Somatosensory function in boys with ADHD and tactile defensiveness

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Abstract

In this study, we tested for deficits in somatosensory function in boys with Attention Deficit Hyperactivity Disorder (ADHD) and tactile defensiveness (TD). The subjects were 67 boys with ADHD, sub-typed as TD (ADHD+TD+) or non TD (ADHD+TD−), matched with 60 "typical" children in the control group. Sixty nine percent of the boys with ADHD were categorized as TD. The groups were compared on three measures: (a) performance scores on subtests of the Sensory Integration and Praxis Test, (b) measurements of the Somatosensory Evoked Potential (SEP) and (c) ratings of the children's affective responses during tactile stimulation. Both ADHD groups differed from the control group on most study measures. No significant differences were found between the two ADHD subgroups on threshold and perceptual tests scores, except for Finger Identification. However, the TD+ group demonstrated significantly higher central SEP amplitudes than did the TD− group. Together, the results support claims that TD is related to central processing of somatosensory information, but not to anomalous tactile perception, with the exception of Finger Identification.
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1. Introduction

Sensory modulation has been defined as the 'capacity to regulate and organize the degree, intensity and nature of responses to sensory input in a graded and adaptive manner' [1]. Sensory Modulation Disorder (SMD) is a disorder whereby individuals routinely demonstrate exaggerated (avoidant and defensive) or inappropriate responses to benign sensory input. SMD is a generalized disorder that effects modulation across sensory systems, including the tactile, vestibular, auditory, and olfactory systems [2,3]. The ability to modulate sensory input is critical for the developing child's quality of life, efficiency of interaction within his physical and human environment, and optimal performance and participation in daily life challenges [4].

As such, efforts to study sensory modulation deficits (SMDs) fit the call from the World Health Organization (WHO) to identify risk factors that may interfere with children's participation in life activities [5]. In this regard, Simeonsson et al. suggested that Sensory Modulation Disorder be included as a special category in the International Classification of Functioning, Disability, and Health (ICF), as formulated by the WHO [5,6].

With the exception of parent or child rating scales which assess typical behavioral responses to routine sensory stimulation, available research findings have identified few reliable indicators of SMD in children. Little is known about the underlying causes of this disorder and it remains unclear whether SMD should be considered independently from the co-occurring "base" disorders (such as ADHD, retardation, and Fragile X syndrome) [7–9].

The focus in this paper is on one subtype of SMD, tactile defensiveness (TD), which refers to hyper-sensitive responses to routine tactile stimulation [10]. Our aim was to provide a broad somatosensory profile of children with and without TD that includes measures derived from a range of discrimination tasks and electrophysiological recordings of the somatosensory evoked potential (SEP). Such a profile could afford a set of objective markers of TD and would link it to aberrant neural responsiveness to somatosensory stimulation.

In order to compile this somatosensory profile, we compared a group of children with ADHD who displayed tactile defensive

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behaviors (as assessed by parent and experimenter ratings of the child's behavior) to a group of ADHD children without TD. A control group was added to establish a normal baseline. A portion of the sample described in this paper was described in a previous publication [11]. The sample of children with ADHD was enlarged in the present study so that they could be divided into two subgroups, one with and one without TD, and then compared on select measures. The control group was enlarged so that it would contain a number of children similar to the groups of children with and without ADHD. In our previous study, we compared children with and without ADHD. Results showed that the ADHD children exhibited larger than normal amplitudes of late sensory evoked potential (SEP) components and that they received lower somatosensory perception scores. In that study, no comparisons were made between subgroups with and without TD [11].

The analyses described in this paper are among the first to test for a relationship between SMD and neural responsiveness to sensory stimuli and the first to connect such data to TD. These data are indispensable in testing the hypothesis that SMD is related to an anomaly in neural activity associated with the processing of sensory information, particularly in the inhibitory or excitatory mechanisms activated by sensory stimuli [12]. In addition, measures of latency (time taken to reach the peaks of the primary components of the waveform) and amplitude (size of the components) can indicate whether SMD is related to faster- or larger-than-normal neural responses, and may also contribute to the assessment as to whether anomalies are present during early and/or later stimulus processing. Recent findings indicate that electrodermal responses of children with SMD have larger amplitude than those of children in the control group, and we expected to find similar results using the somatosensory evoked potential (SEP) [2,7].

The focus on TD rather than other subtypes of SMD was fostered by the increasingly strong evidence that touch plays a crucial role in early childhood development [13,14]. Recent studies have shown that the tactile experience of nonhumans and humans early in life can have long-term effects on neural functioning, the capacity to cope with stress, and emotional development (attachment, emotional regulation, exploration, and learning) [15]. As a result, supportive evidence of neural dysfunction and the identification of objective markers of TD could significantly contribute to our understanding of a disorder with potentially widespread detrimental effects on child development.

In summary, our goal was to find empirical support relating TD to electrophysiological and psychophysical measures in order to test for objective measures that discriminate between children with and without TD. Our working hypothesis was that amplitude and/or latency measures would show significant differences between groups, reflecting a neurological basis for TD. Further, we expected to find that scores reflecting the severity of TD would be related to parameters of the SEP, such that children with more extreme TD would also show extreme SEPs. We did not pose a directional hypothesis regarding SIPT scores, because, to date, scores on discriminative tasks have not discerned between children with and without TD.

2. Method

2.1. Participants

One hundred and twenty seven boys were tested, 67 diagnosed with ADHD, and 60 in the control group. The sample included only boys because it is not clear whether ADHD is driven by the same psychological dysfunction in boys and girls and whether both genders share the same etiology [16]. Selection inclusion criteria stipulated that the subjects, regardless of group placement, be between 5 and 11 years of age, be of normal intelligence as assessed by the Wechsler Preschool and Primary Scale of Intelligence (WIPPSI) tests, and had been free of medication for at least one month prior to testing. Furthermore, participants in the study had to have been born without apparent complications after a full gestation period and show no apparent physical or neurological deficits on the standard assessment protocol proposed by Touwen [17].

Children were diagnosed with ADHD if they showed eight out of the 14 criteria for ADHD according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) [18] and received scores above the standardized cut-off point on the Conners Rating Scale [19]. The children in the ADHD group were divided into two subgroups: ADHD with TD (TD+, $n=46$) and ADHD without TD (TD−, $n=21$) based on the results of two measures. The first measure was derived from parents' ratings on the Touch Inventory for Preschoolers (TIP), a checklist focusing on identifying children with hypersensitivity to benign tactile stimulation [20]. The second measure used to differentiate between TD+ and TD−, the Sensory Reactivity Score, was based on experimenter's blind ratings of the children's behavior [21]. Children with ADHD who scored above the cut-off on both measures were labelled as TD+ (ADHD+TD+; $N=46$). Children with ADHD who scored below the cut-off on both measures were labelled as TD− (ADHD+TD−; $N=21$). All of the control children ($N=60$) and all of the ADHD+TD− children met neither of the two TD criteria.

The children in the control group (ADHD−TD−) were selected from public schools and were matched in age to one of the children diagnosed with ADHD (combined sample of TD+ and TD−). None of the children in the control group met any of the criteria for ADHD. Of the 67 children in the control group, seven children were dropped from the study due to not having met the inclusion criteria after testing, leaving the control group with a total of 60 participants. The average age of the controls, ADHD+TD+, and ADHD+TD− groups was 8.2 (SD=1.6), 7.5 (SD=1.3), and 7.3 (SD=1.0) years old, respectively.

3. Measures

3.1. Touch Inventory for Preschoolers (TIP) [20]

Assessment of TD was made on the basis of parents' ratings on the Touch Inventory for Preschoolers, which focuses on the child's typical responses to routine tactile stimuli [20]. Content and construct validity were established by the consensus of 30 experts who assessed 73 items. The final version of the measure

includes 46 items that were rated as representative, relevant and accurate markers of TD with internal consistency (Alpha Cronbach) of .89 [20,21]. The cut-off score for TD was calculated as one standard deviation above the mean score of a sample of 60 typical children described in a previous report [11].

3.2. The Sensory Reactivity Score [22]

This measure was derived from rating scales designed to quantify the intensity of overt signs of touch-related discomfort and agitation [23]. In the present study, test–retest and inter-rater reliability were examined by coding and re-coding of 10 films, and both reliability measures were found to be significant ($r=.90$ and $r=.85$, $p<.05$; respectively). Construct validity was established by six experts (two neuro-paediatricians, a physiologist, a psychologist and two occupational therapists) who were in full agreement that this measure accurately assesses the construct of TD. In addition, construct validity was supported by the significant correlation between children's observed reactivity to touch and parents' ratings of TD in the present study ($r=.83$, $p<.001$).

A rating of reactivity (e.g., signs of discomfort) was made by the experimenter while each of four scalp-electrodes used for the electrophysiological recordings were being attached to the head and body of the subject. This context was used to obtain a reactivity score because the attachment of electrodes is tactile-intensive, involving scratching of the epidermis and the application of electrode gel and electrodes on the skin. Ratings, made after each electrode placement, were based on the children's overt behavior (verbal and nonverbal) on a 0 (no display of discomfort) to 4 (display of intense discomfort) Likert scale.

3.3. Sensory Integration and Praxis Test (SIPT) [24]

The SIPT is a standardized reliable and valid assessment composed of a battery of tests designed to evaluate various aspects of sensory processing in children. Content and construct validity of the SIPT have been demonstrated through a number of factor analytic studies and cluster analyses. In addition, multiple discriminant analyses demonstrated significant differences between groups of normal children and those with dysfunction (e.g. learning disabilities and brain dysfunction). Inter-rater reliability and test–retest reliability were found to be satisfactory ($r=.54–.94$; $r=.54–.94$, respectively) [24].

In the current study, the five somatosensory subtests of the SIPT, used to examine suprathreshold tactile perception, included: (a) Finger Identification, in which the child is asked to point to the finger(s) previously touched by the examiner; (b) Graphesthesia, in which the child is asked to duplicate a design traced on the back of his/her hand by the examiner; (c) Localization of Tactile Stimuli, in which the child is asked to place his/her finger on a spot that was previously touched by the examiner; (d) Manual Form Perception, in which the child must identify the visual counterparts of various plastic geometric forms that are held and manipulated one at a time, either in one hand, or in both hands simultaneously; and (e) Kinaesthesia, which requires a child to move his/her finger from one location to another, with eyes occluded, following the passive movement of the finger between both locations by the examiner.

3.4. Somatosensory Evoked Potential (SEP)

Following Desmedt, [25] Huttunen and Homberg, [26] Taylor and Fagan [27] and others, the SEP was attained by the delivery of rectangular electrical stimuli, 0.2 ms in duration, at a rate of 2 per second to the median nerve at the wrist, thereby exciting a major somatosensory pathway involved in cutaneous sensation. The stimulus intensity was set at the minimum current for each participant necessary to evoke a thumb twitch, in order to ensure that the sensory fibres in the nerve are stimulated to approximately the same extent in all subjects.

For recording the SEP, 4 silver-cup electrodes were placed over the skin at Erbs point (ERB), the 7th cervical vertebra (C7), the forehead (Fpz), and on the scalp over the contralateral somatosensory cortex (C3). From this matrix, differential recordings for three channels of neural activity were obtained. The recorded activity was averaged ($N=512$) using a time-frame of 63 ms. The recorded activity was also filtered (2–300 Hz bandpass) so that the SEP components would not be masked by electrical "noise" and could be clearly read and measured. Scalp positivity resulted in an upward deflection for all three channels. To confirm reproducibility, two responses were elicited from each participant and the average of the two recordings was derived.

Recordings obtained from the ERB-Fpz channel were used to measure the primary peripheral component of the SEP, generated in the afferent median nerve at the brachial plexus. This wave is labelled P9 because its positive peak appears approximately 9 ms after the onset of the electrical pulse. From the C7-Fpz channel, the SEP's negative peak was measured at 13 ms (N13), which reflects stimulus-evoked neural activity at the entrance to the spinal cord. Finally, the C3-Fpz recording channel provided measurement of the SEP's N20 and P23 waves, which represent the neural response of the somatosensory cortex.

Typically, measures of latency (e.g. ms of time from stimulus onset to the generation of a primary wave of the SEP) and amplitude (size of a primary wave) are derived from the recorded responses and reflect neural transmission time and stimulus-evoked physiological responsiveness, respectively. In this study, seven response parameters were measured from the averaged responses. Three were central conduction times (CCT) calculated as the time difference (ms) between P9 and N20, N13 and N20, P13 and N23. Central conduction time was the measure of choice for latency measures because the CCTs are not influenced by subjects' arm-length, as are absolute latency measures. The final four measures were amplitudes, measured from the preceding peak of opposite polarity to the peak of N13, N20 and P23.

4. Procedure

The experimental protocol was approved by the Committee for Human Experimentation, Hebrew University — Hadassah Medical School, Jerusalem. Each of the participants was tested individually in a quiet room according to a standard protocol. Sensory reactivity ratings were made by one of two

Table 1
Mean SIPT subtest scores of control (C), ADHD+TD+ (TD+), and ADHD+TD− (TD−) groups and results of between-group comparisons

	C	TD+	TD−	ANOVA	C vs. TD+	C vs. TD−	TD− vs. TD+
	M/SD	M/SD	M/SD	F/p	p<	p<	p<
FI	12.15	6.91	9.00				
	2.65	2.62	2.82	.0001	.0001	.0001	.015
GRA	22.91	14.75	12.80				
	3.47	4.65	4.32	.0001	.0001	.0001	NS
LTS	14.77	20.79	20.53				
	4.03	7.49	6.11	.0001	.0001	.001	NS
KIN	23.31	30.33	31.16				
	6.78	9.88	11.10	.001	.001	.004	NS
MFP	17.06	12.25	12.73				
	2.20	4.24	3.61	.0001	.0001	.0001	NS

Note that regardless of the sign of the t score, the performance of the ADHD children on all of the tests was poorer than that of controls. (FI = finger identification, GRA = graphesthesia, LTS = localization of a tactile stimulus, KIN = kinesthesia, MFP = manual form perception).

experimenters during attachment of the four scalp-electrodes used for the SEP recordings. Thereafter, the SEP was recorded, while the children watched a cartoon on a monitor in order to minimize extraneous body movements. In the final stage of testing, the SIPT was administered. While the child was being tested, the accompanying parent filled in the TIP questionnaire in the waiting room. Using this procedure, experimenters were blind as to group placement (ADHD+TD+; ADHD+TD−), since the TIP was not scored until after the testing-session. Half of the sessions were carried out by an experimenter who was aware of the ADHD status of the children and half of the sessions were carried out by an experimenter who was unaware of the children's ADHD status.

5. Data analysis

Differences between groups on the SIPT were examined by a repeated measure MANOVA, with group (ADHD+TD+;

Table 2
Mean (SD) of SEP central conduction times (ms), SEP amplitudes and post-hoc comparisons (Scheffé)

	C	TD−	TD+	ANOVA	C vs. TD−	C vs. TD+	TD− vs. TD+
	M/SD	M/SD	M/SD	F/p	p<	p<	p<
CCT1	8.32 (1.13)	8.09 (.71)	8.20 (.90)	NS			
CCT2	5.99 (1.32)	6.11 (.48)	6.29 (.86)	NS			
CCT3	9.31 (1.81)	9.57 (.85)	9.95 (1.34)	NS			
P9	1.69 (1.45)	1.93 (1.23)	2.61 (1.45)	NS			
N13	2.05 (1.29)	2.70 (1.29)	3.60 (1.39)	10.86 (.001)	NS	.0001	.05
N20	1.07 (.56)	1.45 (.63)	2.17 (.58)	9.16 (.001)	NS	.0001	.05
P23	2.54 (2.0)	2.63 (1.37)	5.50 (3.82)	12.07 (.001)	NS	.0001	.001

One-way ANOVAs and contrasts were not run because the multivariate analysis did not yield a significant main effect of group.
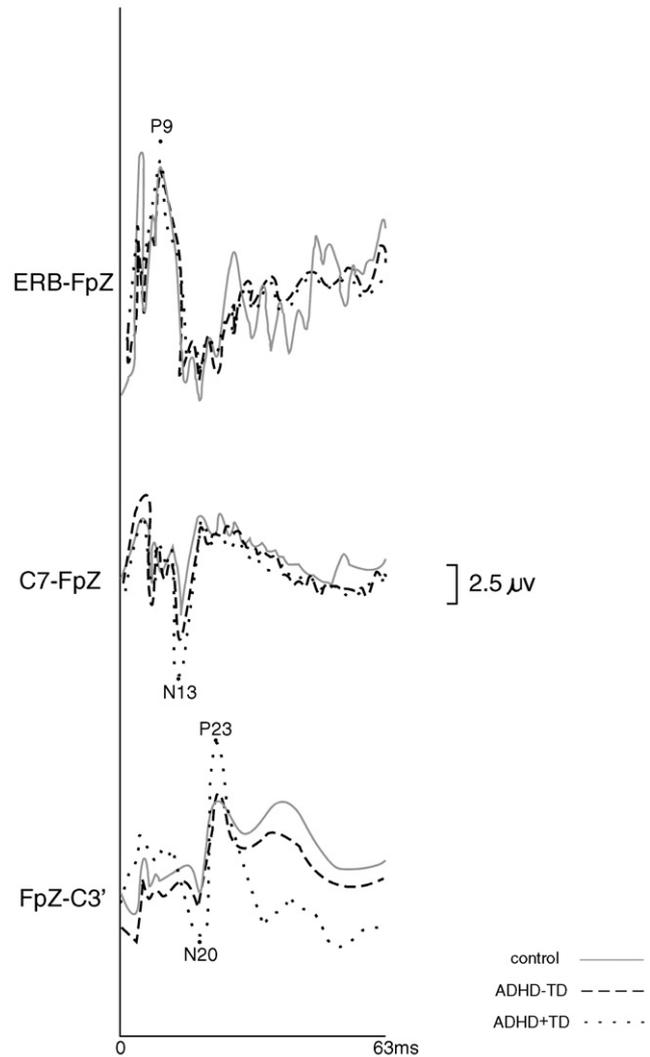

Fig. 1. Sample SEP recordings of three different boys (control, ADHD+TD+, ADHD+TD−). No differences can be seen between the boys on the amplitude of P9, however, the N13, N20 and P23 amplitudes of the ADHD+TD+ group are larger than those of the control and ADHD+TD− group. See text for details.

ADHD+TD−; control group) as the between-group variable and the five subtests of the SIPT as the repeated measure. The same analysis was applied to SEP measures, taken separately. This design was selected over a series of paired contrasts, to limit the number of statistical tests applied to the data and to test for scores that discriminated children in the TD+ group from those in the TD− and control groups. For all tests, the age of the child was entered as a covariate due to the age-difference between the ADHD and control groups.

6. Results

6.1. Preliminary analyses

Prior to data analyses, t-tests were used to test for differences between the children with ADHD used in the previous study and those added to the sample for the present study. No differences (means, variance, range) were found on any demographic or performance measure, including the SEP.

Similarly, no differences were found between children who comprised our previous control group and those who were added to the group for the purpose of this study.

6.2. Grouping by TD+ and TD−

According to the TIP and reactivity scores, 69% (46/67) of the children with ADHD were designated as TD. Mean TIP scores of the control, TD−, and TD+ groups (with standard deviations in parentheses) were 61.51 (8.24), 64.28 (13.80), and 112.30 (24.00) respectively, and mean sensory reactivity scores were .71 (.26), 1.00 (1.28), and 3.36 (1.04) respectively. Paired contrasts showed that both measures distinguished the ADHD+TD+ group from the two other groups, but not the ADHD+TD− group from the control group.

6.3. Group differences on the SIPT

Analysis of the SIPT scores indicated a significant difference across groups ($F(2, 124)=14.15$, $p<.0001$), reflecting higher scores of the control group as compared to those of the ADHD+TD+ and ADHD+TD− groups (Table 1). The only difference between the TD+ and TD− group was shown in Finger Identification (Table 1).

6.4. Group differences on SEP

The MANOVA applied to the amplitude measures yielded a between-group effect ($F(2, 124)=4.65$, $p<.014$). Post-hoc univariate (Scheffé) tests revealed that the N13, N20, P23 amplitudes of the ADHD+TD+ group were significantly larger than those of the control and ADHD+TD− group. There were no differences between these groups on the amplitude of P9, and no differences between controls and children with ADHD but without TD on any amplitude measure (Table 2; Fig. 1).

The same analyses were applied to CCT measures. The between-group MANOVA yielded no significant group effect and no group by CCT interaction. Further, an exploratory comparison between the two ADHD groups did not show a significant difference on any CCT measure.

To strengthen these findings, we tested for an association between the variables used to assess TD (TIP scores and sensory reactivity scores) and SEP amplitudes among children in the ADHD+TD+ group. The results of these analyses revealed a significant relationship between TIP scores and N13, N20, and P23 amplitudes ($r=.31–.37$, $Ps<.001$), but not between TIP scores and P9. Similarly, the correlations between reactivity scores of the TD+ group and amplitudes (N13, N20, and P23) were significant ($r=.23–.37$, $Ps<.05$), but the correlation between reactivity scores and the amplitude of P9 was not.

7. Discussion

In the present study, we compared boys with ADHD with and without TD to a control group on a range of somatosensory discrimination tasks and recorded cortical and sub-cortical potentials evoked by a somatosensory stimulus. Our aim was to identify discriminative markers of TD in order to link them to neural dysfunction. The results show that the ADHD+TD+ group was discernable from the control group on most measures, but, more importantly, it was distinguishable from the ADHD+TD− group by larger central SEP amplitudes. Moreover, analyses revealed a linear relation between scores used to evaluate TD (TIP scores and sensory reactivity scores) and the amplitudes of these components among the children with TD. No differences between the two ADHD groups were found on the amplitude of the P9 component of the SEP, neural conduction times, or four of five of the SIPT subtests. As in previous studies, children's performance on relatively simple somatosensory tests did not discriminate between the ADHD groups, suggesting that TD, as assessed here and elsewhere, is not related to deficits on these kinds of tasks.

Our results show that TD is marked by anomalous central responses to a somatosensory stimulus and, as such, constitute the first direct evidence of a link between SMD and atypical central neural processing, within a male population. In addition, they also indicate that the exaggerated central SEP amplitudes that were related to ADHD in a previous report are more likely associated with TD or a combination of TD and ADHD than to ADHD per se [11].

The present data support claims that TD is a discernable entity, marked by anomalous physiological responses to somatosensory stimuli. Furthermore, the data are consistent with the contention offered previously, that TD, and perhaps SMD in general, is related to disruptions in neural inhibition [12]. The fact that TD was found to occur in a large portion of the ADHD population (46/67) may be related to findings of central hypoperfusion (below normal levels of cerebral blood flow) [28,29].

In addition, a recent case-study on three children with ADHD described the benefits of extended release Valproate (EVA) on the children's clinical symptoms and a reduction of "giant" SEPS [30]. Since EVA is considered to be an enhancer of γ aminobutyric acid (a GABAergic agent), a primary inhibitory transmitter in the vertebrate CNS, the favourable response of the children may point to deficits in neural inhibition as a basis for the large amplitude central components of SEPs as described in this report.

Given this, further research on TD and other subtypes of SMD is called for in order to understand the underlying bases of these disorders, as well as to investigate these effects among girls. Such investigation is vital to designing specific assessment protocols, treatments, and effective interventions.

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