Audiologic evaluation and communication disorders in a group of Egyptian children with autistic features

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Objectives
The aim of this work was to perform an audiological evaluation on a group of children with autistic features and to correlate the results with the intelligent quotient, communication skills, and sensory integration function of these children.
Study design
Descriptive cross-sectional study.
Patients and methods
The study was carried out on 25 Egyptian children with autistic features and 25 age-matched and sex-matched typically developing children. All the children's age ranged from 4 to 9 years. All the children were subjected to the following: history taking, basic audiological evaluation, transient evoked otoacoustic emissions, N100, and P300. The children were also subjected to communication assessment, sensory integration dysfunction questionnaire, psychometric evaluation, and the Childhood Autism Rating Scale. The results obtained from the two groups were then compared. In addition, correlation studies were carried out for all the results obtained.
Results
Autistic children presented with normal hearing sensitivities and cochlear function, and delayed N100 and P300 latencies and small P300 amplitudes compared with the control group. There was a significant negative correlation between N100 latency and verbal and nonverbal communication abilities. In addition, there was a significant correlation between P300 latency and amplitude and each of the following: intelligent quotient, the Childhood Autism Rating Scale, and dynamic assessment of verbal and nonverbal communication. The auditory and visual modalities of the sensory integration dysfunction score correlated positively with P300 latency but not amplitude.
Conclusion
The auditory deficits in autism involve controlled attention processes, speed of perceptual classification, and allocation of attention. N100 is a correlate of the level of communication and language development rather than a marker of autism. P300 abnormalities affect verbal and nonverbal communication, mental development, autistic features, and sensory integration function in autism and may be used as a tool to assess the prognosis of autism.

Keywords:
autism, communication skills, N100, otoacoustic emission, P300, sensory integration dysfunction

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Introduction

Autism is one of the pervasive developmental disorders that constitute a group of developmental disorders of the brain, characterized by qualitative impairments in verbal and nonverbal communication, social interaction, and social imagination, with a restricted range of interests and often stereotyped repetitive behaviors and mannerism [1].

The etiology of autism remains unclear primarily due to the fact that it does not result from a single dysfunction but is multifaceted in nature. Consequently, attempts to elucidate the underlying causes of autism have ranged from the identification of abnormalities in the genome to the description of structural/functional abnormalities in the brain [2].

Reports of stimulus-processing abnormalities in autism extend across all sensory modalities. Of the sensory modalities, the most evidently affected by autism seems to be not the visual, but the auditory modality. Some authors have also suggested that auditory abnormalities should be included among the diagnostic criteria of the disorder. In addition, a link has been suggested between the presence of early auditory abnormalities and the development of language and communication deficits in autism. However, the possible relationship between

auditory modality and the characteristic of social and communicative deficits remains poorly understood [3].

Bruneau *et al.* [4] reported that electrophysiological indexes of auditory processing in individuals with autism have provided incomplete and conflicting evidence of normality or dysfunction at various levels of the auditory system. In the studies aimed at determination of the neurobiological mechanisms involved in autism, a variety of neurophysiological tests have been used. The organization of sensory pathways can be assessed by evoked potentials. These are markers of different stages of processing from sensory registration to cognitive processing [5].

Well-functioning auditory skills constitute a solid foundation for the acquisition of speech and language [6,7]. A compromised ability to process and decode the rapid acoustic stimuli that characterize speech results in the most severe language disorders in autism: verbal auditory agnosia or word deafness [8]. Some researchers [6] suggest that children with speech and language deficits have temporal processing deficits, whereas others [9] propose that the deficits arise from impaired phonologic coding. The exact nature of this deficit, however, remains controversial. In an attempt to resolve this problem, we recorded event-related brain potentials, which represents one method to access neural events underlying language processing, which thus yields information not available from behavioral measures [10].

Event-related potentials (ERPs) are particularly useful for studying the relation between cognitive and brain processes, in that they can provide information about the spatiotemporal pattern of brain activity during specific task performance. Thus, ERPs can be used to examine the underlying pathophysiology of deviant cognitive processing in autism [11]. Examples of auditory cortical potentials include N100, P100, P200, and P300.

Despite several large-scale efforts, the relationship between auditory-evoked potentials (AEPs) and autism has not yet been clearly delineated. This is not surprising, given that autism is a complex, genetic disorder with multiple and/or interacting causal factors [5]. We hope that this study sheds light on one of the core aspects of autism; the observed cognitive, behavioral, and communicative manifestations of this mysterious disorder. A complete understanding of autism is needed, not only for prophylaxis for future generations but also for more effective treatment and coping strategies for individuals with this disorder [12].

The aim of this work is to carry out an audiological evaluation on a group of children with autistic features and to correlate the results with the intelligent quotient (IQ), communication skills, and sensory integration function of these children.

Patients and methods

This study was carried out on 50 Egyptian children in the age group of 4–9 years; 25 were healthy typically developing children free from any history of hearing problems or language difficulties and these constituted the control group. The other 25 children had been previously diagnosed with autistic features according to the *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., Text Revision criteria [13] and these constituted the autism group. The autism group fulfilled the following inclusion criteria: normal middle ear function as detected by tympanometry, and mild and moderate autistic features as diagnosed by the Childhood Autism Rating Scale (CARS) [14].

Children in the autism group were selected from the Phoniatric outpatient clinic of Kasr El-Aini Hospital and the Hearing and Speech Institute. The audiological assessment was carried out in the Audiology outpatient clinic of Kasr El-Aini Hospital. This research was carried out between October 2007 and December 2008, and the study protocol was approved by the Otolaryngology Department Council of Cairo University. A written consent to participate in this research was obtained from the children's parents before commencement of the study. Information on age, prenatal, natal, and postnatal and developmental history, in addition to medical reports was obtained through an interview that had been carried out with the parents before the commencement of the study. Thereafter, each child under study was subjected to the following:

(1) *Basic audiological assessment*: Play audiometry using warble tones was performed at frequencies of 500 Hz, 1, 2, and 4 kHz using a dual-channel clinical audiometer (AC 40; Interacoustics, Assens, Denmark) with TDH 39 earphones. Immittancemetry including tympanometry and the acoustic reflex threshold measurement (ipsilateral and contralateral at frequencies 500 Hz, 1, 2 and 4 kHz) was also performed using an Interacoustics AZ-26 middle ear analyzer (Interacoustics) calibrated according to the ISO standards. Immittancemetry was performed to exclude children with middle ear pathologies.
(2) *Transient evoked otoacoustic emissions* (*TEOAEs*): TEOAEs were performed to verify the play audiometry results. This was done using a nonlinear click stimulus in a sound-treated room; each ear was tested separately. Intensity was adjusted to an approximately 80 decibel sound pressure level (dBSPL). Responses to 260 sets of stimuli were assessed in a time window of 20 ms starting 4 ms after stimulation. The responses of stimulus sets were averaged on each of two buffers (A and B). The average amplitude of these two wave forms represented the overall echo level in dB SPL. In addition, by a simple cross-correlation of the two waveforms, the entire reproducibility percent was computed. These two parameters were used in the assessment of the TEOAE response. TEOAE was considered to be present if the entire reproducibility percent was at least 50% [15].

The 'Vivosonic digital processing technique' was used for TEOAE testing using the Vivosonic audiometer model

V-500 (BDT version 4; Vivosonic Inc., Toronto Canada) with ER-3A insert earphones. It is a wireless system for electrophysiological assessment and hearing screening, whose main platform is scalable, and thus did not require sedation of the patients.

(1) *Auditory brainstem response* (*ABR*): The research team in this study had performed a previous research on the correlation between communication skills, sensory integration dysfunction, and ABR results on the same patient and control groups as those used in this research [16]. ABR was performed at a 100 dB normalized hearing level using a rarefaction click. The absolute and interpeak latencies of waves I, III, and V at 20 c/s and wave V′ (70 c/s) were analyzed. The 'Vivosonic digital processing technique' was also used. The details of the testing procedure have been described in the above-mentioned study.

(2) *N100 and P300 ERPs*: ERPs were assessed while the child was sitting comfortably in a sound-treated room after placing circumaural headphones. The child was instructed to count the rare (target) stimulus they heard and report this number after each trial. Younger children or autistic children were asked to raise their hand upon hearing the target stimuli. Each recording was preceded by a training session to ensure that the child had understood the task properly. The stimuli were presented within an 'odd-ball paradigm' using a tone burst stimulus of 250–300 ms with the frequent stimulus (1000 Hz) and the rare (target) stimulus (4000 Hz). Responses to 30 target infrequent tones and 70 frequent tones were obtained. The stimuli were presented at a rate of 0.5/s at a level of 90 dB SPL. Recording electrodes were placed at Cz, Fz, and Pz electrode sites of the 10–20 international system of electrode placement. P300 was identified as the maximum positive going deflection between 250 and 700 ms and N100 was identified as the negative deflection at about 100 ms. Latency was measured from the time of the stimulus onset to the highest peak of the wave. Amplitude was measured at the highest peak relative to the pre stimulus baseline. ERPs were assessed using an evoked response audiometer (Amplaid model MK 12; Amplaid, S.A.E., Milan, Italy).

(3) *Psychometric evaluation*: The IQ of each child was calculated using the Stanford Binet Intelligence scale [17]. The distribution of children with autism according to IQ was as follows: 50% mild mental retardation, 40% borderline IQ, 5% below average IQ, and 5% average IQ.

(4) *The Childhood Autism Rating Scale* [14]: CARS was used to diagnose and rate the severity of autism. The distribution of autistic patients according to CARS was as follows: 50% had mild autistic features and 50% had moderate autistic features. Children with severe autistic features were excluded from this study to facilitate cooperation during test procedures.

(5) *Assessment of communication skills*: Communication assessment included the evaluation of the following:
   (a) The ability of the child to use signals to interact with others socially (11 items).
   (b) The forms used by the child to indicate his needs (20 items).
   (c) Behavior (positive or negative behaviors that are socially unacceptable (two items).
   (d) Intentionality: to what extent the child uses his or her signals to intentionally indicate his needs (six items).
   (e) Readability of communication behaviors in terms of movement clarity, frequency, and consistency, how well people understand the child, and to what extent he or she can use repair strategies (three items).
   (f) Capacity of symbols, verbal or nonverbal (four items).
   (g) Vocal and motor imitation (two items)
   (h) Reasoning: understanding object function and using tools for solving problems (two items) [18].
   For each of the constituent items, each child was assigned a score of 0–3 depending on the level of performance of the child; scores were graded in an ascending order from 0 to 3. Thereafter, the constituent items of each section were summed up to obtain the score for this section, for example behavior score, intentionality score, etc. These scores were then summed up to obtain the total communication score [16].

(6) *Sensory integration dysfunction questionnaire*: The sensory integration dysfunction questionnaire included questions addressing dysfunctions in the following sensations:
   (a) Auditory dysfunction: e.g., negatively responds to unexpected noises (eight items).
   (b) Visual dysfunctions: e.g., tilts head/close one eye to look at an object (nine items).
   (c) Olfactory–gustatory dysfunction: e.g., mouths/chews on nonfood object or makes excessive use of smell to explore (eight items).
   (d) Vestibular dysfunction: e.g., gets car sick or dizzy easily (eight items).
   (e) Tactile dysfunction: e.g., dislikes being touched even in a friendly and affectionate way (eight items) [19,20].
   For each of the constituent items, each child was given a score of 0–2 depending on the frequency of occurrence of the behavior demonstrating a certain sensory dysfunction (0 = never, 1 = sometimes, and 2 = frequently). Thereafter, the constituent items of each sensation were summed up to obtain the dysfunction score for this sensation, for example, auditory dysfunction score, visual dysfunction score, etc. All these were then summed up to obtain the total sensory dysfunction score [16].

Statistical analysis

An IBM-compatible personal computer was used to store and analyze the data. Calculations were carried out using the statistical software package for the social sciences

(SPSS, version 10; Armonk, New York, USA). Data were tabulated and statistically analyzed to evaluate the differences between the groups under study in terms of various parameters. Comparison of autistic children and the control group in terms of hearing threshold levels, TEOAE, N100, P300, sensory integration dysfunction score, and dynamic assessment of verbal and nonverbal communication was performed. The correlation between the ABR results (from the fore-mentioned study) with N100 and P300 results was assessed. In addition, correlations between the N100 and P300 results with age, IQ, CARS, sensory integration dysfunction score, and dynamic assessment of verbal and nonverbal communication were assessed. Statistical analysis included the arithmetic mean, SD, Hypothesis Student's (t) test, and Pearson's correlation coefficient. The correlation between variables was assessed using the Pearson correlation test. This test determines whether the changes in one variable are accompanied by a corresponding change in the other variable. A significant correlation may be positive, indicating that the change in the two variables is in the same direction, or negative, indicating that the change in the two variables is in the opposite direction. The results were considered nonsignificant if P value was greater than 0.05, significant if P value was less than 0.05, and highly significant if $P<0.01$.

Results

All autistic children included in this study had normal hearing thresholds in both the ears as indicated by the Play audiometry test. Moreover, comparison of their thresholds with the control group showed no significant differences at all test frequencies ($P>0.05$). Their normal peripheral auditory function was confirmed by TEOAE results.

TEOAE response (dB SPL) and reproducibility percent (%) showed a statistically nonsignificant difference ($P>0.05$) between the patients and the controls. The TEOAE response was 13.794 ± 5.261 and14.618 ± 4.298 in the patient and the control groups, respectively (t-test = 0.61). Reproducibility (%) was 50.641 ± 6.593 and 60.112 ± 21.273 in the patient and the control groups, respectively (t-test = 1.44).

The N100 latency results and the P300 amplitude and latency results (demonstrated in Table 1) showed a highly statistically significant difference between the patients and the controls ($P<0.01$).

Comparison of the sensory integration dysfunction score between the autistic and the control groups showed a highly statistically significant difference in the auditory and visual modalities and the total score ($P<0.01$) and a statistically significant difference in the olfactory, vestibular, and tactile modalities ($P<0.05$).

Dynamic assessment of verbal and nonverbal communication between autistic and control groups showed a highly statistically significant difference between both the groups on all items of assessment ($P<0.01$).

Correlation between audiological results and various parameters under study

Results of the auditory brainstem response correlation coefficient

From the results of the research mentioned in the previous section [16] on the correlation between communication skills, sensory integration dysfunction, and ABR results, it was evident that ABR waves III, V, and interpeak latencies III–V (20 c/s) were significantly affected in children with autistic features. These parameters were correlated with the results of N100 and P300 obtained in the present research on the same sample population. Table 2 shows a nonsignificant correlation between ABR latencies (waves III, V and waves III–V at 20 c/s), N100, and P300 amplitude and latency in the autistic group.

N100 correlation coefficient results

Table 3 shows a nonsignificant correlation in the results of N100 latency, age, IQ, and CARS in the autistic group. In addition, the correlation coefficient between the N100 latency and the total sensory dysfunction score showed a nonsignificant correlation ($r = 0.192$).

The correlation coefficient between the N100 latency results and the dynamic assessment of verbal and nonverbal communication showed a highly significant negative correlation in the autistic group in all items of assessment ($P<0.001$) (Table 4). In other words, an increase in the dynamic assessment score is accompanied by a decrease in N100 latency.

P300 correlation coefficient results

The correlation coefficient between the P300 amplitude and latency and N100 latency results in the autistic group was nonsignificant (Table 5).

Table 3 shows that a nonsignificant correlation exists between P300 amplitude, latency, and age in the autistic group. However, a highly significant positive correlation between P300 amplitude and IQ and a highly significant negative correlation between P300 latency and IQ were also found. Considering the relation between P300 results and CARS, the P300 amplitude showed a highly significant negative correlation with CARS, whereas the P300 latency showed a highly significant positive correlation with CARS. Hence, a decrease in IQ is associated with a smaller and delayed P300 response, whereas the decrease in CARS is accompanied by a larger and earlier P300 response.

Table 6 shows that there was no significant correlation between the P300 amplitude results and each of the sensory modalities and the total score in the sensory dysfunction questionnaire.

However, P300 latency results revealed a highly significant positive correlation with the auditory modality and a significant positive correlation with the visual modality and no significant correlation with the olfactory, vestibular, tactile modalities, and the total score of the sensory dysfunction questionnaire. This means that an increase in the auditory and visual dysfunction scores is associated with more delayed P300 latencies.

Table 1 N100 latency results and P300 amplitude and latency results of autistic patients as compared with the control group

	Mean ± SD			
	Patient's group	Control group	*t*-test	Significance
N100 latency (ms)	174.321 ± 6.672	97.548 ± 7.012	39.85	HS
P300 amplitude (μV)	5.158 ± 2.558	12.908 ± 2.009	11.91	HS
P300 latency (ms)	376.404 ± 11.868	295.156 ± 31.828	11.96	HS

HS, highly significant ($P<0.01$).

Table 2 Correlation coefficient between auditory brainstem response latencies (ms) and N100 latency (ms) and P300 latency (ms) and amplitude (μV) results in the autistic group

ABR	N100 latency correlation (*r*)	Significance	P300 amplitude correlation (*r*)	Significance	P300 latency correlation (*r*)	Significance
Wave III	0.264	NS	−0.214	NS	0.344	NS
Wave V	0.127	NS	−0.309	NS	0.233	NS
Waves III–V	0.167	NS	−0.241	NS	0.230	NS

ABR, auditory brainstem response; −, negative correlation.
NS, nonsignificant ($P>0.05$).

Table 3 Correlation coefficient between N100 latency, P300 amplitude and latency, age, intelligent quotient, and Childhood Autism Rating Scale in the autistic children

	Age correlation (*r*)	Significance	IQ correlation (*r*)	Significance	CARS correlation (*r*)	Significance
N100 latency (ms)	−0.184	NS	−0.121	NS	0.125	NS
P300 amplitude (μV)	0.121	NS	0.913	HS	−0.978	HS
P300 latency (ms)	−0.142	NS	−0.895	HS	0.976	HS

CARS, Childhood Autism Rating Scale; IQ, intelligent quotient; −, negative correlation.
HS, highly significant ($P<0.01$); NS, nonsignificant ($P>0.05$).

Table 4 Correlation coefficient between N100 latency (ms), P300 amplitude (μV), and latency (ms) results and dynamic assessment of verbal and nonverbal communication in the autistic group

Dynamic assessment	N100 latency correlation (*r*)	P300 amplitude correlation (*r*)	P300 latency correlation (*r*)
Section I Survey of functions/meanings	−0.942**	0.952**	−0.969**
Section II Forms used to indicate specific requests/needs/emotions	−0.901**	0.991**	−0.976**
Section III Behaviors	−0.869**	0.956**	−0.967**
Section IV Intentionality	−0.924**	0.958**	−0.960**
Section V Readability of communication behaviors	−0.504**	0.491*	−0.525**
Section VI Capacity of symbols	−0.703**	0.689**	−0.693**
Section VII Imitation	−0.924**	0.958**	−0.960**
Section VIII Reasoning	−0.754**	0.831**	−0.632**
Total	−0.934**	0.991**	−0.990**

−, negative correlation.
*Significant ($P<0.05$).
**Highly significant ($P<0.01$).

Table 5 Correlation coefficient between P300 amplitude (μV) and latency (ms) and N100 latency (ms) results in the autistic group

P300	N100 correlation (*r*)	*P* value	Significance
Amplitude	−0.179	>0.05	NS
Latency	0.170	>0.05	NS

−, negative correlation.

P300 amplitude showed a positive highly significant correlation with the dynamic assessment of verbal and nonverbal communication, except section V, which showed a positive significant correlation, whereas P300 latency showed a highly negative significant correlation with the dynamic assessment of verbal and nonverbal communication (Table 4). In other words, the decrease in all communication scores is associated with a smaller and delayed P300 response (as IQ).

Discussion

Autism is a pervasive developmental disorder that affects sensorimotor and cognitive domains [21]. It is known that

Table 6 Correlation coefficient between P300 amplitude (μV) and latency (ms) results and sensory integration dysfunction score in the autistic children

Sensory dysfunction score	P300 amplitude correlation (*r*)	Significance	P300 latency correlation (*r*)	Significance
Auditory	−0.128	NS	0.552	HS
Visual	−0.128	NS	0.448	S
Olfactory	−0.031	NS	0.324	NS
Vestibular	−0.066	NS	0.206	NS
Tactile	−0.066	NS	0.206	NS
Total	−0.043	NS	0.299	NS

−, negative correlation.
HS, highly significant ($P<0.01$); NS, nonsignificant ($P>0.05$); S, significant ($P<0.05$).

autistic patients have an impaired ability to regulate auditory input [4]. The mechanisms underlying such phenomena and the possible relationship between these and the characteristic social and communicative deficits remain poorly understood [3]. The possible disorders that can affect various levels of auditory processing can be evaluated by studying AEPs [22]. This led us to attempt the determination of the level of auditory disorder through AEPs and assess its relation to communication abilities and sensory integration deficits in children with autistic features.

We studied the sensory processing of the auditory modality as it is the most evident sensory modality affected by autism [3]. There is also increasing evidence that abnormal cortical processing of auditory stimuli is one of the core deficits in autism [2]. A comprehensive audiological assessment is a key component in the differential diagnosis of children both at risk for autistic spectrum disorder as well as for those diagnosed with the disorder [23].

Play audiometry performed for a number of autistic children was within normal hearing thresholds. In addition, there was no evidence of peripheral auditory dysfunction in children on the autistic spectrum as indicated by TEOAEs. Thus, aberrant processing of auditory input at this level of the auditory system (peripheral level) does not appear to contribute to the auditory processing disorders reported in individuals with autism.

These results suggest that outer hair cell activity is not different between the two groups. Thus, the cochlear mechanism responsible for the sensitivity, frequency resolution, and dynamic range of the normal ear appears to be functioning identically in children with autism and control children [24]. Previous researches have yielded similar results [24,25].

However, our otoacoustic emission (OAE) results were in disagreement with the findings obtained by Ornitz [26], who suggested that in autism, the locus of dysfunction lies at the interface between peripheral processing and information processing and that the dysfunction that affects all input modalities and processing abnormalities at the peripheral level would result in distortions in further information processing at all levels of the system. Tas *et al.* [27] suggested that hearing loss may be more common in children with autism than in typically developing children and that autism can occur with permanent hearing loss. Rimland and Edelson [28] suggested that aversive behaviors in autism reflect atypical auditory processing at a peripheral level.

A study that compared the suppression of TEOAEs in high-functioning autistic children with typically developing control participants revealed that all participants in both the groups suppressed TEOAEs under all noise conditions, consistent with the intact function of the olivocochlear efferent auditory pathways [29].

On the basis of our OAE, N100, and P300 results (Table 1), we can postulate that there is no causal relationship between peripheral processing (OAE) and central processing dysfunction (delayed latency of N100 or P300 and decreased amplitude of P300). No systematic overview is available of how, in the same individual, a dysfunction at one level of the auditory pathway affects the processing of auditory information at another level, either above or below it [24].

These findings are important in our studies of higher-level auditory processes. With the knowledge that the auditory periphery is intact, any deficit in auditory abilities can be more accurately attributed to deficits at other levels of the auditory pathway in children on the autistic spectrum. On the basis of our OAE results, there is no evidence of a deficit specific to the auditory function in children with autism located at the level of the middle ear or the cochlea. Emerging evidence suggests that atypical behaviors in response to sound represent a perceptual disorder mediated at higher levels of the auditory system [24].

The authors of this study had carried out a previous research on the correlation between communication skills, sensory integration dysfunction, and ABR findings in a group of children with autistic features (same sample population used in this study) [16]. The results revealed a delay in brainstem propagation, mainly involving the later waves. The effect of autism was mainly on ABR wave latencies and not on amplitudes. The brainstem dysfunction present in autistic children affects communication and sensory integration functions, and hence affects the process of coordination, attention, and arousal, which are part of sensory integration function.

On the basis of these results, a correlation between the ABR results from the previous study and N100 latency and P300 latency and amplitude from the current study was assessed and no significant correlations were found (Table 2). This confirms the notion that autism does not

necessarily affect the entire auditory pathway in the same pattern. This was confirmed from the results of N100 and P300 with CARS (Table 3).

The high reliability of the temporal N100 wave in normal children and the cortical areas involved in its generation make it a potentially relevant index to evaluate cortical auditory processing in autism [4]. N100 results revealed a latency delay in autistic children compared with age-matched normal children (Table 1). This delay indicates that autism is related to ineffective regulation of auditory sensory input [2]. These results argue for dysfunction in the brain areas involved in N100 generation, that is, the auditory associative cortex in the lateral part of the superior temporal gyrus. This dysfunction may reflect slower transmission of information in neuronal pathways and/or in synaptic connections in the secondary auditory cortex in autism [4]. As N100 reflects conscious perception of sound and may also represent some kind of detection or attention-triggering process, delayed N100 latency may indicate failure to allocate attention [10]. Our results support the previous findings of other researches [4,11,30]. In contrast, some studies have reported similar N100 latencies in autistic children compared with controls [31,32], whereas others have reported shorter latencies [33].

In this study, we found that N100 latency does not change with age in autistic children (Table 3). Pang and Taylor [34] postulated that N100 latency changes with age in normal children. Bruneau *et al.* [4] reported that in normal children, the younger the child, the longer the N100 peak latency, due to the immaturity of the mediating structures (different level of myelination, different neurotransmitter activity, etc.). Our findings could therefore indicate a maturational delay of the cortical structures involved in the generation of the N100 wave in autism as N100 does not change with age.

Several studies have found that the N100 latency of the auditory event-related potential is longer in autistic than in normal children. The present study examined whether this characteristic has any relationship with the degree of language impairment, level of intellectual ability of autistic children, autistic features, and sensory dysfunction.

There was no correlation between N100 latency and IQ (expresses mental development) (Table 3). This may indicate that N100 does not reflect processes that are essential to mental development. In addition, there was no correlation between N100 latency and CARS and the total sensory dysfunction scores (Table 3). This may be explained by the fact that N100 does not reflect processes that are essential to behavior [35]. However, correlation studies showed that the longer the latency, the lesser the verbal and nonverbal communicative abilities (Table 4). This may suggest that the abnormal cortical processing in the areas that generate N100 is responsible for communication deficits but not for other autistic features in autism. This finding is supported by the findings of Bruneau *et al.* [4], who suggested that interpretation of atypical N100 is a correlate of the level of language development, rather than a marker of autism *per se*.

In the study by Bruneau *et al.* [4], the pattern of N100 dysfunction suggested that autistic individuals process both verbal and nonverbal auditory stimuli only in the right cerebral hemisphere. This is consistent with other nonelectrophysiological evidence indicating right hemisphere dominance in the processing of verbal and nonverbal auditory stimuli by autistic individuals. In another experimental group, Dawson *et al.* [36] compared ERPs in response to a simple speech stimulus (da) to assess hemispheric activity in three groups of children. In the normal group, only the N100 differed significantly between the two hemispheres (left > right); no hemispheric difference was found in the language-impaired group or the autistic group. Taken as a whole, these results support the hypothesis of role reorganization of the left and right hemispheres for auditory processing during early brain development in individuals with autism rather than attributing dysfunction only to the left hemisphere.

Our P300 results showed that the autistic children showed signs of deficient late-stage auditory perceptual processing (Table 1). The delayed latency indicates that more time was required to complete stimulus evaluation in the auditory modality at the higher processing level. Another explanation by Polich [37] suggested that P300 latency reflects the speed of perceptual classification. Thus, prolonged P300 latency implies that the process of evaluating and categorizing auditory stimuli was more time consuming for these autistic children. Abnormally small P300 amplitudes are a reflection of abnormal functioning of the neuronal generators (temporal, parietal, and frontal areas), as these need to be intact in order for the P300 to attain normal levels [38]. Similar P300 amplitude results have been reported in other studies [31,39,40]. This abnormal functioning was discussed in studies of brain perfusion at rest. A PET study in autistic children showed hypoperfusion in the left superior temporal gyrus (Brodmann's area; BA 22/42), in the right superior temporal gyrus (BA area 22/42), and in the right superior temporal sulcus (BA area 21) [40].

As P300 is generally considered to index controlled attention processes, where latency reflects speed of perceptual classification, and amplitude the allocation of attention [10], some explanations were provided for P300 amplitude attenuation. The first speculates that P300 is considered to reflect a limited capacity mechanism whereby attention is consciously allocated to specific information in the environment. Thus, the reduced P300 amplitude may reflect either a failure to allocate appropriate attention to stimuli or a misallocation of attention resources to less important stimuli [2]. In the second speculation, it was argued that P300 amplitude is inversely related to stimulus probability (i.e. a low probability unexpected stimulus elicits a large P300 amplitude). A smaller P300 amplitude may reflect either a difficulty in attaching significance to unexpected stimuli [33] or a defect related to the modification of

expectancies on the basis of previous experience. This may explain why individuals with autism, who have rigid expectations and thus difficulty in extracting information in a way that leads to a reintegration of previously learned information, have smaller P300 amplitudes [41].

However, our P300 results are in disagreement with the findings of Ferri *et al.* [42], who reported higher P300 amplitude in autistic individuals than in normal controls during childhood. They reported similar P300 amplitudes for unattended novel sounds in individuals with autism and mental retardation (aged 6–19 years) and their peer controls. Salmond *et al.* [43] reported that individuals with autism who have average-range cognitive abilities have typical P300 responses.

This study suggests no causal relationship between the long latency of auditory N100 and the smaller P300 amplitude or increased latency (Table 1). This is because P300 and other long latency ERP components are dependent on some aspects of meaning, significance, or implications of an event for the individual, not on the physical properties, *per se*, as is true of N100 [44].

No evidence in this study was obtained to suggest that children with autism may show a change in central auditory processing (measured by P300) with age (Table 3). Our results are in disagreement with Hoeksma *et al.* [38], who reported that children with autism showed a smaller P300 amplitude than controls, whereas adolescents showed no P300 abnormalities. In normal children, a study by Musiek *et al.* [45] showed decreased latency and increased amplitude of P300 from approximately 5 years of age through the mid-teens, indicating an increasing efficiency in information processing with maturity. This could be explained by Nieto Del Rincón [9], who hypothesized that the neurodevelopment process, such as increased myelination and dendritic arborization, may underlie the changes in P300 latency and amplitude observed in normal children and hence this process may be impaired in autistic children.

This study showed a statistically significant correlation between P300 amplitude (positive correlation) and latency (negative correlation) and the level of intellectual ability of autistic individuals (IQ) (Table 3). This may indicate some cognitive abnormality [46]. In addition, our research showed a correlation between P300 (index of attention) and autistic features (measured by CARS) (Table 3). The literature suggests that attentional abnormalities are a rather common finding in autism and may contribute to the clinical features of the disorder [38]. For example, the clinical observation of heightened reactivity to seemingly meaningless stimuli may be an indication of increased distractibility, whereas the reduced interest and repetitive behaviors may be a representation of a deficit in attentional shifting. However, our results were in disagreement with the Belmonte and Carper [35] theory that P300 does not reflect processes that are essential to behavior.

This research showed a significant positive correlation between P300 latency (index of attention) and the auditory and visual modalities of the sensory integration dysfunction score (Table 6). Hence, attentional and arousal impairments could be related to impairments in modulating sensory input (sensory integration). Affection of the visual modality and its correlation to auditory P300 recording may be attributed to the fact that there are multisensory links between visual, auditory, and tactile processing in autism. If early visual experience shapes the development of tactile and auditory perception, then impairments in tactile or auditory experience may equally affect visual perception. This indicates that children with autism have multimodal sensory difficulties [47].

Impaired communication abilities were related to lesser amplitude and a longer latency of P300 (Table 4). This is further supported by the finding that P300 attenuation and delay is specific to the auditory modality as shown by negative findings for visual experiments using letters and written phrases [48]. It has been suggested that individuals with autism show atypical left hemispheric activation during the processing of auditory linguistic stimuli using the P300 response [36].

This study showed significant differences in the sensory integration dysfunction score (especially the auditory domain) between autistic children and their normal peers. Given the prevalence of these findings and their early onset, sensory processing disorders may represent another core diagnostic criterion for autism, a view that is supported by some authors [49]. In addition, our research confirmed that communication deficits are one of the core symptoms of autism [50], as evidenced by the results of the dynamic assessment of verbal and nonverbal communication.

We can conclude from this study that our sample of autistic children did not present with peripheral hearing loss. There is no evidence of a deficit located at the level of the cochlea in autism. The auditory deficits in autism are more consistently manifested in higher aspects of processing that involve controlled attention processes, speed of perceptual classification, and allocation of attention. N100 is a correlate of the level of communication and language development rather than a marker of autism. P300 abnormalities affect verbal and nonverbal communication, mental development, autistic features, and sensory integration function in autism and may be used as a tool to assess the degree of severity of autism. Attention is affected in autism and may be responsible for many autistic features. There are maturational deficits evident at higher centers of the auditory pathway in autism. Sensory processing problems in autistic children may have an impact on intelligence and on autistic features.

We recommend the use of N100 as a valuable tool to evaluate the prognosis of communication intervention programs. In addition, P300 can be used as a prognostic method to investigate differential responding to various interventions and provides measures to objectively subcategorize children on the autistic spectrum.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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