Event-Related Brain Potentials Reveal Differences in Emotional Processing in Alexithymia

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Abstract
The inability to recognize and describe emotions in the self is known as Alexithymia. Such inability, occurring more frequently in men, has been attributed to late suppression of emotion. In the present study we used event-related potentials (ERPs) to examine the locus of processing emotional differences in alexithymia. We tested men, both those scoring high (score > 61) and controls who scored low (score < 51) on the Toronto Alexithymia Scale-20 on an emotional face discrimination task (angry, happy, vs. neutral faces). We assessed three ERP components: P1 (100-200 ms after stimulus onset; an index of early perceptual processing), N170 (140-190 ms after stimulus onset; an index of early facial processing) and P3 (300-600 ms after stimulus onset; an index of late attentional suppression). While controls showed a stronger P3 effect for angry faces relative to happy and neutral faces, Alexithymic men showed no significant differences in P3 across emotions. Alexithymic men showed similar P1 and N170 amplitudes as controls but these components were delayed related to controls. These results suggest that the locus of processing differences between alexithymic men and controls occur both early in perceptual processing and later in conscious processing.

Key Words: Emotional Valence, Alexithymia, ERPs, Repression, Suppression
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Emotion enhances our social interactions with others. However, many individuals have difficulties regulating their emotions or interpreting the emotions of others. One example is alexithymia (literally “no words for emotions,” Oxford English Dictionary), which is a personality syndrome that involves both cognitive and affective deficits in the processing of emotions (Taylor, Bagby & Parker, 1999; see Donges & Suslow, 2016, for a review). One interesting question is where the deficit of emotion processing occurs for alexithymia. Damasio and Carvalho (2013) noted the distinction in emotion perception between early aspects of sensing and perceiving an emotional stimulus, termed exteroception (with emphasis on early limbic system arousal), versus a later internal focus on one’s own internal affective response to an emotional stimulus, termed interoception (later emotional regulation) (see also Pais-Veiera, Wing, & Cabeza, 2016). While the sensory transduction of a threatening stimulus may initially result in a reflexive response (emotional arousal), the later conscious interpretation (emotional regulation) is what we refer to as “feeling” (Damasio & Carvalho, 2013). It is important to separate these two critical stages of emotion perception because one or both may be crucial for a thorough understanding of how emotional stimuli affect an individual. Alexithymia may affect early arousal, later conscious interpretation of emotion, or both. Identifying the locus of emotional processing differences in those who are high in alexithymia can provide for a better understanding of alexithymia and may play a helpful role in its treatment.

In the present study, we used event-related potentials (ERPs) to assess early emotional arousal and later emotional processing (Eimer & Holmes, 2007; Rotshtein et al., 2010) in alexithymic people. ERPs measure the changes in voltage in different areas of the brain in relation to sensory, cognitive or motor events (Luck, 2014). Compared to traditional behavioral data (e.g., response time), ERPs provide a continuous measure of emotion processing starting...
from the moment a stimulus is presented. By examining the time course of ERP components associated with processing emotion, it is possible to gain insight into the temporal locus of deficit for alexithymia.

**Alexithymia and Emotion Processing**

Alexithymia was first studied in the context of psychosomatic disorders and was later extended to individuals diagnosed with posttraumatic stress, eating, and substance use disorders (Taylor, 2004). The deficits observed for alexithymic participants included problems with perceiving and describing feelings, problems in distinguishing between bodily sensations and feelings, a lack of fantasies, and thinking that was externally oriented (Nemiah, Freyberger, & Sifneos, 1976). All these deficits (except for a lack of fantasies) are assessed with the Toronto Alexithymia Scale-20 (TAS-20; Bagby, Parker, & Taylor, 1994; Bagby, Taylor, & Parker, 1994; Taylor et al., 1988)—the most commonly used test of alexithymia (Taylor, 2000). The TAS-20 has demonstrated validity and reliability when used as a single score (Taylor et al., 1988).

Levant (1995, 2011) theorized that socialization agents (parents, teachers, coaches and peers) enforced the norm of restrictive emotionality for boys. This norm restricted the expression of vulnerable emotions like hurt, fear, or sadness, as well as attachment emotions (fondness, caring, loneliness) that reflect a need for another person, because in traditional masculinity ideology (TMI) the most honored way of being a man was to never show vulnerability and never need anyone.

To assess the extent of gender differences in alexithymia, Levant et al. (2006) narratively reviewed 44 studies which examined such gender differences. While few of the 12 studies using clinical samples found gender differences, of the 32 studies using non-clinical samples: 17 found males more alexithymic than females, 1 found females more alexithymic than males, and 14
found no differences between males and females. Levant, Hall, Williams, and Hasan (2009) then conducted a meta-analysis on gender differences in alexithymia literature. An effect size estimate based on 41 existing samples found consistent, although expectedly small, differences in mean alexithymia between women and men (Hedges’ $d = .22$). Men exhibited higher levels of alexithymia.

However, the question remains as to where in emotion processing, such as perception, identification, or expression of these taboo sets of emotions, this deficit occurs for alexithymic men. In a recent study by Levant, Allen, and Lien (2014, Study 2), participants were instructed to make a lexical decision response to the target word, and the issue of interest was whether prime type (positive or neutral) had an effect on target performance (i.e., a priming effect). The interval between the prime onset and the target onset (stimulus onset asynchrony; SOA) was 100, 300, or 500 ms, randomly determined for each trial. Levant et al. reasoned that if inhibition of the prime on the target occurred early as a consequence of automatic repression, a priming effect should be observed at the short SOA. If inhibition of the prime occurred late in processing due to intentional suppression, a priming effect should be observed at the long SOA.

Levant et al. (2014) found that the alexithymic men showed a stronger priming effect (61 ms) at the 500 ms SOA than the control men (8 ms) for emotional taboo words compared to emotional non-taboo words. Also, for just the taboo emotional adjectives (vulnerable or attachment words), the alexithymic men committed more errors (13%) than the controls (8%), although this effect was not significant for emotional non-taboo words (aggression or lust words). This is consistent with the predictions of Levant (2011) in which alexithymic men exhibit gender role strain. These results provided empirical evidence that alexithymic men inhibit vulnerable and attachment adjectives at approximately 500 ms after stimulus presentation. The
finding that significant inhibition did not occur at the 100- or 300-ms SOA suggested that alexithymic men could perceive and identify taboo emotional words, but that they inhibited those words before they could interpret word meaning. In other words, alexithymic men engaged in conscious cognitive suppression rather than preconscious repression.

The evidence for the late suppression view in Levant et al. (2014) is from the semantic priming paradigm. It has been suggested in the literature that other factors, such as word frequency, rather than the nature of word processing itself that may possibly affect processing speed and priming effects (e.g., Allen et al., 2005; Balota & Chumbley, 1984; Monsell, Doyle, & Haggard, 1989). In another emotional priming study, Vermeulen, Luminet, and Corneille (2006) found evidence for less affective priming for those with higher alexithymia scores. Suggesting that processing differences occur early in processing during “automatic” stages. In their study, an emotional prime (a drawing of an emotional face) was presented for 100 ms, followed by a 100 ms SOA, then by a target valenced word. This effect held stable in replication which controlled for additional covariates. Taken together, Levant et al. (2014) and Vermeulen, Luminet, and Corneille (2006) suggest that alexithymia may disrupt emotional processing in both early “automatic” perceptual stages and later “conscious” stages.

The purpose of the present study, therefore, was two-fold. First, instead of using a semantic priming paradigm (Levant et al., 2014), we used an emotional face discrimination task, in which participants determined the emotion on a face that appeared in the center of the screen. This non-linguistic emotional task allowed us to directly test emotional processing without introducing other potential confounds. Second, in addition to the behavioral data (response time and accuracy), we used online electrophysiological measures (i.e., event-related potentials [ERPs]). In particular, ERPs have the temporal precision to measure early perceptual arousal
(the P1 ERP component is measured 100-200 ms after stimulus presentation; Luck, 2014) and later stimulus categorization (the P3 ERP component is measured 300-600 ms after stimulus presentation; Lien et al., 2012). The key time after stimulus presentation for conscious awareness to occur appears to be approximately greater than 200 ms (Dehaene & Changeux, 2011; Luck, 2014). Therefore, ERPs recorded up to approximately 200 ms are assumed to involve preconscious (automatic) processes, and those recorded after 200 ms are assumed to involve conscious processes. Response time and accuracy measures conflate processing stages beyond the stage of interest (e.g., response execution effects that occur after stimulus categorization/identification) whereas ERPs do not (Luck, 2014).

**Emotion Processing and Event-Related Potentials**

Vermeulen, Luminey, De Sousa, and Campanella (2008) used ERP measures to study emotional perception in alexithymia. They found that early attentional perception of anger using the N2b (an index of an attentional switch needed to take new information into account; Suwazono, Machado, & Knight, 2000) and the P3a components (which like N2b is an index of attentional orientation; Halgren & Marinkovic, 1995) occurs significantly later in processing for those high in alexithymia. This delay in processing relative to controls indexes an attentional deficit.

One potential limitation of using N2b and P3a (measured as peak amplitude occurring later than 300 ms post-stimulus onset) as used in Vermeulen et al. is that the timeframe did not measure preconscious processes (Dehaene & Changeux, 2011). Several studies suggest that preconscious processing occur in the first 200 ms of stimulus onset (Fisch et al., 2009; Galliard et al., 2009; Melloni et al., 2007) and that there is a significant increase in conscious access after 200 ms (Fisch et al., 2009; Galliard et al., 2009). To avoid this limitation, the present study
measured the P1 (100-200 ms) and N170 (140-190 ms) components which are measured in completion before 200 ms. In addition to the ERP components P1 and N170 as an index of early preconscious processing, we also used the P3 component as an index of later “conscious” processing. Below we described each ERP component in details.

**P1.** The P1 ERP component is influenced by early visual sensory perception. Specifically, it is sensitive to stimulus parameters such as luminance, and is modulated by selective attention (see review by Hillyard, Vogel, & Luck, 1998) and the subject’s state of arousal (Vogel & Luck, 2000). A greater positive amplitude is indicative of a stronger P1 effect (e.g. strong arousal of the subject is linked to a greater positive P1 amplitude). While the source location of the component appears to be in the extrastriate cortex (Di Russo et al., 2002), the amygdala has been shown to modulate this component by presumably influencing the subject’s arousal. Rotshtein et al. (2010) found that epilepsy patients before surgery with no damage to the amygdala showed an emotionally valenced P1 effect (i.e., a stronger emotional response to fearful faces than to neutral faces). However, after surgery to prevent migration of seizures, these same individuals no longer showed a valenced P1 effect to such emotional faces. Likewise, Smith, Weinber, Moran, and Hajcak (2013), Pollock et al. (2012) and Houston et al. (2018) found emotionally valenced P1 effects using angry faces. Thus, the P1 facial emotional discrimination response is likely based on amygdalar modulation of the secondary visual cortex (Eimer & Homes, 2007; Rotshtein et al., 2010; Vuilleumier & Pourtois, 2007) that occurs early in emotional processing. In the present study, we used the P1 component to index early “preconscious” arousal of emotional faces.

**N170.** The N170 component is modulated by facial perception and is named for its negative peak that occurs at roughly 170 ms post-stimulus onset (Bentin, Allison, Puce Perez, &
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McCarthy, 1996). Compared to other images such as cars, faces elicit a negative deflection in polarity typically between 140-190 ms in occipito-temporal locations (see Rossion & Jaques, 2012 for a review). Although Eimer and Holmes (2002) and Smith et al., (2013) found little evidence for sensitivity of the N170 to facial expression, Blau (et al., 2007) found a stronger effect for negative, relative to neutral faces, meaning that the amplitude was lower for negative faces than neutral faces. Krombholdz, Schaefer, and Boucsein (2007) also found a larger negative deflection in the N170 for angry compared to happy faces.

**P3.** The P3 effect observed in the facial emotional discrimination task is likely an index of later conscious emotional regulation (Houston et al., 2018; Houston et al., in press). Pollatos and Gramann (2011) used IAPS stimuli in attempt to identify the locus of emotional processing deficits in Alexithymia. Similar to the present study, they used the P1 and P3 ERP components to measure early versus later processing differences, respectively. They found reduced ERP amplitudes for both components in the high alexithymia condition. Furthermore, the amplitude of the components covaried, providing support to the assumption that deficits in alexithymia occur early in processing and that these deficits contribute to later deficits. The present study aims to extend these findings to facial perception.

**Other Alexithymia Studies Using ERP Methods.** Franz et al. (2004) tested 20 men scoring higher on the TAS-20 (alexithymic men) and 20 men scoring lower on the TAS-20 (controls) using a visual “oddball” task (20% of the visual stimuli were considered to be arousing and unpleasant and 80% were considered to be pleasant—the stimuli were pictures from the International Affective Picture System, or IAPS, Lang et al., 1988). They found significantly higher P2 (a component occurring 150-260 ms after stimulus presentation that is an index of spatial attention) ERPs for alexithymic men than for controls. Franz et al. concluded that
alexithymia was likely not due to a lack of incoming perceptual information because the “high TAS-20” group showed evidence of perceptual arousal. Also, Campanella et al. (2012) tested 15 women and 15 men on an “emotional oddball” task similar to the Franz et al. (2004) study, except Campanella et al. (2012) examined sex and TAS-20 scores. Campella et al., used happy and fearful faces from the Ekman and Friesen (1976) pictures. Campanella et al. (2012) found that TAS-20 scores predicted the N2 ERP (which is modulated by spatial attention) component amplitude better than sex. In particular, individuals with higher TAS-20 scores (mean = 63) showed lower N2 amplitude than individuals scoring lower on the TAS-20 (mean = 42), but level of alexithymia did not influence P3.

With regard to ERP research on alexithymia, Franz et al. (2004) and Campanella et al. (2012) have observed group effects for P2 and N2 amplitude. Franz et al. (2004) found higher-amplitude P2 responses for alexithymic men than controls, whereas Campanella et al. (2012) found lower-amplitude N2 components for alexithymic individuals than controls. These discrepancies may be due to the stimuli chosen for the task. Emotionally valenced non-facial pictures featured in the IAPS database (such as animals, landscapes, or scenes with weapons), and emotional faces may lead distinct group differences afforded by separate mechanisms. However, there are two limitations to these earlier ERP studies. First, while alexithymic men show an attenuated response to vulnerable and attachment emotions, they do not show a blunted response to stimuli associated with aggression and lust (Levant, 1995; Levant et al., 2014). The IAPS pictures used by Franz et al. (2004) appeared to be primarily associated with aggression, although the Campanella et al. (2012) stimuli did index vulnerable emotions (i.e., they used fearful faces in one condition). Second, to ensure that visual stimuli are being processed preconsciously, it is important to measure responses before 200 ms after stimulus presentation.
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(Dehaene & Changeux, 2011). This is, ERP measurements will need to be taken within the range of 0-200ms post-stimulus onset to assure the stimulus has not reached conscious processing.

The Present Study

The present study aims to examine the locus of differences in emotional face perception between those high in alexithymia and controls. Previous research has provided evidence of these differences occurring later during “conscious processing” and the present study aims to replicate previous findings, by measuring “preconscious” electrophysiological reactivity in conjunction. We measured vulnerable emotion processing (fear—elicited by angry faces) and non-vulnerable emotional processing (happy and neutral faces) in both alexithymic men and control samples using Nim-Stim human faces (Tottenham et al., 2009). Also, we measured both preconscious perceptual processing (an index of repression) and conscious cognitive identification (an index of suppression) using P1 (100-200 ms after stimulus presentation), N170 (140-190 ms after stimulus presentation) to index facial processing and P3 (300-600 ms after stimulus presentation) ERP components, respectively.

Emotional facial discrimination is of particular interest with regard to alexithymia because “happy faces” and “neutral faces” are not assumed to be threatening, whereas “angry faces” are assumed to result in a fear response (e.g., Ohman, 2002). Based on Levant (1995, 2011) and Levant et al. (2014), we assume that the fear response to a threatening stimulus would constitute a vulnerable emotion that is a “taboo emotion” in alexithymia. Therefore, according to the late suppression view, alexithymic men will show evidence of attenuated P3 amplitude relative to controls for angry (negatively valenced) faces. This hypothesis is in line with Levant et al. (2014) which found group differences in priming at longer SOAs but not shorter (although
with the greater sensitivity of ERPs relative to behavioral measures). We hypothesize that control participants will have a higher P1 amplitude for angry faces than neutral or happy faces, as in Pollock et al. (2012) and the alexithymic men will show no emotional valence effect. Similarly, we also hypothesize that controls will show a lower N170 amplitude for angry faces than neutral or happy faces and that alexithymic men will show no emotional valence effect.

**Method**

**Participants**

Nineteen men (age range: 18-26 years, mean = 20.79, SD = 0.55) scoring > 62 on the TAS-20 (alexithymic men) and 19 controls (age range: age range: 18-27 years, mean = 21, SD = 0.59) scoring < 51 on the TAS-20 participated in the present study (see table 1 for TAS-20 means and subscores. Independent sample t-tests were conducted between group means for each subscores and the TAS-20). All participants were undergraduates recruited from undergraduate psychology courses at the University of Akron in exchange for course credit. Initially 927 individuals were screened on a web-based version of the TAS-20, and then a subset of these individuals, 38 out of 927 were tested in the laboratory-based EEG-based experiment. All participants reported normal corrected vision.

**Apparatus, Stimuli, and Procedure**

We adopted the methods from Houston et al. (2018, in press). For the emotion perception task, stimuli were presented centrally on a 23-inch Dell LCD monitor appending approximately 9.53° (height) by 6.75° (width). Each trial consisted of a single stimulus presentation consisting of a color image of a face presented against a black background. Thirty images, taken from the NimStim database (Tottenham et al., 2009), were used that comprised
three emotional expressions (happy, angry, and neutral) from 10 different actors (5 men, 5 women). The actors in these images were a mixture of African-, Asian-, European-, and Latino-American descent. The emotional expressions in the NimStim database were standardized so that angry and happy faces were extreme versions of these emotional expressions. Each face was presented 40 times, including 36 practice trials.

Each trial started with the presentation of a white fixation cross on a black background that persisted for 800 ms. After a 100, 300, or 900 ms delay, randomized within blocks, the stimuli appeared and remained until a response was collected (see figure 1). The delay was included to make the task feel less repetitive and predictable. Participants were asked to determine the emotional expression of the target face by pressing the keys “V”, “B”, and “N” for angry, happy, and neutral emotions, respectively, as quickly and accurately as possible.

Participants performed one practice block of 36 trials, followed by 16 experimental blocks of 72 trials each for a total of 1,152 experimental trials. Mean reaction time and accuracy feedback was provided after each block.

**EEG Recording and Analyses**

Electrophysiological data were recorded using a 32 channel Neuroscan EEG amplifier (Synamp1) using Quik-caps with silver chloride (AgCl) electrodes referenced to the average of the right and left mastoid. We recorded a horizontal electrooculogram (HEOG) from the outer corner of both eyes and a vertical electrooculogram (VEOG) above and below the left eye to control for ocular artifacts. Impedance did not exceed 5kΩ. EEG, HEOG, and VEOG values were amplified using a Synamps1 amplifier (Compumedics, Victoria, Australia) and the signals were digitized at 500 Hz, using the 10-20 system of electrode locations.

Waveforms were analyzed using the ERPLab 7.0 and EEGLab v.14.1.1 toolboxes in
Matlab 2014a (Delorme & Makeig, 2004; Lopez-Calderon & Luck, 2014). Prior to artifact detection, a half-amplitude high pass filter of 0.1 Hz with a 12 decibel/octave roll-off was applied to the data. Independent components were estimated from the continuous data using the runica algorithm in EEGLab. With the assistance of the MARA toolbox plugin (Winkler, Haufe, & Tangermann, 2011), highly probable artifact components were identified and rejected in the continuous data files using spatial and spectral waveform characteristics. After component rejection, 1500 ms epochs were set and time-locked to 200 ms pre-stimulus onset. In EEGLab a 200 ms pre-stimulus onset was used as a baseline for artifact rejections. This rejection process resulted in the loss of 15.37% of experimental trials, which did not differ across groups ($M_{\text{controls}} = 14.59\%$, $M_{\text{alexithymic men}} = 16.15\%$).

The P1, N170, and P3 ERP components were computed utilizing the ERPLab toolbox. For the analysis, the P1 waveform was operationalized as the positive amplitude in microvolts ($\mu$V) at O1, and O2 in the window of 100-200 ms after stimulus onset. The N170 ERP measurement used the electrodes T7, T8, O1 and O2 in the window of 140 to 190 ms after the stimulus onset. The P3 ERP measurement was established by taking the amplitude ($\mu$V) at P3, P4 and Pz in the window of 300 to 600 ms after the stimulus onset.

**Results**

**Behavioral Analyses**

A 2 (Group: alexithymic men vs. control) x 3 (Valence: angry, happy, or neutral faces) analysis of variance (ANOVA) was carried out on response time (RT) and percentage of accuracy data separately. Table 2 shows the mean RT and percentage of accuracy for each of these conditions. Whenever appropriate, $p$ values were adjusted using the Greenhouse-Geisser epsilon correction for non-sphericity. Latencies less than 100 ms were excluded from the
analysis, and incorrect responses were excluded from both the accuracy and ERP data.

Even though the alexithymic men showed a trend toward having longer latencies (mean RT = 948 ms) than the controls (mean RT = 871 ms), the main effect of group did not reach significance, $F(1, 36) = 2.71, p = .11, \eta^2_p = .07$. There was a main effect of valence, $F(2, 72) = 10.95, p < .001, \eta^2_p = .23$ (mean RT: angry = 939 ms, happy = 884 ms, neutral = 907 ms); pairwise comparisons revealed that both angry and neutral faces took longer to respond to than happy faces, $F(1, 36) = 6.64, p = .014, \eta^2_p = .16$, and $F(1, 36) = 4.36, p = .04, \eta^2_p = .09$, respectively. There was no Group x Valence interaction, $F(2, 72) = .27, p = .76, \eta^2_p = .01$.

For accuracy, there was also a trend toward poorer performance for alexithymic men (94% accuracy) than for controls (96%), $F(1, 36) = 3.09, p = .09, \eta^2_p = .08$. There was a main effect of valence, $F(2, 72) = 11.21, p < .001, \eta^2_p = .24$ (accuracy: angry = 93%, happy = 95%, neutral = 97%). Pairwise comparisons revealed that both angry and happy faces showed lower accuracy than neutral faces, $F(1, 36) = 15.47, p < .001, \eta^2_p = .30$, and $F(1, 36) = 13.89, p < .001, \eta^2_p = .28$, respectively. The Group x Valence interaction was not significant ($F < 1.0$), due to similar trends of higher accuracy for neutral faces than happy and angry faces for both groups (alexithymic men accuracy: angry = 92%, happy = 95%, neutral = 97%; controls accuracy: angry = 95%, happy = 96%, neutral = 98%).

**ERP Analyses**

The P1, N170 and P3 data were analyzed using a three-factor ANOVA with emotional valence (angry, happy, or neutral) and electrode location as within-group variables and group
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(alexithymic men vs. control) as a between-group variable. P1 waveforms were maximal over occipital channels s (O1 and O2; see Figure 2 for the pooled P1 as a function of emotional valences for the alexithymia and control groups, respectively). The N170 was maximal over occipital and temporal channels (T7, T8, O1 and O2; see Figure 3 for the pooled N170 as a function of emotional valences for the alexithymia and control groups, respectively). P3 waveforms were maximal over parietal channels (P3, Pz, P4; see Figures 4a and 4b for the pooled P3 as a function of emotional valences for the alexithymia and control groups, respectively). Table 3 shows the mean amplitude of each ERP components.

**P1.** The 2 (group: alexithymic men vs. controls) x 3 (emotional valence: angry, happy, neutral) x 2 (electrode location: O1, O2) ANOVA yielded a reliable main effect of emotional valence, $F(2, 72) = 26.03$, $p < 0.001$, $\eta^2_p = 0.31$ (mean: angry = 2.39 µV, happy = 2.53 µV, neutral = 2.95 µV). Both angry and happy faces resulted in a significantly lower P1 amplitude than did neutral faces, $F(1, 36) = 30.71$, $p < 0.001$, $\eta^2_p = 0.46$, and $F(1, 36) = 40.59$, $p < 0.001$, $\eta^2_p = 0.54$, respectively, but angry and happy amplitudes approached significance, $F(1, 36) = 3.39$, $p = .07$, $\eta^2_p = 0.08$, with a trend for higher amplitude for happy faces than angry faces. Most importantly, the main effect of group and its interactions with other variables did not reach statistical significance (all $p$’s > .10).

**N170.** The 2 (group) x 3 (emotional valence: angry, happy, neutral) x 3 (electrode location: O1, O2, T7, T8) ANOVA yielded a reliable main effect for emotional valence, $F(2, 72) = 17.12$, $p < 0.001$, $\eta^2_p = 0.66$ (angry = 1.67 µV, happy = 1.50 µV, neutral = 2.10 µV ) and electrode $F(3, 108) = 28.87$, $p < 0.001$, $\eta^2_p = 0.42$ (O1 = 3.25 µV, O2 = 3.16 µV, T7 = 0.375 µV, T8 = 0.25 µV ). The N170 for the angry and happy faces were significantly more negative (indicating a stronger effect) than neutral faces, $F(1, 36) = 12.40$, $p = 0.001$, $\eta^2_p = 0.25$, and $F(1,
36) = 12.40, \( p < 0.001 \), \( \eta^2_p = 0.49 \), respectively, but angry and happy faces did not significantly differ, although it approached significance, \( F(1, 36) = 3.70, p = 0.06 \), \( \eta^2_p = 0.09 \). The occipital electrode O1 and O2 did not significantly differ from each other \( (p = .72) \), but pairwise comparisons reveal that they each significantly differed from both temporal electrode sites T7 (O1: \( F(1, 36) = 33.18, p < 0.001 \), \( \eta^2_p = 0.47 \); O2: \( F(1, 36) = 30.41, p < 0.001 \), \( \eta^2_p = 0.45 \) ) and T8 (O1: \( F(1, 36) = 27.64, p < 0.001 \), \( \eta^2_p = 0.30 \); O2: \( F(1, 36) = 26.01, p < 0.001 \), \( \eta^2_p = 0.41 \) ). The temporal electrodes did not significantly differ from each other \( (p = .27) \). No other effects were significant.

**P3.** The 2 (group) x 3 (emotional valence: angry, happy, neutral) x 3 (electrode location: P3, Pz, P4) ANOVA yielded a reliable main effect for emotional valence, \( F(2, 72) = 7.55, p < 0.01 \), \( \eta^2_p = 0.17 \) (angry = 6.24 \( \mu \)V, happy = 5.72 \( \mu \)V, neutral = 5.85 \( \mu \)V). Both neutral and happy faces resulted in a significantly lower P3 amplitude than did angry faces, \( F(1, 36) = 14.55, p < 0.01 \), \( \eta^2_p = 0.28 \), and \( F(1, 36) = 6.30, p = 0.0166 \), \( \eta^2_p = 0.15 \), respectively, but happy and neutral amplitudes did not differ, \( F(1, 36) = 0.76, p = 0.38 \), \( \eta^2_p = 0.02 \). There was also a main effect for electrode location, \( F(2, 72) = 9.90, p < .001 \), \( \eta^2_p = 0.22 \) (P3 = 5.27 \( \mu \)V, Pz = 6.275 \( \mu \)V, P4 = 6.272 \( \mu \)V). The larger ERP amplitude in the P4 electrode site (located in the right hemisphere, see Figures 4a and 4b) compared to the P3 electrode site (located in the left hemisphere) may have been due to hemispheric specialization for emotion perception in the right hemisphere (see General Discussion for details).

Although the main effect for group did not reach statistical significance, \( F(1, 36) = 1.75, p = .19 \), \( \eta^2_p = 0.05 \) (alexithymic men = 5.23 \( \mu \)V, controls = 6.56 \( \mu \)V), it interacted significantly with emotional valence, \( F(2, 72) = 5.75, p < .01 \), \( \eta^2_p = 0.14 \) (controls: angry = 7.11 \( \mu \)V, happy = 6.29 \( \mu \)V, neutral = 6.27 \( \mu \)V; alexithymic men: angry = 5.37 \( \mu \)V, happy = 5.16 \( \mu \)V, neutral = 5.44
To interpret this interaction, we examined simple main effects of emotional valence separately by group. For controls, pairwise comparisons reveal a simple main effect of emotional valence, $F(1, 18) = 10.31, p < .001, \eta^2_p = 0.36$ (angry differed from happy ($F(1, 18) = 15.97, p < .001, \eta^2_p = 0.47$) and neutral ($F(1, 18) = 18.11, p < .001, \eta^2_p = 0.49$); but happy did not differ from neutral ($F(1, 18) = 0.01, p = .92, \eta^2_p = 0.001$). To the contrary, there was no simple main effect of emotional valence for alexithymic men, $F(1, 18) = 1.42, p = 0.26, \eta^2_p = 0.07$.

The 3-way interaction between Group, Emotion Valence, and Electrode was also significant, $F(4, 144) = 12.23, p < .001, \eta^2_p = 0.27$. The effect was driven by a valence effect for the control group ($F(2, 36) = 10.31, p < .001, \eta^2_p = 0.36$), which was not significant for the high alexithymia group ($F(2, 36) = 1.42, p = .258, \eta^2_p = 0.02$). The valence effect interacted with site for the control group ($F(4, 72) = 24.71, p < .001, \eta^2_p = 0.31$) but only approached significance for the high alexithymia group ($F(4, 72) = 2.64, p = .062, \eta^2_p = 0.03$). This interaction was driven by a strong valence effect for the control group in the left hemisphere at the P3 electrode location ($F(2, 36) = 25.31, p < .001, \eta^2_p = 0.42$, controls: angry = 6.63 µV, happy = 5.52 µV, neutral = 4.65 µV) that was not present for the alexithymic men group ($F(2, 36) = 2.34, p = .011, \eta^2_p = 0.09$; alexithymic men: angry = 5.10 µV, happy = 4.76 µV, neutral = 5.54 µV). Contrary to expectations there were no significant differences found in P3 amplitude between the groups for each emotion (angry: $F(1, 36) = 2.05, p = .161, \eta^2_p = 0.05$, happy = $F(1, 36) = 0.67, p = .420, \eta^2_p = 0.02$, neutral = $F(1, 36) = 9.87, p = .736, \eta^2_p = 0.003$). At the P3 electrode location, both angry and happy faces elicited a stronger effect than neutral faces for controls, $F(1, 18) = 38.37, p < .001, \eta^2_p = 0.67$, and $F(1, 18) = 9.87, p = .006, \eta^2_p = 0.35$, respectively. Angry faces also elicited a stronger effect than happy faces for controls ($F(1, 18) = 22.90, p < .001, \eta^2_p = 0.55$).
Peak Latency and Source. Peak latency for the P1, N170 and P3 components were analyzed using a 2 (group) X 3 (emotional valence: angry, happy, neutral) ANOVA. The P1 latency analysis found significantly different peak latency between groups, $F(2, 36) = 10.23, p < 0.01$, $\eta^2_p = 0.24$ (a shorter P1 latency onset for controls than for alexithymic men: Control = 142.33 ms, Alexithymic men = 165.5 ms). The N170 revealed shorter latencies for controls (174.4 ms) than Alexithimics (194.5 ms), $F(2, 36) = 7.75, p < 0.01$, $\eta^2_p = 0.18$ (see Figure 5). The P3 analysis yielded no significant main effect of peak latency for group, $F(2, 36) = 1.72, p = 0.2$, $\eta^2_p = 0.05$. Note that peak differences for the P3 are less common because the component in general has a far less distinct peak than earlier ERP components (Kiesel, Miller, Jolicœur & Brisson, 2008; Luck, 2005). No other effects involved emotional valence were significant.

The source analyses on P1, N170, and P3 ERP components revealed similar activity across groups (Figures 6a-6c, respectively). The P1 component has the majority of its activation in the parietal and occipital areas, the N170 has the majority of its activation in the parietal, occipital and temporal areas and the P3 seems to be largely concentrated in the parietal area with some central activation.

Correlations between ERP Components, TAS-20 and Behavioral Data

Pearson correlations were run between ERP components (P1, N170 and P3), the TAS-20 (total score, difficulty describing feelings, difficulty identifying feelings and externally-oriented thinking) and behavioral results (response time and accuracy). Of these correlations only three were statistically significant. The P3 amplitude was negatively correlated to response time ($r = -0.49, p = 0.002$) meaning that faster response times were linked to higher amplitude. As expected, P1 and N170 were positively correlated ($r = 0.51, p < .001$) because they share the same occipital electrode locations and have an overlap in time measurement. Unlike Pollatos
and Gramann (2011) which used pictorial stimuli, this study found no correlation between either P1 or N170 with the P3 component, suggesting that they are measuring distinct and separate processes. The TAS-20 subscale “difficulty identifying feelings” was approached a negative correlation to accuracy on the emotion perception task ($r = -0.34$, $p = .049$, controlling familywise error, significance was set at 0.0083). The total score of the TAS-20 approached significance to a negative correlation with accuracy ($r = -0.21$, $p = .066$). Surprisingly the ERP components and the TAS-20 or any of its sub-measures were not correlated.

**Discussion**

We assessed Levant et al.’s (2014) claim of a later locus of emotional inhibition (the late suppression view) for alexithymic individuals in the context of the model proposed by Damasio and Carvalho (2013). Damasio and Carvalho emphasized that early emotional arousal (based on exteroceptive processing, or emotional arousal, by the amygdala using sensory information) and later emotional regulation (based on interoceptive processing by the anterior insula and dorsal anterior cingulate cortex; Pais-Vieira et al., 2016) result in emotion perception and the experience of “feelings.” The goal of the present study was to use ERP measures to assess whether exteroceptive emotional arousal (as indexed by the P1 and N170 components) or interoceptive emotional regulation (as indexed by the P3 ERP component) resulted in the known emotional inhibition observed in alexithymia in men (Bagby et al., 1994a, 1994b; Levant, 1995, 2011; Taylor, 2000, 2004).

As was noted earlier, the semantic priming method used by Levant et al. (2014) may have been affected by factors other than vulnerable emotions (e.g., word frequency effects; Allen et al., 2005; Balota & Chumbley, 1984; Monsell et al., 1989). Thus, instead of semantic priming, we used an emotional face discrimination task (Houston et al., 2018a, 2018b) because this task
would allow us to directly distinguish between early perceptual inhibition (perhaps similar to repression effects mentioned in earlier alexithymia research) and later cognitive inhibition (perhaps similar to suppression effects mentioned in earlier alexithymia research).

We found that alexithymic men show a different pattern for P3 in relation to emotional faces, but not P1 and N170, compared to controls. While controls showed a stronger P3 effect for angry faces, alexithymic men did not. There are likely two possibilities why this might be the case. Either individuals high in alexithymia are intentionally inhibiting these emotions, or there is a general hypo-reactivity across all emotions. Furthermore, we found peak latency differences for the P1 and the N170 components. That is, when we assessed the point in time that the P1 and N170 components peaked in amplitude, alexithymic men showed a significantly later peak than did controls. This may be evidence of a general inhibition of early visual perceptual information on the part of alexithymic men relative to controls. Unlike the P1 and N170 ERP components, the P3 latency analysis yielded no significant main effect of peak latency for group. As we mentioned above, peak differences for the P3 are less common because the component in general has a far less distinct peak than earlier ERP components (Kiesel, Miller, Jolicœur & Brisson, 2008; Luck, 2005). Thus, our findings provide converging evidence for Levant et al.’s (2014) claim for conscious processing as a locus of emotion processing differences between alexithymic men and controls and an earlier disruption of emotional processing as found in Pollatos and Gramann (2011) and Vermeulen et al., (2006). Note that neither Franz et al. (2004), Campanella et al. (2012), nor Levant et al (2014) provided support for preconscious (repression) being a causal mechanism in emotional processing differences between alexithymic participants and controls (but see Pollatos & Gramann, 2011; Vermeulen et
al., 2006). Surprisingly, TAS-20 and ERP amplitude were not correlated across sub-scales and ERP components.

**Relations to Previous Studies**

Levant et al. (2014) used behavioral measures (RT and accuracy) and observed relatively weaker semantic priming effects at the long SOA (500 ms) for alexithymic men than for controls—but not for shorter SOAs (100 or 300 ms). Using ERP methods, Franz et al. (2004) also failed to find evidence of a perceptual identification deficit on the part of alexithymic participants (although Campanella et al., 2012, did find an attenuated N2 amplitude for alexithymic participants relative to controls). While this finding assists in better understanding the locus of emotion perception deficits in alexithymia, the N2 was measured from 160-250 ms after stimulus presentation, meaning the measurement was taken before and after the “consciousness threshold, 200ms”, meaning that it is not clear whether this effect is conscious or preconscious. In a behavioral priming experiment by Vermeulen et al., (2006), they found evidence for less efficient early processing in those high in alexithymia. However, the evidence was observed at an SOA of 300 ms, which may have been long enough to enter conscious awareness (Dehaene & Changeux, 2011; Fisch et al., 2009, Galliard et al., 2009; Melloni et al., 2007).

The present study provides converging evidence to the findings of Franz et al. (2004; electrophysiological evidence) and Levant et al. (2014; behavioral evidence) and suggests later processing stages (suppression) as a mechanism for alexithymia and Normative Male Alexithymia. The P3 ERP component is thought to be an index of stimulus categorization without contamination from response execution (Kutas, 1988). Thus, the present results suggest that alexithymic men show greater emotional regulation (specifically inhibition) of a fear
response (identification) elicited by angry faces (but not the emotional response from happy or neutral faces) relative to controls.

In addition to the later stages of processing, we also found evidence for the early locus of emotional perception deficit for alexithymia men. First, onset latencies for P1 and N170 components occurred later for those high in alexithymia relative to controls. This effect appears to be a general inhibition, as this effect was not influenced by the emotion of the facial stimuli. Evidence for later “conscious” processing differences were found by a group by emotional valence interaction, where angry faces caused an increased P3 component for controls, but not for those high in alexithymia. It appears that the effect seen for the P3 component reflects an independent operation than P1 and N170 because they are not correlated. Surprisingly, the group differences were only significant in the ERP data and not in the behavioral data. The behavioral data trended for better performance by the control group for both response time and accuracy. Additionally, TAS-20 score and accuracy did not interact, perhaps the stimuli used caused a ceiling effect due to a relatively low level of difficulty.

**Hemispheric Differences in Processing Emotions**

One notable finding in the present study is that both groups showed a larger P3 ERP amplitude in the right hemisphere (P4 electrode site) than the left hemisphere (P3 electrode site). This finding is consistent with the common finding that the right hemisphere plays a larger role in emotional processing than the left (Blonder, Bowers, & Heilman, 1991; Heilman, Watson, & Bowers, 1983). Lesions to the right parietal lobe (the location of the P4 electrode) have been shown to severely impact a patient’s ability to identify emotional expressions (Adolphs et al., 2000). It was noted by Sifneos (1988) that right hemisphere damage leads to symptoms similar to alexithymia (at least in respect to facial recognition). Jessimer and Markham (1997) used a
behavioral chimeric facial emotional recognition paradigm to examine hemispheric processing differences in alexithymia. Those scoring higher on the TAS-20 used a less overall leftward perceptual bias in rating faces, which provided support for the right hemisphere dysfunction model of alexithymia.

Although both groups showed significantly higher P3 amplitude on the right-hemisphere electrode (P4) than on the left-hemisphere electrode (P3), the hemispheric difference was not accompanied by an emotional valence effect. However, the observed 3-way interaction occurred because the P3 electrode site showed a strong valence effect in controls with large differences in amplitude for emotional stimuli compared to neutral stimuli (Angry = 6.63 µV, Happy = 5.53 µV, Neutral = 4.65 µV), but this was not found for alexithymic men (Angry = 5.46 µV, Happy = 5.36 µV, Neutral = 5.74 µV). That is, we observed a valenced P3 effect for controls at the left-hemisphere electrode location, but alexithymic men failed to show emotional valence effects on the P3 ERP component at any of the three electrode locations (P3, Pz, or P4). These results suggest that differences in later attentional processing occur in the right hemisphere, but group differences in emotional regulation occur in the left hemisphere (alexithymic men show an attenuated response for angry faces relative to controls that is especially pronounced at electrode location P3). Although, we acknowledge that this finding is largely data-driven and this was a post-hoc evaluation. Finally, the group difference on P3 ERP was not due to difficulty in expressing vulnerable emotions (i.e., response execution) because this ERP component does not measure response execution (Kutas, 1988).

**Categorial vs. Dimensional Approach on Alexithymia**

In the present study, we used a categorical rather than a dimensional definition of alexithymia so that an extreme-groups design could be employed. That is, using the traditional
TAS-20 cut-off score of greater than 61 (Bagby et al., 1994a, 1994b; Taylor et al., 1988), we used just two categories of “low” (TAS-20 < 51) and “high” (TAS-20 > 61). In a dimensional view of alexithymia, there would be more of a continuous approach. That is, to what degree does an individual experience the symptoms of alexithymia? While we used an extreme-groups design, we did have a range of TAS-20 scores within the low and high categories. In Figure 7, P3 amplitude is plotted as a function of TAS-20 score for just angry faces (i.e., the condition that differed most across the two groups). Key to the present concern of dimensionality of alexithymia is that, especially for the individuals in the group with high TAS-20 scores, there is minimal variability in amplitude across TAS-20 scores (although somewhat more variability for the group with lower TAS-20 scores). Thus, based on the present data with an imposed restriction of range, there is minimal support for a dimensional concept of alexithymia.

However, it is important to note that the present study was not designed to test a dimensional model of alexithymia. Therefore, additional research is necessary to address this issue. On the other hand, even if alexithymia is dimensional, the present findings show that individuals scoring high on the TAS-20 exhibit an inhibition of the P3 ERP component for angry faces (compared to happy and neutral faces) relative to controls with lower TAS-20 scores.

**Limitations**

The present study includes several limitations. First, the participants were not screened for differences in educational level, IQ, handedness, substance abuse, externally oriented thinking, substance misuse or mental health. It is possible that participant characteristics or comorbid conditions may have affected the results. Alexithymia has been shown to be often comorbid with depression and anxiety (Bagby, Taylor et al., 1994) and furthermore depression and anxiety have been shown to partially account for decoding deficits in the processing of
emotional facial expressions for those high in alexithymia (Grynberg et al., 2012). The present study found no significant differences in the behavioral task for either response time or accuracy. Accuracy was high for both groups (control 96%, high alexithymia 94%), perhaps a more difficult task would have found larger behavioral and electrophysiological differences.

Future Directions

We suggest that men, are shaped to a certain extent to avoid vulnerable emotional expression (Levant, 1995, 2011). This was not directly tested in the present study but we expect to do so in future research. Future studies may measure Traditional Male Ideology (TMI) and include a group of female participants to further understand the role of socialization on development. Additionally, it might be important to examine if there are any differences between present day male ideology and male ideology in the past. Additionally, future studies may use the three factors of the TAS-20 (difficulty describing feelings, difficulty identifying feelings and externally-oriented thinking) to better understand the roles that each of them plays in emotional processing and motivation.

References


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Table 1

**TAS-20 composite and sub-scale scores for alexithymic men and controls**

<table>
<thead>
<tr>
<th>Condition</th>
<th>TAS-20</th>
<th>Describing Emotions</th>
<th>Identifying Emotions</th>
<th>External Oriented Thinking</th>
<th>Mean</th>
<th>SE</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>SE</th>
<th>Minimum</th>
<th>Maximum</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alexithymia</td>
<td>67.11</td>
<td>18.37</td>
<td>26.32</td>
<td>22.42</td>
<td>39.11</td>
<td>1.76</td>
<td>61</td>
<td>94</td>
<td>39.11</td>
<td>1.37</td>
<td>26</td>
<td>49</td>
<td>&lt; .001*</td>
</tr>
<tr>
<td>Control</td>
<td>18.37</td>
<td>26.32</td>
<td>22.42</td>
<td>67.11</td>
<td>11.32</td>
<td>0.84</td>
<td>11</td>
<td>25</td>
<td>11.32</td>
<td>0.81</td>
<td>5</td>
<td>18</td>
<td>&lt; .001**</td>
</tr>
<tr>
<td></td>
<td>22.42</td>
<td>39.11</td>
<td>39.11</td>
<td>67.11</td>
<td>11.32</td>
<td>0.84</td>
<td>11</td>
<td>25</td>
<td>11.32</td>
<td>0.81</td>
<td>5</td>
<td>18</td>
<td>&lt; .001**</td>
</tr>
</tbody>
</table>

*P<.05; **P<.001
Table 2

*Response Time (in ms) and Accuracy (in percentage) as a Function of Group (Alexithymic Men vs. Control) and Emotional Valence (Angry, Happy, Neutral)*

<table>
<thead>
<tr>
<th>Emotional Valence:</th>
<th>Angry</th>
<th>Happy</th>
<th>Neutral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alexithymic Group:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Response Time:</td>
<td>972 (157)</td>
<td>926 (157)</td>
<td>946 (155)</td>
</tr>
<tr>
<td>Accuracy:</td>
<td>92.0 (7.9)</td>
<td>94.8 (3.6)</td>
<td>96.8 (2.8)</td>
</tr>
<tr>
<td>Control Group:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Response Time:</td>
<td>905 (152)</td>
<td>841 (125)</td>
<td>868 (155)</td>
</tr>
<tr>
<td>Accuracy:</td>
<td>94.9 (3.3)</td>
<td>95.8 (2.6)</td>
<td>97.9 (1.3)</td>
</tr>
</tbody>
</table>

Note: The standard deviation is shown in parentheses.
Table 3

*Event-Related Potentials (ERPs in µV) as a Function of Group (Alexithymic Men vs. Controls), Emotional valence (Angry, Happy, Neutral), and Electrode Site (P1: O1, O2; N170: O1, O2, T7, T8; P3: P3, Pz, P4)*

<table>
<thead>
<tr>
<th>Emotional Valence:</th>
<th>Angry</th>
<th>Happy</th>
<th>Neutral</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>P1 ERP:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alexithymic Group:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O1 Electrode Site:</td>
<td>1.96 (1.57)</td>
<td>2.27 (1.32)</td>
<td>2.62 (1.35)</td>
</tr>
<tr>
<td>O2 Electrode Site:</td>
<td>1.75 (1.89)</td>
<td>2.03 (1.70)</td>
<td>2.40 (1.66)</td>
</tr>
<tr>
<td>Control Group:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O1 Electrode Site:</td>
<td>3.17 (2.65)</td>
<td>3.18 (2.43)</td>
<td>3.54 (2.41)</td>
</tr>
<tr>
<td>O2 Electrode Site:</td>
<td>2.71 (2.69)</td>
<td>2.62 (2.58)</td>
<td>3.23 (2.66)</td>
</tr>
<tr>
<td><strong>N170 ERP:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alexithymic Group:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O1 Electrode Site:</td>
<td>2.58 (2.48)</td>
<td>2.72 (2.03)</td>
<td>3.55 (2.21)</td>
</tr>
<tr>
<td>O2 Electrode Site:</td>
<td>2.78 (2.70)</td>
<td>2.32 (2.45)</td>
<td>3.46 (2.44)</td>
</tr>
<tr>
<td>T7 Electrode Site:</td>
<td>0.23 (0.99)</td>
<td>0.47 (1.25)</td>
<td>0.36 (0.92)</td>
</tr>
<tr>
<td>T8 Electrode Site:</td>
<td>0.27 (1.09)</td>
<td>0.18 (1.24)</td>
<td>0.42 (0.95)</td>
</tr>
<tr>
<td>Control Group:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O1 Electrode Site:</td>
<td>3.47 (3.60)</td>
<td>3.15 (3.35)</td>
<td>4.00 (3.47)</td>
</tr>
<tr>
<td>O2 Electrode Site:</td>
<td>3.34 (3.51)</td>
<td>2.90 (3.52)</td>
<td>4.14 (3.53)</td>
</tr>
<tr>
<td>T7 Electrode Site:</td>
<td>0.49 (1.14)</td>
<td>0.25 (1.18)</td>
<td>0.43 (1.37)</td>
</tr>
<tr>
<td>T8 Electrode Site:</td>
<td>0.21 (1.79)</td>
<td>-0.01 (1.34)</td>
<td>0.43 (1.60)</td>
</tr>
<tr>
<td><strong>P3 ERP:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alexithymic Group:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P3 Electrode Site:</td>
<td>5.10 (3.40)</td>
<td>4.75 (3.18)</td>
<td>4.96 (3.27)</td>
</tr>
</tbody>
</table>
## EMOTIONAL PROCESSING DIFFERENCES IN ALEXITHYMIA

<table>
<thead>
<tr>
<th>Electrode Site</th>
<th>Pz Electrode Site: 5.54 (3.39)</th>
<th>5.36 (3.21)</th>
<th>5.62 (3.21)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P4 Electrode Site: 5.46 (2.75)</td>
<td>5.36 (2.56)</td>
<td>5.74 (2.65)</td>
<td></td>
</tr>
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</table>

### Control Group:

<table>
<thead>
<tr>
<th>Electrode Site</th>
<th>P3 Electrode Site: 6.63 (3.16)</th>
<th>5.53 (2.61)</th>
<th>4.65 (2.37)</th>
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</thead>
<tbody>
<tr>
<td>Pz Electrode Site: 7.44 (3.50)</td>
<td>6.70 (3.01)</td>
<td>6.99 (3.48)</td>
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</tr>
<tr>
<td>P4 Electrode Site: 7.25 (2.97)</td>
<td>6.65 (2.87)</td>
<td>7.17 (3.22)</td>
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</tr>
</tbody>
</table>

### Overall Means:

<table>
<thead>
<tr>
<th>Component</th>
<th>P1: 2.39</th>
<th>2.52</th>
<th>2.95</th>
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<tbody>
<tr>
<td>N170:</td>
<td>1.67</td>
<td>1.49</td>
<td>2.10</td>
</tr>
<tr>
<td>P3:</td>
<td>6.24</td>
<td>5.72</td>
<td>5.85</td>
</tr>
</tbody>
</table>

Note: The standard deviation is shown in parentheses.
Figure 1. Behavioral stimuli presentation

Fixation (800 ms)  Delay (100, 300 or 900 ms)  Target (Until response)  Audio Feedback (100 ms)
Figure 2. P1 by emotion (collapsed across group)

Figure 3. N170 by emotion (collapsed across group)
Figure 4a: P3 component for individuals with Alexithymia measured (300-600ms, collapsed across P3, Pz and P4)

Figure 4b: P3 component with Controls measured (300-600ms, collapsed across P3, Pz and P4)
Figure 5. N170 (measured from 140-190ms, Collasped across T7, O1, T8 and O2)

Figure 6a: P1 Source location.

Figure 6b: N170 Source location.
Figure 6c: P3 Source location.
Figure 7: P3 (Collapsed across p3, pz and p4) Amplitude (µV) for angry faces plotted as a function of TAS-20 score.