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- **Title:** Autonomic readiness for social threats in patients with social anxiety disorder
- **Running Title:** Autonomic readiness in social anxiety disorder
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Abstract

Objectives: Pathological anxiety is characterized by dysregulated arousal and lower heart rate variability (HRV) associated with emotional dysregulation. This study explored the connection between peripheral and central autonomic nervous system activity during emotional processing in social anxiety disorder (SAD).~~Pathological anxiety in social anxiety disorder (SAD) is characterized by dysregulated arousal and altered cardiac autonomic responses, with lower heart rate variability (HRV), which is associated with emotional dysregulation. This study explored the connection between peripheral and central autonomic nervous system activity during emotional processing in individuals with SAD.~~

Methods: Thirty-two patients with SAD and 41 healthy controls engaged in a passive viewing task alternating between neutral and angry faces. The root mean square of successive differences (RMSSD) was measured during the resting state (baseline RMSSD) and emotional processing (task RMSSD). We examined the relationships between brain activation during emotional processing and these RMSSD measures.~~We examined the relationships between brain activation during emotional processing and the root mean square of successive differences (RMSSD) in both resting and task conditions.~~

Results: Unlike the controls, the SAD group exhibited a trend level toward significant correlations of baseline RMSSD with left anterior insula activity during neutral face processing ($p = .058$) and significant correlations with both left anterior insula and right amygdala activities during angry face processing ($p = .027$ and $p = .046$, respectively). In the controls, task-related RMSSD correlated with neural activities in the right amygdala and right dorsomedial prefrontal cortex during neutral face processing ($p = .017$ and $p = .004$, respectively), while in the SAD group, a correlation emerged with the right parahippocampal gyrus ($p = .044$). Notably, only in the control group did RMSSD, measured during neutral face processing, significantly correlate with neural activation during the processing of angry faces

($p = .035$).

Conclusions: This study delineates distinct autonomic and neural response patterns to emotional stimuli in SAD patients, highlighting increased autonomic readiness and diminished flexibility in response to social threats.

Keywords: Social anxiety disorder; Peripheral and central autonomic nervous system; Autonomic readiness.

1. Introduction

Social anxiety disorder (SAD) is characterized by fear and avoidance of social situations [1]. Research has highlighted the roles of emotional hyper-reactivity and dysregulation in response to perceived threats in individuals with SAD [2]. Pathological anxiety features dysregulated arousal and altered cardiac autonomic responses [3]. Furthermore, specific somatic symptoms such as hyperventilation, palpitations, and perspiration are recognized indicators of altered autonomic activity in pathological anxiety [4].

The neurovisceral integration model posits that the central autonomic network (CAN), comprised of interconnected brain regions, is essential for regulating physiological, cognitive, and emotional functions [5]. The CAN includes prefrontal areas like the ventromedial prefrontal cortex (VMPFC) and anterior cingulate cortex (ACC), as well as subcortical structures such as the hypothalamus and amygdala [5-7]. This network influences heart rate variability (HRV), which measures the variation in the time interval between heartbeats (R-R interval). This variability is thought to result from the dynamic interaction between sympathetic and parasympathetic nervous activities [8, 9]. It is an index of how strongly individuals can regulate emotion and autonomic responses in the body [10], reflecting autonomic flexibility and parasympathetic nervous system activity.

Previous studies have explored the relationship between HRV and brain activity in both healthy individuals and patient groups. Research involving healthy adult participants found that higher HRV correlates with stronger functional connectivity between the amygdala and the medial prefrontal cortex (MPFC) during rest [11]. Furthermore, higher resting vagally-mediated HRV may indicate effective regulation of amygdala activity by the prefrontal cortex, enhancing emotion regulation [12]. In studies focusing on individuals with post-traumatic stress disorder (PTSD), patients displayed lower high-frequency HRV (HF-HRV) while processing neutral and trauma-related words [13]. HF-HRV, a noninvasive measure of vagal

influences on parasympathetic heart innervation [14], reflects the body's adaptive physiological and emotional flexibility in response to immediate needs [10]. Another study on individuals with generalized anxiety disorder (GAD) observed that higher baseline connectivity between the bilateral amygdala and the prefrontal cortex/cingulum acts as a protective factor, leading to smaller decreases in HRV following mood induction [15]. These findings emphasize HRV's critical role as an indicator of the interplay between cardiac and neural activities, particularly in emotion regulation. They suggest that higher HRV is generally linked to better emotional regulation and neural connectivity in healthy individuals, while lower HRV may indicate emotional dysregulation in patient groups such as those with PTSD and GAD.

Considering that physiological symptoms are distressing and can exacerbate anxiety in individuals with SAD, it is pertinent to explore the connections between the central and peripheral autonomic systems in this group. Previous neuroimaging studies have demonstrated that patients with SAD often show emotional hyper-reactivity in anticipation of, or in response to, potential social threats. However, the mechanisms by which neural activation in response to these threats correlates with autonomic regulation during threat anticipation and confrontation remain largely unexplored in SAD. Clarifying these mechanisms could provide vital insights into the psychophysiological bases of SAD and help develop more targeted treatments. Therefore, our study aims to investigate the relationship between the peripheral autonomic nervous system, as measured by HRV, and central autonomic nervous system activity during emotional processing in patients with SAD. Specifically, we examined the correlation between resting state HRV and neural activation, as well as the relationship between task-related HRV and neural responses while passively processing emotional faces during a task.

2. Materials and Methods

2.1. Participants and measurements

Thirty-two patients with SAD and 41 healthy controls were included in the study. This sample was part of a larger cohort previously reported in other studies [16, 17]. Four patients with SAD and one control participant were excluded from the current study due to the unavailability of physiological data.

All participants underwent comprehensive clinical assessments using the Mini International Neuropsychiatric Interview [14, 15] and the Hamilton Anxiety Scale (HAS) [18], following an initial screening involving the Liebowitz Social Anxiety Scale (LSAS) [19], Social Interaction Anxiety Scale (SIAS), Social Phobia Scale (SPS) [20], the brief version of the Fear of Negative Evaluation Scale (B-FNE) [21], and the Beck Depression Inventory (BDI) [22]. The inclusion criteria for the study were as follows: for the SAD group, LSAS ≥ 30 , SIAS ≥ 34 , and/or SPS ≥ 24 ; for the control group, SIAS < 34 , SPS < 24 , B-FNE < 48 , and BDI < 21 . Additionally, all participants in both groups were right-handed and had a minimum of 12 years of education. Exclusion criteria for both groups included any history of medical, neurological, or psychiatric illnesses, with the exception of SAD and related secondary depressive disorders in the patient group. Three patients with SAD were diagnosed with comorbid depressive disorders. Seven patients were on regular medication regimens, primarily consisting of serotonergic antidepressants. Additionally, one patient had been prescribed benzodiazepines for use on an as-needed basis; however, this medication was not taken on the day of the scanning procedure.

All participants provided written informed consent to ensure their voluntary agreement and understanding of their participation in the study. The study protocol was approved by the Institutional Review Board of Seoul National University Hospital (2304-130-1426), Republic of Korea.

2.2. Design and procedure

To acquire brain imaging and HRV data in both resting and task states, participants underwent resting-state and localizer-run scans within the MRI scanner. For the resting-state scan, participants were instructed to lie still with their eyes closed, remaining awake and alert. This scan lasted for 5 minutes. Following the resting-state scan, a functional localizer run, previously used in another study [17], was conducted. This run involved a passive viewing task where participants were asked to maintain their focus on a green square dot at the center of the screen, aimed at identifying specific brain areas involved in emotional processing. To localize these regions of interest (ROIs), a neutral or angry face was displayed at the center of the screen. Additionally, a peripheral checkerboard pattern was shown in the upper right visual field to localize brain regions responsible for processing target stimuli for the subsequent phase, with data reported in a previous study [17]. Each trial included the presentation of the peripheral checkerboard, followed by a central angry face and then a central neutral face, each displayed for 10 seconds. These conditions were interspersed with 10 seconds of fixation on the green square dot, and the sequence was repeated eight times during the scan, yielding 8 minutes of total duration (Fig.1).

2.3. Brain imaging acquisition and analysis

A 3.0 Tesla MR scanner (Magnetom TrioTrim, Siemens Medical Solutions, Erlangen, Germany) was used to acquire brain images. Functional images were acquired using an echo planar imaging sequence with the following parameters: a matrix size of 64×64 , 34 slices each 3.4 mm thick, a spatial resolution of $3.4 \times 3.4 \times 3.4 \text{ mm}^3$, echo time (TE) of 30 ms, repetition time (TR) of 2 seconds, a field of view (FOV) of $220 \times 220 \text{ mm}$, and a flip angle of 80 degrees. High-resolution anatomical images were obtained using a T1-weighted MP-RAGE sequence

with parameters including a matrix size of 256×256 , 208 slices each 1.0 mm thick, a spatial resolution of $1.0 \times 1.0 \times 1.0 \text{ mm}^3$, TE of 1.89 ms, TR of 1670 ms, a FOV of $250 \times 250 \text{ mm}$, and a flip angle of 9 degrees.

We used statistical parametric mapping (SPM) 12, based on MATLAB, for data preprocessing. To ensure stable magnetic fields, we discarded the first three volumes of the functional localizer run. Preprocessing steps included slice timing correction, realignment, segmentation, coregistration, normalization, and spatial smoothing. Images were realigned to the mean image and resliced using 7th-degree B-spline interpolation. T1 images were segmented and spatially normalized using MNI/ICBM (Montreal Neurological Institute/International Consortium for Brain Mapping) templates in SPM. The images were then smoothed with a 6 mm full-width at half-maximum Gaussian kernel.

Functional MRI data were analyzed using a general linear model. For each participant, a first-level analysis was conducted where experimental conditions were modeled as separate regressors convolved with a canonical hemodynamic response function. These conditions included target, neutral, and angry faces. The angry-target contrast for each participant was computed and then entered into a second-level random effects analysis to examine group-level effects. One-sample t-tests were performed for this contrast, with the statistical threshold set at $p < 0.001$ (uncorrected) and a minimum cluster size of 10 contiguous voxels [23]. Significant clusters from the angry-target contrast were defined as functional ROIs (Table 2). Beta values for neutral face-target and angry face-target contrasts were then extracted from these ROIs for further analysis.

2.4. HRV data acquisition and analysis

Photoplethysmography (PPG) signals were acquired during a resting state baseline and during the localizer run using BIOPAC (BIOPAC Systems Inc., Goleta, CA, USA), with the

PPG sensor attached to the participant's index finger while inside the MRI scanner. The signals were processed using AcqKnowledge (BIOPAC Systems Inc., Goleta, CA, USA) and Mindware HRV (MindWare Technologies Ltd., Gahanna, OH, USA) software. Data preprocessing involved segmentation, manual editing, visual inspection, and artifact correction. Initially, we manually extracted 10-second segments from the full recording that corresponded to each stimulus presentation (target, neutral, and angry). In cases of extreme noise, mean value smoothing with a smoothing factor of 50 was applied to isolated sections. As each stimulus was presented eight times throughout the localizer run, the eight 10-second segments for each condition were concatenated to create continuous 80-second sections for analysis. Manual editing ensured data quality, excluding segments shorter than 50 seconds from the final analysis. Following segmentation, we applied Mindware's artifact correction algorithm, defining artifacts as beats outside the 40-200 bpm range or those significantly deviating from the expected beat distribution. Peaks were visually inspected using AcqKnowledge software; those that significantly deviated from the natural PPG shape or size were either merged with nearby peaks or removed. Peaks with excessively large IBI or amplitude gaps were also removed. Finally, the root mean square of successive differences (RMSSD) was extracted from the auto-calculated HRV statistics and frequency powers provided by Mindware software. RMSSD is a time-domain measure indicative of parasympathetic nervous system activity [24]. The RMSSD measured during the resting state prior to the passive viewing task was defined as baseline RMSSD, while the RMSSD measured during emotional processing was defined as task RMSSD.

Regarding task HRV, data from six out of 41 participants in the control group and three out of 32 participants in the SAD group were excluded due to poor data quality.

2.5. Statistical analysis

Statistical analyses were conducted using SPSS (version 26; SPSS Inc, Chicago, IL, USA). For both beta values and RMSSD, outliers exceeding 2.5 standard deviations from the group mean were identified and excluded from the analysis. One or two participants per group were excluded from each analysis, resulting in a varying number of participants included in each analysis. However, no participants were excluded from all analyses.

To examine the relationship between RMSSD and neural activity during facial emotion processing, a simple linear regression analysis was utilized to assess relationships between RMSSD and beta values obtained from functional ROIs. Additionally, to explore whether the association between RMSSD and brain activity during emotional processing differed between the groups, we conducted a general linear model (GLM) analysis. In the GLM, the dependent variable was the beta value of the ROI, and independent variables included the group, baseline or task RMSSD, and the interaction between RMSSD and group. In the correlation analyses, no formal correction for multiple comparisons was applied. A significance level of $p < 0.05$, uncorrected was applied across all analyses.

3. Results

All descriptive statistics are presented in Table 1. There were no significant differences in demographic variables between the two groups including age, sex, and educational level. The SAD group demonstrated higher levels of anxiety and depressed mood, as well as elevated social anxiety scores compared to the control group.

3.1. Brain regions associated with processing angry faces

As indicated in Table 2, the brain regions activated during the processing of angry emotions include the amygdala, anterior insula, parahippocampal gyrus (PHG), dorsomedial prefrontal cortex (DMPFC), VMPFC, frontal pole, inferior frontal gyrus, and visual cortex. Considering

previous studies that have identified brain regions associated with neural activation changes during emotional processing in SAD patients [2, 25-35], we proceeded with further analysis using the amygdala, anterior insula, PHG, DMPFC, and VMPFC as ROIs.

3.2. Baseline and Task RMSSD

Baseline and task RMSSD data were presented in Table 3. To examine the mean differences in RMSSD across conditions (baseline, neutral, and angry) between groups, a 2x3 repeated measures ANOVA was performed, with condition as the within-subject variable and group as the between-subject variable. The analysis revealed no significant main effects for group ($F_{1,57} = 0.264, p = .609, \eta^2 = .005$) or condition ($F_{1,212,69,093} = 2.673, p = .100, \eta^2 = .045$). However, a significant interaction effect between group and condition was observed ($F_{1,212,69,093} = 5.835, p = .014, \eta^2 = .093$).

In post-hoc analysis, between-group t-tests showed no significant differences in mean RMSSD during baseline, neutral face processing, or angry face processing between the SAD and control groups. However, within-group paired t-tests revealed significant changes in the control group between baseline and task RMSSD. Specifically, there was a significant reduction in RMSSD from baseline to neutral face processing ($t(33) = 2.609, p = .014$) and from baseline to angry face processing ($t(34) = 2.494, p = .018$). In contrast, no significant changes were observed in the SAD group between baseline and task RMSSD conditions.

3.3.2. Correlation between baseline RMSSD and the neural activity during emotional processing

During the processing of neutral faces, no significant correlation was found between baseline RMSSD and the beta values of the ROIs, including the amygdala, anterior insula, PHG, DMPFC, and VMPFC in the control group. However, in the SAD group, baseline RMSSD

exhibited a trend toward explaining the beta value of the left anterior insula during the processing of neutral faces ($R^2 = 0.118$, $\beta = -0.344$, $F_{1,29} = 3.886$, $p = .058$) (Figure 2A). In the GLM analysis, the group-by-RMSSD interaction was significant ($F_{1,67} = 4.803$, $p = .032$, $\eta^2 = .067$). These results suggest that the correlation between baseline RMSSD and left anterior insula activity during neutral face processing significantly differs between groups. There were no significant results in other brain regions, such as the amygdala, PHG, DMPFC, and VMPFC in the SAD group.

Similarly, during the processing of angry faces, no significant correlation was observed between baseline RMSSD and the beta values of the aforementioned ROIs in the control group. However, in the SAD group, significant correlations were found between baseline RMSSD and the beta values of the left anterior insula ($R^2 = 0.157$, $\beta = -0.397$, $F_{1,29} = 5.415$, $p = .027$) and right amygdala ($R^2 = 0.135$, $\beta = -0.367$, $F_{1,28} = 4.360$, $p = .046$) (Figures 2B and 2C). Finally, the GLM analysis revealed a significant group-by-RMSSD interaction for the left anterior insular ($F_{1,67} = 4.980$, $p = .029$, $\eta^2 = .069$), and showed marginal significance for the right amygdala ($F_{1,66} = 3.646$, $p = .061$, $\eta^2 = .052$), respectively. These results show that the correlation between baseline RMSSD and left anterior insula activity during angry face processing differs significantly between groups, with the correlation between baseline RMSSD and right amygdala activity showing a trend-level difference. For other ROIs, no significant correlations were found in the SAD group.

3.43. Correlation between task RMSSD and the neural activity during emotional processing

During the processing of neutral faces, significant correlations were observed in the control group between task RMSSD and the beta values of the right amygdala ($R^2 = 0.160$, $\beta = -0.400$, $F_{1,33} = 6.284$, $p = .017$) and right DMPFC ($R^2 = 0.222$, $\beta = -0.472$, $F_{1,33} = 9.443$, $p = .004$) (Figures 3A and 3B). However, no significant correlations were found between task RMSSD

and the beta values of other ROIs including the left anterior insula, left amygdala, PHG and VMPFC in control group. In the SAD group, while no significant results were found in other brain regions, a notable correlation did emerge between task RMSSD and neural activity in the right PHG ($R^2 = 0.148$, $\beta = -0.384$, $F_{1,26} = 4.5$, $p = .044$) (Figure 3C). Additionally, in the control group, RMSSD measured during the processing of neutral faces was significantly correlated with beta values of right PHG during the emotional processing of angry faces ($R^2 = 0.132$, $\beta = -0.363$, $F_{1,32} = 4.856$, $p = .035$). Such correlations were not observed in the SAD group.

Conversely, during the processing of angry faces, no significant correlations between task RMSSD and the beta values of the ROIs were observed in either the control or SAD groups.

4. Discussion

This study explored the correlation between peripheral and central autonomic network activity during emotional processing in patients with SAD. The findings revealed that during the resting state, only the SAD group demonstrated a correlation between baseline HRV and brain activation during emotional processing, reflecting their autonomic readiness toward social threats. Conversely, during task performance, HRV and brain activation correlated in the control group, while the SAD group showed a weaker correlation compared to controls. These results highlight distinct patterns of autonomic readiness and neural activation in response to emotional stimuli between the control and SAD groups.

Resting-state HRV is linked to various functions critical to our survival, reflecting cardiac autonomic control [36] and influencing decision-making processes concerning overall health in everyday life [37]. Additionally, HRV ensures that acute stress responses are adaptive through interactions between the sympathetic and parasympathetic branches of the autonomic nervous system (ANS), allowing physiological functioning to return to baseline levels upon the cessation of stress when encountering a demanding external or internal stimulus [38].

Higher resting HRV is positively associated with a more flexible and adaptive range of psychophysiological responses, preparing individuals for dynamic environmental challenges [39-42].

In the control group, resting HRV did not significantly correlate with brain activity during the processing of neutral and angry faces, suggesting that individuals with high autonomic adaptability may not have needed to prepare in advance for social threats. In contrast, patients with SAD show a correlation between adaptability in the resting state, as indicated by resting HRV, and neural activation when processing social threats. This relationship was evident not only during the processing of angry faces but also during neutral faces. These results align with previous findings that individuals with SAD perceive neutral faces as emotionally ambiguous rather than neutral stimuli [43], leading them to interpret these expressions negatively, contributing to the persistence of anxiety [44]. Therefore, the correlation between baseline HRV and brain activation observed in the SAD group during the processing of both angry and neutral faces suggests that individuals with SAD perceive neutral faces as threatening stimuli [45-47]. Consequently, the observed correlation between baseline RMSSD and neural activity during tasks in the SAD group suggests that, compared to the control group, patients may be in a state of heightened readiness for external emotional stimuli.

The correlation between HRV measured during the task and brain activation was particularly notable during the processing of neutral faces, more so in the control group. This observation can be interpreted within the framework of a task design where neutral and angry faces are alternately presented, creating 'contexts' that necessitate different cognitive responses to meet situational demands [48]. In this context, the phase during which neutral faces are presented may be viewed as a preparatory stage for the upcoming situation, reflecting a state of autonomic readiness. Given that HRV is associated more with the function of adapting and preparing for situations rather than merely reflecting the immediate state, it suggests that task HRV is more

linked to preparatory measures than to brain activation occurring at the time of task performance. The scarcity of references reporting a direct correlation between task HRV and neural activation during specific emotional processing supports this hypothesis. This is further underscored by findings that, unlike in the SAD group, the control group exhibited a significant correlation between RMSSD measured during the processing of neutral faces and neural activation during the processing of angry faces. This comparison highlights that in the SAD group, the association between HRV and brain activation during the task is weaker, implying that autonomic readiness is less pronounced and reflects their maladaptive responses to social stimuli.

This study has several limitations that warrant discussion. First, the reliability of HRV analysis in our task design requires consideration. We used segments of only 80 seconds to analyze HRV for each condition. However, existing literature suggests that blocks as short as 12.5 seconds are sufficient for HF-HRV analyses [13, 14]. Moreover, previous studies have validated the use of concatenated PPG data from individual trials for analysis [49], supporting the validity of our approach. Second, the HRV analysis during the task was compromised by substantial noise, and after the exclusion of extreme outliers, the final analysis was conducted with a limited dataset. Third, no formal correction for multiple comparisons was applied in the correlation analyses, which increases the potential risk of Type I errors. To enhance the generalizability and robustness of the findings, future studies should apply more stringent statistical corrections. ~~To enhance the generalizability and applicability of the findings,~~ Further research is recommended with larger sample sizes and an expanded patient population, including individuals with various anxiety disorders such as generalized anxiety disorder, panic disorder, and specific phobia.

5. Conclusion

In conclusion, individuals with SAD exhibited distinct autonomic and neural response patterns to emotional stimuli compared to healthy controls. For the SAD group, baseline HRV showed negative correlations with neural activity during the processing of both neutral and angry faces, suggesting a distinct autonomic readiness to potential threats, regardless of their valence. During the task, they exhibited weaker correlations between HRV and neural activation than the control group, indicating a diminished ability to prepare for forthcoming stimuli and reduced autonomic flexibility during the processing of emotional stimuli. These findings suggest a connection to the clinical pathology of SAD. Therefore, interventions that aim to increase HRV and enhance peripheral adaptability, such as biofeedback therapy, or therapies that reduce attentional bias to modulate neural activation in the central nervous system, may prove particularly beneficial for SAD patients. Moreover, the correlation between peripheral HRV and neural activation identified in this study could serve as a useful marker for evaluating treatment responses and outcomes in SAD interventions.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

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Author Contributions

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Table 1. Demographic and descriptive characteristics of the participants

Variable	SAD (<i>N</i> = 32)		Controls (<i>N</i> = 41)		χ^2 or <i>t</i>
	<i>N</i>	%	<i>N</i>	%	
Male	17	23.3	22	30.1	0.002
	mean	SD	mean	SD	
Age, in years	25.6	3.2	24.8	3.1	-1.092
Educational level, in years	15.8	2.4	15.6	1.5	-0.399
Liebowitz Social Anxiety Scale, 0-144	76.7	25.6	18.1	7.6	-12.505*
Social Interaction Anxiety Scale, 0-80	52.8	15.1	12.9	6.4	-14.015*
Social Phobia Scale, 0-80	39.5	19.8	4.9	4.9	-9.651*
Brief Fear of Negative Evaluation Scale, 12-60	46.9	9.7	26.3	6.2	-10.445*
Hamilton Anxiety Scale, 0-56	28.8	10.2	6.8	5.1	-10.701*
Beck Depression Inventory, 0-63	16.4	10.3	4.7	5.5	-5.793*

Note: SAD, social anxiety disorder; SD, standard deviation

* $p < .001$

Table 2. Brain regions significantly activated when processing angry faces compared to targets

Area	H	x	y	z	voxels	equiv Z
Amygdala	L	-20	-8	-12	98	7.80
	R	22	-2	-10	41	5.79
Anterior insula / Lateral orbital frontal	L	-40	28	-2	674	5.40
Parahippocampal gyrus	L	-18	-32	-2	37	4.54
	R	22	-30	-2	35	7.66
Dorsomedial prefrontal cortex	R	10	34	60	18	4.59
Ventromedial prefrontal cortex	L	-2	48	-12	12	3.65
Frontal pole	L	-8	60	32	1170	6.59
Inferior frontal	L	-54	22	30	101	4.23
	R	50	34	14	1611	6.24
Visual cortex	R	30	-86	16	24	5.46

Note: H, hemisphere; L, left; R, right; equiv Z, equivalent Z-score

Table 3. Baseline and Task RMSSD

Variable	SAD		Controls	
	mean	SD	mean	SD
RMSSD during baseline	77.2	48.7	100.6	73.1
RMSSD during neutral face processing	71.7	32.3	66.5	30.1
RMSSD during angry face processing	73.9	27.9	72.6	36.5

Note: RMSSD, the root mean square of successive differences; SAD, social anxiety disorder; SD, standard deviation

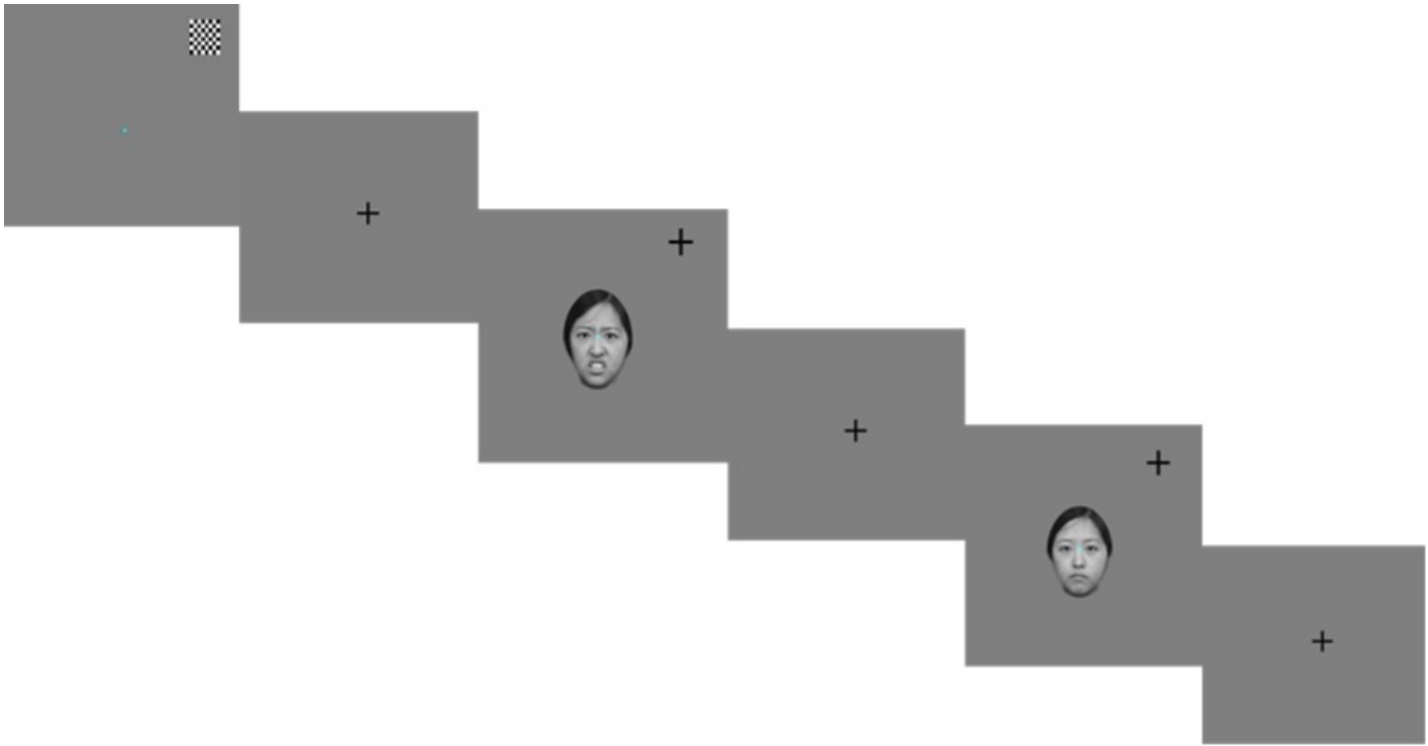


Fig. 1. Fig. 1. An example of the sequence in the passive viewing task

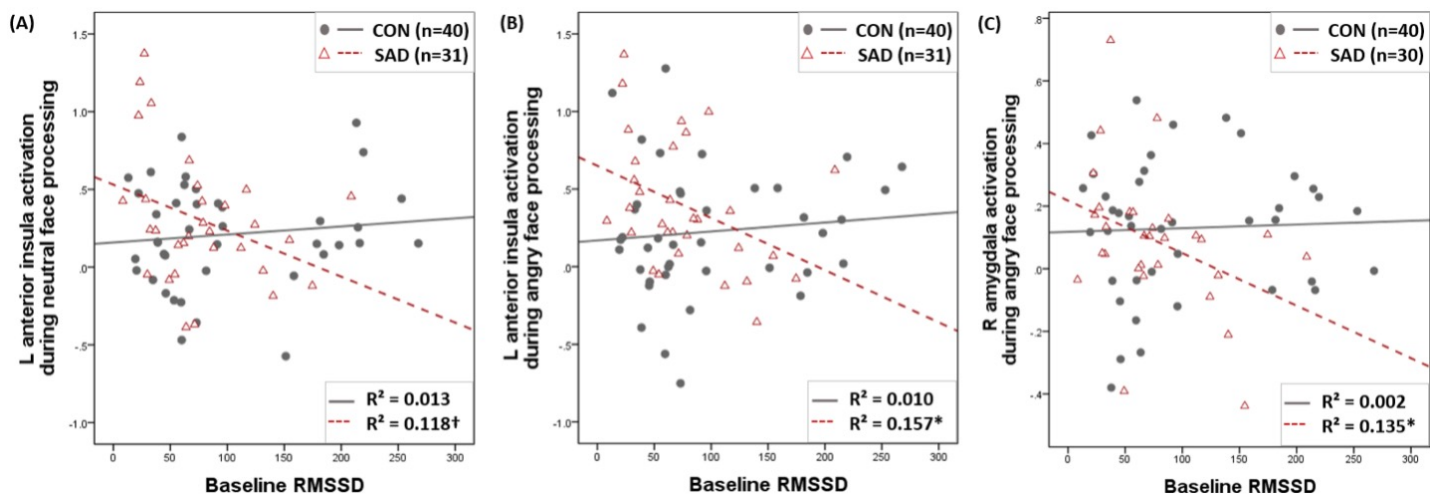


Fig. 2. Fig. 2. The relationship between baseline RMSSD and neural activity in ROIs during emotional processing

Note: CON, control group; SAD, social anxiety disorder group; L, left; R, right;
 $^\dagger p = 0.058$; $^*p < 0.05$

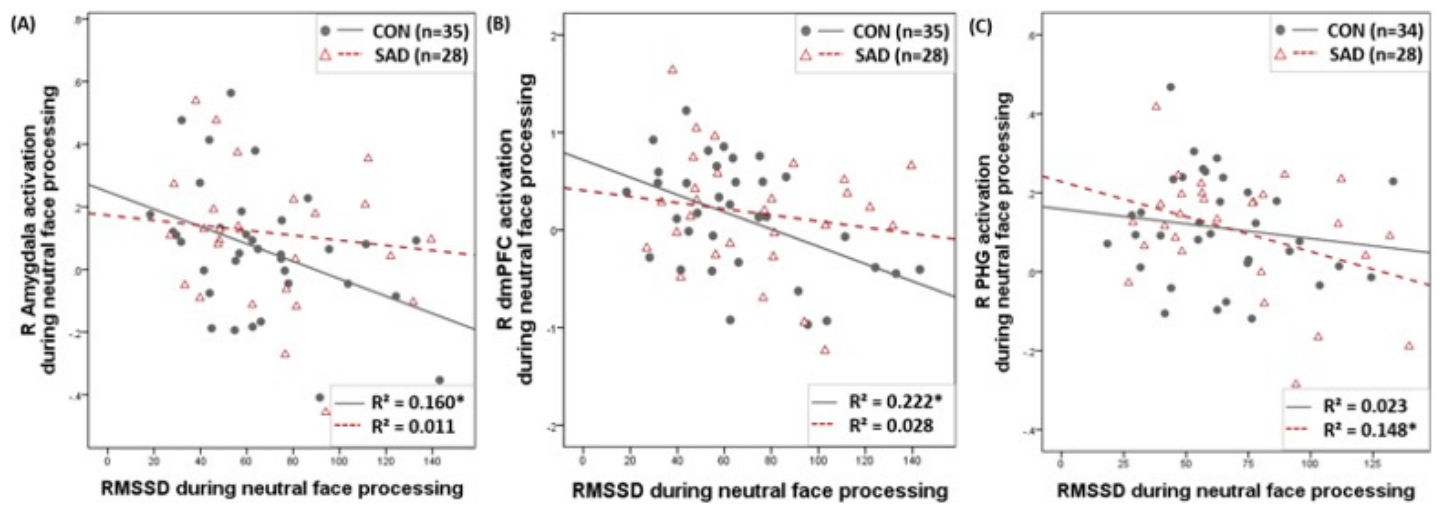


Fig. 3. Fig. 3. The relationship between task RMSSD and neural activity in ROIs during emotional processing

Note: CON, control group; SAD, social anxiety disorder group; R, right; dmPFC, dorsomedial prefrontal cortex; PHG, parahippocampal gyrus; * $p < 0.05$