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Motion and Pattern cortical potentials in adults with high functioning autism spectrum disorder

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Abstract

Purpose:

Autism Spectrum Disorder (ASD) is a condition in which visual perception to both static and moving stimuli is altered. The aim of this study was to investigate the early cortical responses of subjects with ASD to simple patterns and moving radial rings using visual evoked potentials (VEP).

Methods

Male ASD participants (n=9) and typically developing (TD) individuals (n=7) were matched for full, performance and verbal IQ ($p>0.263$). VEPs were recorded to the pattern reversing checks of 50'sidelength presented with Michelson contrasts of 98% and 10% and to the onset of motion – either expansion or contraction of low contrast concentric rings, (33.3% duty cycle at 10% contrast).

Results

There were no significant differences between groups in the VEPs elicited by pattern reversal checkerboards of high (98%) or low (10%) contrast. The ASD group had a significantly larger N160 peak (1.85 x) amplitude to motion onset VEPs elicited by the expansion of radial rings ($p=0.001$). No differences were evident in contraction VEP peak amplitudes nor in the latencies of the motion onset N160 peaks. There was no evidence of a response that could be associated with adaptation to the motion stimulus in the inter-stimulus interval following an expansion or contraction phase of the rings.

Conclusion

These data support a difference in processing of motion onset stimuli in this adult, high functioning ASD group compared to the TD group.

Key Words:

Autism Spectrum Disorder, Motion onset, Pattern reversal, VEPs, Contrast

Introduction:

Autism Spectrum Disorder (ASD) is a neurodevelopmental condition affecting approximately 1:100 individuals, with a higher prevalence amongst males [1]. Individuals with ASD have difficulties in three core diagnostic domains of: reciprocal social interaction, communication and repetitive behaviours and restricted interests [2]. Whilst, the direct aetiology of ASD remains unclear, several studies indicate a complex genetic origin, which may be influenced by environmental factors such as hormones or inflammation to disrupt neural maturation in the brain, [3-5]. There is some evidence of this in MRIs of individuals with ASD who display an increased white matter bulk and reduced long range connectivity between regions of the brain, most notably laterally, but also from anterior to posterior. The general model is one of local over-connectivity and reduced long range connectivity between functional regions of cortex [6-11].

Several theories have been proposed to explain the ASD phenotype. One suggests that ASD is a result of weak central coherence (WCC) [10,12]. This means that individuals with ASD have difficulty in assimilating and making sense of the whole. The idea of weak coherence is supported by elevated motion coherence thresholds in children [13,14]. Poor performance in this motion domain suggests a difference in the processing of simple motion stimuli. In addition ASD individuals outperform typical observers in static tasks such as visual search [15-17] and embedded figures tasks [14, 18, 19] which support a difficulty with

grasping the gestalt and being drawn into the finer detail of objects. Thus, according to WCC theory, there is a natural cognitive bias towards the local over the global perspective and superior performance in tasks requiring the detection of detail.

An alternative model, proposed by Mottron et al (2006) [20] suggests that enhanced perception in sensory cortex contributes to ASD. Evidence for this is found in enhanced pitch discrimination in the auditory domain [21]. In the visual domain, Mottron's group revealed a difference in thresholds for orientation discrimination of first and second order gratings. First order gratings are those in which spatial contrast is defined by luminance, and processed in V1, whilst second order gratings are those defined by texture and draw upon extra-striate regions for correct orientation discrimination [22]. The ASD group was superior at determining the orientation for the first order task, but their performance was inferior for the second order task, compared to the comparison group [23]. This enhanced perception of simple stimuli implies that there are differences in the way that visually salient features are initially processed by V1. It is argued that these differences in early sensory processing are fed forward to higher cortical regions, where they impair ASD performance for more complex stimuli. The enhanced perception theory was supported by findings that individuals with ASD have higher than normal visual acuity [24], but this was subsequently shown not to be the case [25, 26].

Most visual processing studies of complex stimuli, e.g. motion, in individuals with ASD have used imaging or psychophysical methods; few have looked at electrophysiological responses. For example Mottron et al 2006, using

rotating, translating, spiralling or expanding/contracting motion stimuli, found second order (texture defined) motion discrimination thresholds were higher in an ASD group compared with a matched comparison group, but first order (luminance defined) motion discrimination thresholds were not significantly different [23,27]. Therefore, for both static and moving, complex, texture defined stimuli ASD discrimination thresholds were greater. However for simple luminance defined stimuli superior performance was seen only in the static domain with no differences in motion discrimination thresholds for the first order motion defined stimuli.

There has not always been agreement in all findings with respect to motion processing in ASD, in part reflecting the varied stimuli and heterogeneity of the clinical groups studied, e.g. Milne et al (2002) described increased motion discrimination thresholds in children, whilst Del Viva et al (2006) found no differences in a more tightly matched group of children based on IQ measures [28]. For reviews see [29,30]. In one large recent study of 89 ASD and 52 adolescents no group differences in biological motion, motion coherence and form-from motion were detected, although individuals with the lowest IQs performed most poorly on the biological motion task [31].

The motion onset VEP in humans has a major motion related component (N160) occurring between 150 and 200 msec around the extra striate temporo-occipital and associated parietal cortical areas with high contrast sensitivity [32-35]. The preceding P1 component is related to pattern processing at the onset of the motion stimulus [32,34] and associated with the striate cortex [35] whilst the

P2 component occurring at ~ 220 msec with wide inter-subject variability [32] is believed to be associated with motion detection and is highly susceptible to motion adaptation [36].

Our aim was to assess early cortical responses to pattern reversal stimuli to ascertain if, using electrophysiology, these cortical potentials differed between groups and might further support theories of enhanced perception demonstrated by orientation discrimination thresholds. Furthermore, we wished to examine the motion onset- evoked potentials to help our understanding of the differences in motion perception seen in adult high functioning ASD individuals.

Methods

Participants

Cognitive measures of ability were used to match the groups for verbal, performance and full intelligence quotient, (IQ), as measured by the Wechsler Adult Intelligence Scale (WAIS-III^{UK}). Participants with ASD were diagnosed according to conventional criteria. A review of available medical records and assessment with the Autism Diagnostic Observational Schedule (ADOS) [37] confirmed that all met DSM-IV-TR criteria for ASD. The Autism Quotient (AQ) was used as a further measure to characterize the individuals on their severity of ASD [38]. Male adults with ASD (n=9) and typically developing (TD) males (n=7) were recruited, age ranged 23-56 years with the ASD group being significantly ($p=0.023$) younger (ASD 36.6 ± 11.8 and TD 48.9 ± 5.5 years). The groups differed on the AQ score ($p<0.001$) but not on measures of IQ ($p>0.263$) (Table 1). Research and Ethical Approval was obtained by City University Senate

Research Committee, all experiments were in accordance with the declaration of Helsinki.

	ASD (n=9)	**TD (n=7)**	**p**
Age	36.6 ± 11.1[22.9-55.7]	48.9 ± 5.0[41.8-55.8]	0.023
FIQ	111 ± 17 [81-134]	104 ± 16 [77-128]	0.411
PIQ	110 ± 16 [84-136]	100 ± 14 [75-122]	0.263
VIQ	109 ± 16 [81-135]	106 ± 15 [82-125]	0.666
AQ	31 ± 8 [22-42]	13 ± 7 [4-21]	<0.001

_____Insert Table 1 near here_____

Stimuli

High and low contrast pattern reversal checks and radially expanding and contracting, low contrast, circles [39], were generated using a *CRS visage* system. Stimuli were displayed on a NGC *CRT* 32 inch Multisynch monitor and viewed binocularly at 1m. Pattern stimuli were black and white checks of 50' side length, (0.85cpd) of high (98%) or low (10%) Michelson contrast, with 3 phase reversals per second. Motion stimuli also had 10% contrast and consisted of expanding and contracting radial rings, based on the stimuli designed by Kremlacek et al 2004 The duty cycle was 33.3% consisting of 300ms expansion 600ms stationary interstimulus interval, 300 ms contraction and a further 600 ms stationary interstimulus phase. Stimuli were corrected for equal visibility in a 30 degree stimulus field using the cortical magnification factor (CMF) = 1/(0.1x eccentricity +1). The rings had a constant expansion or contraction temporal frequency of 5 c/s across the whole stimulus field, the local motion velocity increased (5-25 deg/s) while spatial frequency was decreasing (1-0.2 c/deg). Contrast modulation of the

motion stimuli used a sine function so that the maximal contrast was either 10% or 90%. The expansion or contraction stimuli occurred randomly and were always separated by an interstimulus interval. The VEPs to each event were epoched and evaluated separately. A central red fixation dot was present during recordings.

Recordings

VEPs to these stimuli were extracted from the EEG recorded with a *Neuroscan* multi- channel system and 40 channel *Quik-Cap*. Electrode impedance was < 5kΩ. Each stimulus run lasted 2 minutes and each stimulus was randomly presented 3 times. A grand average of the VEPs from each of the three stimulus runs was computed for each individual. The grand average for each individual for each run was then used to compute the group grand average as shown figures (1-3). There were no differences in the number of traces rejected due to artefacts for each group. The amplitudes were calculated from peak to peak and the time to peak from stimulus onset to the peak. EEG recordings were epoched off line from -50 to 300 ms with ± 100μV cut-off and filtered between 1Hz to 30Hz, using Fz as reference.

Data Analysis

The largest amplitude signal occurred at Oz to the pattern P4 to motion stimuli. The grand averages for each individual of each of the stimulus runs were used in the statistical calculations. The amplitude and time to the major peaks N80 and P100 of pattern reversal VEPs (high and low contrast) and N160 (expand and contract) of motion onset VEPs were compared between groups (ASD and TD) using multiple ANOVA with age as a covariant (MANCOVA) to control for the

differences between groups on this measure. Following significant multivariate analysis, the univariate ANCOVA's for each factor were analysed and adjusted using the sequentially rejective Bonferroni-Holm method [40]. Student's t-test was used for comparisons between groups for age and IQ measures with $p<0.05$ as significant. All data are presented as mean ± SD with calculations performed with IBM SPSS statistics 19.

Results

Pattern Reversal VEPs

For pattern reversal high and low contrast checks a positive (P100) component was evident over Oz in both groups. There were no significant differences between the groups in either amplitude or latency of the high or low contrast pattern reversal VEPs. For the high contrast pattern reversal condition, the equality of covariance was not significant (Box's *M* 18.5, F=1.2, p=0.262). A one-way MANCOVA revealed no significant multivariate main effect for group, though the power was low. Wilks' λ =0.840, $F(4,10)=0.475$, p=0.754, power to detect the effect was 0.121. Given there was no overall effect of group on the high contrast pattern responses follow up univariate analyses were also non significant ($p>0.328$) with low power to detect any effects > 0.075.

Similarly, for the low contrast pattern reversal response the equality of covariance was also non-significant (Box's *M*=14.6, F=0.9, p=0.456). The one-way MANCOVA did not reveal a multivariate main effect for group. Wilks' λ = 0.885, $F(4,10)$ =0.326, p=0.854, power to detect the effect was also low for this low contrast stimulus 0.097. Follow up univariate ANCOVAs revealed no

significant effects on amplitude or latency of N80 or P100 peaks (p>0.340) and power to detect the effects >0.050. Therefore, we did not find any significant findings in the VEP responses to high or low contrast pattern reversal stimuli between the ASD and TD group with age as a covariate (Figure 1 and table 2).

_____insert figure 1 near here_____

Motion onset VEPs

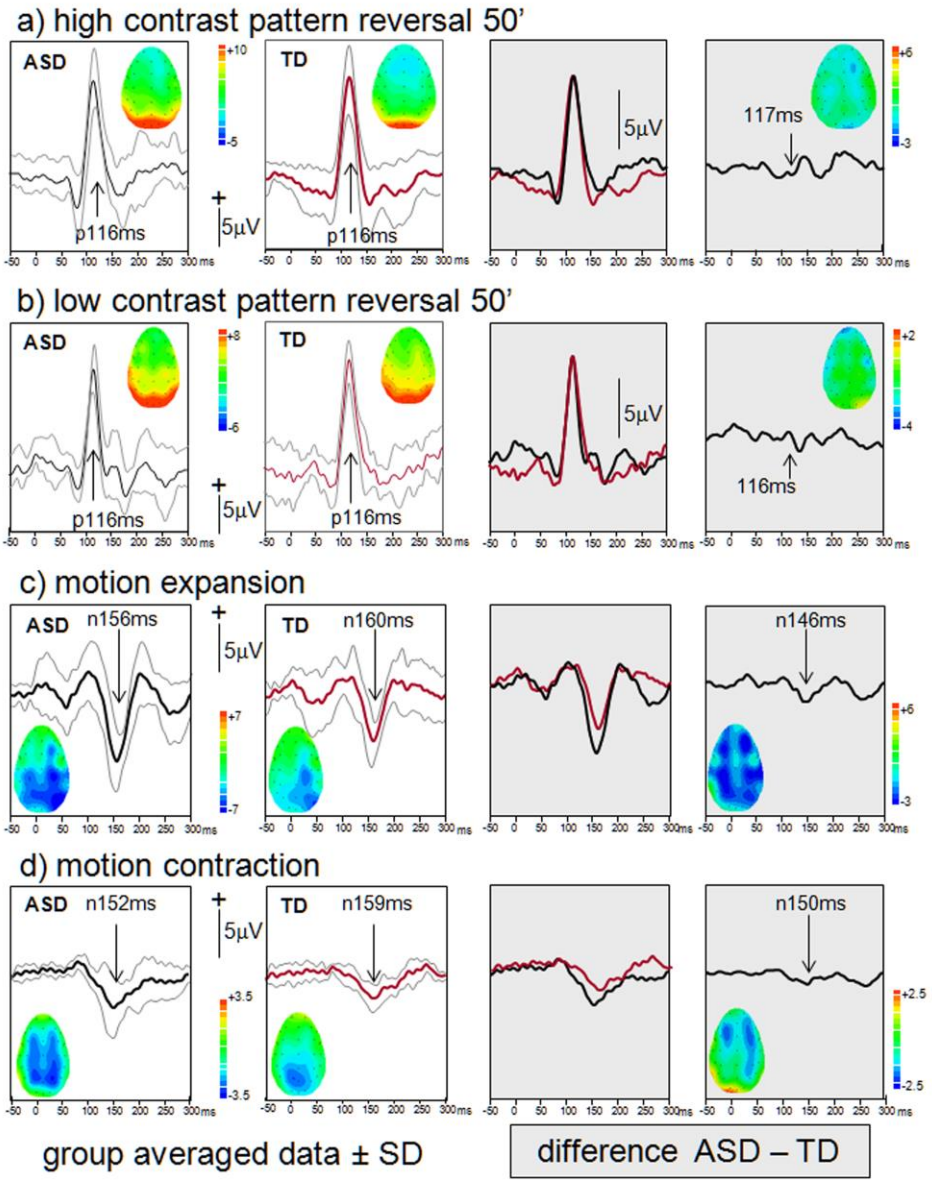
The main factor of interest was whether the major N160 component of the motion elicited response differed between groups. The amplitudes of the P1 and P2 were variable and not analysed in this series as most did not exceed the noise level of >2μV [35]. For the motion onset responses the equality of covariance was also non-significant (Box's *M* 22.6, *F*=1.5, p=0.127). The one-way MANCOVA revealed a significant multivariate effect for group, Wilks' λ =0.229, *F*(4,10)= 8.4, p=0.003, with a high power to detect the effect of 0.969. Given the significance of the overall test, the univariate main effects for group were examined using the Bonferroni-Holm adjusted p-values for the four tests. There was a significant univariate main effect of group for the N160 expanding amplitude, with adjusted p-value of 0.0125: *F*(1,13)=19.8, p=0.001 with a high power of 0.984 to observe this effect. The N160 contracting amplitude was not significant at the adjusted p-value of 0.016: *F*(1,13)=6.5, p=0.025 with observed power of 0.652. The times to peak for the N160 amplitudes for expanding *F*(1,13)=1.4, p=0.256 observed power 0.196 and contracting rings *F*(1,13) =2.7, p=0.126, observed power 0.328 were not significant between groups.

Therefore, overall the ASD group had a significantly larger amplitude N160 motion onset VEP to the expanding motion stimulus compared to the TD group. This effect was present for the contracting stimulus but failed to reach significance once repeated measures were taken into account. (Figure 2 and table 2 for descriptive values).

The multivariate analysis took into account the differences in age as the time to the N160 peak increases with age [33,42]. If age is not used as a covariate then the overall results are the same with a significant difference in the expanding amplitude of the N160 peak (p=0.003) and non-significant effect on the N160 contracting amplitude (0.040) after correction for multiple measures. There were no significant differences between groups on the times to the N160 peaks (p>0.071).

Table 2 SUMMARY RESULTS FOR VISUAL EVOKED PATTERN AND MOTION POTENTIALS

High Contrast Pattern Reversal				
	Component	**ASD**	**TD**	**(*F*, p)**
Amplitude	N80	4.3 ± 2.7	-3.5 ± 1.5	0.5, 0.491
Latency		82 ± 2	82 ± 2	0.2, 0.624
Amplitude	P100	15.9 ± 3.6	15.8 ± 5.6	0.2, 0.694
Latency		115 ± 2	116 ± 2	1.0, 0.328
Low Contrast Pattern Reversal				
Amplitude	N80	-2.7 ± 1.3	-2.6 ± 1.5	0.1, 0.842
Latency		83 ± 4	83 ± 5	0.0, 0.958
Amplitude	P100	9.0 ± 2.8	7.4 ± 1.7	0.1, 0.849
Latency		116 ± 2	117 ± 2	1.0, 0.849
Motion Expansion				
Amplitude	N160	-10.4 ± 3.3	-5.6 ± 1.4	19.8, 0.001
Latency		157 ± 9	161 ± 7	1.4, 0.256
Motion Contraction				
Amplitude	N160	-7.2 ± 2.5	-4.7 ± 1.5	6.5, 0.025
Latency		168 ± 8	175 ± 5	2.7, 0.126



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Discussion

We assessed two areas of visual perception that previous psychophysical investigations suggest differ in the autistic population. Our VEP data show differences in the main motion related N160 component between 150 and 200 ms. Recent findings using VEP data and neuroimaging techniques confirm that the N160 originates from extrastriate cortex, most likely near V3/V3A and MT/V5

[35,46]. However we did not find differences in the low spatial frequency components of pattern contrast VEPs processed by V1 [35,47].

Jemel et al (2010) found that ASD subjects did not show any spatial tuning of the pattern reversal VEP N80 to mid and high spatial frequencies, in contrast to typically developing subjects, and suggested this contributes to altered visual perception [43]. This implies atypical cortical processing in ASD with respect to simple stimuli. These authors did not observe any differences between the groups when low spatial frequency gratings were used and found no differences in the properties of the P100. Our data support these observations. Although we did not vary spatial frequency, we used a check size (0.85 cpd) close to Jemel et al's low spatial frequency stimuli, and at high (90%) and low (10%) spatial contrasts there were no group differences in either N80 or P100 amplitudes or timings ($p>0.159$).

Our adult, high functioning ASD group did show significantly larger motion onset VEP negative peak amplitudes (N160) to radially expanding low contrast rings, than the TD group. The N160 component has been associated with the perception of global coherent motion and local pattern characteristics [44-47], stimulus velocity and spatial frequency [33]. The preceding P1 component is influenced by spatial contrast and relates to activity in V1 [35]. For most of our participants the P1 of the motion onset VEP was small and ill- defined, (< 2μV) and could not be fully analysed. Although the ASD group were younger than the TD group, and time to peak of the N160 increases with age [32], peak latency was similar between the groups. The finding of larger N160 amplitudes in the ASD group remained significant when age was not included as a covariate

To our knowledge these data are the first electrophysiological evidence of differences in motion processing in ASD. There is some fMRI evidence of altered motion processing in ASD; for example whilst biological motion recognition typically uses a unitary parietal-temporal axis, whilst ASD individuals utilised a different network comprising form and motion centres rather than the unitary network used by the TD group [48]. In a separate study, Koldewyn et al (2011) found reduced activity to biological motion in the posterior superior temporal sulcus, parietal and frontal lobe activity [49]. However, in the psychophysical experiments they found their ASD adolescents had higher thresholds for detecting biological motion than the TD group, but did not find any differences in motion coherence thresholds. This led them to suggest that the deficits of motion processing in adolescence may derive from differences in the higher-order social or attentional networks related to interpreting biological motion rather than the earlier motion centres (V5/MT) [49].

Yet others have described higher thresholds in adolescents and in younger children with ASD in detecting coherence motion too; though these may only be evident in individuals who fit the more classic autistic rather than the Asperger profile [14,50,51]. Mostly ASD performance for motion tasks has been reported as being worse than TD, but a local motion detection advantage has been reported in adolescents with ASD who were better at discriminating the differences in speeds of sequential random dot kinetograms when the inter-stimulus interval was long (3s) but not when the window was short (0.5s) [53].

Discordance in the results of psychophysical tests of motion perception have been attributed to construction of coherence motion stimuli, some of which may provide local grouping cues, and may not therefore be true deficits in global motion processing (Dakin and Frith (2005) [29]. To overcome this, Vandenbroucke et al (2008) [52] used two moving plaids that could be perceived either as a coherent whole or as two transparent gratings sliding over each other. No significant difference was found between groups in the duration of either percept. This may reflect the low spatial frequency of the plaids and mid to high spatial frequencies might better reveal differences, as shown by Jemel et al's electrophysiological findings [43].

Clinical differences in high and low functioning individuals on the ASD spectrum, along with age and the demands of the complexity of the studies may also contribute to discrepant conclusions as proposed by Kaiser and Shiffrar (2009) [54]. In addition, individuals with ASD show an altered behavioural style of how they attend to the world [55]. Although the motion after effect [56], and psychophysical motion coherence thresholds [57] can be modulated by attention, our ASD and TD groups showed similar artefact rejection rates during the acquisition the of motion onset VEPs and its unlikely that attention to the stimuli affected these data.

Our objective electrophysiological findings of a difference in the motion-onset VEP to an expanding ring in a small sample of high functioning ASD adults provides evidence supporting an underlying difference in the cortical response to motion in ASD rather than to low spatial frequency pattern reversal checks. The

difference in the cortical response to motion onset may be the result of altered connectivity between visual centres and higher cortical regions [9] or to the changes in cortical structures that are seen in ASD individuals [6-8].

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Figure and table legends

Figure 1 legend

Four rows of traces corresponding to each stimulus condition are displayed: a) high contrast 50' pattern reversal checks, b) low contrast pattern reversal checks, c) motion expansion and d) motion contraction stimuli. The 1st and 2nd columns show group grand averaged traces ±1 SD for the ASD and the TD group respectively. In the 3rd column the mean waveforms from each group are overlapped to illustrate the amplitude difference between groups. In the 4th column the arithmetic difference between these traces is shown as a waveform and as a map. Maximal pattern reversal VEP data were taken from Oz and the motion VEP data from P4. Isopotential maps are shown at the latency at which the peak occurs. The main response to motion onset N2 occurred at (N160) is

significantly larger for the ASD group to the expanding rings ($p=0.001$), but not for contraction.

Table 1 Participant details. ASD: autism spectrum disorder, TD: typically developing, FIQ: full intelligence quotient, PIQ: performance intelligence quotient, VIQ: verbal intelligence quotient, AQ: autism quotient.

Table 2 legend

Summary of the major VEP components of pattern and motion onset stimuli for ASD and TD groups. (Amplitude in micro volts and latency in milliseconds). Univariate results shown with $p<0.0125$ as significant.

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