Validating the Diagnosis of Sensory Processing Disorders Using EEG Technology

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KEY WORDS
- behavior
- brain
- electroencephalography (EEG)
- event-related potential (ERP)
- pediatric
- sensory gating
- sensory integration
- sensory processing
- sensory processing disorder (SPD)

OBJECTIVE. This study tested the assumption of sensory integration theory that states that a relationship exists between brain function and the behavioral manifestations of sensory integrative dysfunction.

METHOD. Electroencephalographic measures were used to examine brain processing in 28 children with sensory processing disorders (SPD) and 25 children who were typically developing, ages 5–12 years.

RESULTS. Children with SPD demonstrated less sensory gating than children who were typically developing. A significant relationship between sensory gating and age was found in children who were typically developing but not in children with SPD. Brain activity correctly distinguished children with SPD from children who were typically developing with 86% accuracy.

CONCLUSION. These results present empirical evidence that children with SPD display unique brain processing mechanisms compared to children who are typically developing and provide external validity for the diagnosis of SPD.

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A. Jean Ayres's theory of sensory integration has generated more research and controversy than any other theory developed by an occupational therapist (Bundy & Murray, 2002). These fervent controversies have emphasized how particularly important it is to find more precise methods to study the phenomenon of sensory integration and the treatment of children with sensory processing disorders (SPD). One approach yet to be pursued in the study of sensory integration is to directly test the assumptions of the theory itself. Bundy and Murray (2002) articulated five assumptions of the sensory integration theory related to the neural and behavioral bases of sensory integration (pp. 10–12). The assumptions that are most germane to validating the theory of sensory integration relate to the relationship between brain maturation or function and behavioral manifestations of sensory integrative dysfunction, which was eloquently stated by Short-Degraff:

> Sensory integration theory assumes that the brain is immature at birth and also is immature [or dysfunctional] in some individuals with learning problems. The goal of sensory integration therapy is to provide stimulation that will address certain brain levels (primarily subcortical), enabling them to mature [or function more normally], and thereby assisting the brain to work as an integrated whole. (Short-Degraff, 1988, p. 200) [Bracketed material added by Bundy & Murray, 2002, p. 11]

Of these assumptions, two can be postulated as hypotheses that can be directly tested using a brain imaging technique commonly used by neuroscientists. First, Ayres's (1972, 1989) theory proposes that behavioral expressions of sensory integration dysfunction are related to immaturity or malfunction in brain processing. This assumption leads to the hypothesis that, when presented with

discrete sensory stimuli, the brain activity of children whose behaviors are indicative of a sensory processing disorder will differ from the brain activity of children who are typically developing. More important, Ayres's theory also proposes that sensory integrative therapy will change neural mechanisms. This assumption leads to the hypothesis that the brain activity of children with SPD observed after therapeutic intervention will differ from brain activity observed before intervention. However, before this assumption can be tested, evidence for the validity of the first assumption should be demonstrated. Interestingly, despite the fact that Ayres began writing articles about sensory integration in the 1960s (e.g., Ayres, 1964, 1965, 1969), these two hypotheses have yet to be directly tested. For example, brain imaging studies that examine the neural processing mechanisms in children with SPD and children without disorders have yet to be conducted to determine whether group differences do indeed exist. Mulligan (2002) stated that, if we are to establish professional consensus regarding the validity of the sensory integration theory, support for the basic assumptions of the theory are needed.

Sensory Integration and SPD

Sensory integration is a therapeutic approach that has been used for many years by occupational therapists and has a strong potential to enhance occupational performance in children. Ayres described sensory integration as an approach used to enhance the brain's ability to organize sensory input for use in functional behaviors (1972, 1979). According to Ayres (1972), the essential principle in sensory integrative therapy is to provide the child with experiences rich in sensory input, in a guided manner, to produce an adaptive response (i.e., functional behavior) deemed more effective than previously observed behaviors. Therefore, the central features of Ayres's sensory integrative theory and intervention focus on the fact that the brain organizes sensory input in order for the individual to participate in meaningful occupations (Parham, 2002).

Occasionally the terms that professionals use to describe the children who receive therapy guided by sensory integration theory can be ambiguous. For example, the term *sensory processing* is often used interchangeably with *sensory integration,* and controversy exists over interchanging these terms (Mulligan, 2002). In the neurosciences, *sensory integration* is used to specifically describe the combining of signals from two or more senses in the central nervous system (Calvert, Spence, & Stein, 2004; Hicks, Molotchnikoff, & Ono, 1993). In contrast, Ayres (1972) defined *integration* as "the interaction and coordination of two or more functions or processes in a manner which enhances the adaptiveness of the brain's response" (pp. 25–26). She further defined the integrative process as filtering, organizing, and integrating sensory information and did not specifically limit it to the combination of two or more senses. Thus, her definitions of the term *sensory integration* seem to give a much broader representation of processing than just the integration of several senses, the definition often used by neuroscientists. When conducting neuroscience research on children who have difficulties in processing sensory information, classifying them as having SPD better captures all aspects of the problems observed in these children, especially filtering, organization, and integration as described very early on by Ayres (1972). The electroencephalographic brain imaging techniques described in this article are commonly used by neuroscientists (Handy, 2005; Luck, 2005) and are ideal for measuring the filtering and organization aspects of sensory processing.

Electroencephalography and Event-Related Potentials

To examine the association among brain structure, function, and behavior related to sensory processing abilities, real-time measures of brain activation during the processing of sensory stimuli provide the most convincing data. Electroencephalography (EEG) and event-related potentials (ERPs)—functional neuroimaging methods—are ideal techniques that may offer occupational therapists new strategies for studying SPD. To obtain EEG data, one or more metallic sensors are placed on the scalp to detect very small (10–50 microvolts) and continuous voltage changes, which are then amplified and digitized.

Because EEG measures electrical activity of the cortical regions of the brain, it can provide a more accurate assessment of the processing of sensory stimuli by the brain than can peripheral measures. Although peripheral measures such as electrodermal activity and heart rate do have the advantage of measuring an individual's *interaction* with the environment (Stern, Ray, & Quigley, 2001), investigators can only *infer* from such measures what processes may be invoked in the central nervous system to produce the changes observed at the periphery. Electroencephalographic methodologies provide the advantage of directly measuring brain activity.

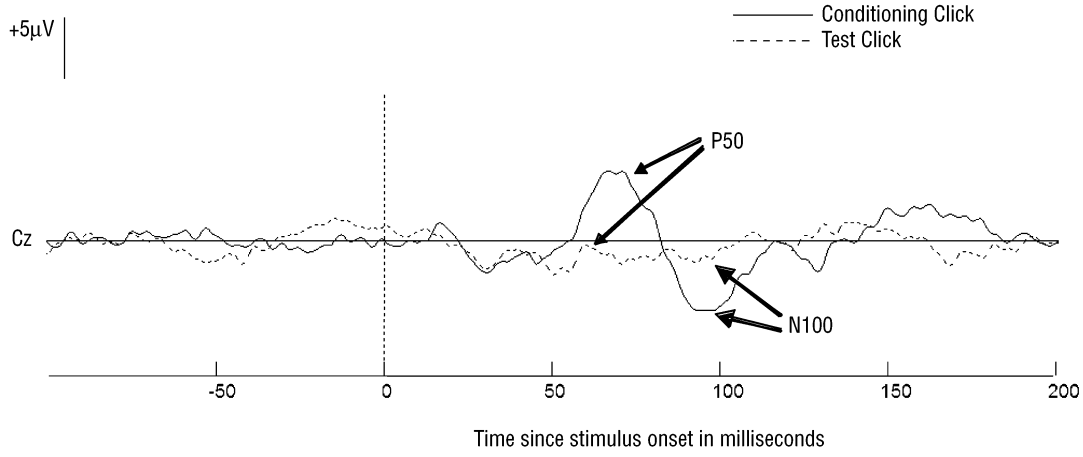
ERPs are graphical displays of the brain's electrical activity typically associated with a specific, defined event. ERPs are obtained by time-locking the EEG to the occurrence of each event (e.g., the onset of a sensory stimulus), creating segments around the event, and then averaging together the segments of the multiple presentations of the event (Segalowitz & Davies, 2004). Thus, an ERP waveform

provides information about the temporal aspects of information processing of stimulus events. Two aspects of the ERP waveform can be measured: amplitude and latency. Amplitude is measured in microvolts (μV) and can be positive or negative based on a relative baseline of zero. Latency is usually measured in milliseconds (ms) post stimulus onset. The ERP components are defined as deflections from baseline and labeled with a *P* for positive deflections and with an *N* for negative deflections (see Figure 1a for an example of an ERP and component labels for an auditory stimulus). The number in the label for a component indicates the latency in milliseconds from the time of the stimulus presentation to the peak of the deflection. As illustrated in Figure 1a, P50 represents the positive deflection occurring about 50 ms after the stimulus presentation. The N100 denotes the negative deflection in the proximity of 100 ms after the stimulus presentation or event.

Studying Sensory Processing Disorders Using EEG/ERP Methodology

Sensory processing has been studied using EEG/ERPs for many years in the general adult population and also in several disorders such as schizophrenia (e.g., Boutros, Belger, Campbell, D'Souza, & Krystal, 1999; Freedman, Adler, & Waldo, 1987), attention deficit hyperactivity disorder (ADHD) (Olincy et al., 2000), and autism (Kemner, Oranje, Verbaten, & van Engeland, 2002). Most of the studies involving clinical populations have used auditory

1a. This brain response of a child from the control group displays a large difference in the P50 peak amplitude of the first click (solid line) when compared to the amplitude of the second click (dashed line).



1b. This brain response of a child from the group with SPD shows minimal difference in the P50 peak amplitude of the first click (solid line) when compared to the amplitude of the second click (dashed line).

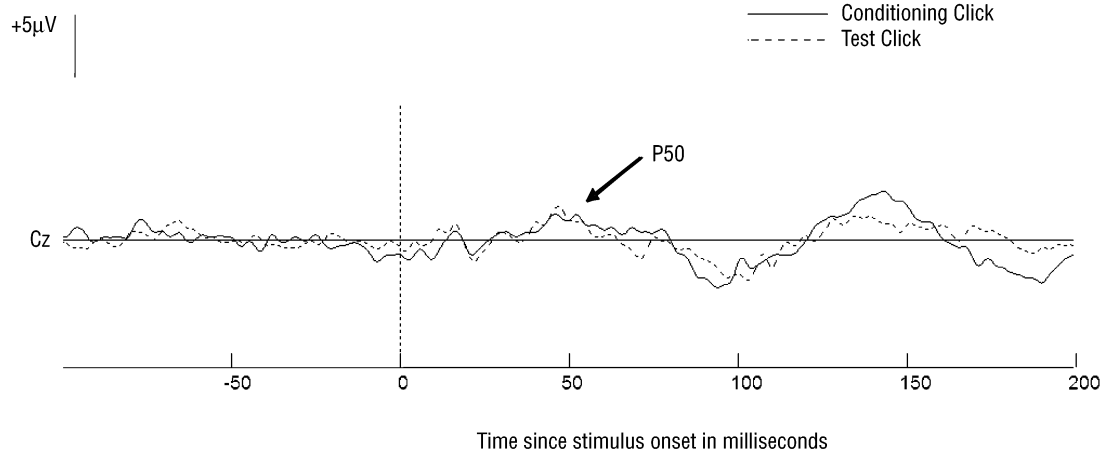


Figure 1. ERP waveforms for two children representing their brain response to the sensory gating paradigm. The averaged brain response to the presentations for the first click sound, the Conditioning click, is shown as a solid line. The second click sound, the Test click, is shown with a dashed line.

stimulation, although a few have used visual (e.g., Dawson et al., 2002; Karrer, Karrer, Bloom, Chaney, & Davis, 1998) or somatosensory (e.g., Arnfred & Chen, 2004) stimulation. Furthermore, of the few studies that investigated the developmental changes in sensory processing in children, the majority have used auditory stimuli (e.g., Čeponinené, Rinne, & Näätänen, 2002; Kraus et al., 1993; Moore & Guan, 2001). Given the rich history of ERP studies of sensory processing of auditory stimuli, it would be prudent in the planning of initial studies of children with SPD to borrow from these specific EEG/ERP methodologies (i.e., paradigms).

After carefully critiquing the literature, we concluded that two auditory paradigms seemed most likely to reveal information regarding the underlying brain mechanisms reflecting the type of behaviors observed in children with SPD. Specifically, the sensory gating paradigm evaluates the brain mechanisms for suppressing repeated or irrelevant stimuli. The second paradigm is the sensory registration paradigm, which evaluates the consistency of brain responses to a variety of auditory stimuli. Use of these two ERP paradigms may allow for the direct evaluation of the brain processing mechanisms of children with and without SPD based on Ayres's (1972) theory. The sensory gating paradigm addresses the function of the brain's ability to filter sensory information, whereas the sensory registration paradigm addresses the brain's ability to organize sensory information. These are two of the three brain functions that Ayres (1972) described as being needed for a person to perform.

The sensory gating paradigm has been used for more than 30 years to study auditory processing dysfunction, primarily in patients with schizophrenia (e.g., Boutros et al., 1999; Freedman et al., 1987). However, sensory gating has more recently been used to study processing in people with ADHD (Olincy et al., 2000), traumatic brain injury (Arciniegas et al., 1999), and autism (Kemner et al., 2002). In the auditory sensory gating paradigm, the participant listens to repeated presentations of a pair of auditory click sounds. The clicks are separated from each other by 500 ms. The component of interest in this paradigm is the positive deflection around 50 ms post stimulus, called the P50. In adults and some children without SPD, the brain has a smaller positive response about 50 ms after the second click stimulus, or Test click, when compared to the brain's response to the first click stimulus, or Conditioning click (see Figure 1a). The reduction of the amplitude of the P50 component to the second click compared to the first click presumably represents enhanced suppression or gating.

Most studies on sensory gating have examined adults; only four published studies have examined children (Freedman et al., 1987; Kemner et al., 2002; Marshall, Bar-Haim, & Fox, 2004; Myles-Worsley et al., 1996) and one involved infants (Kisley, Polk, Ross, Levisohn, & Freedman, 2003). Controversy remains concerning the developmental trajectory of sensory gating (Freedman et al., 1987; Kisley et al., 2003; Marshall et al., 2004; Myles-Worsley et al., 1996). Kisley et al. (2003) demonstrated that gating is present at some level in some infants during sleep. Two studies indicated that sensory gating becomes stronger with age (Freedman et al., 1987; Marshall et al., 2004). In contrast, another study indicated that children age 10 years have adult-like levels of sensory gating (Myles-Worsley et al., 1996).

The second ERP paradigm of interest in the present study, the sensory registration paradigm, was designed based on two studies conducted in children with autism (Bruneau, Garreau, Roux, & Lelord, 1987; Lincoln, Courchesne, Harms, & Allen, 1995). In this paradigm, auditory tones are presented at different frequencies and intensities. The term *registration* describes the fact that, when several auditory stimuli are presented to control individuals, distinct brain responses are elicited for each of the different auditory stimuli. Accordingly, each tone is uniquely "registered" in the brain and can be displayed by an identifiable and dependable brain response or waveform, hence the name *sensory registration*. Use of this paradigm allows for examining whether children with SPD have more difficulty in processing auditory stimuli in a consistent and ordered manner when compared to a control group.

In the sensory registration paradigm, two ERP components, N100 (the negative deflection occurring about 100 ms post stimulus) and P200 (the positive deflection occurring about 200 ms post stimulus), are sensitive to changes of stimuli intensity and frequency. Participants with autism, compared to children without disabilities, did not show the normal increase in amplitude to increased intensity (Bruneau et al., 1987; Lincoln et al., 1995). Given the results of these two studies, this paradigm has the potential to measure the organization of brain processing of auditory stimuli at different frequencies and intensities in children.

Rationale and Purpose of Study

Behavioral data have provided occupational therapists much information about children with SPD, beginning with the work of Ayres (1965). This knowledge includes how to recognize these children, what types of intervention to use, how long to provide treatment, and how to measure treatment effectiveness. However, if occupational therapists can demonstrate empirically that children with SPD exhibit brain processing mechanisms—unlike the brain processing observed in children without disorders—this neurophysiological evidence may then be viewed by others

as additional evidence to support the diagnostic category of SPD.

This research study is the initial step to test the assumptions of sensory integration theory by demonstrating that behavioral expressions of dysfunction in children with SPD are related to dysfunction or malfunction in brain processing. Using EEG techniques, we examined the underlying brain mechanisms in children with SPD to further validate the diagnostic category. The specific research question was, "Is there evidence of differences in brain processing of auditory stimuli, as measured by electroencephalography, in children with SPD compared to children who are typically developing?" Also addressed was the second research question, "Can EEG techniques be useful in the diagnosis of SPD?"

Methods

Participants

Fifty-three children, ages 5–12 years, participated in the study. Twenty-eight of these children (22 boys, 6 girls) were classified as having SPD and were referred to the project by several occupational therapists in the community. In most of these cases, the occupational therapy evaluations included the Sensory Profile (Dunn, 1999) and clinical observations; however, other assessments, such as motor skill assessments, may have been used. Based on the assessment results, all of the children were classified as having a modulation dysfunction (Hanft, Miller, & Lane, 2000; McIntosh, Miller, Shyu, & Hagerman, 1999). Five of these children were reported by parents as having a speech delay; 3 had ADHD; 1 had a learning problem; and 6 children were reported to have a combination of ADHD, learning problems, and reading disability in addition to the SPD. These 28 children had no additional reported conditions except as listed previously. The control group, 25 of the children (13 boys, 12 girls), were recruited from an existing database of children who were typically developing, with no known neurological or behavioral disorders.

The mean age of the groups did not significantly differ. The two groups did demonstrate significant differences on 5 of the 7 subsections and on the total score of the Short Sensory Profile (McIntosh, Miller, & Shyu, 1999). See Table 1 for means, standard deviations, and the results of the *t* tests evaluating the differences between the groups on these measures. For the children who were typically developing, the mean scores for each subsection were found to be within the range of "Typical Performance." For the children with SPD, the mean scores for 3 subsections (underresponsive/seeks sensation, auditory filtering, low energy/weak) and total score were found to be within the range of "Definite Difference," and 2 subsections (tactile sensitivity, visual/auditory sensitivity) were within the range of "Probable Difference."

Procedures

Upon volunteering, the parent of each participant was contacted to schedule a visit to the Human Development Lab at the Colorado State University. The parent was mailed an information packet and was asked to fill out the consent forms and the Sensory Profile before visiting the laboratory. At the beginning of the visit, parental consent was confirmed, and testing procedures were reviewed verbally with the child participant. After the child signed a form indicating his or her interest in participating, the child was prepped for EEG recording and given a brief training on strategies he or she could use to help minimize movement and eye-blink artifacts in the recordings. The hearing threshold of the participant was then assessed using a brief click stimulus (3 ms) and a stepping procedure (Levitt, 1971). After threshold screening, two continuous EEG recordings were obtained while the participant completed the sensory gating paradigm and the sensory registration paradigm (see following paragraphs for a description of

Table 1. Results of *t* Test Comparisons of the Two Groups of Children on the Short Sensory Profile

	Children Who Were Typically Developing		Children With Sensory Processing Disorders		Results of *t* Tests		
Variable	*M*	(*SD*)	*M*	(*SD*)	*t* value	*df*	*p* value
Age	8.34	(1.88)	7.71	(1.80)	1.25*	51	.216
Short Sensory Profile Subscale							
Tactile Sensitivity	32.76	(2.38)	26.89	(5.72)	4.97	37.0	< .0001
Taste/Smell Sensitivity	16.60	(4.00)	14.64	(5.17)	1.51*	51.0	.133
Movement Sensitivity	13.64	(1.25)	13.04	(2.27)	1.22	43.0	.230
Underresponsive/Seeks Sensation	27.80	(3.19)	21.57	(5.51)	5.10	44.1	< .0001
Auditory Filtering	24.08	(2.90)	17.21	(4.72)	6.45	45.5	< .0001
Low Energy/Weak	28.40	(2.08)	21.07	(7.42)	5.01	31.7	< .0001
Visual/Auditory Sensitivity	20.96	(2.26)	17.21	(3.78)	4.42	44.9	< .0001
Short Sensory Profile—Total	164.24	(11.48)	131.64	(20.95)	7.12	42.8	< .0001

Note. Short Sensory Profile (McIntosh, Miller, & Shyu, 1999).

*Equality of variances confirmed rather than unequal variance assumed.

each). Each paradigm lasted about 20 min. Breaks of 2- to 3-min duration were taken between the paradigms. The presentation order of two paradigms was counterbalanced except when the child showed signs of short attention span or restlessness during the preparation for EEG (30% of the children with SPD); then the sensory registration paradigm was administered first to ensure that these data would be collected. The entire visit lasted about 1.5 hours.

EEG/ERP Recording Parameters

EEG recordings were collected with a 32-channel BioSemi ActiveTwo EEG/ERP Acquisition System (BioSemi, WG-Plein 129, 1054 SC Amsterdam, Netherlands). Only data from the Cz electrode site, which is at the top of the head in the central position of the midline, were analyzed. Two bipolar electro-oculograms (EOGs) were recorded from electrodes placed on the left and right outer canthus for horizontal movements and on the left supraorbital and infraorbital region for vertical movements. All recordings were made with an A-D sampling rate of 1024 Hz, a gain setting of 1000, and bandwidth of 268 Hz (high passed at 0 Hz and low passed at 268 Hz). The left earlobe was used as the reference for the sensory gating paradigm, and averaged earlobes were used as the reference for the sensory registration paradigm.

Sensory Gating ERP Paradigm and Analyses

Sensory gating (P50) paradigm. The study used a modified sensory gating paradigm, which consisted of presenting a total of 120 pairings of the click sounds while the participants watched a silent movie meant to visually entertain. The click sounds, 3 ms in duration, were presented at approximately 85 dB SPL (decibels sound pressure level). The paired clicks had an interstimulus interval (ISI) of 500 ms and a 10-second duration between pairs.

Measures of sensory gating—P50 component and T/C ratio. Measures of P50 component (see Figure 1) amplitude and latency at the Cz site were obtained by processing the EEG signals using BrainVision Analyzer software (Brain Products GmbH, Zeppelinstrasse 7, 82207 Gilching, Germany, 2002).

EEG processing consisted of (a) digitally filtering using a 10–200 Hz band pass, (b) segmenting the continuous signal into epochs with durations of 100 ms before the click stimulus onset through 200 ms post stimulus, (c) performing artifact rejection where epochs with deviations greater than ±100 μV on any of the EEG channels or the bipolar EOG channels were eliminated, and (d) applying a baseline correction using the 100 ms prestimulus period. Averaged ERP waveforms for the first click (Conditioning) and the second click (Test) were obtained.

P50 amplitude and latency measurements were obtained from each averaged ERP waveform using computer software known as ERPScore (Segalowitz, 1999). The P50 component of the averaged waveform for each click in the pair was identified and scored as the most positive peak between 40 and 85 ms after the stimulus onset. The P30 component, the most positive peak between 25 and 40 ms after the stimulus, also was scored, as was the maximum negativity between the P30 and P50 peaks.

The T/C ratio was then calculated. In the sensory gating paradigm, often the first click is called the Conditioning (C) click because it conditions the brain to be prepared for additional incoming stimuli. The second click is called the Test (T) click because it tests whether the brain prepared for further incoming stimuli. Traditionally, the brain's gating response is reported using the T/C ratio, which is computed by dividing the peak-to-peak amplitude of the P50 component of the Test click (second click) by the peak-to-peak amplitude of the P50 component of the Conditioning click (first click). The peak-to-peak amplitudes are measured as the amplitude of the P50 peak relative to the preceding negativity. Thus, a small value for the T/C ratio represents better sensory gating ability, whereas a larger T/C ratio represents less sensory gating ability.

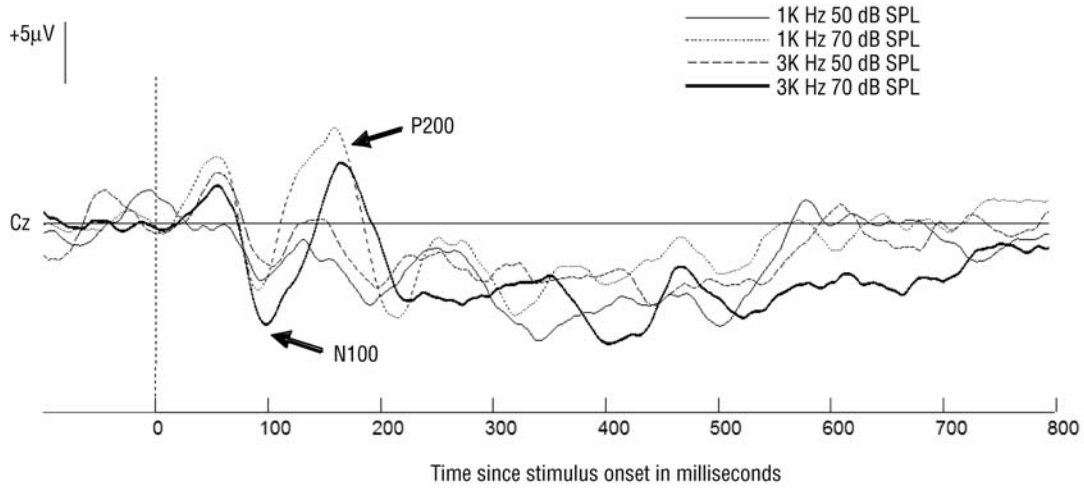
Statistical analyses of the sensory gating P50 component and T/C ratio. Differences in mean peak-to-peak amplitude measures for the P50 component were evaluated using a 2×2 mixed analysis of variance (ANOVA) design. The between factor, Groups, consisted of the two levels, children with SPD and children who were typically developing. The within factor, Clicks, also consisted of two levels, Conditioning click and Test click. Post hoc Tukey *t* tests were then used to compare differences between the two groups at each click. Differences in the T/C ratios between the groups were evaluated using an independent *t* test.

Sensory Registration ERP Paradigm and Analyses

Sensory registration (N100–P200) paradigm. In this paradigm, continuous EEG signals were recorded while the participant stared at a fixed symbol on a computer screen and listened to four different auditory stimuli. The auditory stimuli consisted of pure tones (sinusoidal waves), 2 with frequencies at 1000 Hz and 2 at 3000 Hz, and each frequency was presented at either one of two intensity levels, 50 dB SPL or 70 dB SPL. The stimuli were presented in blocks of 100 trials, 25 trials of each of the stimuli, in random order with a 2-second ISI. Four blocks of trials were presented with each block, taking about 3.5 min. At the conclusion of each block, the participant was given a 30-second break to rest his or her eyes, blink, or move about in the chair.

Measures of sensory registration—N100 and P200 components. The measures obtained from the sensory registration paradigm included the N100 and the P200 (see Figure 2a). Data were processed similarly to the P50 component with the following exceptions: (a) EEG signals were digitally filtered using a .23–30 Hz band pass, and (b) EEG signals were segmented into epochs with durations consisting of 100 ms before the click stimulus onset through 800 ms post stimulus. Averaged ERP waveforms for each of the 4 auditory stimuli were then obtained. Two children who were typically developing and 1 child with SPD had average waveforms based on less than 20 epochs and therefore were excluded from subsequent analysis as will be outlined below. N100 and P200 amplitude and latency measurements were

2a. This brain response of a child from the control group displays similar peak latencies of the early components (i.e., N100 and P200) of the waveforms for each tone. Note that the amplitudes of the loud tones (70 dB) are larger than the amplitudes of the soft tones (50 dB).



2b. This brain response of a child from the group with SPD shows considerable variability in the peak latencies and peak amplitudes of the early components of the waveforms for each tone. These waveforms appear to be more disorganized than those shown in 2a for the child from the control group.

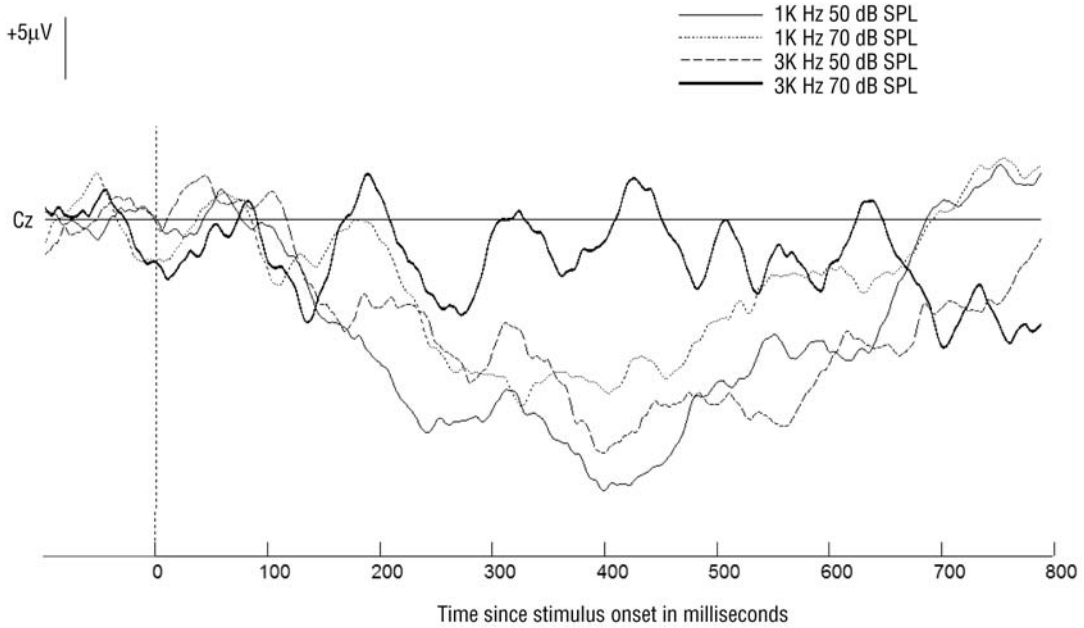


Figure 2. ERP waveforms for two children representing their brain response to the sensory registration paradigm. The averaged brain responses to the presentations of each of the 4 tones are shown as separate lines.

obtained using ERPScore (Segalowitz, 1999). The N100 component was scored as the most negative peak between 80 and 120 ms after the stimulus onset. The P200 component was scored as the most positive peak between 180 and 240 ms after the stimulus.

Statistical analyses of the sensory registration N100 and P200. Differences in mean N100 and P200 amplitude and latency measurements were evaluated using 4 ANOVAs each using a $2 \times 2 \times 2$ mixed ANOVA. The between factor, Groups, consisted of the 2 levels, children with SPD and children who were typically developing. The first within factor, Frequency, consisted of 2 levels, the 1000 Hz and 3000 Hz tones. The second within factor, Intensity, consisted of 2 intensity levels, the soft and loud. Post hoc Tukey *t* tests were used to compare differences between the two groups at each of the 4 auditory stimuli.

Results

The results of the 5 ANOVA procedures, 1 *t* test, and 1 multiple regression analysis are reported below. To guard against Type I errors, the significance of the outcomes were evaluated against a test-wise alpha level of .007, determined by dividing the family-wise alpha level of .05 by the number of statistical procedures performed (.05/7).

Sensory Gating Paradigm

As shown in Figure 1a, a representative child from the control group displayed a large difference between the P50 peak amplitude of the first click (solid line) and the second click (dashed line). However, as seen in Figure 1b, which shows the waveform from a child from the group with SPD, the difference between the amplitudes of the first click (solid line) and the second click (dashed line) is negligible. The 2 × 2 mixed ANOVA revealed only a significant main effect for Clicks, $F_{(1, 51)} = 59.58$, $p < .0005$, partial $\eta^2 = .54$. Post hoc Tukey *t* tests showed that within each group the differences between the Conditioning and the Test clicks were statistically significant, although the effect was stronger for children who were typically developing, $t_{(1, 51)} = 8.71$, $p < .005$, than for children with SPD, $t_{(1, 51)} = 6.61$, $p < .005$. Thus, both groups showed inhibition or gating of the second click. However, analysis of the P50 T/C ratios using an independent *t* test (one-tailed) revealed that, as expected, children with SPD demonstrated a mean of .77 (*SD* = .42), which was higher than the mean T/C ratio of .58 (*SD* = .31) for children who were typically developing, $t_{(1, 51)} = 1.79$, $p = .04$, $\eta^2 = .06$. Although children with SPD as a group showed less gating than children who were typically developing, this difference was not statistically significant when evaluated against an adjusted alpha level of .007.

Sensory Registration Paradigm

Results from the sensory registration ERP paradigm demonstrated that the brain responses of children who were typically developing to changes in the intensity and frequency of the 4 stimuli presented in the ERP paradigm were more organized when compared to the responses of the children with SPD. See Figure 2a for a representative sensory registration waveform for children in the control group and Figure 2b for a representative waveform from the group of children with SPD.

Inspection of the N100 component showed that the mean amplitude of the children who were typically developing was greater for the 1000 Hz stimuli at both intensities, whereas the children with SPD had a greater mean amplitude for the 3000 Hz stimuli at both intensities (see Table 2). The ANOVA revealed a significant main effect for Intensity, $F_{(1, 48)} = 17.97$, $p < .0005$, partial $\eta^2 = .27$. The main effect for Groups was not significant, $F_{(1, 48)} = 1.76$, $p < .68$, partial $\eta^2 = .004$, and post hoc Tukey *t* tests showed that the two groups of children did not significantly differ in N100 amplitudes when each of the auditory stimuli were examined separately. Inspection of the mean N100 latencies showed that the children with SPD had shorter latencies compared to the children who were typically developing. However, the ANOVA procedure revealed that none of the main effects were significant.

Inspection of the amplitudes of the P200 component showed that the mean amplitude of the children who were typically developing was greater for 3 of the 4 auditory stimuli, whereas the children with SPD had a greater mean amplitude for the 3000 Hz tone at the soft intensity. The ANOVA procedure revealed a significant main effect for Intensity, $F_{(1, 48)} = 26.65$, $p < .0005$, partial $\eta^2 = .36$. The main effect for Groups was not significant, $F_{(1, 48)} = .12$, $p = .73$, partial $\eta^2 = .003$, and post hoc Tukey *t* tests showed that the two groups of children did not significantly differ in their P200 amplitudes when each of the auditory stimuli were examined separately. Inspection of the mean P200 latencies showed that the children with SPD had longer latencies compared to the children who were typically developing for all but the 3000 Hz tone at the loud intensity. However, the ANOVA procedure revealed that none of the main effects were significant, and post hoc comparisons showed that no significant differences between groups existed for any of the stimuli.

Exploring Individual Differences in Sensory Gating

To address possible factors accounting for the variability observed in the ERP P50 gating T/C ratios within and between groups, we proposed a model for sensory gating

Table 2. Mean Amplitude and Latencies of the N100 and P200 ERP Components in the Sensory Registration Paradigm

	Auditory Stimuli			
	1000 Hz @ 50 dB	1000 Hz @ 70 dB	3000 Hz @ 50 dB	3000 Hz @ 70 dB
N100 Amplitudes (μV)				
Children Who Were Typically Developing	7.46 (3.72)	9.39 (4.19)	7.60 (4.38)	8.64 (4.75)
Children With SPD	5.80 (3.70)	8.38 (3.76)	7.63 (4.21)	9.59 (4.77)
P200 Amplitudes (μV)				
Children Who Were Typically Developing	7.04 (4.60)	10.77 (8.75)	6.37 (4.58)	11.05 (8.04)
Children With SPD	6.19 (3.49)	10.37 (8.10)	7.18 (4.38)	10.35 (6.92)
N100 Latencies (ms)				
Children Who Were Typically Developing	119.13 (30.92)	119.79 (25.37)	131.94 (35.94)	111.17 (19.80)
Children With SPD	128.85 (32.63)	133.27 (38.64)	130.22 (33.11)	128.09 (29.86)
P200 Latencies (ms)				
Children Who Were Typically Developing	178.43 (35.80)	180.79 (33.15)	182.55 (37.25)	168.13 (21.68)
Children With SPD	178.61 (40.94)	184.92 (40.20)	187.28 (40.92)	181.03 (27.50)

Note. ERP = event-related potentials; dB = decibels; μV = microvolts; ms = milliseconds; SPD = sensory processing disorders.

that consisted of two principal components, maturation and organization of brain responses to auditory stimuli. The maturation component was simply defined as the participant's chronological age. Based on previously reported research (Marshall et al., 2004), the expected relationship was that older children would show smaller sensory gating T/C ratios than younger children. Organization of brain response to auditory stimuli was defined as the participant's interrelationship of the peak-to-peak amplitudes and latencies of the N100 and P200 components of the average waveforms for the two loud intensity auditory stimuli of the sensory registration paradigm. The operational assumption here was that the less variable these components were within an individual, the smaller the individual's sensory gating T/C ratio would be. This model was tested using a 3-step multiple regression procedure. The predicted dependent variable was the P50 T/C ratio from the sensory gating paradigm. The predictors—the independent variables—were age entered in the first step; the N100 amplitudes and latencies of the two loud intensity auditory stimuli of the sensory registration paradigm were entered in the second step; and the P200 amplitudes and latencies of the two loud intensity auditory stimuli were entered last. When this analysis was performed with data from both child groups, a nonsignificant relationship was found, R^2 = .32 (Adj. R^2 = .17), $F_{(9, 40)} = 2.11$, $p = .051$.

However, when the regression analysis was performed using data just from the children who were typically developing, a statistically significant relationship between the variables was found, R^2 = .84 (Adj. R^2 = .72), $F_{(9, 13)} = 7.41$, $p = .001$. Age accounted for 32% of the variance (F Change$_{(1, 21)} = 9.97$, $p = .005$), N100 amplitudes and latencies accounted for 49% (F Change$_{(4, 17)} = 10.82$, p = .0002), and P200 amplitudes and latencies accounted for 3% (F Change$_{(4, 13)} = .56$, $p = .70$). Thus, only for the children who were typically developing, the 3-step regression analysis revealed that the proposed model—consisting of measures of maturation and brain processing of simple auditory stimuli presented in the sensory registration paradigm—could account for a very large percent (84%) of the variability in the P50 T/C ratios observed in sensory gating paradigm. When the regression analysis was performed using data just from the children with SPD, no relationship between the dependent and the independent variables was found, R^2 = .35 (Adj. R^2 = .002), $F_{(9, 17)} = 1.0$, $p = .47$. Interestingly, in contrast to the children who were typically developing, age accounted for only 1.4% of the variance in the children with SPD, F Change$_{(1, 25)} = .35$, $p = .56$.

This discrepancy between the maturation effects observed in children who were typically developing and the failure to find maturation effects in children with SPD might be better understood by examining the zero order correlations between age and T/C ratios of the P50 component for each group separately. For the children who were typically developing, a statistically significant relationship was found between age and the T/C ratios of the P50, r = –.60, $p = .001$. Thus, the evidence suggests that for the children who were typically developing, sensory gating improves (i.e., T/C ratios become smaller) as a function of being older in age. However, children with SPD did not provide evidence of a relationship between age and the T/C ratios of the P50 as r = –.08, $p = .67$. Thus, children with SPD do not demonstrate improved sensory gating as a function of being older.

Diagnosing Children With SPD

To understand how brain processing in children with SPD differs from the brain processing in children who are typically developing, we further examined the relationship between sensory gating and sensory registration measures. First, we derived a prediction equation for P50 T/C ratios, using the unstandardized coefficients obtained for each of

the variables in the regression analysis performed on children who were typically developing, as reported previously. Using this equation, predicted P50 T/C ratios were calculated for each child in both groups. The predicted ratios were then subtracted from their actual P50 T/C ratios obtained in the sensory gating paradigm to produce a difference score for each child. These difference scores were then plotted as a function of their corresponding obtained T/C ratios. As seen in Figure 3, difference scores of the children with SPD are distributed on either side of the children who were typically developing. Thus, this procedure demonstrates that when P50 T/C difference scores are derived from the prediction equation based on the regression analysis of the children who were typically developing, the children with SPD can be shown to be hyperresponsive or hyporesponsive in their sensory gating when compared to the gating of the children who were typically developing.

Discussion

To our knowledge, this study is the first to examine brain processing in children identified by occupational therapists as having SPD and to compare the results to brain processing observed in children without disorders. We explored two research questions. The first addressed whether the groups differed in EEG measures of brain processing of auditory stimuli. The second explored whether electroencephalographic techniques would be useful in diagnosing SPD. Our results revealed that, as a group, children with SPD demonstrated less auditory sensory gating than an age-matched peer group of children without disorders ($p = .04$), although not significantly different when evaluated against the adjusted alpha level. Furthermore, children with SPD did not show a significant relationship between sensory gating and age, although such a relationship was found in children who were typically developing. Finally,

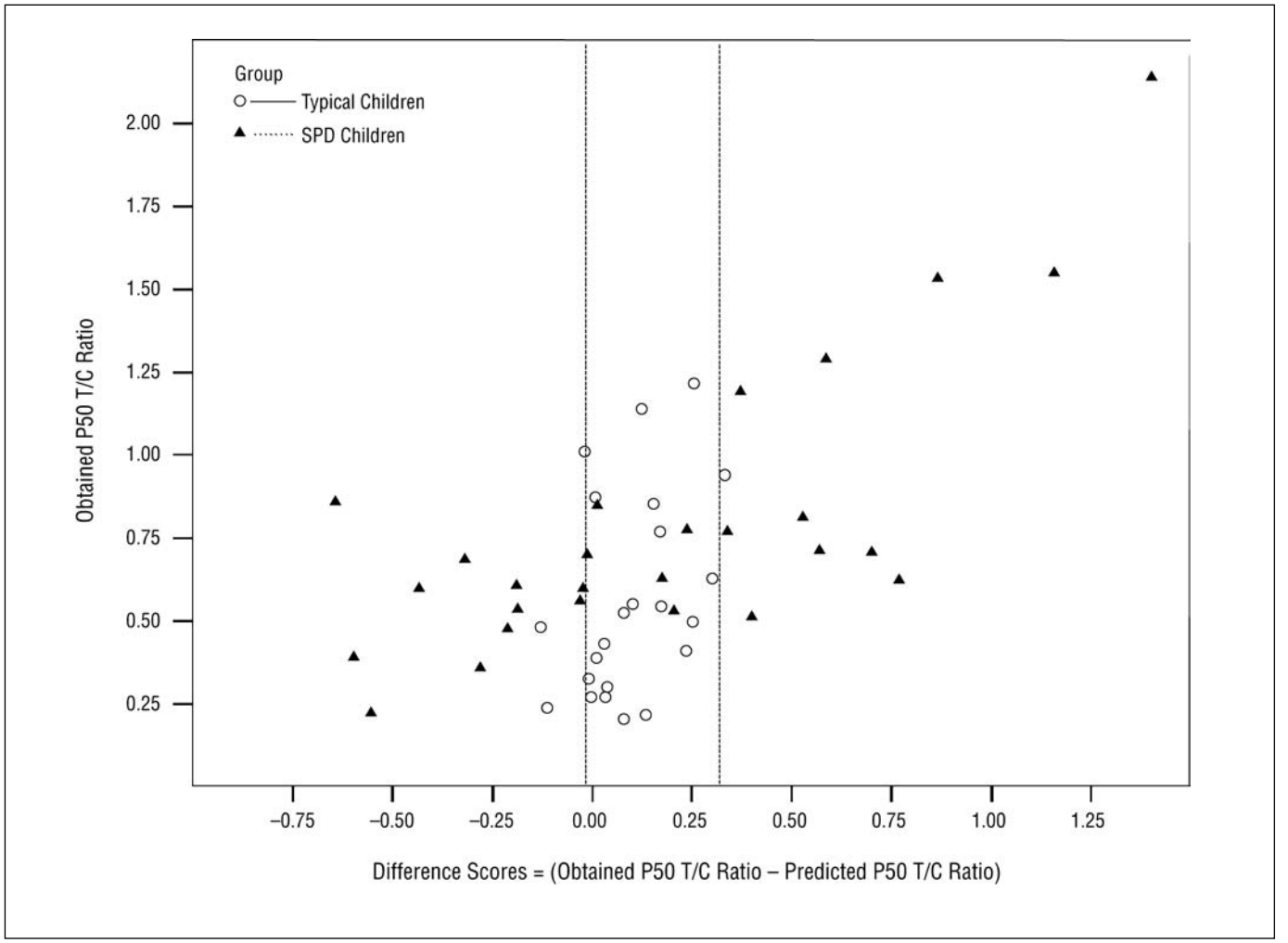


Figure 3. Illustration of the results of the statistical method used to separate the children with SPD from children in the control group (children who were typically developing) based on brain responses to auditory stimuli. Obtained P50 T/C ratios of each child are plotted as a function of the difference between the obtained ratios and the predicted ratios.

we demonstrated that differences between actual sensory gating measures and predicted sensory gating—based on an equation using age and measures of brain processing to simple auditory stimuli—can be used to diagnosis SPD. In the following paragraphs we discuss how the results from this study support the assumption of sensory integration theory that behavioral expressions of dysfunction in sensory integration are related to immaturity or malfunction in brain processing.

Occupational therapists referred children to this study on the basis of their clinical judgment that the children had SPD. One of the purposes of this study was to externally validate that children who are seen by occupational therapists for SPD are indeed a distinct group of children, different from their age-matched peers in the general population. The degree to which these recruitment strategies produced sample groups representative of their respective populations was validated in this study by the scores on the Short Sensory Profile (McIntosh, Miller, & Shyu, 1999) and also by EEG measures.

The results of the Short Sensory Profile data collected in our laboratory for both groups of children confirm that parents of children with SPD reported observing aberrant behaviors, whereas parents of children who were typically developing did not. Although not novel, this independent behavioral data collected in our laboratory provides validity that the children often classified as having SPD by therapists are significantly different from their age-matched peers based on family observations alone. The unique contribution of this study is the fact that electroencephalographic measures from the sensory gating paradigm also showed differences between the two groups of children.

Specifically, the mean T/C ratio for the children with SPD was found to be larger than the mean T/C ratio of a peer group of children without disorders. Because larger T/C ratios represent less sensory gating ability compared to smaller T/C ratios, this finding may be interpreted that children with SPD are deficient in their ability to suppress (i.e., filter out) repeated or irrelevant sensory input and fail to selectively regulate the sensitivity of cortical responses to additional incoming sensory stimuli. The decreased ability of children with SPD to gate out or suppress irrelevant auditory stimuli, as shown in this study, may explain certain behavioral manifestations such as distraction, impulsiveness, abnormal activity level, disorganization, anxiety, and emotional liability, often observed in children with SPD (Cohn, Miller, & Tickle-Degnen, 2000; Miller, Reisman, McIntosh, & Simon, 2001). When a child is unable to automatically suppress incoming sensory information, he or she may become inundated with the incoming information. The child's response to inundation may be expressed behaviorally either by "acting out" or by withdrawing when sensory stimulation in the environment becomes too overwhelming.

Differences between the two groups also were observed regarding evidence of maturation of sensory gating abilities. Maturation was found to be one of the factors accounting for the variability in the T/C ratio measures in the children who were typically developing but not in the children with SPD. In our analyses for developmental trends, the age of the child was used as a variable representing the accumulative time for maturation of brain processes. Our results therefore suggest that, as a group, the auditory gating abilities of children with SPD do not change as a function of either biologically driven maturity (e.g., physical growth) or the accumulation of experiences across time (e.g., learning). The children in our typically developing group did show a significant relationship between sensory gating and age; thus, we infer that sensory gating improves as children without disorders mature. This finding is consistent with several other studies that reported sensory gating in children who were typically developing (Freedman et al., 1987; Marshall et al., 2004). Further investigations on maturation of sensory gating should be conducted to more fully determine the developmental trajectory.

Additional support for the validity of a diagnosis category of SPD was discovered when we explored the relationship of the children's brain responses to basic auditory stimulation to their sensory gating abilities. Multiple regression analyses revealed that age and brain responses to simple auditory stimuli presented in the sensory registration paradigm (i.e., the N100 and P200 ERP components) could account for 84% of the variance in the P50 T/C ratios of the sensory gating paradigm of the children without disorders. In contrast, the children with SPD were found to be more variable in their responses to simple auditory stimuli and, as a result, predicting sensory gating from their brain responses to simple auditory stimuli was considerably less reliable. This finding further contributes to the inference that the brain processing of simple auditory stimuli may be less organized in the children with SPD. On a behavioral note, if a child's brain is not able to organize simple sensory stimuli, it is unlikely that the child will be able to organize more complex incoming sensory input in a manner that results in functional adaptations or responses.

Results from this study support the validity of sensory processing disorders as a diagnosis by providing evidence of differences in brain processing of auditory stimuli, as measured by EEG, in children with SPD compared to children who were typically developing. When assessed from a development perspective, ERP measures of brain processing ability—such as the P50 T/C ratios from the sensory gating

paradigm, along with N100 and P200 from the sensory registration paradigm—may be used as a possible marker of SPD. However, because this is the first study of its kind, further studies of brain processing are needed to confirm the P50 gating deficits obtained in this study. Such studies also should explore the existence of other possible EEG/ERP markers of sensory processing deficits that may be used in the future to cross-validate the diagnosis of SPD in individual children.

Demonstrating that differences in brain processing exist between groups of children with and without SPD should be considered only the first step in establishing the validity of new clinical markers of the disorder. A more powerful demonstration of the validity of a new marker is to show the degree to which the marker may be used to successfully diagnose an *individual* as having or not having the disorder. This goal was the basis of our second research question, "Can EEG techniques be useful in the diagnosis of SPD?" Our approach to answering this question was to demonstrate that, by using a prediction equation based on the brain responses of the children who were typically developing, we could determine with 86% accuracy the group membership of *each* child in both child groups. That is, we could determine by just the ERP responses whether a given child had a sensory processing disorder. Furthermore, the children with SPD also were shown to be either hyperresponsive or hyporesponsive in their sensory gating relative to the gating of the children who were typically developing. This result is congruent with the clinical observations of therapists, who often describe children with SPD as being either hyposensitive or hypersensitive to their environment (Hanft et al., 2000; McIntosh, Miller, Shyu, & Hagerman, 1999) and further strengthens the validity of the clinical diagnosis.

Our study found that ERP measures of the central nervous system show that some children with SPD may be hyperresponsive and others may be hyporesponsive to auditory stimuli. These findings corroborate the results of McIntosh and colleagues (1999), who used a peripheral nervous system measure, electrodermal response (EDR) (McIntosh, Miller, Shyu, & Hagerman, 1999). In the McIntosh et al. study, the group labeled as *hyperresponsive* children showed more EDR responses that had greater magnitude and habituated more slowly than a control group. In contrast, children in another group labeled as *nonresponders* showed significantly smaller magnitude EDR responses than the control group. Our data show similar results to those of McIntosh et al. in that hyperresponsiveness and hyporesponsiveness represent a continuum (see Figure 3). This finding is consistent with some ideas expressed in earlier reports (Fisher & Murray, 1991; Royeen & Lane, 1991; Williams & Shellenberger, 1994). However, Lane (2002) suggested that describing these behavioral responses along a continuum may be too simplistic. Further exploration of the factors that account for the variance in sensory gating in children with SPD is needed to clarify this issue.

The data depicted in Figure 3 highlight another important aspect regarding the children who were classified as having SPD; namely, that these children are qualitatively different from their peer group of children who were typically developing. If children with SPD were merely quantitatively different, their T/C ratios would appear only as extreme points of the normal distribution positioned at the extreme, in one or the other tails, of the normal distribution. This was not the case. Furthermore, if children with SPD were merely quantitatively different, the prediction equation used for children who were typically developing also should have applied to the children with SPD. This was found not to be the case. Rather, our regression results suggest that the children referred for this study as having SPD displayed brain processing mechanisms that were deviant from the brain processing mechanisms seen in the children who were typically developing. The linear pattern of the difference scores observed in Figure 3 for the children with SPD suggests that one or more additional variables—not in the prediction equation for children who were typically developing—may exist and may account for the unique variability observed in the gating performance in children with SPD. Because no such linear pattern is seen in the difference scores for the children who were typically developing, the additional variable or variables that explain the unique pattern of variability in children with SPD represents a qualitative difference between the two groups. Further study is needed to determine the nature of the variable or variables that place children with SPD on both extremes of children who are typically developing. These variables may be behavioral in nature (e.g., parent report of child's behavior on the Sensory Profile) or perhaps more biological (e.g., family history of psychiatric disorders).

Pennington (2002) suggested that most disorders are first defined behaviorally or based on a set of symptoms defining a phenotype of a disorder. Occupational therapists, beginning with Ayres, have engaged in research (for review, see Mulligan, 2002) that helped define phenotypes of dysfunction for sensory integration and SPD. Their studies of sensory integration and SPD have helped define the behavioral phenotype. Pennington (1997) advised that, although genetic and brain studies of disorders cannot progress without carefully designed behavioral and neuropsychological phenotypes, brain and genetic studies can force revisions in the definition of phenotypes, refining both the definition and the diagnostic process. With technologies currently available and the behavioral phenotypes that have been

described by therapists, the time seems appropriate to examine brain and genetic factors that may help explain the behavioral phenotypes of SPD. If, through careful research, occupational therapists and their colleagues can demonstrate a refined definition of SPD that includes evidence of brain and genetic factors along with traditional behavioral definitions, the diagnosis and need for therapeutic interventions will be further substantiated.

The results of the present study illustrate how studies of brain processing may refine the definition of SPD. The measures of brain processing we used were the P50 from the sensory gating paradigm and the N100 and P200 components from the sensory registration paradigm. Because components in auditory ERP waveforms occurring after 25–30 ms post stimulus are shown to be generated in the cortex (Celesia & Brigell, 1999), the P50, N100, and P200 components are believed to be measures reflecting activity at the cortical level rather than at the subcortical level. Thus, the results of the present study suggest that processing of auditory stimuli at a cortical level is different in children with SPD when compared to children who are typically developing. Consequently, when explaining sensory integration theory, we may no longer want to describe the disorder as being only a subcortical problem; rather, SPD may involve processing difficulties in both subcortical and cortical brain regions. However, because this study measured processing only in the cortex, future studies are needed to assess processing in subcortical brain regions, as well as other processing mechanisms in cortical brain regions, to more fully define the underlying brain mechanisms of SPD.

In conclusion, our results support the assumption of the sensory integration theory that neural processing mechanisms are different in children with SPD when compared to age-matched peers who are typically developing. In addition, we demonstrated that brain activity as measured in two ERP paradigms could be used to correctly classify children with SPD and distinguish them from a group of children who were typically developing with 86% accuracy. These data provide empirical evidence that children seen by occupational therapists for intervention of SPD can be distinguished from children who are typically developing on the basis of neural or brain processing mechanisms alone. These neurophysiological findings provide evidence to support the validity of the diagnostic category of SPD, which before this study had been defined primarily through behavioral measures. ▲

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