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Somatosensory evoked potentials in children with autism

Hanan Galal Azouz [a], **Mona Khalil** [a], **Hayam Moustafa Abd El Ghani** [b,*], **Hatim Mohamed Hamed** [a]

[a] *Department of NeuroPediatrics, Faculty of Medicine, Alexandria University, Egypt*
[b] *Department of Physical Medicine, Reumatology and Rehabilitation, Faculty of Medicine, Alexandria University, Egypt*

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Abstract *Introduction:* Autism is a neurodevelopmental disorder in the category of pervasive developmental disorders (PDD), which is characterized by widespread abnormalities of social interactions, communication, and severely restricted interests and highly repetitive behavior. **Children with autism show sensory and perceptual abnormalities. They have either hyposensitivity or hypersensitivity to sensory, auditory, and visual stimuli.**

Objectives: The aim of this work was to study somatosensory evoked potential (SSEPs) changes among children with autism, and their relation to somatosensory manifestations and severity of autism.

Subjects: Thirty children with autism aged 2–12 years were included in the study, all of them fulfilling criteria of the Diagnostic and Statistical Manual of Mental Disorders (DSM–IV–TR).

Methods: All cases were subjected to thorough history taking including autistic symptoms and sensory abnormalities, comprehensive medical examination, psychiatric assessment according to DSM–IV–TR criteria for diagnosing autism, assessment of severity of autism using Childhood Autism Rating Scale (CARS) and measurement of somatosensory evoked potentials elicited by median nerve stimulation at wrist.

Results: The majority of the cases were males (86.7%), according to CARS 53.3% were classified as mild to moderate autism, while 46.7% were severe. Sensory abnormalities were present in 56.7% of cases.

Abbreviations: PDD, pervasive developmental disorders; ASD, autism spectrum disorders; DSM-IV-TR, diagnostic and Statistical Manual of Mental Disorders, 4th edition; SEPs, somatosensory evoked potentials; CNS, central nervous system; CARS, Childhood Autism Rating Scale.

* Corresponding author.
E-mail addresses: hananazouz@hotmail.com (H.G. Azouz), drMonaKhalil@yahoo.com (M. Khalil), Hayam_mstf@yahoo.com (H.M.A.E. Ghani), HatimHamed42@yahoo.com (H.M. Hamed).

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Somatosensory abnormalities were present in 36.76% of the cases. There was a statistically significant relationship between sensory symptoms with SSEP abnormalities ($P = 0.040$). The presence of abnormal SSEPs was not statistically associated with higher score in CARS.

Conclusions: Children with autism have abnormal SSEP changes and were significantly related to the presence of sensory abnormalities, indicating central cortical dysfunction of somatosensory area. On the other hand, these abnormal SSEP changes were not related to the severity of autism.

1. Introduction

Autism is a neurodevelopmental disorder in the category of pervasive developmental disorders (PDD), which is characterized by widespread abnormalities of social interactions, communication, and severely restricted interests and highly repetitive behavior.[1] These conditions are suggested to be present at birth and are diagnosable by 18 months of age.[2]

Children with autism show sensory and perceptual abnormalities. They have both hyposensitivity and hypersensitivity to sensory, auditory, and visual stimuli.[3] Sensory disorders are included among the most prominent features of Pervasive Developmental Disorders and are reported to play an important role in children's intervention planning, as well as outcome. Sensory disturbances were reported in Kanner's original description of autism[4], and have been reported consistently in the clinical literature.[5,6] Though not currently part of the diagnostic criteria for Autism spectrum disorders (ASD), the presence of unusual sensory behaviors has been proposed for inclusion in updated diagnostic criteria for The Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-V), highlighting emerging consensus that sensory abnormalities are central features of ASD. Reports of abnormal sensory function that span the visual, auditory, gustatory, and tactile domains reinforce the "multisensory" nature of sensory processing alterations in ASD[7], and emerging evidence suggests that abnormalities also extend to the selective integration of information across the different sensory modalities.[8]

Sensory processing involves the ability to take in, organize and make sense of different kinds of sensations received by the brain. Rates of sensory processing dysfunction may be as high as 90% in individuals with Autism Spectrum Disorder.[9–12]

Somatosensory perception plays a central role in the early stages of human development. Impaired somatosensory processing is found in a range of neurodevelopmental disorders and is associated with deficits in communication, motor ability, and social skills in these disorders. Given the central role of touch in early development, both experimental and clinical approaches should take into consideration the role of somatosensory processing in the etiology and treatment of neurodevelopmental disorders.[13] Somatosensory evoked potentials (SEPs) would be expected to provide information about somatosensory function in children with autistic disorder. Somatosensory Evoked Potentials (SEPs or SSEPs) are useful, noninvasive means of assessing somatosensory system functioning. Somatosensation has four main submodalities, touch, proprioception, pain, and thermal sensation. Distinct receptor neurons transmit information further to the central nervous system (CNS).[14,15]

The aim of this work was to study somatosensory evoked potential changes among children with autism, and their relation to somatosensory manifestations and severity of autism.

2. Subjects

Thirty children with autistic disorder were included in this study, aged 2–12 years, all of them fulfilling the criteria of the Diagnostic and Statistical Manual of Mental Disorders (DSM–IV–TR).[16] The sampled children were recruited from outpatient neurology clinic at Alexandria University Children's Hospital.

Exclusion criteria included: Children with other psychiatric disorders, and children with other pervasive disorders such as Rett syndrome, childhood disintegrative disorder.

Thirty normally developing age and sex matched controls were included in the study for comparison of somatosensory evoked potentials .They were recruited from those attending outpatient clinics at Alexandria University Children's Hospital other than neurology clinic.

An informed consent was obtained from the parent or caregiver before the procedures.

3. Methods

All children included in the study were subjected to the following:

1- Thorough history taking including the socio-demographic characteristics of the child, information on prenatal history, developmental milestones, and family history, history of autistic disorder, onset, presenting symptoms, and the course of the illness,
2- Comprehensive medical examination.
3- Psychiatric assessment according to DSM-IV-TR criteria for the diagnosis of children with autism[16], and assessment of the severity of the autistic disorder using the Childhood Autism Rating Scale (CARS).[17] The severity of autistic symptoms was categorized according to child's total score to mild- moderate $\leqslant 37$ and severe $\geqslant 37$.
4- Interview of child and parents for assessment of sensory abnormalities.
5- Somatosensory Evoked Potentials (SSEP).[18] using Nihon Kohden Corp Electrodiagnosis Apparatus (MEB- 710 2 K, made in Japan).

EEG electrodes were used for recording. We applied an electrical stimulus to the median nerve at wrist on both sides.

Active recording electrodes were placed on C3/ (for right median nerve stimulation) and C4/ (for left median nerve stimulation). C3/ and C4/ were 2 cm behind C3 and C4 electrode positions of the international 10–20 system of EEG electrode placement.

Cephalic (FP_Z) and non cephalic (Erb's point contralateral to stimulated side) reference electrodes were used. For right median nerve stimulation we use montage C3/ – Lt Erb's (wave P9, P11, P13/14) and C3/ -FP_Z (wave N20–P25) and for left median nerve stimulation we use montage C4/ – Rt Erb's (wave P9, P11, P13/14) and C4/ -FP_Z (wave N20–P25).

The children lay supine on a bed in a quiet room. The examination was performed during sleep using oral chloral hydrate half an hour prior to recording in a dose of 50 mg/kg.

The median nerve at the wrist was stimulated by an electric square-wave pulse of 0.3 ms duration, which was of sufficient intensity to produce a noticeable movement of the thumb delivered at a rate of 1 HZ.

Input was amplified 100,000 times. The filters used were set at 2500–4000 HZ. Two trials of 200 averaged responses were performed and superimposed to test the reproducibility of wave forms.

Peak latencies of P9, P11, P13/14, N20 and P25 were measured (P9 the ERB's point representing the brachial plexus-P11, P13/14, represent cervical segment of spinal cord- N20 and P25, represent the cortical level in the primary somatosensory area) and the interpeak latency for P13/14–N20 was calculated as an estimate of central conduction time as well as that for N20–P25. We also evaluated the peak-to-peak amplitude of N20–P25 to estimate cerebral activities, especially in the primary somatosensory area. S-SEPs were considered abnormal if any latency exceeded a standard deviation (SD) of 2.5 of the control mean value, which was obtained from 30 age matched controls (15 males, 15 females) age range 2–9 years; mean age 6.7 ± 2.08 for the present study, based essentially on data obtained from typically developing children after their parent consent. In addition, the S-SEPs were also considered abnormal if: (1) any component was judged to be absent on visual inspection; (2) the peak-to-peak amplitude of N20–P25 exceeded 10 mV, representing giant SEP; or (3) if the left and right peak-to-peak amplitude of N20–P25 differed more than twofold. On the basis of data obtained from all 30 autistic children, differences of interpeak latencies of P13/14–N20 and N20–P25 between left and right median nerve stimulations were evaluated as well as those of the peak to-peak amplitude of N20–P25.

we did a peripheral conduction study of the sural and median sensory nerves and median and posterior tibial motor nerves in addition to tibial H reflex.[19] All parameters of nerve conduction study were within normal. H reflex latency was normal with no increase in H/M ratio.

4. Statistical analysis of the data

Data were analyzed using SPSS software package version 18.0 (SPSS, Chicago, IL, USA). Test of normality was applied on the data by using Kolmogorov–Smirnov test, Shapiro–Wilk test. D'Agstino test was used if there was a conflict between the two previous tests. Quantitative data was expressed using range, mean, standard deviation and median. Quantitative data was analyzed using student's *t*-test to compare between two groups. Not normally distributed quantitative data were analyzed using Mann Whitney test for comparing two groups. P value was assumed to be significant at 0.05.

5. Results

The mean age of the studied autistic children was 5.77 ± 2.25 years. Age groups 5 < 8 years represented the highest rate among the sample (43.3%), while the lowest rate (23.3%) was among age group 8–12 years. The majority of the sample were males (86.7%), while the rest (13.3%) were females. As regards parents' consanguinity, 10% of the sampled children were having parents with positive consanguinity. As regards the age of onset of autistic features, 43.3% of the autistic children presented at age of 1 < 2 years, 30% presented at age of 2 < 3 years and 26.7% at age of 3 years.

According to CARS, 53.3% of sampled children had scores less than 37 on CARS indicating mild to moderate autistic features, while those who score ⩾37 were 46.7% indicating severe autistic features with a total CARS mean score 39.92 ± 5.88.

Sensory abnormalities were present in 17 children (56.7%) in the form of hyper responsiveness (i.e., behavioral over-reactivity to sensory stimuli) or hypo responsiveness (i.e., behavioral under-reactivity to sensory stimuli), some children had more than one sensory abnormality Table 1.

Table 1 Distribution of autistic children according to sensory abnormalities.

Sensory abnormalities	No. (n = 17)	%
Vision		
Hypo responsiveness	2	10
Hyper responsiveness	1	
Auditory		
Hypo responsiveness	1	16.7
Hyper- responsiveness	4	
Smell		
Hypo responsiveness	–	3.33
Hyper- responsiveness	1	
Touch		
Hypo responsiveness	4	
Hyper- responsiveness	2	20
Proprioception(body sense)		
Hypo responsiveness	3	10
Hyper- responsiveness	–	
Vestibular(movement sense)		
Hypo responsiveness	2	
Hyper- responsiveness	–	6.66
Pain		
Hypo responsiveness	4	13.33
Hyper- responsiveness	–	
Temperature		
Hypo responsiveness	2	10
Hyper- responsiveness	1	
Taste		
Hypo responsiveness	1	3.33
Hyper- responsiveness	–	

6. SSEP results

Table 2 shows that 11 cases (36.7%) had peak latency, interpeak latency, and/or amplitude abnormalities as determined by SSEP. There was a statistically significant difference in peak latency of N20 among the autistic children on both sides and the control group. There were no statistically significant differences between the autistic children and control regarding P9, P11, P13/14, and P25 on both sides Table 3 shows statistically significant differences between cases and controls in the interpeak latency of P13-N20 and N20-P25 for both right and left median nerve stimulation, while the interpeak latency of P11-P13/14 was of no statistical significance. As regards the peak to peak amplitude of N20–P25 of autistic children and control, P–P amp of N20–P25 ranged between 1.17–10.70 μv for right median and 0.42–9.17 μv for left median and there were statistically significant differences for both right and left stimulation in cases and the control group. ($P = 0.001$ for right side and $P = 0.002$ for left side) Table 4.

Table 2 Peak latency of SSEP among autistic children and control.

Wave latency (ms)	Control ($n = 30$)	Cases ($n = 30$)			
		Right median	$^{MW}p_1$	Left median	$^{MW}p_2$
P 9					
Range	4.20–6.80	4.0–7.0	0.430	4.20–7.80	0.282
Mean ± SD	5.39 ± 0.94	4.89 ± 0.67		5.27 ± 1.01	
Median	5.50	5.10		5.0	
P 11					
Range	6.95–8.25	6.80–10.40	0.490	6.80–10.80	0.615
Mean ± SD	7.78 ± 0.41	8.03 ± 0.84		8.20 ± 1.05	
Median	7.85	8.0		7.90	
P 13/14					
Range	9.45–11.60	8.80–14.0	0.850	9.40–16.60	0.875
Mean ± SD	10.52 ± 0.68	10.52 ± 1.22		10.95 ± 1.68	
Median	10.55	10.60		10.60	
N 20					
Range	15.35–17.0	7.60–23.40	$<0.001^*$	16.80–24.0	$<0.001^*$
Mean ± SD	16.15 ± 0.59	17.48 ± 2.28		17.87 ± 1.50	
Median	16.08	17.60		17.20	
P 25					
Range	24.30–27.10	19.25–30.0	0.987	20.0–37.20	0.150
Mean ± SD	25.83 ± 0.86	25.25 ± 2.58		25.05 ± 3.16	
Median	25.83	26.40		25.20	

$^{MW}p_1$: p value for Mann Whitney test between right median of cases and control group; $^{MW}p_2$: p value for Mann Whitney test between left median of cases and control group.
* Statistically significant at $p \leqslant 0.05$.

Table 3 The inter peak latency of SSEP between autistic children and control group.

Interpeak latency (ms)	Control ($n = 30$)	Cases ($n = 30$)			
		Right median	$^{MW}p_1$	Left median	$^{MW}p_2$
P11- P13/14					
Range	1.60–3.90	1.40–4.60	0.245	1.60–7.0	0.614
Mean ± SD	2.92 ± 0.88	2.49 ± 0.81		2.79 ± 1.08	
Median	3.18	2.20		2.30	
P13-N20					
Range	6.15–7.45	3.20–9.40	0.032^*	1.30–12.30	0.045^*
Mean ± SD	6.81 ± 0.44	7.29 ± 1.43		7.42 ± 2.06	
Median	6.93	7.60		7.70	
N20-P25					
Range	6.40–10.60	2.20–10.70	$<0.001^*$	2.60–13.20	0.036^*
Mean ± SD	8.77 ± 1.36	7.46 ± 2.52		7.17 ± 2.33	
Median	8.80	8.0		8.0	

$^{MW}p_1$: p value for Mann Whitney test between right median of cases and control group; $^{MW}p_2$: p value for Mann Whitney test between left median of cases and control group.
* Statistically significant at $p \leqslant 0.05$.

Table 4 Peak to peak amplitude of N20–P25 of autistic children and control group.

Peak to peak amplitude (μv)	Control (n = 30)	Cases (n = 30)			
		Right median	$^{MW}p_1$	Left median	$^{MW}p_2$
N20–P25					
Range	5.25–7.74	1.17–10.70	0.001*	0.42–9.17	0.002*
Mean ± SD	6.82 ± 0.86	4.96 ± 2.07		4.99 ± 1.80	
Median	7.26	5.0		4.63	

$^{MW}p_1$: p value for Mann Whitney test between right median of cases and control group; $^{MW}p_2$: p value for Mann Whitney test between left median of cases and control group.
* Statistically significant at $p \leqslant 0.05$.

Table 5 SSEP abnormalities in autistic children with somatosensory symptoms (no = 7).

Somatosensory symptoms	SSEP					
	Rt			Lt		
	Peak	Interpeak	P–P	Peak	Interpeak	P–P
Hyposensitivity to pain	Delayed P25	Prolonged N20–P25		Delayed P11 P13/14 P25	Prolonged P11-P14 P13-N20 N20-P25	
Hypersensitivity to touch	Delayed N20	Prolonged P13–N20	Giant wave	Delayed P11		
Hyposensitivity to temperature and touch	Delayed P25	Prolonged P13–N20			Prolonged N20–P25	
Hypersensitivity to touch			More than two fold than lt			
Hyposensitivity to pain		Prolonged P13–N20		Delayed N20	Prolonged P13–N20	
Hyposensitivity to touch	Delayed P13/14 P25–N20	Prolonged P13–N20		Delayed P13/14 P25–N20	Prolonged P13–N20 N20–P25	
Hyposensitivity to temperature and Proprioception	Delayed P11 P25	Prolonged P13–N20 N20–P25		Delayed P11,P25 P13/14	Prolonged P13–N20 N20–P25	

Table 6 Distribution of autistic children according to the severity of autistic symptoms and SSEP changes.

		SSEP		Total
		Normal	Abnormal	
CARS	Mild-moderate	10 (62.5%)	6 (37.5%)	16 (100%)
	Severe	9 (64.3%)	5 (35.7%)	14 (100%)
Total		19 (63.3%)	11 (36.7%)	30 (100%)
X^2		0.010		
P		0.610		

Table 5 shows that the total number of children who had SSEP changes and somatosensory symptoms were seven children (63.63%). The most common somatosensory symptoms were in the form of hyposensitivity to touch, pain and temperature and associated with prolonged interpeak latency of P13/14-N20 and N20–P25, while hypersensitivity to touch was associated with increased peak to peak amplitude of N20–P25.

Table 6 shows that six children (37.5%) with abnormal SSEP had mild to moderate autistic symptoms, while five children (35.7%) with abnormal SSEP had severe autistic symptoms. Normal SSEP was found in 10 children (62.5%) with mild to moderate autistic symptoms and 9 children (64.3%) with severe autistic symptoms. There were no statistically significant relationships between SSEP changes and severity of autism. (X^2 = 0.010, P = 0.610).

7. Discussion

In the present study 56.7% of the autistic children had sensory abnormalities, 20% to touch, 16.7% to auditory, 13.3% to

pain, 10% to temperature, Proprioception and vision, 6.6% to Vestibular, 3.3% to taste, as regarded that some children had more than one sensory abnormality. Klintwall et al.[20] studied 208 children with autism, 76% of them had sensory symptoms. The most commonly reported individual types of abnormalities were over-reactivity to sound (44%) and under-reactivity to pain (40%). Under-reactivity to cold and heat were reported for 22% and 7% respectively. Over-reactivity to touch had been noted in 19%. Abnormal reactions to visual stimuli were seen in 19%. Oversensitivity to smell was reported in 5%. Moreover, Bromley et al.[21] found that from a sample of 75 children; 71% were hypersensitive to sound, 52% to touch, 41% to smell and 40% to taste. They also found that 23% of children were hypersensitive to pain and 45% were hyposensitive to pain. The differences between the rates reported in these studies and the present study may be due to the large sample size compared to our sample size.

Kostović and Judas[22] emphasized that sensory abnormalities are very disabling, and have received little attention, but that insight into their neurobiologic basis may open the way to effective treatments.

In the present study, SSEP abnormalities were found in 11 children, indicating the presence of frequent somatosensory pathway dysfunction in autistic children, and this is in accordance with previous studies of SSEP on autistic children.[18,23] Regarding the peak latency of recorded waves, none of our patients had prolonged peak latency of P9 which suggests that conduction up to the brachial plexus was normal.[24] Prolonged latency of P13/14 was recorded in three children only but of no statistical significance, it has been ascertained that this wave is generated in the caudal brain stem (anatomic origin of P13/14).[25] P25 was delayed in five children but not statistically significant. It has been speculated that P25 may arise from the crown of posterior central gyrus.[26]

On the contrary, important and statistically significant delayed latency were recorded in N20 by Rt and Lt median nerve stimulation, it has been speculated that the generator of N20 may be located in Brodmann area 3b (the anterior bank of the posterior central gyrus facing the central sulcus). N20 and P25 represent the initial response of primary somatosensory cortex to stimulation of median nerve.[27]

The results of the current study recorded that there were statistically significant differences between children with autism and control as regards interpeak latency of P13/14-N20 and N20–P25. The prolongation of these interpeak latencies indicates central conduction slowing of somatosensory pathway between the brain stem and the sensory cortex.

This was in agreement with the study done by Miyazaki M, et al.[18] who described short-latency somatosensory evoked potentials (SSEP), elicited by median nerve stimulation, in 24 children with autism, and recorded delayed interpeak latency of P13-N20 in 7 children.

Ververi et al.[23] reported slightly higher rate for SSEP abnormalities (47%) of 19 boys with autism examined, nine children (9/19) presented abnormalities (prolonged latencies/interpeak latencies).

However in contrast to the finding of the present study, Hashimoto et al.[28] examined 11 children with autism using SSEP elicited by left median nerve stimulation, noting a prolongation of the interpeak latency of P11–P14, which suggested the presence of brainstem dysfunction.

There was a statistically significant difference in peak to peak amplitude of N20–P25, this was in agreement with the study done by Miyazaki et al.[18] who suggested that most somatosensory dysfunction in autism results from damage to cerebral hemisphere.

The present study showed a significant relationship between SSEP abnormalities and somatosensory symptoms. However four children had abnormal SSEP without somatosensory symptoms, as in previous investigations, patients without clinically detectable sensory loss had abnormal SSEP. One possible explanation is that the SSEP is more sensitive than the clinical examination in detecting decreased sensation.[29]

The recorded two children with hypersensitivity to touch had high peak to peak amplitude of N20–P25. On the other hand children with hyposensitivity to pain, temperature or touch had prolonged interpeak latency of P13/14–N20 and N20–P25. No previous studies focused on this observation. It requires further studies on a large sample of autistic children.

Furthermore, the present study showed no statistically significant relation between the presence of SSEP abnormalities and the severity of autism. Consistent with these finding, Ververi A, et al.[23] did not find any significant association between the presence of abnormal SSEPs and the severity of autism.

From the study, we conclude that children with autism have abnormal SSEP changes and were significantly related to the presence of sensory abnormalities, indicating central cortical dysfunction of somatosensory area. On the other hand, these abnormal SSEP changes were not related to the severity of autism.

Conflict of interest

None declared.

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