

Directionality of Interpersonal Neural Influence in fNIRS Hyperscanning: Validation of a Spectral Causality Approach in a Motor Task

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February 20, 2025

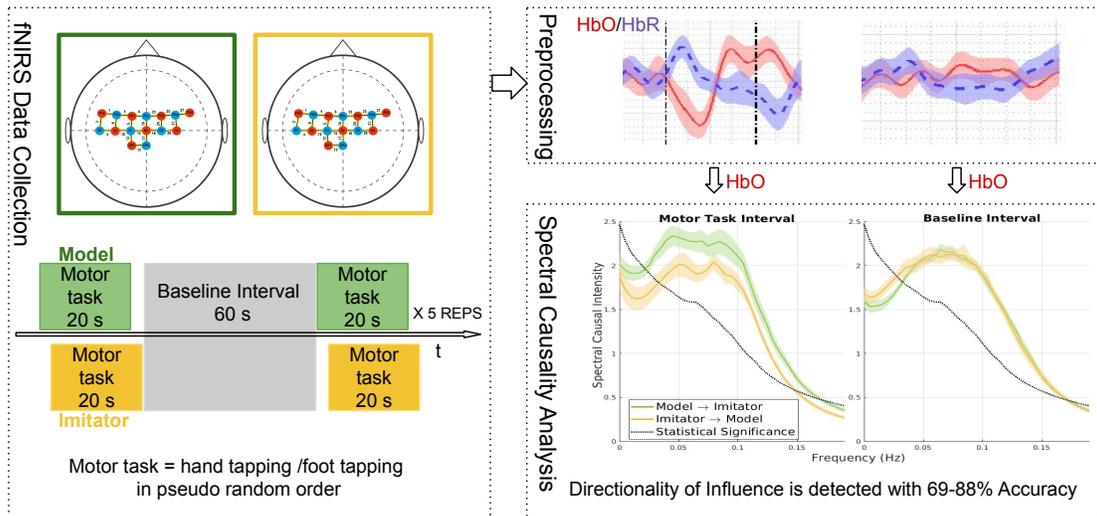
Abstract

Hyperscanning approaches represent a shift from single- to two-person neuroscience, enabling a more profound understanding of the neural mechanisms underlying interpersonal synchronization. In this context, fNIRS has emerged as a valuable tool for measuring brain activity in a natural, unconstrained environment. While interpersonal synchrony using fNIRS hyperscanning has been well studied using statistical association analysis, establishing causal relationships that elucidate the direction of influence remains challenging. This study aimed to investigate the feasibility of testing the direction of influence in dyadic interactions. Since the ground truth of such direction is not available in a natural setting, we validated our approach in an experimental setup in which we controlled the direction of influence between two subjects by assigning them the roles of 'Model' and 'Imitator' of specified motor tasks. A total of 22 participants, hence 11 dyads, completed the task in a within-subject design. We adapted concepts from spectral causal-effect decomposition theories to formulate a new measure of the direction and intensity of influence. The results of this study demonstrate that the direction of influence in the fNIRS data of motor tasks can be detected with an Accuracy in the range of 69-88%. Furthermore, the proposed spectral causality measure was shown to significantly reduce spurious causal relationships due to the confounding effects of physiological processes and measurement artifacts compared to time-domain causal analysis.

Graphical Abstract

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Highlights

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- Causality analysis can reveal the direction of influence in fNIRS hyper-scanning
- Experimental setup with a controlled direction of influence in motor imitation tasks
- Time and spectral group causality methods are investigated for directionality testing
- Spectral causality measure showed 69 – 88% Accuracy in directionality detection
- Spectral causality analysis is more robust to measurement and physiological noises

Directionality of Interpersonal Neural Influence in fNIRS Hyperscanning: Validation of a Spectral Causality Approach in a Motor Task

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Abstract

Hyperscanning approaches represent a shift from single- to two-person neuroscience, enabling a more profound understanding of the neural mechanisms underlying interpersonal synchronization. In this context, fNIRS has emerged as a valuable tool for measuring brain activity in a natural, unconstrained environment. While interpersonal synchrony using fNIRS hyperscanning has been well studied using statistical association analysis, establishing causal relationships that elucidate the direction of influence remains challenging. This study aimed to investigate the feasibility of testing the direction of influence in dyadic interactions. Since the ground truth of such direction is not available in a natural setting, we validated our approach in an experimental setup in which we controlled the direction of influence between two subjects by assigning them the roles of 'Model' and 'Imitator' of specified motor tasks. A total of 22 participants, hence 11 dyads, completed the task in a within-subject design. We adapted concepts from spectral causal-effect decomposition theories to formulate a new measure of the direction and intensity of influence. The results of this study demonstrate that the direction of influence in the fNIRS data of motor tasks can be detected with an Accu-

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racy in the range of 69 – 88%. Furthermore, the proposed spectral causality measure was shown to significantly reduce spurious causal relationships due to the confounding effects of physiological processes and measurement artifacts compared to time-domain causal analysis.

Keywords: fNIRS, hyperscanning, interpersonal synchronisation, social interaction; imaging; spectral causality, mutual information decomposition

1 Introduction

Hyperscanning involves the simultaneous recording of brain activity from two or more individuals to determine the temporal relation between both brains (synchronization). Hyperscanning approaches thereby mark a shift from single to two-person neuroscience, allowing a much deeper understanding of the neural mechanisms of interpersonal social interactions [1]. Such research revealed synchronized patterns of brain signals in interacting minds, especially in brain regions involved in social cognition, emotion, and motor control (for an overview, see [2]).

Different hyperscanning measurements such as EEG, fMRI, and functional near-infrared spectroscopy (fNIRS) have been used to investigate interpersonal synchronization during verbal, semi-verbal, and nonverbal interactions [3]. Compared to fMRI and EEG, fNIRS offers significant advantages for monitoring neural activity during natural, unconstrained, real-life interactions. Its high temporal resolution of oxygenation change and its motion tolerance make it particularly valuable for capturing dynamic neural activity in naturalistic settings [4].

While interpersonal synchrony using fNIRS hyperscanning has been well studied using statistical association analysis, e.g., temporal correlation [5] or wavelet coherence [6], establishing causal relationships that elucidate the direction of influence in hyperscanning remains challenging (for an overview, see [3]). This study aimed to go one step beyond the direction-blind statistical association and investigate the feasibility of testing the direction of influence in dyadic interactions using causal discovery methods.

Causal discovery in multivariate time series aims to elucidate the cause-and-effect relationships between variables that evolve over time. The most known classical method is Granger causality (GC) [7]. GC analyzes time series data to determine if one variable can predict future values of another target variable better than using past values of the target variable alone.

30 Recent research has increasingly focused on understanding the synergistic ef-
31 fects of groups of variables acting as a collective subsystem on other groups.
32 This focus is particularly critical in complex systems characterized by in-
33 tricate interdependencies, such as climate-ecosystem interactions and neural
34 activity across distinct brain regions of the same subject [8]. Notable group
35 causality methods are the Trace method [9], the 2GVecCI [10], Vanilla-PC
36 [11] and the Canonical-VAR (MC-VAR) [12]. While these methods operate
37 in the time domain, Faes et al. [8] built on the spectral causality approach
38 of Geweke [13] and proposed a framework based on mutual information rate
39 (MIR) decomposition to assess the interactions among groups of processes,
40 both within specific frequency bands of interest and in the time domain.

41 fNIRS data is often influenced by various sources of noise stemming from
42 measurements and physiological processes, e.g., breathing, heart rate, Mayer
43 waves, etc., [14]. In hyperscanning, these processes typically occur at similar
44 frequency ranges in both participants and can confound the results, leading
45 to spurious associations between participants when using time-domain sta-
46 tistical or causal analysis. Furthermore, the strength of coupling may vary
47 across different frequency bands. To address these challenges, in our study,
48 we adapted the framework of Faes et al., [8] to our problem and then proposed
49 a new measure for the direction and intensity of causal effect relationships
50 in fNIRS data. Since the ground truth of the direction of interpersonal in-
51 fluence is not available in a natural setting, we validated our approach in an
52 experimental setup where we controlled the direction of influence between
53 two subjects. We compared the results of different state-of-the-art group
54 causality methods to the proposed spectral domain causal-effect measure
55 and showed the feasibility of detecting the correct cause-effect direction in
56 fNIRS time series data. To our knowledge, this paper is the first to provide
57 a comprehensive analysis pipeline for identifying the direction of influence in
58 fNIRS data.

59 **2. Materials and Methods**

60 *2.1. Participants*

61 A total of 11 dyads, 22 participants, were recruited from the student
62 population, with a mean age of 23.15 and standard deviation of 2.58. The
63 sample was 21 females and one male. Inclusion criteria required participants
64 to be at least 18 years old and report to be neurologically healthy. Partic-
65 ipants received research participation credits as compensation. The study

66 was conducted following the Declaration of Helsinki and approved by the
 67 ethics review board of the Faculty of Social and Behavioral Sciences of the
 68 University of Jena (FSV 22/063).

69 *2.2. Experimental Design*

70 Participants were invited in dyads to perform a dyadic movement imita-
 71 tion task. In the beginning, each person was assigned to either the role of
 72 Model or Imitator. Both participants were seated opposite each other, so
 73 the Model faced a screen behind the Imitator, invisible to the Imitator. We
 74 presented two 20-second videos on the screen, one showing hand-tapping and
 75 the other foot-tapping. For hand tapping, the video showed a person’s hand
 76 with each finger (excluding the thumb) sequentially tapping on a surface at
 77 a rate of approximately 1.5 Hz. For the foot-tapping task, the video showed
 78 a barefoot tapping on the floor at the same rate. The Model’s task was to
 79 watch the screen and copy the movement with their right hand or foot. The
 80 Imitator’s task was to imitate the movement of the Model.

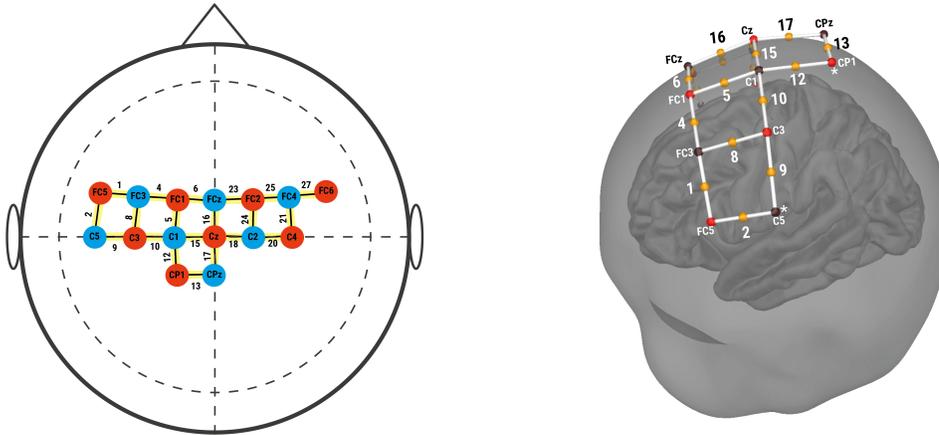


Figure 1: The optodes layout used for fNIRS measurements (2D and 3D views). The brain motor regions are M1 (Channels 12, 13, and 17), PMC (Channels 4, 5, 6, 8, and 16), and PMC/M1 (Channels 10 and 15).

81 A fixation cross was displayed for 60 seconds before each video, serving
 82 as a baseline during which participants were asked not to move. Videos
 83 were presented in a pseudo-randomized order five times each, resulting in
 84 ten trials per Model-Imitator constellation. After a short break, the Model

85 and Imitator switched roles and repeated the experiment with a different
86 stimulus order. The experiment and the video presentation were programmed
87 and controlled using Presentation software (Version 23.0, Neurobehavioral
88 Systems, Inc., Berkley, CA).

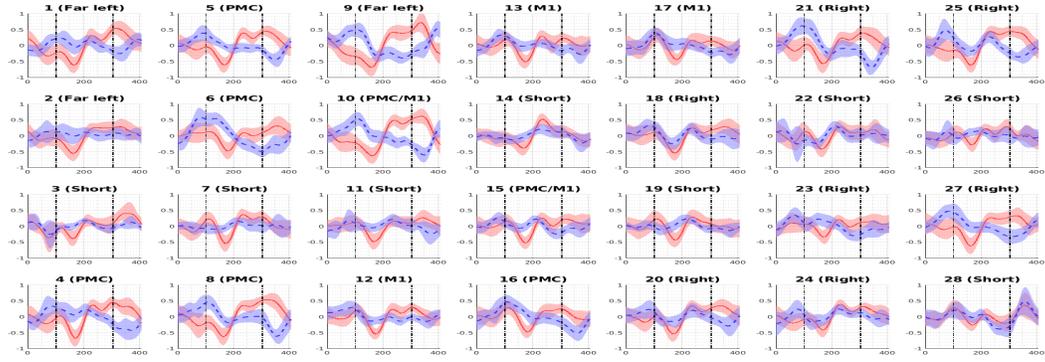
89 *2.3. fNIRS Data Acquisition*

90 Each participant’s cortical hemodynamic activity was recorded using a
91 continuous wave fNIRS system (NIRSport2, NIRx, Germany) with a sam-
92 pling rate of 10.17 Hz and 16 optodes per participant (eight emitters \times eight
93 detectors). Based on a finger- and foot-tapping study by Cockx et al. [15],
94 the optodes were placed to cover the left and right primary motor cortex
95 (M1) and premotor cortex (PMC) (Figure 1) with a distance of 3 cm to
96 allow measurement of cerebral blood oxygenation at 2 to 3 cm depth. Addi-
97 tionally, eight short-distance channels were placed at each emitter position
98 for later offline short-channel correction of non-neuronal signals from long-
99 channel data.

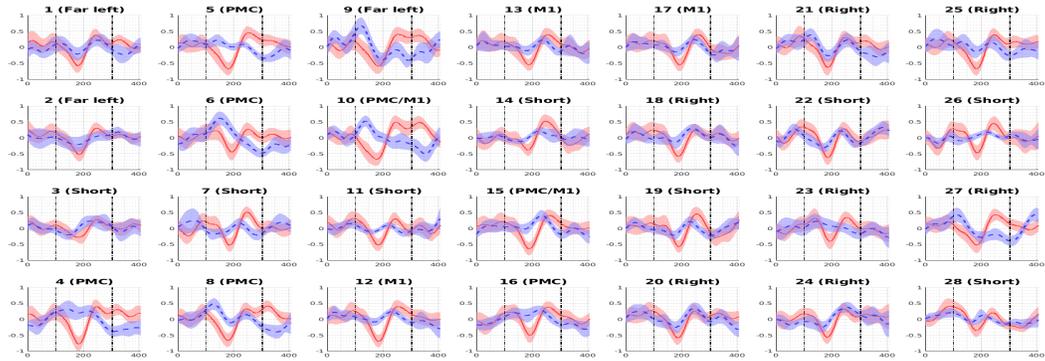
100 *2.4. fNIRS Data Preprocessing*

101 The preprocessing of the fNIRS time series involved the following three
102 steps, which were performed using the *fNIRSFiterPipeline* function of the
103 Homer2 toolbox [16].

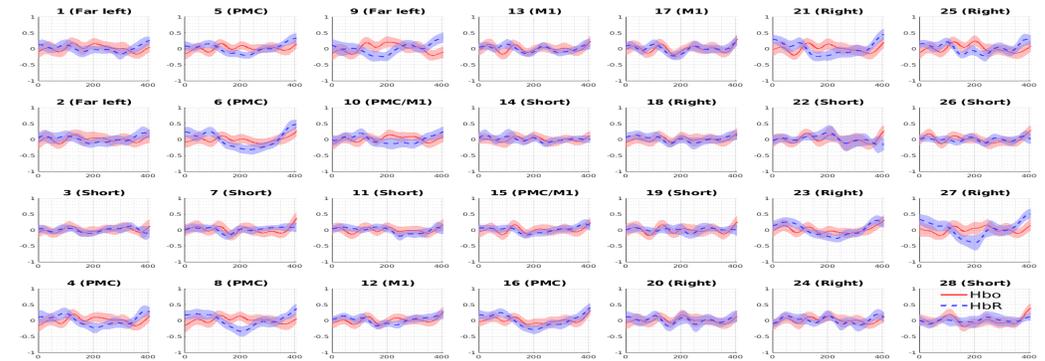
- 104 - Bandpass Filtering: A fifth-order Butterworth bandpass filter was ap-
105 plied with a low cutoff frequency of 0.02 Hz and a high cutoff frequency
106 of 0.15 Hz. The phase of the used filter is almost linear in the pass-
107 band, i.e., all signal components undergo a similar delay, and thus, no
108 influence on causal analysis is expected due to this filtering process.
109 This filtering step removes physiological noise, such as respiratory fluc-
110 tuations (≈ 0.25 Hz), cardiac oscillations (≈ 1 Hz) [3], and slow drifts
111 in the baseline signal, while preserving neural activity in the typical
112 frequency range of interest (≈ 0.029 Hz, depending on the stimulus
113 presentation rate [3]).
- 114 - Hemoglobin Concentration Estimation: Changes in oxy/deoxygenated
115 hemoglobin (HbO/HbR) concentrations were estimated using the mod-
116 ified Beer-Lambert law [17].
- 117 - Normalization: The preprocessed HbO and HbR time series were nor-
118 malized to have zero mean and unit standard deviation.



(a) Hand-Tapping



(b) Foot-Tapping



(c) Baseline

Figure 2: The average and 50% confidence interval of the normalized oxy-/de-oxygenated hemoglobin time series (HbO/HbR) in red/blue color. The time series are averaged for each channel over all dyads and task repetitions (total of 80 intervals) for (a) hand tapping, (b) foot tapping, and (c) baseline tasks. Each subplot spans a 40-second interval (≈ 407 samples). The start and end of the motor task in motor task intervals are marked in vertical lines. For baseline intervals, we show the 40 seconds (407 samples) starting 20 seconds after the end of the motor task.

119 Data quality was assessed in the time domain using the *qualityAssessment*
 120 function within the Homer2 toolbox [16]. A supplementary wavelet-based
 121 visual quality control procedure [6] was implemented before the filtering pro-
 122 cess. Figure 5 in 6 shows examples of good and bad quality HbO signals.
 123 Eight of the eleven dyads exhibited good data quality and were included in
 124 the subsequent analysis.

125 Figure 2 illustrates the preprocessed signals for hand tapping, foot tap-
 126 ping, and baseline intervals. We can see an apparent increase in HbO signal
 127 during motor tasks (Figure 2 (a) and (b)) compared to the baseline condi-
 128 tion (Figure 2 (c)) in the brain motor regions: M1 (Channels 12, 13, and 17),
 129 PMC (Channels 4, 5, 6, 8, and 16), and PMC/M1 (Channels 10 and 15).
 130 Residual periodic fluctuations, likely attributed to Mayer waves, are observ-
 131 able at ≈ 0.1 Hz (\approx two waves in 20 seconds). Some short-distant channels,
 132 such as channels 7 and 14, displayed motor task-related activations. Thus,
 133 employing these channels for noise reduction in the fNIRS time series could
 134 potentially lead to the inadvertent removal of genuine neural activity. Since
 135 our proposed causal intensity measure, detailed in the following section, relies
 136 on the difference in cross-spectral densities of the information flow between
 137 the two participants, we excluded short-distance channels from subsequent
 138 analyses.

139 2.5. The Directionality of Neural Influence: A Spectral Causality Approach

140 To identify the direction of influence between the two participants (Model
 141 and Imitator) within each dyad, we adapted the Spectral Decomposition of
 142 Mutual Information Rate framework (MIR) of [13, 8], hereafter referred to
 143 as Spectr-MIR. In the following, we first provide a brief overview of the
 144 Spectr-MIR method as adapted to our problem and subsequently propose
 145 our definition of the measure quantifying both the intensity and direction of
 146 the causal effect between the Model and the Imitator along with the used
 147 statistical significance test.

148 2.5.1. Spectr-MIR Method

149 Let $X(t_n) \in \mathbb{R}^{L \times 2N}$ be $L \times 2N$ matrix representing N time series of
 150 length L of a specific brain region for both the Model (M) and Imitator (I),
 151 respectively, where $t_n = n\Delta t$ is the time index in iteration n and $\Delta t = 1/f_s$
 152 with f_s the sampling frequency. The matrix $X(t_n)$ can be represented as
 153 the concatenation of the HbO channels of the Model X_M and Imitator X_I
 154 as $X(t_n) = [X_M(t_n) \quad X_I(t_n)]$. The information shared by the two random

155 processes $X_M(t_n)$ and $X_I(t_n)$ per unit of time is defined as the mutual infor-
 156 mation rate (MIR) as follows [18]

$$\text{MIR}_{X_M;X_I} = \lim_{k \rightarrow \infty} \frac{1}{k} \text{MI}(X_M(t_{n-k:n-1}); X_I(t_{n-k:n-1})), \quad (1)$$

157 where $\text{MI}(X_1, X_2)$ denotes the mutual information (MI) shared by the
 158 two variables X_1 and X_2 and defined as

$$\text{MI}(X_1; X_2) = \mathbb{E} \left[\log \frac{p(x_1, x_2)}{p(x_2)p(x_1)} \right]. \quad (2)$$

159 where $p(\cdot, \cdot)$ and $p(\cdot)$ denote joint and marginal probabilities, and \mathbb{E} is the
 160 statistical expectation operator. Using the relation between transfer entropy
 161 and mutual information, it is possible to decompose the MIR into three
 162 components, that is [8, 18]

$$\text{MIR}_{X_M;X_I} = T_{X_M \rightarrow X_I} + T_{X_I \rightarrow X_M} + \text{MIR}_{X_M \cdot X_I}. \quad (3)$$

163 $\text{MIR}_{X_M \cdot X_I}$ represents the instantaneous information shared between X_M and
 164 X_I and $T_{X_i \rightarrow X_j}$ is the entropy transfer from X_i to X_j .

165 Following the methodology of [18, 8], we utilize a state-space modeling
 166 approach to compute all necessary MIR terms. Accordingly, we present the
 167 process $X(t_n)$ as a state space model, i.e.,

$$\begin{aligned} S(t_{n+1}) &= \mathbf{A}S(t_n) + \mathbf{K}W(t_n), \\ X(t_n) &= \mathbf{C}S(t_n) + W(t_n). \end{aligned} \quad (4)$$

168 $S(t_n)$ is the $2N \times p$ state vector of the model, where p is the model
 169 order; \mathbf{A} , \mathbf{C} and \mathbf{K} are the state-space model matrices, and $W(t_n)$ is a
 170 white Gaussian innovation noise vector of zero mean and Covariance ma-
 171 trix $\mathbf{\Sigma}_W = \mathbb{E}[W_n W_n^T]$. Similar to $X(t_n)$, $W(t_n)$ also can be written as
 172 $W(t_n) = [W_M(t_n) \quad W_I(t_n)]$.

173 Taking the Fourier Transform (FT) of the state Equation 4 yields

$$S(\omega) = \mathbf{A}S(\omega)e^{-j\omega} + \mathbf{K}W(\omega)e^{-j\omega}, \quad (5)$$

174 where $S(\omega)$ and $W(\omega)$ are respectively the Fourier transforms of $S(t_n)$
 175 and $W(t_n)$ and ω is the normalized angular frequency. From Equation 5 we
 176 can derive the power spectral density of $X(t_n)$ as $X(\omega) = \mathbf{H}(\omega)W(\omega)$, where

$$\mathbf{H}(\omega) = (\mathbf{I}_{2N \times p} + \mathbf{C}[\mathbf{I}_{2N \times p} - \mathbf{A}e^{-j\omega}]^{-1}\mathbf{K}e^{-j\omega}), \quad (6)$$

177 with \mathbf{I} being the identity matrix. $\mathbf{H}(\omega)$ represents the transfer function relating
 178 the FT of the innovation process $W(t_n)$ to the FT of the process $X(t_n)$
 179 and can be used together with the innovation covariance matrix to derive
 180 the power spectral density (PSD) matrix of the process $X(t_n)$ using spectral
 181 factorization.

$$\mathbf{S}_X(\omega) = \mathbf{H}(\omega)\boldsymbol{\Sigma}_W\mathbf{H}^*(\omega). \quad (7)$$

182 The matrix $\mathbf{S}_X(\omega)$ can be then factorized to get the power spectral den-
 183 sities of X_M and X_I , $\mathbf{S}_{X_M}(\omega)$ and $\mathbf{S}_{X_I}(\omega)$ and the cross-spectral densities
 184 between X_M and X_I , $\mathbf{S}_{X_M X_I}(\omega)$ and $\mathbf{S}_{X_I X_M}(\omega)$. A logarithmic spectral mea-
 185 sure of the interdependence between X_M and X_I is defined by [13].

$$f_{X_I;X_M}(\omega) = \log \frac{|\mathbf{S}_{X_I}(\omega)||\mathbf{S}_{X_M}(\omega)|}{|\mathbf{S}_X(\omega)|}, \quad (8)$$

186 where $f_{X_I;X_M}(\omega)$ is a measure of the total spectral coupling between X_I and
 187 X_M , which, in analogy to the time domain decomposition, can be factorized
 188 into three components.

$$f_{X_I;X_M}(\omega) = f_{X_I \rightarrow X_M}(\omega) + f_{X_M \rightarrow X_I}(\omega) + f_{X_I.X_M}(\omega), \quad (9)$$

189 where $f_{X_{(1)} \rightarrow X_{(2)}}(\omega)$ is a measure of the density of information transferred
 190 from process $X_{(1)}$ to process $X_{(2)}$, and $f_{X_I.X_M}(\omega)$ is the information shared
 191 between the two processes at angular frequency ω . These measures are de-
 192 fined as

$$f_{X_I.X_M}(\omega) = \log \frac{|\mathbf{H}_M(\omega)\boldsymbol{\Sigma}_{W_M}\mathbf{H}_M^*(\omega)||\mathbf{H}_I(\omega)\boldsymbol{\Sigma}_{W_I}\mathbf{H}_I^*(\omega)|}{|\mathbf{S}_X(\omega)|}, \quad (10)$$

$$f_{X_M \rightarrow X_I}(\omega) = \log \frac{|\mathbf{S}_{X_I}(\omega)|}{|\mathbf{H}_M(\omega)\boldsymbol{\Sigma}_{W_M}\mathbf{H}_M^*(\omega)|}, \quad (11)$$

$$f_{X_I \rightarrow X_M}(\omega) = \log \frac{|\mathbf{S}_{X_M}(\omega)|}{|\mathbf{H}_I(\omega)\boldsymbol{\Sigma}_{W_I}\mathbf{H}_I^*(\omega)|}. \quad (12)$$

193 Here, $\mathbf{H}_{(\cdot)}(\omega)$ describes the transfer from $\mathbf{W}_{(\cdot)}$ to $X_{(\cdot)}$ in the frequency domain
 194 and $\boldsymbol{\Sigma}_{W_{(\cdot)}} = \mathbb{E}[W_{(\cdot),n}W_{(\cdot),n}^T]$.

195 In our study, the full state space model, as defined in Equation 4, rep-
 196 represents only the channels of the two regions of interest in the Model and
 197 Imitator and not the channels of all regions in both participants. We justify
 198 our choice by arguing that we are only interested in the inter-dependencies

199 of a specific brain region in both Model and Imitator, regardless of the intra-
 200 dependency of other regions in the same person’s brain. Moreover, focusing
 201 on a specific brain region at a time can benefit from better model fitting due
 202 to lower dimensionality since the intervals of the motor task are only of size
 203 200 samples, which is insufficient to accurately fit a higher dimensionality
 204 model.

205 *2.5.2. Spectral Causal Intensity Measure*

206 Our objective was to measure the intensity and direction of the cause-
 207 effect relationship between a specific region in the brain of the Model and the
 208 same region of the Imitator. Model and Imitator are, in principle, two inde-
 209 pendent entities. In our settings, any bidirectional causality and/or detected
 210 cause-effect during baseline intervals presumably results from some unob-
 211 served factor influencing both participants, such as a physiological process
 212 occurring at the same frequency range, task repetition frequency, or common
 213 noise occurring during signal measurement and acting as a confounder. To
 214 eliminate, as much as possible, any causality due to confounders, we propose
 215 to measure the causal effect of the Model on the Imitator in the frequency
 216 domain at frequency ω as

$$C_{X_M, X_I}(\omega) = f_{X_M \rightarrow X_I}(\omega) - f_{X_I \rightarrow X_M}(\omega) \quad (13)$$

217 *2.5.3. Statistical Significance of Spectr-MIR*

218 To assess the statistical significance of the causal relationships identi-
 219 fied, we use a frequency domain surrogate data method [19]. This approach
 220 preserves the amplitude spectrum of the original HbO time series while ran-
 221 domising the phase information, effectively breaking the temporal depen-
 222 dencies within the data. The following steps are applied to the HbO time
 223 series: 1. Compute the Fourier transform of the original HbO time series. 2.
 224 Replace the original phase of each Fourier coefficient with a random phase
 225 drawn from a uniform distribution between 0 and 2π . 3. Perform the inverse
 226 Fourier transform to produce a surrogate HbO time series. 4. Apply the
 227 same causal inference method described above to the generated surrogate
 228 HbO time series. This procedure is repeated several times to produce an en-
 229 semble of surrogate time series. The spectral causality value of the HbO time
 230 series data is considered significant at a specific frequency only if it exceeds
 231 the spectral causality of the surrogate data at this frequency.

232 *2.6. The Directionality of Neural Influence: Time Domain Causal Analysis*

233 *2.6.1. Spectr-MIR*

234 The time domain causal intensity and direction for the Spectr-MIR method
235 can be obtained by the integration of $C_{X_M, X_I}(\omega)$ over a specific band of fre-
236 quencies from ω_1 to ω_2

$$C_{X_M \rightarrow X_I} = \frac{1}{4\pi} \int_{\omega_1}^{\omega_2} C_{X_M, X_I}(\omega) d\omega \quad (14)$$

237 We define the intensity of the causal effect as the absolute value of $C_{X_M \rightarrow X_I}$,
238 and the direction of the causal effect based on the sign of $C_{X_M \rightarrow X_I}$. Specifi-
239 cally, for $C_{X_M, X_I} > 0$ the direction of influence is from model X_M to Imitator
240 X_I . Otherwise, if $C_{X_M, X_I} < 0$ then the direction of influence is from Imita-
241 tor X_I to Model X_M . For $C_{X_M, X_I} = 0$, we assume there is no causal effect
242 between The Model X_M and Imitator X_I . This definition supports the elim-
243 ination of spurious causal influence between the Model and the Imitator due
244 to noise and physiological processes that the filtering step of preprocessing
245 could not eliminate.

246 *2.6.2. Baseline Time Domain Group Causality Methods*

247 To assess the performance of the proposed time domain causal direction
248 estimation using the Spectr-MIR method, we compare it with the following
249 four state-of-the-art time domain group causality methods.

- 250 - Vanilla-PC Method [11]: A framework for inferring causal directions
251 between groups of variables by applying a series of conditional inde-
252 pendence tests.
- 253 - Trace method [9]: This method infers whether linear relations between
254 two high-dimensional variables X and Y are due to a causal influence
255 from X to Y or from Y to X .
- 256 - 2GVecCI [10]: A non-parametric approach for inferring the causal re-
257 lationship between two vector-valued random variables from observa-
258 tional data based on a series of conditional independence tests.
- 259 - Canonical Granger Causality method (MC-VAR) [12]: This method
260 combines ideas from canonical correlation and Granger causality anal-
261 ysis to yield a measure that reflects directed causality between two
262 regions of interest using optimized linear combinations of signals from
263 each region of interest to enable accurate causality measurements.

264 *2.6.3. Evaluation Metrics*

265 To validate the performance of time domain group causal analysis meth-
266 ods, we calculated the following metrics: Accuracy, false positive rate (FPR),
267 true positive rate (TPR), and F-Score. TPR presents the ratio of intervals
268 of a specific motor task of all dyads and task repetitions where the causality
269 direction is correctly detected from Model to Imitator. Meanwhile, FPR is
270 the ratio of intervals for a specific motor task of all dyads and task repeti-
271 tions where the causality direction is falsely detected from the Imitator to
272 the Model. Accuracy is the ratio of intervals where the causal link from the
273 Model to the Imitator in motor tasks and the absence of causal link in the
274 baseline intervals are correctly predicted. The F-score focuses more on the
275 correctly detected links in motor task intervals. Formally, these metrics are
276 defined as follows.

$$\text{Accuracy} = \frac{\text{TP}+\text{TN}}{\text{TP}+\text{FP} + \text{TN}+\text{FN}} \quad (15)$$

$$\text{FPR} = \frac{\text{FP}}{\text{FP} + \text{TN}} \quad (16)$$

$$\text{TPR} = \frac{\text{TP}}{\text{FP} + \text{TN}} \quad (17)$$

$$\text{F-score} = \frac{\text{TP}}{\text{TP} + 0.5(\text{FP} + \text{FN})} \quad (18)$$

277 Here, TP is the number of trials where the correct direction from Model to
278 Imitator is detected; TN is the number of trials where no causal link from
279 Model to Imitator or from Imitator to Model is correctly detected in baseline
280 intervals. FP is the number of baseline intervals where a causal link is falsely
281 detected; FN is the number of motor task intervals where no causal link is
282 detected in either direction. Accuracy is our primary metric for evaluation,
283 but other metrics help better understand the methods' overall performance.

284 **3. Results**

285 *3.1. Spectral Causal Analysis Results*

286 To evaluate the performance of the Spectr-MIR method in the frequency
287 domain, we applied the method for each motor task and for each of the
288 brain motor regions of interest, namely M1 (Channels 12, 13, and 17), PMC
289 (Channels 4, 5, 6, 8, and 16), and PMC/M1 (Channels 10 and 15) separately.

290 As we noted a delay in the activation of HbO in response to the motor task,
291 we chose to work with an interval length of 224 samples, which is equal to the
292 samples of task interval ($20seconds \times 10.17Hz \approx 204$ samples) and a slight
293 shift of 10 samples (one-second of data) before and after the start and end of
294 the motor task respectively. The spectral causality components (Equation 9)
295 are calculated for each interval and each dyad and then averaged over all
296 dyads and the ten repetitions of the same motor task.

297 In all our experiments, we used the *oir_mir* function of the Matlab toolbox
298 of Faes et al., [8] to calculate the different components of spectral causality
299 with adaptation to our definition of the state-space model as detailed in
300 Section 2.5.1. Experimental results for the spectral group causality analysis
301 using the Spectr-MIR method and for different brain regions are shown in
302 Figure 3. These results indicate that the average spectral causality from
303 Model to Imitator is higher than from Imitator to Model in hand-tapping
304 and foot-tapping in almost all brain regions of interest. However, in baseline
305 intervals of all brain regions, we see almost equal spectral causality in both
306 directions. The statistically significant spectral causality in both directions
307 during baseline intervals can be attributed to the confounding effect of the
308 task repetition frequency as well as the confounding effect of measurement
309 and physiological processes. The causal intensity at a specific frequency can
310 be measured as defined by Equation 13 or directly from the difference between
311 the green and orange lines.

312 Region-wise, we can notice in Figure 3 a higher causal intensity from
313 Model to Imitator in motor task intervals in M1 and PMC/M1 regions com-
314 pared to PMC. The hand-tapping intervals have a higher average causal
315 intensity in the PMC/M1 region, while the causal intensity due to the foot-
316 tapping task is higher in the M1 region. This difference in causal intensity
317 between regions could be because the PMC/M1 covers more lateral parts,
318 M1 covers more central parts, and the hand region is better represented in
319 the lateral areas than the foot region [20].

320 3.2. Time-Domain Causal Analysis Results

321 In this section, we compare the Accuracy of estimating the direction of
322 influence using the time domain Spectr-MIR (Section 2.6.1) with the time-
323 domain group causality baseline methods described in Section 2.6.2: Trace
324 methods [9], Vanilla PC [11], MC-VAR [12] and 2GVecCI [10].

325 The time domain causal intensity and direction for the Spectr-MIR using
326 the integral of Equation 14 is calculated to include only statistically signifi-

327 cant values in the frequency range 0.02-0.15 Hz. The statistical significance
328 for each dyad was estimated using the frequency surrogate data method de-
329 scribed in Section 2.5.3 averaged over 10 surrogates.

330 Results are shown in Figure 4 for the brain regions PMC, M1, and
331 PMC/M1. As a performance measure, we show the Accuracy and the false
332 positive rate (FPR) in Figure 4, while the True Positive Rate (TPR) and
333 F-score are shown in Figure Figure 6 in section 6.

334 For the foot tapping task, the best result of Spectr-MIR is from the M1
335 region (Accuracy= 88%, FPR = 0), and the worst is from the PMC region
336 (Accuracy= 69%, FPR=0.08). On the other hand, for the hand tapping
337 task, the best results are from the PMC/M1 region (Accuracy= 82%, FPR
338 = 0), and the worst is from the M1 regions (Accuracy= 63%, FPR = 0.02).
339 These ROI-wise results are consistent with similar differences in causal in-
340 tensity results in PMC/M1 and M1 regions, as discussed in Section 3.1. For
341 baseline intervals, the absence of causality is best detected in the M1 region
342 (Accuracy= 88%, FPR=0.06) and worst in the PMC/M1 region (Accuracy=
343 75%, FPR = 0.12).

344 On average, Accuracy is higher for the foot-tapping task than the hand-
345 tapping task. The higher Accuracy for foot tapping is probably due to a
346 longer delay in HbO activation between the Model and the Imitator. As
347 noted earlier, Figure 2 shows that the reaction time is longer for the foot
348 than for the hand. This longer delay made it easier for causality methods to
349 detect who leads (the cause) and who follows (the effect).

350 In almost all brain regions of interest, Spectr-MIR has higher accuracy
351 and lower FPR than all other methods, followed by the Vanilla-PC method.
352 The MC-VAR and Trace methods suffer from high FPR. The low FPR of the
353 proposed causal intensity measure, based on the subtraction of the Spectr-
354 MIR spectral causality $f_{X_M \rightarrow X_I}(\omega)$ from $f_{X_I \rightarrow X_M}(\omega)$, allowed the removal of
355 spurious causal effects that could be attributed to measurement artifact or
356 physiological processes.

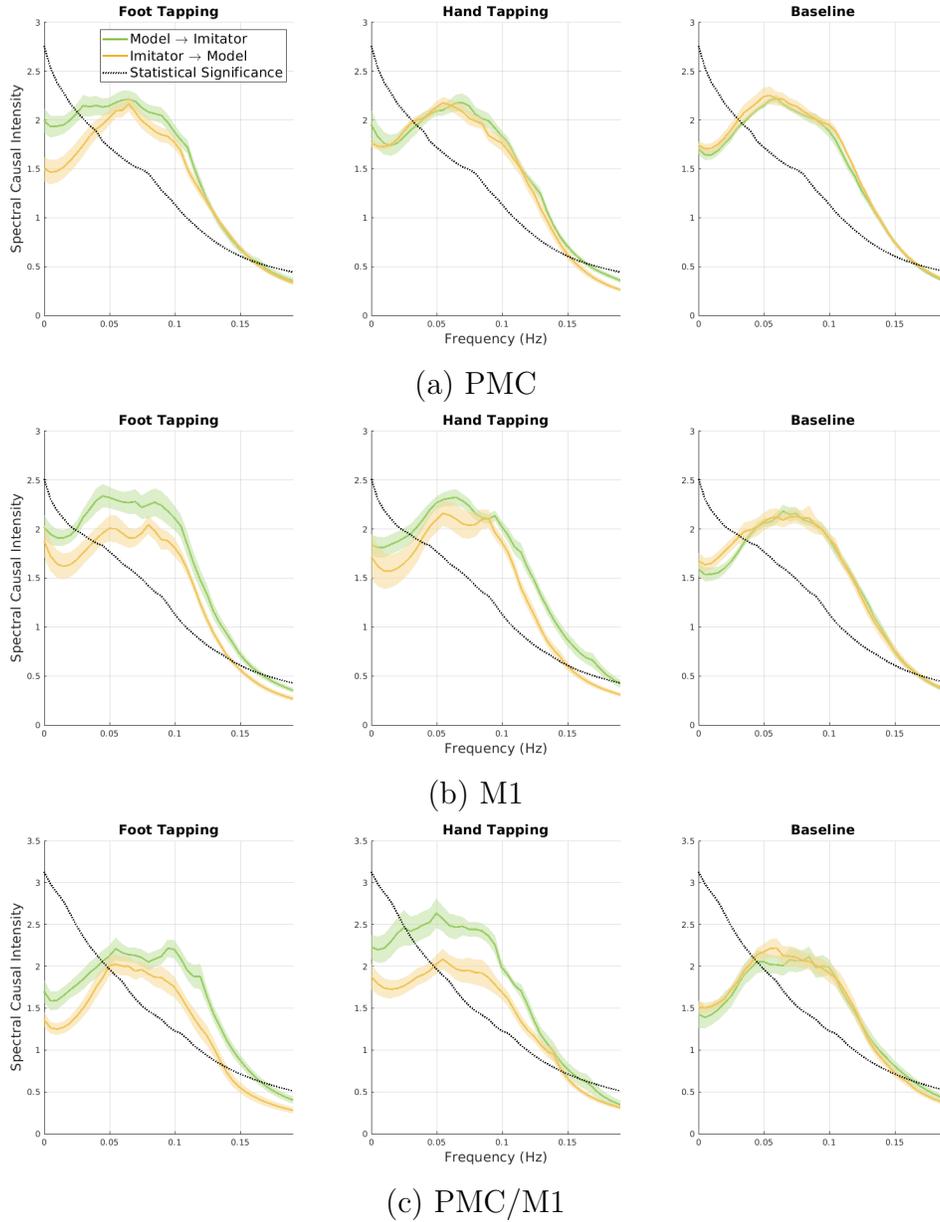


Figure 3: The average and 50% confidence interval of the spectral causality of the normalized HbO time series from Model to Imitator (green plots) and from Imitator to Model (orange plots). The average is calculated for each type of event of all dyads for regions: (a) Premotor cortex (PMC), (b) Primary motor cortex (M1), and (c) PMC/M1. The statistical significance is shown in the dotted black line, which is the average spectral causality of the frequency domain surrogate data. Only spectral causality values higher than this line are considered statistically significant.

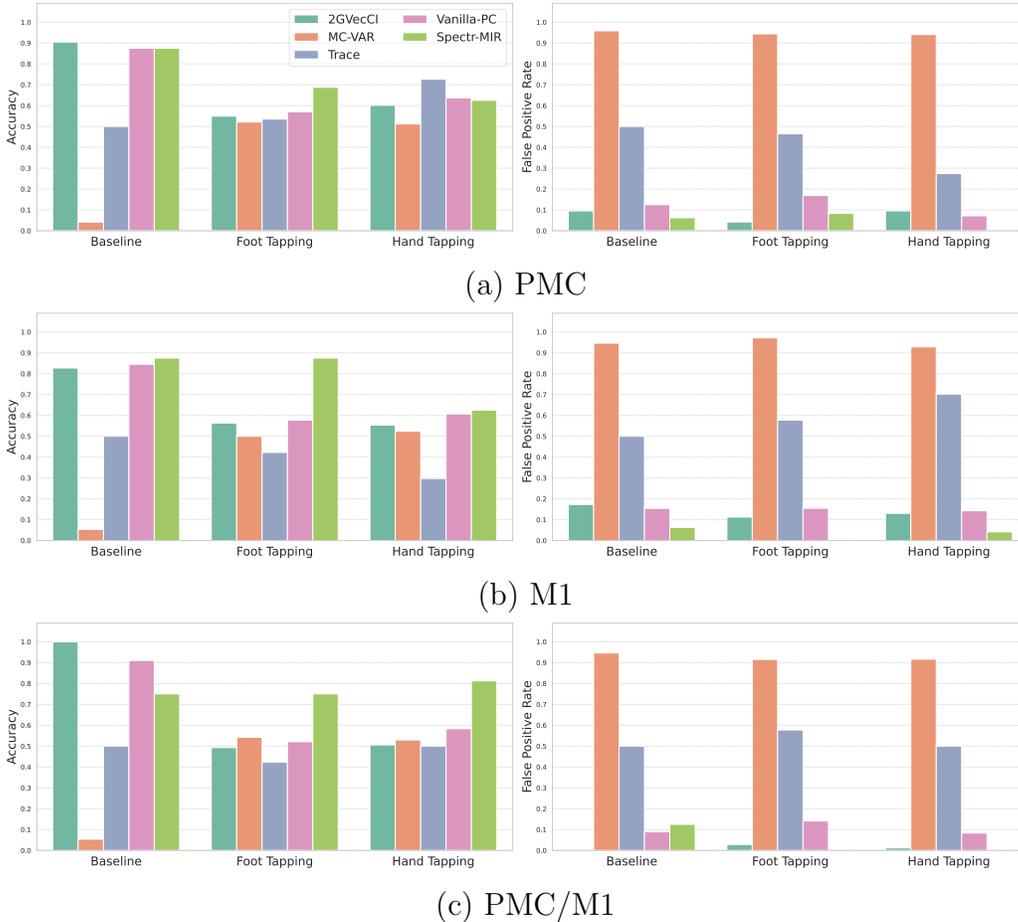


Figure 4: Results of the time domain cause-effect analysis of the normalized HbO time series using the Spectr-MIR method in comparison with four baseline methods: Vanilla-PC [11], 2GVecCI [10], Trace [9], and MC-VAR [12]. The left column shows the Accuracy (higher is better) for the three different regions: (a) premotor cortex (PMC), (b) primary motor cortex (M1), and (c) PMC/M1. The right column shows the false positive rate (FPR) for the same methods and regions (lower is better).

357 3.3. Discussion

358 Both spectral and time domains causal analysis showed that causality
 359 can be accurately derived from fNIRs hyperscanning of motor tasks. Our
 360 causality measure achieved 69 – 88% Accuracy in detecting the causal in-
 361 fluence from Model to Imitator. Moreover, the proposed causality measure
 362 significantly reduced the false positive rate compared to time domain base-
 363 line causality methods (Figure 4). This reduction indicates that the proposed

364 measure enabled the removal of spurious causal effects that might result from
365 task repetition, measurement, or physiological processes. The feasibility of
366 detecting causality in fNIRS was not only significantly above chance but
367 also significantly higher than the Accuracy of four other tested time domain
368 methods. The average spectral cause-effect plots in Figure 3 showed a higher
369 causality from the Model to Imitator than Imitator to Model for hand- and
370 foot-tapping for all regions of interest. Our paradigm was not ambiguous in
371 who leads and who follows in the interaction and was consistent with 10 very
372 well-defined onset blocks of a standardized motor test typically evoking large
373 responses. The motor imitation task deemed us a good testing paradigm, as
374 it allows for valid control of causality - as only the Model saw the instruction,
375 the Imitator’s movement was dependent on the Model. The extracted HbO
376 time series supported the validity of the task: The motor tasks led to an
377 evident signal rise in the expected brain areas. We see evident activation of
378 the averaged HbO time series during the motor tasks compared to baseline
379 (Figure 2). The signal maximum occurred with a delay of about 10 seconds,
380 in line with the temporal evolution of the hemodynamic response function
381 [21], and was faster for the hand than the foot. In the natural setting of
382 fNIRS paradigms, such as cooperation tasks, data onset is less defined, and
383 hence, we expect a worse signal-to-noise ratio for the detection of causal rela-
384 tions. The feasibility of our method for various natural settings needs further
385 investigation.

386 4. Conclusion

387 This study aimed to test whether the direction of influence in dyadic in-
388 teraction can be derived from fNIRs hyperscanning. To this end, we amended
389 the frequency domain mutual information rate decomposition frameworks of
390 Geweke [13] and Faes et al., [8] to fNIRS data of the motor imitation task.
391 We then defined a measure for the direction and intensity of neural influence
392 in frequency and time domains and compared the performance of this mea-
393 sure with four state-of-the-art time domain causal analysis methods. Our
394 study showed that detecting the direction and intensity of neural influence
395 is feasible based on fNIRs data. The usability of the proposed approach in a
396 natural or uncontrolled setting might face new challenges that need further
397 investigation. The varying magnitude and temporal delay in HbO activation
398 in response to different tasks make it more challenging for the causal discov-
399 ery methods to detect the correct cause-effect patterns in a natural setting

400 where several motor or non-motor tasks might occur simultaneously. We
401 argue that using multimodal imaging or different sources of information is
402 vital for causal discovery in fNIRS hyperscanning. Including emotional influ-
403 ence analysis using facial expressions in dyadic interaction [22], body-part-
404 tracking, or verbal signal analysis as additional synchronization measures
405 might support the validation of hyperscanning measurements-based causal
406 discovery results.

407 5. Acknowledgment

408 This research is funded by the Federal Ministry of Education and Research
409 (BMBF) project 01EW2105: "DZPG development funding – 'Jena location'
410 – CIRC - (Mal)adaptive brain circuits of social interaction, FSU" as well
411 as the German Research Foundation (DFG) individual research grant SH
412 1682/1-1.

413 6. Conflicts of Interest

414 The authors declare no conflicts of interest.

415 References

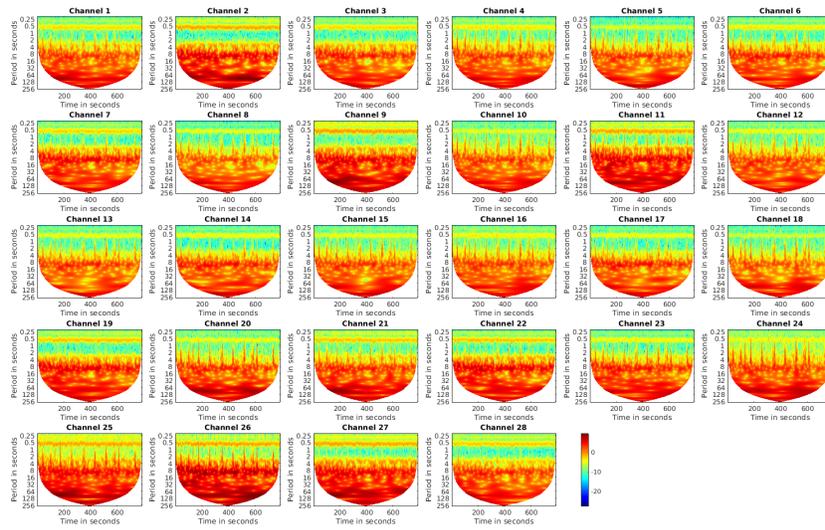
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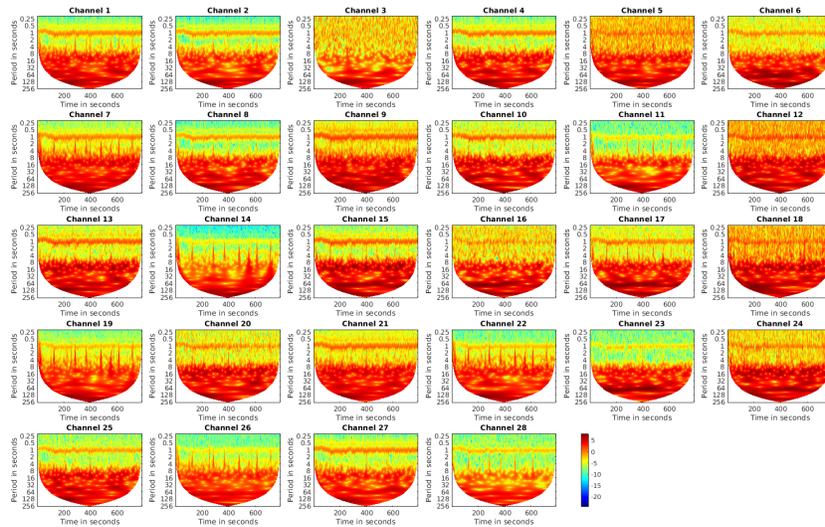
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504 Appendix A: fNIRS data visual quality check using Wavelet trans-
 505 form

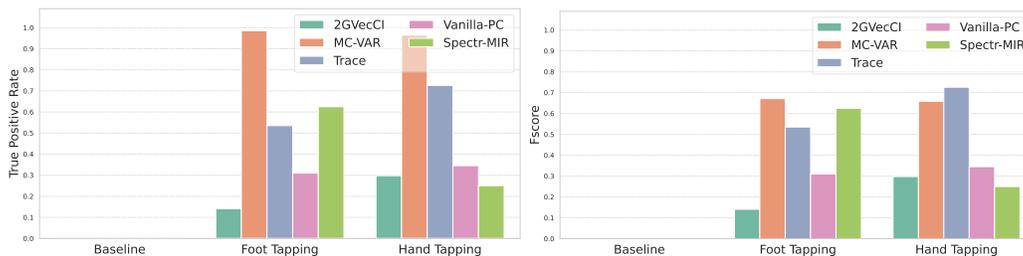


(a) Good quality data

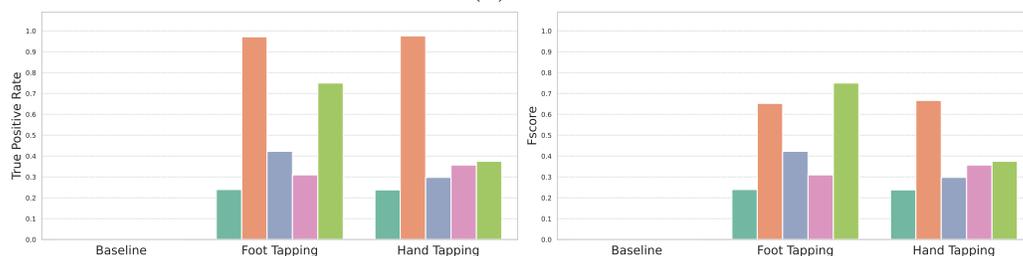


(b) Bad quality data

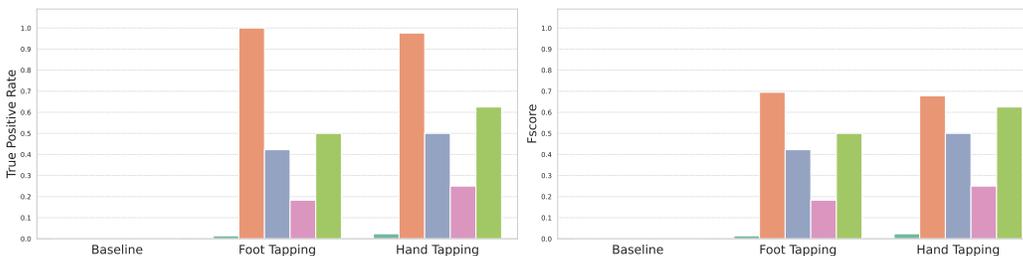
Figure 5: fNIRS data visual quality check using Wavelet transform plots before any filtering: (a) a good quality data with a clear separation between the heart rate at the period of $\approx 1\text{sec}$ and the lower frequencies (higher periods) and (b) a bad quality data where high wavelet values occur at all frequencies and all time samples.



(a) PMC



(b) M1



(c) PMC/M1

Figure 6: The true positive rate (TPR) (left) and F1-score (right) of the time domain cause-effect analysis results for the normalized HbO time series using the Spectr-MIR method in comparison with four baseline methods: Vanilla-PC [11], 2GVecCI [10], Trace [9], and MC-VAR [12] for the three different regions: (a) Primary Motor Cortex (PMC), (b) Motor Cortex (M1), and (c) PMC/M1. MC-VAR shows high TPR and high FPR (Figure 4), indicating that this method detects causal links everywhere.