 0.256) than stroke patients (mean = 2.289, SD = 0.668), indicating smoother reach trajectories ($t(34) = -3.25$, $p = 0.004$). Moreover, this feature was highly correlated with patients' upper-limb function, as measured by the ARAT score (Pearson $r = -0.82$, $p < 0.001$). No significant group differences were observed for maximum grip aperture (Healthy: 1.220 ± 0.089 , Patient: 1.191 ± 0.133 , $p = 0.457$) or time to maximum grip aperture (Healthy: 0.694 ± 0.059 , Patient: 0.688 ± 0.073 , $p = 0.809$). Likewise, these features did not correlate with ARAT scores ($p > 0.6$).

These observations align with prior findings that movement smoothness is a sensitive marker of post-stroke motor deficits and recovery, detecting deficits even in relatively well recovered patients as in the present cohort [52,53].

3.2. Online-rTMS effects on grasping

To assess whether TMS effects differed between patients and healthy controls, we conducted separate one-way ANOVAs for each kinematic parameter. No significant group differences were found for the number of velocity peaks ($F(1, 34) = 0.97$, $p = 0.331$, $\eta^2 = 0.028$), maximum grip aperture ($F(1, 34) = 3.19$, $p = 0.083$, $\eta^2 = 0.086$), or time to maximum grip aperture ($F(1, 34) = 2.16$, $p = 0.150$, $\eta^2 = 0.060$) (Fig. 2).

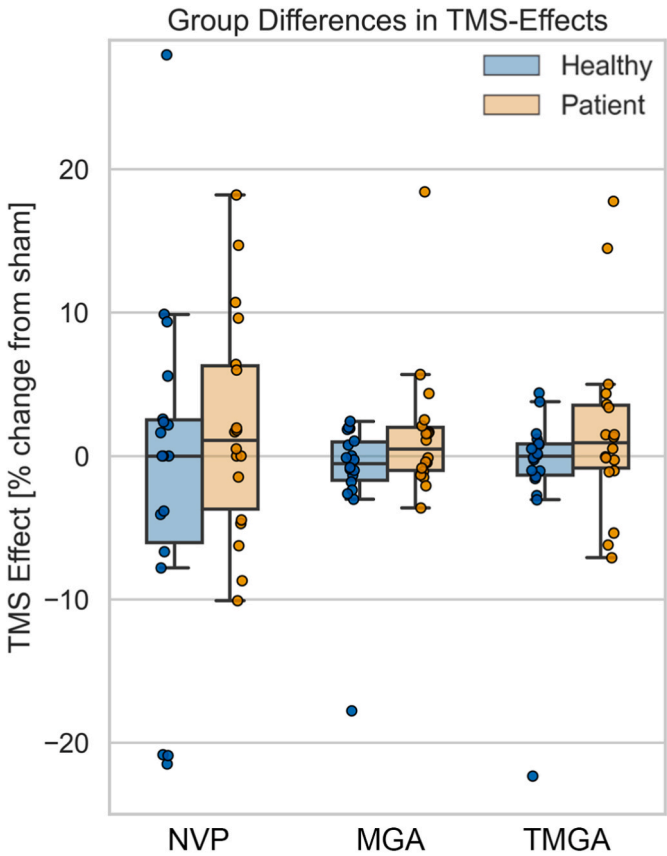


Fig. 2. Boxplots showing percentage performance change from sham during online-rTMS for the number of velocity peaks (NVP), the maximum grip aperture (MGA) and the time to maximum aperture (TMGA). One-way ANOVAs did not detect significant group differences between rTMS effects.

3.3. Hotspot-related TMS-effects

3.3.1. Modulation of functional networks

To assess whether behavioral alterations from online-rTMS were driven by stimulation of a task-relevant functional network, we examined the relationship between E-field overlap and a grasping-related meta-analytic network and grasping performance during rTMS.

The linear model predicting TMS effects on the number of velocity peaks was statistically significant ($R^2 = 0.341$, $p = 0.004$), indicating that greater overlap was associated with more velocity peaks, i.e., less smooth movements ($\beta = 0.80$, $p = 0.003$). No main effect of group ($p = 0.269$) nor an interaction ($p = 0.445$) was found, indicating that the effect of network overlap was consistent across stroke patients and healthy controls. Correspondingly, post-hoc Pearson correlations showed significant associations between E-field exposure in the grasping network and the number of velocity peaks in both patients ($r = 0.59$, $p = 0.010$) and healthy controls ($r = 0.56$, $p = 0.016$; Fig. 3).

In contrast, the corresponding models for maximum grip aperture ($p = 0.142$, $R^2 = 0.154$) and time to maximum grip aperture ($p = 0.052$, $R^2 = 0.211$) did not reach statistical significance.

Taken together, these results suggest that the degree of overlap between the induced E-field and the grasping network was related to induced changes in movement smoothness (number of velocity peaks), but not to grip-shaping parameters (maximum grip aperture, time to maximum grip aperture). This finding was consistent in both healthy individuals and stroke patients, suggesting that smooth execution of grasping relies on the integrated activity of a distributed frontoparietal network, even after cortical reorganization.

Complementing the population-based analyses, overlap between the

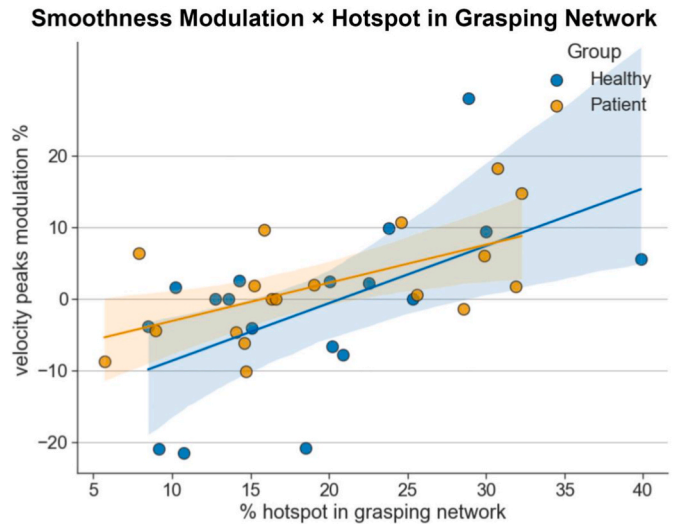


Fig. 3. Correlation of rTMS-induced modulation on the number of velocity peaks and overlap between the E-field hotspot overlap and the grasping network. Patients: Pearson $r = 0.59$ $p = 0.010$; Healthy: $r = 0.56$ $p = 0.016$.

E-field hotspot and each participant's individual task-fMRI activation was likewise associated with the rTMS-induced change in movement smoothness (number of velocity peaks; $R^2 = 0.33$, $p = 0.005$), without a group difference (overlap $\times$ group: $p = 0.22$), whereas no association was found for maximum grip aperture or time to maximum grip aperture (Supplementary Fig. S5).

3.3.2. Modulation of anatomical regions

To examine whether rTMS effects were linked to stimulation of specific cytoarchitectonic regions, multiple linear regressions were computed separately for each kinematic parameter. Each model included the E-field hotspot overlap and cytoarchitectonically defined regions of interest (IPS, IPL, SPL, and S1), as well as group and interaction terms. For all three kinematic parameters, the overall regression models showed a significant fit (FDR-corrected model p -values ≤ 0.048 , Table 1), indicating that E-field exposure in parietal regions jointly explained a significant proportion of variance in TMS-induced grasping changes. Examination of individual regression coefficients revealed that the E-field overlap with IPS was the only regional predictor that showed consistent, significant associations with rTMS effects in healthy participants. The E-field overlap with the IPS was associated with the TMS-induced decline of smoothness (increased number of velocity peaks, $\beta = +58.91$, $p < 0.001$), which was specific for healthy participants (Group $\times$ IPS: $\beta = -57.61$, $p = 0.005$). Similarly, greater IPS overlap significantly reduced maximum grip aperture in healthy participants (β

Table 1
E-field related TMS effects on distinct kinematic features

	NVP	MGA	TMGA
R^2	0.575	0.508	0.442
Adjusted R^2	0.428	0.338	0.248
F-statistic	3.915	2.984	2.284
p-value _{FDR} (model)	0.009 ***	0.021 *	0.048 *
Degrees of Freedom	9.26	9.26	9.26
AIC	257.0	208.1	229.7
BIC	272.8	224.0	245.5

Model summary for multiple linear regressions relating TMS-induced alterations of grasping from regional E-field exposures. Columns show results of models computed for the number of velocity peaks (NVP), maximum grip aperture (MGA) and time to maximum aperture (TMGA). All models identified E-field hotspot overlap in the IPS as the most relevant predictor in healthy participants. A detailed overview on post-hoc correlations is shown in Table 2. p_{FDR} show p -values FDR-corrected for multiple comparisons.

= -25.85, $p = 0.002$), with a trend toward group-specificity (Group $\times$ IPS: $\beta = +19.04$, $p = 0.054$). Finally, greater IPS overlap delayed the time to maximum grip aperture in healthy controls ($\beta = -28.45$, $p = 0.009$), but not in stroke patients (Group $\times$ IPS: $\beta = +38.86$, $p = 0.005$). This reflected an overlap-dependent modulation in healthy individuals, such that lower IPS overlap was associated with preserved or improved performance, whereas higher overlap was linked to performance deterioration.

In contrast, stroke patients showed no significant associations with IPS overlap. Instead, E-field overlap with IPL was related to changes in movement smoothness, as indicated by a significant Group $\times$ IPL interaction ($\beta = +3.34$, $p = 0.049$). This suggests that, after stroke, structure–function relationships may differ, such that grasping smoothness becomes more strongly associated with IPL rather than IPS involvement. Detailed post-hoc correlations are shown in Table 2.

Taken together, these results indicate that in the healthy brain, grasping kinematics are specifically modulated by the extent of IPS involvement, as evidenced by E-field-dependent TMS effects. After a stroke, this relationship is altered, with grasping smoothness showing stronger associations with IPL than with IPS. These findings suggest a reorganization of structure–function relationships following stroke.

4. Discussion

This study demonstrates that the effects of online-rTMS on hand motor function were linked to the spatial distribution of the induced E-field relative to frontoparietal cortical regions and that these effects differed between healthy individuals and individuals with reorganized networks due to chronic stroke [29]. In healthy controls, stimulation-induced behavioral changes were specifically associated with the E-field overlap in the IPS, whereas in patients an analogous association was observed for the IPL rather than the IPS. This provides proof of concept that individualized E-field modeling can improve the interpretation of online-rTMS effects and may more sensitively reveal

cortical reorganization after stroke.

4.1. Specificity of IPS in healthy adults

For smoothness, indicated by the number of velocity peaks, and grip shaping, indicated by the maximum grip aperture and the time to maximum grip aperture, we observed significant associations between the magnitude of rTMS-induced motor interference and the degree of E-field overlap with cytoarchitecturally defined IPS in healthy participants (Fig. 4). These findings are consistent with the well-investigated role of the IPS as a critical node processing reaching and grasping in the healthy brain. During reaching and grasping, as performed in the present study, the IPS may contribute by distinct subregions, especially the anterior IPS, transforming visual object properties into motor commands for grasp shaping [4,5] and the mid-IPS contributing to trajectory planning [6,31,54].

Combining neuronavigated rTMS, E-field modeling and 3D kinematics, this study extends prior work by providing evidence that interfering with parietal cortex affects distinct aspects of hand motor control during reaching and grasping, and that the magnitude of this interference tracks the estimated E-field overlap with the IPS. E-field modeling improved the estimation of the likely cortical engagement [27,36], explaining substantial inter-individual variance in motor outcomes (Table 1) and underscoring the relevance of accounting for anatomical variability when defining neuromodulation targets. The present results further highlight that interference with IPS specifically modulates grip shaping, indicated by the (time to) maximum grip aperture. In contrast, the online control of the reaching trajectory, indicated by the number of velocity peaks, was not only associated with regional interference in the IPS but also a wider distributed grasping network (Fig. 3).

Collectively, these findings suggest that the quality of hand-shaping during grasping may require precision of rTMS in parietal subregions, whereas smooth execution of reach-to-grasp movements may depend more on interference with the frontoparietal grasping network.

4.2. Grasping smoothness and hand shape

Among the kinematic features, the number of velocity peaks (indicating reaching smoothness and online motor control) emerged as the most sensitive behavioral readout, distinguishing patients from controls and correlating strongly with upper-limb function as measured by ARAT. This finding is consistent with number of velocity peaks indexing movement smoothness as a clinically established marker of upper-limb impairment after stroke [55]. As a behavioural measure, however, it is not specific to parietal grasp processing. For example, reduced smoothness may equally reflect compensatory strategies, proximal arm control, or residual impairment [56,57]. In our data, calculating the rTMS-induced change in motor performance (real vs. sham) aimed to isolate the contribution of the transiently perturbed network from potential confounds. Consistent with this, rTMS-induced change of smoothness was associated with E-field exposure across an extended frontoparietal network, reflecting the sensorimotor integration required for real-time control of reach-to-grasp actions [35,52,53].

In contrast, the grasp-specific hand-shaping measures (maximum grip aperture and time to maximum grip aperture) neither differed between groups nor related to clinical impairment. In healthy participants, these markers demonstrated that E-field models could reveal region-specific rTMS effects in the IPS, consistent with the role of the IPS in grip shaping [32,58].

Taken together, the convergence of rTMS responsiveness and clinical relevance points at smoothness as a reliable behavioural marker for future E-field-informed rTMS studies on reach-to-grasp movements.

4.3. Cortical reorganization after stroke

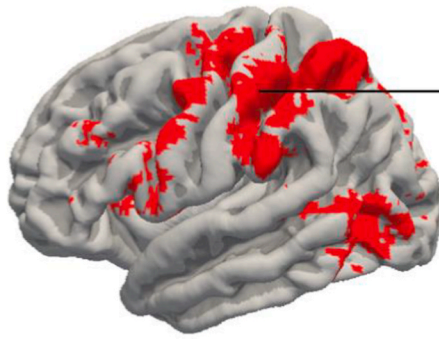
In contrast to the region-specific effects observed in healthy

Table 2
Correlations between TMS effects and regional E-field exposure

Feature	Region	Group	R	p	pFDR	
Velocity Peaks (NVP)	IPS	Healthy	0.75	0.0003	0.0036	**
	IPS	Patient	0.43	0.0763	0.2921	
	IPL	Healthy	0.36	0.1434	0.3824	
	IPL	Patient	0.62	0.0056	0.0444	*
	SPL	Healthy	-0.01	0.9634	0.9838	
	SPL	Patient	-0.03	0.9182	0.9838	
Max. Grip Aperture (MGA)	S1	Healthy	0.40	0.1052	0.3156	
	S1	Patient	0.42	0.0852	0.2921	
	IPS	Healthy	-0.79	0.0001	0.0024	**
	IPS	Patient	-0.11	0.6648	0.9385	
	IPL	Healthy	-0.34	0.1657	0.3901	
	IPL	Patient	0.23	0.3563	0.588	
Time to Grip Aperture (TMGA)	SPL	Healthy	-0.04	0.8624	0.9838	
	SPL	Patient	-0.43	0.0736	0.2921	
	S1	Healthy	-0.14	0.5762	0.8643	
	S1	Patient	0.00	0.9838	0.9838	
	IPS	Healthy	-0.61	0.0074	0.0444	*
	IPS	Patient	-0.07	0.7741	0.9838	
	IPL	Healthy	-0.23	0.3675	0.588	
	IPL	Patient	-0.32	0.198	0.396	
	SPL	Healthy	0.07	0.7909	0.9838	
	SPL	Patient	-0.26	0.3048	0.5627	
	S1	Healthy	-0.03	0.8995	0.9838	
	S1	Patient	-0.33	0.1788	0.3901	

Post-hoc correlations between TMS effects on kinematic features (NVP: number of velocity peaks; MGA: maximum grip aperture; TMGA: time to maximum aperture) and E-field exposure in different parietal regions. p_{FDR} shows all results FDR-corrected for multiple comparisons. IPS: intraparietal sulcus; IPL: intraparietal lobule; SPL: superior parietal lobule; S1: primary somatosensory cortex.

Functional: Grasping Network



Structural: Parietal Cyto Areas

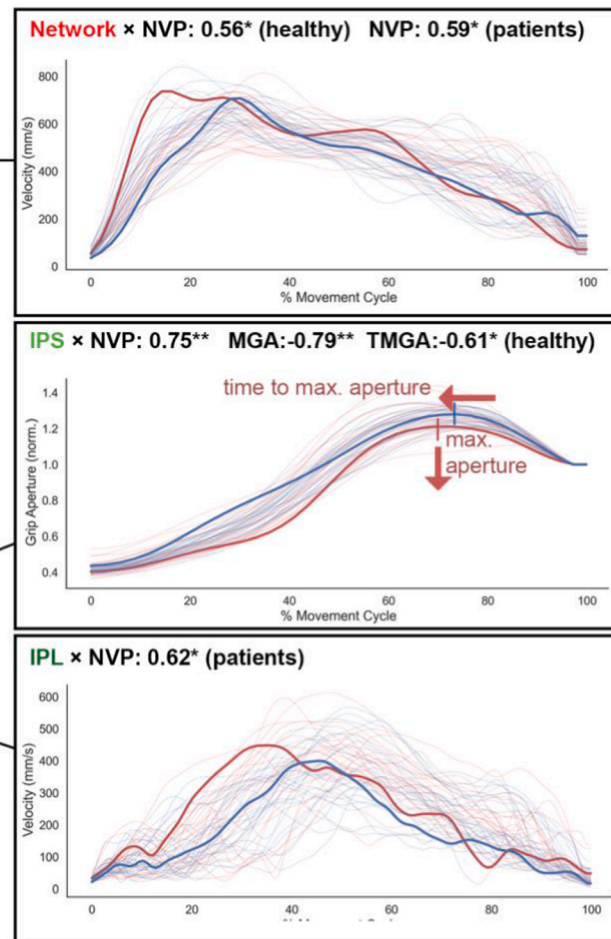
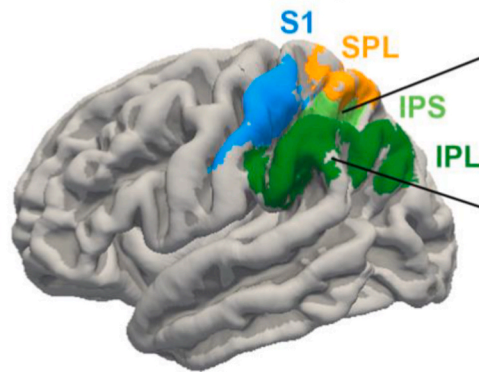


Fig. 4. Summary of associations between the E-field hotspot overlap with functional (top) and structural (bottom) ROIs. For each region linked to rTMS effects, right panels show correlation coefficients for kinematic features: number of velocity peaks (NVP), maximum grip aperture (MGA), and time to maximum grip aperture (TMGA). Single-subject reach-to-grasp kinematic data are plotted as examples for healthy participants (top, middle) and a participant with stroke (bottom, red: real, blue: sham rTMS, median performance plotted as bold line). * $p_{\text{FDR}} < 0.05$, ** $p_{\text{FDR}} < 0.01$, see Table 2 for correlations.

participants, stroke patients did not show significant modulation of motor performance linked to E-field overlap with IPS, but with IPL, a region supporting sensorimotor integration and attentional control [49, 59, 60]. Because lesions in our sample spared the IPS (Fig. S3), this absence of specificity is unlikely to reflect direct damage to the parietal cortex. Instead, both observations, the absence of IPS specificity and the observation of an IPL-linked effect may reflect two facets of an underlying mechanism, namely the disruption of the frontoparietal network into which the intact IPS is embedded.

This pattern links two classical concepts of post-stroke reorganization. The absence of functional specialization in IPS may correspond to *diaschisis*, in which a lesion produces abnormal function in remote but intact regions through disrupted connectivity rather than direct injury [61, 62]. Subcortical lesions such as those in our cohort, with maximal overlap in the internal capsule [29] damage the corticospinal tract, indirectly resulting in an upstream dysfunction of the sensorimotor network [63]. Beyond such outflow effects, subcortical lesions can also disrupt long cortico-cortical association fibres, as shown for frontoparietal pathways in the attentional domain [64, 65]. Regarding motor recovery, increasing evidence suggests that such connections with parietal regions play a critical role for motor recovery [66, 67].

Whereas diaschisis may account for the reduced specificity of the intact IPS, the IPL-linked effect may reflect *vicariation*, a complementary process within the same network, whereby spared regions partially assume the roles of disconnected ones [12, 68, 69]. Experimental work supports the capacity for remote corticocortical reorganization after

focal motor injury. In non-human primates, lesions of the M1 hand area induced axonal sprouting in ventral premotor cortex and the formation of novel connections with somatosensory cortex, demonstrating that structurally intact regions remote from the lesion may undergo substantial anatomical reorganization [69]. Although these findings provide a mechanistic precedent for vicariation, they do not demonstrate a specific transfer of function from IPS to IPL. In humans, the increase of parietofrontal and interhemispheric connectivity after subcortical stroke has been linked to successful motor recovery [15, 70]. The present data are compatible with this hypothesis, showing rTMS-effects on grasping smoothness for a distributed grasping network in patients (Fig. 4, top panel), indicating that grasp control remains processed by spared frontoparietal resources even as its subregional architecture in the parietal cortex appears to be reorganized.

Of note, the interpretation of the present findings is limited by the sample size of 18 patients with heterogeneous lesions, which is underpowered to probe which lesion locations preferentially induce such reorganization. Addressing this question will require lesion- and disconnection-network mapping in larger samples [67, 71].

4.4. Outlook

Although our data cannot answer the question of why reorganization occurs, it does suggest some direct implications for future rTMS applications when targeting the motor system after a stroke. The present study shows that the behavioural impact of stimulation tracks the

modeled cortical engagement rather than the nominal coil target, and that responses in subregions IPS and IPL can differ after stroke. This suggests that E-field modelling can be used to control for interindividual variability after stroke, allowing a more precise interpretation of results. The present findings thus serve as a proof of principle, generating anatomically specific, testable hypotheses for future work. Because individual E-field estimates can also be used prospectively to optimise target selection [40] the same approach may help tailor personalised neuromodulation studies in stroke populations.

5. Conclusion

This study shows that E-field modeling enhances our understanding of online-rTMS effects on grasping and, thereby, may help characterise how parietal contributions to grasp control differ after stroke. In the chronic phase after stroke, we found an altered regional motor response depending on the rTMS-induced field in the parietal cortex. While interference with the IPS disrupted grasp control in healthy individuals, stroke patients showed this association only for the IPL, consistent with concepts of adaptive cortical reorganization. These findings demonstrate how E-field-informed analysis can improve models to guide more accurate interpretations of cortical contributions to motor behavior after stroke.

CRedit authorship contribution statement

Lukas Hensel: Writing – review & editing, Writing – original draft, Visualization, Project administration, Formal analysis, Data curation, Conceptualization. **Fabian Lange:** Writing – review & editing, Data curation. **Hartwig Roman Siebner:** Writing – review & editing, Methodology. **Axel Thielscher:** Writing – review & editing, Methodology. **Gereon R. Fink:** Writing – review & editing, Resources, Funding acquisition, Conceptualization. **Christian Grefkes:** Writing – review & editing, Supervision, Resources, Funding acquisition, Conceptualization.

Ethics declaration

Written informed consent to take part in the study and to publish the article has been obtained from all participants or their legal representatives. The privacy rights of participants have been observed.

This study was performed in compliance with relevant laws, regulatory frameworks and guidelines where the research took place. This study was conducted in accordance with Declaration of Helsinki. This study was approved by the Local ethics committee at the University of Cologne. (Approval No. file no: 17-244)

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Declaration of competing interests

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Hartwig R. Siebner has received honoraria as speaker and consultant from Lundbeck AS, Denmark, and as editor (NeuroImage: Clinical) from Elsevier Publishers, Amsterdam, The Netherlands. He has received

royalties as book editor from Springer Publishers, Stuttgart, Germany, Oxford University Press, Oxford, UK, and from Gyldendal Publishers, Copenhagen, Denmark. Gereon R. Fink serves as an editorial board member of *NeuroImage: Clinical*, *Zeitschrift für Neuropsychologie*, *Zeitschrift für Kinder- und Jugendpsychiatrie und Psychotherapie*, and *Info Neurologie & Psychiatrie*; receives royalties for the books *Funktionelle MRT in Psychiatrie und Neurologie*, *Neurologische Differentialdiagnose*, *SOP Neurologie*, and *Therapiehandbuch Neurologie*; receives royalties for the neuropsychological tests KAS, NP-KISS, and KöpSS; receives honoraria for speaking engagements from the Deutsche Gesellschaft für Neurologie (DGN) and Forum für medizinische Fortbildung FomF GmbH.

The other authors report no competing interests.

All authors have reviewed and approved the manuscript and have disclosed any potential conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.brs.2026.103191>.

References

- [1] Jeannerod M, Arbib MA, Rizzolatti G, Sakata H. Grasping objects: the cortical mechanisms of visuomotor transformation. *Trends in neurosciences* 1995;18: 314–20.
- [2] Schaffelhofer S, Scherberger H. Object vision to hand action in macaque parietal, premotor, and motor cortices. *eLife* 2016;5:e15278.
- [3] Errante A, Ziccarelli S, Mingolla G, Fogassi L. Grasping and manipulation: neural bases and anatomical circuitry in humans. *Neuroscience* 2021;458:203–12.
- [4] Binkofski F, Dohle C, Posse S, Stephan KM, Hefter H, Seitz RJ, Freund HJ. Human anterior intraparietal area subserves prehension. *Neurology* 1998;50:1253–9.
- [5] Grefkes C, Weiss PH, Zilles K, Fink GR. Crossmodal processing of object features in human anterior intraparietal cortex an fMRI study implies equivalencies between humans and monkeys. *Neuron* 2002;35:173–84.
- [6] Grefkes C, Fink GR. The functional organization of the intraparietal sulcus in humans and monkeys. *J Anat* 2005;207:3–17.
- [7] Tunik E, Frey SH, Grafton ST. Virtual lesions of the anterior intraparietal area disrupt goal-dependent on-line adjustments of grasp. *Nat Neurosci* 2005;8:505–11.
- [8] Gardner EP, Babu KS, Reitzen SD, Ghosh S, Brown AS, Chen J, Hall AL, Herzlinger MD, Kohlenstein JB, Ro JY. Neurophysiology of prehension. I. posterior parietal cortex and object-oriented hand behaviors. *J Neurophysiol* 2007;97: 387–406.
- [9] Tunik E, Ortigue S, Adamovich SV, Grafton ST. Differential recruitment of anterior intraparietal sulcus and superior parietal lobule during visually guided grasping revealed by electrical neuroimaging. *J Neurosci* 2008;28:13615–20.
- [10] Rens G, Figley TD, Gallivan JP, Liu Y, Culham JC. Grasping with a twist: dissociating action goals from motor actions in human frontoparietal circuits. *J Neurosci* 2023;43:5831–47.
- [11] Wiesendanger M. Constantin von Monakow (1853-1930): a pioneer in interdisciplinary brain research and a humanist. 2006.
- [12] Finger S. Recovery of function: redundancy and vicariation theories. *Handb Clin Neurol* 2009;95:833–41.
- [13] Lotze M, Markert J, Sauseng P, Hoppe J, Plewnia C, Gerloff C. The role of multiple contralesional motor areas for complex hand movements after internal capsular lesion. *J Neurosci* 2006;26:6096–102.
- [14] Rehme AK, Fink GR, Cramon DY von, Grefkes C. The role of the contralesional motor cortex for motor recovery in the early days after stroke assessed with longitudinal fMRI. *Cerebr Cortex* 2011;21:756–68.
- [15] Schulz R, Koch P, Zimerman M, Wessel M, Bönstrup M, Thomalla G, Cheng B, Gerloff C, Hummel FC. Parietofrontal motor pathways and their association with motor function after stroke. *Brain* 2015;138:1949–60.
- [16] Volz LJ, Vollmer M, Michely J, Fink GR, Rothwell JC, Grefkes C. Time-dependent functional role of the contralesional motor cortex after stroke. *Neuroimage, Clin* 2017;16:165–74.
- [17] Tscherpel C, Hensel L, Lemberg K, Freytag J, Michely J, Volz LJ, Fink GR, Grefkes C. Age affects the contribution of ipsilateral brain regions to movement kinematics. *Hum Brain Mapp* 2020;41:640–55.
- [18] Hensel L, Tscherpel C, Freytag J, Ritter S, Rehme AK, Volz LJ, Eickhoff SB, Fink GR, Grefkes C. Connectivity-related roles of contralesional brain regions for motor performance early after stroke. *Cerebr Cortex* 2021;31:993–1007.
- [19] Knecht S, Sommer J, Deppe M, Steinsträter O. Scalp position and efficacy of transcranial magnetic stimulation. *Clin Neurophysiol* 2005;116:1988–93.
- [20] Lee EG, Rastogi P, Hadimani RL, Jiles DC, Campodoni JA. Impact of non-brain anatomy and coil orientation on inter- and intra-subject variability in TMS at midline. *Clin Neurophysiol* 2018;129:1873–83.
- [21] Davis NJ. Variance in cortical depth across the brain surface: implications for transcranial stimulation of the brain. *Eur J Neurosci* 2021;53:996–1007.

- [22] Minjoli S, Saturnino GB, Blicher JU, Stagg CJ, Siebner HR, Antunes A, Thielscher A. The impact of large structural brain changes in chronic stroke patients on the electric field caused by transcranial brain stimulation. *Neuroimage, Clin* 2017;15: 106–17.
- [23] Antonenko D, Grittner U, Saturnino G, Nierhaus T, Thielscher A, Flöel A. Inter-individual and age-dependent variability in simulated electric fields induced by conventional transcranial electrical stimulation. *Neuroimage* 2021;224:117413.
- [24] Lu H, Li J, Zhang L, Chan SSM, Lam LCW, Studies for the Oas of I. Dynamic changes of region-specific cortical features and scalp-to-cortex distance: implications for transcranial current stimulation modeling. *J Neuroeng Rehabilitation* 2021;18:2.
- [25] Evans C, Johnstone A, Zich C, Lee JSA, Ward NS, Bestmann S. The impact of brain lesions on tDCS-induced electric fields. *Sci Rep* 2023;13:19430.
- [26] Windhoff M, Opitz A, Thielscher A. Electric field calculations in brain stimulation based on finite elements: an optimized processing pipeline for the generation and usage of accurate individual head models. *Hum Brain Mapp* 2013;34:923–35.
- [27] Thielscher A, Antunes A, Saturnino GB. Field modeling for transcranial magnetic stimulation: a useful tool to understand the physiological effects of TMS? In: All authors contributed equally to the work. 2015 37th annu int conf IEEE eng med biol soc (EMBC), 2015; 2015. p. 222–5.
- [28] Huang Y, Datta A, Bikson M, Parra LC. Realistic volumetric-approach to simulate transcranial electric stimulation—ROAST—a fully automated open-source pipeline. *J Neural Eng* 2019;16:056006.
- [29] Hensel L, Lange F, Tscherpel C, Viswanathan S, Freytag J, Volz LJ, Eickhoff SB, Fink GR, Grefkes C. Recovered grasping performance after stroke depends on interhemispheric frontoparietal connectivity. *Brain* 2023;146:1006–20.
- [30] Grefkes C, Ritzl A, Zilles K, Fink GR. Human medial intraparietal cortex subserves visuomotor coordinate transformation. *Neuroimage* 2004;23:1494–506.
- [31] Culham JC, Valyear KF. Human parietal cortex in action. *Curr Opin Neurobiol* 2006;16:205–12.
- [32] Rice NJ, Tunik E, Cross ES, Grafton ST. On-line grasp control is mediated by the contralateral hemisphere. *Brain Res* 2007;1175:76–84.
- [33] Caspers S, Eickhoff SB, Rick T, Kapri A von, Kühlen T, Huang R, Shah NJ, Zilles K. Probabilistic fibre tract analysis of cytoarchitecturally defined human inferior parietal lobule areas reveals similarities to macaques. *Neuroimage* 2011;58: 362–80.
- [34] Davare M, Kraskov A, Rothwell JC, Lemon RN. Interactions between areas of the cortical grasping network. *Curr Opin Neurobiol* 2011;21:565–70.
- [35] Verhagen L, Dijkerman HC, Medendorp WP, Toni I. Cortical dynamics of sensorimotor integration during grasp planning. *J Neurosci* 2012;32:4508–19.
- [36] Puonti O, Leemput KV, Saturnino GB, Siebner HR, Madsen KH, Thielscher A. Accurate and robust whole-head segmentation from magnetic resonance images for individualized head modeling. *Neuroimage* 2020;219:117044.
- [37] Rorden C. From MRICro to MRICron: the evolution of neuroimaging visualization tools. *Neuropsychologia* 2025;207:109067.
- [38] Wagner TA, Zahn M, Grodzinsky AJ, Pascual-Leone A. Three-dimensional head model simulation of transcranial magnetic stimulation. *IEEE Trans Biomed Eng* 2004;51:1586–98.
- [39] Drakaki M, Mathiesen C, Siebner HR, Madsen K, Thielscher A. Database of 25 validated coil models for electric field simulations for TMS. *Brain Stimul* 2022;15: 697–706.
- [40] Lynch CJ, Elbau IG, Ng TH, Wolk D, Zhu S, Ayaz A, Power JD, Zebley B, Gunning FM, Liston C. Automated optimization of TMS coil placement for personalized functional network engagement. *Neuron* 2022;110:3263–3277.e4.
- [41] Hoornweder SV, Nuyts M, Frieske J, Verstraeten S, Meessen RLJ, Caulfield KA. Outcome measures for electric field modeling in tES and TMS: a systematic review and large-scale modeling study. *Neuroimage* 2023;281:120379.
- [42] Yarkoni T, Poldrack RA, Nichols TE, Essen DCV, Wager TD. Large-scale automated synthesis of human functional neuroimaging data. *Nat Methods* 2011;8:665–70.
- [43] Rozzi S, Calzavara R, Belmalih A, Borra E, Gregoriou GG, Matelli M, Luppino G. Cortical connections of the inferior parietal cortical convexity of the macaque monkey. *Cerebr Cortex* 2006;16:1389–417. New York, NY : 1991.
- [44] Gerbella M, Rozzi S, Rizzolatti G. The extended object-grasping network. *Exp Brain Res* 2017;235:2903–16.
- [45] Amunts K, Mohlberg H, Bludau S, Zilles K. Julich-brain: a 3D probabilistic atlas of the human brain's cytoarchitecture. *Science* 2020;369:988–92.
- [46] Geyer S, Schleicher A, Zilles K. Areas 3a, 3b, and 1 of human primary somatosensory cortex. *Neuroimage* 1999;10:63–83.
- [47] Geyer S, Schormann T, Mohlberg H, Zilles K. Areas 3a, 3b, and 1 of human primary somatosensory cortex. Part 2. Spatial normalization to standard anatomical space. *Neuroimage* 2000;11:684–96.
- [48] Grefkes C, Geyer S, Schormann T, Roland P, Zilles K. Human somatosensory area 2: observer-independent cytoarchitectonic mapping, interindividual variability, and population map. *Neuroimage* 2001;14:617–31.
- [49] Caspers S, Eickhoff SB, Geyer S, Scheperjans F, Mohlberg H, Zilles K, Amunts K. The human inferior parietal lobule in stereotaxic space. *Brain Struct Funct* 2008; 212:481–95.
- [50] Scheperjans F, Hermann K, Eickhoff SB, Amunts K, Schleicher A, Zilles K. Observer-independent cytoarchitectonic mapping of the human superior parietal cortex. *Cerebr Cortex* 2008;18:846–67.
- [51] Scheperjans F, Eickhoff SB, Hömke L, Mohlberg H, Hermann K, Amunts K, Zilles K. Probabilistic maps, morphometry, and variability of cytoarchitectonic areas in the human superior parietal cortex. *Cerebr Cortex* 2008;18:2141–57.
- [52] Rohrer B, Fasoli S, Krebs HI, Hughes R, Volpe B, Frontera WR, Stein J, Hogan N. Movement smoothness changes during stroke recovery. *J Neurosci* 2002;22: 8297–304.
- [53] Balasubramanian S, Melendez-Calderon A, Roby-Brami A, Burdet E. On the analysis of movement smoothness. *J Neuroeng Rehabilitation* 2015;12:112.
- [54] Prado J, Clavagnier S, Otzenberger H, Scheiber C, Kennedy H, Perenin M-T. Two cortical systems for reaching in central and peripheral vision. *Neuron* 2005;48: 849–58.
- [55] Schwarz A, Kanzler CM, Lamercy O, Luft AR, Veerbeek JM. Systematic review on kinematic assessments of upper limb movements after stroke. *Stroke A J cerebral Circul* 2019;50:718–27.
- [56] Michaelsen SM, Jacobs S, Roby-Brami A, Levin MF. Compensation for distal impairments of grasping in adults with hemiparesis. *Exp Brain Res* 2004;157: 162–73.
- [57] Michaelsen SM, Magdalon EC, Levin MF. Grip aperture scaling to object size in chronic stroke. *Mot Control* 2009;13:197–217.
- [58] Tunik E, Rice NJ, Hamilton A, Grafton ST. Beyond grasping: representation of action in human anterior intraparietal sulcus. *Neuroimage* 2007;36(Suppl 2): T77–86.
- [59] Rushworth MF, Ellison A, Walsh V. Complementary localization and lateralization of orienting and motor attention. *Nat Neurosci* 2001;4:656–61.
- [60] Shulman GL, d'Avossa G, Tansy AP, Corbetta M. Two attentional processes in the parietal lobe. *Cerebr Cortex* 2002;12:1124–31 (New York, NY : 1991).
- [61] Monakow C von. Die Lokalisation im Grosshirn und der Abbau der Funktion durch kortikale Herde. *Bergmann JF*; 1914.
- [62] Carrera E, Tononi G. Diaschisis: past, present, future. *Brain* 2014;137:2408–22.
- [63] Carter AR, Patel KR, Astafiev SV, Snyder AZ, Rengachary J, Strube MJ, Pope A, Shimony JS, Lang CE, Shulman GL, Corbetta M. Upstream dysfunction of somatomotor functional connectivity after corticospinal damage in stroke. *Neurorehabilitation Neural Repair* 2012;26:7–19.
- [64] He BJ, Snyder AZ, Vincent JL, Epstein A, Shulman GL, Corbetta M. Breakdown of functional connectivity in frontoparietal networks underlies behavioral deficits in spatial neglect. *Neuron* 2007;53:905–18.
- [65] Sperber C, Rosenzopf H, Wawrzyniak M, Klingbeil J, Saur D, Karnath H-O. Spatial neglect after subcortical stroke may reflect cortico-cortical disconnection. *Sci Rep* 2025;15:18544.
- [66] Koch PJ, Park C-H, Girard G, Beanato E, Egger P, Evangelista GG, Lee J, Wessel MJ, Morishita T, Koch G, Thiran J-P, Guggisberg AG, Rosso C, Kim Y-H, Hummel FC. The structural connectome and motor recovery after stroke: predicting natural recovery. *Brain* 2021;144:2107–19.
- [67] Frontzkowski L, Fehring F, Frey BM, Wröbel PP, Reibelt A, Higgen F, Wolf S, Backhaus W, Braaß H, Koch PJ, Choe C, Bönstrup M, Cheng B, Thomalla G, Gerloff C, Quandt F, Schulz R. Frontoparietal structural network disconnections correlate with outcome after a severe stroke. *Hum Brain Mapp* 2024;45:e70060.
- [68] Ward NS, Brown MM, Thompson AJ, Frackowiak RSJ. Neural correlates of motor recovery after stroke: a longitudinal fMRI study. *Brain* 2003;126:2476–96.
- [69] Dancause N, Barbay S, Frost SB, Plautz EJ, Chen D, Zoubina EV, Stowe AM, Nudo RJ. Extensive cortical rewiring after brain injury. *J Neurosci* 2005;25: 10167–79.
- [70] Liu J, Qin W, Zhang J, Zhang X, Yu C. Enhanced interhemispheric functional connectivity compensates for anatomical connection damages in subcortical stroke. *Stroke A J cerebral Circul* 2015;46:1045–51.
- [71] Siegel JS, Ramsey LE, Snyder AZ, Metcalf NV, Chacko RV, Weinberger K, Baldassarre A, Hacker CD, Shulman GL, Corbetta M. Disruptions of network connectivity predict impairment in multiple behavioral domains after stroke. *Proc Natl Acad Sci* 2016;113:E4367–76.