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Faculty of Psychology

MASTER THESIS PROPOSAL

Presented to outline the general concept of the master's thesis in the field of social cognition within the Cognitive Science study program at Ruhr-University Bochum

by the student

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Thesis Title:

Neural-Autonomic Synchrony and Embodied Integration

Temporal Binding of Frontal-Parietal Networks and Autonomic Responses in Conscious Emotional Processing

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List of Acronyms

ANOVA	Analysis of Variance
ANS	autonomic nervous system
BAS	Behavioural Approach System
BIS	Behavioural Inhibition System
DLPFC	dorsolateral prefrontal cortex
ECG	Electrocardiography
EDA	Electrodermal Activity
EEG	Electroencephalography
FAI	frontal asymmetry index
fNIRS	Functional Near-Infrared Spectroscopy
GLM	General Linear Model
GSR	Galvanic Skin Response
HbO2	Oxyhemoglobin
HbR	Deoxyhemoglobin
HRV	Heart Rate Variability
ICA	Independent Component Analysis
NN intervals	Time intervals between consecutive R-peaks
PANAS	Positive and Negative Affect Schedule
PFC	prefrontal cortex
PLV	Phase locking value
RMSSD	Root Mean Square of Successive Differences
ROI	regions of interest
SAM	Self-Assessment Manikin
SCL	Skin Conductance Level
SCR	Skin Conductance Responses
TDDR	temporal derivation distribution repair
VMPFC	ventromedial prefrontal cortex

1 Introduction

The human experience is profoundly shaped by emotions, which orchestrate adaptive responses to environmental challenges and opportunities. Not purely mental events, emotions manifest as complex, integrated states involving subjective feelings, cognitive appraisals, behavioral expressions, and significant physiological changes mediated by the central and autonomic nervous system (ANS) (Barrett et al., 2016; Kreibig, 2010). Understanding the dynamic interplay between neural processing and autonomic physiology is fundamental to emotion science (Critchley, 2005). This brain-body interaction is not merely epiphenomenal; the perception and regulation of bodily states (interoception) actively contribute to emotional feeling and decision-making (Antonio Damasio, 2005). Furthermore, disruptions in this neuro-autonomic dialogue, such as altered heart rate variability or electrodermal hypo/hyper-reactivity, are increasingly recognized as core features of affective disorders like anxiety and depression, underscoring the clinical relevance of investigating these mechanisms (Beauchaine, 2001; Thayer et al., 2009).

Within the brain, the prefrontal cortex (PFC) is critical for orchestrating emotional responses, integrating sensory information with internal goals and modulating subcortical affective circuits (Fuster, 2008). A prominent theoretical framework focuses on hemispheric asymmetry in PFC activity. The motivational direction model posits that relatively greater left PFC activation supports approach-related motivation and positive affect, while relatively greater right PFC activation underlies withdrawal-related motivation and negative affect (Davidson, 2004; Harmon-Jones & Allen, 1996). This asymmetry is not static but dynamically modulated by emotional context and individual predispositions, such as trait approach/avoidance tendencies captured by the Behavioural Inhibition System (BIS) and Behavioural Approach System (BAS) scales (Carver & White, 1994; Rodrigues et al., 2018; Sutton & Davidson, 1997). The effectiveness of emotional induction methods can also influence the observed asymmetry, suggesting that more engaging stimuli may elicit stronger, more differentiated neural responses (Rodrigues et al., 2021).

While the PFC orchestrates central processing, the emotional response simultaneously engages the body via the ANS, adjusting physiology to meet situational demands. Different physiological signals offer complementary views of autonomic activity. Heart Rate Variability (HRV), the analysis of beat-to-beat fluctuations in heart rate derived from Electrocardiography (ECG), provides a non-invasive window into ANS function (Berntson et al., 1997; Malik et al., 1996). Short-term metrics like Root Mean Square of Successive Differences (RMSSD) primarily reflect parasympathetic (vagal) control (Malik et al., 1996; Shaffer & Ginsberg, 2017). Higher vagally-mediated HRV (indexed by RMSSD) generally reflects greater autonomic flexibility and adaptive capacity, linked to effective emotional regulation, while reduced HRV is linked to stress, psychopathology, and impaired executive function (Thayer et al., 2010). In contrast, sympathetic activity can be captured using Electrodermal Activity (EDA) (Galvanic Skin Response (GSR)), which measures changes in skin conductance driven by sympathetically-innervated sweat glands (Boucsein, 2012; Dawson et al., 2007). Phasic increases (Skin Conductance Responses (SCR)) reflect event-related sympathetic bursts, while the tonic Skin Conductance Level (SCL) indicates general arousal (Boucsein, 2012). Capturing both parasympathetic (RMSSD) and sympathetic (EDA) influences allows comprehensive assessment of the body's response during emotion.

The perception of emotion itself is also debated. While traditional appraisal theories emphasize cognitive interpretation as the primary determinant of emotional experience (Scherer et

al., 2001), alternative perspectives like the direct perception account suggest a more immediate recognition of emotional significance based on characteristic patterns of features, akin to perceptual object recognition (Newen et al., 2015). This view aligns well with theories of embodied cognition, which posit that cognitive processes, including emotional understanding, are grounded in the body's sensorimotor and physiological states (Niedenthal et al., 2009). From this perspective, the coordinated activity between neural circuits (like the PFC) and autonomic responses (reflected in RMSSD and EDA) is not just a correlate but an integral part of the emotional percept and experience (Critchley, 2005). Understanding how these systems dynamically couple (synchronize) over time is key to understanding the emergence of subjective feeling states.

Investigating this embodied perspective, where coordinated brain-body activity is integral to emotion, necessitates analytical approaches capturing the temporal coordination (synchrony) between brain and body signals. Neural-autonomic synchrony reflects the functional coupling between central neural oscillations and peripheral physiological rhythms (Thayer et al., 2009). Quantifying this synchrony reveals how effectively these systems interact during emotional challenges. The Phase locking value (PLV) is a robust method for this purpose (Lachaux et al., 1999). PLV measures the consistency of the phase difference between two time series, irrespective of their amplitudes. A high PLV indicates consistent phase alignment, suggesting a stable functional interaction. We will calculate PLV between the EEG signal from the PFC and two different autonomic signals: separately with a continuous HRV-derived signal (reflecting parasympathetic influence) and with a continuous EDA-derived signal (reflecting sympathetic influence). Compared to coherence measures, which are sensitive to both phase and amplitude, PLV's focus on phase makes it particularly adept at detecting transient, potentially non-linear synchronization patterns characteristic of dynamic biological systems responding to stimuli (Cohen, 2014; Valenza et al., 2014). The proposed theoretical integration is depicted in Figure 1.

The present study aims to elucidate neural-autonomic synchrony during the conscious processing of distinct emotional states elicited by validated affective stimuli. We adopt a multimodal approach, integrating high-temporal-resolution EEG to capture rapid prefrontal neural dynamics, ECG to derive measures of primarily parasympathetic autonomic control (RMSSD), EDA to reflect sympathetic sudomotor control, and fNIRS for complementary spatial localization (Pinti et al., 2019; Scholkmann et al., 2014). This combination allows investigating network dynamics while focusing on EEG's temporal resolution for synchrony, ECG/EDA for specific autonomic branches, and fNIRS to localize activity in key prefrontal (emotion regulation) and parietal (attention, bodily signal integration) regions (Critchley, 2005; Pessoa, 2008). While technically demanding, this integrated approach is necessary to investigate how precisely timed brain-body coupling relates to activity within specific neural substrates. We acknowledge that linking the hemodynamic response measured by fNIRS to the specific neural activity whose phase is relevant for autonomic coupling relies on the assumption that significant hemodynamic changes reflect underlying neural engagement relevant to the synchrony analysis, despite the complex and indirect nature of neurovascular coupling (Logothetis & Wandell, 2004). The fNIRS data, therefore, serve not just as post-hoc context, but as a means to spatially constrain and interpret the functional significance of observed EEG-based synchrony patterns, as detailed in the analysis plan. We will utilize standardized, emotionally evocative video clips (positive, negative, neutral) from the E-MOVIE database, validated by Maffei and Angrilli (2019) for their efficacy in inducing targeted affective states. By calculating PLV between prefrontal EEG oscillations (specifically in the alpha [8-13 Hz] and beta [13-30 Hz] bands, which are broadly implicated in cortical processing, attention, and potentially emotional regulation (Allen

et al., 2004; Klimesch, 1999; Koenig et al., 2002)) and relevant autonomic signals (including continuous signals derived from HRV data reflecting vagal activity for brain-heart coupling and continuous phasic EDA-derived signals for brain-sudomotor sympathetic coupling), we will investigate how brain-body interactions involving both ANS branches vary with emotional content and intensity.

Given these considerations, we hypothesize:

(a) **Emotional Modulation of Synchrony:** We predict that neural-autonomic synchrony (PLV) will be significantly enhanced during the processing of both positive and negative emotional stimuli compared to neutral stimuli. We will investigate this using PLV calculated between prefrontal EEG and both continuous HRV-derived signals (reflecting brain-heart parasympathetic coupling) and EDA-derived signals (reflecting brain-sudomotor sympathetic coupling). Rationale: Emotional engagement necessitates greater integration between central command centers and peripheral effectors (cardiac and sudomotor) to prepare for potential action and adapt internal milieu; this heightened integration is expected to manifest as stronger phase coupling involving both ANS branches (Critchley, 2005). While we predict overall enhancement, we will also exploratorily examine whether the nature or dominant branch of synchrony (e.g., EEG-HRV vs. EEG-EDA) differs between positive and negative valence conditions.

(b) **Synchrony and Subjective Arousal:** We predict a positive correlation between the magnitude of neural-autonomic synchrony and subjective ratings of emotional arousal (via the Self-Assessment Manikin (SAM)) specifically during the positive and negative conditions. This correlation will be tested for both PLV using continuous HRV-derived signals (parasympathetic coupling) and PLV using EDA-derived signals (sympathetic coupling). Rationale: Higher subjective arousal reflects a more intense emotional state, likely involving stronger reciprocal signaling and tighter coupling between the brain's assessment and the body's physiological mobilization. This mobilization involves coordinated adjustments in cardiovascular and sudomotor control (potentially reflected in EEG-HRV or EEG-EDA synchrony), leading to more consistent phase relationships between central and peripheral systems (Boucsein, 2012; Valenza et al., 2014).

(c) **Baseline Vagal Tone and Task-Related Synchrony:** We predict that individual differences in baseline parasympathetic regulation, indexed by resting-state RMSSD, will be positively associated with the degree of neural-autonomic synchrony (PLV calculated specifically between EEG and continuous HRV-derived signals) exhibited during the processing of negative emotional stimuli. Rationale: Individuals with higher baseline RMSSD, indicative of greater vagal regulatory capacity (Shaffer & Ginsberg, 2017; Thayer et al., 2010), may be better equipped to dynamically coordinate neural and cardiac activity when facing demanding or threatening situations, resulting in more robust task-related brain-heart phase synchrony.

(d) **Frontal Asymmetry and Branch-Specific Synchrony:** We predict that the direction of prefrontal cortical asymmetry, indexed by alpha band [8-13 Hz] power differences (e.g., F3 vs. F4), may be differentially associated with the strength of phase synchrony involving distinct autonomic branches. Specifically, we will exploratorily investigate whether greater relative right PFC activation (associated with withdrawal/inhibition) correlates with stronger EEG-HRV PLV (parasympathetic coupling), and whether greater relative

left PFC activation (associated with approach/action) correlates with stronger EEG-EDA PLV (sympathetic coupling), particularly during emotional conditions. Rationale: This hypothesis explores whether the functional specialization suggested by the motivational direction model (Davidson, 2004; Harmon-Jones & Allen, 1996), typically assessed via power measures, extends to the domain of phase coupling with the autonomic branch potentially most relevant to that motivational state. We acknowledge this is an exploratory step, bridging asymmetry (power) and synchrony (phase) concepts, aimed at generating hypotheses about potential underlying mechanisms linking cortical motivational states to specific patterns of brain-body interaction.

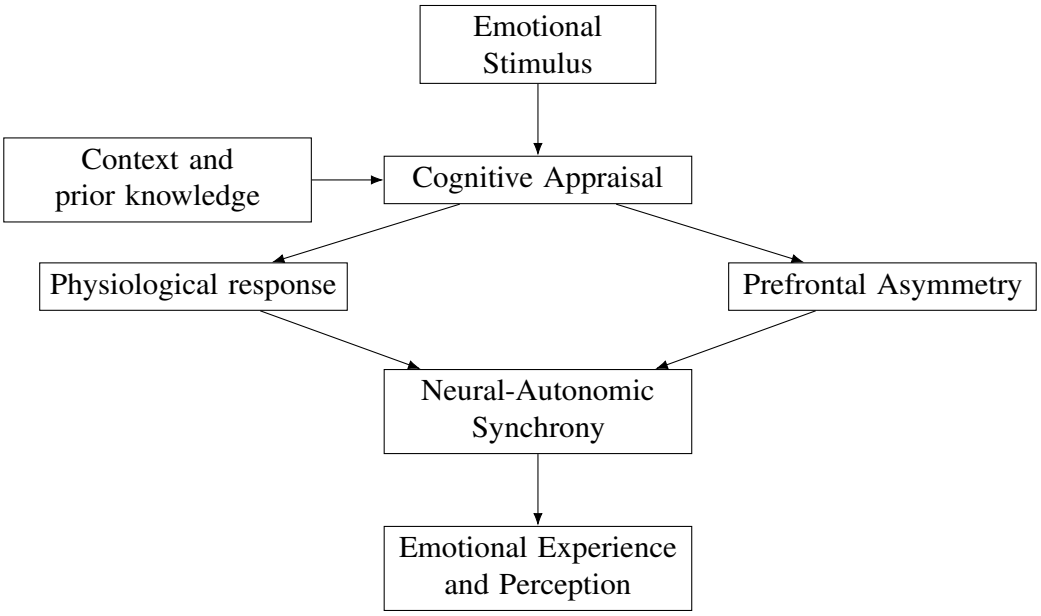

Figure 1: Neural-Autonomic Synchrony in Emotional Experience.

This diagram illustrates the proposed model of emotional processing, highlighting the interplay between neural and physiological components. An emotional stimulus initiates cognitive appraisal, influenced by contextual factors. Simultaneously, the stimulus elicits a physiological response. Cognitive appraisal leads to prefrontal asymmetry, a key neural component, which interacts with the physiological response through neural-autonomic synchrony. This synchronized activity culminates in the emotional experience.

2 Methods

2.1 Electroencephalography

EEG measures electrical brain activity via scalp electrodes, capturing voltage fluctuations from synchronized postsynaptic potentials primarily in cortical pyramidal neurons (Chaddad et al., 2023; Sharma & Meena, 2024). Its excellent temporal resolution (milliseconds) is crucial for capturing the rapid neural dynamics necessary for phase synchrony analysis (PLV) with physiological signals (Cohen, 2014). While spatial resolution is limited by volume conduction, EEG allows tracking oscillatory activity in specific frequency bands linked to cognitive and affective states. In this study, we focus on the Alpha (8-13 Hz) band, often associated with cortical inhibition, attentional processes, and emotional valence processing (particularly in frontal asymmetry contexts (Allen et al., 2004)), and the Beta (13-30 Hz) band, implicated in sensorimotor processing, maintaining the current cognitive set, and potentially arousal states (Engel et al., 2001; Klimesch, 1999). EEG will thus provide the primary high-resolution neural signal for assessing frontal asymmetry (via alpha power) and calculating PLV (using phase information from both alpha and beta bands).

2.2 Functional Near-Infrared Spectroscopy

fNIRS measures cerebral hemodynamic changes (Oxyhemoglobin (HbO2), Deoxyhemoglobin (HbR)) linked to neural activity using near-infrared light (Jöbsis, 1977; Obrig & Villringer, 2003; Scholkmann et al., 2014). This linkage, known as neurovascular coupling, reflects the increased metabolic demand and subsequent blood flow changes in active brain regions. Offering spatial information complementary to EEG (Pinti et al., 2019), fNIRS allows localizing hemodynamic correlates of neural activity within specific cortical regions. In this study, it will be used to identify prefrontal regions of interest (ROI) showing significant task-related activation, which will then inform the analysis of EEG-autonomic synchrony originating from those functionally defined areas.

2.3 Electrocardiography and Heart Rate Variability

ECG records the heart's electrical activity, allowing derivation of HRV by analyzing beat-to-beat variations (Time intervals between consecutive R-peaks (NN intervals)), which reflect ANS modulation of heart rate (Berntson et al., 1997; Malik et al., 1996). We will focus on the time-domain metric RMSSD, calculated as:

$$\text{RMSSD} = \sqrt{\frac{1}{N-1}\sum_{i=1}^{N-1}(NN_{i+1} - NN_i)^2} \tag{1}$$

where NN_i is the i-th interval and N is the number of intervals. RMSSD is sensitive to rapid, high-frequency fluctuations in heart rate and primarily reflects parasympathetic (vagal) influence on the heart (Malik et al., 1996; Shaffer & Ginsberg, 2017). Higher resting RMSSD generally indicates greater vagal tone and autonomic flexibility (Thayer et al., 2010). For the synchrony analysis, a continuous signal representing vagal influence will be derived from the NN intervals (e.g., via interpolation) to compute PLV with EEG signals.

2.4 Electrodermal Activity

EDA (GSR) measures skin conductance changes reflecting sympathetic output from eccrine sweat glands (involved in thermoregulation and emotional sweating, solely innervated by the sympathetic nervous system) (Boucsein, 2012; Dawson et al., 2007). It indicates arousal and

sympathetic activation (Kreibig, 2010). EDA is typically measured via skin surface electrodes (Boucsein, 2012). The signal includes tonic SCL (baseline arousal) and phasic SCR (event-related bursts) (Dawson et al., 2007). As we are interested in dynamic coupling related to emotional events, the continuous phasic EDA signal will be the focus for PLV analysis.

2.5 Experimental Setup

This study employs a multimodal approach, simultaneously recording fNIRS, EEG, EDA, and ECG. fNIRS (NIRSport, 8 sources/7 detectors, 17 channels + short channels (Gagnon et al., 2012)) optodes integrated into an EEG cap will cover prefrontal and parietal cortices (Chen et al., 2024). This coverage targets key regions for emotional processing (PFC (Etkin et al., 2011; Fuster, 2008)) and parietal areas involved in attention modulation and integrating bodily signals relevant to emotion (Critchley, 2005; Culham & Valyear, 2006; Pessoa, 2008). While enabling broader network analysis, parietal signals primarily provide context for the focused frontal-autonomic synchrony investigation. Inter-optode distance $\leq$ 3 cm; sampling 7.81 Hz (Scholkmann et al., 2014). EEG electrodes (10-20 system with 64 electrodes, excluding optode positions (Sharma & Meena, 2024)) and Polybox-integrated ECG (3-lead) / EDA (non-dominant hand fingers) modules will record at 1000 Hz (Luck, 2014). For EEG and fNIRS electrode placement see Figure 2.

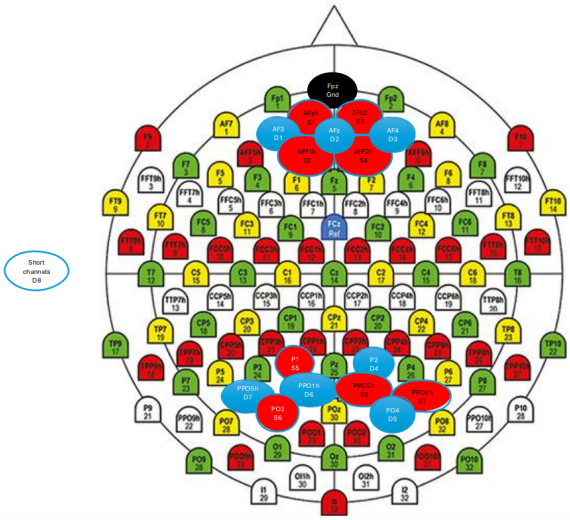

Figure 2: Placement Setup for Simultaneous EEG and fNIRS.

Combined EEG/fNIRS setup. Blue = detectors, Red = sources. Circled sources connect to short channel detectors.

2.6 Experimental Design

This study utilizes a within-subjects design where participants view emotionally evocative video clips intended to induce positive, negative, or neutral affective states. The stimuli consist of standardized clips selected from the E-MOVIE database (Maffei & Angrilli, 2019), chosen for validated effectiveness and translated to German. To manage presentation and minimize order effects, three bundles are created, each containing one randomly ordered scene of each valence (positive, negative, neutral). Bundle presentation order is counterbalanced across participants, with the constraint that the valence of the last scene in one bundle differs from the first scene of the subsequent bundle. We aim to recruit 20-30 participants for this study.

Before the task, participants complete baseline questionnaires (BIS/BAS (Carver & White, 1994; Strobel et al., 2006), Positive and Negative Affect Schedule (PANAS) (Breyer & Bluemke, 2016; Watson et al., 1988)). The experimental procedure involves repeating a specific trial structure

nine times (once for each scene). Each trial involves: 1-min baseline rest, video clip presentation, 2-min recovery rest, and post-stimulus questionnaires (SAM for valence/arousal, emotional adjectives, basic emotion evaluation, familiarity rating (Maffei & Angrilli, 2019)). Breaks and fatigue monitoring are included due to session length.
Participants (target N=20-30) will be right-handed native German speakers (18-30y), screened for neurological/psychiatric history, current psychopharmaceutical use, scalp conditions impeding sensors, and prior brain stimulation participation.

2.7 Data Analysis and Statistics

Data analysis will be conducted using the Python scientific ecosystem (Harris et al., 2020; Virtanen et al., 2020), primarily using MNE-Python (Gramfort et al., 2013) for EEG/MEG, NeuroKit2 (Makowski et al., 2021) for physiological signal processing, MNE-NIRS (Yücel et al., 2021) for fNIRS analysis, and 'statsmodels' (Seabold & Perktold, 2010) and 'pingouin' (Vallat, 2018) for statistics. The significance level will be set at $p < 0.05$. Standard assumptions (e.g., normality, sphericity) will be checked, using corrections (e.g., Greenhouse-Geisser) or non-parametric tests where appropriate (Field, 2024). Effect sizes will be reported for all key findings. Given the multiple hypotheses and comparisons involved, we will primarily control the False Discovery Rate (FDR) using the Benjamini-Hochberg procedure (Benjamini & Hochberg, 1995) applied across relevant families of tests (e.g., ANOVA results, correlation analyses).

Preprocessing and Feature Extraction Standard preprocessing pipelines will be applied using the specified Python libraries. EEG data will undergo filtering and re-referencing, followed by segmentation relative to stimulus onset. Artifacts (e.g., eye blinks, muscle activity) will be identified and corrected using Independent Component Analysis (ICA) (Delorme & Makeig, 2004). For ECG, data will be filtered, R-peaks detected (Pan & Tompkins, 1985), and NN intervals derived to calculate baseline and potentially time-varying RMSSD (see Equation 1) (Malik et al., 1996). EDA signals will be filtered and decomposed into tonic (SCL) and phasic (SCR) components (Benedek & Kaernbach, 2010), with the continuous phasic signal extracted for synchrony analysis (Boucsein, 2012). fNIRS processing involves converting raw optical density to HbO2/HbR concentration changes via the modified Beer-Lambert law (Cope & Delpy, 1988), addressing motion artifacts (e.g., using temporal derivation distribution repair (TDDR) (Scholkmann et al., 2014)), filtering (e.g., 0.01-0.1 Hz) to isolate the hemodynamic response, and calculating average concentration changes relative to baseline within predefined prefrontal ROIs (e.g., dorsolateral prefrontal cortex (DLPFC), ventromedial prefrontal cortex (VMPFC) (Etkin et al., 2011; Motzkin et al., 2015)). These fNIRS results provide spatial context for the EEG synchrony analysis.

Neural-Autonomic Synchrony and Statistical Testing Neural-autonomic synchrony will be quantified using PLV (Lachaux et al., 1999) between prefrontal EEG and continuous autonomic signals. PLV measures the phase difference consistency ($\Delta\phi(t) = \phi_{\text{EEG}}(t) - \phi_{\text{Autonomic}}(t)$) over a time window:

$$\text{PLV} = \left| \frac{1}{N} \sum_{t=1}^{N} \exp(i \cdot \Delta\phi(t)) \right| \tag{2}$$

where N is the number of time points in the window, i is the imaginary unit, and $\phi_{\text{EEG}}(t)$ and $\phi_{\text{Autonomic}}(t)$ are the instantaneous phases of the filtered EEG and the respective continuous autonomic signal (derived from HRV or EDA) at time t. For brain-heart (parasympathetic) coupling, the discrete NN intervals will be interpolated into a continuous signal (e.g., using cubic splines at 4 Hz, a standard approach (Laguna et al., 1998; Shaffer & Ginsberg, 2017)) via 'scipy.interpolate' (Virtanen et al., 2020). For brain-sudomotor (sympathetic) coupling, the

continuous phasic EDA signal will be resampled (e.g., to 4 Hz) to match the HRV-derived signal's rate. Instantaneous phase will be extracted from filtered EEG (Alpha/Beta bands) and continuous autonomic signals via Hilbert transform (Virtanen et al., 2020). PLV (see Equation 2) will then be computed between selected frontal EEG channels (e.g., F4/F3, Fp2/Fp1 (Rodrigues et al., 2021), further refined by fNIRS findings as described below) and the continuous HRV-derived and phasic EDA signals, respectively. The primary analysis window will cover the duration of each stimulus presentation, with comparisons made to PLV calculated during the pre-stimulus baseline. Exploratory analyses might examine temporal dynamics within the stimulus period, and surrogate data testing (e.g., phase shuffling (Cohen, 2014)) may be used to assess significance against chance.
Specific statistical tests will address the hypotheses:

(a) **Emotional Modulation:** We will first analyze the fNIRS data using a General Linear Model (GLM) approach to identify prefrontal ROIs significantly modulated by emotional content (Emotion vs. Neutral contrast) (Yücel et al., 2021). EEG channels spatially corresponding to these functionally identified ROIs will then be selected (Gramfort et al., 2013). The primary test for emotional modulation of synchrony will be a repeated measures Analysis of Variance (ANOVA) on the PLV values (EEG-HRV and EEG-EDA) from these selected channels, with Emotion (Positive, Negative, Neutral) as the within-subjects factor. Significant effects will be followed by FDR-corrected post-hoc tests. This integrated approach ensures that the synchrony analysis focuses on functionally relevant cortical areas.

(b) **Synchrony and Arousal:** Pearson correlations will be computed between subjective arousal ratings (SAM) and the corresponding trial's PLV magnitudes (EEG-HRV and EEG-EDA from the functionally defined channels/ROIs), specifically within the positive and negative emotional conditions.

(c) **Baseline Vagal Tone:** A Pearson correlation will assess the relationship between resting-state RMSSD (baseline parasympathetic tone) and the average EEG-HRV PLV observed during the negative emotional condition (using PLV from the functionally defined channels/ROIs).

(d) **Asymmetry and Branch-Specific Synchrony:** Frontal asymmetry will be quantified using the frontal asymmetry index (FAI), calculated from alpha band power differences between homologous frontal electrodes (e.g., F4/F3, Fp2/Fp1 (Allen et al., 2004; Rodrigues et al., 2021)). The formula is given by:

$$\text{FAI} = \ln(P_{\text{Right}}) - \ln(P_{\text{Left}}) \tag{3}$$

where P_{Right} and P_{Left} represent the alpha band power spectral density at homologous right and left frontal electrode sites, respectively (e.g., P_{F4} and P_{F3}). We will then exploratorily test for correlations (Pearson) between this FAI and the strength of EEG-HRV PLV versus EEG-EDA PLV (from functionally defined channels/ROIs), particularly during emotional conditions, to investigate potential differential links between cortical motivational direction and coupling with specific autonomic branches.

3 Timeline

3.1 Phase 1: Preparation (Months 1-2)

- Movie clip acquisition (Month 1)
- Writing experiment description (Month 1)
- Obtain EEG measurement setup (Month 1)
- Obtain fNIRS measurement setup (Month 1)
- Participant announcement (Month 1)
- Printing new fNIRS Optode holders (Month 1)
- Write declaration of consent (Month 1)
- Apply for internal email account (Month 1)
- Programming task (Month 2)
- Ethics approval (Month 2)
- Laboratory coordination (Month 2)
- Obtain ECG measurement setup (Month 2)
- Initial Literature Research (Months 1-2)

3.2 Phase 2: Data Acquisition and Early Analysis, Writing (Months 3-6)

- Piloting (Month 3)
- Introducing participants (Month 3)
- Ongoing Data Collection (Months 4-6)
- On-the-Go Data Preprocessing and Analysis (Months 3-6)
- Preliminary Data Analysis (Months 3-6)
- Refine Literature Review (Months 3-6)
- Drafting Methods Section (Months 3-6)
- Drafting Introduction Section (Months 3-6)
- Start Drafting Results (Months 5-6)

3.3 Phase 3: Finishing Analysis and Interpretation (Month 7)

- Final Data Preprocessing (Month 7)
- Final Data Analysis (Month 7)
- Finish Drafting Results (Month 7)
- Interpretation of Results (Month 7)
- Drafting Discussion Section (Month 7)

3.4 Phase 4: Finalizing and Submission (Months 8-9)

- Refining Introduction Section (First Week of Month 8)
- Refining Methods Section (First Week of Month 8)
- Refining Results Section (Second Week of Month 8)
- Refining Discussion Section (Second Week of Month 8)
- Polishing and Proofreading (Third Week of Month 8)
- Supervisor Feedback and Revisions (Fourth Week of Month 8)
- Final Revisions (First Week of Month 9)
- Thesis Submission (Second Week of Month 9)
- Buffer (Third and Fourth Week of Month 9)

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