

Electrophysiological abnormalities of spatial attention

in adults with autism during the gap overlap task

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[Acknowledgements]

This study was supported by grants from the Ministry of Health, Labour, and Welfare (Health and

Labour Sciences Research Grants, Research on Psychiatric and Neurological Diseases and Mental Health, H17-kokoro-ippan-004 to KK and NK), and from the MEXT (Grant-in-Aid for Scientific Research for Young Investigators, No. 17790806 to YK), Japan.

[Keywords]

autism; spatial attention; event-related potentials; saccadic eye movement

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Abstract

Objective: We evaluated event-related potentials (ERPs) elicited by attentional disengagement in individuals with autism.

Methods: Sixteen adults with autism, 17 adults with mental retardation and 14 healthy adults participated in this study. We recorded the pre-saccade positive ERPs during the gap/overlap task under which a peripheral stimulus was presented subsequent to a stimulus in the central visual field. Under the overlap condition, the central stimulus remained during the presentation of the peripheral stimulus and therefore participants need to disengage their attention intentionally in order to execute the saccade to the peripheral stimulus due to the preservation of the central stimulus.

Results: The autism group elicited significantly higher pre-saccadic positivity during a period of 100 to 70 msec prior to the saccade onset than the other groups only under the overlap condition. The higher amplitude of pre-saccadic positivity in the overlap condition was significantly correlated with more severe clinical symptoms within the autism group.

Conclusions: These results demonstrate electrophysiological abnormalities of disengagement during visuospatial attention in adults with autism which cannot be attributed to their IQs.

Significance: We suggest that adults with autism have deficits in attentional disengagement and the physiological substrates underlying deficits in autism and mental retardation are different.

Introduction

Autism is diagnosed on the basis of behavioral and developmental features such as impairment of reciprocal social interaction, communication and imagination, and the presence of repetitive and ritualistic behavior. It has been recently pointed out that scientific merits from studies using a comprehensive model assuming a single explanation for autism may be limited, since various behavioral features of autism are largely independent in terms of genetic background as shown by twin cohort studies (Happé et al., 2006). Thus, a bottom-up type approach which explores abnormal or preserved functions in relatively fundamental cognitive processes based on simple cognitive models may be important in the autism research strategy. Researchers have proposed that attentional abnormalities underlie the behavioral features of autism such as inflexibility, repetitive behavior and overselectivity (Lovaas et al., 1979; Casey et al., 1993; Townsend et al., 1996,2001; Wainwright and Bryson, 1996, Senju et al., 2004). Auditory attention has been intensively studied in the literature, particularly using event-related potentials (ERPs). For example, a reduced P300 amplitude (Oades et al., 1988; Lincoln et al., 1993) and abnormal mismatch negativity (Gomot et al., 2002; Kasai et al., 2005) has been reported. On the other hand, spatial attention has been also implicated in the pathophysiology of autism, which has been understudied.

Spatial attention is a function of spatially directed attention and spatial selectivity (Allport,

1989). Posner and Cohen (1984) outlined a model in which the successive components of spatial attention were defined as disengagement, shift, and engagement of attentional sources. In Posner's cuing paradigm, a target stimulus follows a cue stimulus presented on the same side of a target (valid condition) or on the contralateral side of a target (invalid condition). This task makes it possible to investigate the ability to disengage attention from one location and shift attention to its contralateral location. Findings from patients with acquired brain damage suggest that these components are associated with specific brain areas, i.e., disengagement is associated with the parietal cortex, shift is related with superior colliculus, and engagement is associated with thalamus (Posner and Cohen, 1984; Posner and Petersen, 1990).

Most studies on visuospatial attention in adults with autism have used the cuing paradigm that elicits the re-orienting of the direction of the attention (Posner, 1980) and reported behavioral problems in attentional disengagement and shift (Casey et al., 1993; Townsend et al., 1996, 2001; Wainwright and Bryson, 1996), but findings of other studies using eye gaze cue have been mixed (Senju et al., 2004; Swettenham et al., 2003; Kylliäinen and Hietanen, 2004). Other studies reported that autistic children presented the difficulties disengaging attention from the salient object (Hughes & Russell, 1993) and mental concepts (Hughes et al, 1994). On the other hand, studies using the gap paradigm (Saslow, 1967) have shown impairment of attentional engagement in children (van der Geest et al., 2001) and adults (Kawakubo et al., 2004) with autism. In the

gap/overlap task, when a temporal gap is introduced between the disappearance of a central fixation point and the appearance of a new target stimulus, the saccade reaction times are reduced compared to when no gap is introduced (gap effect; Saslow, 1967). This difference in saccade reaction times has been explained by the difference in attentional disengagement (Fischer and Weber, 1993). In the gap condition, in which an initial fixation point disappears before a target appears, attention on the fixation point is disengaged automatically. On the other hand, in the overlap condition, in which the fixation point remains after the target appears, attention on the fixation point is disengaged due to the appearance of the peripheral target stimulus. Therefore, the saccade reaction times in the overlap condition are longer than in the gap condition. It cannot be concluded, however, that individuals with autism show only impairment of attentional engagement but not of disengagement in the gap paradigm. If the engaging of attention to the central fixation point is enhanced, they may show impairment of attentional *disengagement*. In order to clarify whether individuals with autism have an impairment of attentional disengagement in the gap paradigm, further examination under conditions in which attentional engagement to the central visual field is ensured is necessary.

Moreover, little is known of the *physiological* substrates for these visuospatial attentional deficits in autism. In the visual ERP literature, P300 amplitude of individuals with autism under a condition where the target stimulus was presented in the center of visual field was comparable to

that of normal controls (Pritchard et al., 1987; Ciesielski et al., 1990). On the other hand, when the stimulus was presented at various peripheral locations individuals with autism were associated with smaller-than-normal visual P300 amplitude (Kemner et al., 1999; Townsend et al., 2001). To our knowledge, however, no previous studies have directly examined ERPs elicited by attention disengagement in individuals with autism.

Csibra et al. (1997) and Gómez et al. (1996) analyzed saccade-locked ERP during the gap/overlap task in *healthy* adults, and found a slowly developing pre-saccadic positivity elicited at central-parietal electrode sites that was followed by a pre-saccadic spike potential immediately preceding the eye movement. Csibra et al. (1997) concluded that attentional disengagement in the gap/overlap task was reflected in these parietal positive ERP components.

Thus, the goal of this study was to assess the pre-saccade positive ERP components during the gap/overlap task as an electrophysiological index of attentional disengagement in adults with autism. To ensure attentional engagement to the central visual field, our task was different from a typical format of the gap/overlap task in some points that various pictures were used as stimuli instead of a cross and a square and participants were required to discriminate the stimuli. Moreover, to clarify whether the physiological dysfunction of visuospatial attention was specific to autism rather than being attributable to general intellectual disability, we compared the data from adults with autism with those of IQ-matched adults with mental retardation. Previous

studies (Csibra et al., 1997, Gomez et al., 1996) indicated that the pre-saccadic positivity was higher in the overlap condition than in the gap condition. Thus, it is assumed that the pre-saccadic positivity reflects the resource that is needed to disengage the attention. Accordingly, we hypothesized that the autistic group would exhibit impairment in attentional disengagement under the overlap condition that would be reflected as higher or longer pre-saccadic positivity detected in the saccade-locked ERPs. Moreover, we predicted higher presaccadic positivity would be associated with severer autistic symptoms.

Method

Participants

Sixteen adults with autism (11 men and 5 women; mean age [SD], 29.0 [6.5]; mean IQ [SD], 43.6 [14.7]), 17 adults with mental retardation (12 men and 5 women; mean age [SD], 27.5 [5.0]; mean IQ [SD], 40.6 [10.9]), and 14 healthy adults (6 men and 8 women; mean age [SD], 28.5 [5.8]; mean IQ [SD], 105.2 [10.9]) participated in this study. Age (one-way ANOVA, $p=0.70$) and gender (chi-square test, $p=0.23$) were not significantly different among groups. IQs were evaluated with the Wechsler Adult Intelligence Scale-Revises (WAIS-R) for two of autism participants and with the Tanaka-Binet Test for eleven of autism participants and all individuals with mental retardation. For healthy adults IQs were estimated by four subtests of the WAIS-R.

Autism subjects and mentally-retarded subjects did not significantly differ in IQ (t-test, $p=0.52$).

Diagnosis of autism was determined according to DSM-IV criteria and Childhood Autism Rating Scale (CARS: Schopler et al., 1988) by a trained pediatric psychiatrist (clinical experience > 5 years; KW, HK, or MI). We used a CARS score of 27 as the cutoff point (Mesibov et al., 1989).

Autism subjects had significantly higher CARS scores than mentally-retarded subjects (34.1 [SD=4.1] versus 20.3 [3.1]; t-test, $p<0.001$). All participants had normal or corrected to normal vision and did not have any chromosomal or neurological disorders. All participants were right-handed determined using the Edinburgh Inventory. The ethical committee of the Faculty of Medicine, University of Tokyo, approved this study (No. 629). Written informed consent was obtained from all participants and their parents before the experiment.

Stimuli and Apparatus

The stimuli were presented on a 21-inch CRT display with a black background by using STIM software (Neuroscan, Inc.). To encourage the motivation of the participants, we used as stimuli 13 illustrations that consisted of pictures of animals (e.g., dog, frog), daily goods (e.g., glasses, baggage) and vehicles (e.g., bicycle, car), etc. In place of a fixation point a central stimulus (picture stimulus as above) was presented in the central visual field. Peripheral stimuli were presented 14 degree (deg.) to the left or right of the central stimulus. All stimuli were the

same angular size; 1.3 X 1.3 deg.

Please insert Figure 1 about here.

Procedure

Participants were seated 0.60 m away from the CRT monitor with their chin on a chinrest.

Participants initiated each trial by pressing a start button, and after 1000 msec a central stimulus was presented in the center of the CRT monitor. A peripheral stimulus was then presented to the left or right side of the central stimulus for 2000 msec. To minimize the possibility that participants anticipate the timing of the peripheral stimulus onset, the central stimulus was presented at intervals randomly varying between 700 and 1500 msec. To make the task clearer to participants, we instructed them to press a button when a target stimulus, the drawing of a dog, appeared as either the central stimulus or the peripheral stimulus. In some trials, the dog was presented both in the center and the peripheral visual field. Thus, even if the dog was presented in the center, saccades were required. Since it was difficult to ask button pressing at an appropriate timing for three subjects, these subjects were alternatively asked to speak aloud the name of the target stimulus. It was necessary for participants to move their eyes to the peripheral stimulus when it was presented, in order to discriminate the target stimulus from the other

stimuli.

In the gap condition, the central stimulus disappeared 200 msec before the peripheral stimulus was presented, and in the overlap condition, the central stimulus remained during the presentation of the peripheral stimulus. Practice trials were given to each participant to ensure that the instructions had been understood. After the practice trials, 180 trials composed of 45 trials of each of the four conditions ([Gap vs. overlap] X [left- vs. right-side presentation on the monitor]) were presented in a random order in three blocks of 60 trials each. The probability of the appearance of target stimulus was 20%.

Recordings

The scalp electroencephalogram (EEG) was recorded according to the international 10-20 system using Ag/AgCl electrode caps (Neuroscan, Inc.) at 16 electrode sites (F3, Fz, F4, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, Oz, O2) referred to linked earlobes. The horizontal electrooculogram (EOG) was recorded at the outer canthi of the both eyes and the vertical EOG was recorded from electrodes placed below and above the left eye. The bandpass filter was set at 0.15-30 Hz and the sampling rate was 500 Hz. EEGs and EOGs were analyzed using SCAN system with SynAmps (Neuroscan, Inc.).

Data analysis

A saccade was defined as the first moment at which the velocity of the eye exceeded 22 deg/sec. Trials were excluded from further analysis if: 1) the target stimulus was presented in that trial, 2) the eyes moved before the peripheral stimulus onset, and 3) the saccade occurred in the incorrect direction or saccadic reaction times (SRTs) were less than 80 msec. Express saccade was defined as saccade that occurred between 80 and 130 ms after the presentation of the peripheral stimulus (Fischer and Weber, 1993). Trials with EEG range exceeding 100 μ V at any EEG electrode were rejected as artifact. We used the 50-msec period starting 150 msec before the saccade onset as the baseline as no salient activities at any electrode in this period. The four conditions (i.e., [Gap vs. overlap] X [left- vs. right-side presentation on the monitor]) were averaged separately. The mean number of accepted sweeps was above 20 for all conditions and for all groups, and did not significantly differ between conditions or between groups. Pre-saccadic positivity was defined as a slowly developing positivity 100 msec before the saccade onset, and the pre-saccadic spike potential was defined as a sharp positivity peaking immediately before the saccade onset.

For the group comparison of the SRT, repeated measures ANOVA was performed with the group (autism, mental retardation, control) as the between-subject factor, and the condition (gap, overlap) and the presentation side (left, right) as within-subject factors. For all repeated measures ANOVAs, the sphericity assumption was tested using Mauchley's test, and Greenhouse-Geisser

correction was performed when there was a significant violation of the sphericity assumption.

Tukey's honestly significant difference test was used for post-hoc multiple comparisons.

To compare the time course of the pre-saccadic positivity, the averaged amplitude was calculated within 30 msec windows from 100 msec to 70 msec and from 70 msec to 40 msec before the saccade onset. Then repeated measures ANOVA was performed for each of the pre-saccadic positivity amplitude (averaged across 30 msec), with the group as between-subject factor and the condition, the side of presentation and the electrode (C3, Cz, C4, P3, Pz, P4, O1, Oz, O2) as the within-subject factors. In order to determine the latency of pre-saccadic spike potential, the computation of global field power (GFP: Lehmann and Skrandies, 1980) was applied to the waveform for each subject and condition. The individual pre-saccadic spike potential amplitudes were determined as the amplitude at the GFP peak latency. Repeated measures ANOVA was performed to compare the pre-saccadic spike potential amplitudes with the group as between-subject factor and the condition, the side of presentation and the electrode as the within-subject factors.

Spearman's rank-correlation coefficient was used in exploratory analyses of the relationships between SRT and scores on each item of the CARS and IQ, and between pre-saccadic positivity amplitudes (averaged amplitudes over 9 electrodes for both side of presentation under each condition) at -100 to -70 msec and -70 to -40 msec periods, where the

pre-saccadic positivity amplitudes showed significant group differences, and scores on each item of the CARS and IQ. The level for significance was not corrected for multiple comparisons due to the preliminary nature of the correlational analyses.

Results

Behavioral data

Please insert Figure 2 and Table 1 about here.

In this study, individuals with autism did not show more frequent express saccade than the other two groups either in the gap or overlap condition; the express saccade rate under the gap condition was larger than that under the overlap condition in all three groups (autism group; $z=2.10$, $p<0.05$, MR group; $z=-3.62$, $p<0.001$, healthy control group; $z=-3.23$, $p<0.01$).

The repeated measures ANOVA of the SRT data showed a significant main effect of Condition ($F[1,44]=45.7$, $p<0.001$) and Group ($F[2,44]=4.36$, $p=0.019$), and a significant Group X Condition interaction ($F[2,44]=4.29$, $p=0.02$) (Fig. 2). No significant main effect of the side of presentation or other interactions was found (Table 1). We thus performed a 1-way ANOVA for each condition after data for both sides of the presentation were averaged. The main effect of group was significant in the overlap condition ($F[2,44]=4.71$, $p=0.014$) but not in the gap condition ($F[2,44]=2.86$, $p=0.068$). The multiple comparison test under the overlap condition

revealed that the autism group showed significantly longer SRTs than the control group ($p=0.01$), while the mental retardation group were not significantly different in SRTs from either group (p 's=0.15, 0.45, respectively).

Mean correct response to the target stimuli (discrimination of the dog) were 63% in the autism group, 68% in the MR group and 98% in the healthy control group, respectively. Thus, although autism and MR groups performed worse than the control group, these groups were able to understand the task procedure. Additionally, t-test for the correct response between the MR group and the autism group did not show significant difference ($t[1,26]=0.432$, $p=0.67$).

Pre-saccadic positivity

Please insert Figure 3, 4 and 5 about here.

Visual inspection of the ERP waveforms and topographic mappings indicated that pre-saccadic positivity increased around 100 and 40 msec prior to the saccade onset under the overlap condition in the autism group (Fig. 3, 4, 5). The ANOVA of the pre-saccadic positivity amplitude for the 100-70 msec before the saccade onset showed a significant Condition X Side X Group interaction ($F[2,44]=3.52$, $p=0.038$) and a significant main effect of Group ($F[2,44]=3.86$, $p=0.029$). No other significant interactions were found. After the amplitudes at all electrodes were averaged, 2-way ANOVA was performed separately for each condition. Under the gap

condition, no significant main effect of Group ($F[2,44]=0.43$, $p=0.65$) or interaction of Side X Group ($F[2,44]=0.834$, $p=0.44$) was found. On other hand, under the overlap condition, there was a significant main effect of Group ($F[2,44]=5.47$, $p=0.008$) but there was no significant interaction of Side X Group ($F[2,44]=1.64$, $p=0.21$). Multiple comparisons found that the autism group elicited higher pre-saccadic positivity than both mental retardation ($p=0.011$) and control ($p=0.029$) groups in the overlap condition.

In the ANOVA of the pre-saccadic positivity amplitude for the 70-40 msec, there was a significant interaction of Condition X Electrode X Group ($F[16,352]=2.89$, $p=0.018$, $\epsilon=0.31$) and significant main effect of Group ($F[2,44]=3.89$, $p=0.028$). However, there were no other significant interactions. After the amplitudes for both sides of presentation were collapsed, 2-way ANOVA was performed for each condition. Under the gap condition, no significant main effect of Group was found ($F[2,44]=0.40$, $p=0.67$) but there was a significant interaction of Electrode X Group ($F[16,352]=6.36$, $p<0.0001$). The post-hoc analysis for the electrode in the gap condition indicated that, at O2 and Oz, the autism group elicited higher presaccadic positivity than the mental retardation group (O2: $p=0.009$; Oz: $p=0.035$). Under the overlap condition, there was no significant group X electrode interaction ($F[16,352]=0.96$, $p=0.45$) but there was a significant main effect of Group ($F[2,44]=5.63$, $p=0.007$). The multiple comparison test under the overlap condition showed that pre-saccadic positivity in the autism

group was higher than that in the mental retardation group ($p=0.005$).

Pre-saccadic spike potential

Pre-saccadic spike potentials that peaked 10 msec prior to the saccade onset were observed at broad centro-parietal sites in all groups and conditions (Fig. 3,4). For the pre-saccadic spike potential amplitude, the ANOVA showed a significant interaction of Electrode X Group ($F[16,352]=3.94$, $p=0.001$, $\epsilon=0.44$). However, the follow-up analysis for each electrode showed no significant group difference ($F[2,44]=0.37-2.78$, $p=0.073-0.77$). These results suggest that there were no group difference for pre-saccadic spike potential.

Correlations with the clinical data

SRTs were not significantly correlated with IQs or scores on each item of the CARS. There were significant correlations between pre-saccadic positivity amplitude for 100-70 msec under the overlap condition and two items of CARS (Imitation: $r=0.53$, $p=0.041$; Near receptor responsiveness: $r=0.55$, $p=0.034$).

Discussion

To our knowledge, this is the first study that provides electrophysiological evidence for

dysfunction of attentional disengagement indexed by saccade-locked ERPs in individuals with autism. We speculate that the higher pre-saccadic positivity represents inefficiently more resource for attentional disengagement in the autism group. In other words, higher and longer electric activities as indexed by presaccadic positivity would be necessary for the autistic patients to reach a threshold for executing the saccade.

Additionally, a clear differentiation between autism and mental retardation groups suggests that this attentional dysfunction is not a mere consequence of general cognitive disability. Since there was no difference in SRT or correct response rate between autism and mental retardation groups, we may argue against the possibility that the observed neurophysiological difference between groups is merely attributable to the difference in task performance. Furthermore, we found that individuals with autism show attention deficits in the overlap condition but not in the gap condition. The attentional system is thought to be comprised of two mechanisms: one is the exogenous component that is thought to be triggered reflexively by external stimuli; and the other is the endogenous component that is thought to be controlled by internal, volitional, or central executive mechanisms (Posner, 1980). Under the gap condition, where attention on the central stimulus is disengaged automatically by the disappearance of that stimulus, exogenous disengagement occurs, while under the overlap condition, where attention is disengaged intentionally as the central stimulus remains, endogenous disengagement occurs

(Fischer and Weber, 1993). Developmental studies of attentional disengagement (Hood and Atkinson, 1993; Matsuzawa and Shimojo, 1997) have suggested that the inability of young infants to disengage their attention from a stimulus on which they have fixated is responsible for their difficulty in visual orientation. Different developmental time courses were shown for the gap and overlap disengagement, with early maturation of the gap disengagement, i.e. exogenous disengagement, and later maturation of the overlap disengagement ability, i.e., endogenous disengagement. Taken together with the fact that the exogenous attentional disengagement normally occurred in the gap condition in the autism since the central stimulus to which the participants attended disappeared before the peripheral stimulus appeared, the present study suggests that individuals with autism had specific deficits in endogenous attentional disengagement.

According to Posner's model, the disengagement component of visuospatial attention is associated with function of the parietal cortex. Recent neuroimaging evidence has also suggested that parietal cortex is important in visually guided shift of spatial attention (Kincade et al., 2005; Shomstein and Yantis, 2006). Thus, dysfunction in attentional disengagement could be attributable to parietal abnormality proposed in autism (Courchesne et al., 1993; Minshew et al., 1999; Belmonte and Yurgelun-Todd, 2003; Haist et al., 2005; McAlonan et al., 2005). However, the t-maps for group comparison under the overlap condition showed that pre-saccadic positivity

for 100-70 msec occurred significantly higher in the individuals with autism than in the other two groups at broad scalp areas. EEG recording has a limitation in knowing precise anatomical location for the generator due to volume conductance of the intervening tissues. Future studies possibly using simultaneous recording of EEG and fMRI, or using MEG should be necessary to determine the issue.

Consistent with prior studies that recorded the saccade-locked ERP in healthy adults during the gap overlap task without discrimination (Gómez, et al., 1996; Csibra et al., 1997) the present study demonstrates that the pre-saccadic spike potential immediately precedes the saccade onset and is clear at parietal sites in the three groups. Previous studies reported that 6-month-old infants did not elicit the pre-saccadic spike potential (Csibra et al., 1998) and 12-month-old infants (Csibra et al., 2000) and elder adults (Doig and Boylan, 1989) showed smaller amplitude of pre-saccadic spike potential than younger adults. Csibra and some other researchers have claimed that the pre-saccadic spike potential arises from saccade planning circuits of the parietal cortex (Balaban and Weinstein, 1985). Some fMRI studies suggest that the parietal cortex is involved in saccade execution (Nobre et al., 2000; Perry and Zeki, 2000). In the present study the pre-saccadic spike potential amplitude was not significantly different among the three groups; thus, the results suggest that the saccade execution processing in the individuals with autism and mental retardation were similar to healthy adults.

In the autism group, higher pre-saccadic positivity amplitude in the overlap condition was associated with more severe deficits in “Imitation” and “Near receptor responsiveness” items of the CARS. The “Imitation” item evaluates the level of disability to imitate both verbal and nonverbal acts. The “Near receptor responsiveness” refers to a rating of the individual’s response to stimulation of taste, smell, and touch senses (including pain) and whether or not the individuals make appropriate use of these near sense modalities. The sensory responsiveness often plays a part in the overselectivity (Schopler et al., 1980). Significant correlations between the pre-saccadic positivity and these scores suggest that the dysfunction of attentional disengagement may contribute to indifference to action observation of others and preoccupation to non-social stimuli in individuals with autism. However, these interpretations should be regarded as tentative, since our analyses were preliminary in nature and did not make correction for multiple comparisons.

Some limitations of our study should be noted. First, the participants with autism had comorbid of mental retardation. Although our design including a comparison with a separate mental retardation group has partially resolved the problem, future studies should include high functioning autism participants to further eliminate the influence of mental retardation. Second, future investigations should also include children in order to obtain a more comprehensive view of the developmental course of the attentional system in autism.

In summary, the present study, using the gap overlap task, provides electrophysiological evidence for deficits in attentional disengagement in adults with autism. We have also demonstrated that the physiological substrates underlying deficits in visuospatial attention in autism and mental retardation are different.

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Figure Legends

Figure 1. Experimental procedures in the gap condition (left) and the overlap condition (right).

In the gap condition, the central stimulus disappeared 200 msec before the peripheral stimulus was presented, and in the overlap condition, the central stimulus remained during the presentation of the peripheral stimulus. The participants were instructed to press a button when a target stimulus, the drawing of a dog, appeared as either the central stimulus or the peripheral stimulus.

Figure 2. Saccadic reaction times for the autism group (Autism), the mental retardation group (MR) and the control group (Control).

Figure 3. Grand-averaged waveforms at Pz in the gap condition (top) and the overlap condition (bottom). Black lines are for the autism group, dot lines are for the mental retardation group, and gray lines are for the control group.

Figure 4. ERP map series for each condition. AUT, autism (N=16); MR, mental retardation (N=17); CON, control (N=14)

Figure 5. T-maps in the overlap condition. The upside is the t-map representing comparison between the autism group and the control group; the downside is the t-map between the autism group and the mental retardation group.

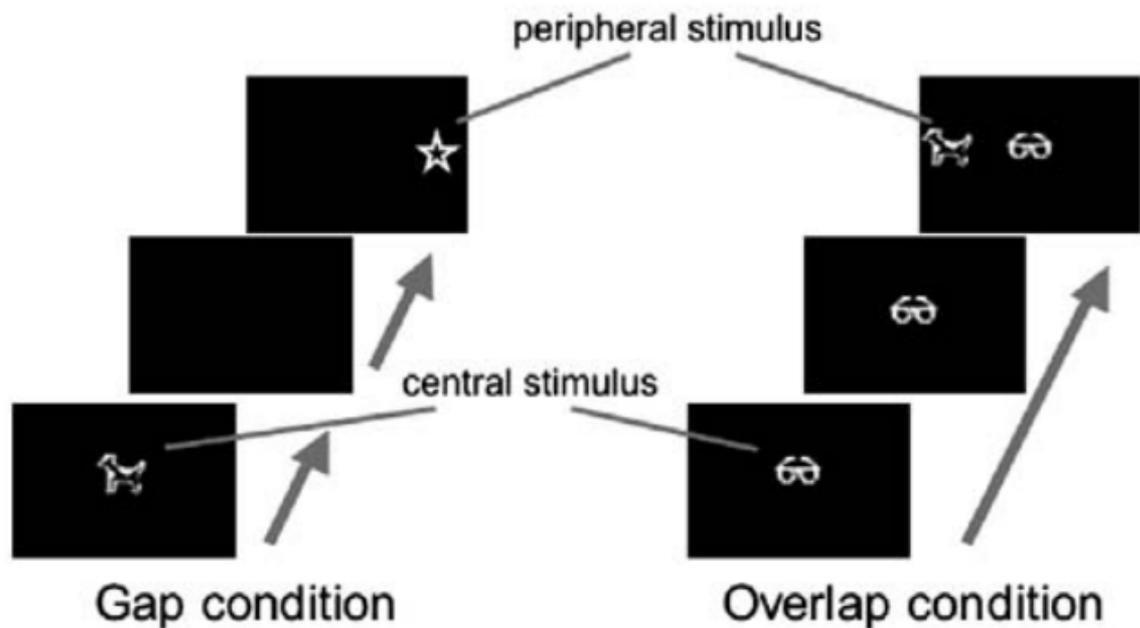


Fig. 1. Experimental procedures in the gap condition (left) and the overlap condition (right). In the gap condition, the central stimulus disappeared 200 ms before the peripheral stimulus was presented, and in the overlap condition, the central stimulus remained during the presentation of the peripheral stimulus. The participants were instructed to press a button when a target stimulus, the drawing of a dog, appeared as either the central stimulus or the peripheral stimulus.

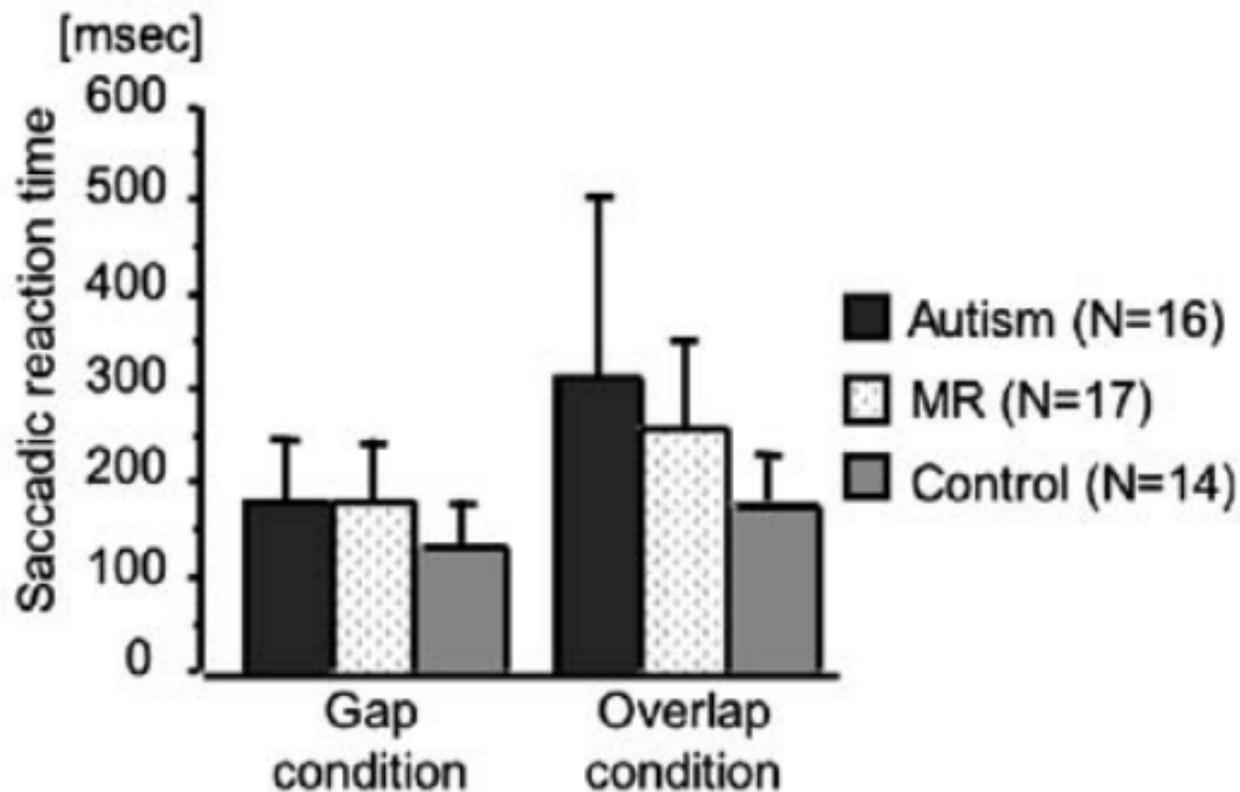


Fig. 2. Saccadic reaction times for the autism group (Autism), the mental retardation group (MR) and the control group (Control).

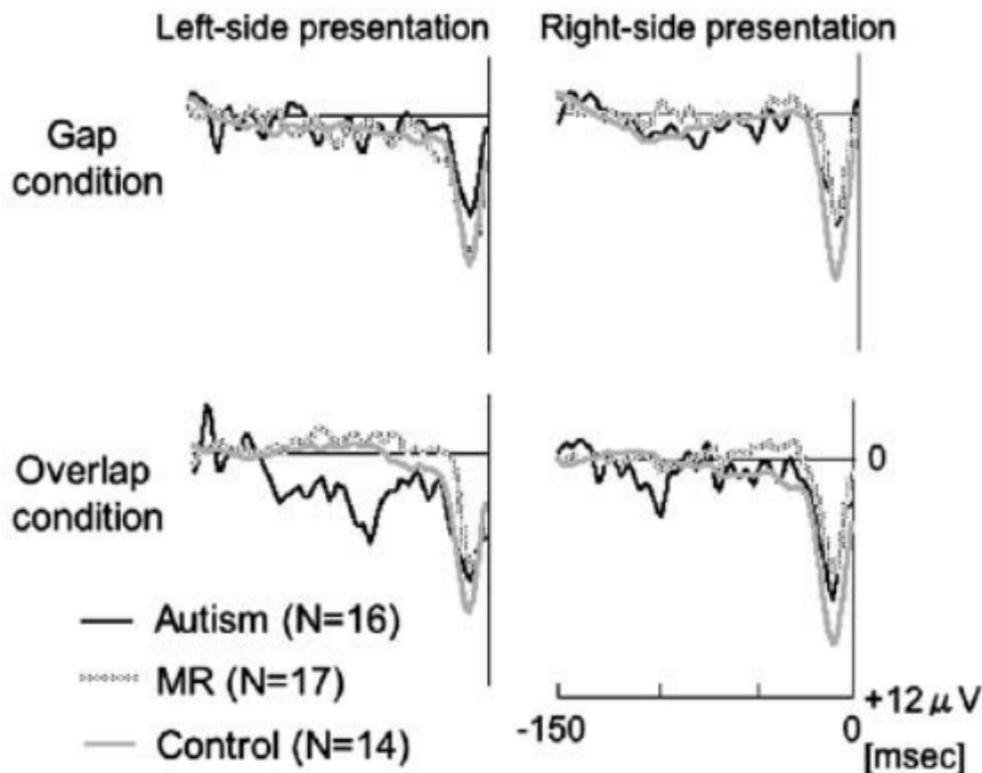


Fig. 3. Grand-averaged waveforms at Pz in the gap condition (top) and the overlap condition (bottom). Black lines are for the autism group, dot lines are for the mental retardation group, and gray lines are for the control group.

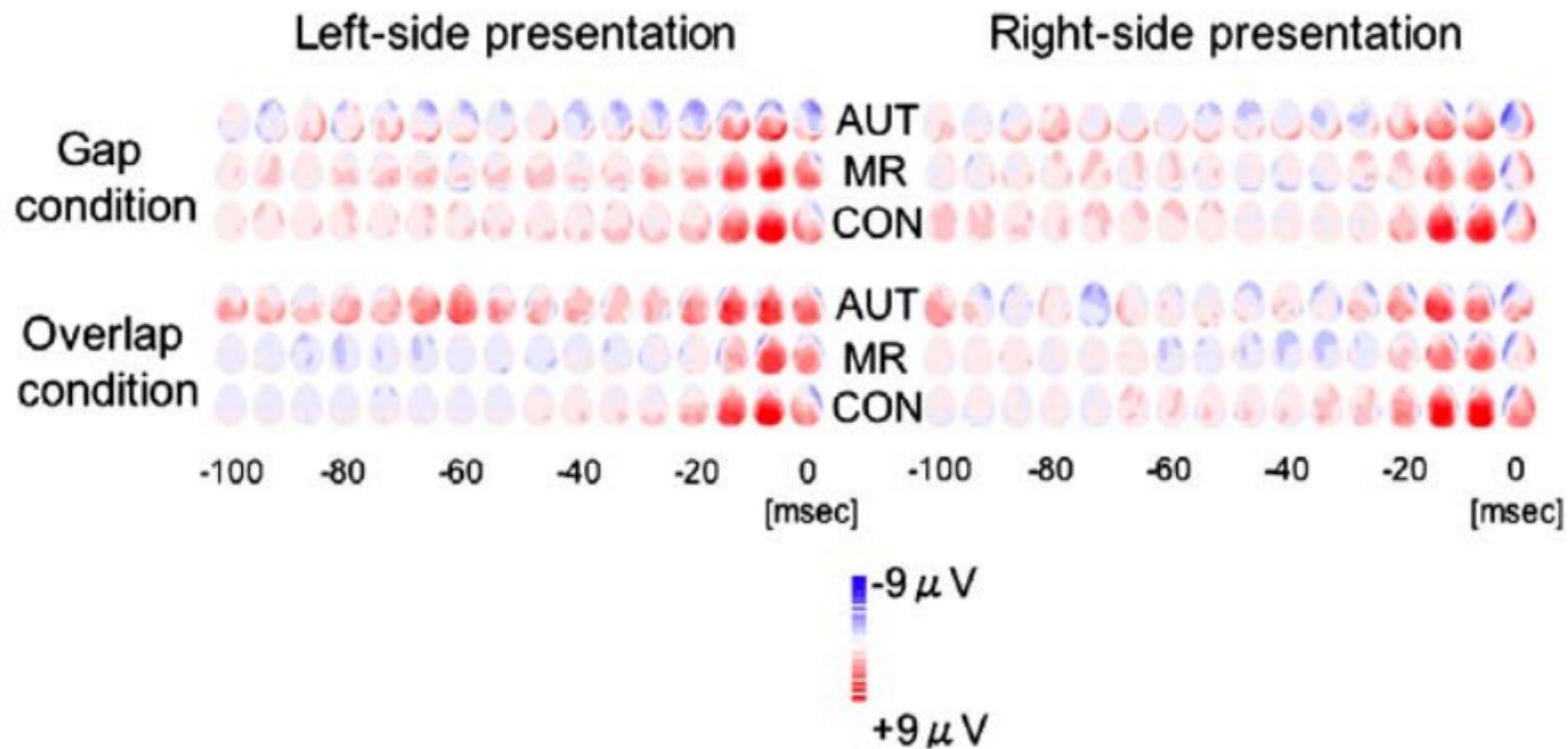


Fig. 4. ERP map series for each condition. AUT, autism ($N = 16$); MR, mental retardation ($N = 17$); CON, control ($N = 14$).

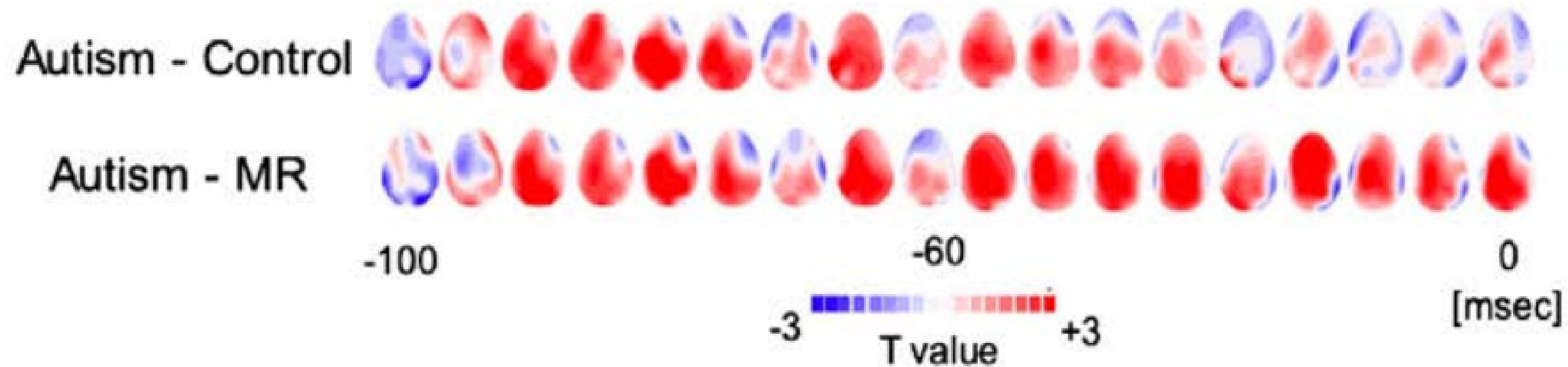


Fig. 5. *T*-maps in the overlap condition. The upside is the *t*-map representing comparison between the autism group and the control group; the downside is the *t*-map between the autism group and the mental retardation group.