Instantaneous neural effects of continual visual neurofeedback after stroke

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Abstract

Objective: Neurofeedback training using motor imagery-based brain-computer interfaces (MI-BCIs) show promising but variable outcomes in chronic stroke rehabilitation. However, little is known on the dual task effects of feedback when simultaneously performing a mental task. This becomes especially relevant for cognitively challenged patient groups. This study aims to investigate instantaneous effects of continual visual feedback on neurophysiological activity while stroke patients practiced MI during BCI training.

Approach: Fourteen chronic stroke patients participated in a 10-week long intervention, with 4 weeks of intense MI-BCI training. During each MI training trial, patients were instructed to mentally imagine either opening or closing their paretic hand while being provided continually updated visual neurofeedback only during the last part of the trial (after 4 seconds). We analysed patient-specific spatial and temporal dynamics of peak frequency power related to MI with and without feedback, and analysed the correlation of changes to blink rate, and clinical motor- and cognitive abilities.

Main results: Individual MI-related peak frequency of event-related desynchronisation (ERD) across sensorimotor areas was mainly found within the Alpha band. Two peak ERD patient profiles were observed: dominant ipsilesional (43% of patients) or contralesional ERD (57% of patients). Independent of ERD profile, we show weakened ERD in anticipation of visual feedback, followed by a restrengthening shortly after feedback onset, not significantly different from the prior no feedback phase. However, bilateral occipital decrease in individual peak frequency suggests increased information processing shortly after feedback onset. Furthermore, ERD was weakened during the last part of the trial (with feedback). This reduction was correlated to increased blink rate, and we show a tendency of higher cognitive ability for patients with greater reduction in ERD during the end of the trial. These results indicate that visual feedback does not interfere, nor significantly help the practice of MI during BCI training.

Significance: This study provides insight in the temporal and spatial dynamics of peak frequency ERD in response to visual feedback while stroke patients practice MI during BCI training. We believe the results will contribute to building a solid foundation of knowledge and understanding for the informed development of BCI technology in stroke rehabilitation.

Keywords
brain-computer interface, motor imagery, electroencephalography, stroke rehabilitation, neurofeedback

1 Introduction

Ischemic stroke is one of the leading causes of long-term functional disabilities worldwide ("2025 Heart Disease and Stroke Statistics," n.d.; Lui et al., 2025). For people with severe motor disabilities after stroke, rehabilitation with a Motor Imagery (MI)-based Brain-Computer Interface (BCI) has shown promising outcomes (Foong et al., 2020; Pichiorri et al., 2011; Prasad et al., 2010). The use of MI is based on findings showing that the activation patterns during MI largely overlap with those of motor execution (Ehrsson et al., 2003; Zich et al., 2015). Stroke patients are in addition shown to be fully capable of performing MI similarly to healthy people (Ang et al., 2008), and the accuracy of extracting MI from electroencephalograpm (EEG) activity does not seem to correlate to motor impairment after stroke (Ang et al., 2011). Therefore, MI constitutes an ecologically valid approach for rehabilitation considering the difficulties stroke survivors face during physical therapy.

Alongside MI, the potential of BCI technology in rehabilitation is in its ability to activate cortical plasticity that can lead to functional neural reorganisation (Engelbregt et al., 2016; Kaiser et al., 2014; Orsborn et al., 2014; Ros et al., 2013). Providing patients with feedback based on their cortical activity (i.e. neurofeedback) facilitates neural learning through reinforcement learning mechanisms. Therefore, the feedback constitutes a crucial part of a BCI to obtain successful outcomes in a rehabilitation context. From a dual task perspective, there is currently very little known on how providing neurofeedback simultaneously as performing MI affects the underlying activity, and hence the ability of performing the MI task and at the same time process the information given by the feedback. One study with healthy participants points to the benefit of performing MI with visual neurofeedback, showing both an increase in functional Magnetic Resonance Imaging (fMRI) BOLD activation as well as stronger EEG event-related desynchronisation (ERD) in sensorimotor areas when feedback is provided (Foong et al., 2020). However, whether this benefit persists in clinical populations still need to be demonstrated. MI is a cognitively demanding task, which requires substantial focus and attentional resources. After stroke, cognitive ability is in addition to motor impairments also often observed to be compromised (El Husseini et al., 2023), and the ability to simultaneously process feedback information might therefore be even more challenging.

In this study, we set out to investigate the instantaneous impact of simultaneous visual feedback on cortical activity and blink rate while chronic stroke patients performed multiple sessions of EEG-based MI-BCI training. We further investigate whether cognitive and motor impairments correlate with observed changes in EEG activity following feedback.

2 Method

2.1 Participants

Sixteen chronic stroke patients participated in the study. Each patient underwent clinical assessments, carried out by a trained physiotherapist at Danderyd University Hospital in Stockholm in Sweden before starting the experiments. Details on the inclusion and exclusion criteria can be found in the supplementary materials. The intervention targeted patients with stroke and persistent moderate to severe arm and hand motor impairment. Two patients interrupted the study due to personal reasons, resulting in a total of 14 patients that were included in the data analyses of this study. Nine of the patients had ischemic stroke and 5 had haemorrhagic stroke, all 14 within the supply area of the middle cerebral artery (MCA). A majority involved both cortical and subcortical regions and 9 out of 14 involved the right hemisphere. Mean age was 52 years and 9 out of 14 participants were males. Time to inclusion was mean 17 (min-max:12-31) months. Neurological assessment at inclusion indicated an overall stroke severity ranging from mild to moderate (median 4, min-max: 0-10) according to the NIH stroke scale, while Fugl-Meyer Assessment for the upper extremity indicated moderate to severe hand motor impairment (mean 25, min-max:11-46, a higher score indicating less motor impairment)

The study adhered to the Declaration of Helsinki and was approved by the Swedish Ethical Review Authority (Dnr 2022-00713-01) as well as the Swedish Medical Agency (CIV-21-10-037847). The study was registered as a clinical trial at clinicaltrials.gov (NCT04847089). All patients participated voluntarily and provided informed written consent.

2.2 Intervention Design

The full duration of the intervention was 10 weeks per patient and contained three different phases. The initial baseline phase was three weeks and consisted of three baseline EEG recordings without feedback (once/week), two fMRI recordings, and three clinical test sessions (once/week). This phase was followed by 4 weeks of BCI training three times per week (i.e. MI with neurofeedback) and four clinical test sessions (once/week). The last evaluation phase was identical to the baseline phase, except for the clinical test sessions that did not include certain tests (described below).

In this study, we only analyse EEG data from the BCI training phase as the focus is on the impact of neurofeedback. Data from the clinical test sessions are analysed throughout the intervention, as some of the tests were performed only during the baseline phase. An overview of the intervention can be found in figure 1A.

2.3 Clinical tests

A battery of clinical tests was conducted by a licensed physiotherapist to evaluate cognitive and motor abilities of the patients at baseline and throughout the intervention. Of relevance here, cognitive function was assessed at baseline using the Montreal Cognitive Assessment scale (MoCa) with a maximum score of 30 (Nasreddine et al., 2005a). Further, we continuously monitored motor function throughout the intervention (once/week) using Fugl-Meyer (FMA-UE) score, both motor and position sensibility scores with a maximum score of 60 and 8 respectively. Assessment of hand spasticity was performed with the NeuroFlexor method (www.aggeromedtech.com) (GÃ¤verth et al., 2014, 2013; Lindberg et al., 2011) and continuously monitored throughout the intervention (once/week).

2.4 Structural MRI scans

Each patient underwent two MRI recordings before and after the neurofeedback sessions (amounting to a total of 4 sessions) to characterise their lesion size and location. Anatomical whole brain spin-echo T1 and T2 weighted sequences were carried out without contrast media (non-invasive). During this sequence, the patient was instructed to lay calm at rest with eyes closed and not think of anything in particular. A Siemens MAGNETOM Prisma 3T scanner was used and all recordings were held at Stockholm University Brain Imaging Centre. Before participating, the patients filled out a screening questionnaire, and before each session they were screened for metal objects. Functional MRI was also acquired while patients performed a baseline MI paradigm (similar to EEG baseline sessions). We did not include this data in the analyses.

2.5 Data collection

EEG activity was recorded and sampled at 1kHz using the actiCHamp amplifier (Brain Products) with 62 active Ag/AgCl EEG electrodes placed on the

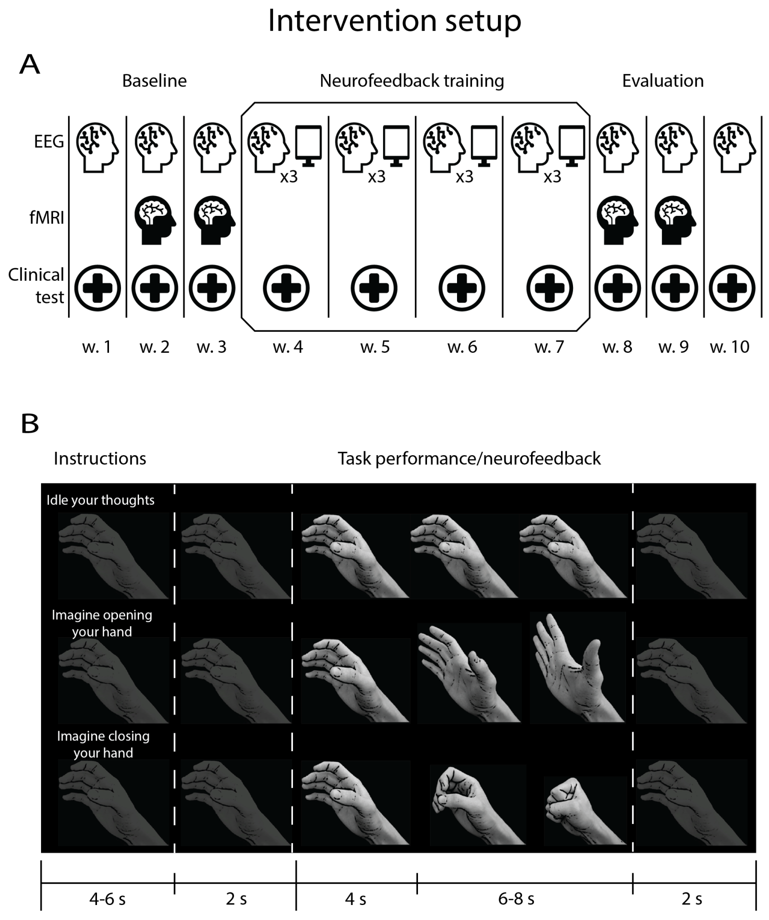

Figure 1. Intervention outline and task protocol. A) The intervention was composed of three phases: baseline (3 weeks), training (4 weeks), and evaluation (3 weeks). Clinical tests were conducted once/week during all phases of the intervention, however, some tests were conducted only at baseline.Structural and functional MRI recordings were conducted during week 2, 3, 8, and 9.. At baseline and evaluation, EEG recordings were performed during practice of motor imagery (MI) and motor execution (ME) tasks. During training, three MI-based neurofeedback sessions were performed each week.. B) The task protocol for the neurofeedback training sessions (outlined in A). During BCI training, three tasks were performed: 1) MI of either open or close the hand, 2) motor observation of previous feedback, and 3) idling. For each trial, following the instruction, a 2 s preparation time with a dark grey hand in a neutral position was presented to the patients. The task onset was then signalled by the hand turning light grey. During the first 4 seconds of each task the hand remained in its neutral position and was then followed by 6-8 seconds of movements that reflected the cortical activity of the patient. Motor observation provided the exact same hand movements as the previous MI trial. For the idle task the hand stayed in a neutral position throughout the duration of the trial.

scalp of the patients according to the 10/10 standard. The ground electrode was placed at Fpz location and the left nose wing was used as location for the reference (electrode TP9). Vertical electrooculography (EOG) was measured as the difference between Fp2 and one electrode placed under the right eye. Two passive Ag/AgCl electrodes (Brain Products) were placed next to the outer canthi of each eye to measure horizontal EOG (ground placed on the left earlobe). Passive Ag/AgCl electrodes (Brain Products) were placed on the flexor and extensor muscles on the paretic forearm to measure two channels of EMG. Two electrodes were placed 2 cm apart on the medial group of antebrachial muscles with a reference electrode placed on the medial epicondyle, and two were placed on the lateral group of antebrachial muscles with a reference electrode placed on the lateral epicondyle. Lastly, two one dimensional accelerometer sensors (Brain Products) were placed on the top of the hand and index finger respectively to measure movements. Prior to the start of each session, the impedance of each active electrode was lowered to below 40 kOhm by application of an electrolyte gel. The impedances were verified and if necessary adjusted, at half the session to ensure good values. The signal of each electrode was inspected carefully before starting each session to ensure minimal drift and noise, and to detect faulty electrodes.

2.6 BCI training sessions

A total of 12 BCI sessions were performed by each patient, with each session split into two blocks with a short break in-between. All experiments were conducted in a quiet and dimly lit room, where the patients were sitting comfortably in front of a computer screen. A trained BCI operator were sitting on the right hand side of the patient in front of three computer screens, monitoring the raw EEG, EMG and EOG signals, the protocol progression and the feedback given to the patient. The operator had full vision of the patient and took notes of anomalies or intervened if instructions were not followed. Instructions were given before the sessions with video and audio and the patients had the opportunity to ask questions.

During the session, task-specific instructions were given at the start of each trial. The patients were instructed to perform three different tasks: 1) MI of opening or closing the hand, 2) motor observation (MO), and 3) idling/rest. For MI, we specifically instructed patients to perform kinaesthetic MI of one movement, i.e. imagine the sensation of opening or closing their hand once and hold the imagined hand position. Following the specific MI task instruction, a dark grey animated hand was kept in a neutral position on the screen for a duration of 2 seconds. By turning light grey, the animated hand signalled the onset of the MI task to the patients. The hand was maintained still in its neutral position for the first 4 seconds and then started to move (i.e. provide feedback) based on the output of a support vector machine (SVM) classifier, reflecting ongoing EEG activity of the patient. For positive feedback, the hand always moved in the direction of the instruction (open or close). Neurofeedback was provided for 6-8 seconds; the exact duration was jittered. An MO trial always followed an MI trial, and patients were instructed to rest their mind and only observe, without performing any mental task, the exact same movements of the animated hand as shown during the previous MI trial (i.e. observing the previously provided feedback). During idle trials the patients were also instructed to rest their mind, but this time the animated screen hand was kept still in its neutral position. For a timeline of each instruction see figure 1B. During each of the two blocks patients performed 10 trials of open hand MI and 10 trials of close hand MI followed by their corresponding MO trial, and 10 trials of idling. This amounted to a total of 40 trials of MI, 40 trials of MO and 20 trials of idling. The order of MI/MO and idle trials was pseudorandomised. Before and after each block within the BCI sessions, the patients were asked to self-evaluate how tired they felt (score of 1, 2 or 3 with 3 being the least tired).

2.7 Online signal processing

The BCI system developed in-house used LabVIEW for online data handling and Matlab for signal processing and feature extraction. The data were streamed using packets of 250 ms with a sliding window of 100 ms through the Py-Corder software to LABVIEW and then to Matlab for online signal processing. Each packet was individually pre-processed by removing the DC offset (subtracting the average). Then blink artifacts were removed using the adaptive filtering method described in (He et al., 2004) with the length M=3 for the finite impulse response filter and a forgetting factor of 0.999 for the recursive least-squares algorithm. EOG data from both horizontal and vertical electrodes were used as reference. This method was chosen since it converges fast and is well suited for an online setting. To mitigate volume conduction, the Surface Laplacian spatial filter was applied based on the method described in (Cohen, 2014).

Feature extraction was performed on each data packet using fast Fourier transform (zero-padding to 1000, fft function in Matlab). Since we are interested in features from the motor cortex, a subset of EEG channels was selected, excluding the peripheral electrodes: [AF7, AF8, F7, F8, Fp1, Fp2, FT7, FT8, P7, P8, T7, T8, TP7, TP8, TP9, TP10]. Subsequently, remaining 46 central channels and a frequency band between 2 and 30 Hz resulted in 1334 features per streamed data packet. To mitigate the influence of outliers, each feature's time series was min-max normalised using the 90th percentile as the upper bound.

2.8 Online classification and prediction

To determine the feedback (i.e. animated hand movements), a linear SVM (fitclinear in Matlab) was used to classify and predict the two classes: MI and MO. The MI class contained both MI of open and close hand movements. The SVM model was not trained on prior data, instead, it was continuously rebuilt from cumulative data during the BCI training. It was retrained every new MI and MO trials. Since the first MI trial does not have a model, the feedback output was determined by a 55% chance of positive feedback. Before the second MI trial (after the first MI and MO and potential idle trial), the first model was built from all features generated during MI and MO. At this time, between approximately 80-120 data packets per task was included in the model, using 1334 features per packet. After the next MI and MO trial passed, the model was rebuilt again using all data from previous MI and MO trials, essentially doubling the amount of data. This continued through the first block, with the model including 20 MI and MO trials in the end. For the second block, all data from the previous block was used as model from the start. The model was then retrained every MI/MO pair of trials. Throughout the session, the SVM model was rebuilt during a short pause between each trial, unnoticed by the patient.

Feedback was provided with each new data packet (streamed every 100 ms) by predicting on the latest 250 ms data using the most current SVM model. If the prediction output was MI, the BCI provided positive feedback in the form of moving the animated hand on the screen one frame in the direction of the task instruction (open or close). On the opposite, if the prediction output was MO, negative feedback was provided by moving the hand towards the neutral position. To reduce jitter, two consecutive predictions of the same class was required to change direction of the animated hand (one prediction required to update the frame of the same class).

2.9 Offline signal processing

The offline analysis was applied on the collected raw data without the need for streamed data packets. Because of that, the pipeline was changed slightly. The continuous raw data was first bandpass filtered between 0.5 and 60 Hz. Adaptive filtering was then applied on the whole time series instead of separate time windows, with the same length M=3 but a slightly lower lambda=0.995 after internal testing. Each individual trial for all patients was manually inspected by visualising the signals of representative channels (AF3, AF4, C3, C4, PO3, PO4). Trials with bad channels or apparent noise from muscle artifacts were flagged for rejection and excluded from the analysis. Lastly, Surface Laplacian (Cohen, 2014) was applied in a similar fashion as the online signal processing.

For the online feature extraction, we used the fast Fourier Transform, excluding the time dimension in favor of fast processing. For the offline analysis we wanted to keep the time dimension and opted to use a complex Morlet wavelet transform instead. It was applied on the data with a wavelet of 7 cycles and a timespan between -4 and 4 seconds. All 62 EEG channels were convolved with the wavelet over 40 frequencies (1-40 Hz). Lastly, power was extracted and converted into decibels.

2.10 Blink detection

To find number of blinks during each trial, the EOG data was first bandpass filtered between 0.5 and 60 Hz. The data was then split into its individual trials, and the blink detection was applied on all trials separately for each block and later cumulated for each patient. The built-in findpeaks function in Matlab was used with maximum peak width set to 1000 ms and minimum peak width set to 50 ms. The threshold was defined as two standard deviations above the mean of each session block. The timepoint of each detected peak relative to each trial was saved in a matrix. For each task, the number of blinks per millisecond was averaged over trials and split in to three time windows representing no feedback, and early and late feedback (1-3s, 5-7s and 8-10s after MI task onset). Each task and window was scaled by dividing by the mean over all time windows.

3 Results

First, we wanted to explore how the patients varied in their EEG response to performing MI with and without feedback. We observed individual differences of the ERD, mainly in terms of peak frequency and location. To illustrate this, we first show the ERD as a contrast between MI and idle trials across all frequencies, spanning from low alpha to high beta frequencies (two representative patients in figure 2Ai and ii). For this, we specifically targeted sensorimotor electrode locations (C1-C4, CP1-CP4) and investigated each hemisphere separately (i.e. lesioned vs. healthy). The time window used for this analysis was chosen out of three alternatives based on which one resulted in the highest ERD: three time-windows of 2 seconds each were predefined, in the beginning, middle and end of the MI trial (1-3 s, 5-7 s, 8-10 seconds after task onset). These windows would thus represent no-feedback, early-feedback and late-feedback. A fixed time window in the beginning of the trial (1-3 s after task onset) was used for idle trials, since we do not expect any time-locked effects in the power for these trials. A clear ERD peak can be observed in 9-11 Hz for SP01, for which the response was mainly contralesional. On the contrary, for SP02, we observe peak frequencies around 8-10 Hz on the ispilesional side. Taking this to all 14 patients, we can show that the majority had a peak frequency within the alpha frequency band (n=11), with a large variability in specific peak frequency (figure 2Aiii). We further discovered an interesting split between the patients in our patient group where 6 patients had stronger ERD for ispilesional sensorimotor channels (referred to as ipsi group) while 8 patients had stronger ERD for contralesional sensorimotor channels (referred to as contra group, 42.86% and 57.14% of our patient group, respectively).

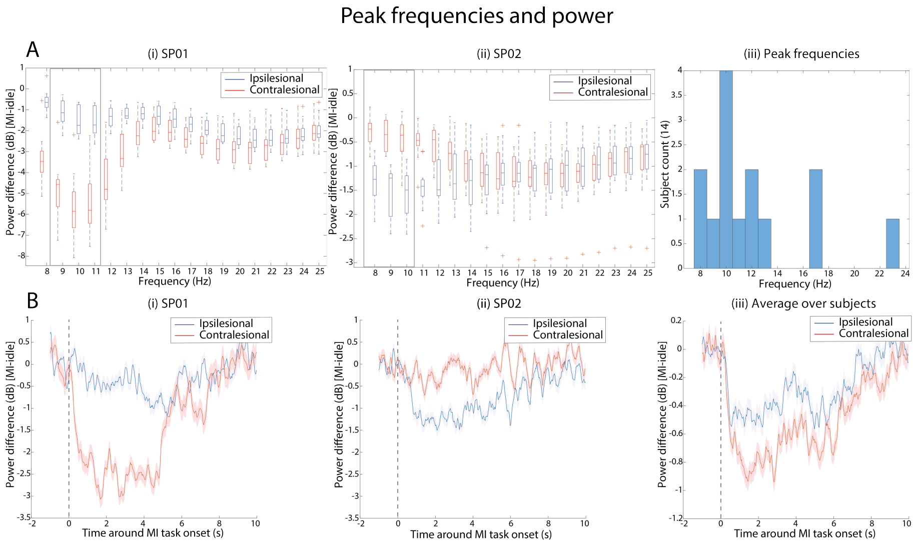

Figure 2. Ipsi and contralesional EEG response to performing MI. A) Average power (MI-idle) over sessions for each frequency in the alpha and beta band for patient SP01 and SP02 (i and ii) during nofb (1-3 s after task onset). Blue boxes show power for channels over the ipsilesional hemisphere, and red boxes show power for channels over contralesional hemisphere. Enclosed boxes highlight peak ERD frequency +-1Hz for that patient. A(iii) The distribution of peak ERD frequencies for all patients (n=14). B) Power across time around MI task onset, averaged over peak frequencies +-1Hz and ipsi- and contralesional hemispheres (blue and red) for patients SP01 and SP02 (i and ii), and all subjects (iii). Shaded area represents standard error of all trials in that task. Dashed lines denote task onset.

We then set out to explore the temporal evolution of peak frequency ERD within trials for ipsi- and contralesional sensorimotor electrode locations. We plotted in time the average power contrast between MI and idle trials of the peak frequency ± 1 Hz , spanning both the no feedback and feedback part of the trial, for the same two representative patients: SP01 and SP02 (figure 2Bi and ii). Following MI task onset of patient SP01, a decrease in power can be observed in both the ipsi- and contralesional hemispheres, however the ERD was significantly more dominant on the contralesional side. Following feedback onset at 4 s, a rather steep reduction in ERD can be observed both ipsi- and contralesionally, followed by a more gradual reduction until the end of the trial. For patient SP02, a strong ipsilesional ERD can be observed following MI task onset. The ipsilesional ERD was stable throughout the no feedback period, while it slowly decreased during the feedback period, following feedback onset. These temporal dynamics can be visualised on a population level (n=14) and we can further observe both strong ipsi- and contralesional ERD activity, with the latter dominant (figure 2Biii).

3.1 The spatial and temporal instantaneous effects of continual feedback

To have a more comprehensive understanding of the spatial and temporal differences of peak frequency ERD between the no feedback and feedback part of MI trials, we show topographical plots of three different time windows: no feedback, and early and late feedback period (averaged activity over 1-3 s, 5-7 s, and 8-10 s after MI task onset, respectively). We divided the patients into the contra- or ipsilesional group based on which hemisphere the peak frequency was dominant. We further quantified the differences by plotting the contrast between each two consecutive time windows. Overall, we reveal strong ipsilesional ERD extending to the contralesional side for the ipsilesional group (figure 3A; n=6 patients). A slight ERD decrease over bilateral sensorimotor areas, together with increased ERD across lateral occipital electrodes can be observed when going from no feedback to early feedback. This ERD pattern is weakened during the late feedback period. For the contralesional group (n=8 patients) we observe strong focal ERD across the contralesional sensorimotor region, in conjunction with an event-related synchonisation (ERS) across lateral more frontally located areas on the ipsilesional side (figure 3A). In contrast to the ipsilesional group, this group does not display any bilateral ERD. The observed pattern is maintained yet slightly weakened during early feedback and then further weakened during late feedback. Similar to the ipsilesional group, lateral occipital ERD can be observed during early feedback.

To statistically quantify changes in peak frequency ERD over sensorimotor areas in figure 3A, we plotted boxplots of the power difference between MI and idle trials, averaged across hemispheric electrode locations [C1, C3, CP1, CP3] and [C2, C4, CP2, CP4], separately for the ipsi- and contra patient groups (figure 3B). A 2-way repeated measures ANOVA for each patient group revealed main effects of power for contra- vs. ipsilesional hemispheres (ipsi group: $p=1.77*10^{-9}$, contra group: $p= 9.86*10^{-17}$) and for time windows (ipsi group: $p=2.66*10^{-14}$, contra group: $p=1.33*10^{-7}$). For both groups, we obtained interaction effects between hemispheres and time windows (ipsi group: $p=0.02$, contra group: $p=4.05*10^{-9}$), indicating that the power changed differently in time depending on hemisphere. Further post-hoc testing using Tukey-Kramer (with correction for multiple comparisons) revealed a significant increase in power during late feedback for both the ipsi- (no fb vs. late fb, $p=8.32*10^{-9}$) and contra patient groups (no fb vs. late fb, $p=3.75*10^{-5}$), whereas no significant difference was found between no and early feedback (ipsi group: no fb vs. early fb, p=0.88; contra group: no fb vs. early fb, p=0.28).

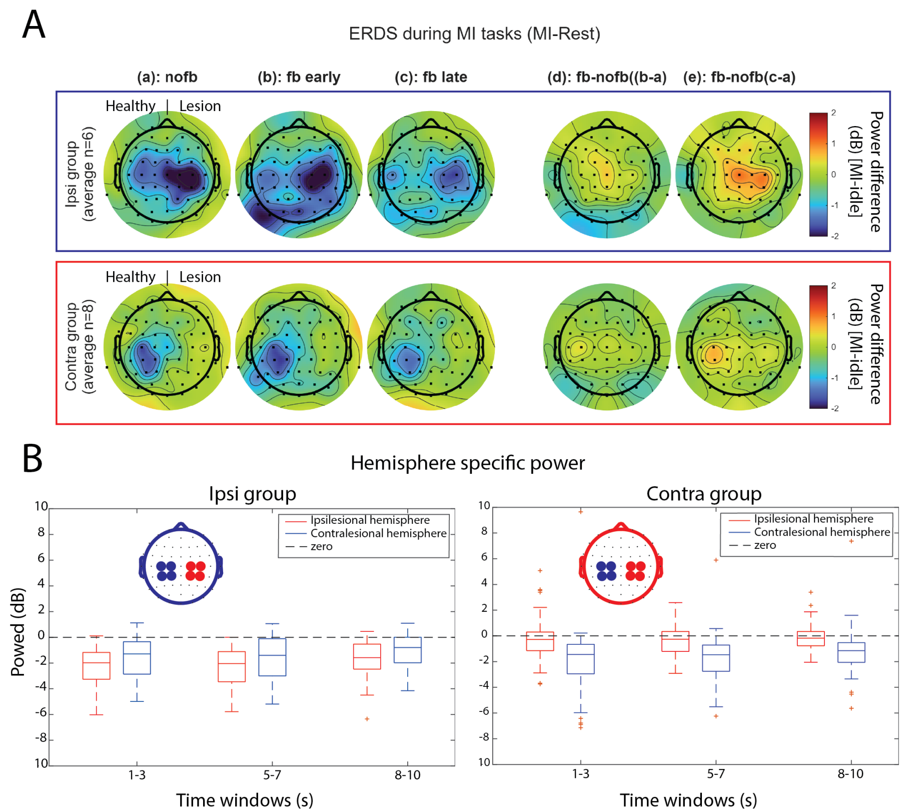

Figure 3. Peak frequency ERD power differences (MI-idle) during no fb (1-3 s), early fb (5-7 s) and late fb (8-10 s after task onset). A) Topographical plots of power with the ipsi (n=6) and contra (n=8) groups separated (blue and red box). The contrast of early fb and no fb, and late fb and no fb are shown in (d) and (e) respectively. B) Peak frequency ERD power difference across patients, averaged over channels [C1, C3, CP1, CP3] and [C2, C4, CP2, CP4] (blue and red dots), of the ipsi (blue head) and contra (red head) group.

3.2 Temporal dynamics of blink rate

To better understand what might have caused the weakened ERD/ERS observed independently of contra/ipsi group during the late feedback phase of the trial, we estimated the blink rate from the recorded EOG data. For coherence, we calculated the number of blinks during the three different time windows corresponding to: no feedback, and early and late feedback (1-3s, 5-7s and 8-10s after MI task onset). We show the normalised blink rate across all blocks and sessions for MI trials, as well as idle trials separately (figure 4). During idle trials, the number of blinks significantly increased when going from no feedback to feedback (Wilcoxon signed rank test with Bonferroni correction for 7 tests: no fb vs. early fb, p=0.00077). However, the rate did not change during the feedback (Wilcoxon signed rank test with Bonferroni correction for 7 tests: early vs. late fb, p=1). On the contrary, during MI trials, there were no significant changes in blink rate when going from no feedback to feedback (Wilcoxon signed rank test with Bonferroni correction for 7 tests: no fb vs. early fb, p=0.28). However, a significant increase in blink rate can be observed during the feedback phase of the MI trial (Wilcoxon signed rank test with Bonferroni correction for 7 tests: early vs. late fb, p=0.0078).

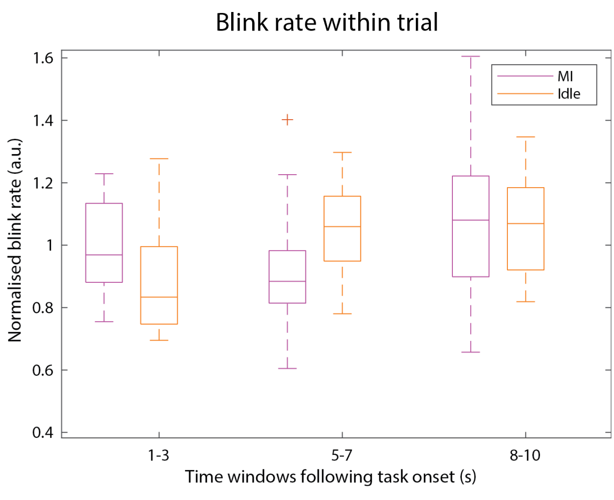

Figure 4. normalised blink rate over the time windows: no fb (1-3 s), early fb (5-7 s) and late fb (8-10 s after task onset) for the MI (purple) and idle (red) trials. Each box represents the average amount of blinks detected over all subjects, normalised to scale all time windows around 1.

3.3 Behavioural correlates of ERD changes during late feedback

We wanted to further understand whether any behavioural measures could explain the observed ERD changes in our group of patients. We therefore correlated the different behavioural metrics with the difference in ERD between the no feedback and the late feedback time windows (figure 5). First, the grand average of self-experienced tiredness (on a scale from 1 to 3) did not correlate with the ERD change (Spearman's rho= 0.30, p=0.29). However, the correlation between ERD change and the MOCA score did show a positive trend (Spearman's rho=0.46, p=0.10), suggesting that patients with higher cognitive ability generally had larger differences of sensorimotor ERD between no feedback and late feedback. Further, neither the FMA-UE motor function (Spearman's rho=-0.02, p=0.94), position sensibility (Spearman's rho=0.05, p=0.85) nor the neuroflexor component (Spearman's rho=-0.00, p=0.99) showed any correlation to ERD change between no feedback and late feedback.

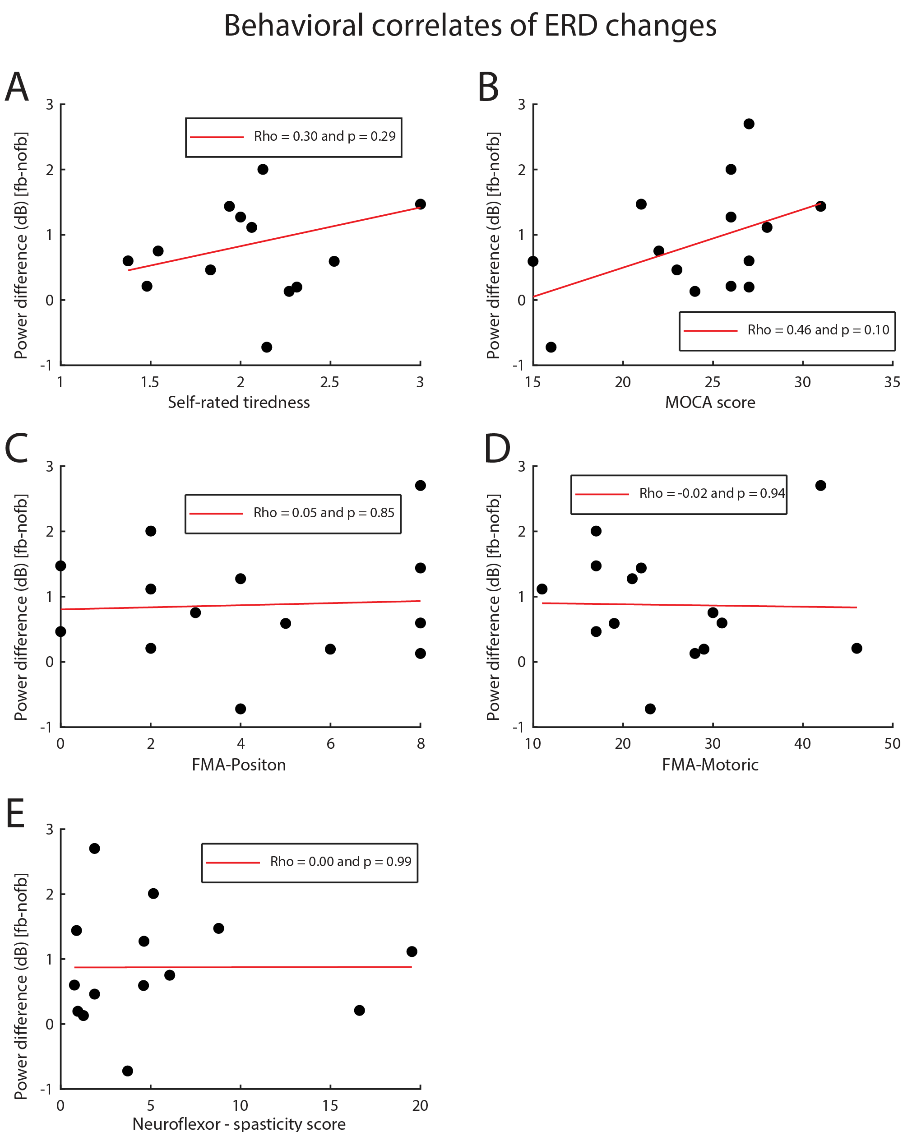

Figure 5. The difference between late fb and no fb peak frequency ERD across sensorimotor regions correlated with A) Self-rated tiredness, B) MOCA score C) FMA-Position sensory, D) FMA-Motor function, and E) Neuroflexor spasticity score. Each dot represents one patient, and the red line corresponds to the spearman correlation.

4 Discussion

In this study we have investigated how visual continual feedback impacts the EEG activity during the practice of MI in a group of chronic stroke patients. Our experimental design allowed us to compare the MI-related EEG activity with and without feedback within the trials. We observed two main profiles of ERDS patterns during MI in our group of patients: i) bilateral sensorimotor ERD with ipsilesional dominance, and ii) focused contralesional sensorimotor ERD and ipsilesional frontocentral ERS. Independent of ERDS profile, we show that the activity follows the same temporal dynamics during trials, with specific effects that can be attributed to the onset of visual continual feedback. We further show time-on-trial neurophysiological effects, that we investigate in conjunction to cognitive and overt behavioural metrics. These results will be discussed in the following.

4.1 Individual variability in neurophysiological MI-related activity

With previously described well-established patterns during MI (McFarland et al., 2000) as a point of departure, one of our first observations were the differences in ERD peak frequency between patients. While almost all patients had their peak frequency within the alpha band, three patients had peak frequencies within the beta band. There are numerous reports showing ERD over sensorimotor areas dominantly on the contralateral side during the performance of upper-limb MI (McFarland et al., 2000; Pfurtscheller et al., 2006), however the subject-specific nature of these activations is less known, especially for stroke patients. In contrast to our results, a rather narrow range of peak frequencies in the alpha band has been shown in a group of healthy participants (10.9Hz$\pm$0.9Hz) (Pfurtscheller et al., 2006). The variability in peak frequency that we observe could potentially be explained by the heterogenous nature of our participant group. Stroke patients with lesions varying in their location and size will reasonably lead to neurophysiological differences that might explain our observations as compared to healthy participants.

In addition to varying peak frequencies, we also show that the laterality of the activity with regards to the location of the lesion varied across our patient group. Eight patients showed focal unilateral ERD across the contralesional sensorimotor cortex in conjunction with ipsilesional ERS located a bit more frontally. On the opposite, there were six patients showing rather unfocused bilateral ERD across the sensorimotor cortex. However, for these patients, ERD dominated on the ipsilesional side of the brain. Bilateral enhanced activation of motor areas is typically observed in the acute phase after stroke (Rehme et al., 2011). This seems to be a temporary phenomenon and depending on motor recovery, the laterality of overactivation will change with time (Ward et al., 2003). There is support for the ERDS patterns observed in our two groups from the literature: namely ipsilesional ERS with contralesional ERD versus ipsilesional ERD over motor areas, and studies have shown that each activity patterns can be specifically correlated with functional recovery (Tangwiriyasakul et al., 2014). In this study, we focus particularly on the impact of visual feedback on ERDS during MI, therefore we merely describe and account for the differences in our analyses without making any inferences on the functional relevance for motor recovery. From the literature, we can conclude that the two profiles of ERDS activity patterns observed in our group of patients are typical patterns observed after stroke. Our next question is how continual visual feedback impacts these ERDS profiles and whether the impact is profile-specific?

4.2 Spatial and temporal instantaneous effects of visual continual feedback

In our experimental design, each MI trial consisted of four seconds of MI without any visual feedback (a hand on the screen was fixed in its neural position) followed by 6-8 seconds of MI with visual feedback (based on previous 250ms of data) that was updated every 100ms (i.e. continual). We performed crossover analyses to investigate the effects of visual feedback on ERDS. We intentionally designed it such that the no-feedback condition always occurred before feedback to ensure that it was pure no-feedback. Otherwise, if feedback was provided before the no-feedback condition, it could have affected the performance of MI and introduced confounds in the data. However, it is important to note that this design does not allow to control for time-on-trial effects (such as changes in arousal, increased attentional lapses or fatigue at the end of the trial). On the other hand, we aimed to mitigate time-on-task effects by having the experimental conditions occur on a trial-level. As such, we first wanted to look at the sensorimotor ERDS in time across the trial to investigate whether the feedback had an impact on the activity and how it evolved throughout the trial. We focused on individual ERD peak frequencies and by averaging across all patients, we show a sharp decrease in power for both ipsi- and contralesional sensorimotor cortex shortly after the onset of the MI-task (figure 2B). In our group of patients, the contralesional ERD was stronger which is reasonable considering that all patients, independent of group, showed contralesional ERD, whereas only the ipsilesional group (n=6) showed ipsilesional ERD during MI. Up until about 3 seconds of MI without feedback ERD remains high, and then temporarily decreases (power increase) in what seems to be in anticipation of the upcoming feedback. Interestingly, this reduction of ERD occurs about half a second earlier on the contra- as compared to the ipsilesional side (figure 2B). Further, for both ipsi- and contralesional ERD, the reduction is maintained for about 2 seconds around the onset of the visual feedback and then restrengthens temporarily before steadily decreasing to its baseline at the end of the trial. We believe there are several interesting observations to be made here: 1) the reduction in ERD before the onset of visual feedback, 2) the restrengthening of ERD at the beginning of visual feedback, and 3) the slow but steady weakening of ERD during the late phase of the trial.

In the healthy population, an increase in beta power has been previously observed in preparation for motor imagery (Vukelić and Gharabaghi, 2015) as well as in anticipation of behaviourally relevant cues (Saleh et al., 2010). The increase in peak frequency power before the onset of visual feedback can therefore be interpreted as a form of anticipation of the visual feedback due to its timely predictive nature. Since alpha frequencies constituted the peak frequency for many of the patients, we extend previous findings by showing that sensorimotor alpha also shows this anticipatory power increase previously observed in beta. We speculate that this increase could reflect anticipatory attentional processes of the up-coming visual stimuli (Kilavik et al., 2013).

The restrengthening of sensorimotor ERD observed shortly after the onset of visual feedback indicates an internal reorientation of focus towards motor imagery, thereby decreasing peak frequency sensorimotor power. This restrengthening could be caused by an increased level of arousal due to the feedback, or it could be due to the patients using the feedback to regulate their cortical activity. Previous reports on healthy participants are somewhat inconsistent in their findings of the impact of feedback on cortical motor activity during MI. While there is some support for stronger broad-band ERD (8-30Hz) across sensorimotor areas when visual neurofeedback is provided (as compared to no feedback) (Zich et al., 2015), another study report initial stronger sensorimotor alpha ERD and weaker beta ERD for MI without feedback as compared to MI with motor observation (not neurofeedback) (Adham et al., 2024). Although significant in its temporal dynamics, the sensorimotor enhancement of ERD early after the feedback onset was not significantly different from the ERD during MI without feedback. This result therefore suggests that visual feedback that is continually updated does not interfere with the ongoing mental processes of performing MI after stroke. However, at the same time, visual feedback does not automatically seem to strengthen peak frequency ERD across sensorimotor regions. Since our experimental protocol incorporated the two conditions (with and without feedback) on a trial-level, our analyses will follow any learning effects reflected in the evolution of ERDS across the session and intervention. Previous report on the effect of visual neurofeedback did not adequately account for evolving ERDS related to learning (Zich et al., 2015), and these effects might therefore be present in the reported condition-differences.

During the late phase of the trial, with feedback, we observe a return to baseline in the sensorimotor ERD. The patients were instructed to mentally imagine the feeling of moving the hand either through opening or closing it one single time and then to hold that hand position in mind. Although there is no way to know the exact duration of the imagined movement, we can speculate that the patients were imagining the hand in a static hold during the last phase of the trial, plausibly around the time when ERD decreased. Beta power has been shown to increase during real static hold of grasp (Spinks et al., 2008) and the oscillations has been shown to be significantly coherent and phase synchronised with EMG of tonically contracting muscles (Baker et al., 1997). Although there are no reports on MI of static hold, static visual imagery has shown an initial suppression of alpha power followed by an enhancement (Hashim et al., 2024). The increase in peak frequency power might therefore be explained by the static hold nature of MI during that period of the trial, however, it can also be explained by cognitive factors, such as reduction in arousal or fatigue. In the next section, we will discuss the presence of such variables affecting the performance and thus the EEG activity.

We further wanted to analyse the spatial effects of visual feedback. We chose three time windows to do this: without feedback, shortly after feedback onset, and at the end of the trial with feedback. Since we observed two very distinct spatial ERD patterns in our patient group, we divided the two groups and analysed them separately (referred to as ipsi- and contralesional group depending on ERD dominance). For both groups, there were no significant differences in sensorimotor ERD between no feedback and early feedback for neither of the hemipsheres. When comparing early and late feedback, sensorimotor ERD was significantly weakened for both groups on the dominant side as well as on the non-dominant contralesional side for the ipsilesinal group. The non-dominant ipsilesional side of the contralesional group did not show any differences, which is reasonable considering that there was no detectable ERD.

For both groups, shortly following feedback onset, we can observe ERD over bilateral occipital regions with dominance on the contralesional side. This is in line with previous studies showing posterior alpha decrease when visual neurofeedback is provided online (Shenoy et al., 2006). Further, occipital alpha has been suggested as an index of inhibition of visual processes, independent of whether it is internally or externally driven (Hohaia et al., 2022). Further, the role of occipital alpha in visual attention (Peylo et al., 2021) and especially contralateral suppression of alpha at parieto-occipital areas during attention orientation (Sauseng et al., 2005) supports our observations of dominant contralesional suppression of alpha if one is considering the screen hand being oriented with fingers pointing to the contralateral side. Even though the sensorimotor ERDS patterns seem to be manifested differently after stroke, occipital alpha does seem to be consistently suppressed contralateral to where the hand is positioned on the screen. This occipital ERD weakens at the end of the trial, suggesting that it was either a temporary visual effect due to the start of hand movements on the screen, or it was because of less screen hand movements at the end of trial.

4.3 Explainability of cognitive and motor behaviour

As our experimental design does not control for effects due to time-on-trial, we wanted to analyse potential biomarkers of such effects. One such biomarker, independent of EEG -based ERDS, is blink rate, which has systematically been observed to increase in relation to time-on-task (Fukuda et al., 2005; Rodriguez et al., 2018). Therefore, we analysed blink rate over three time windows during idle trials and compared the rate over the same time windows of MI trials (figure 4). Interestingly, we observe rather opposite temporal patterns between idle and MI trials. Indeed, during idle trials, blink rate significantly increased during the later part of the trial (for the second and third time windows). During idle trials, the screen hand was fixed so it is interesting to note that blink rate was modulated in the absence of any visual changes on the screen, indicating that any change in blink rate was related to time-on-trial. During MI trials, blink rate remained at the same level during no feedback and early feedback, however increased significantly during late feedback. There was no significant difference between blink rates of idle and MI trials for the third time window (i.e. late feedback during MI trials), however, they significantly differed during the second time window (i.e. early feedback during MI trials). This increase in blink rate during the late phase of the trial which was independent of whether the patients were performing MI with visual feedback or idling demonstrates a clear relationship to the time-on-trial. As we are not measuring time-on-task in the sense that we did not analyse blink rate as a function of time within sessions. Instead, we are observing modulations of the blink rate related to time within trials. We show that these modulations were also reflected in the peak frequency sensorimotor ERD, relevant to the MI task, suggesting that patients had difficulty in performing MI until the end of the trial. Taken together, these results suggest that, overall, patients either became bored, less aroused, or used less cognitive resources at the end of the trial, independent of whether they performed MI or were just idling. However, the onset of visual feedback seems to have postponed this effect. Attentional processing has been proposed as a main driver for blink modulation (Ponder and Kennedy, 1927). Blinks have further been reported to occur temporally correlated to the onset of external stimuli (Siegle et al., 2008) with suppression of blinks in relation to task structure (Hoppe et al., 2018). There is mounting evidence that blinks influence information processing by suppression in phases when task-relevant information needs to be processed (Wascher et al., 2015). Our findings are in line with these theories as blink rate tends to decrease shortly after visual feedback onset in MI trials and it is significantly different from idle trials. Considering that ERD was restrengthened during early feedback of MI trials (figure 2B), we propose that attention was reoriented internally to performing MI.

We also analysed the patients' self-rated tiredness (on a scale from 1-3) averaged during the sessions to see whether it correlated with the time-on-trial effects observed in the ERD (figure 5). We found no such correlation, suggesting that overall self-perceived tiredness does not explain the reduction of ERD during late feedback.

Further, we also wanted to investigate whether the observed time-on-trial effect might have been related to any cognitive and/or motor disabilities of our patients. To do so, we correlated the peak frequency power difference between no feedback and late feedback windows with different behavioural metrics. In essence, in our patient group, we observe a weak non-significant relationship between cognitive ability, as measured with MOCA (Nasreddine et al., 2005b), and time-on-trial ERD decrease. There is a clear tendency for a more pronounced reduction in ERD during late feedback (as compared to no feedback) for patients with normal cognitive function (scores > 25). This is surprising as it might indicate that mild cognitive impairments attenuate differences in arousal and internal focused attention. Further, we did not find any correlation between motor impairments, in terms of Fugl-Meyer Assessment motor and position sensibility scores, and reduction in ERD during late feedback, suggesting that independent of motor disability, the patients were similarly impacted by time-on-trial.

5 Conclusion

This work analyses the instantaneous effects of continual visual feedback during MI tasks in chronic stroke patient EEG data. We compare ERD changes during MI with and without early and late feedback within the same trial. The results from this study show that for our patient group (n=14), peak ERD frequencies varied around Alpha, with a few patients (n=3) showing peak frequencies in Beta. Using the peak ERD frequencies for each patient we could separate our patient group into patients with dominant ipsilesional ERD and those with strong contralesional ERD dominance (42.86% and 57.14% of patients in each group, respectively). Independently of group, we observe a strong enhancement of ERD during no feedback and a restrengthening during early feedback, and lastly a weakening of ERD towards the end of the trial. Blink rate modulation within trials suggests that the weakened ERD at the end of the trial (with feedback) is related to a mental state of less arousal combined with less information processing. We further show that a weak trend of cognitive ability being positively correlated to the reduction in ERD at the end of the trial, suggesting that mild cognitive impairment attenuates the time-on-trial effects in peak frequency ERD. Finally, we show that self-perceived tiredness, as well as motor impairment and spasticity does not impact the observed time-on-trial effects.

We believe these results contribute to the development of BCI technology for stroke rehabilitation as we show that visual feedback, provided to stroke patients, does not interfere with individual peak frequency sensorimotor rhythms during simultaneous practice of motor imagery. The potential interaction between cognitive ability after stroke and time-on-trial effects during MI calls for further targeted investigations and supports personalised strategies.

6 Authors contributions

Conceptualisation, EA and MJA; Methodology, EA, MJA, JP and SP; Patient recruitment, SP; EEG data collection, MJA and EA; MRI data collection, MJA, JP and SP; Clinical data collection, SP; EEG data analysis, EA and MJA; MRI analysis, JP; Writing – original draft, EA and MJA; Writing – review and editing, EA, MJA, JP and SP; Funding acquisition, EA and MJA; Supervision, EA.

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Supplementary materials

Exclusion criteria

Inclusion criteria: Eligible participants will fulfill the following inclusion criteria: age > 18 years; > 6 months since first time stroke onset and with remaining hemiparesis in upper extremity; able to participate fully in the intervention, able to perform Functional Magnetic Resonance Imaging (fMRI); able to passively extend the wrist 15 degrees and extend fingers fully with a neutral position of the wrist.

In addition, participants need to be able to voluntarily control the power of their grip when requested according to the Visuomotor force tracking method and/or according to the clinical assessment of a therapist (while holding the patient's hand). According to the Fugl-Meyer Upper Extremity (UE) scale participants should accomplish <14 points on the hand subscale and <2 points in finger flexion and extension (C) in addition to < 47 points on the total motor score (equivalent to moderate disability in the upper extremity.

Exclusion criteria: Exclusion criteria are other neurological or musculoskeletal disease/injury, or contagious disease. If the research person is regularly treated with botulinum toxin in the upper extremity, 3 weeks should have passed before the first baseline assessment.

Since this study protocol involves non-invasive magnetic resonance imaging, additional exclusion criteria are current or history of epilepsy, severe hearing or visual impairments, metal implants in the brain/skull cochlear implants, any implanted neurostimulator, cardiac pacemaker or cardiac implants of metal, infusion device, any other neurological disorder, pregnancy, current or history of severe psychiatric disorder with need for pharmacological treatment.