NEURAL CORRELATES OF EYE-GAZE DETECTION IN YOUNG CHILDREN WITH AUTISM

Sarah J. Grice[*][1], **Hanife Halit**[*][1], **Teresa Farroni**[1,4], **Simon Baron-Cohen**[2], **Patrick Bolton**[3] and **Mark H. Johnson**[1]

([1]Centre for Brain and Cognitive Development, School of Psychology, Birkbeck College, University of London, UK; [2]Autism Research Centre, Department of Experimental Psychology and Psychiatry, Cambridge University, UK; [3]Institute of Psychiatry, Social, Genetic and Developmental Psychiatry Research Centre, King's College London, Strand, London, UK; [4]Dipartimento di Psicologia dello Sviluppo e della Socializzazione, University of Padua, Italy, *joint first authors)

ABSTRACT

Various reports have demonstrated difficulties in eye-gaze processing in older children and adults with autism. However, little is known about the neural or developmental origin of such difficulties. In the present study, we used high-density Event-Related Potentials (HD-ERPs) to record the neural correlates of gaze processing in young children with autism, and their age-matched controls. In addition, to determine normal gaze processing development we also tested a non-autism adult group. The data obtained from the children with autism resembled that previously observed in typical 4-month old infants. In contrast, the control group showed the same pattern as typical adults. These findings suggest that the neural correlates of gaze direction processing may be delayed in young children with autism.

Key words: autism, autistic spectrum disorder: face perception, ERP, HD-ERP, developmental disorder, eye-gaze

INTRODUCTION

Autism comprises a spectrum of neurodevelopmental disorders characterised by social communication difficulties. Previous research suggests that one component contributing to these difficulties is an abnormality in processing faces. In the typically developing child, the ability to process facial information is thought to be vital for the development of social communication (Baron-Cohen, 1995). Unsurprisingly, a number of aspects of face processing have been found to be atypical in autism, including attention to faces in infancy, face recognition, and the identification of emotional expression (Langdell, 1978; Hobson, 1988; Tantam et al., 1989; Boucher and Lewis, 1992; Davies et al., 1994; Osterling and Dawson, 1994; Teunisse and De Gelder, 1994; Boucher et al., 1998; Celani et al., 1999; Klin et al., 1999; Adolphs et al., 2001). Abnormalities have been found in both behavioural and functional brain imaging tasks (Schultz et al., 2000; Pierce et al., 2001). In the present study we investigate a different aspect of face processing; eye-gaze perception. Behavioural abnormalities of eye-gaze perception and joint attention, as well difficulties understanding the mentalistic relevance of gaze, have already been documented in the disorder (Baron-Cohen et al., 1996, 1997, 2001; Charman et al., 1997; Dawson et al., 1998; Leekam et al., 1998). In this study we sought to determine the neural basis of this perceptual abnormality by using the high-density event-related potential (HD-ERP) technique with young children with autism.

Face processing is known to undergo a protracted developmental course in typical development (de Haan, 2001). According to one view, cortical specialisation for face processing is thought to occur as a result of extensive experience of discriminating between faces (Gauthier and Nelson, 2001). In adults, a region of the medial fusiform gyrus, 'the fusiform face area' (FFA), is specialised for faces compared to other classes of visual stimuli (Kanwisher et al., 1997). By contrast, studies using fMRI have shown that this pattern of specialisation may be aberrant or absent in adults with autism (Schultz et al., 2000; Pierce et al., 2001). Some have suggested that the apparent lack of functional specialisation in the FFA in autism is due to a lack of experience with processing faces (Schultz et al., 2000; Grelotti et al., 2002). In typical development, expertise with faces is thought to be initiated in early infancy by an innate sub-cortical mechanism which predisposes the individual to orient to face-like visual stimuli (Johnson and Morton, 1991). Since it is only possible to diagnose autism from about 18 months of age, it is currently impossible to identify autism-specific behaviour in early infancy. However, in a retrospective study of home videotapes of first birthday parties (Osterling and Dawson, 1994), children later diagnosed with autism were found to spend less time than typically developing children looking at faces, suggesting that they may lack the tendency to orient toward faces during early infancy.

One previous study has investigated the neural correlates of face processing in children with autism using HD-ERPs (Dawson et al., 2002). The HD-ERP technique using the Geodesic sensor net (Tucker, 1993) is ideal for use with children with developmental disorders since it is non-invasive, can be rapidly applied, and gives a millisecond by

millisecond measurement of changes in the brain's naturally occurring electrical activity. Using this technique, Dawson et al. (2002), suggest that the neural correlates of face recognition are abnormal in children with autism aged 3-4 years. Specifically, ERPs measured from typically developing children differentiate between familiar and unfamiliar faces, and also between familiar and unfamiliar objects. Although children with autism showed a normal discriminatory response to object familiarity, the neural correlates of face familiarity were abnormal. In this autism group, there was no difference between the brain's response to familiar compared to unfamiliar faces. These results support the view that face processing impairments are present from early in the autistic child's life.

Abnormalities of eye contact in autism have been documented ever since the first reports of the syndrome (Kanner, 1968). More recently, individuals with autism have been shown to display a range of difficulties with eye-gaze processing (Baron-Cohen et al., 1996, 1997, 2001; Charman et al., 1997; Dawson et al., 1998; Leekam et al., 1998). If these abnormalities are present early in life then they are likely to have major developmental consequences for social communication (Kleinke, 1986; Hains and Muir, 1996). The importance of mutual gaze has even been noted from birth, as newborns prefer to look at faces with direct compared to averted gaze (Batki et al., 2000; Farroni et al., 2002). In addition, in older infants, the perception of averted gaze can elicit an automatic shift of attention in the same direction, developing the foundations of joint attention. Research suggests that this automatic shift of spatial attention in response to changes in gaze direction is present from at least four months of age (Hood et al., 1998; Farroni et al., 2000). The difference between mutual and averted gaze is also reflected in differences in neural processing at this age. A recent study found an enhancement of a face-sensitive ERP component when viewing faces with direct compared to averted gaze. These results are consistent with at least two possible explanations. One hypothesis is that there may be a specific mechanism, termed the 'Eye Direction Detector' (EDD), that detects the presence of eyes and represents their direction and behaviour (Baron-Cohen, 1994). A different possibility is that mutual gaze may facilitate face processing in young infants (Farroni et al., 2002).

Electrophysiological studies of adults have consistently shown that faces elicit a negative potential that occurs most prominently over occipito-temporal scalp regions and peaks between 120-170 msec after stimulus onset (Bentin et al., 1996). This ERP component, the N170, is sensitive to faces in that it is of larger amplitude and shorter latency to faces compared with most other stimuli, including hands, feet, trees, cars,

letters or words (Botzel and Grusser, 1989; Bentin et al., 1996; George et al., 1996). Attempts to investigate the developmental trajectory of the N170 from childhood to adulthood have found that the component increases in amplitude and decreases in latency with age (Taylor et al., 2001). Face sensitive ERPs have also been studied in infancy. For example, the infant N290 component (de Haan et al., 2002; Halit et al., 2003), is thought to be the developmental pre-cursor to the adult N170 (Bentin et al., 1996). They share some functional properties and occur as the first waveform negativity over occipito-temporal areas after the presentation of a face stimulus. The underlying neural activity in both adults, children and infants is thought to be related to the earliest stages of face processing (Eimer, 2000; de Haan et al., 2002). Farroni et al. (2002) studied the effects of eye-gaze direction in 4 month-old infants and found that direct gaze elicited a larger negativity than averted gaze. The scalp distribution of the response was largest over mid-line scalp locations, as opposed to the more lateral distribution seen in the adult N170. A study (Taylor et al., 2001) that attempted to investigate the effect of direction of gaze on the N170 in adults found no difference for direct compared to averted gaze. However, these data may not be directly comparable to that obtained from infants as different scalp locations were used and the experiment did not utilise the same paradigm i.e., passive viewing.

The aims of the current study were two-fold. First, to investigate the neural correlates of eye-gaze perception in young children with autism compared to typically developing age and sex matched controls. Second, to investigate the neural correlates of eye-gaze perception in typical adults without autism in order to ascertain the characteristics of the final state. The objective was to investigate the behaviour of the face-sensitive component in all groups at both mid-line and lateral scalp locations. Although this was a somewhat exploratory study, we predicted that the neural correlates of eye-gaze sensitivity in children with autism would look significantly different to that of typically developing children of the same age.

EXPERIMENT ONE

Methods

Participants

The final sample consisted of 10 children with autism (9 males) with a mean age of 61 months (range: 42-87 months), and 10 individually age and sex-matched control children with a mean age of 63 months (range: 42-85 months). All children with autism had previously received an independent

TABLE I
Ages and CARS Scores for Individual Participants in the Autism Group

Participant	Age (months)	CARS Total Score	CARS Verbal Score*
1	42	40	4
2	46	42	4
3	53	47.5	4
4	56	36	1.5
5	58	33	1
6	65	40	3
7	65	43	4
8	68	37	2
9	75	43	2
10	85	44.5	4

*CARS Verbal Score
1 = Normal verbal communication, age and situation appropriate
2 = Mildly abnormal verbal communication
Speech shows overall retardation. Most speech is meaningful; however, some echolalia or pronoun reversal may occur. Some peculiar words or jargon may be used occasionally.
3 = Moderately abnormal verbal communication
Speech may be absent. When present, verbal communication may be a mixture of some meaningful speech and some peculiar speech such as jargon, echolalia, or pronoun reversal. Peculiarities in meaningful speech include excessive questioning or preoccupation with particular topics.
4 = Severely abnormal verbal communication
Meaningful speech is not used. The child may make infantile squeals, weird or animal-like sounds, complex noises approximating speech, or may show persistent, bizzare use of some recognizable words or phrases.

diagnosis of autism by a clinician. Diagnostic status according to DSM-IV criteria was confirmed in all cases by clinical judgement of a psychologist experienced in the diagnosis of autism. All children with autism were judged on the basis of observation and parental report to score well above 30 on the Childhood Autism Ratings Scale (CARS) (see Table I). One child who scored highly on the CARS but whose diagnosis was questioned on the basis of clinical judgement was further evaluated, and found to reach criteria for Autistic disorder, by use of the Autism Diagnostic Interview – Revised (Lord, et al., 1994), and the Autism Diagnostic Observation Schedule – Generic (Lord, et al., 1989). All of the age-matched control sample scored 15 on the CARS ('Non-Autistic' scores classed as between 15-30) and were judged to be typically developing, with no history of any developmental delay or family history of autism. Exclusionary criteria for both the control and autism group included the presence of a neurological disease or disorder of known etiology (e.g., Tuberous Sclerosis), physical abnormalities, or history of head injury. An additional 6 (3 autism) children were tested but were excluded from further analysis due to eye and/or body movements that resulted in recording artefacts (n = 5) or due to a procedural error (n = 1 autism). Parents of all children tested gave informed written consent.

Stimuli

The stimuli (see Figure 1) were full colour photographic images of 3 different human female faces directing their gaze straight-on to the viewers (Direct Gaze) or averted to either the right or left (Averted Gaze), and were identical to those used in a previous study (Farroni et al., 2002). The faces were presented against a grey background and subtended
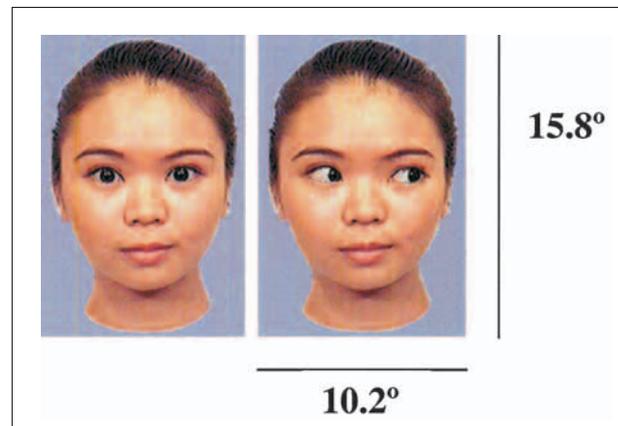

Fig. 1 – *Examples of experimental stimuli showing direct and averted gaze. Measures in the figure indicate the visual angle of the stimuli when viewed from a distance of 90 cm. Stimuli were presented individually.*

a horizontal angle of 10.2° and a vertical angle of 15.8° when viewed from a distance of 90 cm.

ERP Recording

ERPs were recorded using a Geodesic sensor net consisting of 128 silver-silver chloride electrodes evenly distributed across the scalp (Tucker, 1993). A ground electrode was positioned at the back of the head above the neck. All bio-electrical signals were recorded using EGI NetAmps (Eugene, OR). The signals were recorded referenced to the vertex, with a bandpass filter of 0.1-100 Hz and with gain set to 10,000 times. EEG was recorded continuously throughout the test sessions with a sampling rate of 250 Hz. Stimulus duration was 1000 msec with a variable inter-stimulus interval between 800-1200 ms. In order to be able to eliminate trials containing artefacts caused by eye movements the electro-oculogram (EOG) was recorded from electrodes positioned above both eyes and on the outer canthi.

General Procedure

After the sensor net was applied, each child passively viewed the faces while seated on a chair or a carer's lap in a dimly lit booth, approximately 90 cm from a 21 inch-computer monitor mounted in a black background. The child was readily observable to the experimenter at all times via a video camera situated directly beneath the monitor. An experimenter observed the child's behaviour and stimuli were presented only when the child was watching a fixation point which consisted of various multi-coloured cartoon images appearing centrally on the screen. Stimuli were presented in random order and with equal probability up to a maximum of 150 trials of each condition or until the child became too fussy or bored to attend. Between trials the experimenter could activate a number of different noises via a speaker located out of sight beneath the presentation monitor, and present coloured patterns on the monitor to re-orient the child's attention to the screen if required.

Aspects of Procedures Specific to Autism Group

Children with autism were selected on the basis that they had previously successfully taken part in another ERP study in our laboratory, and formed a subgroup of children who had been recruited from specialist schools in and around the London area. The experimental test session was tailored to meet the requirements of each child. A parental questionnaire (contained in parent pack) was used to establish each child's likes and dislikes. The questionnaire was also used as a guide to how the child would most likely respond to a new environment and new people, as well as to wearing the net. Children who were reported to have difficulties with new people and/or places or head touching/washing or other tactile sensitivities etc. were visited in their homes approximately a week prior to testing. The purpose of the visit was to familiarise the child to the net and experimenters in their own environment. The net was introduced to the child by both experimenter and parent, and children were encouraged to touch and wear the net. In two cases, parents requested that they retain the net in order to provide continual familiarisation prior to visiting the lab.

Geodesic Net Application and Testing

Children from both groups were encouraged to wear the net by use of play and reward. Rewards was chosen by parents to be the most motivating for each individual child and usually consisted of the child's favourite food or toy. The system often utilised during the test session was that children were encouraged to attend to at least 5-10 stimulus presentations at the end of which they were rewarded. This procedure continued until the child became too fussy or restless to attend.

ERP Waveform Analysis

The continuous EEG recording was divided to create segments from 200 msec pre-stimulus onset to 600 msec post-stimulus onset (i.e., 800 msec segments). Data were edited for artefacts and digitally filtered offline with a 30 Hz low-pass elliptical filter. Data from each sensor were removed if they contained artefacts created by movement or poor contact. The entire trial was excluded if data from more than 12 sensors were removed or if the trial contained an eye-blink. The video recording of each individual participant's behaviour was viewed off-line. Trials were removed if the video revealed that the participant was looking away during the trial. Data were baseline-corrected and then individual participant averages were computed for each trial type (Minimum = 10 trials per condition). Individuals with more than 10 bad channels in their averages were excluded from further analysis. The average number of trials making up these individual averages for the autism group was 31 (SD10, Range 15-52) and for the control group was 40 (SD10, Range 22-52). Missing data for children with 10 or fewer bad channels were interpolated using spherical spline interpolation from the individual participant averages. Data were re-referenced to the average reference. ERP data analyses were carried out on two previously identified face-sensitive components, namely the N170 (termed here 'N170') and a related mid-line component (termed here 'mid-line N170') similar to that showing sensitivity to gaze direction in typically developing 4 month-olds (Farroni et al., 2002). The timing and scalp distribution of the N170 in response to Direct Gaze was similar for the autism group and the control group: the N170 peaked around 220 msec after stimulus onset and occurred most prominently over occipito-temporal sites. All participants showed clear N170 peaks (except over the mid-line anterior sites, see below). The effects of gaze on the amplitude and latency of the N170 were tested by computing two measures: (1) Peak Amplitude (μV) within the time-window 180-300 msec, and (2) Peak Latency (msec) by calculating the time at which the peak occurred. Peak amplitudes were identified using peak detection software (EGI Transave).

These measures were analysed in a $2 \times 2 \times 3$ Mixed ANOVA with group (autism, control group) as a between participant factor, and gaze (direct/averted), channel group (Left, Medial, Right) as within participant factors. Greenhouse-Geisser corrected p-values were used for within-participants factors when appropriate. Sensors that made up the channel groups were: Left – 64, 65, 66, 69, 70, 74, Medial – 71, 75, 76, 82, 83, 84, and
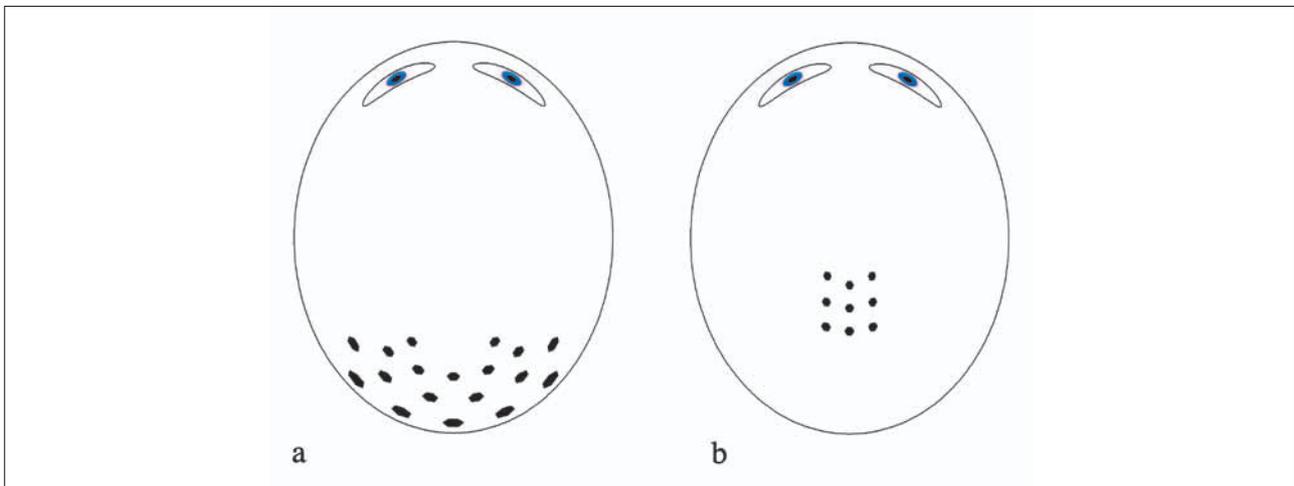
Fig. 2 – *Diagram of head showing electrode groups for a) N170 and b) anterior mid-line N170 analysis.*

Right – 85, 89, 90, 91, 95, 96 (Johnson et al., 2001) (see Figure 2a). The mid-line N170 component was more anterior in our current study compared to the previous infant study. Since, like the infant study, there was no consistently identifiable peak at these midline recording sites a mean (as opposed to peak) analysis of the component was carried out from 200-260 msec, therefore there are no latency data. Sensors that made up the midline group included: 54, 61, 62, 67, 68, 73, 79, 80 (see Figure 2b).

Results

The analysis of the N170 peak amplitude revealed that there were no main effects of group [F (1, 18) = .293, p > .05], or gaze [F (1, 18) = .160, p > .05] and no interaction between group and gaze [F (1, 18) = .165, p > .05]. There was, however, a main effect of channel [F (2, 36) = 17.22, p < .05], which showed that for both groups the N170 was largest over the right hemisphere compared to the left hemisphere leads [t (19) = 2.797, p < .05], and both left and right channel groups were significantly larger than the medial channel group [left, t (19) = 3.48, p < .05; right, t (19) = 5.52, p < .05]. There was no three-way interaction [F (2, 36) = .943, p > .05]. Analysis of the latency results for the N170 reflected a similar pattern, with no difference in latency by group [F (1, 18) = .021, p > .05], or different gaze directions [F (1, 18) = .018, p > .05], and no interaction between group and gaze [F (1, 18) = 1.193, p > .05].

The main effect of channel was significant [F (2, 36) = 5.825, p < .05] because the left hemisphere peak occurred significantly earlier than both the medial channel group [t (19) = 3.486, p < .05] and right hemisphere channel group peak [t (19) = 2.976, p < .05].

More interesting was the analysis of the anterior mid-line scalp electrodes. The mean amplitude of the midline-N170 did not differ overall by group [F (1, 18) = .021, p > .05], but the there was a differential effect across group to changes in eye-gaze [F (1, 18) = 4.981, p < .05]. This was because the midline-N170 amplitude to direct gaze was significantly larger (more negative) compared to averted gaze for the autism group alone [t (9) = 2.827, p < .05] (see Figures 3a, 4, and 5a) and not for the control group [t (9) = .399, p > .05] (see Figures 3b, 5b).

Discussion

Previous ERP studies on gaze processing have shown a clear effect over midline channels when 4-month-old infants passively view direct or averted gaze (Farroni et al., 2002). In the present study, our autistic sample showed an effect very similar to that observed in infants, whereas the effect was absent in the age-matched controls. One interpretation of these findings is that the neural correlate of eye-gaze processing in our autistic sample shows developmental delay relative to age-matched controls. In order to ascertain whether the lack of effect displayed by the control group was characteristic of the culmination of gaze processing development, we tested a non-autism adult group.

If the adult group show the same lack of effect as the age-matched control group then we may conclude that the age-matched control group are displaying functionally mature neural correlates of eye-gaze processing.

EXPERIMENT TWO

Methods

Participants

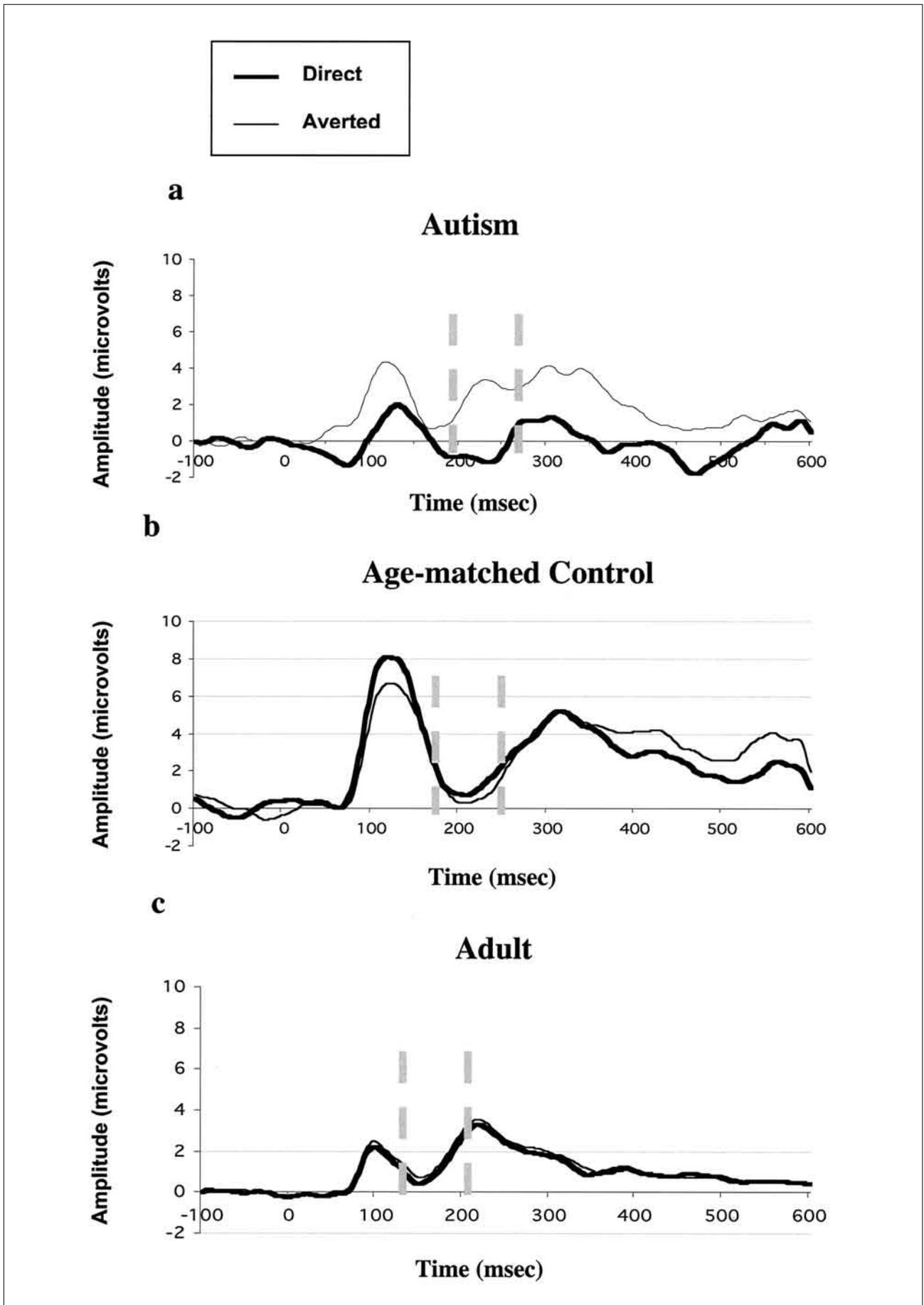The final sample consisted of 10 adults with a mean age of 28.6 years (range = 20-40 years).

Fig. 3 – *ERP waveform averaged across electrodes included in mid-line N170 channel group for direct and averted gaze a) autism group b) age matched controls, and c) adult group. Vertical grey bars indicate time window used in analysis.*
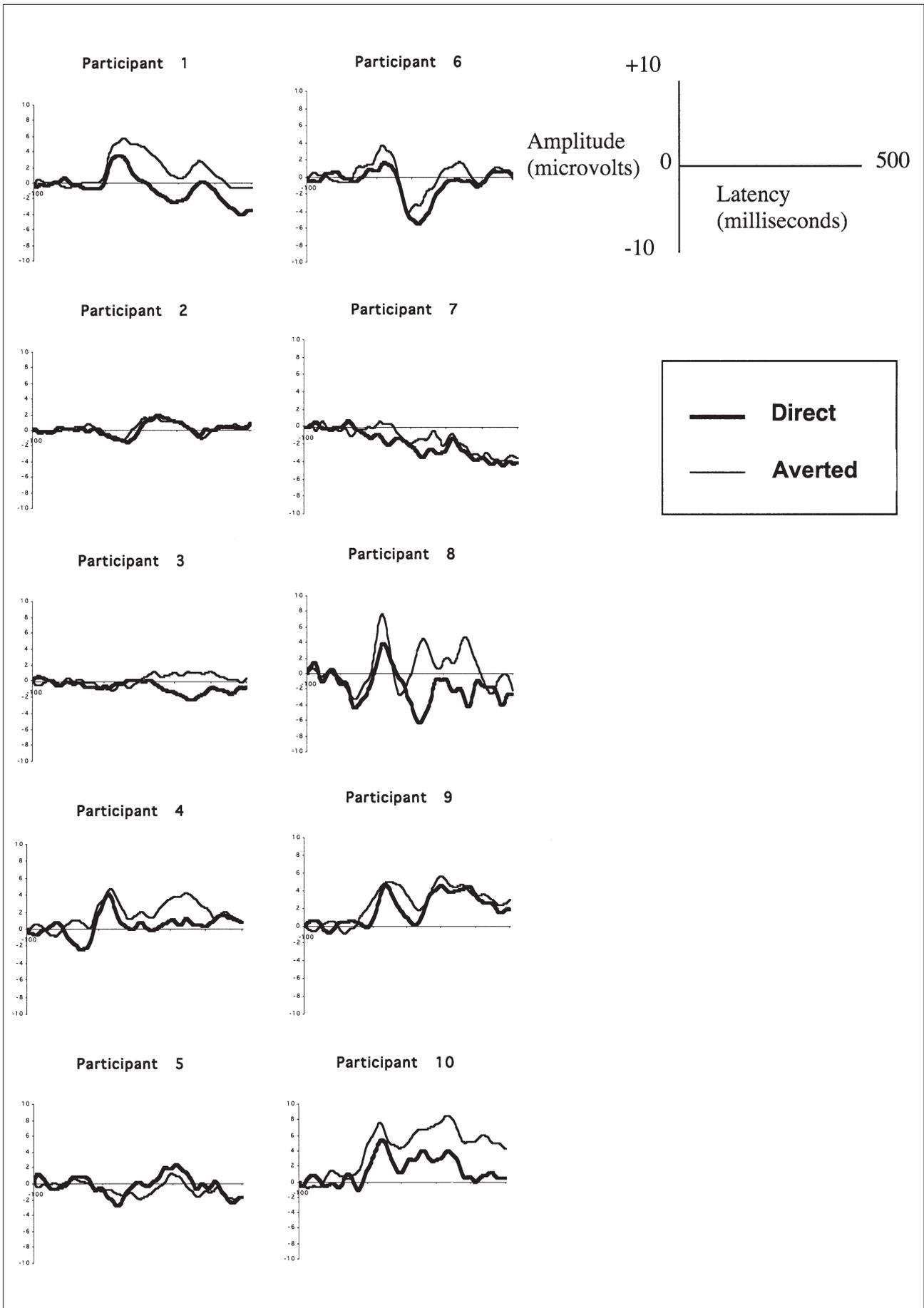
Fig. 4 – *Average ERP waveforms from midline channel group for each individual autism participant in order of chronological age. The thick line represents the waveform to direct gaze stimuli, and the thin line represents the waveform to averted gaze stimuli.*
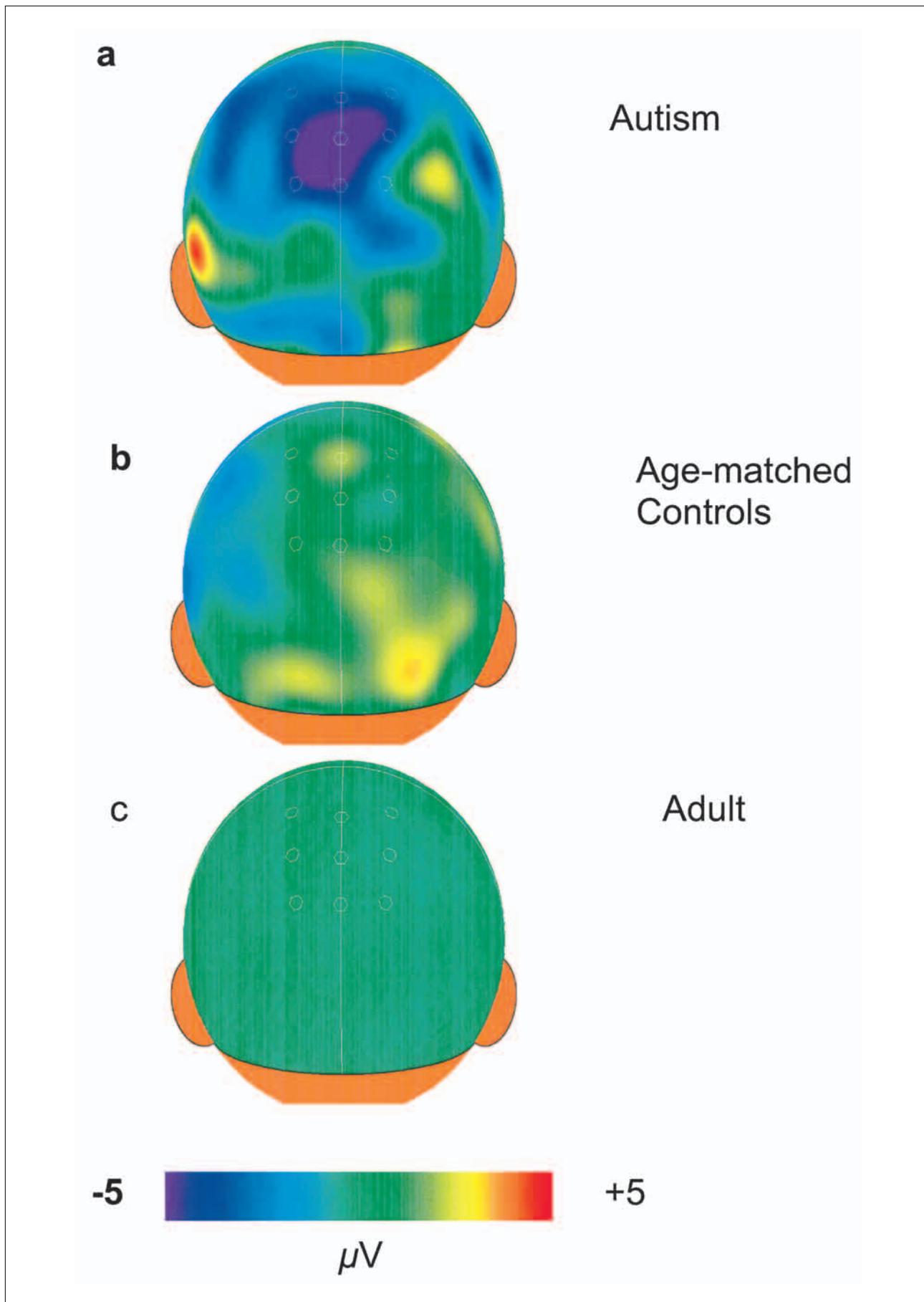
Fig. 5 – *Spherical spline interpolations for the surface distribution of the average amplitude difference obtained for direct minus averted gaze for a) autism group (200-260 msec after stimulus onset) b) age-matched control group (200 - 260 msec), and c) adult group (140-200 msec).*

None of the adult group had any history of developmental delay or family history of autism. Exclusion criteria were the same as for the previous experiment. An additional adult was tested but was excluded from further analysis due to eye movements that resulted in recording artefacts. All participants gave informed written consent.

Stimuli and ERP Recording

The stimuli and ERP recording procedure were exactly the same as for the previous experiment.

General Procedure

After the sensor net was applied, each adult passively viewed the faces while seated approximately 90 cm from a 21 inch-computer monitor mounted in a black background. The adult was readily observable to the experimenter at all times via a video camera situated directly beneath the monitor. An experimenter observed the individual and stimuli were presented only when they were watching a fixation point that consisted of various multi-coloured cartoon images appearing centrally on the screen. Stimuli were presented in random order and with equal probability, until the individual had seen 150 trials of each condition (direct or averted gaze).

ERP Waveform Analysis

The treatment of the ERP data was the same as for the previous experiment. The average number of trials making up each individual averages was 135 (SD 10, Range 120-150). The timing and scalp distribution of the N170 was similar to other face processing studies, peaking around 160 msec after stimulus onset and occurring most prominently over occipito-temporal sites. The effects of gaze on the amplitude and latency of the N170 were tested by computing two measures: (1) Peak Amplitude (μV) within the time-window 120-200 msec, and (2) Peak Latency (msec) by calculating the time at which the peak occurred. These measures were analysed in a 2×3 ANOVA with gaze (direct/averted) and channel group (Left, Medial, Right) as within participant factors. Since the previous experiment had analysed a mean amplitude measure for mid-line N170 scalp regions, the same approach was also used for these adult data despite the presence of a clear peak. Sensors that made up the midline group were the same as those in the previous experiment. However, inspection of waveforms from individuals revealed that the component peaked on average approximately 60 ms earlier than for the previous groups. For this reason, the time-window used to compute the average amplitude measure was 140-200 ms.

Results

The results of the analysis were very similar to those of the age-matched control group in Experiment One. Analysis of the N170 amplitude revealed that there was no effect of gaze [$F (1, 9) = .355, p > .05$]. There was, however, a significant effect of channel group [$F (1, 9) = 5.223, p < .05$], which was because the left and right channels, while not significantly different from each other [$t (9) = .951, p > .05$], were both more negative in amplitude than the medial channel group leads {left [$t (9) = 3.313, p < .05$]; right [$t (9) = 2.305, p < .05$]}. There was no significant interaction of gaze with channel group [$F (2, 18) = .120, p > .05$]. The latency data also revealed no effect of gaze [$F (1, 9) = .556, p > .05$]. There was no main effect of channel [$F (1, 9) = 2.438, p > .05$] or interaction of gaze with channel [$F (2, 18) = .620, p > .05$]. Similarly, the analysis of the mid-line-N170 component revealed that there was no significant difference in amplitude for direct compared to averted gaze conditions [$F (1, 9) = 2.101, p > .05$] (see Figures 3c and 5c).

Discussion

Experiment Two was conducted to investigate whether the adult N170 shows sensitivity to gaze direction. The results showed that the N170 (over all scalp locations analysed) did not differ to direct or averted eye-gaze suggesting that both stimuli elicit equivalent underlying neural processing.

GENERAL DISCUSSION

A previous study has shown that the neural correlates of gaze processing in infants are enhanced by direct compared to averted gaze (Farroni et al., 2002). In the present study, our autistic sample showed an effect very similar to that observed in infants, whereas the effect was absent in the age-matched and adult control groups. Overall, these results suggest that the neural correlates of eye-gaze processing in our autistic sample reflect developmental delay relative to age-matched controls.

While it is likely that the posterior negativity observed in infants and children corresponds to the adult N170 (de Haan et al., 2002; Halit et al., 2003), it remains unclear whether the eye gaze effect observed in infants and in the current autistic sample shares common neural generators with the face-sensitive negativity. On the assumption that they do have the same neural generators, one possible interpretation of the results is that direct gaze causes deeper processing of faces from very early in life, whereas averted gaze does not initiate an increase in face processing at this stage of development. The significance and importance of

averted gaze may then develop in the first years of life (Farroni, et al. 2000). By early childhood, the functional relevance of direct and averted gaze may be such that both elicit equivalent neural processing. This is reflected in the equivalent sensitivity of the face-sensitive N170 to differences in gaze direction in both our adult and child control group. This hypothesis suggests delayed development in learning the significance of averted eye-gaze in young children with autism. An obvious alternative interpretation is that the functional relevance of direct gaze decreases over development such that by early childhood it becomes equivalent to that of averted gaze. However, since the amplitude of the N170 to averted gaze increases over developmental time (as opposed to a reduction in amplitude to direct gaze) the current findings are less consistent with this second hypothesis.

Another, equally interesting, possibility derives from previous ERP studies on the development of face processing in children. Specifically, Taylor and colleagues (Taylor et al., 2001) have argued that early in typical development, face-sensitive ERP components are modulated by the eyes rather than overall face configuration. Applied to the current data, this idea suggests that information about the eyes is more evident in the scalp-recorded ERP of young infants and young children with autism, while the equivalent components in non-autistic children and adults reflect processing of the overall configuration of a face. This hypothesis is consistent with the view that autistic children are developmentally delayed in their processing of faces, and/or that they use a more featural and less configural strategy for processing faces compared to controls. Evidence for the latter hypothesis has been frequently reported in studies of older children and adults with autism (Frith, 1989).

Overall, our results are broadly consistent with those obtained in the only other published ERP study in young children with autism (Dawson et al. 2002). In both studies, young autistic children had an ERP waveform that contained the same basic components as seen in age-matched controls. In other words, there were no gross abnormalities in the early visual ERP components observed. Further, in Dawson et al. (2002) typically developing children showed an effect of face familiarity at a posterior face-sensitive component termed the "P400" that was absent in their autistic sample. In the present study we recorded this component in both child groups, but discrimination of gaze direction occurred at a shorter latency component, the equivalent of the adult "N170". Therefore, in both studies there was atypical modulation of a mid-latency component in the autistic group.

At present our conclusions must be tentative for a number of reasons. One reason for caution is that our sample of children with autism was selected on the basis that they had previously successfully

completed an earlier ERP experiment. About a third of those individuals recruited for the earlier ERP experiment yielded sufficient data. It therefore remains possible that the subset of children we studied in the present experiment does not reflect the abilities of the larger population of those with autism. However, such issues are endemic even to behavioural studies of developmental disorders early in life. A related issue to that of sampling bias is the heterogeneous nature of the autism group studied. The autistic children tested for this study ranged from moderate to high functioning and included children with and without spoken language. Despite this heterogeneity, all participants showed the same gaze effect[1]. Such consistency across our apparently disparate autism group suggests that any delay in the neural development related to eye-gaze processing may be a fundamental characteristic of the autistic spectrum.

Our study is also limited to some extent by our use of a passive-viewing ERP paradigm. For example, the absence of a gaze-direction effect in adults and our age-matched controls could simply be explained by use of the passive viewing paradigm that may not elicit sufficient attention from typical children and adults. However, this is unlikely since various studies have shown differences in ERPs to manipulations of faces using identical passive viewing paradigms (e.g., Halit et al. 2001, de Haan et al. 2002). Another limitation is that while we excluded trials with eye movements, we cannot completely rule out differences in scanning faces, or initial foveation. Indeed, while the age-matched control group showed a trend for a larger P1 to the direct gaze face, the group with autism showed the opposite tendency (although this varied between individuals, see Figure 4). This could potentially be explained by greater foveation of the face in response to direct gaze in the control group, and increased foveation of the face with averted gaze in the autism group. However, this would not explain the difference in later face-sensitive components. Finally, as with all ERP experiments the lack of difference in neural activity as recorded by electrical activity at the scalp surface cannot be taken as conclusive evidence of common neural mechanisms since subcortical activation is largely undetectable by this method.

While we are cautious about drawing specific conclusions, the present study does allow us to conclude that young children with autism can differentially process direct and averted gaze when viewing faces. The results also offer some support

---

[1]For one child the N170 was unusually early compared to the rest of the children in the autism and control groups. The time window used to analyse the data did not capture the N170 over the mid-line scalp regions for this child, but instead captured later P2 activity. When the window was individually adjusted to capture his early N170, this individual with autism also showed the significant effect (these adjusted data were not included in the overall analysis).

to previous behavioural studies suggesting the presence of eye-gaze processing abnormalities in older individuals with autism. Further, and of equal importance, we have helped establish that it is possible to study the neural correlates of visual cognition in autism during early childhood. Whether differential ERPs can be used as part of diagnostic package remains a topic for further research.

REFERENCES

ADOLPHS R, SEARS L and PIVEN J. Abnormal processing of social information from faces in autism. *Journal of Cognitive Neuroscience, 13:* 232-240, 2001.

BARON-COHEN S. How to build a baby that can read minds: Mechanisms in mindreading. *Cahiers de Psychologie/Current Psychology of Cognition, 13:* 513-552, 1994.

BARON-COHEN S. *Mindblindness: An Essay on Autism and Theory of Mind.* Cambridge, MA: MIT Press, 1995.

BARON-COHEN S, COX A, BAIRD G, SWETTENHAM J, NIGHTINGALE N, MORGAN K, DREW A and CHARMAN T. Psychological markers in the detection of autism in infancy in a large population. *British Journal of Psychiatry, 168:* 158-163, 1996.

BARON-COHEN S, JOLLIFFE T, MORTIMORE C and ROBERTSON M. Another advanced test of theory of mind: Evidence from very high functioning adults with autism or Asperger syndrome. *Journal of Child Psychology and Psychiatry, 38:* 813-822, 1997.

BARON-COHEN S, WHEELWRIGHT S, HILL J, RASTE Y and PLUMB I. The "reading the mind in the eyes" test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry, 42:* 241-251, 2001.

BATKI A, BARON-COHEN S, WHEELWRIGHT S, CONNELLAN J and AHLUWALIA J. Sex differences in human neonatal social perception. *Infant Behaviour and Development, 23:* 223-229, 2000.

BENTIN S, ALLISON T, PUCE A, PEREZ E and McCARTHY G. Electrophysiological studies of face perception in humans. *Journal of Cognitive Neuroscience, 8:* 551-565, 1996.

BOTZEL K and GRUSSER OJ. Electric brain potentials evoked by pictures of faces and non-faces: A search for "face-specific" EEG-potentials. *Experimental Brain Research, 77:* 349-360, 1989.

BOUCHER J and LEWIS V. Unfamiliar face recognition in relatively able autistic children. *Journal of Child Psychology and Psychiatry, 33:* 843-859, 1992.

BOUCHER J, LEWIS V and COLLIS G. Familiar face and voice matching and recognition in children with autism. *Journal of Child Psychology and Psychiatry, 39:* 171-181, 1998.

CELANI G, BATTACCHI MW and ARCIDIACONO L. The understanding of the emotional meaning of facial expressions in people with autism. *Journal of Autism and Developmental Disorders, 29:* 57-66, 1999.

CHARMAN T, SWETTENHAM J, BARON-COHEN S, COX A, BAIRD G and DREW A. Infants with autism: An investigation of empathy, pretend play, joint attention, and imitation. *Developmental Psychology, 33:* 781-789, 1997.

DAVIES S, BISHOP D, MANSTEAD AS and TANTAM D. Face perception in children with autism and Asperger's syndrome. *Journal of Child Psychology and Psychiatry, 35:* 1033-1057, 1994.

DAWSON G, CARVER L, MELTZOFF AN, PANAGIOTIDES H, McPARTLAND J and WEBB SJ. Neural correlates of face and object recognition in young children with autism spectrum disorder, developmental delay, and typical development. *Child Development, 73:* 700-717, 2002.

DAWSON G, MELTZOFF AN, OSTERLING J, RINALDI J and BROWN E. Children with autism fail to orient to naturally occurring social stimuli. *Journal of Autism and Developmental Disorders, 28:* 479-485, 1998.

DE HAAN M. The neuropsychology of face processing during infancy and childhood. In M Luciana and CA Nelson (Eds), *Handbook of Developmental Cognitive Neuroscience.* Cambridge, MA: MIT PRESS, 381-398, 2001.

DE HAAN M, PASCALIS O and JOHNSON MH. Specialization of neural mechanisms underlying face recognition in human infants. *Journal of Cognitive Neuroscience, 14:* 199-209, 2002.

EIMER M. Event-related brain potentials distinguish processing stages involved in face perception and recognition. *Clinical Neurophysiology, 111:* 694-705, 2000.

FARRONI T, CSIBRA G, SIMION F and JOHNSON MH. Eye contact detection in humans from birth. *Proceedings of the National Academy of Sciences, USA, 99:* 9602-9605, 2002.

FARRONI T, JOHNSON MH, BROCKBANK M and SIMION F. Infants' use of gaze direction to cue attention: The importance of perceived motion. *Visual Cognition, 7:* 705-718, 2000.

FRITH U. *Autism: Explaining the enigma.* Oxford: Basil Blackwell, 1989.

GAUTHIER I and NELSON CA. The development of face expertise. *Current Opinion in Neurobiology, 11:* 219-224, 2001.

GEORGE N, EVANS J, FIORI N, DAVIDOFF J and RENAULT B. Brain events related to normal and moderately scrambled faces. *Cognitive Brain Research, 4:* 65-76, 1996.

GRELOTTI DJ, GAUTHIER I and SCHULTZ RT. Social interest and the development of cortical face specialization: What autism teaches us about face processing. *Developmental Psychobiology, 40:* 213-225, 2002.

HAINS SM and MUIR DW. Infant sensitivity to adult eye direction. *Child Development, 67:* 1940-1951, 1996.

HALIT H, DE HAAN M and JOHNSON MH. Cortical specialisation for face processing: Face sensitive event-related potentials in 3 and 12 month-old infants. *Neuroimage, 19:* 1180-1193, 2003.

HOBSON RP, OUSTON J and LEE A. What's in a face? The case of autism. *British Journal of Psychology, 79:* 441-453, 1988.

HOOD BM, WILLEN JD and DRIVER J. An eye direction detector triggers shifts of visual attention in human infants. *Psychological Science, 9:* 53-56, 1998.

JOHNSON MH, DE HAAN M, OLIVER A, SMITH W, HATZAKIS H, TUCKER LA and CSIBRA G. Recording and analyzing high-density event-related potentials with infants using the geodesic sensor net. *Developmental Neuropsychology, 19:* 295-323, 2001.

JOHNSON MH and MORTON J. *Biology and Cognitive Development: The Case of Face Recognition.* Oxford, UK: Blackwell, 1991.

KANNER L. Autistic Disturbances of Affective Contact, 1943. Riedited in: *Acta Paedopsychiatrica, 35:* 98-136, 1968.

KANWISHER N, McDERMOTT J and CHUN MM. The fusiform face area: A module in human extrastriate cortex specialized for face perception. *Journal of Neuroscience, 17:* 4302-4311, 1997.

KLEINKE CL. Gaze and eye contact: A research review. *Psychological Bulletin, 100:* 78-100, 1986.

KLIN A, SPARROW SS, DE BILDT A, CICCHETTI DV, COHEN DJ and VOLKMAR FR. A normed study of face recognition in autism and related disorders. *Journal of Autism and Developmental Disorders, 29:* 499-508, 1999.

LANGDELL T. Recognition of faces: An approach to the study of autism. *Journal of Child Psychology and Psychiatry, 19:* 255-268, 1978.

LEEKAM SR, HUNNISETT E and MOORE C. Targets and cues: Gaze-following in children with autism. *Journal of Child Psychology and Psychiatry, 39:* 951-962, 1998.

LORD C, RUTTER M, GOODE S and HEEMSBERGEN J. Autism diagnostic observation schedule: A standardised observation of communicative and social behaviour. *Journal of Autism and Developmental Disorders, 19:* 185-212, 1989.

LORD C, RUTTER M and LE COUTER A. Autism-diagnostic interview – revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 24:* 659-685, 1994.

OSTERLING J and DAWSON G. Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders, 24:* 247-257, 1994.

PIERCE K, MULLER RA, AMBROSE J, ALLEN G and COURCHESNE E. Face processing occurs outside the fusiform 'face area' in autism: Evidence from functional MRI. *Brain, 124:* 2059-2073, 2001.

SCHULTZ RT, GAUTHIER I, KLIN A, FULBRIGHT RK, ANDERSON AW, VOLKMAR F, SKUDLARSKI P, LACADIE C, COHEN DJ and

Gore JC. Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and asperger syndrome. *Archives of General Psychiatry, 57:* 331-340, 2000.

Tantam D, Monaghan L, Nicholson H and Stirling J. Autistic children's ability to interpret faces: A research note. *Journal of Child Psychology and Psychiatry, 30:* 623-630, 1989.

Taylor MJ, Edmonds GE, McCarthy G and Allison T. Eyes first! Eye processing develops before face processing in children. *Neuroreport, 12:* 1671-1676, 2001.

Taylor MJ, Itier RJ, Allison T and Edmonds GE. Direction of gaze effects on early face processing: Eyes-only versus full faces. *Brain Research and Cognitive Brain Research, 10:* 333-340, 2001.

Teunisse JP and De Gelder B. Do autistics have a generalized face processing deficit? *International Journal of Neuroscience, 77:* 1-10, 1994.

Tucker D. Spatial sampling of head electrical fields: The geodesic sensor net. *Electroencephalography and Clinical Neurophysiology, 87:* 154-163, 1993.

Mark H. Johnson, Centre for Brain and Cognitive Development, School of Psychology, Birkbeck College, University of London, 32 Torrington Square, London WC1E 7JL, UK.
e-mail: mark.johnson@psychology.bbk.ac.uk