Sensory Overresponsivity and Anxiety in Typically Developing Children and Children With Autism and Attention Deficit Hyperactivity Disorder: Cause or Coexistence?

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KEY WORDS
- anxiety
- attention deficit disorder with hyperactivity
- autistic disorder
- sensation disorders

OBJECTIVE. To explore the relationship between sensory overresponsivity (SOR) and anxiety in children with autism, attention deficit hyperactivity disorder, and typical development.

METHOD. Path analysis was used to examine the primary SOR model (Green & Ben-Sasson, 2010) using both physiological and behavioral data.

RESULTS. The magnitude of physiological responses to sensory challenge was a mediator variable between predictors (baseline arousal and attention) and outcomes (anxiety and physiological recovery). Behavioral SOR was correlated with anxiety but not with physiological variables.

CONCLUSION. The intensity or magnitude of sensory responsivity mediates the relationship between baseline arousal and attention and outcome anxiety and physiologic recovery from sensory challenge. Behavioral tools used to measure SOR do not reflect physiological responsiveness; this mismatch warrants further investigation. SOR can prevent children from participating in the occupations of childhood; the greater the understanding of SOR, the more successful occupational therapy practitioners will be in developing effective interventions.

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Anxiety and sensory overresponsivity (SOR) coexist within many neurodevelopmental disorders, influencing participation and occupational performance (Ben-Sasson, Carter, & Briggs-Gowan, 2009; Pfeiffer, Kinnealey, Reed, & Herzberg, 2005; Reynolds, Lane, & Gennings, 2010). The relationship between anxiety and SOR has rarely been addressed, although recently Green and Ben-Sasson (2010) presented both causal and noncausal models to explain the development and maintenance of these conditions. Their theoretical article focused on children with autistic spectrum disorders (ASD), but the concepts have the potential for broader application.

Core features of ASD are well established and include impairments in social interaction and communication and the presence of repetitive behaviors. In addition to these core diagnostic features, atypical sensory processing has been reported in up to 95% of children with ASD (Baranek, David, Poe, Stone, & Watson, 2006; Leekam, Nieto, Libby, Wing, & Gould, 2007; Tomchek & Dunn, 2007). Such behaviors may take the form of sensory underresponsivity, sensory seeking, or sensory avoiding and sensory sensitivity. Although moderated by age, sensory underresponsivity has been reported to be common in ASD relative to comparison groups, followed by SOR and sensory seeking (Ben-Sasson et al., 2009). Sensory processing disorders have also been linked with attentional disorders in children with ASD; Liss, Saulnier, Fein, and

Kinsbourne (2006) identified a cluster of children with ASD, SOR, and attentional overfocusing and a cluster with ASD, sensory underresponsivity, and sensory seeking.

Coupled with these hallmark signs of ASD are comorbidities, among which are anxiety disorders. Reviews of the coexistence of ASD and anxiety disorders have concluded that among children and youth with ASD, anxiety disorders are highly prevalent, clinically significant, and varied as to specific type of anxiety disorder (MacNeil, Lopes, & Minnes, 2009; White, Oswald, Ollendick, & Scahill, 2009). Pfeiffer and colleagues (2005) identified a link between sensory sensitivity and anxiety in children and teens with Asperger syndrome, a finding that was paralleled by a population study of Japanese children (Tsuji et al., 2009). Recent work by Joosten and Bundy (2010) suggested that children with ASD and intellectual disability demonstrated SOR that likely contributed to increased anxiety. This provides an interesting backdrop for understanding the potential link between ASD and anxiety, but the populations investigated by these two groups of researchers represent ends of the ASD spectrum. Thus, the link between anxiety and SOR for children with ASD continues to require further examination.

Although the diagnostic group of ASD remains of prominent interest to researchers and clinicians alike, the coexistence of and potential relationship between anxiety and sensory processing disorders is also of interest in children with other diagnoses. Approximately 25% of children with attention deficit hyperactivity disorder (ADHD) also have anxiety disorder, a rate that is elevated when ADHD is seen in conjunction with conduct or oppositional defiant disorders (Abikoff, 2002; Jensen et al., 2001; Pliszka, 2003; Schatz & Rostain, 2006). Moreover, a subgroup of children with ADHD can be identified as having comorbid SOR with elevated levels of anxiety (Reynolds & Lane, 2009). Behaviorally, children with ADHD have been reported to have difficulties in all areas of sensory modulation (sensory overresponsiveness, sensory underresponsiveness, sensory seeking) and in their emotional responses to sensation (Dunn & Bennett, 2002; Kalpogianni, 2002; Lane, Reynolds, & Thacker, 2010; Yochman, Parush, & Ornoy, 2004). Physiologically, children with ADHD may demonstrate elevated levels of electrodermal response to the initial presentation of sensory stimuli (orienting response; Mangeot et al., 2001) during the Sensory Challenge Protocol (SCP; Miller, Reisman, McIntosh, & Simon, 2001), and children with ADHD with and without tactile overresponsivity have shown different electroencephalogram patterns in response to touch (Parush, Sohmer, Steinberg, & Kaitz, 2007).

A link between anxiety and SOR, possibly related to faulty information processing, has previously been proposed (Ayres, 1972; Johnson, 1975). In seeking a better understanding of this relationship, Green and Ben-Sasson (2010) proposed three models worthy of further study: (1) anxiety caused by, or a symptom of, SOR (primary anxiety model); (2) SOR caused by, or a symptom of, anxiety (primary SOR model); and (3) the presence of both anxiety and SOR, linked by way of another factor (noncausal model). Work from our lab suggests that physiological (electrodermal activity [EDA]) and neuroendocrine (salivary cortisol) responses to challenging sensory stimulation may have a modulatory role in the development and/or perpetuation of anxiety-related behavior (Reynolds et al., 2010; Lane et al., 2010). Therefore, we tested the primary SOR model using physiological and neuroendocrine data from our laboratory. Although this model was originally proposed for children with ASD, we used data collected on children for whom the SOR–anxiety link has been proposed: children with ASD, children with ADHD, and typically developing children (TYP).

Method

Participants

The Virginia Commonwealth University Institutional Review Board approved all investigations in the Sensory Processing and Stress Evaluation (SPASE) Lab before participant recruitment began. Data for the current study are from participants in two separate studies in the SPASE Lab. Convenience sampling provided participants ages 6–10 yr in one of three groups: ASD, ADHD, or TYP. Children with psychological diagnoses other than ADHD and ASD, significant motor impairments such as cerebral palsy, or any known endocrine or metabolic dysfunctions were excluded. We screened all children for intelligence using a two-subtest battery of the Wechsler Abbreviated Scale of Intelligence (Psychological Corporation, 1999); children needed IQ ≥70 to be included. We conducted phone interviews with parents to verify diagnoses and ensure that children met the inclusion criteria. We asked parents of children with ASD to provide a copy of documentation verifying ASD diagnosis (i.e., Autism Diagnostic Interview [Lord et al., 2000] or the Autism Diagnostic Observation Schedule [Lord, Rutter, & Le Couteur, 1994] results). We recruited children with a diagnosis of ADHD under the guidance of the university's division chair in the Department of Psychiatry, who confirmed the diagnosis of ADHD through either formal assessment or interview.

Procedures

Procedures followed for all participants have been previously reported (Reynolds et al., 2010; Reynolds, Lane,

& Thacker, 2011). Following telephone screening, we mailed assessment measures, a child information form, and consent forms to parents. We performed the IQ screening before the physiological testing; no child scored <70 on the screening, and so all children were included in the study.

Following the IQ screening, we introduced the children to the SPASE lab, explained the procedures, applied electrodes, and collected baseline saliva samples. Children watched a 6-min clip from the movie *Apollo 13* (Grazer & Howard, 1995) to allow time for the electrodes to accommodate to the skin. Immediately following the movie clip, we took the second baseline sample of cortisol. We then told the child that he or she would be preparing for a spaceship trip.

A detailed description of the entire SCP is available in Miller et al. (2001); our lab procedures are described in Reynolds et al. (2010). We collected electrodermal activity and electrodermal responsivity (EDR) measures throughout the SCP. Following administration of the SCP, which took approximately 20 min, we collected seven additional samples of saliva at 5-min intervals. During the postchallenge sampling, children rested and watched a silent cartoon. The final cortisol sample, taken at 30 min following completion of the SCP, reflected a return to baseline in the current analyses.

Measures

Sensory Profile. The Sensory Profile (Dunn, 1999) is a 125-item parent-report questionnaire designed to examine a child's behavioral responses to environmental sensation. The Sensory Profile was normed on a sample of 1,037 children without disabilities ages 3–10 yr representing four major regions of the United States. The original scoring identified deficits in sensory systems (auditory, visual, tactile) and general responsiveness to sensory input, along with the estimated factor scores. Internal consistency examination produced Cronbach's α ranges for the sensory processing categories of .64 (Multisensory Processing)–.86 (Touch Processing); Cronbach's α for the factors ranged from .72 (Fine Motor/Perceptual) through .92 (Emotionally Reactive; Dunn, 1999). Construct validity between sensory processing and the School Function Assessment (SFA; Coster, Deeney, Haltiwanger, & Haley, 1998), Behavioral Regulation section, was determined to be moderate, with *r* values ranging from −.10 (Behavior Regulation/Assistance and Touch Processing) to .67 (Behavioral Regulation/Assistance and Visual Processing); somewhat stronger correlations were documented between factor scores and this same SFA section, ranging from −.20 (Behavioral Regulation/Adaptations and Oral Sensory Sensitivity) to −.72 (Behavioral Regulation/Assistance and Low Endurance/Tone). Correlations are negative because lower SFA scores but higher Sensory Profile scores indicate more typical performance.

Additional analyses led to the identification of sensory processing quadrants, considered to reflect the interaction between a nervous system threshold continuum and a self-regulation–responsiveness continuum. Cronbach's α for quadrant groupings ranged from .87 to .93 (Dunn, 2006). Although Dunn (2006) identified four quadrants, we used scores for Sensory Sensitivity and Sensory Avoiding to reflect sensory overresponsivity in the current study. Parents of children with ASD and some typically developing children completed the Sensory Profile.

Sensory Overresponsiveness Inventory. The Sensory Overresponsiveness (SensOR) Inventory (Schoen, Miller, & Green, 2008) is the only available caregiver report tool that specifically identifies people with SOR. Version 1.4, used in the current study, is a 76-item questionnaire on which parents indicate with *yes* (1) or *no* (0) whether various stimuli bother their child. Stimuli items reflect responsiveness to the tactile, auditory, visual, movement, taste, and smell environments. For this study, we calculated a total SensOR score to reflect overall SOR, parallel to the sensory sensitivity factor from the Sensory Profile. Internal consistency reliability for the SensOR Inventory is reported to be high within each domain (Cronbach's α = .65–.88); the SensOR Inventory was shown to have strong discriminant validity, distinguishing between people with and without SOR within each domain ($p < .05$ to $p < .001$; Schoen et al., 2008) and having strong concurrent validity with the overresponsive scales on the Short Sensory Profile ($r = .47$, $p < .01$; McIntosh, Miller, Shyu, & Dunn, 1999).

Revised Children's Manifest Anxiety Scale. The Revised Children's Manifest Anxiety Scale (RCMAS) is a 37-item self-report tool used to measure anxiety in children ages 6–19 yr (Reynolds & Richmond, 2005). It includes 28 items that measure traits related to anxiety and 9 items that comprise a Lie Scale or social desirability score (e.g., "I am *always* good"). A RCMAS Total Anxiety (RTA) score and subscale scores (physiological anxiety, worry/oversensitivity, and social concern/concentration) are calculated; we used only the RTA score in the current analysis.

The RCMAS has been shown to have high internal consistency reliability (Cronbach's α = .89 for Total score). Construct validity was established through strong correlations with scores from the State–Trait Anxiety Inventory for Children ($r = .88$), the Multidimensional Anxiety Scale for Children ($r = .76$), the Screen for Child Anxiety Related Emotional Disorders ($r = .76$), and the Spence Children's Anxiety Scale ($r = .76$; Muris, Merckelbach, Ollendick, King, & Bogie, 2002). High Lie Scale scores may reflect inaccurate self-report, idealized

sense of self, or inattention during the questionnaire process, providing a safeguard for child report. No children in the current study scored above 13 on the Lie Scale, and all RCMAS scores were considered valid for analysis. Higher scores on the RCMAS indicate greater levels of anxiety.

Electrodermal Response Measures. A measure of eccrine sweat gland activity, EDRs reflect sympathetic nervous system (SNS) responses to sensory stimuli. We assessed EDRs during a 3-min baseline, throughout the SCP, and during a 3-min recovery. Collection of EDRs followed the procedures recommended by Fowles and colleagues (1981) and previously reported for the SPASE Lab (Lane et al., 2010).

Several electrodermal measurement parameters have been used to assess tonic and phasic sympathetic activity and response to sensation, both of interest in the current study. In this study, tonic EDA, or skin conductance level (SCL), was averaged across a 3-min baseline (SCLbase) and during a 3-min recovery (SCLrec), providing a measure of baseline arousal and arousal level during recovery. The phasic, or reactivity, variable used was mean response magnitude across all domains. As is typical in studies evaluating magnitude of skin conductance responses, our magnitude data were positively skewed and required logarithmic transformation before analysis (Boucsein, 1992; Dawson, Schell, & Filion, 1990).

Salivary Cortisol. The children were instructed not to eat, drink, chew gum, or brush their teeth for 30 min before coming to the lab. In addition, parents were asked to withhold their child's ADHD medication (if applicable) for 24 hr before testing to eliminate any potential neurochemical interactions and interparticipant variation. We collected saliva as previously reported (Reynolds et al., 2010). Changes in cortisol take approximately 5 min to register and approximately 15–20 min to peak in human saliva (Hiramatsu, 1981; Schmidt, 1998). The entire SCP takes approximately 20 min to complete, making the initial cortisol response to the SCP (Cortbase) expected immediately following SCP completion and a return to tonic level expected by 30 min post SCP.

Analysis

We examined an adaptation of the primary SOR model proposed by Green and Ben-Sasson (2010). This model suggests that SOR becomes linked with specific objects and events and may result in specific phobias or avoidance, hypervigilance, hyperarousal, and subsequent generalized anxiety because of the unpredictability and uncontrollability of the sensory input. In modeling these parameters, we gauged SOR through the use of parent-report tools, the Sensory Profile, or the SensOR Inventory; scores were normalized by converting all to standard scores to represent a single SOR variable. We used SCLbase and Cortbase to reflect tonic arousal at the start of the SCP, average response magnitude (SRMag) to represent response to sensory challenge, and SCLrec to reflect a return to baseline arousal. RTA served as a measure of generalized anxiety.

We used path analysis (Wright, 1921) to test the mediation model, graphically represented in Figure 1. Path analysis is used to describe the directed relationships between a set of observed variables. General linear models (e.g., regression analysis, canonical correlation analysis, analysis of variance, multivariate analysis of covariance) can be represented as path analysis. Path analysis may also be considered as a submodel of structural equation models in which latent variables are used in the analysis. All model parameters are tested simultaneously in path analysis. Path analysis is well suited to test mediation models in which the effect of X (predictor) on Y (outcome) depends on the value of Z (mediator; MacKinnon, 2008).

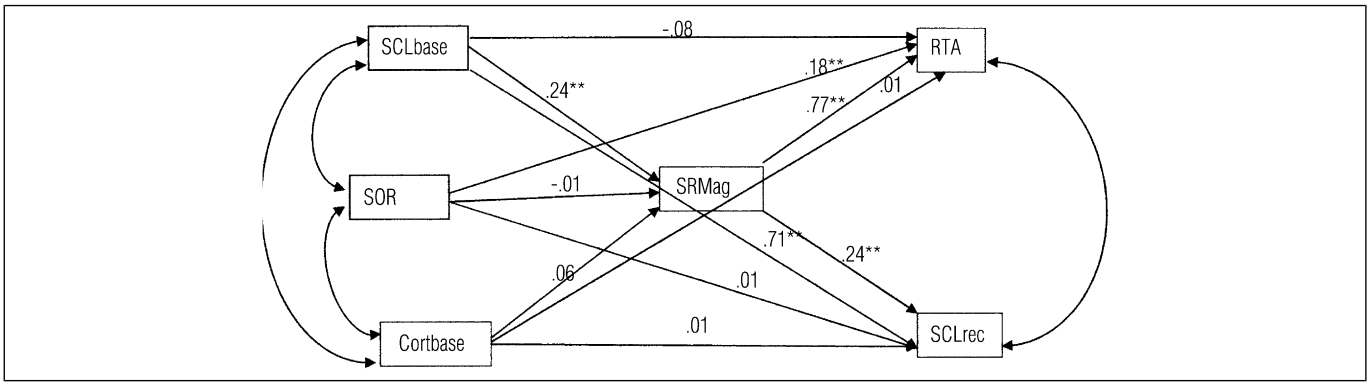

Figure 1. Sensory responsivity mediates between baseline and outcome measures.

Cortbase = baseline cortisol; RTA = Total Anxiety score on the Revised Children's Manifest Anxiety Scale; SCLbase = baseline skin conductance level; SCLrec = skin conductance level during recovery; SOR = sensory overresponsivity; SRMag = mean magnitude of the specific response.

We investigated the hypothesis that the SRMag mediates the relationship between predictors (SCLbase, Cortbase, SOR) and outcomes (RTA and SCLrec). The total effect of a predictor and an outcome is the sum of the direct effect of predictor on outcome (e.g., SCLbase → RTA) plus the indirect effect of the predictor on outcome through the mediator (SCLbase → SRMag → RTA). This later, indirect effect represents the mediation pathway. Thus, in addition to estimating and testing the path coefficients in Figure 1, we tested the statistical significance of specific mediational pathways. We used the full-information maximum likelihood method to estimate the model, which uses all available data without inserting values for each missing datum (imputation). We used the Mplus program (Muthén & Muthén, 1998–2010) to estimate the mediational model.

Results

Demographic data for the children are shown in Table 1. The mediational model converged properly. Standardized parameter estimates appear in Figure 1. The SRMag fully mediates the relationship between SCLbase and RTA. That is, the relationship between SCLbase and RTA becomes null after controlling for SRMag. The relationship between SCLbase and SCLrec is partially mediated; that is, the effect of SCLbase on SCLrec remains significant after taking into account the fact that SRMag is a significant mediator of the effect of SCLbase on SCLrec. Because the effects of both Cortbase and SOR on the SRMag are not significant, the SRMag does not mediate the effect of Cortbase and SOR on the two outcome variables (RTA and SCLrec). The direct effect of SOR on RTA was significant. Significant indirect effects (pathways) are given in Table 2.

Discussion

Children with SOR demonstrate defensive or exaggerated avoidant responses to everyday sensations that people with more typical sensory processing do not find bothersome. Available tools rely on adult report of child sensory processing, and as such, behavioral measures of SOR rely on secondary information. In the mediation model we identified, parent-reported SOR did not influence the magnitude of the child's physiological response to sensory challenge. This finding is consistent with the findings of Schoen, Miller, Brett-Green, and Nielsen (2009), in which SCL magnitude was unrelated to scores on the Short Sensory Profile in children with ASD. Although no research to date has examined differences in child versus parent perception of SOR, researchers have indicated that the relationship between child and parent perception of behavior is weak, particularly for behaviors that are difficult to observe (e.g., Achenbach, McConaughy, & Howell, 1987). Because we did not ask the children to complete self-reports of SOR or to engage in a performance assessment of sensory modulation, we can only surmise that parent and child perceptions might differ. In the future, measures that directly involve the child will become invaluable in developing a more thorough understanding of the perception of SOR.

Parent-reported SOR was strongly linked with our measure of generalized anxiety, the RCMAS Total Anxiety score. This tool is completed by the child but administered aloud to the child by the parent. Thus, the parent may influence the child's responses to the questions about anxiety, resulting in a measure of anxiety that reflects as much parent perception as child perception. We have found SOR and anxiety to be related in other investigations (Lane et al., 2010; Reynolds & Lane, 2009). This relationship provides some support for Green and Ben-Sasson's (2010) primary SOR model in that it points to SOR as a cause of child anxiety.

Our path model indicates that the intensity of response to sensory stimuli serves as a mediating variable between baseline arousal and attention, on the one hand, and anxiety (RTA) and recovery (SCLrec), on the other. Baseline SCL is commonly used as a measure of tonic arousal and attention; SCLs are influenced by multiple brain regions associated with these functions, including

Table 1. Child Demographics

Group	*N*		Mean Age ± *SD* (mo)	Mean Cognitive Score ± *SD*
	Girls	Boys		
ASD (*n* = 23)	2	21	109.2 ± 20	95.5 ± 18
ADHD (*n* = 38)	11	27	101.5 ± 22	83.0 ± 16
TYP (*n* = 70)	35	35	103.7 ± 22	112.7 ± 14

Note. ADHD = attention deficit hyperactivity disorder; ASD = autism spectrum disorders; *SD* = standard deviation; TYP = typically developing.

Table 2. Indirect Effects (Pathways)

Specific Indirect Effect	*p*
SCLbase → SRMag → RTA	<.001
Cortbase → SRMag → RTA	ns
SOR → SRMag → RTA	ns
SCLbase → SRMag → SCLrec	.05
Cortbase → SRMag → SCLrec	ns
SOR → SRMag → SCLrec	ns

Note. Cortbase = baseline cortisol; ns = not significant; RTA = Total Anxiety score on the Revised Children's Manifest Anxiety Scale; SCL = skin conductance level; SCLbase = baseline SCL; SCLrec = SCL during recovery; SOR = sensory overresponsivity; SRMag = mean magnitude of the specific response.

the reticular formation, the limbic system, and the frontal cortex (Dawson, Schell, & Filion, 2007; Lee et al., 1988; Mangina & Beuzeron-Mangina, 1996; Sequeira, Hot, Silvert, & Delplanque, 2009). High arousal and hypervigilance, reflecting activation of these central nervous system regions, influence the magnitude of response to sensation. SCLrec is seen to reflect the ability of the SNS to return to a state of homeostasis following response to a perceived threat. Our model suggests that the magnitude of responsiveness to a sensory challenge determines both the ability of the nervous system to recover from the challenge, in this case return to baseline following the perceived threat of unpredictable and uncontrollable sensory challenge, and generalized anxiety, supporting the primary SOR model proposed by Green and Ben-Sasson (2010). Thus, although our current measurement tools do not allow us to adequately measure child-perceived SOR, tonic arousal and attention are related to anxiety and recovery of the SNS by way of the mediator variable, magnitude of response to sensory challenge. The relationship between our measured variable, mean EDR to sensory challenge, and child self-perception of SOR needs further investigation.

Clinical Relevance

Our findings indicate that the strength of the physiological response to sensation is influenced by the initial state of arousal and attention. This finding is highly consistent with earlier suggestions by Ayres (1972) that defensive responses to (tactile) sensation vary with the child's emotional state and other experiences in that single day. Moreover, our model indicates that the response to sensation itself influences overall anxiety and the ability to return to a baseline state. Clinically, this concept is important to capture as we speak with parents, teachers, and administrators because it reframes the child's response as being attributable to the state of the nervous system and not to willfulness to behave poorly in various environments.

Unfortunately, available tools measuring sensory modulation in children rely on parent report to reflect how the child feels about routine sensory stimuli. Parent report is likely to reflect the parent's perception of the child's response across several days' time. In both this and prior work, we have shown that parent reflections on sensory responsivity correlate with measures of anxiety, and this notable relationship appears for typically developing children as well as children with ASD and ADHD. In the current study, SOR was causally related to anxiety, indicating that clinicians must understand both SOR and anxiety disorders; the two likely coexist in at least some children. That this work supports Green and Ben-Sasson's (2010) primary SOR model suggests that treatment of SOR and anxiety may need to begin by addressing sensory responsivity.

Tonic arousal level influences autonomic nervous system responsivity to sensory challenge. Moreover, the response of the autonomic nervous system to a sensory challenge mediates the extent of anxiety and the ability of the child to recover from challenge. Clinically, these complex relationships may help explain and validate the reports that children may find a given sensation bothersome on one day, in one context, but not on another day or in a different context. Both SOR and anxiety have the potential to interfere with participation in the routine activities of childhood; our best clinical approach will capitalize on development of a better understanding of these linkages.

Limitations

Path analysis requires large data sets; this requirement necessitated grouping children across diagnostic groups for the current study. It would be intriguing in future work to examine these same variables in each group separately. It would also have been interesting to look at SOR within each sensory system because the literature suggests that responsiveness across sensory systems has some consistency (McIntosh, Miller, Shyu, & Hagerman, 1999) or can differ substantially (e.g., Liss et al., 2006). The available data set for the current analysis did not allow for these more detailed analyses.

Implications for Occupational Therapy Practice

The results of this study have the following implications for occupational therapy practice:

- Arousal and attentional states are important to consider when working with children with SOR.
- Children showing SOR may also show anxiety, and anxiety may be made worse when bothersome sensations are experienced. Behaviorally this child may "recover" from an environmental "sensory challenge" more slowly than other children.
- Clinically, the complex relationship among arousal, anxiety, and SOR documented in this study may help explain and validate the variability in sensory responsivity often reported by parents and teachers.

Conclusion

Occupational therapy researchers must develop either child self-report or child performance tools to measure sensory responsivity and determine their relationship to

parent report tools. We must also examine the child-based tools relative to the developing physiological measures of central nervous system responsiveness to sensory challenge. We have demonstrated that the intensity or magnitude of sensory responsivity mediates the relationship between baseline arousal and attention, on one hand, and outcome measures of anxiety and central nervous system recovery, on the other. SOR can prevent children from participating in the occupations of childhood; the greater our understanding of SOR, the more successful occupational therapy practitioners will be in developing effective interventions. ▲

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