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Electrocortical measures of information processing biases in social anxiety disorder: A review

Anita Harrewijn*[a,b,d], Louis A. Schmidt[c,e], P. Michiel Westenberg[a,b,f], Alva Tang[c,g], & Melle J.W. van der Molen[a,b,h]

a. Developmental and Educational Psychology, Leiden University, Wassenaarseweg 52, 2333 AK Leiden, The Netherlands

b. Leiden Institute for Brain and Cognition, Leiden University, P.O. Box 9600, 2300 RC, Leiden, The Netherlands

c. Department of Psychology, Neuroscience & Behaviour, McMaster University, 1280 Main Street West, Hamilton, Ontario L8S 4K1, Canada

d. anitaharrewijn@gmail.com

e. schmidtl@mcmaster.ca

f. westenberg@fsw.leidenuniv.nl

g. tanga6@mcmaster.ca

h. m.j.w.van.der.molen@fsw.leidenuniv.nl

*Corresponding author:

Anita Harrewijn, MSc

Phone: +1 240 750 4038

Abstract

Social anxiety disorder (SAD) is characterized by information processing biases, however, their underlying neural mechanisms remain poorly understood. The goal of this review was to give a comprehensive overview of the most frequently studied EEG spectral and event-related potential (ERP) measures in social anxiety during rest, anticipation, stimulus processing, and recovery. A Web of Science search yielded 35 studies reporting on electrocortical measures in individuals with social anxiety or related constructs. Social anxiety was related to increased delta-beta cross-frequency correlation during anticipation and recovery, and information processing biases during early processing of faces (P1) and errors (error-related negativity). These electrocortical measures are discussed in relation to the persistent cycle of information processing biases maintaining SAD. Future research should further investigate the mechanisms of this persistent cycle and study the utility of electrocortical measuresin early detection, prevention, treatment and endophenotype research.

Key words:

Delta-beta correlation, EEG, ERN, event-related potentials, information processing biases, P1, P2, social anxiety disorder, spectral measures

1. Introduction

Social anxiety disorder (SAD) is a highly prevalent and debilitating disorder characterized by fear and avoidance of social or performance situations that might lead to scrutiny and/or negative evaluation by others (Rapee & Spence, 2004; Spence & Rapee, 2016). It is posited that social anxiety is expressed along a severity continuum (Rapee & Spence, 2004). That is, many people experience symptoms of social anxiety without meeting the clinical diagnostic criteria for SAD. When social anxiety symptoms hinder someone's daily-life functioning to such an extent that they avoid social situations, these people often meet the diagnostic criteria for SAD (APA, 2013). SAD is among the most prevalent psychiatric disorders, with a life-time prevalence ranging from 5.0% to 12.1% in the United States (Grant et al., 2005; Kessler, Berglund, Demler, Jin, & Walters, 2005). Patients with SAD have an increased risk for developing comorbid disorders, such as other anxiety disorders, depression, and substance abuse (Grant et al., 2005; Rapee & Spence, 2004; Spence & Rapee, 2016). Therefore, the identification of mechanisms underlying and maintaining SAD is of critical importance to improve (preventive) interventions for SAD.

Many cognitive-behavioral studies have demonstrated that information processing biases play an important role in the development and maintenance of SAD (Bögels & Mansell, 2004; Clark & McManus, 2002; Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004; Morrison & Heimberg, 2013; Wong & Rapee, 2016). Information processing biases might be displayed as biases in attention (e.g., hypervigilance, or self-focused attention) (Bögels & Mansell, 2004), interpretation (e.g., evaluating own behavior very critically, or interpreting social situations in a negative way), memory (e.g., selectively retrieving negative information), and imagery (e.g., experiencing images of oneself performing poorly in social situations) (Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004; Morrison & Heimberg, 2013). Cognitive models posit that patients with SAD exhibit a persistent cycle of information

processing biases, which perpetuate different stages of processing (i.e., automatic and controlled) and reinforce socially anxious behaviors over time. These information processing biases are triggered when the person is confronted with a socially stressful situation, repeated while in the situation, and carried forward in time when anticipating similar future events (Clark & McManus, 2002; Morrison & Heimberg, 2013). Electrocortical measures that are related to social anxiety could provide more insight in these information processing biases. So, to delineate electrocortical measures underlying the different stages of this persistent cycle of information processing biases, we reviewed EEG measures during rest, anticipation of, and recovery from socially stressful situations, as well as event-related potential (ERP) measures during the processing of socially threatening stimuli.

We reviewed electrocortical measures of SAD, because EEG/ERP offers an online, objective and direct measure of brain activity. Of note, the future utility of potential electrocortical measures is highlighted by the relative ease of application and cost-effectiveness (Amodio, Bartholow, & Ito, 2014; Luck, 2005). Most importantly, the high temporal precision of ERPs is very useful for capturing the precise timing of information processing biases during stimulus processing (Amodio et al., 2014; M. X. Cohen, 2011; Ibanez et al., 2012; Luck, 2005). The goal of this review was to provide a comprehensive overview of the most frequently studied EEG and ERP measures during rest, anticipation, stimulus processing, and recovery. These electrocortical measures may give insight into the mechanisms underlying and maintaining the persistent cycle of information processing biases in SAD, and might eventually be used in early detection, prevention, treatment and endophenotype research.

1.1 Focus

To delineate electrocortical measures related to the information processing biases in SAD, we reviewed studies that have reported on EEG spectral characteristics during rest, anticipation and recovery from a socially stressful situation, as well as ERPs during stimulus processing. Given that the social anxiety literature on EEG spectral characteristics has largely focused on power of the alpha frequency band and the correlation between the power of delta and beta frequency bands, these two EEG metrics were included in our review (Table 1). These EEG metrics were studied during resting state, in which participants sat still for a certain period of time, or during impromptu speech preparation tasks.

With respect to ERPs, studies on social anxiety have primarily investigated stimulus processing in face processing and in cognitive conflict paradigms. ERPs give precise insight in the timing of biases in processing of faces and errors/feedback. To put the ERPs into context and to show that differences in ERPs are not caused by differences in behavior, we also reported on behavioral findings in the tasks. Studies using face-processing paradigms typically include negative emotional faces as socially threatening stimuli because they communicate social dominance (Öhman, 1986) or disapproval for violated social rules or expectations (Averill, 1982, as discussed in Kolassa and Miltner, 2006). In this review, we further distinguished between explicit and implicit face processing paradigms (Table 2) to examine the effects of task-relevant (explicit) versus task-irrelevant (implicit) faces on the modulation of early and late ERP components (Schulz, Mothes-Lasch, & Straube, 2013). In explicit paradigms, participants are required to direct their attention to the emotional valence of stimuli. In implicit paradigms, participants are presented with emotional faces, but are required to direct their attention to different aspects of stimuli (e.g., indicating the gender of stimuli, or responding to a target replacing the faces). Our review focused on the early P1,

N170, and P2 components, and the late P3 and late positive potential (LPP) components, since studies on social anxiety have examined these ERP components[1].

A recent and very relevant line of ERP research in social anxiety has focused on ERP components of feedback processing and performance monitoring in cognitive conflict paradigms. We reviewed ERP studies that have focused on the N2, feedback-related negativity (FRN), error-related negativity (ERN), correct response negativity (CRN), and positive error (Pe) components in these cognitive conflict paradigms (Table 3)[2].

We included studies reporting on patients diagnosed with SAD, as well as high socially anxious individuals, because both are expressions of social anxiety at the more severe end of the continuum (Rapee & Spence, 2004). We also reviewed studies examining constructs related to SAD, such as fear of negative evaluation, social withdrawal, shyness, and behavioral inhibition, since these constructs share common symptoms of SAD (Stein, Ono, Tajima, & Muller, 2004). Fear of negative evaluation is considered as a hallmark cognitive feature of SAD, whereas social anxiety is a more complete measure encompassing behavioral and affective symptoms (Carleton, McCreary, Norton, & Asmundson, 2006). Social withdrawal is a behavioral style commonly observed in childhood that is characterized by a lack of engagement in social situations or solitary behavior, such as playing alone (Rubin & Burgess, 2001). Shyness is a personality dimension defined as self-preoccupation and inhibition in social situations (Cheek & Buss, 1981). Behavioral inhibition is a temperament observed in infancy as negative reactivity to novel social and nonsocial stimuli (Hirshfeld-Becker et al., 2008). While these constructs are different, they are related to each other and to

[1] For studies using face processing paradigms, we did not report on the C1, N1, P150, N250, FN400, correct-response negativity (CRN), vertex positive potential (VPP), early posterior negativity (EPN), contralateral delay activity (CDA), and stimulus-preceding negativity (SPN) components, because very few (only 1 to 3) studies have investigated these components in relation to social anxiety.

[2] For studies using cognitive conflict paradigms, we excluded results on the N1, P150, P2, P3, LPP, CDA, and SPN components, because very few (only 1 to 2 studies) have reported on these components in social anxiety.

a greater risk of developing SAD (Clauss & Blackford, 2012; Hirshfeld-Becker et al., 2008; Stein et al., 2004).

We focused our review on studies of adults, due to several factors that hinder a comprehensive comparison between adult and child studies. For instance, brain development should be taken into account when comparing spectral EEG measures and ERPs between adults and children. Brain development is associated with a decline in total EEG power, as well as a shift from dominant slow wave (theta) activity to the dominant alpha rhythm as seen in adults (Marcuse et al., 2008; Segalowitz, Santesso, & Jetha, 2010). Such age-related differences in spontaneous EEG activity question the similarity in the functional significance of electrocortical measures when compared between age groups. Also, different methodological approaches might be required in quantifying these spectral measures (e.g., spectral band-width of alpha power should be different between young children and adults), which does not happen often in the literature. With regard to the ERP technique, comparing data between child and adult samples might be complicated by other factors, such as information processing efficiency, strategies used to allocate attention, and even task instructions (Segalowitz et al., 2010). Therefore, we focused mainly on electrocortical studies in adults, but we included a paragraph on developmental studies at the end of the review (Table 4 and 5).

This review is organized as follows: First, we describe briefly the information processing biases in social anxiety as recognized in the cognitive-behavioral literature. These cognitive-behavioral findings (e.g., attention biases, hyperviliance/avoidance tendencies) can be used as an information processing framework (Clark & McManus, 2002) for interpreting the electrocortical measures of SAD. Second, we give an introduction to EEG spectral characteristics and then review studies on spectral EEG analyses at rest, during anticipation of and recovery from socially stressful situations. Third, we introduce the ERP method, and

review studies that report on early and late ERP components in response to facial stimuli and ERP components in cognitive conflict paradigms as potential indices of information processing biases in social anxiety. Lastly, we conclude by relating our findings to the persistent cycle of information processing biases that maintains SAD, and discussing the utility of electrocortical measures of SAD. We also describe current methodological challenges in electrocortical studies, and developmental studies involving these EEG and ERP measures of SAD.

1.2 Search strategy

We searched Web of Science for electrocortical studies in socially anxious individuals, using the key terms *EEG or ERP or oscillation** and *social anxi* or social anxiety disorder or fear of negative evaluation or social withdrawal or shy* or behavioral inhibition*, combined with *resting state, anticipation, recovery, face, stimulus processing, emotion, error, or performance monitoring*. We also searched the reference list of the articles for additional studies, and searched for other publications of the authors of the articles. The data search was conducted before February 16th, 2017. The inclusion criteria for studies were including participants older than 18 years, who displayed SAD, high social anxiety, fear of negative evaluation, social withdrawal, shyness, or behavioral inhibition (as determined by standardized, validated measures). We included all published papers that were written in English. The data search resulted in a total of 35 studies.

2. Information processing biases in social anxiety

Cognitive-behavioral studies have repeatedly shown that socially anxious individuals display information processing biases in attention, interpretation, memory, and imagery (for extensive reviews, see Bögels and Mansell, 2004; Clark and McManus, 2002; Heinrich and

Hofmann, 2001; Hirsh and Clark, 2004). These information processing biases can occur before, during, and after social situations (Hirsch & Clark, 2004).

Prior to a social situation, socially anxious individuals may exhibit information processing biases because they anticipate that negative events might result from the social encounter (Clark & McManus, 2002; Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004). An example of a socially stressful situation is public speaking. Research has shown that feelings of anxiety can be evoked in anticipation of performing a public speech (Westenberg et al., 2009). This anticipatory anxiety enhances perceptual processing and directs attention to socially threatening stimuli such as emotional faces (Wieser, Pauli, Reicherts, & Muhlberger, 2010). During the anticipation of a socially stressful situation, socially anxious individuals display memory biases. For example, high socially anxious individuals selectively retrieved negative impressions about oneself, and patients with SAD selectively retrieved past social failures (Clark & McManus, 2002). Patients with SAD estimated the chance of negative social events higher than controls or patients with other anxiety disorders (Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004). Furthermore, patients with SAD estimated the consequences of negative social events and evaluation by others as more severe than controls or patients with other anxiety disorders (Hirsch & Clark, 2004).

Cognitive models posit that information processing biases during anticipation might steer attentional focus towards potentially threatening social cues (Bögels & Mansell, 2004; Clark & McManus, 2002; Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004; Morrison & Heimberg, 2013). This notion is in line with the hypervigilance-avoidance theory of attentional function in anxiety disorders (Mogg et al., 1997). This theory states that socially anxious individuals process socially threatening stimuli in two stages: initial vigilance (i.e., allocating attention to threatening stimuli), followed by avoidance of these stimuli (after 500-1000 ms) (Bögels & Mansell, 2004; Mogg, Bradley, DeBono, & Painter, 1997).

These information processing biases impact the thoughts and beliefs in socially anxious individuals after such socially stressful situations, triggering post-event rumination. For example, shortly after a social situation, patients with SAD interpreted ambiguous social situations in a negative way, and mildly negative situations in a catastrophic way (Brozovich & Heimberg, 2008; Clark & McManus, 2002). Socially anxious individuals displayed a recall bias, they were more likely to remember past negative social situations (Brozovich & Heimberg, 2008; Clark & McManus, 2002). Further, socially anxious individuals displayed prolonged and more perseverative self-focused thoughts and negative interpretations of themselves after a socially stressful situation (Brozovich & Heimberg, 2008).

Although these information processing biases seem to be triggered by a socially stressful situation, there is also evidence suggesting that information processing biases occur spontaneously, and hence are not restricted to a specific social situation. However, because there is no overt behavioral response linked to spontaneous information processing biases, much of this research stems from studies of "intrinsic" measures of brain functioning during rest, which are thought to reflect a history of brain activation in goal-directed, purposeful processing states (Sylvester et al., 2012). Indeed, resting-state functional MRI (fMRI) studies have shown that social anxiety was related to an imbalance between the amygdala and prefrontal cortex, which is linked to emotion dysregulation (Miskovic & Schmidt, 2012). Moreover, some EEG studies have shown social anxiety is related to differential resting brain activity linked to negative emotion and withdrawal-related social behaviors (Miskovic et al., 2011; Schmidt, 1999).

Together, there is accumulating evidence from cognitive-behavioral studies suggesting that socially anxious individuals display information processing biases during various contexts. Although these studies have offered important insights into the characteristics of information processing biases, they were not able to delineate the exact nature and time-

course of these biases. This is mainly due to constraints of subjective dependent variables (e.g., self-report data), as well as a limitation in isolating specific processes (e.g., stimulus detection, categorization, response selection). Electrocortical studies provide a direct and objective index of information processing with high temporal resolution (Amodio et al., 2014; M. X. Cohen, 2011; Kotchoubey, 2006; Luck, 2005), and could yield a richer understanding of how social anxiety is maintained. Such results could provide valuable insight in unraveling disorder-specific biological measures that in turn could facilitate early diagnosis and (preventive) intervention.

3. Spectral EEG measures related to information processing biases in social anxiety

The degree of synchronous firing of pyramidal neurons measured at the scalp with EEG is reflected in neuronal oscillations of different frequencies (Knyazev, 2007; Von Stein & Sarnthein, 2000). The range of frequencies in the human EEG that are typically examined in electrocortical studies include the delta (1 to 3 Hz), theta (4 to 8 Hz), alpha (8 to 13 Hz), beta (13 to 30 Hz), and gamma (30 to 100 Hz) bands. Rhythmic changes in the strength of oscillatory activity in a certain frequency band can be induced by various mental operations, and is reflective of different brain functions (Knyazev, 2007). In addition, the cross-talk between low and high EEG frequency bands – represented by indices of amplitude-amplitude or phase-amplitude coupling – have been suggested to reflect the functional communication between distant brain regions (Bastiaansen, Mazaheri, & Jensen, 2012; Schutter & Knyazev, 2012). In the social anxiety literature, researchers have mainly focused on alpha power, and the correlation between delta and beta power. Thus, our review is limited to these spectral EEG measures (Table 1).

3.1 Frontal alpha asymmetry

An influential theory on hemispheric asymmetry and emotion suggests that individual differences in positive and negative affect can be quantified in terms of asymmetry patterns in frontal alpha power (Davidson, 1992, 1998). More specifically, relatively greater left frontal cortical activity is related to approach behavior, whereas relatively greater right frontal cortical activity is related to withdraw behavior (Davidson, 1992, 1998). However, it should be noted that there is no simple correspondence between positive/negative affect and approach/avoidance behavior. For example, anger is a negative emotion related to approach behavior and was also related greater left frontal cortical activity (Harmon-Jones & Allen, 1998; Harmon-Jones, Gable, & Peterson, 2010). Frontal alpha asymmetry is typically measured by subtracting log-transformed left lateralized frontal alpha power from log-transformed right lateralized frontal alpha power (Allen, Coan, & Nazarian, 2004). Since alpha power is inversely related to cortical activity, positive alpha asymmetry scores reflect relatively greater left frontal cortical activity (i.e., decreased left frontal alpha power), and negative alpha asymmetry scores reflect relatively greater right frontal cortical activity (i.e., decreased right frontal alpha power) (Allen et al., 2004). Frontal alpha asymmetry has been examined in relation to the behavioral approach and avoidance systems (Carver and White, 1994). Some studies have shown that right frontal alpha asymmetry is related to behavioral inhibition (Coan & Allen, 2004), whereas other studies have shown that this relation is more complex and not related to behavioral inhibition alone (Coan & Allen, 2003).

3.2 Frontal alpha asymmetry in social anxiety

3.2.1 Rest

Frontal alpha asymmetry has often been studied during resting state EEG measurements (or baseline), in which participants are asked to sit still during a certain period of time, with their eyes open or closed. The literature on frontal alpha asymmetry during

resting state in social anxiety appears to be mixed. For example, patients with SAD showed increased left frontal activity after cognitive-behavioral therapy (Moscovitch et al., 2011). However, this study did not include a control group nor a treatment control condition, so it cannot be concluded that SAD patients showed increased right frontal activity compared to controls before treatment. Frontal alpha asymmetry during resting state has also been investigated in relation to constructs related to social anxiety, such as shyness in nonclinical samples. For example, greater right frontal activity has been observed in adults scoring high on shyness versus those scoring low on shyness (Schmidt, 1999). In contrast, other studies have found no difference in resting frontal alpha asymmetry between patients with SAD and controls (Davidson, Marshall, Tomarken, & Henriques, 2000), between high and low socially anxious individuals (Beaton et al., 2008; Harrewijn, Van der Molen, & Westenberg, 2016), and between high and low socially withdrawn individuals (Cole, Zapp, Nelson, & Perez-Edgar, 2012).

3.2.2 Anticipation

Cognitive models have highlighted the importance of information processing biases when socially anxious individuals anticipate exposure to feared social situations. Patients with SAD typically anticipate a more negative outcome in social situations and have more negative expectations about their own performance in social situations. Patients with SAD fear behaving in an inappropriate way, because it might result in negative evaluation by others (Clark & McManus, 2002; Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004).

Typically, anticipatory anxiety in SAD is examined via impromptu speech preparation tasks, in which participants are asked to prepare a speech on a general topic or on personal characteristics. An example of a social performance task is presented in Figure 1. Some studies have shown that frontal alpha asymmetry is related to social anxiety during

anticipation in such socially stressful situations (Cole et al., 2012; Davidson et al., 2000). For example, Davidson et al. (2000) examined frontal alpha asymmetry in patients with SAD while they were anticipating to perform a speech about an unknown topic and while preparing this speech when they were informed about the topic. Patients with SAD showed increased right anterior temporal activity during anticipation and planning compared to resting state (Davidson et al., 2000). Likewise, high socially withdrawn individuals showed increased right frontal activity during anticipation of performing their own speech, when they watched a video of a confederate talking in an anxious way, but not when the confederate talked in a non-anxious way (Cole et al., 2012). Other studies have found no effect of social anxiety between high versus low socially anxious individuals during anticipation of a speech (Beaton et al., 2008; Harrewijn et al., 2016), or between high versus low shy individuals during anticipation of a social interaction (Schmidt & Fox, 1994). Although Beaton et al. (2008) did not find a difference between high and low socially anxious individuals, shyness was related to increased right frontal activity in their sample, but only after controlling for depression.

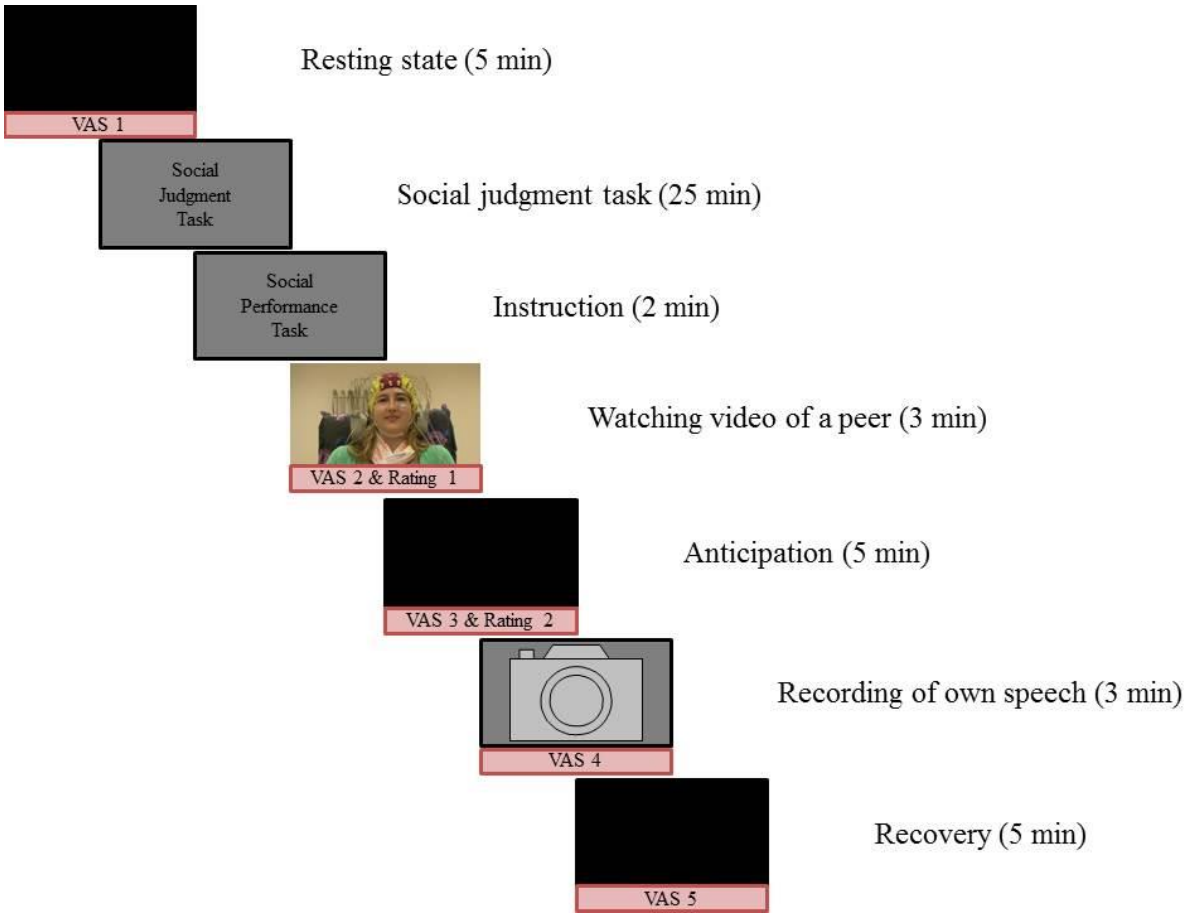

Figure 1. Example of a social performance task. This task includes a recovery phase after giving the speech, which is a novel compared to usual designs that measure only resting state and anticipation. Reprinted from Cognitive, Affective & Behavioral Neuroscience, Harrewijn, A., Van der Molen, M.J.W., & Westenberg, P.M., Putative EEG measures of social anxiety: Comparing frontal alpha asymmetry and delta-beta cross-frequency correlation, Copyright (2016), with permission.

The mixed findings among these studies can be explained in several ways. First, the effect of social anxiety might only be measurable at extreme levels of social anxiety. That is, the effect was significant for patients with SAD (Davidson et al., 2000), who presumably experience more social anxiety, than high socially anxious individuals. However, the sample size in the study of Davidson et al. (2000) was rather small (14 patients with SAD), and thus these results need to be interpreted with caution. Furthermore, Cole et al. (2012) only found

increased right frontal activity in high socially withdrawn individuals in the anxious condition. Tasks without such an anxiety-inducing condition might not elicit an increase in frontal alpha asymmetry, such as in Harrewijn et al. (2016). Second, the effect of social anxiety might only be measurable if the control group shows no anxiety during the task. For example, control participants in the study of Davidson et al. (2000) showed no increase in subjective anxiety during anticipation, whereas low socially anxious participants in the study of Harrewijn et al. (2016) showed an increase in subjective anxiety. An increase in subjective anxiety in control participants might render the inability to detect significant group differences in frontal alpha asymmetry. Third, Davidson et al. (2000) focused on the difference between anticipation and resting state, whereas most studies only focused on anticipation (Beaton et al., 2008; Cole et al., 2012; Harrewijn et al., 2016; Schmidt & Fox, 1994). However, no effect of social anxiety was found when analyzing the difference between anticipation and resting state data in the Harrewijn et al. (2016) study. Fourth, the effect of social anxiety on frontal alpha asymmetry during anticipation might also be related to differences in the duration of the anticipation period. Studies that did not find frontal alpha asymmetry effects (Harrewijn et al., 2016; Schmidt & Fox, 1994) used relatively longer anticipation periods (i.e., 5-6 minutes) compared to studies that used shorter anticipation periods (Beaton et al., 2008; Cole et al., 2012). Particularly, Davidson et al. (2000) used an anticipation period of 3 minutes and a planning condition of 2 minutes that presented new information (topic of the speech), which might have increased participants' anxiety again during this phase. Overall, null effects in studies that have employed longer anticipation periods might be due to a habituation effect. That is, if the anticipation period is longer, participants' anxiety might habituate and less right frontal activity is shown towards the end. Possible habituation effects should be examined in future studies by comparing frontal alpha asymmetry of various time-bins during the anticipation period.

3.2.3 Recovery

Recovery from a socially stressful situation, such as performing a speech, might induce increased post-event processing in socially anxious individuals. According to various cognitive-behavioral studies (Brozovich & Heimberg, 2008; Clark & McManus, 2002), post-event processing in social anxiety is characterized by rumination and perseverative thinking (e.g., negative beliefs about past performance during a social situation). This enhanced retrieval of negative memories and a focus on negative assumptions are believed to maintain social anxiety symptoms (Brozovich & Heimberg, 2008). Potentially, post-event processing during recovery stages of a social performance task might be tracked by frontal alpha asymmetry. Only two studies have measured frontal alpha asymmetry during recovery from giving a speech. These studies failed to detect differences in frontal alpha asymmetry between patients with SAD and controls (Davidson et al., 2000) and between high and low socially anxious individuals (Harrewijn et al., 2016). Although the apparent scarcity of studies should be taken into account, these studies suggest that post-event processing in social anxiety is not reflected in patterns of frontal alpha asymmetry.

3.3 Delta-beta cross-frequency correlation

Another EEG metric that has been of interest in examining information processing biases in social anxiety during resting state, anticipation and recovery, is the cross-frequency correlation between the power (i.e., amplitude) of delta and beta oscillations, hereafter referred to as delta-beta correlation. Although different metrics of cross-frequency coupling exist, such as phase-phase or phase-amplitude coupling (M. X. Cohen, 2014), our focus is on the amplitude-amplitude coupling between the delta and beta frequency bands since this is the only metric that has been used in the social anxiety literature. We reviewed studies that have

employed a similar experimental design as reviewed for the frontal alpha asymmetry studies (e.g., comparing resting state, as well as activity during anticipation of and recovery from a socially stressful situation).

Neural oscillations in the delta frequency range (1 to 3 Hz) are slow-wave oscillations that are hypothesized to stem from subcortical regions, whereas neural oscillations in the beta range (13 to 30 Hz) are fast-wave oscillations that are hypothesized to stem from cortical regions (Miskovic et al., 2011; Putman, Arias-Garcia, Pantazi, & Van Schie, 2012; Schutter & Knyazev, 2012; Schutter, Leitner, Kenemans, & Van Honk, 2006; Schutter & Van Honk, 2005; Velikova et al., 2010). It is posited that the cross-frequency correlation between slow- and fast-wave oscillations acts as an electrophysiological signature of the crosstalk between cortical and subcortical brain regions (Schutter & Knyazev, 2012). This is endorsed by a source localization analysis revealing that delta-beta correlation is associated with activity in the orbitofrontal and anterior cingulate cortex (Knyazev, 2011). Several studies have shown that positive delta-beta correlation is increased in anxious states, and interpreted this as increased communication between cortical and subcortical brain regions (Schutter & Knyazev, 2012). Delta-beta correlation was increased in anxiogenic situations in individuals scoring both high and low on general anxiety (Knyazev, Schutter, & Van Honk, 2006). Another study showed that participants with the largest increase in positive delta-beta correlation in an anxiogenic situation, also tended to have higher state anxiety scores (Knyazev, 2011). In contrast, Putman (2011) found no relation between delta-beta correlation and behavioral inhibition. So, some caution in interpreting delta-beta correlation is warranted, because there are some contradicting results, most research comes from one research group, the functional role of amplitude-amplitude coupling is unclear (Canolty & Knight, 2010), and it could be debated whether delta power solely reflects subcortical activity (Amzica & Steriade, 2000; Blaeser, Connors, & Nurmikko, 2017; Harmony, 2013).

3.4 Delta-beta cross-frequency correlation in social anxiety

3.4.1 Rest

The findings about delta-beta correlation at rest are mixed. Miskovic et al. (2011) showed that delta-beta correlation before cognitive-behavioral treatment was higher than after treatment in patients with SAD. However, when pretreatment delta-beta correlation of patients with SAD was post hoc compared with controls, there was no difference (Miskovic et al., 2011). Delta-beta correlation was increased in high compared to low behaviorally inhibited males (Van Peer, Roelofs, & Spinhoven, 2008). In contrast, two studies have reported no differences between high and low socially anxious individuals (Harrewijn et al., 2016; Miskovic et al., 2010). Overall, despite the small amount of studies, it seems that delta-beta correlation during resting state is not related to social anxiety.

3.4.2 Anticipation

As an electrocortical measure of social anxiety, delta-beta correlation seems more promising when socially anxious individuals are anticipating a socially stressful situation. That is, patients with SAD displayed increased positive delta-beta correlation during anticipation before treatment compared to low socially anxious individuals (post hoc comparison). This increased positive delta-beta correlation during anticipation in patients with SAD decreased after cognitive-behavioral treatment, and there was no difference between patients with SAD after treatment and low socially anxious individuals (Miskovic et al., 2011). High socially anxious individuals also displayed increased positive delta-beta correlation during anticipation compared to low socially anxious individuals (Miskovic et al., 2010). Another study has found increased negative delta-beta correlation in high compared to low socially anxious individuals (Harrewijn et al., 2016). The authors argue that negative

delta-beta correlation could still be interpreted as increased crosstalk between cortical and subcortical regions, only in a different direction. Negative delta-beta correlation possibly reflects the known imbalance between subcortical and cortical brain regions in general anxiety (Bishop, 2007), and more specifically in SAD (Bruhl, Delsignore, Komossa, & Weidt, 2014; Cremers et al., 2015; Miskovic & Schmidt, 2012). Together, these studies highlight the potential of delta-beta correlation as a sensitive electrocortical measure of SAD when individuals are anticipating a socially stressful situation.

3.4.3 Recovery

Despite the importance of post-event processing in social anxiety, only one study has examined delta-beta correlation during recovery from a socially stressful situation. In this study, Harrewijn et al. (2016) examined delta-beta correlation during recovery from giving a presentation about their positive and negative qualities. Results showed that high socially anxious individuals showed increased negative delta-beta correlation compared to low socially anxious individuals (Harrewijn et al., 2016). This effect was interpreted as reflecting the imbalance between cortical and subcortical regions during recovery (Harrewijn et al., 2016). This is in line with findings from cognitive-behavioral studies suggesting that socially anxious individuals engage in post-event rumination after a socially stressful situation (Brozovich & Heimberg, 2008; Clark & McManus, 2002). Thus, the addition of a recovery phase in social performance paradigms seems valuable, and future studies should validate whether delta-beta correlation during recovery is a possible electrocortical measure of SAD.

3.5 Discussion of spectral EEG measures

The studies reviewed above provide insight in the potential of frontal alpha asymmetry and delta-beta correlation as electrocortical measures of SAD. Based on the available studies,

it seems that delta-beta correlation is more strongly associated with SAD, relative to frontal alpha asymmetry.

Frontal alpha asymmetry during resting state and recovery was not related to social anxiety. However, frontal alpha asymmetry during anticipation appears to be a possible electrocortical measure of SAD, but only when the anxiety is extreme. This might suggest that frontal alpha asymmetry is not a trait-measure of SAD, but might be related to SAD in certain highly stressful states. Thibodeau, Jorgensen, and Kim (2006) have suggested that the mixed findings in alpha asymmetry literature could be related to comorbidity with depression. Unfortunately, only few studies in social anxiety have reported on depression as well. Two studies with participants with high levels of depression revealed an effect of social anxiety on frontal alpha asymmetry (Moscovitch et al., 2011; Schmidt et al., 2012). Beaton et al. (2008) found the relation between frontal alpha asymmetry and shyness when controlling for concurrent depression. In contrast, there was no effect of social anxiety in a sample with low levels of depression (Harrewijn et al., 2016).

Delta-beta correlation during anticipation and recovery appears to be more promising as a electrocortical measure of SAD. Functionally, delta-beta correlation is suggested to reflect the crosstalk between cortical and subcortical regions that is related to anxiety (Knyazev, 2011; Knyazev et al., 2006; Schutter & Knyazev, 2012). Indeed, source-localization analyses have shown that delta-beta correlation was associated with activity in the orbitofrontal and anterior cingulate cortex (Knyazev, 2011). Increased delta-beta correlation in social anxiety converges with fMRI studies that have found an imbalance between cortical and subcortical regions in general anxiety (Bishop, 2007), but also more specific in SAD (Bruhl et al., 2014; Cremers et al., 2015; Miskovic & Schmidt, 2012). This imbalance between cortical and subcortical regions also concurs with information processing biases that are found in cognitive-behavioral studies (Bögels & Mansell, 2004; Clark & McManus, 2002;

Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004). For example, increased anticipatory anxiety could be related to increased amygdala activation (Miskovic & Schmidt, 2012). However, some caution in this interpretation is warranted because the exact functional role of amplitude-amplitude correlation remains unclear (Canolty & Knight, 2010), it could be debated whether delta power solely stems from subcortical regions (Amzica & Steriade, 2000; Blaeser et al., 2017; Harmony, 2013), and most studies are performed by one research group. So, research on the exact meaning of delta-beta correlation, and independent replication of this effect is necessary. The effects were found in anticipation and recovery, which suggests that a certain level of stress-induction, or an anxious state, is necessary to find electrocortical measures of SAD.

4. ERPs related to information processing biases in social anxiety

To delineate electrocortical measures of SAD that are directly related to stimulus processing in face processing and cognitive conflict paradigms, we focused on ERP studies. ERPs are electrical potential changes in the brain that are time-locked to a certain stimulus and offer fine-grained information about the temporal dynamics of information processing (Koivisto & Revonsuo, 2010; Luck, 2005). ERPs provide objective insights into very early and late stages of stimulus processing (Luck, 2005). ERPs that are elicited as early as 100 ms after stimulus presentation are presumably modulated by physical characteristics of the stimulus rather than cognition (Herrmann & Knight, 2001; Luck, 2005). However, highly salient stimuli or changes in the order of stimulus presentation have been known to influence these early ERP components, reflecting stimulus-driven or bottom-up effects on attention (Knudsen, 2007; Luck, 2005). Early components that have been most frequently studied in social anxiety are the P1, N170 and P2.

In contrast, late ERP components are less influenced by variations in the physical characteristics of a stimulus, and reflect post-perceptual processing related to stimulus categorization, response selection/activation, and emotional reactivity evoked by stimuli (Eimer & Driver, 2001; Hajcak, MacNamara, & Olvet, 2010). These late ERP components mostly reflect top-down effects on attention (Luck, 2005), a process through which neuronal sensitivity to specific task-relevant stimuli is increased (Knudsen, 2007). Late components that have been frequently studied in social anxiety are the P3 and late positive potential (LPP).

Due to its ability to distinguish between these early and late processing stages, ERPs offer objective measures to examine information processing biases in social anxiety. Here we focused on ERP components that are elicited by explicit or implicit face processing (Table 2) and cognitive conflict (Table 3) paradigms.

4.1 Early ERP components in face processing paradigms

4.1.1 P1

The P1 is an early positive ERP component that peaks 90-110 ms after stimulus onset. The P1 was previously seen as a stimulus-driven response that is not influenced by intentions, goals, and tasks (Eimer & Driver, 2001; Luck, 2005). However, more recent studies show that attention does influence the P1, as amplitude of the P1 increases to stimuli in an attended location compared to stimuli in an unattended location (Luck & Kappenman, 2013). The effect of attention of the P1 is maximal at the lateral occipital lobe and has been associated with activation in the lateral occipitotemporal cortex (Luck & Kappenman, 2013). Moreover, P1 amplitudes are enhanced in response to emotional faces compared to neutral faces in healthy adults. This suggests that enhanced attention is recruited in response to threat-related stimuli, and might be related to activity in the extrastriate visual cortex as seen in fMRI studies (Vuilleumier & Pourtois, 2007).

In explicit tasks, in which attention to emotion is required to complete the task, increased P1 amplitude in response to faces seems to be related to social anxiety (Figure 2). Patients with SAD showed increased P1 amplitude in response to schematic faces (i.e., line drawings of faces with different emotional expressions) in an emotion identification task and in a modified Stroop task (Kolassa et al., 2009; Kolassa, Kolassa, Musial, & Miltner, 2007). Increased P1 amplitude in response to pictures of faces was found in high versus low socially anxious participants in a modified Stroop task and in an emotional oddball paradigm (Peschard, Philippot, Joassin, & Rossignol, 2013; Rossignol, Campanella, et al., 2012). In the emotional oddball paradigm, P1 amplitude was increased in response to emotional faces versus neutral faces in high socially anxious individuals, whereas in low socially anxious individuals P1 amplitude was increased only in response to angry faces (Rossignol, Campanella, et al., 2012). This result indicates that high socially anxious individuals show a global hypervigilance towards emotional faces (Rossignol, Campanella, et al., 2012). This increased P1 amplitude was not related to any behavioral measures.

Also, increased P1 amplitudes may not be specifically linked to social anxiety, since patients with spider phobia also showed increased P1 amplitude when identifying faces (Kolassa et al., 2009). Furthermore, high socially anxious individuals showed increased P1 amplitude in response to colored rectangles in a modified Stroop task (Peschard et al., 2013), which suggests that increased P1 amplitudes reflect a more generic novelty response rather than early allocation of attention towards faces.

The effect of group (SAD, spider phobia, healthy controls) on P1 amplitude just failed to reach significance in one study (Kolassa & Miltner, 2006). That is, P1 amplitude did not differ between patients with SAD, patients with spider phobia, and healthy controls in a modified Stroop task. However, scores on the fear survey schedule were positively related to P1 amplitude only in patients with SAD (Kolassa & Miltner, 2006). This might be a power

issue in this study, since only 19 patients with SAD were included. Most studies have shown that social anxiety is related to increased P1 amplitude in response to emotional faces in explicit tasks.

In implicit tasks, in which attention is directed to stimulus characteristics other than the emotional valence, increased P1 amplitude also seems to be related to social anxiety (Figure 2). Patients with SAD showed increased P1 amplitude in response to angry-neutral face pairs in a dot probe task, which was interpreted as an early hypervigilance to angry faces (Mueller et al., 2009). Patients with SAD showed an increased P1 amplitude in response to angry and neutral faces compared to happy faces in a face learning task, whereas controls did not show this effect of emotion (Hagemann, Straube, & Schulz, 2016). This might have been an novelty effect, the P1 effect was only present when the faces were shown for the first time, there was no effect of social anxiety on the P1 if the faces were shown for the second time in the test phase of this learning task (Hagemann et al., 2016). In the implicit condition of a modified Stroop task, patients with SAD showed increased P1 amplitude in response to all faces, compared to patients with spider phobia and healthy controls (Kolassa et al., 2007). High socially anxious individuals showed increased P1 amplitude in response to all faces in a dot probe task (Helfinstein, White, Bar-Haim, & Fox, 2008). P1 amplitude was also increased in the implicit condition of a modified Stroop task in high compared to low socially anxious individuals (Peschard et al., 2013), and in a spatial cueing task in individuals with high compared to low fear of negative evaluation (Rossignol, Campanella, Bissot, & Philippot, 2013; Rossignol, Philippot, Bissot, Rigoulot, & Campanella, 2012).

In contrast to previous studies, Rossignol, Fisch, Maurage, Joassin, and Philippot (2013) showed that high socially anxious participants had decreased P1 amplitude in response to faces in an attention-shifting paradigm. One reason for this contrasting finding might be that the stimuli are less threatening in this task, because they used faces and bodily postures of

artificial humans. Artificial humans might not convey the same social evaluative threat as real humans. Another reason might be that participants can direct less attention to the face or bodily posture in the study of Rossignol, Fisch, et al. (2013), because the cue has no function in the rest of the task. In most other studies, the faces indicated the location of the target in some trials (Helfinstein et al., 2008; Mueller et al., 2009; Rossignol, Campanella, et al., 2013; Rossignol, Philippot, et al., 2012). Also, this contradicting finding might be related to the overall slower response to targets in high socially anxious individuals in this task, since most other studies did not find behavioral differences between individuals with and without social anxiety (Hagemann et al., 2016; Kolassa et al., 2007; Mueller et al., 2009; Peschard et al., 2013; Rossignol, Campanella, et al., 2013; Rossignol, Philippot, et al., 2012). Furthermore, Kolassa and Miltner (2006) found no difference in P1 amplitude between patients with SAD, patients with spider phobia and healthy controls in the implicit condition of a modified Stroop task. However, as discussed above, this might be due to low power. Taken together, the majority of the reviewed studies provide evidence that social anxiety is related to increased P1 amplitude in implicit tasks.

The abovementioned studies all examined the P1 component in response to faces with a direct gaze. However, averted gazes might also elicit atypical electrocortical responses in socially anxious individuals due to their ambiguous nature (Schmitz, Scheel, Rigon, Gross, & Blechert, 2012). High socially anxious individuals showed increased P1 amplitude in response to viewing averted faces, although this finding did not reach statistical significance (Schmitz et al., 2012), possibly because the averted gazes were not threatening enough to elicit responses in high socially anxious individuals.

Two studies have focused on the P1 component in response to targets replacing the facial stimuli to measure whether the initial hypervigilance was maintained or followed by avoidance. On the one hand, in a dot-probe task, Mueller et al. (2009) showed *decreased* P1

amplitude in response to targets, interpreted as reduced processing of emotionally salient locations at later stages of stimulus processing. On the other hand, in a spatial cueing task, Rossignol, Campanella, et al. (2013) showed *increased* P1 amplitude in response to targets, interpreted as maintained attention to the location of emotional cues. These contradicting findings could be linked to different processing stages as there were timing differences between the two tasks. In addition, the task of Mueller et al. (2009) might require more attention, because participants had to compare the target with the fixation cross, instead of just responding to the target as in Rossignol, Campanella, et al. (2013). Future research should clarify the information processing biases in later phases of dot-probe or spatial cueing tasks.

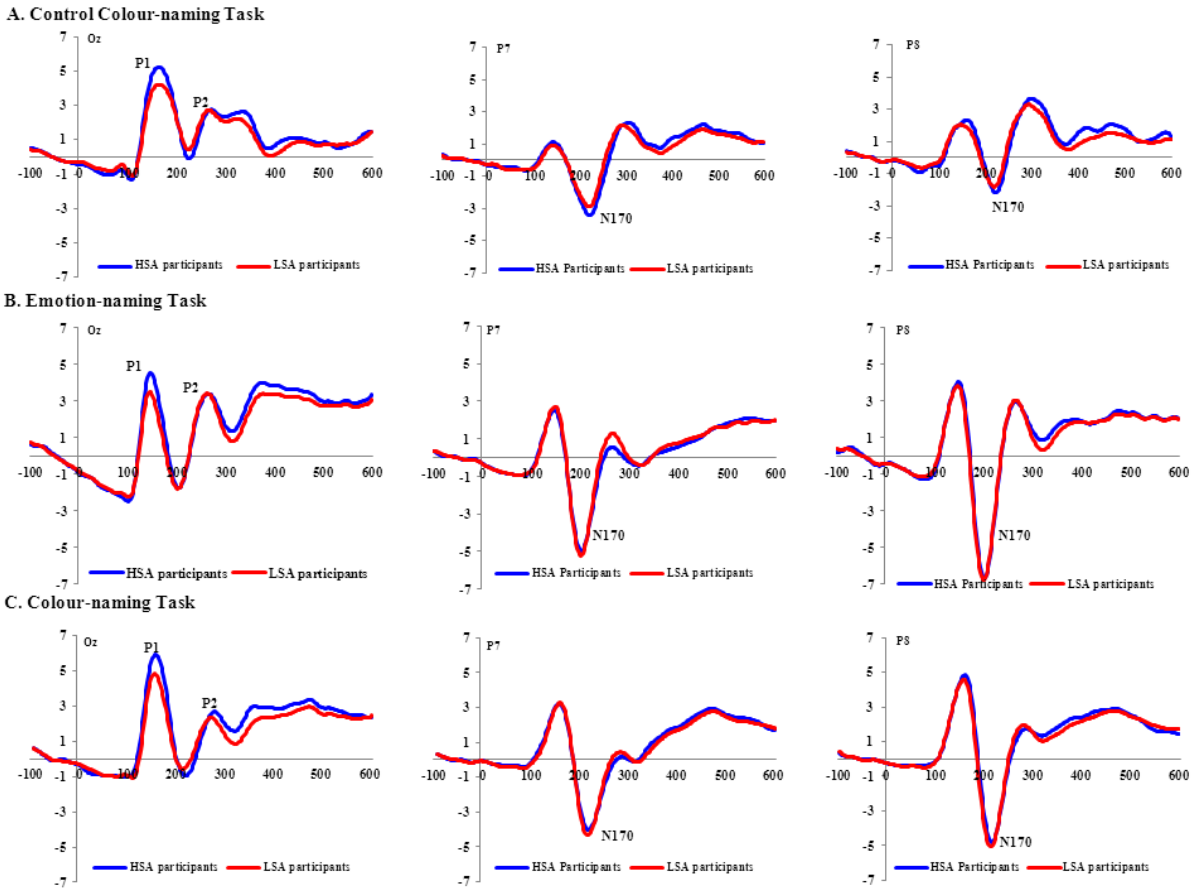

Figure 2. Social anxiety is related to increased P1 amplitude in response to explicit (emotion-naming task) and implicit tasks (color-naming task). High and low socially anxious individuals performed a modified Stroop task (3 conditions: color-naming of rectangles (A), emotion-naming of emotional faces (B), and color-naming of emotional faces (C)). Reprinted

from Biological Psychology, 93, Peschard, V., Philippot, P., Joassin, F, & Rossignol, M., The impact of the stimulus features and task instructions on facial processing in social anxiety: An ERP investigation, 88-96, Copyright (2013), with permission from Elsevier.

To conclude, most studies have shown that social anxiety is related to increased P1 amplitude. It should be noted that these studies have included relatively few participants (12 to 21 participants in the socially anxious groups), and the effect sizes are medium to high (η_p^2 ranging from 0.09 to 0.29). The relation between social anxiety and P1 amplitude is in line with the reviews of Staugaard (2010) and Schulz et al. (2013). The P1 is an early component that is mostly seen as a stimulus-driven or bottom-up response (Luck & Kappenman, 2013). Increased P1 amplitude to emotional faces is suggested to reflect enhanced attention to threat-related stimuli (Vuilleumier & Pourtois, 2007). Given these functions of the P1, SAD might be related to information processing biases with underlying mechanisms linked to attention to threatening social stimuli in early phases of stimulus processing. Indeed, cognitive-behavioral studies have shown that SAD is related to hypervigilance to threatening stimuli (Bögels & Mansell, 2004; Clark & McManus, 2002; Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004; Morrison & Heimberg, 2013), and the P1 component might be the electrocortical measure of this early hypervigilance.

According to Jetha, Zheng, Schmidt, and Segalowitz (2012), the P1 component in response to emotional faces might be related to amygdala sensitivity to fear-related emotional faces. That is, the amygdala might have a causal role in fear processing as indexed by the P1 component (Rotshtein et al., 2010). The P1 component in response to fearful versus neutral faces was decreased in pre-operative patients with medial temporal lobe epilepsy, and patients with more severe amygdala damage showed lower P1 amplitudes (Rotshtein et al., 2010). In line with this hypothesis, fMRI studies in socially anxious individuals have shown increased

amygdala activation in response to emotional faces (Miskovic & Schmidt, 2012; Schulz et al., 2013). So, this increased amygdala activation when viewing emotional faces, might be related to increased P1 amplitude. On the other hand, Mattavelli, Rosanova, Casali, Papagno, and Lauro (2016) showed that the medial prefrontal cortex influenced P1 amplitude during emotional face processing. They applied transcranial magnetic stimulation to the medial prefrontal cortex and found that P1-N1 amplitude in the right hemisphere decreased in response to happy and neutral faces (and not in fearful faces) during an explicit task. The authors suggested an early influence of top-down processing on face processing (Mattavelli et al., 2016). fMRI studies have also shown activation of the medial prefrontal cortex during face processing, albeit less substantial than amygdala activity (Miskovic & Schmidt, 2012; Schulz et al., 2013). Future research should clarify the influence of the amygdala and/or medial prefrontal cortex on P1 amplitude during face processing.

4.1.2 N170

The N170 is an early negative deflection in the ERP and is thought to measure early perceptual encoding and face categorization. This interpretation of the N170 seems contradictory to the early P1 findings in response to emotional faces. However, Vuilleumier and Pourtois (2007) interpret the P1 in response to faces as an index of rapid emotional processing based on crude visual cues, and the N170 as an index of full visual categorization. The N170 peaks 130-200 ms after stimulus onset and is predominantly distributed at occipitotemporal electrodes (Luck, 2005; Pratt, 2013; Rossion & Jacques, 2013). Some studies have found that N170 amplitude is related to emotional expressions, whereas others have not found this sensitivity to emotion (for a review, see Vuilleumier & Pourtois, 2007).

In explicit tasks, the N170 does not seem to be modulated by social anxiety. Patients with SAD, patients with spider phobia and controls showed no differences in N170 amplitude

in response to schematic faces in an emotion identification task and in a modified Stroop task (Kolassa et al., 2009; Kolassa et al., 2007). In response to pictures of emotional faces, N170 amplitude did not differ between high and low socially anxious participants in a modified Stroop task (Peschard et al., 2013) and in an emotional oddball paradigm (Rossignol, Campanella, et al., 2012). Only one study revealed increased N170 amplitude at right temporo-parietal electrodes when identifying angry faces in a modified Stroop task in patients with SAD compared to patients with spider phobia and healthy controls (Kolassa & Miltner, 2006). This contradicting finding could be caused by the use of more personal and ecologically valid stimuli in the study of Kolassa and Miltner (2006). They presented pictures of the entire face (Kolassa & Miltner, 2006), whereas other studies presented schematic (Kolassa et al., 2009; Kolassa et al., 2007) or trimmed faces without ears and hair (Peschard et al., 2013; Rossignol, Campanella, et al., 2012). However, most explicit tasks showed no influence of social anxiety on N170 amplitude.

N170 amplitude was also not modulated by social anxiety during tasks, in which participants' attention should be focused on stimulus characteristics other than emotion (implicit tasks). Patients with SAD showed no difference in N170 amplitude in the learning and test phases of a face learning task, compared to controls (Hagemann et al., 2016). Patients with SAD, patients with spider phobia and healthy controls also showed no difference in N170 amplitude in the implicit condition of a modified Stroop task with faces (Kolassa & Miltner, 2006), and with schematic faces (Kolassa et al., 2007). Studies reported no difference in N170 amplitude between high and low socially anxious individuals in an attention-shifting paradigm (Rossignol, Fisch, et al., 2013), in the implicit condition of a modified Stroop task (Peschard et al., 2013), and in a viewing task with direct and averted eye gazes (Schmitz et al., 2012), and between individuals with high and low fear of negative evaluation in a spatial cueing task (Rossignol, Campanella, et al., 2013). Only one study contradicts this finding, by

showing decreased N170 amplitude in patients with SAD in response to emotional faces in a dot-probe task (Mueller et al., 2009). However, they included only 12 patients with SAD, which might have been statistically underpowered (although the effect size was large, η_p^2 = 0.20). Furthermore, this dot-probe task was probably more difficult than the other dot-probe tasks, and therefore not comparable. That is, in Mueller et al. (2009), patients with SAD had to compare the target with the fixation cross, instead of reporting on only one aspect of the target, such as the location, or direction (Rossignol, Campanella, et al., 2013; Schmitz et al., 2012). Therefore, we conclude that social anxiety does not influence N170 amplitude in implicit tasks.

In sum, social anxiety is not related to N170 amplitude in both explicit and implicit face processing paradigms. Social anxiety also had no influence on behavioral performance in most of these studies. Only one study showed that high socially anxious individuals responded slower to the target than low socially anxious individuals in an attention-shifting paradigm (Rossignol, Fisch, et al., 2013). Patients with SAD and patients with spider phobia rated the angry schematic faces as more arousing, but they did not show differences in valence ratings, emotional classifications and reaction times (Kolassa et al., 2009). In his review, Staugaard (2010) concluded that differences between high socially anxious individuals and controls were mainly visible in the early P1 and N170 component. However, here we update this conclusion by showing that social anxiety is related to increased P1 amplitude, but not to changes in N170 amplitude, as most of the studies presented in the previous review of Staugaard (2010) were dated. Given that the N170 component in response to faces is not different between SAD and healthy controls, this implies that the N170 is not related to hypervigilance or threat detection strategies in socially anxious individuals.

4.1.3 P2

The P2 is a positive ERP component that peaks 150-250 ms after stimulus onset at anterior scalp sites (Luck, 2005). The P2 is an early electrocortical index of selective attention. That is, the P2 is increased in response to targets relative to non-targets or homogeneous stimuli. The P2 component is responsive to specific stimulus features, and is often increased in response to an infrequent target stimulus (Hajcak, Weinberg, MacNamara, & Foti, 2013; Luck, 2013). The P2 component is also associated with affective evaluation: P2 amplitude is typically increased in response to pleasant or unpleasant stimuli compared to neutral stimuli (Hajcak et al., 2013). Indeed, P2 amplitude was increased in response to emotional faces, which was interpreted as reflecting the rapid representation of emotional importance in prefrontal regions (Eimer & Holmes, 2007; Moser, Huppert, Duval, & Simons, 2008).

The P2 component seems to be unrelated to social anxiety when participants are asked to focus their attention on the emotional expression of a face. P2 amplitude did not differ between patients with SAD, patients with spider phobia and controls for happy, angry, and neutral faces in a modified Stroop task (Kolassa & Miltner, 2006), nor for schematic faces that changed from neutral to gradually more angry, happy and sad faces in an emotion identification task (Kolassa et al., 2009). Furthermore, during a modified Stroop task, high socially anxious individuals did not differ in P2 amplitude from low socially anxious individuals (Peschard et al., 2013). Differences between high and low socially anxious individuals appeared only during a modified version of the Eriksen flanker task. Low socially anxious individuals displayed increased P2 amplitude in response to flankers consisting of happy or surprised compared to angry or disgusted faces, which was interpreted as a positive bias. High socially anxious individuals did not show this positive bias (Moser et al., 2008). However, it should be noted that this interaction was only significant at trend level (η_p^2 = 0.08), and was mainly driven by the effect in controls. In the other tasks, there was also no

effect of emotion of the face in socially anxious individuals (Kolassa et al., 2009; Kolassa & Miltner, 2006; Peschard et al., 2013). The P2 results were unrelated to behavioral performance in these explicit tasks.

The results of implicit tasks on the relation between social anxiety and P2 amplitude are mixed. On one hand, in spatial cueing tasks, individuals with high fear of negative evaluation showed an increased P2 amplitude compared to individuals with low fear of negative evaluation in response to neutral, angry, disgusted, and happy faces (Rossignol, Philippot, et al., 2012), and in response to angry-neutral compared to fear-neutral face pairs (Rossignol, Campanella, et al., 2013). Helfinstein et al. (2008) found a trend towards increased P2 amplitude in high compared to low socially anxious individuals in a dot-probe task. On the other hand, patients with SAD and controls showed no difference in P2 amplitude in the learning and testing phases of a face learning task (Hagemann et al., 2016). There was also no difference in P2 amplitude in the implicit condition of a modified Stroop task between patients with SAD, patients with spider phobia, and healthy controls (Kolassa & Miltner, 2006) and high and low socially anxious individuals (Peschard et al., 2013). In an attention-shifting paradigm with pictures of artificial humans (faces and bodily posture), Rossignol, Fisch, et al. (2013) found an overall decrease in P2 amplitude in high versus low socially anxious individuals. However, there was also no difference in P2 amplitude between high and low socially anxious individuals in a change detection task, though P2 amplitude was negatively correlated with task performance in self-focus trials in high socially anxious individuals (Judah, Grant, & Carlisle, 2016). Taken together, social anxiety was related to increased P2 amplitude in spatial cueing and dot-probe tasks (Helfinstein et al., 2008; Rossignol, Campanella, et al., 2013; Rossignol, Philippot, et al., 2012), although these studies included only few participants (12-14 participants) in the socially anxious groups. Social anxiety was not related to increased P2 amplitude in attention-shifting, face learning, change

detection and Stroop tasks (Hagemann et al., 2016; Judah, Grant, & Carlisle, 2016; Kolassa & Miltner, 2006; Peschard et al., 2013; Rossignol, Fisch, et al., 2013). Social anxiety is unrelated to task performance in most of these studies, with the exception that high socially anxious individuals respond slower to targets in the attention-shifting paradigm (Rossignol, Fisch, et al., 2013).

These findings suggest that the sensitivity of the P2 component as a measure of SAD seems to depend on explicit vs. implicit task instructions. During explicit tasks, there was no effect of social anxiety on P2 amplitude, suggesting that all participants mobilized their attentional resources to the same extent and showed the same level of emotional evaluation. However, in implicit spatial cueing and dot-probe tasks, individuals with social anxiety showed increased P2 amplitude, whereas individuals without social anxiety did not process the emotional faces when they were not required to. Functionally, the P2 component is an index of selective mobilization of attentional resources to certain stimuli (Hajcak et al., 2013; Luck, 2013). Thus, in specific implicit tasks, enhanced P2 amplitude might be related to an early emotional evaluation of affective stimuli. This coincides with information processing biases reported in cognitive-behavioral studies, which show that SAD is related to a focus on negative information (Bögels & Mansell, 2004; Clark & McManus, 2002; Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004). Nevertheless, this effect should first be replicated in future studies with more participants.

4.2 Late ERP components in face processing paradigms

4.2.1 P3

The P3 is a positive deflection in the ERP typically observed 300-500 ms after stimulus onset and is distributed at frontocentral and centroparietal scalp sites (Hajcak et al., 2013; Polich, 2007). P3 amplitude is enhanced in response to infrequent targets in classic

oddball paradigms, but is also sensitive to the amount of attention given to a stimulus (Luck & Kappenman, 2013; Polich, 2013). Polich (2007) proposed that the P3 comprises two subcomponents: the earlier component – P3a – has a frontocentral scalp topography, and is implicated in novelty detection (Friedman, Cycowicz, & Gaeta, 2001; Herrmann & Knight, 2001); the later component – the P3b – has a centroparietal scalp topography, and reflects the voluntary shift in attention towards target stimuli (Herrmann & Knight, 2001). According to Polich (2007), this 'family' of P3 components is thought to subserve a neural mechanism implicated in inhibiting extraneous brain activation to enhance the allocation of sufficient attentional resources during stimulus detection (P3a), and this process is guided by the contents of working memory specific to the task at hand (P3b). Emotional stimuli are also known to modulate the P3 (Hajcak et al., 2013). In the social anxiety literature, the paradigms employed typically generated the P3b component (hereafter referred to as the P3), but when appropriate we distinguish between the P3a and P3b.

Most studies that have used explicit tasks to measure the P3 component have found no effect of social anxiety. For instance, there was no difference in P3 amplitude between patients with SAD, patients with spider phobia and controls in response to schematic faces in a modified Stroop task (Kolassa et al., 2007). There was also no difference in P3 amplitude between high and low socially anxious individuals in an emotional oddball task (Rossignol, Campanella, et al., 2012). These two studies showed no effect of social anxiety on behavioral performances. In addition, P3 amplitude did not differ between individuals with high and low fear of negative evaluation in an identification task (Rossignol, Anselme, Vermeulen, Philippot, & Campanella, 2007), and between high and low behaviorally inhibited males in an approach-avoidance task (Van Peer et al., 2007). However, social anxiety had an influence on behavior in these tasks. Individuals with high fear of negative evaluation detected disgusted faces before angry faces in all conditions, whereas individuals with low fear of negative

evaluation did not show this differentiation (Rossignol et al., 2007). Individuals with high behavioral inhibition showed more state anxiety and tension during the task, but no differences in task performance (Van Peer et al., 2007). Only one study has found an effect of social anxiety on P3 amplitude in an emotional oddball task (Sewell, Palermo, Atkinson, & McArthur, 2008). That is, healthy participants were presented with happy, angry and neutral faces that were displayed in an upright and inverted position. Self-reported social anxiety was positively related to P3 amplitude in response to upright-presented, angry faces, suggesting an attentional bias towards processing threatening faces (Sewell et al., 2008). This contradicting finding might be related to task instructions to selectively focus on angry or happy faces, and analysis of only the unattended faces (Rossignol, Campanella, et al., 2012; Sewell et al., 2008). Taken together, it seems that social anxiety does not modulate the P3 component.

For implicit tasks, there seems to be no effect of social anxiety on P3 amplitude. P3 amplitude did not differ between patients with SAD and controls in the implicit condition of a modified Stroop task with schematic faces (Kolassa et al., 2007), nor between high and low socially anxious individuals in an attention-shifting paradigm (Rossignol, Fisch, et al., 2013), and individuals with high and low fear of negative evaluation in a spatial cueing task (Rossignol, Philippot, et al., 2012). Social anxiety affected task performance in the attention-shifting paradigm, showing that high socially anxious individuals responded overall slower to targets than low socially anxious individuals (Rossignol, Fisch, et al., 2013).

To conclude, there is no effect of social anxiety on the P3 component in explicit and implicit tasks, which corroborates prior discussion of the P3 in social anxiety (Staugaard, 2010). The P3 component is an index of the voluntary shift in attention towards target stimuli (Herrmann & Knight, 2001) and is also related to emotional content (Hajcak et al., 2013). The findings suggest that social anxiety is not related to an altered voluntary shift in attention, nor to aberrant processing of emotional content as indexed by the P3 component.

4.2.2 LPP

Studies that examined ERPs in response to the emotional content of stimuli have often found a positive deflection extending the traditional time-window of the P3. This component is coined the LPP, a sustained positive deflection that could last for seconds (Hajcak et al., 2013). The LPP is suggested to reflect the encoding and storage of intrinsically motivating stimuli, as it is larger after pleasant and unpleasant stimuli compared to neutral stimuli (Hajcak et al., 2010; Hajcak et al., 2013). Additionally, the LPP has been related to emotion regulation (Hajcak et al., 2010; Hajcak et al., 2013).

In explicit tasks, there are contradicting results regarding the LPP. For example, LPP amplitude was increased in angry or disgusted target faces in a modified version of the Erikson flanker task in high versus low socially anxious participants (Moser et al., 2008), whereas no difference in LPP amplitude was found in a modified Stroop task between patients with SAD, patients with spider phobia and controls in response to schematic faces (Kolassa et al., 2007). This difference might be related to arousal: Kolassa et al. (2007) used schematic stimuli that could be less arousing than real pictures, and Moser et al. (2008) showed 3 faces at the same time (a target face and two flanking faces) which could be more threatening for participants.

In an implicit face learning task, the LPP at a right central scalp site was increased in patients with SAD in response to learned versus novel faces task, but not in controls. However, this effect was the same for patients with SAD and controls in the left central or other parietal scalp sites (Hagemann et al., 2016). The LPP was also increased in response to faces with averted gaze compared to faces with direct gaze in high versus low socially anxious individuals (Schmitz et al., 2012). This result was interpreted to show the facilitated

processing of negative stimuli during more detailed and sustained processing stages (Schmitz et al., 2012).

Most of these studies have found that social anxiety is related to an increased LPP, in absence of behavioral differences. This might suggest that social anxiety is related to increased processing of intrinsically motivating stimuli, and/or emotion regulation (Hajcak et al., 2010; Hajcak et al., 2013). However, this suggestion should be confirmed in future studies since only few studies focused on the LPP in social anxiety and the effect sizes are medium (η_p^2 ranging from 0.07 to 0.13).

4.3 ERP components in cognitive conflict paradigms

A recent and very relevant line of ERP research in social anxiety has focused on ERP components that are related to feedback processing and conflict monitoring. In general, these studies assume that the socially anxious brain shows aberrant processing of cues that communicate performance errors or social rejection. Indeed, cognitive-behavioral studies revealed that socially anxious individuals are sensitive to signs that could convey social threat (Bögels & Mansell, 2004; Clark & McManus, 2002; Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004). ERP components of interest are typically a class of medial-frontal negativities related to cognitive and attentional control, including the N2, FRN, ERN, and CRN, and the Pe (Gehring, Liu, Orr, & Carp, 2013; Van Noordt, Desjardins, & Segalowitz, 2015; Van Noordt & Segalowitz, 2012).

4.4.1 N2

The N2 is a negative component that peaks 200-350 m after stimulus presentation, and, depending on the task, has a frontocentral or centroparietal scalp distribution. It is proposed that the N2 component consists of at least three subcomponents: a frontocentral

component associated with cognitive control, a frontocentral component associated with novelty or mismatch, and a posterior component associated with visual attention (Folstein & Van Petten, 2008).

First, the frontocentral N2 related to cognitive control did not differ between high and low socially anxious individuals in a modified version of the Eriksen flanker task (Moser et al., 2008), nor between individuals with high and low behavioral inhibition in a approach-avoidance task (Van Peer et al., 2007). The latter task showed increased state anxiety and tension in individuals with high behavioral inhibition, but no differences in task performance (Van Peer et al., 2007). Second, the frontocentral N2 related to novelty or mismatch was decreased in individuals with high fear of negative evaluation while detecting change in the intensity of anger during an emotional oddball task (Rossignol et al., 2007). Individuals with high fear of negative evaluation detected disgust before anger in all conditions, whereas individuals with low fear of negative evaluation did not show this pattern. However, it should be noted that only few individuals with high fear of negative evaluation (n = 10) participated (Rossignol et al., 2007). Third, the more posterior N2 component in response to the target tone in a standard two-tone oddball paradigm was increased in patients with SAD compared to controls (Sachs et al., 2004). These few studies suggest that social anxiety is differentially related to various types of the N2 component, but this should be confirmed in future research.

4.4.2 FRN

The FRN is a frontocentral negative deflection peaking around 250-300 ms after a feedback stimulus (Gehring et al., 2013). The FRN component is increased when feedback is unexpected or reflects poor performance (Van Noordt & Segalowitz, 2012). However, recent studies showed that depending on the likelihood of an outcome, the FRN component might be sensitive to both negative and positive information (Ferdinand, Mecklinger, Kray, & Gehring,

2012; Oliveira, McDonald, & Goodman, 2007). Cao, Gu, Bi, Zhu, and Wu (2015) found that patients with SAD displayed an increased FRN in response to acceptance feedback from peers. This was interpreted to reflect a violation of negative feedback expectancies, since socially anxious participants anticipated a larger proportion of negative peer feedback in this study (Cao et al., 2015). A difficulty with this interpretation is that expectancies were not recorded during the EEG experiment (on a trial-to-trial basis), but as an overall Likert-scale measure prior to the task to index general expectancies about the social evaluative outcome. Van der Molen et al. (2014) did measure participants' expectancy per trial during EEG recording, but did not find an association between the FRN and social anxiety. The FRN was only sensitive to feedback that violated participants' expectancies (Van der Molen et al., 2014). Further, the FRN did not differ in amplitude between high and low socially anxious individuals in trial-and-error learning task. There was only a marginal difference in FRN amplitude before learning between high and low socially anxious individuals when participants received false feedback about increased heart rate (to increase self-focus) (Judah, Grant, Frosio, et al., 2016). Taken together, studies have found mixed findings on the influence of social anxiety on the FRN component. A possible FRN effect might be related to the severity of symptoms, since the effect is significant in patients with SAD (Cao et al., 2015), marginally significant in high socially anxious individuals (Judah, Grant, Frosio, et al., 2016), and not significant in healthy participants (Van der Molen et al., 2014).

4.4.3 ERN

The ERN (or error negativity (Ne)) is a frontocentral negative deflection in the ERP that typically occurs about 50 ms after people make mistakes (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). Many studies have linked the ERN to activity in the anterior cingulate cortex (Holroyd & Coles, 2002; Van Veen & Carter, 2002; Yeung & Cohen, 2006), an

important hub in the conflict monitoring network (Yeung & Cohen, 2006). Functionally, the ERN seems to reflect an error monitoring system, but it remains uncertain whether the ERN reflects a conscious or unconscious process of error detection (Wessel, 2012). It has been shown that the ERN is sensitive to motivational relevance of errors and individual differences in trait affect (Larson, Clayson, & Clawson, 2014). For example, ERN amplitudes are larger in individuals with perfectionistic or anxious tendencies, a finding that has been interpreted to reflect chronic conflict detection due to pathological worrying (Moser, Moran, & Jendrusina, 2012; Weinberg, Olvet, & Hajcak, 2010). In addition, the ERN is sensitive to social motivational factors, when performance is evaluated by others (Hajcak, Moser, Yeung, & Simons, 2005; Van Meel & Van Heijningen, 2010).

Patients with SAD showed an increased ERN compared to controls in a flanker task (see Figure 3) (Endrass, Riesel, Kathmann, & Buhlmann, 2014; Kujawa et al., 2016). An interesting finding was that the augmented ERN in SAD patients (children and adults) in the Kujawa et al. (2016) study persisted after SAD patients received treatment (i.e., cognitive-behavioral therapy or SSRI pharmacological treatment), suggesting these treatment options have little effect on desensitizing the error-detection mechanism in SAD. The ERN was also larger in high compared to low socially anxious individuals in a trial-and-error learning task, in which participants learned stimulus-response mappings (Judah, Grant, Frosio, et al., 2016). Sensitivity of the ERN to performance evaluation by a peer was recently shown in a study by Barker, Troller-Renfree, Pine, and Fox (2015). In this study, high and low socially anxious individuals performed a flanker task in two different conditions: alone or under peer observation. Results indicated that high socially anxious individuals showed larger ERN amplitudes when they were observed rather than when they were alone (Barker et al., 2015).

Several explanations have been offered for the increased ERN in SAD. For example, Kujawa et al. (2016) argued that patients with SAD monitor their own behavior more closely

and are more sensitive to errors. This could be related to increased self-focused attention in social situations (Bögels & Mansell, 2004; Clark & McManus, 2002), but also to perfectionism as shown by the tendency to uphold high performance standards by patients with SAD (Clark & Wells, 1995). Alternatively, Moser, Moran, Schroder, Donnellan, and Yeung (2013) suggested that increased ERN amplitude in anxious apprehension might be related to processing inefficiency, caused by increased cognitive load, and increased compensatory mechanisms. Although this interpretation was not specific for SAD, it suggests that individuals with SAD are more distracted by their errors and need to use compensatory mechanisms. At the behavioral level, a candidate compensatory mechanism is post-error slowing – a well-known increase in reaction time observed on the trial following an error (Danielmeier & Ullsperger, 2011; Gehring & Fencsik, 2001). Surprisingly, however, few studies have reported on post-error slowing in SAD, but the provisional evidence available suggests no significant differences in post-error slowing between SAD participants and controls (Endrass et al., 2014). Additionally, these reviewed ERN studies did not provide evidence of task performance differences (e.g., number of trials correct or % errors) between SAD and control participants, an observation that speaks to the notion that the augmented ERN in SAD might be reflecting a sensitive error-detection process, rather than an error compensation mechanism. However, examining behavioral measures such as post-error slowing in future ERN studies on SAD should validate this suggestion. Finally, it should be noted that only few studies have focused on the ERN component in relation to social anxiety, though the effect sizes are large for patients with SAD ($\eta_p^2 = 0.12$ in Kujawa et al. (2016) and $\eta_p^2 = 0.16$ in Endrass et al. (2014)) and medium for high socially anxious individuals ($\eta_p^2 = 0.08$ in Judah, Grant, Frosio, et al. (2016), and $\eta_p^2 = 0.11$ in Barker et al. (2015)). Thus, increased ERN amplitude appears to be a promising electrocortical measure of SAD.

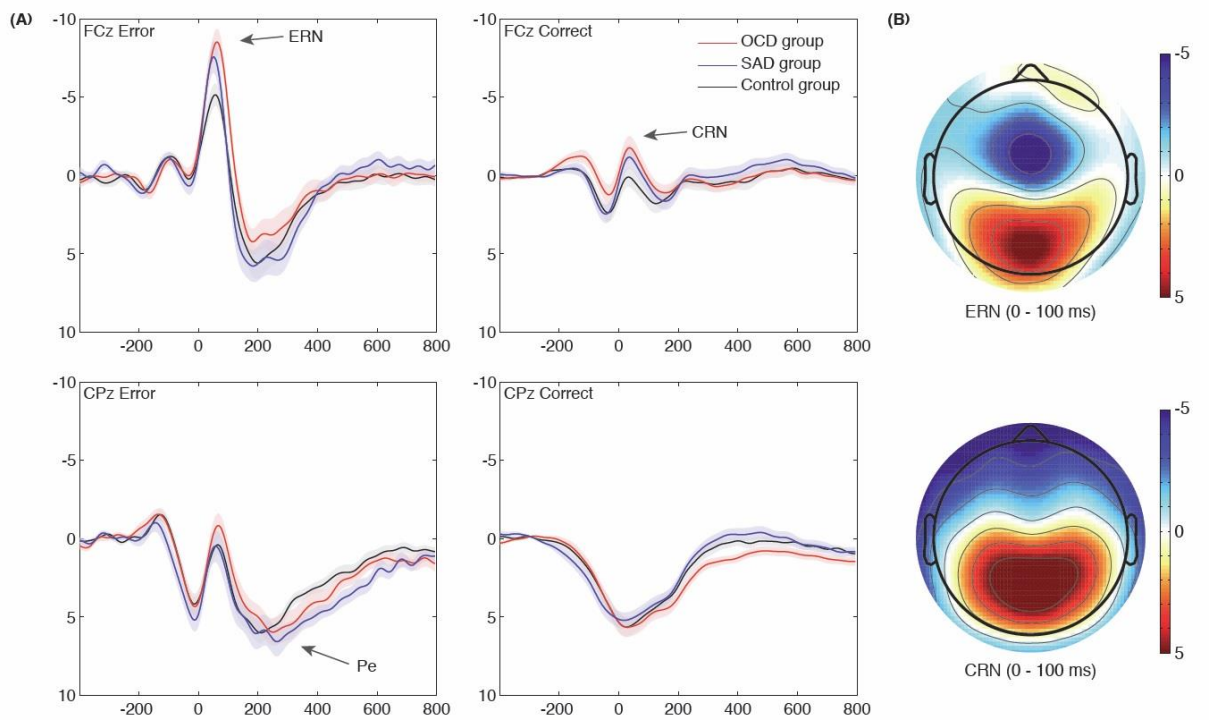

Figure 3. Social anxiety is related to increased ERN in patients with SAD and obsessive-compulsive disorder after errors in a flanker task. Note: negative values are plotted upwards. Reprinted from Journal of Abnormal Psychology, 123, Endrass, T., Riesel, A., Kathman, N., & Buhlmann, U., Performance monitoring in obsessive-compulsive disorder and social anxiety disorder, 705-714, Copyright (2014), with permission from American Psychological Association.

4.4.4 CRN

The CRN is often studied concurrently with the ERN. The CRN resembles the ERN (negative deflection 50 ms after feedback), but is measured in response to correct rather than incorrect responses. The CRN component is usually smaller than the ERN component, but has a similar frontocentral scalp distribution (Gehring et al., 2013). Patients with SAD showed increased CRN amplitude in a flanker task (Endrass et al., 2014), and high socially anxious individuals showed increased CRN amplitude in a trial-and-error learning task (Judah, Grant, Frosio, et al., 2016). Moser et al. (2008) found no overall increased CRN amplitude in high

socially anxious individuals. Nevertheless, high socially anxious individuals showed no difference in flanker interference effect in the CRN component between threatening and reassuring faces, whereas low socially individuals showed no flanker interference effect for threatening faces. This was interpreted as a positive bias that is lacking in high socially anxious individuals (Moser et al., 2008). In contrast, there was no difference in CRN amplitude between high and low socially anxious individuals in a flanker task performed alone nor when observed by a peer (Barker et al., 2015). Studies measuring both the ERN and CRN components have found that the effect of social anxiety on the ERN is larger than on the CRN (Barker et al., 2015; Endrass et al., 2014). Therefore, more studies are needed to draw conclusions about the possible influence of social anxiety on the CRN.

4.4.5 Pe

The Pe is also often studied in the same paradigms as the ERN and CRN. The Pe is a centroparietal, positive deflection 200-400 ms after an error, which might be related to an affective response, awareness, or adapting response strategies (Gehring et al., 2013). Most studies have shown no difference in Pe amplitude between patients with SAD and controls (Endrass et al., 2014) and between high and low socially anxious individuals (Barker et al., 2015) in flanker tasks. However, high socially anxious individuals showed marginally increased Pe amplitude compared to low socially individuals in a trial-and-error learning task. Furthermore, high socially anxious individuals showed a greater increase in Pe amplitude from trials before to after learning than low socially anxious individuals (Judah, Grant, Frosio, et al., 2016). The difference in these findings are probably related to the difference in tasks.

5. Discussion

The goal of this review was to give a comprehensive overview of the most frequently studied EEG spectral and ERP measures during rest, anticipation, stimulus processing, and recovery. Studies on EEG spectral characteristics have shown that delta-beta correlation during anticipation and recovery is a promising electrocortical measure, possibly reflecting the alleged imbalance between cortical and subcortical brain regions (Bishop, 2007; Bruhl et al., 2014; Cremers et al., 2015; Miskovic & Schmidt, 2012). The ERP studies have shown information processing biases during early processing of faces and errors. Increased P1 amplitude in response to emotional faces is associated with social anxiety, reflecting hypervigilance to threatening stimuli (Bögels & Mansell, 2004; Clark & McManus, 2002; Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004; Morrison & Heimberg, 2013). Another electrocortical measure of SAD is increased ERN amplitude, possibly reflecting increased self-focused attention (Bögels & Mansell, 2004; Clark & McManus, 2002) or perfectionism (Clark & Wells, 1995). Finally, increased P2 amplitude was related to social anxiety, but only in implicit spatial cueing and dot-probe tasks. This might be related to a focus on negative evaluation as reported in cognitive-behavioral studies (Bögels & Mansell, 2004; Clark & McManus, 2002; Heinrichs & Hofmann, 2001; Hirsch & Clark, 2004). The reviewed studies did not provide evidence that frontal alpha asymmetry nor the N170, P3, LPP, N2, FRN, CRN and Pe components are electrocortical measures of SAD.

Cognitive-behavioral studies have proposed that SAD is maintained by a persistent cycle of information processing biases (Clark & McManus, 2002; Morrison & Heimberg, 2013). That is, attention biases are elicited by socially threatening stimuli, repeated while in the social situation, and carried forward over time by anticipation (Morrison & Heimberg, 2013). Indeed, we have shown that social anxiety is related to hypervigilance to threatening stimuli, such as faces and errors. Repetition within a social situation has not yet been studied, since ERPs are an average across multiple trials. The next step of the persistent cycle of

information processing biases – carried forward over time by anticipation – has only partly been studied. We have found that social anxiety is related to increased delta-beta correlation during anticipation and recovery, but it is unknown whether this carries the attention biases forward over time and thus plays a role in the maintenance of SAD. Such a mechanism has been found in healthy participants, where anticipatory anxiety before giving a speech enhanced early ERP responses to angry faces (Wieser et al., 2010), but remains to be established SAD. Taken together, increased amplitudes of the P1 to faces and the ERN to errors, and delta-beta correlation during anticipation and recovery might be possible electrocortical measures underlying the persistent cycle of information processing biases that maintains SAD. Future studies should investigate how hypervigilance is repeated within the situation, and whether is it carried forward over time during anticipation and recovery.

Another important avenue for future research is to investigate how these information processing biases are linked to behavior in patients with SAD. One important question is whether information processing biases during the early stages of stimulus processing (e.g. hyperviligance) trigger a cascade of biases during further processing stages. Most studies have focused on the ERPs individually, but it would also be important to know how the early biases influence later processing of stimuli. Another important question is how these information processing biases influence behavior. A promising field of research would be to examine whether ERN activity impacts subsequent decision-making (e.g., post-error slowing), which has only been scarcely studied in relation to SAD (Endrass et al., 2014). Future studies should continue this line of research in SAD, since such work would not only contribute to our understanding of information processing biases in SAD, but also into the psychological processes indexed by the ERN more generally. Another way of investigating the link with behavior is by using more ecologically valid paradigms, such as social performance tasks or social feedback tasks.

Electrocortical measures of SAD could be useful in research on early detection, prevention and treatment of SAD. Future studies should investigate whether amplitudes of the P1 and ERN, and delta-beta correlation can be used to identify persons at risk for developing SAD at a young age. Understanding the factors influencing the development of SAD in relation to functional brain development might be useful for developing preventive interventions. In addition, it would be valuable to know how such electrocortical measures could predict treatment response. For instance, it might be that persons who are sensitive to errors (those with an increased ERN component) need a different focus in treatment than persons who are displaying information processing biases during anticipation or recovery (those with increased delta-beta correlation). Recent studies with facial stimuli have shown that P1 amplitude might be a predictor of treatment outcome and N2 and LPP amplitudes might be predictors of treatment response in anxiety disorders (Bunford et al., 2017; Hum, Manassis, & Lewis, 2013). However, only very few electrocortical studies have focused on predicting treatment response in anxiety disorders (Lueken et al., 2016). Another interesting avenue for future research is to examine whether these electrocortical measures could help in unraveling the genetic basis of SAD. For example, these electrocortical measures can be tested as possible endophenotypes of SAD (Glahn, Thompson, & Blangero, 2007). This is a relatively new approach that has yielded promising results in depression and schizophrenia research (Bramon et al., 2005; Glahn et al., 2012; Glahn et al., 2007), and might be particularly fruitful in SAD research given the relatively high heritability (Isomura et al., 2015). Research on electrocortical measures of SAD should take the next step by validating these measures and studying how they could be used best to reduce social anxiety symptoms. In the following paragraphs, we discuss methodological and developmental considerations that should be addressed in future studies.

5.1 Methodological considerations

One issue that hampered delineating electrocortical measures of SAD is the diversity of experimental paradigms that have been used in the social anxiety literature. Furthermore, even when using similar paradigms, differences between ERP results can emerge due to the diversity in methodological strategies, such as ERP component scoring, filter and reference settings, the number of trials required to obtain the ERP of interest, and timing differences (J. Cohen & Polich, 1997; Hajcak et al., 2013). In addition, there are numerous inconsistencies in the names and definitions of electrocortical measures. For example, the often-used term 'cross-frequency coupling' could refer to different measures of electrocortical brain activity (Schutter & Knyazev, 2012). One of the challenges in cognitive electrophysiology is therefore to use unambiguous and consistent terminology (M. X. Cohen & Gulbinaite, 2014). It should also be noted that not all studies reported effect sizes, which makes it difficult to interpret and compare the effects of social anxiety across studies.

Future studies should also examine whether these electrocortical measures are specific to SAD. The studies reviewed above have focused mainly on participants with SAD or heightened symptoms of social anxiety. A few studies have already compared patients with SAD with patients with spider phobia as well as healthy controls (Kolassa et al., 2009; Kolassa et al., 2007; Kolassa & Miltner, 2006). However, specificity should also be studied by comparing patients with SAD and patients with other disorders that have a high comorbidity with SAD (such as generalized anxiety disorder or depression). Moreover, it should be investigated whether the electrocortical measures are specifically related to socially threatening stimuli (faces in most paradigms). Notably, high socially anxious individuals also displayed increased P1 amplitude in response to colored rectangles (Peschard et al., 2013), which questions the specificity of this electrocortical measure.

We have focused on constructs related to SAD, such as fear of negative evaluation, social withdrawal, shyness, and behavioral inhibition, because these constructs share common symptoms of SAD (Stein et al., 2004). However, some findings were only found in individuals characterized by these related constructs (e.g. the relation between shyness and right frontal cortical activity in Beaton et al. (2008)), which questions the generalizability of these findings to SAD. Given that not all shy and behaviorally inhibited individuals develop SAD (Spence & Rapee, 2016), future research should investigate which electrocortical measures are related to developing SAD. In addition, future research should also focus on the diagnostic utility of these electrocortical measures by investigating their specificity, sensitivity, and diagnostic value.

5.2 Developmental considerations

One of the objectives of examining electrocortical measures of SAD is to evaluate whether they can be used to detect individuals at risk for developing this debilitating disorder. Therefore, it is important to study these possible electrocortical measures in children. SAD has a relatively late onset and usually develops during early adolescence (Haller, Kadosh, & Lau, 2014), and early detection of SAD in younger children typically involves the assessment of personality/temperamental constructs that have been interpreted as precursors of the disorder (e.g., behavioral inhibition and shyness). However, the key question is whether the EEG measures associated with behavioral inhibition or shyness are also related to SAD, since not all children with these related constructs eventually develop SAD (Spence & Rapee, 2016). In addition, the integration of findings from adult and child studies is complex due to age related differences in spontaneous EEG activity and the need for different methodological approaches. While being aware of these concerns, we here shortly describe electrocortical studies that have included children that might be at risk of developing SAD (Table 4 and 5).

With respect to frontal alpha asymmetry studies, the pattern of findings observed in children mimics the inconsistencies in the adult literature. For example, Fox et al. (2001) showed that children classified as behaviorally inhibited at 4 months exhibited increased right frontal activity at 9 and 14 months of age. In healthy children, increased right frontal activity was related to socially inhibited behavior (Henderson, Fox, & Rubin, 2001; Henderson, Marshall, Fox, & Rubin, 2004). In contrast, others did not find an association between frontal alpha asymmetry and SAD-related constructs, such as shyness (Schmidt et al., 1999; Theal-Honey and Schmidt, 2006) or social withdrawal (Fox et al., 1995; Hannesdottir et al., 2010).

Notably, in contrast to the adult studies reviewed earlier, there is no evidence of an early hypervigilance towards threatening stimuli or novelty in children (as indexed by early ERPs). For example, studies examining face processing in behaviorally inhibited children (Thai, Taber-Thomas, & Perez-Edgar, 2016), as well as novelty detection in an auditory oddball paradigm in behaviorally withdrawn children (Bar-Haim, Marshall, Fox, Schorr, & Gordon-Salant, 2003) did not find evidence of early hypervigilance as indexed by the early ERPs.

Developmental studies focusing on late ERPs revealed mixed results. Some studies found an enhanced LPP in children and adolescents with SAD to emotional faces (Kujawa, MacNamara, Fitzgerald, Monk, & Luan Phan, 2015), and a larger P3 to target and standard tones in shy children (Tang, Santesso, Segalowitz, & Schmidt, 2016). However, the novelty P3 was not associated with shyness (Tang et al., 2016), or behavioral inhibition in adolescence (Reeb-Sutherland et al., 2009). Although, a combination of high behavioral inhibition and high P3 amplitudes to novel sounds in adolescence, indicative of heightened attentional orienting, were more likely to have clinical anxiety diagnoses (Reeb-Sutherland et al., 2009).

Developmental studies of ERPs in cognitive conflict paradigms report mixed findings on the N2 component. Shyness did not affect N2 amplitude in a three-stimulus auditory oddball task (Tang et al., 2016), nor in a flanker task (Henderson, 2010). However, high behaviorally inhibited children showed increased N2 amplitude during a flanker task, and a combination of high behavioral inhibition and increased N2 amplitude predicted more withdrawal and less assertiveness in a social exclusion task (Lahat, Walker, et al., 2014). In addition, behavioral inhibition was related to social reticence at age 7 in children who showed increased N2 amplitude during a Go-NoGo task (Lamm et al., 2014). Shy children with increased N2 amplitudes reported higher levels of social anxiety (Henderson, 2010). In behaviorally inhibited children, N2 amplitude predicted a bias away from angry faces in a dot-probe task (Thai et al., 2016).

In terms of the FRN, mixed findings have been reported in developmental studies. For example, Lackner, Santesso, Dywan, Wade, and Segalowitz (2014) found that shyness was related to a decreased FRN to monetary feedback (no difference between wins or losses), whereas Kessel, Kujawa, Proudfit, and Klein (2015) reported an increased difference in FRN between wins and losses in social anxiety. Kujawa, Arfer, Klein, and Proudfit (2014) found that a greater difference in FRN between social acceptance and social rejection feedback was related to social anxiety.

ERN amplitude was the only electrocortical measure that was consistently found across adult and child studies. Behaviorally inhibited children (Lahat, Lamm, et al., 2014) and adolescents (McDermott et al., 2009) demonstrated a larger ERN in a flanker task, and increased ERN amplitude in behaviorally inhibited adolescents was related to a higher risk for anxiety disorders (McDermott et al., 2009). Furthermore, differences between ERN and correct-response negativity amplitude in 7-year-old children predicted SAD symptoms at age 9 (Lahat, Lamm, et al., 2014). It should be noted however that the ERN is not specific to

SAD, but also found in other anxiety disorders in children (Wauthia & Rossignol, 2016). The CRN and Pe are each studied in only one developmental study and were not related to social anxiety (Lahat, Lamm, et al., 2014; McDermott et al., 2009).

Taken together, only the ERN component has been linked to social anxiety in both child and adult studies. This might suggest that the ERN could play a role in the early detection of SAD, although this should be confirmed in longitudinal studies. However, it should be noted that the studies in children and adults use different paradigms that render comparisons of the results and any long-term associations difficult. Accordingly, future studies should address the issues of measurement equivalence and adopt longitudinal designs to confirm the developmental associations. Nevertheless, these results speak to the importance of context to provide specificity in uncovering electrocortical measures of SAD. Contexts that involve social evaluation may be more salient for individuals who are socially anxious, particularly during adolescence – an important period for the development of SAD (Haller et al., 2014). Thus, brain functioning during social rejection or exclusion events in socially anxious individuals across development may provide more specific measures to understand the electrocortical mechanisms related to SAD.

6. Conclusion

In sum, social anxiety is related to delta-beta correlation during anticipation of and recovery from a socially stressful situation, increased P1 amplitude in response to processing emotional faces, and increased ERN amplitude after making errors. Together, these electrocortical measures might underlie the persistent cycle of information processing biases that maintains SAD. However, these electrocortical measures represent only a part of this persistent cycle, so future research should investigate repetition within the social situation and whether hypervigilance might be carried forward over time by information processing biases

during anticipation and recovery. The influence of early ERPs on later ERPs and the link between electrocortical measures and behavior should also be studied to gain more insight in the psycho(physio)logical mechanisms maintaining SAD. Given the abovementioned methodological and developmental concerns, we also call for studies that examine these electrocortical measures in larger samples using longitudinal designs. Such studies should validate these electrocortical measures and investigate whether these measures could (1) be identified at young age, (2) be used to prevent the development of SAD, (3) play a role in treatment of SAD (e.g. if they could predict treatment response), and (4) be seen as endophenotypes of SAD and thereby give insight in genetic mechanisms.

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Table 1

Overview of studies about frontal alpha asymmetry and delta-beta correlation in social anxiety.

Author	**Participants**	**Sex ratio of target group (F:M)**	**Protocol**	**Behavioral results** (socially anxious relative to control)	**EEG Results** (socially anxious relative to control)
Frontal alpha asymmetry					
A. Resting state					
Moscovitch et al., 2011	Patients with SAD Pre and post CBT	11:12	Resting state	-	Increased left frontal activity after CBT (F3/4; $\eta_p^2 = 0.15$)
Schmidt, 1999	High-shy/high-social High-shy/low-social Low-shy/high-social Low-shy/low-social (extreme groups)	All 10:0	Resting state	-	Increased right frontal activity (F3/4)
B. Anticipation of and recovery from socially stressful situation					
Schmidt & Fox, 1994	High-shy/high-social High-shy/low-social Low-shy/high-social Low-shy/low-social (extreme groups)	All 10:0	Instruction Anticipation Social interaction	Low-social more socially anxious nonverbal behavior	No difference
C. Combined studies (both resting state and anticipation/recovery of/from socially stressful situation					
Davidson et al., 2000	Patients with SAD vs controls	14 patients with SAD (ratio unclear)	Resting state Instruction Anticipation Planning Speech Recovery	More anxiety in each condition, increase in anxiety during anticipation	RS: No difference ANT: Increase in right anterior temporal activity from resting state to anticipation and from resting state to planning (T3/4), same for lateral frontal activity (F7/8)* REC: No difference

Beaton et al., 2008	HSA vs LSA participants (extreme groups)	19:5	Resting state Instruction Anticipation Speech	-	RS: No difference ANT: No difference. After controlling for depression, only shyness was related to increased right frontal activity
Harrewijn et al., 2016	HSA vs LSA participants (extreme groups)	23:0	Resting state Social judgment task Instruction Watch video Anticipation Speech Recovery	More nervous at each time point (except baseline); more avoidance after video	RS: No difference (r = -0.12) ANT: No difference (r = -0.06) REC: No difference (r = -0.03)
Cole et al., 2012	High vs low socially withdrawn participants (median split)	12:9	Resting state Instruction Watch video Anticipation Speech	No influence on performance rating	RS: No difference ANT: After watching anxious video, increased right frontal activity during watching video and anticipation (F3/4; d = 0.81)
Delta-beta correlation					
A. Resting state					
Van Peer et al., 2008	High vs low behaviorally inhibited participants (extreme groups)	0:20	Resting state Cortisol vs placebo administration	-	Increased positive delta-beta correlation Delta-beta correlation increased after cortisol administration in both groups (Fz)
B. Combined studies (both resting state and anticipation/recovery of/from socially stressful situation					
Miskovic et al., 2011	Patients with SAD Pretreatment 1 Pretreatment 2 Midtreatment Posttreatment	12:13	Resting state Anticipation Speech	More anxiety during anticipation than resting state	RS: Decreased positive delta-beta correlation from pretreatment to midtreatment and from pretreatment to posttreatment (F3, F4, C4, P4, O2) ANT: Decreased positive delta-beta correlation from pretreatment to posttreatment (F3, F4, C3, C4, P3, P4, O1)

Harrewijn et al., 2016	HSA vs LSA participants (extreme groups)	23:0	Resting state Social judgment task Instruction Watch video Anticipation Speech Recovery	More nervous at each time point (except baseline); more avoidance after video	RS: No difference ANT: Increased negative delta-beta correlation (F3/F4/Fz) REC: Increased negative delta-beta correlation (F3/F4/Fz)
Miskovic et al., 2010	HSA vs LSA participants (extreme groups)	24 HSA (ratio unclear)	Resting state Instruction Anticipation Speech	More nervous, less confident, calm and prepared	RS: No difference ANT: Increased positive delta-beta correlation (F4)

* p-level between 0.05 and 0.1
Effect sizes are displayed when reported.
Note: SAD = social anxiety disorder; CBT = cognitive-behavioral therapy; RS = resting state; ANT = anticipation; REC = recovery; HSA = high socially anxious; LSA = low socially anxious.

Table 2

Overview of studies about early and late ERPs in face processing paradigms in social anxiety.

Author	**Participants**	**Sex ratio of target group (F:M)**	**Task**	**Stimuli**	**Behavioral results** (socially anxious relative to control)	**Early ERPs**			**Late ERPs**	
						P1	**N170**	**P2**	**P3**	**LPP**
A. Explicit tasks (and studies with both explicit and implicit instructions marked with +) - attention to emotion necessary to complete the task										
Kolassa & Miltner, 2006 +	Patients with SAD vs patients with spider phobia vs controls	9:10	Modified Stroop task (identify gender or expression)	Angry, happy, and neutral faces	No difference	P1 no diff Relation with FSS in patients with SAD	N170 ↑ angry faces, right hemisphere, during emotion identification	P2 no diff Longer latency*		
Kolassa et al., 2007 +	Patients with SAD vs patients with spider phobia vs controls	9:10	Modified Stroop task (identify colour or expression)	Schematic stimuli of angry, happy, and neutral faces	No difference	P1 ↑ overall	N170 no diff		P3 no diff	LPP no diff

Kolassa et al., 2009	Patients with SAD vs patients with spider phobia vs controls	7:8	Emotion identification task	Schematic faces that morphed into more and more intensely angry, happy or sad faces	Angry faces more arousing (also in spider phobia patients), no difference in valence ratings, emotional classifications, reaction times	P1 ↑ overall (also in spider phobia patients)	N170 no diff	P2 no diff	
Moser et al., 2008	HSA vs LSA participants (extreme groups)	15:6	Modified Eriksen Flanker task	Threatening (anger, disgust), and reassuring (happy, surprise) faces	No difference			P2 no diff threatening - reassuring faces* $\eta_p^2 = 0.08$	P3/LPP ↑ threatening target faces $\eta_p^2 = 0.12$
Peschard et al., 2013 +	HSA vs LSA participants (extreme groups)	9:9	Modified Stroop task (identify colour or expression) Control color-naming task	Angry, happy, and neutral faces (upright and inverted) Red, blue, and green-coloured rectangles	No difference in Stroop task, faster in control colour-naming task	P1 ↑ all tasks $\eta_p^2 = 0.11$	N170 no diff	P2 no diff	

Rossignol et al., 2012a	HSA vs LSA participants (extreme groups)	8:4	Emotional oddball task	Frequent stimuli: neutral faces Deviant stimuli: angry, disgusted, fearful, and happy faces	No difference	P1 ↑ $\eta_p^2 = 0.29$ all faces, emotional > neutral $\eta_p^2 = 0.12$	N170 no diff	P3b no diff
Rossignol et al., 2007	High vs low FNE (cut off)	10:0	Identifiy deviant stimuli	Morphed faces: mix of angry and disgusted faces Frequent stimuli: 35% angry or disgusted Deviant stimuli: 5% and 65% angry or disgusted)	Disgust detected before anger, independent of conditions (not in controls)			- N2a/P3a: earlier latencies for disgust faces. - P3b: no diff
Sewell et al., 2008	Healthy participants	12:9	Emotional oddball paradigm	Frequent stimuli: neutral faces Deviant stimuli: angry and happy faces	Not reported			Corr P3 with SA: upright > inverted faces

Van Peer et al., 2007	High vs low BI (extreme groups)	0:20	Approach-avoidance task After cortisol and placebo administration	Angry and happy faces	More state anxiety and tension, no differences in task performance				P3 ↑ angry > happy, only in avoidant trials after cortisol administration $\eta_p^2 = 0.20$	
B. Implicit tasks - attention to emotion not necessary to complete the task										
Hagemann et al., 2016	Patients with SAD vs controls	16:5	Face learning task	Angry, happy, and neutral faces	No difference Lower accuracy *	*Learning* P1 ↑ for neutral and angry faces (no effect emotion in controls) $\eta_p^2 = 0.09$ *Test* P1 no diff	*Learning* N170 no diff *Test* N170 no diff	*Learning* P2 no diff *Test* P2 no diff		*Learning* LPP ↑ for angry > neutral & happy (not in controls, not in P4) $\eta_p^2 = 0.13$ *Test* LPP ↑ for learned > novel faces (not in controls) - only for C4 electrode $\eta_p^2 = 0.07$

Mueller et al., 2009	Patients with SAD vs controls	8:4	Modified dot-probe task	Angry-neutral, or happy-neutral face pairs	No difference (but when tested separetely SAD showed hypervigilance to anger vs happiness, and controls not)	P1 ↑ angry-neutral pairs $\eta_p^2 = 0.18$ ↓ probes emotionally < neutrally cued trials $\eta_p^2 = 0.20$	N170 ↓ $\eta_p^2 = 0.20$		
Helfinstein et al., 2008	HSA vs LSA participants (extreme groups)	12:0	Dot-probe task with neutral and social threat words as primes	Angry-neutral face pairs	Less accurate on incongruent trials than congruent trials (not in LSA)*	P1 ↑ overall		P2 ↑ overall*	
Judah et al., 2016b	HSA vs LSA participants (extreme groups)	11:9	Change detection task Cue to elicit self-focus	Disgusted and neutral faces	No difference			P2 no diff Neg corr P2 and task performance in self-focus trials (not in LSA)	
Rossignol et al., 2012b	High vs low FNE (extreme groups)	11:3	Spatial cueing task	Angry, disgusted, happy, and fearful faces	No difference	P1 ↑ overall Longer latency for probe arrow*		P2 ↑ overall	P3 no diff
Rossignol et al., 2013b	HSA vs LSA participants (extreme groups)	8:8	Attention-shifting paradigm with faces/bodily postures as cue	Angry, happy, and neutral faces and bodily postures of	Overall slower response	P1 ↓ overall $\eta_p^2 = 0.13$	N170 no diff	P2 ↓ overall $\eta_p^2 = 0.13$	P3 no diff $\eta_p^2 = 0.00$

				artificial humans					
Schmitz et al., 2012	HSA vs LSA participants (median split)	13:13	View eye gaze Report location of white dot	Neutral photos of direct or 30° left/right averted gaze	No difference	P1 ↑ averted > direct gazes* $\eta_p^2 = 0.09$	N170 no diff		LPP ↑ averted > direct gazes $\eta_p^2 = 0.13$
Rossignol et al., 2013a	High vs low FNE (extreme groups)	11:2	Spatial cueing task	Neutral-angry, neutral-happy, neutral-disgust, and neutral-fear face pairs	No difference	P1 ↑ overall $\eta_p^2 = 0.17$ ↑ in targets replacing emotional face > neutral face $\eta_p^2 = 0.16$	N170 no diff	P2 ↑ neutral-anger > neutral-fear $\eta_p^2 = 0.10$	

* p-level between 0.05 and 0.1

Effect sizes are displayed when reported.

Note: SAD = social anxiety disorder; diff = difference; FSS = Fear survey schedule; SPAI = social phobia and anxiety inventory; HSA = high socially anxious; LSA = low socially anxious; FNE = fear of negative evaluation; SA = social anxiety; BI = behavioral inhibition.

Table 3

Overview of studies about ERPs in cognitive conflict paradigms in social anxiety.

Author	**Participants**	**Sex ratio of target group (F:M)**	**Task**	**Stimuli**	**Behavioral results** (socially anxious relative to control)	**N2**	**FRN**	**ERN**	**CRN**	**Pe**
Cao et al., 2015	Patients with SAD vs controls	13:7	Island Getaway task	Neutral faces with feedback indicating social acceptance or rejection	Lower peer-acceptance expectancy in real life and in the task		FRN ↑ positive vs negative feedback $\eta_p^2 = 0.13$ Δ FRN ↑ (rejection - acceptance)			
Endrass et al., 2014	Patients with SAD vs patients with OCD vs controls	17:7	Flanker task	Arrows	No difference			ERN ↑ $\eta_p^2 = 0.16$	CRN ↑ $\eta_p^2 = 0.16$	Pe no diff $\eta_p^2 = 0.05$
Kujawa et al., 2016	Patients with SAD vs patients with GAD vs controls Pre and posttreatment	13:5	Flanker task	Arrow heads	No increase in reaction time between pre and posttreatment (as in controls)			Δ ERN (error - correct response) ↑ $\eta_p^2 = 0.12$		
Sachs et al., 2004	Patients with SAD vs controls	12:13	Standard two-tone oddball paradigm	Tones	No difference	N2 ↓				

Barker et al., 2015	HSA vs LSA participants (extreme groups)	13:12	Flanker task, alone and peer observation condition	Arrow heads	No difference			ERN ↑ peer condition vs alone, not in LSA $\eta_p^2 = 0.11$	CRN no diff	Pe no diff
Judah et al., 2016a	HSA vs LSA participants (extreme groups)	26 HSA (ratio unclear)	Trial-and-error learning task	Contour line drawings as stimuli Faces (neutral, happy, disgusted) provided performance feedback	No difference		FRN ↑ in self-focus trials before learning*	ERN ↑ $\eta_p^2 = 0.08$	CRN ↑ $\eta_p^2 = 0.08$	Pe ↑* Greater increase after learning $\eta_p^2 = 0.14$
Judah et al., 2016b	HSA vs LSA participants (extreme groups)	11:9	Change detection task Cue to elicit self-focus	Disgusted and neutral faces	No difference	N2pc for disgust faces in standard trials (LSA in self-focus trials) $\eta_p^2 = 0.16$				
Moser et al., 2008	HSA vs LSA participants (extreme groups)	15:6	Modified Eriksen Flanker task	Threatening (anger, disgust), and reassuring (happy, surprise) faces	No difference	N2 no diff			CRN no difference beteen threatening and reassuring $\eta_p^2 = 0.11$	

Rossignol et al., 2007	High vs low FNE (median split)	10:0	Identifiy deviant stimuli	Morphed faces: mix of angry and disgusted faces Frequent stimuli: 35% angry or disgusted Deviant stimuli: 5% and 65% angry or disgusted)	Disgust detected before anger, independent of conditions (not in controls)	N2b ↓ while detecting change in intensity of anger	
Van Peer et al., 2007	High vs low BI (extreme groups)	0:20	Approach-avoidance task After cortisol and placebo administration	Angry and happy faces	More state anxiety and tension, no differences in task performance	N2 no diff	
Van der Molen et al., 2014	Healthy participants	31:0	Social judgment paradigm	Acceptance or rejection feedback	No correlation FNE and percentage of negative judgments. Positive correlation FNE and RT for predicting acceptance and rejection		FRN no effect

* p-level between 0.05 and 0.1
Effect sizes are displayed when reported.
Note: FRN = feedback-related negativity; ERN = error-related negativity; CRN = correct-response negativity; Pe = positive error; SAD = social anxiety disorder; OCD = obsessive-compulsive disorder; diff = difference; GAD = generalized anxiety disorder; HSA = high socially anxious; LSA = low socially anxious; FNE = fear of negative evaluation; BI = behavioral inhibition; RT = reaction time.

Table 4

Overview of studies about frontal alpha asymmetry related to social anxiety in children.

Author	**Participants**	**Sex ratio of target group (F:M)**	**Protocol**	**Results** (socially anxious relative to control)
Fox et al., 2001	Continuously inhibited, and change children At 9, 14, 24 and 48 months (based on mean scores)	4:8	Resting state	Increased right frontal activity at 9, 14, and 48* months (F3/F4)
Schmidt et al., 1999	High, middle, and low shy groups 7 years (extreme/middle groups)	4:6	Resting state Instruction Anticipation (3 parts)	Behavior: More anxious behaviors in 2nd and 3rd part of anticipation RS: No difference ANT: No difference Increased right frontal activity (F4) from 2nd to 3rd part of anticipation
Theall-Honey & Schmidt, 2006	High vs low shy children 4.5 years (extreme groups)	10:10	Resting state Watch affective videoclips (sad, anger, happy, fear) Speech	Behavior: More behavioral signs of verbal anxiety during speech RS: No difference (F3/F4), increased right central activity (C3/4) Videoclips: Increased right central activity in fear videoclip (C3/4)
Hannesdottir et al., 2010	Healthy children at age 4.5 and 9	8:12	Age 4.5 (EEG) Resting state Cognitive control task Age 9 (HR) Resting state Instruction Anticipation Speech	Behavior: Correlations between child reported internalizing symptoms and anticipatory anxiety before speech (positive), and between anticipation anxiety and HR (positive) and HRV (negative) RS: No effect on behavior at age 9

			Recovery	
Henderson et al., 2001	Healthy children at 9 and 48 months	51:46	Resting state	Negative reactivity predicted social wariness at age four in infant boys with right frontal activity at 9 months (F3/4)
Henderson et al., 2004	Healthy children at age 4	80-67	Resting state	Solitary-passive and reticent social play groups show increased right frontal activity (F3/4)
Fox et al., 1995	Healthy children at age 4	28:20	Resting state	No effect on inhibition/social retinence, only effect on social competence/sociability (F3/4)

* *p*-level between 0.05 and 0.1.
Effect sizes are displayed when reported.
Note: RS = resting state; ANT = anticipation; HR = heart rate, HRV = heart rate variability.

Table 5

Overview of studies about ERPs related to social anxiety in children.

Author	**Participants**	**Sex ratio of target group (F:M)**	**Task**	**Stimuli**	**Results** (socially anxious relative to control)
A. Explicit tasks - attention to emotion necessary to complete the task					
Kujawa et al., 2015	Children with anxiety disorders and healthy controls 7-19 years	All 53 (ratio unclear)	Emotional face-matching task with shape-matching trials	Angry, fearful, and happy faces	- Behavior: No difference - LPP ↑ for angry and fearful faces ($\eta_p^2 = 0.12$ and $\eta_p^2 = 0.09$)
B. Implicit tasks - attention to emotion not necessary to complete the task					
Thai et al., 2016	Community sample, children with BI were oversampled 9-12 years	50:49	Dot-probe task	Angry-neutral and neutral-neutral face pairs	- Behavior: No difference - P1, N170, N2 no effect of social anxiety - P1 to probes replacing angry faces no effect (↑ in BN) ($\eta_p^2 = 0.05$) - ↑ N2 predicted bias away from angry faces in BI
C. Cognitive conflict paradigms					
Bar-Haim et al., 2003	High vs low socially withdrawn children 7-12 years (extreme groups)	11:12	Passive listening	Tones	- No behavior - P1-N1 no difference
Lackner et al., 2014	High vs low shy adolescents 12-14 years (extreme groups)	22 (ratio unclear)	Money game	Feedback indicating win or loss	- Behavior: No difference - FRN ↓

Lahat et al., 2014a	High vs low BI children 7 years (median split)	28:26	Flanker task	Fish	- Behavior: No difference - ERN ↑ ($\eta_p^2 = 0.12$) - CRN no difference - BI group was positively related to SAD symptoms, in children with relatively large ERN-CRN
Lahat et al., 2014b	High vs low BI children 7 years (median split)	40 or 41 (ratio unclear)	Flanker task	Fish	- Behavior: No difference - N2 ↑ ($\eta_p^2 = 0.09$) - Greater withdrawal and lower assertiveness in high BI children with ↑ N2
McDermott et al., 2009	High vs low BI children 15 years (median split)	41 (ratio unclear)	Flanker task	Letters	- Behavior: No difference - ERN ↑ - Pe no difference - ↑ ERN related to higher risk for anxiety disorders in high BI children
Reeb-Sutherland et al., 2009	Adolescents who were high or low BI as children (latent class analysis) 13-16 years	23:20	3-stimulus auditory oddball	Tones and noises as novel stimuli	- No behavior - Novelty P3 no difference - Higher novelty P3 amplitudes = more likely to have anxiety diagnoses
Henderson, 2010	Healthy children 9-13 years	36 (ratio unclear)	Modified version of Eriksen Flanker task	Arrow heads	- Behavior: No difference - N2 no effect - Shyness predicted social anxiety in children with relatively large N2
Kessel et al., 2015	Community sample 8-10 years	175:215	Monetary reward task	Green arrow indicated win, red arrow indicated loss	- No behavior - ↑Δ FRN associated with social anxiety
Kujawa et al., 2014	Community sample 10-15 years	8:11	Island Getaway task	Feedback indicating social acceptance or rejection	- Behavior: Less rejection of co-players - ↓ (more negative) Δ FRN associated with social anxiety

Lamm et al., 2014	Healthy children 7 years	58:48	Go/No-Go task	Neutral animal pictures	- Behavior: Postive relation between BI and accuracy and reaction time - Negative association between BI and N2 amplitude - Early BI was positively associated with social reticence at age 7, if N2 was increased
Tang et al., 2016	Healthy children 10 years	26:27	3-stimulus auditory oddball	Target, novel, standard tones	- Behavior: No difference - N2 no effect - P3 ↑ for target and standard tones, longer latency

Effect sizes are displayed when reported.
Note: BI = behavioral inhibition; BN = children without behavioral inhibition; CRN = correct response negativity.