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MATURATION OF SENSORY GATING PERFORMANCE IN CHILDREN WITH AND WITHOUT SENSORY PROCESSING DISORDERS

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Abstract

Recent interest in sensory gating in children with and without neuropsychological disorders has resulted in a number of studies and the results regarding the developmental trajectory of sensory gating are inconsistent. We investigated the maturational course of sensory gating in samples of typically developing children and children with sensory processing deficits (SPD) and compared their performance to adults. Besides gating ratios, we also examined the brain responses to conditioning and test click stimuli in the sensory gating paradigm separately to clarify if the changes in click amplitudes could explain the maturational change in the T/C ratio in children. Eighteen adults with no known disorders, 25 typical children, and 28 children with SPD participated in this study. The children ranged in ages between 5 and12 years. The three groups differed in their P50 and N100 ERP components. Both child groups displayed significantly less gating than the adults. Children with SPD demonstrated significantly less gating and more within-group variability compared to typical children. There were significant relationships between age and T/C ratios and between age and peak-to-peak amplitude of the conditioning click in typical children but not in children with SPD. Typical children demonstrated significantly smaller brain response amplitudes to the clicks as compared to adults . These findings suggest that there is a maturational course of sensory gating in typical children and if there is a maturational trajectory in children with SPD it appears to be different than typical children. In addition, children with SPD were found to be lacking in their ability to filter out repeated auditory input and failed to selectively regulate their sensitivity to sensory stimuli.

Keywords

Sensory Gating; EEG/ERP; Children; Development; Sensory Processing Deficits

Sensory gating is the brain's natural response to attenuate redundant or irrelevant sensory stimuli (Adler et al., 1982; Braff, Swerdlow, & Geyer, 1995; Freedman, Adler, & Waldo, 1987). The gating response is conceptualized as the brain's capacity to selectively regulate its sensitivity to sensory stimuli. Sensory Gating is a critical underlying psychophysiological and protective mechanism of brain function which directs processing resources to important environmental stimuli (Myles-Worsley et al., 1996). A number of neurotransmitters and

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receptor systems, such as the nicotinic cholinergic, dopaminergic, gamma-aminobutyric acidergic, glutamatergic, noradrenergic, and serotonergic systems have been identified as mediating sensory gating (see Freedman, et al., 2003 and Potter, Summerfelt, Gold, & Buchanan, 2006 for reviews). The prefrontal cortex, Heschl's gyrus and surrounding areas, and hippocampus have been associated with sensory gating processing (e.g., Grunwald et al., 2003; Rosburg et al., 2004). Sensory gating has been associated with the cognitive functions of attention, processing speed, and working memory (see Potter et al., 2006 for review).

An auditory event-related potential (ERP) obtained during electroencephalogram (EEG) recordings, known as sensory gating or P50 paradigm, has been specifically utilized to measure this regulatory mechanism (e.g., Freedman, et al., 1987; Nagamoto, Adler, Waldo, & Freedman, 1989; Myles-Worsley et al., 1996; Waldo & Freedman, 1986). The traditional sensory gating paradigm uses a very short duration auditory "click" stimulus (e.g., 3 milliseconds [ms]) presented as pairs with an inter-stimulus interval (ISI) of 500 ms. Typically 40 to 80 pairs are presented with an 8 to 10 second inter-trial interval (ITI) between the pairs (e.g., Freedman et al., 1987; Myles-Worsley, et al., 1996; Nagamoto et al., 1989).

Two components, the P50 and N100, have been examined in the averaged evoked potential obtained from the sensory gating paradigm (e.g., Boutros, Belger, Campbell, D'Souza, & Krystal, 1999; Boutros, Korzyukov, Jansen, Feingold, & Bell, 2004). The P50 component is the most positive peak occurring within the time period ranging from 40–90 ms after auditory stimuli onset (e.g., Boutros et al., 1999; Boutros et al., 2004; Freedman et al., 1987; Kemner, Oranje, Verbaten, & van Engeland, 2002; Myles-Worsley et al., 1996). The N100 is the most negative peak with the peak latency of 90 to 120 ms after stimulus onset (e.g., Boutros et al., 1999; Boutros et al., 2004). Several research groups suggest that the P50 and N100 components may play different roles in the sensory gating process (Boutros, et al., 1999; Boutros et al., 2004; Kisley, Noecker, and Guinther, 2004). Specifically, the P50 may represent stimulus filtering and N100 reflecting passive attention switching (Kisley, et al., 2004). This supposition of separate functions emphasizes the importance of investigating both the P50 and N100 components.

The degree of sensory gating, or brain's inhibitory response, is often measured by a ratio obtained by dividing the peak-to-peak amplitude of the P50 (or N100) component of the second click, commonly labeled "Test" click by the peak-to-peak amplitude of the P50 (or N100) component of the first click, often labeled the "Conditioning" click. This ratio is referred to as the T/C ratio (Freedman et al., 1987). A small T/C ratio represents a large inhibitory capacity and thus, good sensory gating ability. Sensory gating is an essential brain function because the failure to inhibit an influx of irrelevant or distracting information may lead to processing inappropriate stimuli which may result in perceptual or attentional deficits (Braff & Geyer, 1990). Sensory gating has been studied in healthy individuals and in individuals with various disorders that have difficulty dealing with attention, perception, or integration of sensory stimuli such as traumatic brain injury (e.g., Arciniegas et al., 1999), migraine (e.g., Ambrosini, De Pasqua, Afra, Sandor, & Schoenen, 2001; Siniatchkin, Kropp, & Gerber, 2003), schizophrenia (e.g., Freedman et al., 2002; Adler et al., 2004), and attention-deficit/ hyperactivity disorder (ADHD; e.g., Olincy et al., 2000). Of these disorders, schizophrenia has been most widely studied and most of these studies reported a significant deficit in sensory gating in patients with schizophrenia compared to control adults (e.g., Adler et al., 1998; Clementz & Blumenfeld, 2001; Olincy et al., 2000). With relatively few studies of sensory gating abilities in other disorders, the association of sensory gating deficits to other disorders is inconclusive. However, Olincy, et al 2000 reported that sensory gating in adults with ADHD was not impaired compared to adults without ADHD. Accordingly, the study of sensory gating can be used to illuminate neurophysiology deficits underlying some disorders.

Sensory Gating in Children

Recently a growing interest has focused on sensory gating performance in children with and without disorders (Brinkman & Stauder, 2007; Davies & Gavin, 2007; Fiedler, Debus, Neubauer, Kienle, & Kurlemann, 2006; Freedman et al., 1987; Kemner et al., 2002; Marshall, Bar-Haim, & Fox, 2004; Myles-Worsley et al., 1996; Orekhova et al., 2008). One interest in studying sensory gating in children is to determine if the P50 or N100 T/C ratios might be useful in identifying and explaining the neurophysiology underlying child disorders. A first important step in determining the usefulness of these measures in childhood clinical populations is to document possible developmental trajectories in typically developing children.

In the first few documented studies investigating sensory gating in children there was some controversy whether or not sensory gating exhibited developmental trajectories (Freedman, et al., 1987 and Myles-Worsley et al., 1996). Freedman and colleagues (1987) examined sensory gating in 163 participants ranging in age from 18 months to 65 years old. Within the sample, there were a total of 108 typical children, ages 18 months to 19 years. The participants were assigned to six age groups: children 1–8 years of age, pre-adolescents 9–12 years, adolescents 13–19 years, young adults 20–29 years, adults 30–44 years, and middle-aged adults 45–65 years. They reported that the P50 T/C ratio in typical children ranged from 0 to 1.0 (the authors did not provide means in the article) and, although the younger participants varied widely in their ability to demonstrate sensory gating, the mean P50 T/C ratio did decrease during late childhood and adolescence. They also revealed age-related changes in the latency of the auditory evoked-potential P50 component. The youngest age group (1 to 8 years of age) demonstrated a rapid decrease in latency. The latency stabilized starting with the 9 – 11 age group and latency remained stable into adulthood. They concluded that the P50 T/C ratio did not reach adult levels until the end of adolescence.

Another study conducted by the same research group examined the developmental and genetic influences on the P50 sensory gating in 127 participants, ages 10–39 years (Myles-Worsley et al. 1996). Contrary to the results of the earlier study by this group (Freedman, et al., 1987), the more recent results indicated that there was not a significant age-related change in the P50 T/C ratio between children and adults. The mean P50 T/C ratio of children (.27) was similar to that of adults (.25). However, there was a significant difference in the latency of response to the conditioning click across the age groups revealing that latency decreased with age. These disparate findings may be explained by the fact that only older children and adolescents (ages 10 to 19 years) were involved in the Myles-Worsley et al. (1996) study and children/infants as young as 18 months were included in the Freedman et al. study (1987). Conceivably a large proportion of the sensory gating maturation occurs before the age of 10 years.

Several recent cross sectional studies were designed to disambiguate sensory gating across ages in typically developing children (Brinkman & Stauder, 2007; Marshall, Bar-Haim, & Fox, 2004). Brinkman and Stauder (2007) examined the maturation of P50 and sensory gating in typical children (n = 87) ages 5 to 12 years compared to adults (n=35) ages 18 to 30 years. They found a significant negative correlation between age and P50 T/C ratio with sensory gating abilities improving with age. However, analysis of variance (ANOVA) and post hoc tests revealed that while the youngest group of children (ages 5 to 7 years) were significantly different from the other three groups (8 to 9 years; 10 to 12 years; and 18 to 30 years), the latter three groups did not differ from each other. The mean sensory gating ratio at Cz for each age group was 1.14 (SD = .80) for 5 to 7 year olds; .77 (SD = .35) for 8 to 9 year olds; .72 (SD = .24) for 10 to 12 year olds; and .72 (SD = .30) for adults. Their results regarding the latency of the P50 revealed that there were no significant differences in the latency of the P50 across age groups for either the conditioning or test stimulus. Although, when they examined age effects

of latency in the youngest group of children ages 5 to 7 they found a significant negative correlation between age and latency of the conditioning stimulus. They concluded that sensory gating is mature by 8 years of age.

When Brinkman and Stauder (2007) examined the relationship of age to the amplitude of the conditioning click and the testing click, they revealed that the youngest group (5 to 7 year olds) had a significantly smaller P50 amplitude to the conditioning click as compared to the three older groups. Thus, the age related increase in sensory gating they found is likely due to the increase of amplitude to the conditioning stimulus. Interestingly, the group of children 10 to 12 years of age had a significantly larger P50 amplitude to the testing stimulus than the adult group. Age related changes in T/C ratio will be opposite in these two age groups. If the response to the conditioning click increases with age, the T/C ratio will decrease with age for 5 – 7 year olds. In contrast, as in 10 to 12 year olds, the amplitude of the test click is larger than that in the adult group, thus, the T/C ratio could be smaller in that child group than in the adult group showing an increase in T/C ratio with age. These data suggest that opposite age trends may be found depending on the age groups used in a particular study. This may help explain some of the controversy in the literature concerning the maturation effects of sensory gating ratios. Thus, carefully examining not only the T/C ratio, but in cases where the ratio differs between groups, it is imperative to determine if one or both click amplitudes contribute to the group differences to more fully understand the nature of the age differences. Notable is the fact that the T/C ratios in the Brinkman & Stuader (2007) study even for the adults is quite large (T/C ratios of over .70) indicating that the inhibition of the second stimulus was not even at the 50% level as is found in many previous studies involving adults.

Marshall and his colleagues (2004), examined the development of sensory gating in participants ages 7–13 years by comparing 3 groups of children; a group of 10 socially outgoing children, a group of 12 socially withdrawn children, and an unselected group of 10 children (i.e., those who did not meet criteria for the other 2 groups). Their results revealed that P50 gating did not differentiate the three groups. The mean P50 T/C ratio for all participants was . 90 with a standard deviation of .53. For the children who showed any P50 gating (i.e., P50 T/C ratio less than 1.0), the P50 sensory gating was better in older children than younger children and reached adult levels by 13 years of age. Marshall et al. (2004) reported that the age related increase in the P50 sensory gating was due to an age related decrease in amplitude to the test stimulus, in children ages 7 to 13 years of age.

Children with Sensory Processing Deficits (SPD)

Several studies have begun to address whether or not sensory gating is a neurophysiological marker that will distinguish between typically developing children and children with clinical neurological disorders such as centrotemporal spikes and sharp waves (CTS) which may develop into benign rolandic epilepsy (Fiedler et al., 2006) and behavioral disorders such as autism (Kemner et al., 2002; Orekhova et al., 2008). Fiedler et al. (2006) found that children ages 8 to 14 years of age with CTS showed a significant sensory gating deficit compared to age match peers of typically developing children. They state that their data may confirm the assumption of a cholinergic pathology in CTS.

Focusing more on the effects of deficits in sensory processing on behavior, Kemner et al. (2002) conducted a study comparing the sensory gating of typically developing children to children with autism, a neurological disorder that includes the symptom of having “unusual reactions to the environment such as hypersensitivity or hyposensitivity to sound” (Kemner et al., 2002, pp 214). Following the suggestion of Kootz, Marinelli and Cohen (1982) that people with autism have a problem in the filtering of sensory input and, hence, withdraw from social contact, Kemner et al. (2002) examined 12 children with autism and 11 children without autism

aged 7–13 years using the sensory gating paradigm. Kemner et al. (2002) reasoned that if evidence for P50 sensory gating anomalies was found it would suggest that impaired suppression of the second stimulus in the P50 paradigm may be associated with an increase of irreverent or distracting sensory information that may underlie the behavioral deficits (Braff & Geyer, 1990). Interestingly, they reported that the mean P50 T/C ratio for typical children was .47 (SD =.50) and for children with autism the mean was .28 (SD =.36). Children with autism appeared to be overly responsive in their gating as compared to the typically developing children. However, despite this difference in the group means, their analyses showed that differences between children with autism and the control children with regard to P50 amplitude and suppression were not statistically significant. This is consistent with the findings from Orekhova and colleagues (2008) which reported unimpaired P50 suppression in children with autism as a group when compared to control group of children.

Not all children who exhibit hypersensitivity or hyposensitivity to every day sensory stimuli also exhibit problems with social interaction and communication severe enough to be diagnosed as having autism. However, these children with milder sensory processing deficits still often display abnormal behaviors in response to sensory stimuli suggesting they experience challenges dealing with sensory information from the environment in their daily activities (McIntosh, Miller, Shyu, & Hagerman, 1999). Individuals with these characteristics have been classified as having sensory processing deficits (SPD). Such behaviors disrupt an individual's ability to achieve and maintain an optimal range of performance necessary to adapt to challenges in life (Schaaf, Miller, Seawell, & O'Keefe, 2003). The manifestations of SPD may include distraction, impulsiveness, abnormal activity level, disorganization, anxiety, and emotional lability that produce deficient social participation, insufficient self-regulation and inadequate perceived competence (Miller, Reisman, McIntosh, & Simon, 2001). A cautious estimate of the prevalence of SPD suggests that based on parents' perceptions, 5.3% of the kindergarten children may have SPD (Ahn, Miller, Milberger, & McIntosh, 2004). Despite the fact that specific therapeutic interventions have been advocated for and provided to children with SPD for over 40 years (Ayres, 1972; Bundy & Murray, 2002), studies have yet to be conducted to examine the neurophysiological basis of the aberrant behavior that these children display. The use of the sensory gating P50 paradigm and other event-related paradigms may allow us to dissect the sensory processing chain of events that leads to the behavioral characteristics of over reactivity to sensations experienced in everyday activities.

Purpose of this Study

The contradictory findings for the developmental trajectory of sensory gating in children with and without neurodevelopmental disorders motivated the design of this present study. In keeping with the recent focus related to sensory gating performance in children with various disorders, the main purpose of this study is to determine if measures of sensory gating can distinguish between a group of typically developing children (typical) and a group of children with known sensory processing deficits (SPD). Specifically, we predict that children with SPD will show less sensory gating ability as measured by the P50 and N100 T/C ratios compared to typically developing children. In addition, this study also focuses on the maturational course of sensory gating in a cross sectional sample of children with and without SPD over the age range of 5–12 years compared to a group of adults. We predict that sensory gating will be associated with age in the typically developing group of children, such that older children show a more enhanced sensory gating (smaller T/C ratios) than younger children. However, due to the behavioral manifestations in response to everyday sensory exposure in children with SPD across childhood, we predict that sensory gating ability will not improve in the group of children with SPD.

Method

Participants

Three groups of participants were recruited for this study. An adult control group consisted of healthy 18 adult volunteers with equal numbers of males and females between 20 and 55 years of age ($M = 33.28$; $SD = 11.35$). The second group consisted of twenty-five typically developing children (typical; 13 male and 12 female) between 5 and 10 years of age ($M = 8.33$; $SD = 1.88$) recruited from the local community through schools, youth organizations or from families who participated in past research projects in this lab. Adult participants and typical children in the study were screened to ensure that they were free of neurological injuries, disabilities, and family histories of psychological disorders through self-reports for the adult participants and by parent reports for the children.

The third group consisted of twenty-eight children with SPD (22 male and 6 female) between 5 and 12 years of age ($M = 7.70$; SD = 1.80) who were referred to the study by local practitioners in the medical community. The two child groups did not significantly differ in age, $t(51) = 1.25$, $p = .22$. The large number of males in this group was consistent with sensory processing deficits being more prominent in males than females (Ahn et al., 2004). However, because (Brinkman & Stauder, 2007) reported that they did not find a gender difference in sensory gating performance, the disparity in gender was deemed not likely to affect the results of this study.

To validate differences between the sensory processing abilities of the typical children and those of the children diagnosed as having SPD, we used a behavioral measure known as the Sensory Profile (Dunn, 1999). This parental report measures how his or her child processes sensory information in everyday situations and profiles the sensory system's effect on functional performance. A multivariate analysis of variance (MANOVA) revealed that typical children had significant higher scores than SPD children (Wilks' lambda = .24, $F(22,30) = 4.22$, $p < .0005$), suggesting that SPD children displayed significant difficulties processing sensory information. Table 1 reports the F values and the associated probability and effect sizes for each of the subscales of the Sensory Profile that served as the dependent variables in this MANOVA. These results indicated that the two child groups did differ on sensory processing characteristics such as auditory, vestibular, and multisensory processing, which provided face validity that children in the group referred by the medical community were more abnormal in their responses to stimuli in everyday activities compared to the children in the typical group.

Besides the primary diagnosis by a medical specialist that these children had a sensory processing disorder, parental reports revealed that five children were also diagnosed as having attention-deficits/hyperactivity disorder (ADHD), two children had learning difficulties or disabilities (LD), five children had delayed speech, seven children had combined ADHD and LD, and one child had combined LD and delayed speech. None of the children in this group had been diagnosed as having schizophrenia, bipolar disorder, or autism nor did they have a family history of these disorders. There were no significant differences in P50 and N100 T/C ratios between children with SPD only and children with SPD and other comorbid disorders. Only six of the children with SPD were on various medications; four for ADHD (2 Adderall XL, 1 Stratera, 1 specific medication not listed), one for ADHD and obsessive-compulsive disorder (OCD) (Stratera and Zoloft), and one for depression (2.5 mg of Abilify). To determine whether to include or exclude children with SPD on medications, we compared the P50 and N100 T/C ratios of children with SPD not on medications to those on medications. There was no significant difference in P50 T/C ratios between children with SPD not taking medications (mean T/C ratio = .75; SD = .44) and children with SPD taking medications (mean T/C ratio = .83; SD = .37), $t = .383$, p = .705. For the N100 T/C ratio there was a significant difference between the children with SPD on medications (mean N100 T/C ratio = .48; SD = .22)

displaying more sensory gating than those children with SPD not on medications (mean N100 T/C ratio = 1.14; SD = .95), t = 2.826, p = .010 (this test was adjusted for unequal variances). In addition, there were no significant differences on any of the subscales or total score on the sensory profile for children with SPD taking medications as compared to those not on medications. Based on these analyses it was decided the most conservative approach was to include all children with SPD in further analyses because there was no significant difference between on the P50 T/C ratio and sensory profile based on comorbidities or medications and the difference on the N100 T/C ratio showed that those on medications displayed more sensory gating than those not on medications.

Informed consent was obtained from the adult participants. For the child participants, parent permission and child assent were obtained. All procedures used in this study were approved by the human research committee of the local university.

Procedure

The participants were tested in a relaxed sitting position in high back chair in an attempt to support the head to decrease tightening of neck muscles that may cause EEG artifacts. Prior to the EEG recording, they were provided with a short training period on how to reduce artifacts caused by eye blinks and muscle activity.

All adult participants and child participants (via parent) reported no hearing deficits. Auditory threshold screening was conducted on all participants by binaurally presenting single clicks of 3 ms duration varying in intensity as determined by a simple up-down procedure (Levitt, 1971). During this procedure the participant was asked to press a mouse button each time he or she heard the click sound. Upon a response within one second of the click presentation, the intensity for the next presentation was decreased by one step. If no response occurred, then the intensity was increased by two steps. For the first four downward sweeps (i.e., successive responses) the step size was 5 db and thereafter the step size was 2.5 db. Procedure ended after 13 downward sweeps were completed. The threshold was computed by averaging the lowest intensity levels obtained during the last nine sweeps. Among the participants, only one child with SPD was unable to perform the threshold testing. This child either did not understand the directions given for the task or how to respond appropriately during the task. We included this participant as the parent reported that the child had hearing within normal limits and subsequent testing revealed that this child did have hearing levels within the normal range. This child, who was a member of the SPD group, had ERP measures more in line with the typical children, thus, we felt that to remove the child from the analysis would inappropriately bias the outcome. Mean thresholds for adult participants was 27 dB SPL (range = 26 to 29.57 dB SPL, *SD* = 0.87). The mean threshold in typical children was 29.4 dB SPL (range = 25.39 to 39.67 dB SPL, *SD* = 4.14) and mean threshold in children with SPD was 29.86 dB SPL (range = 23.76 to 38.85 dB SPL, *SD* = 3.81). After the threshold testing and prior to the recording of sensory gating paradigm, one pair of clicks was presented to each participant to verify that the click with the intensity of 85 dB SPL would not produce a startle reaction. None of the participants displayed a startle reaction any time during the recordings.

Electrophysiological Paradigm

The sensory gating paradigm was presented using the E-Prime software (Psychological Software Tools, Pittsburgh, PA) running on a laptop computer. The click sounds, 3 ms in duration, were presented as pairs with 500 ms stimulus onset asynchrony (SOA). The pairs of click stimuli were presented continuously with an inter-trial interval (ITI) of 10 seconds to each participant binaurally through the ER-3A insert earphones (Etymotic Research) while he or she sat quietly and watched a silent Wallace and Gromit movie. The EEG activity of the participant was continuously recorded until a total of 120 pairs of clicks were presented.

Electrophysiological Recording Methods and Apparatus

EEG activity was recorded using the portable BioSemi ActiveTwo EEG system (BioSemi Inc., Amsterdam, Netherlands) with 32 pin-type Ag-AgCI sintered Active-electrodes inserted into a lycra head cap with locations based upon the American Electroencephalographic Society nomenclature guidelines (1994). EEG was recorded with the Common Mode Sense (CMS) active electrode and Driven Right Leg (DRL) passive electrode as the reference and ground respectively (http://www.biosemi.com/faq/cms&drl.htm). Electrooculograms (EOG) were recorded from individual electrodes placed on the left and right outer canthus for horizontal movements and on the left supraorbital and infraorbital region for vertical movements. Four additional electrodes were placed on the left and right ear lobes and left and right mastoids. EEG signals were sampled at an analog-to-digital conversion rate of 1024 Hz with a bandwidth of 268 Hz.

ERP Waveform and Component Analysis

All offline EEG analyses were conducted using the Brain Vision Analyzer software (Brain Products GmbH, München, Germany). The left ear lobe was used as the offline reference. The four individual EOG channels were converted to a vertical and a horizontal bipolar EOG. All ERP waveforms were segmented time locked to the stimulus, averaged, and scored relative to 100 ms baseline prior to the stimulus. Although all 32 sites were recorded and included in the data reduction, we only used Cz site in the analyses.

P50 component analysis—Based on procedures similar to those outlined in Freedman, et al. (1987), peak-to-peak measures for the P50 component were obtained in the following manner. The continuous EEG was filtered offline with a band pass setting of 10 to 200 Hz (24 dB/octave) and then segmented into epochs representing either the conditioning or test click with a duration of 100 ms pre-stimulus onset to 200 ms post-stimulus onset. Segments with deviations greater than ± 100 μV on any of the EEG channels or the bipolar EOG channels were eliminated. The non-rejected segments were baseline corrected and then averaged to create ERP waveforms for both the conditioning and test clicks in order to measure the P50 component for each participant. The mean number of non-rejected segments for adults were 119.17 (*SD* = 0.86) for the conditioning click and 119.22 (*SD* = 0.73) for the test click. The mean number of non-rejected segments for the typical children were 115.96 (*SD* = 4.39) for the conditioning click and 116.12 (*SD* = 4.34) for the test click. For the children with SPD the mean number of non-rejected segments for the conditioning click were 109.32 (*SD* = 17.65) and 110.21 (*SD* = 17.48) for the test click. None of participants were excluded for the P50 component analyses.

P50 amplitude and latency measurements were obtained from each averaged ERP waveform using computer software known as ERPScore (Segalowitz, 1999) which is a computer scoring program that provides peak amplitude and latency values within a set window automatically and allows for the visual inspection of the average waveform. Waveforms for all participants were scored by one of the authors (W-PC). To validate the accuracy of these measures, an inter-rater reliability measure was determined by having a second individual who was blinded to group membership independently scored 60% of the data. Pearson product moment correlations between the two individuals' amplitude measures of the P50 component revealed $r(38) = .98$ for the conditioning click (click 1) and $r(38) = .88$ for test click (click 2). The peak-to-peak amplitude of the P50 component for adult participants was defined as the difference in μV between the peak of the P50 component and its preceding negative trough. These components were scored in the following manner. The P50 peak was identified as the most positive peak between 40 to 70 ms after the stimulus onset. In order to identify the maximum negative trough preceding the P50 peak, the P30 was scored as the most positive peak between 25 to 40 ms after the stimulus and then the maximum negative point between the P30 and P50

peaks was marked as the negative trough preceding the P50. The selection of the P50 peak for the test click in adult participants was determined the same way as the P50 to the conditioning click, with the restriction that the peak latency must be within ± 10 ms of the of the latency of conditioning click (Oranje et al., 1999).

For child participants, the peaks were identified like the adults with a few exceptions. The P50 peak was identified as the most positive peak between 40 to 80 ms after the stimulus onset. The longer latency period for children was used based on previous reports of P50 components showing longer latencies compared to adults (e.g., Freedman et al., 1987). For child participants, the test click P50 Peak was identified as described for the P50 of the conditioning click and needed to be within ± 20 ms of the latency of conditioning click. This modification from the scoring procedure used for adults was based on visual inspection of the ERPs that revealed, especially for the younger children (i.e., 5 and 6 years of age) and children with SPD, that the latency variability between the conditioning click of the P50 and the test click of the P50 could be greater than the 10 ms window used for adults. Thus, for all children we used a ± 20 ms window. The P50 T/C ratio for each participant was calculated by dividing the peak-to-peak amplitude of the P50 component of the test click (T) by the peak-to-peak amplitude of the P50 component of the conditioning click (C).

N100 component analysis—Peak-to-peak measures for the N100 component were obtained in the following manner. The continuous EEG was filtered offline with a band pass setting of .23 to 30 Hz (12 dB/octave) and then segmented into ERPs representing either conditioning or test clicks with a duration of 100 ms pre-stimulus onset to 800 ms post-stimulus onset. Artifact rejection criteria and averaging techniques were the same procedures used for the P50 component. The data were bandpass filtered with a different setting for the N100 because the frequencies of interest here are much lower as compared to those of the P50 which is about 40Hz (Clementz, Blumenfeld, & Cobb, 1997). Using a different bandpass filter setting for measuring the P50 and later components such as the N100 is a common practice with investigators examining the sensory gating phenomenon (e.g., Anokhin, Vedeniapin, Heath, Korzyukov, Boutros, 2007; Kisley, et al., 2004). However, since a different band pass filter setting was used to measure the N100 component, eye blink artifacts were no longer filtered out and a larger number of segments were rejected in all groups but particularly in the child groups. The mean number of non-rejected segments for adults were 101.61 (*SD* = 17.85) for the conditioning click and 102.22 (*SD* = 18.70) for the test click. The mean number of non-rejected segments for the typical children were 50.36 (*SD* = 18.03) for the conditioning click and 57.77 (*SD* = 17.83) for the test click. For the children with SPD the mean number of non-rejected segments for the conditioning click were 43.54 (*SD* = 18.44) and 46.81 (*SD* = 19.18) for the test click. To correct for this imbalance in the number of segments and to ensure that the ERP components to be scored were free from biases due to a difference in the number of segments used to generate the averaged ERP waveform, for each adult 50 non-rejected segments were randomly selected, baseline corrected and then averaged to create the averaged ERP. For the child participants, 3 typical and 2 with SPD were excluded for the N100 component analyses due to an insufficient number (less than 20) of non-rejected segments. One child with SPD had a N100 T/C ratio over 5 *SD*s above the group mean and was also excluded for the N100 component analyses.

N100 amplitude and latency measurements were obtained from each averaged ERP waveform by also using the ERPScore software (Segalowitz, 1999). Waveforms for all participants were also scored by one of the authors (W-PC) and validated by a second individual in same manner as the P50 measures. Pearson product moment correlations between the two individuals for the N100 component revealed an $r(38) = .995$ for the conditioning click (click 1) and $r(38) = .93$ for test click (click 2). The N100 component in adult participants was identified as the most negative peak between 70 to 150 ms after the stimulus onset while in child participants the

N100 was defined as the most negative deflection between 70 to 180 ms. The maximum positive peak preceding the N100 in adult participants was scored between 20 to 60 ms while in child participants was 40 to 100 ms. The peak-to-peak amplitude of the N100 component was defined as the difference in μV between the N100 peak and the maximum positive peak preceding the N100 (Kisley, et al., 2003). Peak-to-peak measures of the N100 were used in recent studies investigating sensory gating and the window used in this present study was based on these previous studies (Boutros, et al., 2004; Kisley, et al., 2003). Other investigators have used N100 peak relative to baseline (Anokhin, et al., 2007; Kisley, et al. 2004). However, due to previous reports of the N100 being above baseline in young children (Ponton, Eggermont, Kwong, & Don, 2000), the peak-to-peak method was used for this study. The selection of the N100 peak for the test click in adult participants was determined within ± 25 ms of the latency of conditioning click whereas in children the test click was within ± 40 ms of the latency of conditioning click. The test/conditioning (T/C) ratio was computed to quantify gating abilities. This continuous variable is interpreted as robust gating when the T/C ratio approaches 0, and as the T/C ratio approaches 1.0 there is less gating (Boutros & Belger, 1999; Cromwell, Mears, Wan, & Boutros, 2008).

Statistical Analysis

In keeping with past studies, the statistical analyses for this study were performed only on measures obtained for the central (Cz) midline site. Differences between adult participants, typical children, and children with SPD in the P50 and N100 T/C ratios were each evaluated using a one-way analysis of variance (ANOVA) and *a priori* tests, specifically, pair-wise orthogonal contrasts (Kirk, 1995, pp. 127–131). Prior to conducting all ANOVAs the assumptions of normalcy and homogeneity of variance were examined. Levene's test was used to evaluate homogeneity of variance and if the test was significant, the appropriate adjustment of the degrees of freedom was applied. Differences between groups for peak-to-peak amplitudes of the P50 component and the N100 component were each evaluated using a two-factor 3 × 2 ANOVA with Group (3 levels: adults, typical children, and SPD children) as a between subject factor and Click (2 levels: conditioning and test clicks) as a within measure. ANOVAs with the same design were conducted to evaluate differences in the latencies of the two components. *A priori* tests and *a posteriori* tests (Tukey's HSD) were used to compare cell means (Kirk, 1995) for the peak-to-peak and latency measures. Pearson Product-Moment Correlation procedures were used to examine if the age relates to the P50 and N100 gating measures (i.e., T/C ratio, amplitude and latency) in typical children and children with SPD respectively. All statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) for Windows software, 14.0 version.

Results

The mean T/C ratio, peak-to-peak amplitude, and peak latency of both P50 and N100 components for each group, along with standard deviations are shown in Table 2. In general, children demonstrated less gating in both P50 and N100 components and displayed more within-group variability when compared to adults. Typical children displayed enhanced P50 gating and less within-group variance as compared to children with SPD. However, the Levene's test for homogeneity of variance was not significant (p = .06). Furthermore, the distributions of scores for the two normal groups were normally distributed and the distribution of children with SPD was only slightly skewed towards higher T/C ratios.

Comparisons of Sensory Gating Performance in Adults and Children – P50 Measures

P50 T/C Ratio—The results of a one-way ANOVA indicated that for the P50 T/C ratio there were statistically significant differences between the three groups, $F(2,68) = 5.12$, $p = .008$ (see Figure 1). We conducted two *a priori* orthogonal comparison tests, the adults compared

to the average of the 2 child groups and the second test compared the mean of the typical child group to the mean of the group of children with SPD. The *a priori* tests revealed that adults had a significantly smaller mean P50 T/C ratio than the average of both child groups, $t(68) = -2.44$, $p = .017$, $d = .67$. Typical children also demonstrated a statistically significant smaller mean P50 T/C ratio than children with SPD, $t(68) = -2.01$, $p < .049$, $d = .49$.

P50 Peak-to-Peak Amplitude Measures—The two-factor ANOVA revealed a statistically significant main effect for Click, $F(1,68) = 114.31$, $p < .001$, but not a significant main effect for Group, $F(2,68) = .81$, $p > .05$, *ns*. The *a priori* tests revealed that the mean peak-to-peak amplitude of the conditioning click of the P50 component was significantly different from that of the test click of the P50 component for each group (adults – $t(68) = 7.55$, $p < .0005$, $d = 1.40$; typical children – $t(68) = 5.95$, $p < .0005$, $d = 1.20$; children with SPD – $t(68) = 4.78$, $p < .0005$, $d = .96$). The ANOVA also revealed a significant Group × Click interaction, $F(2,68) = 4.23$; $p = .019$, but none of the *post hoc t*-tests were significant.

P50 Latency Measures—The two-factor ANOVA with the Greenhouse-Geisser correction revealed a significant main effect for Click, $F(1,68) = 8.74$, $p = .004$, but no significant main effect for Group, $F(2,68) = .15$, $p > .05$, *ns*. The *a priori* tests between two clicks for each group revealed that the peak latency of the conditioning click of the P50 component was significantly different from that of the test click of the P50 component for both children groups (typical children – $t(68) = 2.27$, $p < .025$; SPD children – $t(68) = 2.23$, $p < .025$), but not for the adult group $t(68) = .84$, $p > .05$, *ns*. The Group × Click interaction was not significant $F(2,68) = .398$; $p > .05$, *ns*.

Comparisons of Sensory Gating Performance in Adults and Children – N100 Measures

N100 T/C Ratio—As mention above, six of the children's data were not included in the analyses for N100 due to an insufficient number of trials after eye artifact rejection. Thus, the analyses for N100 include 18 adults, 22 typical children, and 25 SPD children; a total of 65 participants. The results of a one-way ANOVA indicated that for the N100 T/C ratio there were also statistically significant differences between the three groups, $F(2,62) = 3.76$, $p = .029$ (see Figure 2). We conducted two *a priori* orthogonal comparison tests, the adults compared to the average of the 2 child groups and the second test compared the typical child group to the group of children with SPD. These 2 comparison tests were adjusted for significant Levene's test. The *a priori* tests revealed that adults had a statistically significant smaller mean N100 T/C ratio than compared to the average of the two child groups, $t(58.46) = -3.87$, $p < .0005$, $d = .72$. The N100 T/C ratio was not significantly different between the two child groups, $t(44.7) = -.80$, $p = .428$, $d = .23$.

N100 Peak-to-Peak Amplitude Measures—The two-factor ANOVA revealed that there was not a statistically significant main effect for Group, $F(2,62) = 3.06$, $p = .054$, *ns*, but a statistically significant main effect for Click, $F(1,62) = 66.81$, $p < .0005$ was found. The planned comparisons using the *a priori* tests revealed that the mean peak-to-peak amplitude of the conditioning click of the N100 component was significantly different from that of the test click of the N100 component for each group (adults – $t(62) = 7.31$, $p < .0005$, $d = 2.04$; typical children – $t(62) = 3.31$, $p < .005$, $d = .92$; children with SPD – $t(62) = 3.21$, $p < .005$, $d = .73$).

The ANOVA also revealed a statistically significant Group × Click interaction, $F(2,62) = 7.20$; $p = .002$). The *post hoc t*-tests only found that the mean peak-to-peak amplitude of the conditioning click of the N100 component in adults was significantly different from that in typical children, $t(62) = 3.78$, $p < .001$, $d = 1.01$ and from children with SPD, $t(62) = 3.71$, p

< .001, d = .88. Typical children did not demonstrate any significant differences as compared to children with SPD for either click.

N100 Latency Measures—The two-factor ANOVA with the Greenhouse-Geisser correction revealed that there was not a significant main effect for Group, F (2,62) = 2.73, p = .073, *ns*, but a significant main effect for Click, F(1,62) = 14.88, p < .0005 was found. The *a priori* tests revealed that the mean peak latency of the conditioning click of the N100 component was significantly different from that of the test click of the N100 component for adults, t(62) = 4.99, p < .0005, d = 1.51, and SPD children, t(62) = 1.93, p < .05, d = .21, but not for typical children, t(62) = .53, p > .05, *ns*.

The ANOVA also revealed a significant Group × Click interaction, F (2,62) = 8.29; p = .001. The *post hoc* t-tests only found that the mean peak latency of the test click of the N100 component in adults was significantly different from that in typical children, t(62) = 3.22, p < .01, d = .92.

Maturation of Sensory Gating Performance in Children with and without SPD

The Relationship of Conditioning and Test Click Amplitudes and P50 T/C Ratio with Age—For all child participants, results of Pearson correlation analyses revealed that the age was significantly correlated with the P50 T/C ratio, r(51) = −.32, p = .020 and the peak-to-peak amplitude of the conditioning click, r(51) = .28, p = .041, but not the peak-to-peak amplitude of test click, r(51) = −.05, p = .709. However, when the two child groups were examined separately, the correlation coefficients of these variables revealed a significant relationship only for the typical children and not for the children with SPD (see Figure 3). Results of Pearson correlation analyses for the group of typical children revealed that the age was significantly correlated with the P50 T/C ratio, r(23) = −.60, p = .001, and the peak-to-peak amplitude of the conditioning click, r(23) = .46, p = .022, but not for the test click, r(23) = −.11, p = .588. No statistically significant age related correlations were found for children with SPD: P50 T/C ratio, r(26) = −.08, p = .672; conditioning click, r(26) = .11, p = .572; test click, r(26) = .07, p = .733.

The Relationship of Conditioning and Test Click Amplitudes and N100 T/C Ratio with Age—For all child participants, results of Pearson correlation analyses revealed that the age was not significantly correlated with the N100 T/C ratio, r(45) = −.24, p = .102, the peak-to-peak amplitude of the conditioning click, r(45) = .13, p = .390, or the peak-to-peak amplitude of test click, r(45) = .06, p = .714. However, when the groups were examined separately, a relationship with age was found for the typical children but not for the children with SPD (see Figure 4). For typical children, results revealed that the age was significantly correlated with the N100 T/C ratio, r(20) = −.55, p = .008, and the peak-to-peak amplitude of the conditioning click, r(20) = .43, p = .045, but not for the test click, r(20) = .08, p = .741. Children with SPD did not display any significant age related results: P50 T/C ratio, r(23) = .004, p = .985; conditioning click, r(23) = −.09, p = .670; test click, r(23) = .07, p = .756.

The Relationship of P50 with N100 T/C Ratio Controlling for Age—Two-tailed Pearson Product-Moment correlations revealed that for typical children, there was no significant relationship between their P50 and their N100 T/C ratios, r(20) = .08, p = .714 (see Figure 5); a finding similar to the adult group, r(16) = .27, p = .278. However, a partial Pearson correlation conducted for typical children with age removed revealed that there was a relationship between the P50 and N100 T/C ratios that approached but was not significant, r (19) = −.40, p = .073. For SPD children, a Pearson correlation revealed that there was a significant relationship between the P50 and N100 T/C ratios, r(23) = .51, p = .010. The partial

Pearson correlation revealed that this significant relationship between the P50 and N100 T/C ratios, $r(22) = .51$, $p = .011$, continued to exist after age was removed.

Discussion

The present study investigated sensory gating in children with and without Sensory Processing Disorder (SPD) as compared to normal adults. The results indicated that the three groups did differ in sensory gating abilities as measured by both P50 and N100 ERP components and their corresponding T/C ratios. Adults without disabilities displayed significantly enhanced sensory gating as compared to either child group. Children with SPD demonstrated the least amount of sensory gating and more within-group variability, in comparison to typical children. These results suggest that children with SPD are deficient in their ability to filter out repeated auditory input and fail to selectively regulate their sensitivity to sensory stimuli. The children with SPD included in this study were heterogeneous, in that some of the children were on medications and some of the children had comorbid diagnoses. Thus, the results of this study could be impacted by the heterogeneity of the individuals in the SPD group and the results should be cautiously interpreted.

To further understand the differences in sensory gating among the three groups included in this study, we examined the individuals' averaged brain responses to each auditory stimulus in the click pair (i.e., the conditioning and test clicks). Since sensory gating is measured as a ratio, individual differences in either the denominator of the ratio (the amplitude of conditioning click), or numerator of the ratio (the amplitude of test click), or both can result in an outcome where one group has a larger mean T/C ratio than the other group. If the mean amplitude of the conditioning click is smaller for individuals in one group than that of the other group while having similar amplitudes for the test click, the resulting larger group's mean T/C ratio is not due to the lack of suppression but due to a smaller response to the initial stimulus. If the mean amplitude of the test click is larger for individuals in one group than individuals in the other group while the amplitude of the conditional click is similar for individuals in both groups, the resulting larger group mean T/C ratio does represent a lack of suppression of brain response to the test stimulus, hence showing a difference in gating abilities. Thus, by examining the amplitudes of brain responses to the each auditory stimulus in the click pair a more informative interpretation of the differences of mean T/C ratios between groups can be achieved.

In the present study the peak-to-peak analyses of both the P50 and the N100 components revealed significant Group × Click interactions though none of the post hoc *t*-tests were significant. These results suggest that since neither the amplitude of the first stimulus nor the second stimulus was significantly different between the child groups, changes in both stimuli may have contributed to the larger mean T/C ratio in the group of children with SPD. However, when examining the mean amplitude for the two child groups, the absolute difference in the mean amplitude for the P50 component of the first stimulus, the conditioning click, was found to be negligible (.08 μV). In contrast, for the second stimulus, the test click, the SPD group had a larger mean amplitude by .49 μV when compared to the mean amplitude of the typical children (see Table 2). Thus, compared to the amplitude difference between the typical and SPD groups for the first stimulus, there was a considerably greater absolute difference in the amplitude to the second stimulus (60%). This suggests that the larger amplitude of the second click greatly contributed to the larger T/C ratio in the children with SPD. It is important that these findings be replicated, as the results in this present study were not statistically conclusive. Pursuing this line of analyses could lead to information about the underlying neural mechanisms in sensory processing deficits and could assist in differentiating sensory processing deficits across a variety of diagnoses.

The second purpose of this study was to examine the maturation of sensory gating in children with and without sensory processing disorders. The significant post hoc tests between the typically developing children in this study and adults reveal that the children ages 5 to 12 years of age do not exhibit mature sensory gating brain mechanisms. Typical children did show a significant relationship between sensory gating and age, a variable representing the accumulative time for maturation of brain processes, suggesting that typical children as a group improve their gating abilities as they mature. Thus, our results suggest that maturation is one of the factors accounting for the variability in the T/C ratio measures in the typical children. These findings are consistent with Freedman et al. conclusions that maturation results in the decrease of both mean T/C ratio and its variance (i.e., sensory gating increases with age and becomes more stable in adults; Freedman et al., 1987). Marshall and colleagues (2004) demonstrated a maturational effect of the T/C ratio and also a large standard deviation in children but did not have an adult control group in which to compare the results. In our analyses, age was not significantly related to the sensory gating ability in children with SPD. This result suggests that children with SPD as a group do not improve their ability to gate out repeated or irrelevant sensory information as a function of the biological-driven maturity (e.g., physical growth), at least in the same trajectory as typically developing children. Studies that include more children under the age of 10 are needed to more fully understand the developmental trajectory in clinical groups and in typically developing children.

Conversely, this maturational trend found in sensory gating is at odds with the study done by Myles-Worsley et al. (1996), which found no significant differences in sensory gating between normal adults and typical children. The inability to detect a developmental trend for sensory gating in the Myles-Worsley et al. study, which included children ages 10 and older, may be related to the lack of younger children in their study. Sensory gating may be mostly mature by the age of 10 years and sensory gating ability may not change appreciably from age 10 years to adulthood. Based on speculation that the frontal cortex may be involved in P50 suppression (Knight, Staines, Swick, & Chao, 1999; Weisser, et al., 2001), Marshall et al. (2004) acknowledge that the maturation of sensory gating may be related to the developmental changes in executive and attentional capacities which are prefrontal cortical functions. Brinkman and Stauder (2007) concluded that sensory gating is mature by 8 years of age. Additional studies are needed to understand the trajectory of sensory gating in typically developing children and the relationship of the maturation of sensory gating to prefrontal cortical functions.

Investigation of these functions may also help explain the differential developmental changes in the amplitudes in the P50 component of the conditioning and test stimuli. We found that there was an age-related increase in the peak-to-peak amplitude of the conditioning click in typical children, but there was not a relationship between age and amplitude of the test click. These findings indicate that the failure of younger typical children to demonstrate as robust sensory gating as adults is due to children exhibiting a smaller brain response to the conditioning stimulus, rather than having a larger response to the test click. Thus, the maturational increase in sensory gating abilities may be in an increased response to the initial stimulus and not at all related to a change in the suppression to the second stimulus, consistent with the findings of the younger children in the Brinkman & Stauder (2007) study. This is in contrast to the findings in the Marshall et al. (2004) study where they found a significant age-related decrease in the peak-to-peak amplitude of the second click, but not to the conditioning click. The older children in the Brinkman and Stauder (2007) study also showed a significant age-related decrease to the second stimulus. Further studies are needed to determine if the maturational changes in sensory gating in children are due to the increase in the amplitude of the conditioning click or due to the decrease in the amplitude of the test click or whether the relationship may change depending on the age of the child. Determining this factor may be important especially when comparing typical children to children with disorders.

The finding in this present study that the P50 and N100 T/C ratios did not correlate in the adult group (consistent with the findings of Kisley, et al., 2004) and the typical child group provide evidence to corroborate the premise that the P50 and N100 represent distinct aspects of sensory processing introduced by Boutros and colleagues (1999) and further supported by more recent studies (Boutros, et al., 2004; Kisley, et al., 2004). One explanation for the lack of relationship between the N100 and P50 T/C ratios could be that the N100 may represent more complex processing than the P50 (Näätänen & Picton, 1987). Another interesting finding in this study, is that for children with SPD who were on medications for ADHD and depression displayed significantly more sensory gating for the N100 as compared to children with SPD who were not on medications. In contrast, there were no differences in these subgroups of children with SPD for the P50 T/C ratio. This may suggest that attention may be more involved in the N100 than in the P50 T/C ratio. An unexpected finding in this present study was that for the typical children, when age was controlled for, the correlation between the P50 and N100 T/C ratios approached significance. More puzzling was the finding that a significant relationship existed between the P50 and N100 T/C ratios for the children with SPD, whether or not the variance for age was statically removed. Further studies are needed to clarify the meaning of these results, but they could suggest that the dissociation of P50 and N100 sensory processing occurs with maturation. Furthermore, clinical populations that exhibit difficulty in dealing with everyday sensory experiences early in life may fail to develop the complex sensory processing that is observed in healthy adults. The neural substrates for the sensory processing deficits in children with SPD could be further illuminated by examining hemispheric specific responses through dipole analyses (Hanlon, et al., 2005). Dipole analyses could not be reliably performed on these present data as only 32 electrode site were recorded.

In conclusion, our results demonstrate that normal adults, typical children, and children with SPD show significant differences in sensory gating T/C ratios. Children with SPD show significantly less sensory gating abilities than typical children. This difference in gating abilities appears to be most related to a larger response to the test click, indicating that children with sensory processing disorders have difficulty in suppressing repeated sensory input. In addition, our results indicate that in typical children there is an increase in gating abilities with increasing age, but there is not a developmental change in sensory gating in children with sensory processing disorders. This difference in maturational affects may be due to the increase in the magnitude of the response to the conditioning click in typical children. The P50 and N100 T/C ratios were not related in adults and in typical children, though, when age was accounted for in the children, the relationship between the P50 and N100 strengthened appreciatively but not enough to reach statistical significance. In contrast, the P50 and N100 T/C ratios were significantly related in the children with SPD. These findings demonstrate that the neurophysiological mechanisms to process auditory information are significantly different between children and adults and between typical children and children with sensory processing disorders (SPD) and may help clarify the underlying neural mechanisms to the behavioral manifestations observed in children with SPD.

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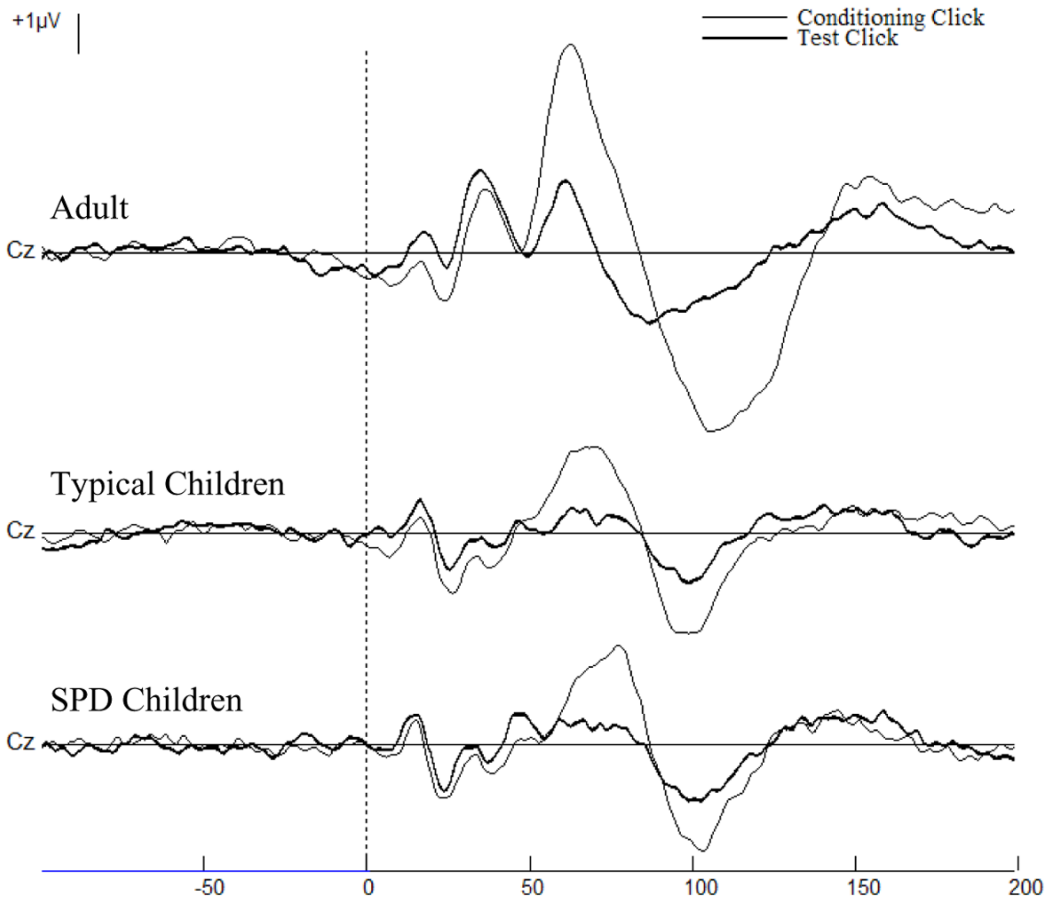

Figure 1.
Grand averages of sensory gating ERP at Cz with 100 ms baseline prior to the stimulus filtered with a bandpass of 10 Hz to 200 Hz for the P50. The thin line represents that conditioning click (first click) and the thick line represents the test click (second click).

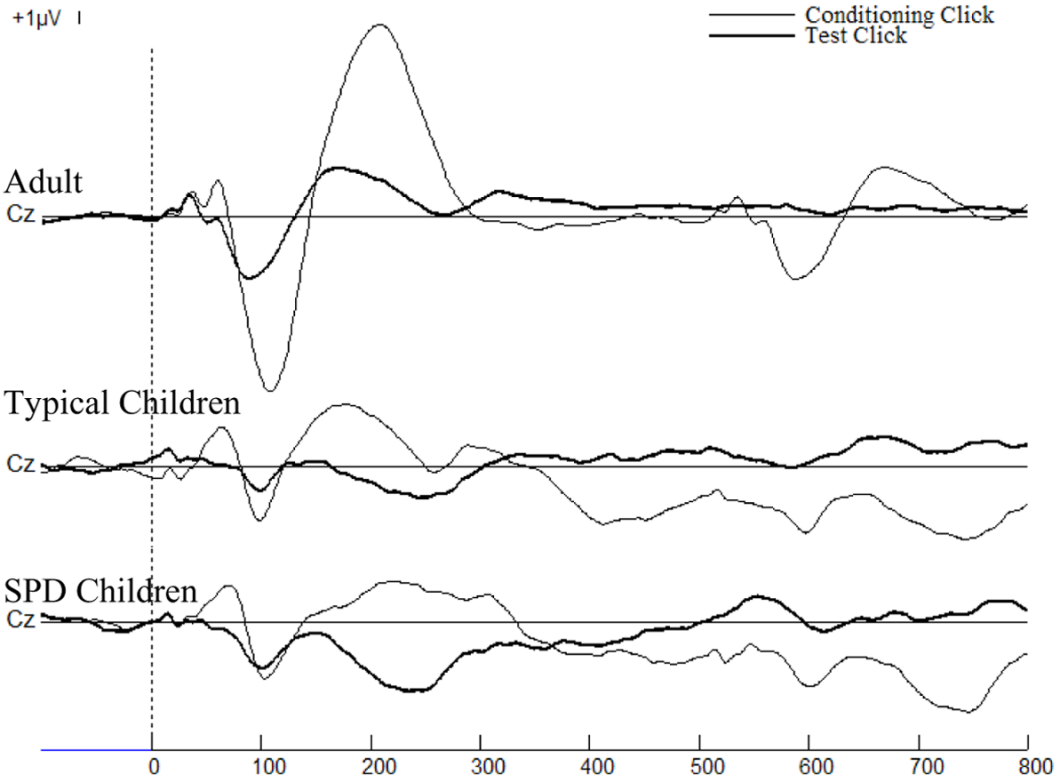

Figure 2.
Grand averages of sensory gating ERP at Cz with 100 ms baseline prior to the stimulus filtered with a bandpass of .23 Hz to 30 Hz for the N100. The thin line represents that conditioning click (first click) and the thick line represents the test click (second click).

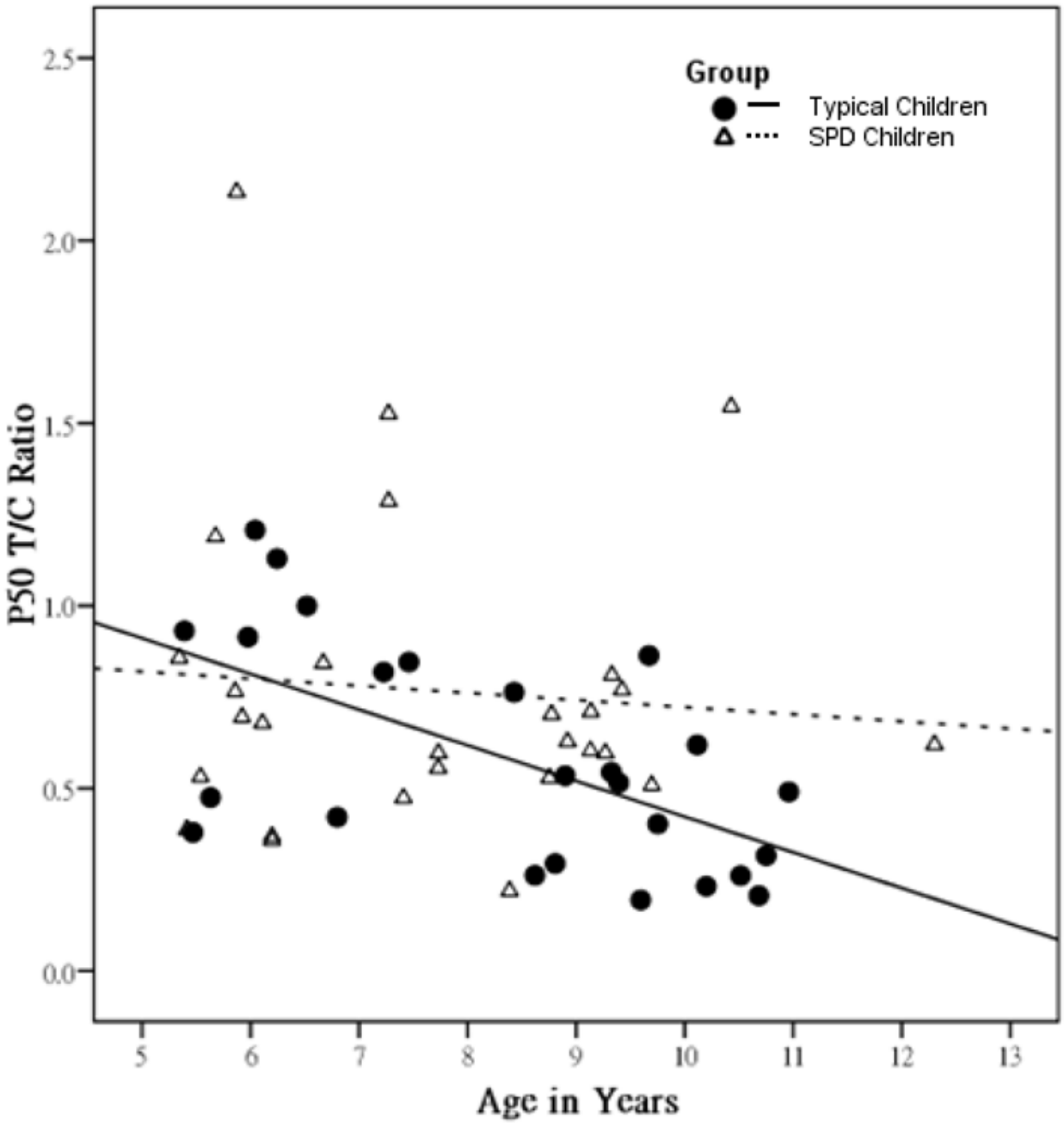

Figure 3.
Scatter Plot for the P50 T/C ratio by age for the two child groups. The P50 T/C ratios for individual typical children are represented by a circle and the P50 T/C ratios of individual children with SPD are represented by a triangle.

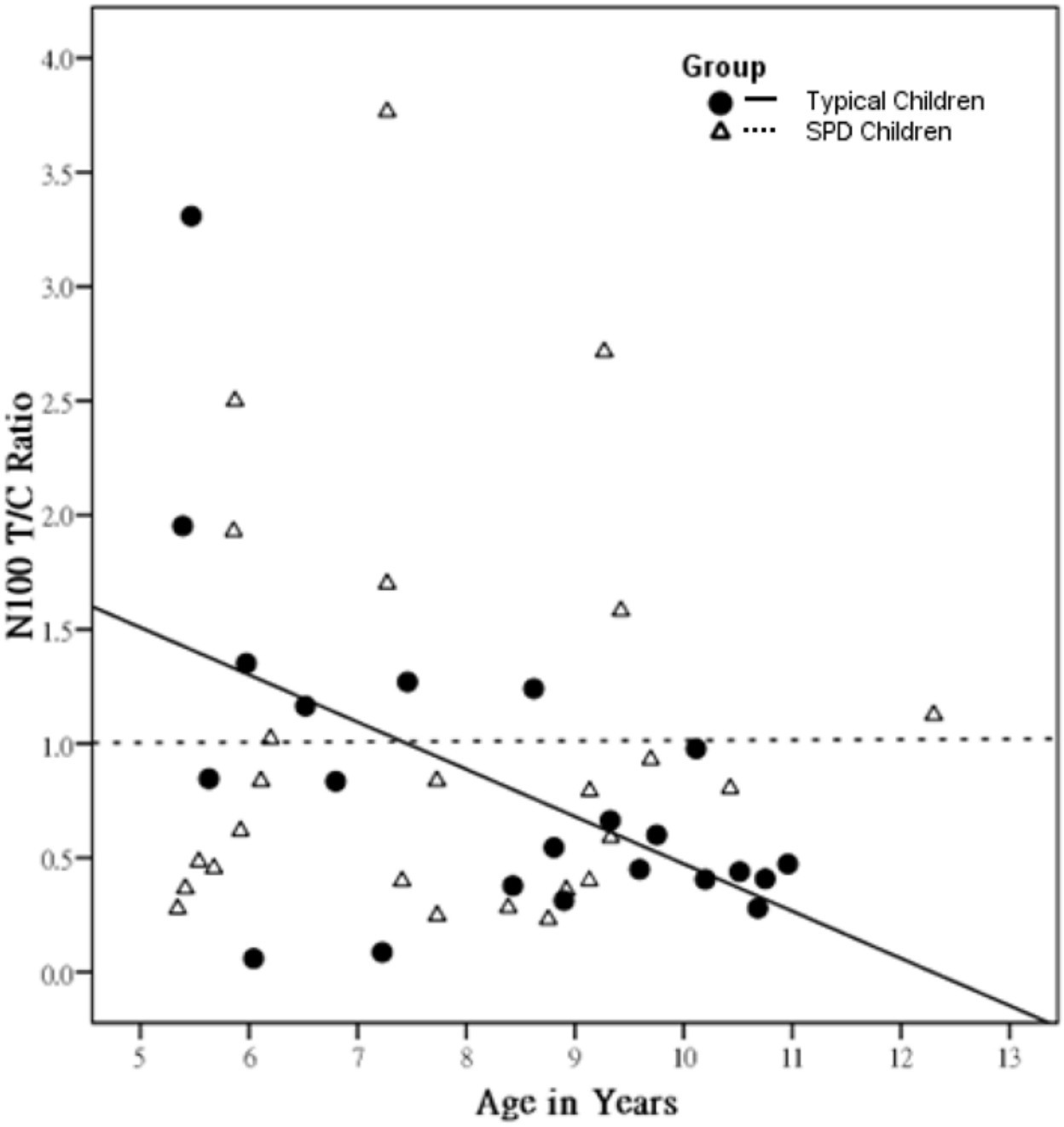

Figure 4.
Scatter Plot for the N100 T/C ratio by age for the two child groups. The N100 T/C ratios for individual typical children are represented by a circle and the N100 T/C ratios of individual children with SPD are represented by a triangle.

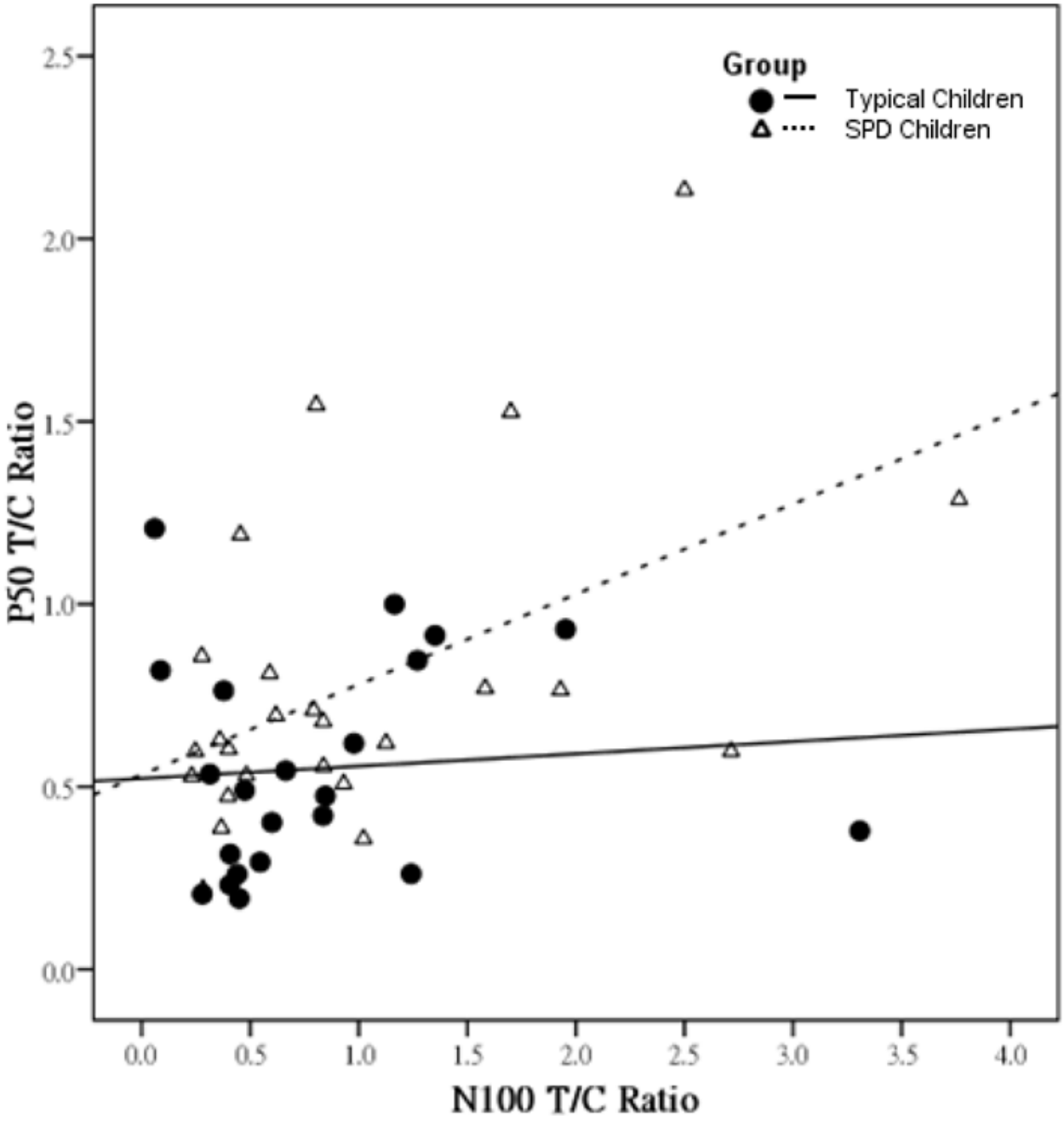

Figure 5.
Scatter Plot for the P50 T/C ratios by N100 T/C ratios for each of the two child groups. The T/C ratios for individual typical children are represented by a circle. The T/C ratios for individual SPD children are represented by a triangle. The regression lines for each group are zero order correlations, without the variance of age removed.

Table 1

MANOVA Results for the Sensory Profile in the two child groups.

Sections and Factors	*F*	*P*	Effect Size Partial η^2	Power
Auditory processing	29.12	<.0005	.36	1.00
Visual processing	10.27	.002	.17	.88
Vestibular processing	26.58	<.0005	.34	1.00
Touch processing	34.59	<.0005	.40	1.00
Multisensory processing	32.28	<.0005	.39	1.00
Oral sensory processing	8.23	.006	.14	.80
Modulation of movement affecting activity level	20.47	<.0005	.29	.99
Modulation related to body position and movement	9.24	.004	.15	.85
Sensory processing related to endurance/tone	21.36	<.0005	.30	1.00
Modulation of sensory input affecting emotional responses and activity level	10.77	.002	.17	.90
Modulation of visual input affecting emotional responses and activity level	24.79	<.0005	.33	1.00
Emotional/social responses	21.36	<.0005	.30	1.00
Behavioral outcomes of sensory processing	26.72	<.0005	.34	1.00
Items indicating thresholds for response	6.46	.014	.11	.70
Sensory seeking	13.23	.001	.21	.95
Emotionally reactive	29.12	<.0005	.36	1.00
Low endurance/tone	21.36	<.0005	.30	1.00
Oral sensory sensitivity	3.28	.076	.06	.43
Inattention/distractibility	42.01	<.0005	.45	1.00
Poor registration	20.93	<.0005	.29	.99
Sensory sensitivity	2.19	.145	.04	.31
Sedentary	.99	.323	.02	.17
Fine motor/perceptual	18.17	<.0005	.26	.99

Table 2

Mean T/C Ratio, Peak-to-Peak Amplitude (in μV), and Peak Latency (in ms) for each of the 3 groups. Standard Deviations are shown in parentheses.

	T/C Ratio	Amplitude Conditioning Stimulus	Amplitude Test Stimulus	Latency Conditioning Stimulus	Latency Test Stimulus
P50 Component					
Adult	.46 (.16)	6.28 (2.63)	2.74 (1.41)	62.42 (3.27)	60.86 (4.03)
Typical	.58 (.31)	5.04 (2.44)	2.67 (1.37)	64.88 (10.94)	61.25 (9.89)
SPD	.77 (.42)	4.96 (2.28)	3.16 (1.33)	64.28 (11.06)	60.90 (11.07)
N100 Component					
Adult	.41 (.23)	17.22 (6.91)	6.28 (3.11)	107.64 (9.70)	92.57 (10.20)
Typical	.82 (.72)	10.85 (5.81)	6.37 (3.68)	118.52 (35.13)	119.98 (39.09)
SPD	1.01 (.90)	11.14 (6.99)	7.06 (3.81)	111.17 (23.28)	106.22 (24.60)