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# Frontal P300 and Alexithymia

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by

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Abstract

Event related potential (ERP) methodology provides a measure of brain electrical activity in response to an experimental stimulus. The P300, an endogenous component of the ERP, is associated with cognitive activity and is purported to reflect stimulus recognition and information processing. Previous results from our lab suggest that alexithymic individuals, those who are unable to identify and label their own emotional state, show reduced early cognitive processing of affectively neutral auditory stimuli in the frontal regions of the brain. Based on the premise that alextihymia could be the result of an increased left hemisphere activation or reduced activation of the right hemisphere it was expected that the P300 amplitude of the left hemisphere would be greater than that of the right hemisphere. Specifically, the left frontal P300 amplitude was expected to be greater than the central and posterior P300 amplitudes of the left hemisphere; as the discrepant activity in the prefrontal cortex and the anterior cingulate cortex (ACC) has been found in patients with alexithymia (Berthoz et al., 2002; Kano et al., 2003; Lane et al., 1997). In the present study, alexithymia correlated with the P300 amplitude. However, results varied for each hypothesis by TAS-20 factor scores and not the TAS-20 total score. Future studies focusing on the specific factors that comprise the multifaceted construct of alexithymia (e.g., as measured by the TAS-20) may provide more insight into the dimension of emotion and brain/behavior relationships.

## Introduction

Alexithymia (from the Greek "a" for lack, "lexis" for word, and "thymos" for emotion) is a disturbance in emotional processing that is manifested clinically by difficulties in identifying and verbalizing feelings, in elaborating fantasies, and by a tendency to focus on the somatic sensations accompanying emotional arousal. Research in the field of alexithymia has stemmed from neurophysiological, psychoanalytic, social learning, developmental, and genetic points of view. A review of the neurophysiological literature illustrates that alexithymia is posited to be a result of: (1) a deficit in interhemispheric communication or (2) a dysfunction in the right cerebral hemisphere. Hoppe and Bogen (1977) observed an impoverishment of dreams and fantasies and a decreased ability to describe feelings in patients with cerebral commisurotomies. Houtveen, Bermond, & Elton (1996) classified two types of alexithymia: Type I alexithymia – the absence of emotional experience but presence of thoughts that accompany emotions, and Type II alexithymia – the presence of emotional experience but absence of accompanying thoughts. This deficit in affect is postulated to result from the improper communication between limbic and neocortical systems in the brain or a deficit in interhemispheric transfer.

Electrophysiological measures have provided a means of examining the brain's response to experimental stimuli. The event-related potential (ERP) methodology is commonly employed to determine physiological correlates associated with psychological constructs, such as cognitive processes, emotionality, and personality constructs (Berger, Starratt, & Starratt, 2004). Latencies and amplitudes of specific components of the ERP can be examined to ascertain these relationships between physiological and psychological

variables. Within the ERP, the P300 is a component associated with cognitive and subjective factors such as stimulus recognition and information processing (Andreassi, 2000). The P300 is elicited by the use of an auditory oddball task which aides in the production of a well-defined peak (Berger, Starratt, & Starratt, 2004).

The aim of the present study was to explore the relationship between the P300 component and alexithymia. Based on the neurophysiological propositions of the development of alexithymia, (either a deficit in interhemispheric communication or a dysfunction in the right cerebral hemisphere), it was hypothesized that P300 amplitudes would be positively correlated with scores on a measure of alexithymia – the Toronto Alexithymia Scale (TAS-20). In particular, we expected that alexithymic individuals, (those with high TAS-20 scores), would show an enhanced left frontal P300 amplitude to affectively neutral auditory stimuli compared to the left central and posterior P300 amplitudes. Further, we expected the left frontal P300 amplitude to be greater than the right frontal P300 amplitude for alexithymic individuals.

## Literature Review

Event-related potentials will be introduced, highlighting the P300 component. The auditory oddball paradigm is also described as the task used to elicit a well-defined P300. A brief history of alexithymia will be reported followed by the neurophysiology of emotion and alexithymia, and finally the development of the Toronto Alexithymia Scale (TAS-20). The hypotheses and rationale will conclude the section.

### *Event-Related Potentials*

The event-related potential, or ERP, is a neurophysiological measure of the changes in the electrical activity of the brain in response to a specific sensory or cognitive event. ERPs are derived from electroencephalographic (EEG) recordings which reflect the constant and spontaneous electrical activity of the brain. ERPs are employed to identify the brain's response to experimental stimuli in an effort to understand how the brain processes information. Short latency components reflect sensory signals from receptors via ascending pathways to the cortex (Kotchoubey, Lang, Bostanov & Birbaumer, 2002). ERPs are of cortical origin and generally investigated between 100 and 1000 ms. The ERP methodology provides a non-invasive technique that allows for tracking of processing from stimulus presentation at complex levels (Kotchoubey et al., 2002).

An underlying assumption is that EEG activity which is not related to the event will vary randomly across epochs, form "background noise" of the EEG, and average to zero. The resultant ERP waveform represents activity that displays a fixed temporal relationship to the event, which is the event-related potential (Rugg & Coles, 1995).The

ERP waveform is generally illustrated as having three separate epochs, each with distinct peaks or components. Early brainstem waves (also called short latency components) occur in the interval from 0 to 10 ms following presentation of a stimulus. Early cortical waves, or middle latency components occur in the interval from 10 to 80 ms following stimulus onset. Late cortical waves or long latency components, including the N100, P200, N200 and P300 components (also called N1, P2, N2 and P3, respectively), occur at 80 to 1000 ms post stimulus (Hillyard, Mangun, Woldorff, & Luck, 1995; Woods, 1995).

*Components of the ERP*

In contrast to brainstem waves, the components of late cortical waves are labeled according to their polarity (N refers to negative polarity and P to positive polarity) and the number indicates the approximate time of occurrence following the presentation of a stimulus. To analyze the ERP waveform, amplitudes and latencies of the components of interest are determined. Amplitudes are measured in microvolts (μV) and latencies are measured in milliseconds (ms) (Drake, 2004). For example, the P300 (or P3) is a major cognitive component which occurs at approximately 300 ms following stimulus onset and has a positive deflection (Hillyard et al., 1995).

The ERP wave is composed of both exogenous (reflecting stimulus variables) and endogenous (reflecting cognitive variables) components. Exogenous ERP components occur in all three latency epochs, regardless of the direction of attention, and are contingent upon the physical properties of the stimulus that is presented. Specifically, brainstem waves, middle latency components and the N1 and P2 components of the long latency components are considered to be exogenous (Näätänen, 1992). Conversely,

endogenous components of the ERP typically occur during the long latency epoch, include N2 and P3 components, and emerge only in response to specific cognitive manipulations (Woods, 1995).

*N100.* N1 is the negative peak that occurring approximately 100 ms following stimulus presentation, The N1 component is theorized to represent the brain's processing of sensory information. When a stimulus is presented, the N1 component is believed to reflect the point when the brain first recognizes that a stimulus has been presented (Drake, 2004). This is especially true in auditory stimuli research. The N1 is an exogenous component and has been related to attention (Andreassi, 2000).

*P300.* The P300 is perhaps the most-studied ERP component in investigations of selective attention and information processing. It was first identified by Sutton, Braren, Zubin, and John (1965) who discovered that the P300 was the major component associated with anticipation of the stimulus. This extensively researched component of the ERP wave is manifested as a positive peak and occurs approximately 300 ms after the presentation of a stimulus. The P300 ERP component is considered to be a useful measure because P300 amplitude is interpreted as a measure of central nervous system (CNS) activity and as specifically reflective of information processing during instantiation of both the memory representation and the context in which a stimulus occurs (Polich, 1998). The P300 is an endogenous component and has been associated with cognitive processes such as decision making, attention, discrimination, stimulus relevance, and information delivery (Andreassi, 2000). The P300 is considered as the peak of the waveform where alexithymics engage in the cognitive processing of the deviant/infrequent stimuli in an auditory-oddball paradigm.

*ERP Methodology*

The International Ten-Twenty System (Jasper, 1958; Appendix C) was developed to facilitate the standardized placement of electrodes for the electrophysiological measurement of cortical activity. Within this system, electrode sites are designated with a letter that indicates their proximity to particular regions of the brain, specifically the frontal (F), parietal (P), central (C), temporal (T), and occipital (O) regions. Numbers denote proximity of electrode locations to the midline with odd numbers designated for the left hemisphere and even numbers for the right. For example, left and right hemisphere electrode sites at frontal locations would be labeled F3 and F4 respectively (Drake, 2004).

*Auditory Oddball Paradigm*

ERP components can be elicited using visual, auditory, or tactile stimulation. A common task used to elicit the waveform components using auditory stimuli is the auditory oddball paradigm. It is perhaps one of the most well-studied perceptual discrimination tasks. Essentially, participants are asked to discriminate target (or infrequent) tones from standard (or frequent) tones, and make a response indicating the detection of the target tone. The task has been well-studied electrophysiologically, with the P300, an event-related potential (ERP), identified as a neural correlate of the underlying target detection processes (Polich, 2007).

The auditory oddball task has therefore been employed as a useful tool for stimulus presentation in ERP studies as it produces a well-defined P300 (Polich, 1986, 2007). For the purposes of the current alexithymia study, this paradigm involves the

presentation of a string of stimuli comprised of 2 different tones: a high auditory frequency tone (the standard) occurring approximately 80% of the time and a low auditory frequency tone (the deviant) occurring approximately 20% of the time. The deviant tones/stimuli are randomly inserted in a sequence of standard, frequent stimuli where the participant counts the infrequent/rare events (Kotchoubey et al., 2002).

The use of the auditory oddball task can be seen in the work of Bermond, Righart, Ridderinkhof, & Moormann (2007). To clarify the effects of alexithymia on the P300 response, participants engaged in a visual oddball task while scalp EEG was recorded. The oddball task consisted of a series of non-target stimuli, intermixed with less frequent target stimuli, emotional stimuli, and neutral stimuli. The results of this study indicated P300 amplitudes were enhanced for emotionally-charged pictures compared with neutral pictures.

The inverse relationship between P300 amplitude and target stimulus probability has been well-established. Larger amplitudes are obtained with low probability in auditory, visual, and somatosensory modalities (Polich, 2003). The results illustrate that P300 amplitude and response time increased, and peak latency decreased, as the target stimulus interval increased (Gonsalvez, Barry, Rushby, & Polich, 2007).

*Alexithymia*

The concept of alexithymia evolved from the clinical observations of psychosomatic disorders. A description of alexithymic characteristics was reported by MacLean (1949) when he noted psychosomatic patients' inability to verbalize their feelings. Ruesch (1948) noted similar limitations in symbolism and verbalization in post traumatic and psychosomatic patients, and considered them to have an 'immature' or 'infantile personality'. Patients were unimaginative, socially conforming, and unresponsive to insight-oriented psychotherapy (Taylor & Taylor, 1997). Sifneos (1967), and Nehmiah and Sifneos (1970) coined the term alexithymia to describe a disturbance in the experience and expression of emotion that had been observed in individuals with "psychosomatic" illnesses. The term alexithymia was formed from the Greek words *a* = lack, *lexis* = word, *thymos* = mood/emotion which literally translates to a lack of emotion. The symptoms observed by Sifneos were an impoverished fantasy life (a utilitarian way of thinking), the use of action to avoid conflicts in stressful situations, a marked constriction in experiencing emotions, and difficulty in finding appropriate words to describe their feelings (Sifneos, 1973).

Alexithymia was once viewed as a categorical construct which proposes that the trait is either present or absent (Gündel et al., 2004). Nowadays, alexithymia is conceptualized as multifaceted and dimensional which indicates the trait has a normal distribution in the general population devoid of relation to intelligence, socioeconomic status, educational level or culture (Taylor, Bagby, & Parker, 1997). It is described as a cognitive and affective disorder of emotional processing that impairs the ability to develop cognitive representations of emotions (Parker, Bagby, Taylor, Endler, &

Schimtz, 1993). Alexithymia is now characterized by three factors: a) impaired ability to identify one's own feelings and to discriminate between physiological sensations and emotion; b) decreased ability to describe emotion; and c) a reliance on externally oriented thinking (i.e., avoidance of exploration of one's own thoughts and feelings) (Parker et al., 1993). These skills appear to be relatively normally distributed with approximately 10% of the normal population showing characteristics of alexithymia.

Alexithymics demonstrate significant impairments in both the verbal and non-verbal recognition of emotion stimuli (Lane et al. 1996). Alexithymics are reportedly literal, unimaginative, utilitarian, and lack insight, humor and personal meaning in life (Haivland and his colleagues, 2002). Supporting data reveals that alexithymics lack the emotional experience of love, happiness, and joy, thereby exhibiting an anhedonic quality in their lives (Taylor, Bagby and Parker 1997). Taylor and his colleagues (1997) illustrated that while alexithymic individuals experience feelings, they are more likely to experience negative or unpleasant emotions, with a limited capacity to regulate them effectively through cognitive or other psychological processes. Some researchers have reported that alexithymics demonstrate a limited ability to show empathy in social interactions (Krystal 1979; Taylor, Bagby and Parker 1997; Bekendam 1997).

Clinical research on alexithymia has focused on relationships between alexithymic characteristics and medical and psychiatric disorders. Alexithymia is a personality trait believed to increase an individual's vulnerability to such psychiatric and medical conditions as depression, post-traumatic stress disorder, substance abuse, pain and eating disorders, hypertension, obesity and gastrointestinal dysfunction, and other psychosomatic conditions (Sifneos 2000; Taylor 2000; Porcelli et al. 1999).

It is believed that alexithymia can be either neurogenic (caused by biological abnormalities) or psychogenic (caused by past experience or psychological trauma). There is no recognized functional distinction between neurological and psychological strains of alexithymia, and hence no clinical test to ascertain the cause (Lumley, Stettner, & Wehmer, 1996). Although the etiology of alexithymia remains unknown, several theories suggest a neurobiological basis for this disorder (e.g., MacLean, 1949; Houtveen, et al., 1996; Lumley & Sielky, 2000) and a few studies have demonstrated psychophysiological differences in neural processing related to alexithymia (e.g., Berthoz, et. al., 2002; Starratt, Starratt, Berger & Florville, 2003). Alexithymia describes the cognitive-affective dimension of emotion. If a person cannot identify their own emotions or describe emotion, impaired affect regulation and interpersonal communication skills could result (Taylor & Bagby, 1997).

*Neurophysiology of Emotion*

The brain has evolved through a progressive elaboration of circuits surrounding the brainstem core (Derryberry & Tucker, 1992). Primitive brainstem structures regulate autonomic, endocrine, and motor activities of the body which are fundamental to the expressive and instrumental functions of emotion (Derryberry & Tucker, 1992). The limbic system differentiated to provide greater sensitivity to emotional signals in the environment, flexible emotional responses, and a greater capacity for learning. Then, the cortical structures began to differentiate, from the paralimbic regions surrounding the limbic system and progressing toward the most recent neocortical fields (Derryberry & Tucker, 1992). The evolving cortex afforded higher resolution representation of the emotional environments, which resulted in highly refined emotional responses, and

enhanced cognitive capabilities related to anticipation and planning (Derryberry & Tucker, 1992). As the higher levels of the brain evolved, descending connections were established with the primitive brainstem structures. Consequently, the descending projections allow the cognitive processes of the cortex to regulate the emotional functions of the limbic system and brainstem. This allows for finely tuned peripheral responses and provides mechanisms through which emotion may influence learning and cognition (Derryberry & Tucker, 1992).

The anterior cingulate cortex (ACC) is a heterogeneous structure that helps to orchestrate the motor, neuroendocrine, and autonomic responses to emotional stimuli (Vogt, Finch, & Olson, 1992). The ACC has direct monosynaptic connections to the vagal nuclei (responsible for intake of air into the lungs and further expiratory activity) in the brainstem (Hurley, Herbert, Moga, & Saper, 1991). The transmission of emotion information to the ACC permits the completion of a feedback loop to modulate sympathetic arousal. Failure of such transmission could lead to exaggerated and persistent sympathetic discharge.

If this is true, the current model provides a physiological basis for an emotion regulation theory (Schwartz, 1983), which states that conscious awareness of emotions promotes both psychological (promoting optimal adaptation by integrating all available information, including that derived from emotions) and physiological health. Alexithymia may be associated with a dysfunction in the ACC activity during emotional arousal due to a deficit in blood flow (Lane et al., 1997).

*Neurophysiology of Alexithymia*

The first published interest in the underlying neurobiological mechanisms of alexithymia was shown by Hoppe & Bogen (1977) who observed 12 "split-brain" individuals (i.e. patients who had undergone cerebral commissurotomies for treatment of intractable epilepsy). These patients appeared to be deprived of "hemispheric bisociation" (a term developed by Hoppe) which facilitates the verbal expression of emotions (Hoppe & Bogen, 1977). A lack of fantasy and ornate dream content was also observed and reported in Hoppe's interviews of the epileptic patients with cerebral commissurotomies. Buchanan, Waterhouse, & West (1980) examined patients with agenesis of the corpus callosum and observed a right hemispheric deficit. Voeller (1986) observed the occurrence of alexithymia more often in patients with stroke who had a lesion in the right rather than the left cerebral hemisphere. All three populations mentioned above exhibited symptoms of alexithymia.

Researchers have speculated that alexithymia is associated with deficits in the ACC activity during emotional arousal. In a study using functional magnetic resonance imaging with 16 healthy participants, Berthoz et al. (2002) reported that alexithymics' level of activity in the ACC and mediofrontal region of the brain differed from controls during emotional stimuli processing. Kano and colleagues (2003) demonstrated that alexithymia is related to differences in blood flow in the ACC, during facial expression recognition tasks.

The functional commissurotomy model posits that the alexithymic brain does not effectively transfer emotional information from the right hemisphere to the language

centers of the left hemisphere (Taylor, 2000). Research conducted with alexithymic and nonalexithymic veterans diagnosed with PTSD, supports the hypothesis that a dysfunction in interhemispheric communication may account for alexithymia (Zeitlin et al. 1989).

Houtveen et al. (1996) examined Type II alexithymia (the presence of emotional experience but absence of accompanying thoughts) in a neurologically normal undergraduate population and investigated the relationship between Type II alexithymia and interhemispheric processing. The study results indicated that those who scored high in alexithymia showed reduced communication between the right and left brain hemispheres compared to those who scored low in alexithymia. Taylor and colleagues (1997) suggested that alexithymia is associated with impairment in interhemispheric communication or a dysfunction in the right cerebral hemisphere. Parker et al. (1999) and Zeitlin et al. (1987) supported that alexithymic males displayed a bidirectional interhemispheric transfer deficit and not a dysfunction in either hemisphere.

Preliminary neurobiological hypotheses postulate a deficit in interhemispheric communication or a dysfunction in the right cerebral hemisphere. Based on these hypotheses, it is still unclear if alexithymia is a direct result of a deficit in the right hemisphere or if a dysfunction in the right hemisphere causes an over-activation of the left hemisphere which results in the disruption of interhemispheric communication and integration. Taylor and Bagby (2000) reported that, although these correlational studies have detected neural differences associated with alexithymia, they do not imply cause-effect relationships. The results acquired from these studies can be used to formulate a holistic psychobiological model of alexithymia, in which neural correlates may interact

with environmental influences during early development such as the quality of emotional interaction and attachment with the caregiver, and/or the presence of traumatic or neglectful experiences.

*The Toronto Alexithymia Scale (TAS-20)*

The TAS-20 was developed using a combined empirical and rational method of scale construction. Taylor and Bagby (2000) explained that the second and third factor together adequately measure the deficits in daydreaming and other imaginative activity originally assessed by a fourth factor in the original TAS-26. The TAS-20 provides a reliable and valid method of measuring the construct alexithymia (Taylor, 2000) and has been replicated in both student and psychiatric populations in North America (Parker, Taylor and Bagby 2003). In a community sample of approximately 2000 adults, Parker, Taylor and Bagby (2003) administered the TAS-20 and used factor analysis to report that the test was replicable in the entire sample (internal reliability coefficient = .86).

The test consists of twenty statements, for which an individual must choose a response on how much they agree or disagree with each statement. Responses on a five point Likert scale range from strongly disagree to strongly agree. In addition to yielding a total alexithymia score (high scores indicating alexithymia), the twenty-item Toronto Alexithymia Scale (TAS-20) also assesses three unique factors: 1) difficulty identifying feelings and distinguishing them from bodily sensations of emotion, 2) difficulty describing feelings to others, and 3) an externally oriented style of thinking (Parker et al., 1993). A total score above or equal to 61 on the TAS-20 signifies an individual as

"alexithymic" and a score below or equal to 51 signifies an individual as "non-alexithymic".

The literature indicates that alexithymia may be the result of a deficit in the right hemisphere and the dysfunction may lie in the ACC activity (Lane et al., 1997). If the dysfunction in the right hemisphere causes an over-activation of the left hemisphere (Buchanan, Waterhouse, & West, 1980; Voeller, 1986), then an enhanced amplitude of the left frontal P300 should be observed. Following this rationale, the present study investigated the relationship between alexithymia and the left frontal P300 with the following stated hypotheses.

*Hypotheses*

*Hypothesis 1:* TAS-20 scores will be positively correlated to P300 amplitudes.

It was expected that as TAS-20 total and factor scores increased, P300 amplitudes at all sites (frontal, central, and parietal) would also increase. This would effectively indicate that those high in alexithymia would generate higher amplitudes in reaction to deviant/infrequent stimuli.

*Hypothesis 2:* Alexithymic individuals, (those with high TAS-20 scores), will show an enhanced left frontal P300 amplitude to affectively neutral auditory stimuli compared to the left central and posterior P300 amplitudes.

Since it has been proposed that alexithymia could be the result of a hyperactive left hemisphere or reduced activation of the right hemisphere it was expected that the P300 amplitude of the left hemisphere will be greater than that of the right hemisphere.

Specifically, the left frontal P300 amplitude was expected to be greater than the central and posterior P300 amplitudes, given that the prefrontal cortex and the ACC have been implicated in patients with alexithymia (Berthoz et al., 2002; Kano et al., 2003; Lane et al., 1997).

*Hypothesis 3:* The left frontal P300 amplitude will be greater than the right frontal P300 amplitude for alexithymic individuals.

To further test the theory that a dysfunction in the right hemisphere causes an over-activation of the left hemisphere, this hypothesis was investigated. More importantly, we were curious as to whether this difference can be detected by use of the ERP methodology.

## Methods

As part of a larger electrophysiological study, 350 neurologically normal undergraduate students were scheduled for two sessions where they: (1) completed the Toronto Alexithymia Scale (TAS-20) and (2) underwent auditory oddball ERP recording.

### *Participants*

Participants in the present study consisted of 162 students (34 men and 128 women) from Barry University in South Florida. Within the total sample, ages ranged from 18 to 53 years old with a mean age of 23 (sd = 5.8). Participants were divided into high and low alexithymic groups based on their total TAS-20 score. There were 16 participants in the low alexithymic group (total TAS-20 score $\leq$ 29), ages ranged from 18 to 40 years old with a mean age of 24.4 (sd = 6.6). In the high alexithymic group (total TAS-20 score $\geq$ 61), the sample consisted of 19 participants whose ages ranged from 16 to 32 years old with a mean age of 22.8 (sd = 4.6).

Participants were scheduled for two appointments, the first to complete the psychometric evaluation and consent forms documentation, and the second to record ERP data. If professors accepted extra credit for research participation, documentation of participation was provided for students. Participants earned up to three extra credit unit slips for the two sessions required for this study. They were informed of all testing procedures prior to consent and voluntary participation was highlighted. Each participant was informed that minimal discomfort would be experienced during the study. Demographic information including gender, age, handedness, and history of head injuries were collected (Appendix A). Identifying information was not included on any of the

measures, data, or records. Consent forms were kept separately in a locked filing cabinet in the principal investigators office. Deception was not used.

*Measure*

Participants completed the 20-item Toronto Alexithymia Scale (TAS-20; Parker et al., 1993; Appendix B). This is a common self-report measure of alexithymia. The TAS-20 assesses three factors: (a) difficulty identifying feelings and distinguishing them from bodily sensations of emotion, (b) difficulty describing feelings to others, and (c) an externally oriented style of thinking (Parker et al., 1993). For clinical purposes, a high score (61 and above) on the TAS-20 indicates the presence of alexithymia and a low score (51 and below) is considered normal. The TAS-20 uses a 5-point Likert format ranging from 1 (*strongly disagree*) to 5 (*strongly agree*) for participants to rate themselves on statements describing traits of alexithymia. The possible total scores range from 20 to 100. To obtain the total score, the TAS-20 score form is used (Appendix F). The responses for items 4, 5, 10, 18, and 19 are reverse-scored after which all 20 responses are combined for a total score. Participants were divided into high and low alexithymic groups based on the $10^{th}$ and $90^{th}$ percentile of TAS-20 scores. Participants who had a score of 61 and above on the TAS-20 were considered high in alexithymia and those who had a score of 29 and below were considered low in alexithymia.

*ERP Recording and Stimuli*

Testing was occurred in the Psychophysiology Lab in the Psychology Department of Barry University. PsyLab hardware and software was used for ERP data acquisition, storage, artifact rejection, and the initial stages of processing for each participant. The

PsyLab EEG8 is an eight channel biological amplifier unit that also permits impedances to be monitored. The International Ten-Twenty System (Appendix C) was used to record ERP data from multiple electrode locations. Accordingly, the standardized pattern of electrode placement was followed: midline, right, and left hemisphere locations over frontal, temporal, occipital, and parietal brain areas. These locations were demarcated as F3, F4, C3, C4, P3, and P4 scalp sites. The earlobes were used as a reference electrode which measured electrical activity of the body *without* cortical input and the forehead was used as a ground. Two electrodes were also placed near the right eye (one directly above the eye and one at the outer canthus) to monitor eye movement. Electrodes were attached and removed following standard procedures for infection control (Society for Psychophysiological Research Guidelines, Putnam, 1992) and were sterilized between uses (the method for this study has been described in Drake, 2004).

PsyLab hardware and software was used to amplify and record EEF and EOG (i.e., eye muscles) signals. Amplifiers were set at a gain of 200μV for active electrode sites. Upper and lower band pass limits were set at 0.1 and 100 Hz, respectively. A 1000 ms epoch was digitized on a trial-by-trial basis with a sampling rate of one data point every 10 ms, to yield a total of 100 points per digitized epoch. A pre-stimulus interval of 100 ms was included in the digitizing procedure for the purposes of baseline correction. Using PsyLab programming, trials contaminated by artifact were eliminated from the data set prior to waveform averaging.

For each participant, approximately 200 tones were employed in an oddball paradigm where 20% of the tones were higher pitched target tones. The 200 trials were presented with an average inter stimulus interval (ISI) of 2-seconds in an ignore condition

where participants were simply asked to listen to the tones. Auditory stimuli (100 ms sinusoidal tone pulses) were generated by a PsyLab tone generator and presented binaurally via headphones at an intensity equivalent to conversational speech. Standard and deviant tones were 40 Hz and 85 Hz, respectively.

Data for all participants submitting complete TAS-20 data and showing an ERP waveform free of artifact were included in the final analyses. All data were stored on the personal computer on which they were gathered. Initial calculation of average waveforms for each participant was accomplished on PsyLab software. Each participant's averaged waveform gathered in response to the deviant tone, at each of the six electrode locations, was downloaded for further analysis. Baseline correction of individual participant data and the calculation of grand average waveforms for each group were conducted in Excel. Statistical analysis was conducted using Statistical Package for the Social Sciences (SPSS).

*Procedure*

After an explanation of all procedures, participants signed an informed consent (Appendix D) and an Authorization to Link form (Appendix E). A copy of the informed consent was give to each participant who then completed the demographic questionnaire (Appendix A) and the TAS-20 (Appendix B). Each participant was then seated in a comfortable chair in the psychophysiology lab where his/her head was measured and electrode sites were marked. Each electrode site was first cleaned with a topical antiseptic microbicide (Betadine), followed by the application of NuPrep (a slightly abrasive topical gel used to reduce skin impedance). A small amount of Ten 20 Conductive Paste (white,

opaque adhesive paste) was placed on electrodes before they were applied to the marked sites on the scalp. The same procedure was used for earlobe, forehead, and eye electrode placements. Earlobe electrodes were attached with ear clips. Impedance for all electrodes was maintained at under 5kOhms.

During ERP testing, participants were asked to relax and sit comfortably with their eyes open. Headphones were placed on the participant to present auditory stimuli in an oddball paradigm. Each participant was tested in two experimental conditions. For the first condition, the participant was instructed to focus their eyes on a comfortable spot in front of them, blink as little as possible, and simply listen to the tones. A short break (2-5 minutes) followed the first condition, during which time the headphones were removed and the participant was able to shift positions while remaining seated. For the second condition, the headphones were replaced and the participant was instructed to count the higher pitched (deviant) tones. The data gathered in this attend condition was not a focus of the present study and will not be referenced. Each session lasted approximately 10 minutes. All data was gathered and stored on PsyLab software on a personal computer, locked in the psychophysiology lab, for further statistical analysis. After the ERP sessions, electrodes and remaining paste were removed from the participants scalp with a cotton swab and water. Any questions were then answered and credit units were distributed to the participant. The entire procedure lasted between 1 hour 30 minutes and 2 hours per participant.

## Results

The demographics of the sample are displayed in Table 1. Gender and age have already been reported in the Methods section for the three groups, total, low alexithymics, and high alexithymics. Handedness and head injury are also reported as the measured neuropsychological demographics.

**Table 1**

*Demographic characteristics of sample*

| Variables | Low Alexithymics | High Alexithymics | Total Sample |
|---|---|---|---|
| N | 16 | 19 | 162 |
| Gender | | | |
| Male | 5 | 2 | 34 |
| Female | 11 | 17 | 128 |
| Age | | | |
| Range | 18 – 40 | 18 – 32 | 18 – 53 |
| Mean | 24.4 | 22.8 | 23 |
| Handedness (%) | | | |
| Right Handed | 92.9 | 85.7 | 81.6 |
| Left Handed | 0 | 14.3 | 16.3 |
| No Preference | 7.1 | 0 | 2.1 |
| Head Injury (%) | | | |
| Yes | 0 | 0 | 4.3 |
| No | 100 | 100 | 95.7 |

P300 was coded as the largest positive amplitude between 270 and 340 ms. An alpha level of .05 was used to establish significance for all statistical tests. All data were analyzed using Predictive Analytics Software (PASW), formerly known as the Statistical Package for Social Sciences (SPSS). Correlations were used to evaluate relationships between variables and ANOVAs were conducted to investigate the dependent variable P3 amplitude across the left brain regions (frontal, central, and parietal) for TAS-20 scores. A total of four TAS-20 scores were used as follows: total score, factor 1 –difficulty identifying feelings and distinguishing them from bodily sensations of emotion, factor 2 difficulty describing feelings to others, and factor 3 an externally oriented style of thinking (Parker et al., 1993). Within group analyses compared right and left hemisphere amplitudes of the P3 component of the ERP waveform. Results will be discussed as they relate to hypotheses.

*Hypothesis 1: TAS-20 scores will be positively correlated to P300 amplitudes.*

Hypothesis 1 was partially supported. Table 2 reports the Pearson's r and p values for the correlation conducted between TAS-20 scores and P300 amplitudes. Of the six measured P300 amplitudes, the left frontal region was the only brain region that was significantly positively correlated with TAS-20 factor 1 (difficulty identifying feelings and distinguishing them from bodily sensations of emotion); $r = .208$, $p = .008$. No other brain region P300 was significantly correlated with any other TAS-20 score. This finding lends partial support to our original hypothesis that TAS-20 scores would be positively correlated with P300 amplitudes.

**Table 2**

*Correlations of TAS-20 scores and P300 amplitudes*

| TAS-20 Score | Frontal P300 | | Central P300 | | Parietal P300 | |
|---|---|---|---|---|---|---|
| | Left | Right | Left | Right | Left | Right |
| Total | .126 | -.087 | -.034 | -.031 | .009 | .057 |
| Factor 1 | .208* | -.002 | .032 | .003 | -.004 | .053 |
| Factor 2 | .043 | -.120 | -.069 | -.077 | .006 | -.002 |
| Factor 3 | -.027 | -.105 | -.085 | -.035 | -.017 | .035 |

***$p < .01$**

*Hypothesis 2:* Alexithymic individuals, (those with high TAS-20 scores), will show an enhanced left frontal P300 amplitude to affectively neutral auditory stimuli compared to the left central and posterior P300 amplitudes.

Four within-subjects ANOVAs were conducted for the TAS-20 total, factor 1, factor 2, and factor 3 scores against the dependent measure, P300 amplitude across each brain region. Each of the four analyses used only the participants with TAS-20 total scores above 61 placing them within the alexithymic category. Only one of the four ANOVAs yielded a statistically significant effect. The analysis of variance performed on the data for TAS-20 factor 1 scores (difficulty identifying feelings and distinguishing them from bodily sensations of emotion) yielded a significant interaction effect; $F = 4.157$, $p = .048$. Post hoc analyses demonstrated that the left frontal P300 is statistically

significantly greater than both the central and parietal P300 values. The results indicate that alexithymic individuals, (those with high TAS-20 scores), illustrate an enhanced left frontal P300 amplitude to affectively neutral auditory stimuli compared to the left central and posterior P300 amplitudes only when the alexithymic participants are grouped on TAS-20 Factor 1 (difficulty identifying feelings).

**Table 3**

*Means and Standard Deviations of the left P300 amplitudes*

| *N*: Low Alexithymics = 16, High Alexithymics = 19 | | Frontal P300 | | Central P300 | | Parietal P300 | |
|---|---|---|---|---|---|---|---|
| TAS-20 Score | | Mean | SD | Mean | SD | Mean | SD |
| Total | High | 10.78 | 6.64 | 3.12 | 4.42 | 1.52 | 5.73 |
| | Low | 8.59 | 6.19 | 4.76 | 4.62 | 1.15 | 4.25 |
| Factor 1 | High | 10.24 | 6.67 | 4.50 | 4.94 | 1.60 | 5.32 |
| | Low | 5.59 | 5.15 | 3.82 | 4.39 | 1.34 | 4.73 |
| Factor 2 | High | 9.24 | 6.69 | 3.63 | 4.76 | 1.15 | 5.12 |
| | Low | 8.69 | 4.98 | 4.98 | 4.92 | 1.74 | 4.84 |
| Factor 3 | High | 7.84 | 6.30 | 2.39 | 4.67 | .66 | 4.88 |
| | Low | 9.07 | 5.65 | 5.24 | 4.55 | 2.07 | 4.58 |

*Hypothesis 3:* The left frontal P300 amplitude will be greater than the right frontal P300 amplitude for alexithymic individuals.

Four ANOVAs were conducted to determine the relationship between the left and right frontal P300 amplitudes for alexithymic individuals (those with high TAS-20 scores). The ANOVA performed on P300 amplitudes for alexithymics based on TAS-20

total scores yielded a statistically significant hemispheric difference, $F = 10.369$, $p = .003$. That is, alexithymics demonstrated a greater left frontal P300 than the right frontal P300. Results for alexithymics based on TAS-20 factor 1 scores yielded a significant hemispheric different P300 amplitudes, $F = 6.716$, $p = .013$. The ANOVA performed for the TAS-20 factor 2 scores and factor 3 scores were statistically insignificant. The means and standard deviations are presented in Table 4.

**Table 4**

*Means and Standard Deviations of the frontal P300 amplitudes*

| *N*: Low Alexithymics = 16, High Alexithymics = 19 | | Left Frontal P300 | | Right Frontal P300 | |
|---|---|---|---|---|---|
| TAS-20 Score | | Mean | SD | Mean | SD |
| Total | High | 10.78 | 6.64 | 4.51 | 5.25 |
| | Low | 8.59 | 6.19 | 7.91 | 6.89 |
| Factor 1 | High | 10.24 | 6.67 | 5.81 | 4.53 |
| | Low | 5.59 | 5.15 | 5.46 | 6.75 |
| Factor 2 | High | 9.24 | 6.69 | 4.71 | 5.31 |
| | Low | 8.69 | 4.98 | 7.28 | 6.41 |
| Factor 3 | High | 7.84 | 6.30 | 4.24 | 4.65 |
| | Low | 9.07 | 5.65 | 7.24 | 5.73 |

## Discussion

The results indicate that as alexithymia scores increase, some P300 amplitudes also increase which lends partial support to Hypothesis 1. Interestingly, when correlations were conducted between the TAS-20 and P300s of different brain regions, only one significant relationship was revealed. That is, only the relationship between TAS-20 factor 1 and the left frontal P300 was significantly and positively related. While some neurobiological hypotheses postulate that a dysfunction in the right cerebral hemisphere causes an over-activation of the left hemisphere, this was not fully supported by the current findings (Berthoz et al., 2002; Kano et al., 2003; Lane et al., 1997). The only significant positive correlation was the left frontal P300 amplitude for TAS-20 factor 1 (difficulty identifying feelings). This partially supported our original hypothesis.

The proposed hypothesis that the left frontal P300 would be greater than the central and parietal P300 was partially supported. There was a significant interaction effect shown for the TAS-20 factor 1. That is, the mean of the left frontal P300 was clearly greater than that of the central and parietal means for individuals high in alexithymia (Table 3), there was also a large fluctuation in individual amplitudes as seen by the standard deviations. This may suggest that instead of an overall hemispheric association with a global index of alexithymia, the recorded amplitude may be specific to the factors measured by the TAS-20 related to specific brain regions. As mentioned earlier, the P300 is an endogenous component that has been associated with cognitive processes such as decision making, attention, discrimination, stimulus relevance, and information delivery (Andreassi, 2000). These results are consistent with the previous statement as alexithymic individuals (those with high TAS-20 scores); demonstrate a

higher P300 amplitude for the left frontal site than the left central and parietal site. The findings also revalidate that the P300 is the peak of the waveform where alexithymics engage in the cognitive processing of the deviant/infrequent stimuli in an auditory-oddball paradigm.

A significant difference was observed between the left frontal P300 amplitude and the right frontal P300. Since the P300 is associated with automatic attentional processes, this finding suggests that alexithymics have increased attentional processing for affectively neutral novel auditory stimuli both relative to low alexithymics and relative to their own left frontal attentional processing. Perhaps this reflects a hemispheric attentional "bias" that reduces the efficiency of attending to the emotional qualities of novel stimuli. Further research using novel affectively laden auditory stimuli in an oddball paradigm would help clarify this as a possibility. Other findings from our lab report an enhanced right hemisphere P300 amplitude among those low in alexithymia (Berger, Starratt, & Starratt, 2004). For the current study, the opposite was found true; there was an enhanced left frontal P300 amplitude noted for individuals high in alexithymia. It is noteworthy that the Berger study used a median split to divide the sample into high and low alexithymia groups. The current study used an extreme groups method, taking the highest and lowest 10% of TAS-20 scores to define high and low alexithymia groups. This may help to explain the difference in results between the two studies. The significant difference was not only seen in the TAS-20 factor 1 scores but also the total score. The reason for this difference using the same equipment and methodology remains to be worked out.

As Taylor and Bagby (2000) reported, correlational studies have detected neural differences associated with alexithymia; however, they do not imply cause-effect relationships. These findings, if validated by follow-up investigations, can be used toward the development of a holistic psychobiological model of alexithymia, in which neural correlates may interact with environmental influences during early development such as the quality of emotional interaction and attachment with the caregiver, and/or the presence of traumatic or neglectful experiences. The results obtained were related to specific factor deficits and not a total TAS-20 score, therefore, further research is needed to clarify the neural correlates of the factors that comprise the alexithymia construct.

Developments in the field of alexithymia and neurophysiological measures continue to inform alexithymia research. The fMRI procedure has shown great consistency in detecting neural correlates and hemispheric differences with all types of stimuli. Based on these results, the ERP methodology may not be the most sensitive measure to assess factor differences. Further research on the construct of alexithymia and neurophysiological correlates will aide in understanding the hemispheric asymmetries detected. As Parker et al., (1993) stated, alexithymia is a multifaceted construct, future research may focus on the neurophysiological responses that are correlated with a particular factor of the alexithymia construct as measured by the three factor scores. Based on the present study's results, TAS-20 Factor 1 would be a noteworthy starting point as there is currently little research focusing on specific factors and neurophysiological correlates of alexithymia. Research addressing these more specific questions may further verify the cerebral constituents of emotion related to the construct

of alexithymia. This may inherently afford new insight into fundamental questions regarding brain/behavior relationships.

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# APPENDICES

Appendix A

Demographic Questionnaire

Gender: __ Male (1)

__ Female (2)

Age: _____

Please indicate whether you are right or left handed.

_____ Right handed (1) _____ Left handed (2) _____ No preference (3)

If not specified (10)

Have you ever had a serious head injury?

_____ Yes (1)

_____ No (2)

If not specified (10)

If yes, please explain:

Ethnicity

_____ American Indian or Alaska Native (1)

_____African American/ Black (2)

_____ Asian (3)

_____ Caucasian (4)

_____Hispanic (5)

_____Native Hawaiian or other Pacific Islander (6)

_____Other (7)

_____Caribbean (8)

If not specified (10)

Appendix B

## Toronto Alexithymia Scale (TAS-20)

Sex: M / F Age: Date: ID#:

**TAS-20**

Using the scale provided as a guide, indicate how much you agree or disagree with each of the following statements by circling the corresponding number. Give only one answer for each statement.

Circle 1 if you STRONGLY DISAGREE
Circle 2 if you MODERATELY DISAGREE
Circle 3 if you NEITHER DISAGREE NOR AGREE
Circle 4 if you MODERATELY AGREE
Circle 5 if you STRONGLY AGREE

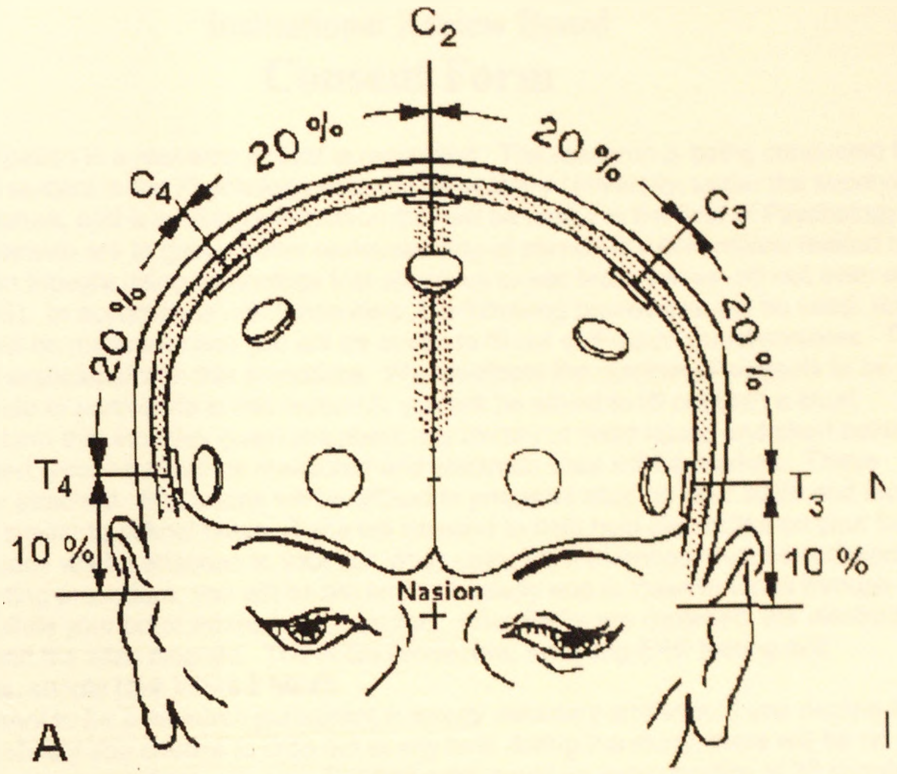
| | | Strongly Disagree | Moderately Disagree | Neither Disagree Nor Agree | Moderately Agree | Strongly Agree |
|---|---|---|---|---|---|---|
| 1. | I am often confused about what emotion I am feeling. | 1 | 2 | 3 | 4 | 5 |
| 2. | It is difficult for me to find the right words for my feelings. | 1 | 2 | 3 | 4 | 5 |
| 3. | I have physical sensations that even doctors don't understand. | 1 | 2 | 3 | 4 | 5 |
| 4. | I am able to describe my feelings easily. | 1 | 2 | 3 | 4 | 5 |
| 5. | I prefer to analyze problems rather than just describe them. | 1 | 2 | 3 | 4 | 5 |
| 6. | When I am upset, I don't know if I am sad, frightened, or angry. | 1 | 2 | 3 | 4 | 5 |

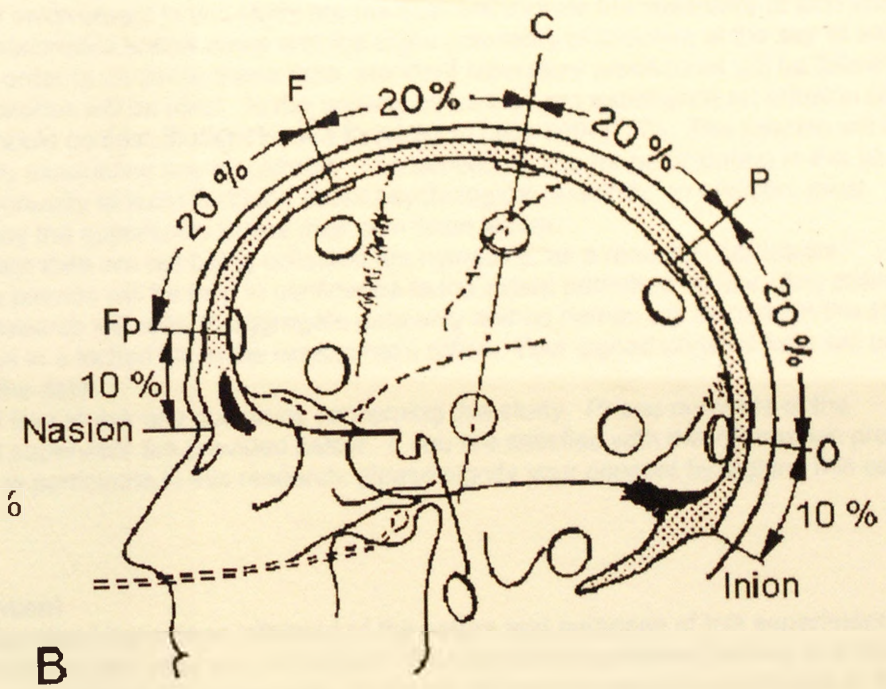
| | | |
|---|---|---|
| 7. | I am often puzzled by sensations in my body. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 8. | I prefer to just let things happen rather than to understand why they turned out that way. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 9. | I have feelings that I can't quite identify. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 10. | Being in touch with emotions is essential. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 11. | I find it hard to describe how I feel about people. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 12. | People tell me to describe my feelings more. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 13. | I don't know what's going on inside me. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 14. | I often don't know why I am angry. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 15. | I prefer talking to people about their daily activities rather than their feelings. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 16. | I prefer to watch "light" entertainment shows rather than psychological dramas. | 1_____ 2 _____ 3 _____ 4 _____5 |

| | | |
|---|---|---|
| 17. | It is difficult for me to reveal my innermost feelings, even to close friends. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 18. | I can feel close to someone, even in moments of silence. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 19. | I find examination of my feelings useful in solving personal problems. | 1_____ 2 _____ 3 _____ 4 _____5 |
| 20. | Looking for hidden meanings in movies or plays distracts from their enjoyment. | 1_____ 2 _____ 3 _____ 4 _____5 |

Appendix C

The International Ten-Twenty System



Appendix D

Informed Consent

# Barry University

Institutional Review Board

# Consent Form

Your participation in a research project is requested. The research is being conducted by Elise Drake, a student in the Psychology department at Barry University, under the supervision of Dr. Gerene Starratt, and is seeking information that will be useful in the field of Psychology. The aims of the research are to gain a better understanding of personality dimensions related to the study of human thought using technology that allows us to see brain waves without even piercing your skin (EEG). In accordance with these aims, the following procedures will be used: your brain waves will be measured and you will be asked to fill out self-report questionnaires. There is no discomfort associated with this procedure. We anticipate the number of subjects to be 60.

If you decide to participate in this research, you will be asked to fill out both a short demographic form that includes questions about any history of head injury, and short personality measures. Next, your head will be measured and electrode sites will be marked. These electrodes are attached. Electrodes will be affixed to prepared sites on your scalp and face with a white paste similar to school paste. Tape will be used to help hold electrodes on your face in place. Electrodes will be attached to your ear lobes using clips (earrings must be removed). During the testing procedure, you will be asked to sit quietly and to listen to tones through headphones while your brain waves are measured. After ERPs are recorded, the electrodes will be removed and the sites cleaned. The entire procedure, including ERP testing and questionnaires, should take about 2 hours.

Your consent to be a research participant is strictly voluntary and should you decline to participate or should you choose to drop out at any time during the study, there will be no adverse effects on your grades and you will earn 1 unit of extra credit for every portion of 30 minutes that you are here.

The risks of involvement in this study are minimal and include the possibility of skin irritation in response to electrodes and/or paste and the slight possibility of infection at the site of electrode attachment. In order to minimize these risks, standard laboratory procedures will be followed and only sterile electrodes will be used. In the unlikely event that you experience an irritation of concern, you should contact Student Health Services at (305-899-3750). The session will be terminated if you experience any discomfort. The benefits to you for participating in this study include the opportunity to learn firsthand about psychological research. In addition, most participants enjoy the opportunity to see their own brain waves.

Although your data are not being collected anonymously, as a research participant, information you provide will be held in confidence to the extent permitted by law. Any published results of the research will refer to aggregate data only and no names will be used in the study. Data will be kept in a locked file in the researcher's office. Your signed consent form will be kept separate from the data.

Please feel free to ask any questions concerning this study. Phone numbers of the researcher and supervisor are provided below. If you are satisfied with the information provided and are willing to participate in this research, please signify your consent by signing this consent form.

**Voluntary Consent**

I acknowledge that I have been informed of the nature and purposes of this experiment by _____ and that I have read and understand the information presented above, and that I have received a copy of this form for my records. I give my voluntary consent to participate in this experiment.

| | | |
|---|---|---|
| _____ | _____ | Dr. Gerene Starratt |
| *Signature of Participant* | *Date* | (305) 899-4575 or (305) 899-3270 |
| _____ | _____ | _____ |
| *Researcher* | *Date* | *Supervisor* |

Appendix E

Authorization to Link

**Barry University**
**Department of Psychology**

11300 NORTHEAST SECOND AVENUE
MIAMI SHORES, FLORIDA 33161-6695
*Direct Number* (305) 899-3270
*Main Campus* (305) 899-3000

## Authorization to Link Data

I (Please Print)_____, give my permission for the Psychology Department researchers listed below to access data related to my participation in other Psychology Department experiments in which I have participated. I understand that this data will be linked by way of the unique participant code that I have provided below and that my name will not be exchanged with the data. I also understand that confidentiality of my data will be maintained as stipulated by Barry University's Institutional Review Board.

Participant's signature: _____

Researcher's signature: _____

Date: _____

In order to establish your unique participant code, please provide the following information:

Your mother's birth month: _____

Your father's birth month: _____

Your birth date month_____ day_____ year_____

Appendix F

**Toronto Alexithymia Scale (TAS-20)**

**Score Form**

1. **General Score**
   a. Reverse score the following items (i.e. 1=5, 2=4, 3=3, 4=2, 5=1)
      4
      5
      10
      18
      19
   b. After reverse scoring, total all 20 items.

**TOTAL SCORE:** _____ ≥ 61 = Alexithymic
≤ 51 = Non Alexithymic

2. **Factor Scores**

| Factor 1: Difficulty Identifying Feelings | | Factor 2: Difficulty Describing Feelings | | Factor 3: Externally Oriented Thinking | |
|---|---|---|---|---|---|
| Item | Score | Item | Score | Item | Score |
| 1. | _____ | 2. | _____ | 5. | _____ |
| 3. | _____ | 4. | _____ | 8. | _____ |
| 6. | _____ | 11. | _____ | 10. | _____ |
| 7. | _____ | 12. | _____ | 15. | _____ |
| 9. | _____ | 17. | _____ | 16. | _____ |
| 13. | _____ | | | 18. | _____ |
| 14. | _____ | | | 19. | _____ |
| | | | | 20. | _____ |
| **Total F1** | _____ | **Total F2** | _____ | **Total F3** | _____ |