Electroencephalography in Children With and Without Sensory Processing Disorders During Auditory Perception

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OBJECTIVE. We sought to determine whether children with sensory processing disorder (SPD) differ from typically developing children on a neurophysiological measure, the P300 component of event-related potentials produced in response to brief auditory stimulation.

METHOD. We used electroencephalographic measures (i.e., N200 and P300 components) to examine auditory processing in 20 children with SPD and 71 typically developing children, ages 5–10 yr.

RESULTS. Children with SPD demonstrated significantly smaller P300 amplitudes and shorter N200 latencies than typically developing children. Brain activity correctly distinguished children with SPD from typically developing children with 77% accuracy. We also found a significant relationship between the neurophysiological measures and functional performance on sensory and motor tasks.

CONCLUSION. This study presents empirical evidence that children with SPD display unique brain processing mechanisms compared with typical children and, therefore, provide further evidence for the neural deviations associated with SPD.


According to the existing literature, 1 in 20 children has a sensory processing disorder (SPD; Ahn, Miller, Milberger, & McIntosh, 2004), in which a person has difficulty organizing sensory stimuli to make an adaptive response. Children with SPD often display aversion to movement or touch, unfocused attention, and poor coordination as a result of their disorganized interpretation of stimuli (Bundy & Murray, 2002). Several postulates regarding subtypes of SPD have been developed. For example, Bundy and Murray (2002) categorized this dysfunction into two subtypes: dyspraxia and poor modulation. Children with dyspraxia exhibit poor motor planning and coordination, whereas children identified with modulation disorders fail to appropriately regulate their behavior because of inadequacies in processing specific attributes of the sensory information (Miller, Anzalone, Lane, Cermak, & Osten, 2007). In this study, we focused on the modulation subtype.

Despite an extensive history, SPD remains a controversial subject and continues to be a popular area of research in occupational therapy (Bundy & Murray, 2002). Although most research on SPD has used behavior measures, Miller (2003) advocated that “more objective and direct methods are required to characterize the population with sensory processing impairments” (p. 6). One such objective measure is electroencephalography (EEG).

EEG measures voltage changes at the scalp that are related to cortical neuronal activity (Stern, Ray, & Quigley, 2001). One method for making
inferences about the meaning of voltage changes is to examine event-related potentials (ERPs). In this method, a participant experiences an event, such as listening to a presented tone, at multiple times throughout the EEG recording. Segments of the EEG corresponding to the time of the tone presentations are averaged, producing an averaged ERP. (See Figure 1 for an example of an averaged ERP to an auditory stimulus and the component labels.) Then, both the latency and the amplitude of the major peaks in the averaged ERP (i.e., the components) are measured and compared between individuals or groups. Latency, typically measured in milliseconds, involves the timing of the component; that is, how much time elapsed between stimulus presentation and the component. Amplitude involves the amount or change in voltage (measured in microvolts) and can “reflect variations in the degree to which some process is invoked” (Rugg & Coles, 1995, p. 31). Amplitude and latency values are quantitative and objective measures of neural activity that can help researchers illustrate the relationship between physiologic processing and behavioral manifestations (Banaschewski & Brandeis, 2007).

Examining actual neuronal activity in response to a stimulus is a technique that has only recently been used to test the assumption that sensory processing difficulties are a manifestation of neurological processing deficits (Davies, Chang, & Gavin, 2009, 2010; Davies & Gavin, 2007). Studies using the sensory registration paradigm (Davies & Gavin, 2007; Davies et al., 2010) have established ERP research as a verifiable measure of the differences between children with SPD and typically developing children. The sensory registration paradigm involves two different auditory stimuli, each played at a soft and a loud volume, presented multiple times while the participant stares at a fixed mark on a computer screen.

The ERP data from the four stimuli measure an individual’s ability to discriminate and organize auditory stimuli. Using only the amplitude and latency of ERP components generated by the sensory registration paradigm, Davies et al. (2010) correctly classified children identified with the modulation subtype of SPD and typically developing children with 95.6% accuracy. This high level of accuracy suggests that neurophysiological responses to simple auditory stimuli may correctly predict sensory modulation difficulties in children.

In the Davies et al. (2010) study, the highly accurate group categorization was largely based on the P300 component. This late component is indicative of additional cognitive activity (Polich, 2007). P300 has become the most studied ERP component (Wu, Liu, & Quinn-Walsh, 2008). Other P300 research has found significant differences between neurotypical individuals and individuals with schizophrenia (Klein, Berg, Rockstroh, & Andresen, 1999; Weisbrod, Hill, Niethammer, & Sauer, 1999), attention deficit hyperactivity disorder (Barry, Johnstone, & Clarke, 2003; Sugawara, Sadeghpour, Traversay, & Ornitz, 1994; Sunohara et al., 1999), autism (Lincoln, Courchesne, Harms, & Allen, 1995), and epilepsy (Naganuma et al., 1997). Although those studies have demonstrated significant differences between the mean P300 amplitudes of individuals with and without these neurological disorders, they have yet to demonstrate relationships between the P300 and the functional behaviors that are used to define the disorder in individuals.

Despite the insights gained from the previous research, additional studies are still needed to investigate the relationship between the P300 component and SPD. In this study, we sought to determine whether children with SPD differ from typically developing children on a neurophysiological measure, the P300 component of ERPs.
produced in response to brief auditory stimulation. Such an outcome would replicate, in part, the findings of Davies et al. (2010).

We examined differences in brain processing between the two groups of children from two viewpoints. First, do the groups differ in either mean amplitude or mean latency measures of the P300 ERP component? Second, can the individual differences in the late ERP component be used to accurately classify children according to their diagnostic category? Extending beyond the Davies et al. study, our third research question focused on whether a relationship between the P300 and functional behaviors often used to diagnose children with SPD can be demonstrated. Specifically, does a significant relationship exist between the amplitude and latency of the P300 ERP component and scores on the Short Sensory Profile (SSP; McIntosh, Miller, Shyu, & Dunn, 1999) and the Clinical Observation of Motor and Postural Skills (COMPS; Pollock, Kaplan, & Law, 2000)?

Method

Participants

This study was performed using a subsample of a larger ongoing study. A total of 91 children ages 5–10 yr were recruited from two sources, creating two independent groups. The first group consisted of 20 children with SPD (mean \( M = 7.0 \), standard deviation \( SD = 1.6 \)) who were referred to the study by the medical community. This group consisted of 14 boys and 6 girls. The unbalanced male-to-female ratio is representative of the population with SPD (Ahn et al., 2004). The second group consisted of 71 typically developing children (\( M = 7.5 \), \( SD = 1.5 \)) from the community. The typically developing children who volunteered for the study had no known neurological diagnosis and did not have a history of receiving any special services. Two of the typically developing children were subsequently excluded because of missing data on EEG measures or COMPS scores. Although the typically developing children were as a group slightly older than the children with SPD, the difference was not significant (\( t[87] = 1.33, p = .19 \)). Each participant’s group membership was independently confirmed in the laboratory using two behavioral assessments.

Behavioral Assessments

The SSP is a norm-referenced screening tool appropriate for children ages 3–10 yr. It includes seven subscales: Auditory Filtering, Low Energy–Weak, Movement Sensitivity, Tactile Sensitivity, Taste–Smell Sensitivity, Underresponsive–Seeks Sensation, and Visual–Auditory Sensitivity. The SSP has acceptable internal consistency reliability and construct validity (McIntosh et al., 1999); it is derived from the Sensory Profile (Dunn, 1999) and formatted as a caregiver questionnaire. The SSP was completed by the children’s parents before visiting the lab. Children with SPD scored significantly lower than the typically developing children on all seven SSP subscales as well as the total score (Table 1).

The COMPS is a short screening test used to identify motor difficulties with both postural and praxis components for children ages 5–15 yr. The COMPS has been found to have acceptable reliability and validity (Pollock et al., 2000). This assessment was administered during the participants’ second visit to the laboratory for this study. As with the SSP, children with SPD also scored significantly lower than typically developing children on the COMPS measure (see Table 1).

Procedure

Informed consent was obtained from the parents of all participants, and procedures for this research study were approved by the human research committee at Colorado
State University. Additionally, children were informed of the study’s procedures in a child-friendly manner, and all children agreed to participate by signing an assent form. The participants visited the lab twice. On both visits, each participant completed the EEG procedures described in the paragraphs that follow.

The participants sat in a relaxed position in a high-backed chair, and the EEG sensors, which were contained in a stretch-fabric cap, were placed on their head. Strategies to reduce artifacts in the EEG recordings caused by eye blinks, movement, and muscle activity were explained and demonstrated to the participants. Next, resting EEG recordings were taken. Earphones (ER-3A; Etymotic Research, Elk Grove Village, IL) were then inserted, and an auditory threshold screening was conducted. EEG recordings were conducted during two ERP paradigms: (1) a sensory gating paradigm and (2) a sensory registration paradigm. Each paradigm required the participants to listen to paired clicks or tones varying in frequency and intensity. In this study, however, we report only the results of data collected during the sensory registration paradigm on the first visit. The order of paradigm presentation was counterbalanced between participants. Participants watched a silent Wallace and Gromit film (Schelley et al., 1996) to keep them engaged during each paradigm.

The auditory sensory registration paradigm was adapted from Lincoln et al. (1995) and Davies et al. (2010). For this paradigm, four different auditory stimuli were presented in both ears using E-Prime software (Psychological Software Tools, Pittsburgh, PA). The stimuli were 1 kHz at 50 dB sound processing level (SPL), 1 kHz at 70 dB SPL, 3 kHz at 52 dB SPL, and 3 kHz at 73 dB SPL. We examined only the 3 kHz data in this study. The duration of each stimulus was 50 ms with 10-ms onset and offset ramps. The interstimulus interval was 2 s. Stimuli were presented in 4 blocks of 100 stimuli presentations with 30-s breaks between blocks.

**EEG Recording and Analysis**

EEG activity was recorded using a 32-channel BioSemi Active Two EEG system (BioSemi Inc., Amsterdam, the Netherlands). The electrodes were located in accordance with the 10–20 system (American Electroencephalographic Society, 1994). EEG was recorded with the Common Mode Sense active electrode as the reference and the Driven Right Leg passive electrode as the ground (BioSemi, Inc., n.d.). Electrooculograms (EOGs) 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 more individual electrodes were placed on the left and right earlobes and mastoids. EEG signals were sampled at an analog-to-digital rate of 1024 Hz with a bandwidth of 268 Hz.

We conducted all EEG and ERP analyses offline using the Brain Vision Analyzer2 software (Brain Products GmbH, Munich, Germany). The left and right earlobe recordings were averaged and used as the offline reference. The four individual EOG channels were converted to a vertical and a horizontal bipolar EOG. The EEG recordings were filtered with a band pass of 0.23–30 Hz (12 dB/octave). The EEG was segmented about each auditory stimulus with a duration of 100 ms prestimulus onset to 800 ms poststimulus onset. Eye-blink artifacts were removed using a regression procedure. Segments with deviations greater than ±100 μV on any of the EEG channels or the bipolar EOG channels were eliminated. Nonrejected segments for each auditory stimulus were then baseline corrected using the prestimulus period of −100 to 0 ms and averaged to create ERP waveforms for each participant, from which the ERP components were measured.

We used methods from Lincoln et al. (1995) and Davies et al. (2010) to measure peak-to-peak amplitude and latency for N200 and P300 components. The most negative peak 240–290 ms after stimulus onset was defined as N200. The most positive peak 360–410 ms after stimulus onset was defined as P300. We calculated the peak-to-peak amplitude of P300 by subtracting the N200 peak amplitude from the P300 peak amplitude. A computer program, Brainwaves Peak Picker, created by the Brainwaves Research Laboratory at Colorado State University, was used to provide automatic scoring and visual inspection and, when necessary, to allow manual marking of components. Two teams of independent raters completed the visual inspection of automatic marking of components. To increase reliability of any manual adjustment of component values, all values were checked and agreed on by the opposite team of raters. Congruent with Davies et al. (2010), we examined only the amplitude and latency measurements obtained from the Fz and Pz sensors placed on the scalp over the frontal lobe and the parietal lobes, respectively, along the sagittal midline.

**Statistical Analysis**

To answer the first research question, we used a $2 \times 2 \times 2$ analysis of variance (ANOVA) to evaluate whether the two child groups differed in mean amplitude or mean latency measures of the P300 ERP component. The first factor, Group, was a between-subjects factor with two levels, (1) typically developing children or (2) children with SPD. The second factor, a within-subject factor, was
Results

The mean amplitude and latency values for children with SPD were less than those for typically developing children at each electrode site for each of the two auditory stimulus intensities (Table 2). ANOVA for the peak-to-peak amplitude of the P300 revealed a significant difference between the two groups ($F[1, 87] = 5.11, p = .026, \eta_p^2 = 0.06$). We also found significant main effects for Intensity ($F[1, 87] = 14.14, p < .0005, \eta_p^2 = 0.14$) and for Electrode Site ($F[1, 87] = 36.55, p < .0005, \eta_p^2 = 0.30$). The Intensity × Electrode Site interaction was also significant ($F[1, 87] = 4.75, p = .032, \eta_p^2 = 0.05$). ANOVA for the latency of the N200 also revealed a significant difference between the two groups ($F[1, 87] = 4.33, p = .040, \eta_p^2 = 0.05$). We found a significant main effect for Electrode Site ($F[1, 87] = 4.96, p = .029, \eta_p^2 = 0.05$) but not for the main effect of Intensity. However, the Intensity × Electrode Site ($F[1, 87] = 10.33, p = .002, \eta_p^2 = 0.11$) and the Intensity × Electrode Site × Group ($F[1, 87] = 5.18, p = .025, \eta_p^2 = 0.06$) interactions were significant.

To determine which P300 amplitude and N200 latency measures in combination might best predict the group membership of each child participant, we calculated zero-order correlations between each measure and group membership using a point-biserial correlation approach. We chose the three variables with the highest correlation coefficients to serve as the predictor variables in the discriminant analysis: (1) P300 peak-to-peak amplitude of the 3 kHz stimulus at 73 dB SPL measured at Fz ($r_{pb} = -.21, p = .046$); (2) P300 peak-to-peak amplitude of the 3 kHz stimulus at 73 dB SPL measured at Pz ($r_{pb} = -.20, p = .058$); and (3) N200 latency of the 3 kHz stimulus at 53 dB SPL measured at Fz ($r_{pb} = -.31, p = .003$). Because the distribution of ages and gender was different in each group, we also entered age and gender into the discriminant analysis as predictor variables. The results of this discriminant analysis showed that typically developing children and children with SPD were significantly distinct from each other (Wilks’ $\Lambda = .77, p = .001$). The discriminant analysis correctly classified 77% of all child participants: 77% correct classification for typically developing children and 79% correct classification for children with SPD. The standardized canonical discriminant function coefficients were .30 for Fz and .40 for Pz of the P300 amplitudes for the 3 kHz stimulus presented at 73 dB; .81 for the N200 latency of Fz for the 3 kHz stimulus presented at 52 dB; .67 for age; and .08 for gender.

The distribution of discriminant scores derived from the ERP components is depicted in Figure 2 as a function of the corresponding discriminant scores derived from the two behavioral measures, the SSP and the COMPS. As expected, the discriminant analysis using the two behavioral measures showed that typical children and children with SPD were significantly separated from each other (Wilks’ $\Lambda = .47, p < .0005$). This second discriminant analysis correctly classified 92% of all child participants, with 93% correct classification for typically developing children and 90% correct classification for children with SPD. Correlation analysis revealed a statistically significant relationship between the two discriminant functions ($r = .38, p = .0003$).

Discussion

A major purpose of this study was to investigate differences in brain processing between children with SPD and typically developing children. The results of the ANOVA...
confirmed that children with SPD demonstrate brain processing of simple auditory stimuli that is significantly different from that of their age-matched peers.

An additional goal of this study was to see whether neurophysiological measures could correctly classify children as belonging to either a sensory modulation deficit category or a typically developing category. The discriminant analysis confirmed that late components of the sensory registration paradigm could correctly classify children with SPD with 79% accuracy. These results are consistent with previous research using the auditory sensory registration paradigm and children with SPD as well as with studies investigating the late components in people with other disabilities.

Our final goal was to determine the extent to which measures of brain activity during sensory processing relate to behavioral measures typically used to evaluate sensory processing deficits. We found a statistically significant relationship ($r = .38$). These neurophysiological findings contribute to the body of knowledge for understanding the neural manifestations of SPD.

Davies et al.’s (2010) previous findings, combined with this study’s results, show that the sensory registration paradigm can be used to find differences between children with SPD and their typically developing peers. Differences in sensory registration suggest that the manifestations of SPD may be the result of atypical neurophysiological functioning in relation to the discrimination

Table 2. Mean P300 Amplitude and Mean N200 Latency of the Event-Related Potential (ERP) Components Obtained at Two Electrode Sites for Two of the Auditory Stimuli Used in the Sensory Registration Paradigm

<table>
<thead>
<tr>
<th>ERP Components</th>
<th>Auditory Stimuli</th>
<th>3 kHz 52 dB</th>
<th>3 kHz 73 dB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean P300 amplitudes, μV</td>
<td>Fz Site</td>
<td>Pz Site</td>
<td>Fz Site</td>
</tr>
<tr>
<td>Typically developing children</td>
<td>7.54 (3.08)</td>
<td>5.31 (2.47)</td>
<td>9.05 (3.71)</td>
</tr>
<tr>
<td>Children with SPD</td>
<td>6.99 (1.82)</td>
<td>4.46 (2.06)</td>
<td>7.21 (3.10)</td>
</tr>
<tr>
<td>Mean N200 latencies, ms</td>
<td>Fz Site</td>
<td>Pz Site</td>
<td>Fz Site</td>
</tr>
<tr>
<td>Typically developing children</td>
<td>281.54 (48.20)</td>
<td>289.35 (51.37)</td>
<td>273.10 (46.88)</td>
</tr>
<tr>
<td>Children with SPD</td>
<td>244.29 (47.83)</td>
<td>280.57 (38.64)</td>
<td>262.55 (54.47)</td>
</tr>
</tbody>
</table>

Note. Standard deviations appear in parentheses. SPD = sensory processing disorder.

Figure 2. The distribution of discriminant scores derived from the event-related potential measurements depicted as a function of their corresponding discriminant scores derived from the two behavioral measures, the SSP and the COMPS.

Note. The centroids for each set of discriminant scores are marked as dotted lines. SSP = Short Sensory Profile; COMPS = Clinical Observation of Motor and Postural Skills.
and organization of stimuli. Discrimination and organization deficiencies would manifest as inappropriate reactions to everyday sensory stimuli. For example, if a child has difficulty determining the difference between a ringing phone and a fire alarm or does not consistently recognize a ringing phone, his or her response may be inappropriate, atypical behavior. Equipped with this information regarding neural mechanisms associated with SPD, professionals are better able to provide individually tailored services to children with SPD.

Late components have proved to be important physiological markers to distinguish typically developing children from children with SPD (Davies et al., 2010) and other disabilities (Barry et al., 2003; Klein et al., 1999; Lincoln et al., 1995; Naganuma et al., 1997; Sugawara et al., 1994; Sunohara et al., 1999; Weisbrod et al., 1999). We found that the children with SPD displayed smaller P300 amplitudes than their typically developing peers. These findings are inconsistent with Davies et al.’s (2010) findings demonstrating that children with SPD had, in general, larger P300 amplitudes than their typically developing peers. A methodological difference between these two studies might account for the discrepancy. In the Davies et al. (2010) study, the children listened to the auditory stimuli while sitting quietly, staring at a fixed symbol on a computer screen, with a short break about every 4 min. In our present study, the children watched a silent movie. The distraction of the movie might have changed the manner in which the children processed the auditory stimuli. Decreased P300 amplitudes have been related to difficulty discriminating stimuli (Sugawara et al., 1994) and increased task difficulty (Wu et al., 2008) and, in general, are smaller in people with disabilities (Linden, 2005). In our study, having the children watch a movie could have distracted them from listening to the auditory stimuli. In the Davies et al. (2010) study, in which the children were not distracted by a movie, children with SPD may have exhibited more capacity allocated to processing the auditory stimuli, resulting in a larger P300 than in typically developing children (Kok, 2001).

The third purpose of this study was to examine the relationship between the neurophysiological measures and functional performance on sensory and motor tasks. As shown in Figure 2, when the results of the ERP measures and the behavioral measures are combined, the classification of children belonging to the SPD or typically developing categories is clear. Sensitivity of the combined neurophysiological and behavioral measures for classifying children with SPD is shown in the lower, dark gray quadrant in Figure 2. The upper, light gray quadrant shows the level of specificity, which is the ability of this combination of measures to correctly classify children who are typically developing. The figure also illustrates that only 1 child with SPD was incorrectly placed in the quadrant with the typically developing children and only 1 typically developing child was misplaced in the quadrant with the children with SPD. Several other children in both groups were not clearly classified in either group, as shown by the cases that stray into the two unmarked quadrants.

Despite the ease and standardization of using auditory stimuli for EEG recordings, other forms of stimuli, such as visual, tactile, or proprioceptive stimuli, may provide additional ERP data about sensory processing in relation to modulation abilities. Moreover, the silent Wallace and Gromit film (Schelley et al., 1996) used in this study to entertain the participants may have implications for sensory processing that need to be considered in subsequent studies, because watching the film may have altered the amount of attention paid to the auditory stimuli. Generalizability of the results may be limited because of convenience sampling. Future research should include studies of various disability groups and the use of ERP data to differentiate these groups from children with SPD. Subsequent research in the field could also examine the utility of EEG as a measure of intervention effectiveness.

Conclusion

The purpose of this study was to provide neurophysiological evidence showing that children with the modulation subtype of SPD, compared with typically developing children, have statistically significant differences in brain processing of simple auditory stimuli. The late components of the ERPs obtained from a sensory registration paradigm correctly classified 77% of the children. This study’s results will help practitioners understand the underlying physiological mechanisms responsible for atypical behavior. Consequently, compensatory or remedial therapeutic approaches can target the foundational physiological origins of the disorder rather than the behavioral manifestations.

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References


