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Magnocellular Impairment in Autism Spectrum Disorders as Assessed by Visual Evoked Potentials

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By

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Abstract

Motion perception in Autism Spectrum Disorders (ASD) has become a salient topic in the past few years. Several studies have found links between abnormal motion processing and symptomatology in ASDs, suggesting that impaired magnocellular function may underlie some of the various symptoms and outcomes in individuals with ASD. The following study explored achromatic motion perception processing differences in high-functioning adolescents with Autism Spectrum Disorder (ASD). Previous studies have shown abnormalities in the way that individuals with ASD perceive motion, including biological and coherent motion, but few studies have examined pattern-reversal and expanding and contracting motion. Abnormalities can have implications for ASD symptomatology, including emotional/facial expression processing, and difficulty with integration of visual information with other cognitive information, such as social cues and communication systems. Two motion stimuli, a reversing checkerboard and an expanding and contracting dartboard, were displayed for ASD male participants and male and female neurotypical control groups. Visual-evoked potentials (VEPs) were used to examine differences in time-to-peak amplitude, area amplitude, and latency of the VEP in participants. Results showed that there are magnocellular impairments in ASD. There were several main effects for check size and contrast and significant 3-way interactions for contrast, check size and diagnosis in P1 area amplitude and N1 fractional area latency for all three groups, and P1 peak amplitude for ASD males and neurotypical males. Results imply that contrast and size in environment have an effect on how individuals with ASD process visual stimuli, which can explain some of their symptomatology and difficulty with other forms of processing that follow the magnocellular process. Future research can focus on successful interventions that aim to correct these deficits in visual-motion processing and as a result, help treat the individuals with innovative therapies that improve quality of life. These results can also have implications for early diagnosis and treatment, and possibly prevention.

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1. Introduction

Autism Spectrum Disorder (ASD) is a developmental disorder that results in impairments in verbal and nonverbal communication, understanding and coping with the social environment and failure to develop normal social relationships (Baron-Cohen et al., 1985). In addition to cognitive and social impairments, perceptual deficits have been discovered that can be instrumental to understanding the disorder and can aid in early diagnosis and treatment. Specifically, perceptual scientists have proposed that impairments of the magnocellular pathways can lead to deficient processing of moving stimuli, which can result in dysfunctional higher-order processing (Milne et al., 2005). New DSM-V criteria for ASD include 3 severity levels. Level 1 “requiring support” includes deficits in social communication causing noticeable impairments in social interactions and unsuccessful responses to social overtures of others. Level 2 “requiring substantial support” shows marked deficits in verbal and non-verbal social communication skills and impairments in social interactions even with supports in place. Level 3 “requiring very substantial support” includes severe deficits in verbal and nonverbal social communication skills and very limited initiation of social interactions (apa.org).

We assessed the impact of ASD on the magnocellular system using visual-evoked potentials (VEPs). The question of interest concerned whether or not autistic individuals have a different response to moving stimuli than neurotypical individuals, and if so, how

altered early visual signals affect recognition of moving stimuli, and ultimately, of the visual world.

Common effects of ASD are poor cognitive and social skills, including difficulty tracking gaze, lack of pretend play, poor processing of facial expressions, delayed language development, and repetitive behaviors such as hand flapping and echolalia (Frith, 1998). Vision abnormalities have become a wide focus of research in ASD due to the vagaries of visual perception implicated by poor gaze and difficulty with emotional facial processing (Gepner, 2002). Additionally, the repetitive movements and a seeming obsession with objects in motion have drawn more attention to study of the motion pathways in autism. Because movement is essential to comprehending the visual world, deficits in visual processing can have severe implication for processing of emotional and social meaning of visual stimuli. Several types of visual-evoked potentials, including steady-state VEPs and motion-VEPs have been used to test magnocellular function in a variety of patient populations in order to understand how deficits in this system relate to behavior and symptomatology of particular disorders.

The magnocellular pathway of the visual system is one of at least three systems that carries specific visual information via ganglion cells to the lateral geniculate nucleus (LGN), and from there, to primary visual cortex (Kaplan and Shapley, 1986). Motion processing systems carry a signature low-contrast threshold with a saturating response characteristic. Another main pathway is the parvocellular pathway, thought to be mostly in charge of color vision processing and acuity, including processing of fine

details, and has a higher contrast threshold than does the magnocellular pathway (Kaplan and Shapley, 1986). The magnocellular pathway is considered largely insensitive to color when luminance is balanced, has a higher contrast sensitivity than the parvocellular pathway, is responsive to lower spatial frequencies and higher temporal frequencies, and has transient responses. The fibers of the magnocellular pathway are thick, and thus, speed of transfer is high due to faster impulse conduction (Liu et al., 2006). This quick conduction speed has been dubbed "the magnocellular advantage" by Laycock et al (2007). Such an advantage is thought to be instrumental in integrating global information processed by the magnocellular system and projecting it to several areas of the brain involved in visual processing, including primary visual cortex (PVC). Thus, a deficit in magnocellular processing removes this advantage, and causes problems in the integration and transmission of visual information to these critical processing areas. Several studies of visual processing in ASD have focused on abnormal facial processing, which occurs in separate brain areas, but visual motion processing studies took longer to develop momentum.

Measurement of electrophysiological correlates of motion processing in people was pioneered by Clarke (1973). He was one of the first to look at changes in the motion of a patterned field, giving rise to use of the pattern-reversal stimuli in subsequent studies. Since then, disagreement on when the motion-specific components occur has been mostly resolved, and interest has surged in the past decade in motion processing as it relates to a variety of settings, including disorders like Dyslexia and ASD. Pattern

reversal is the most common stimulus used in clinical settings (Harding et al, 1996), and was used in the current project.

Dakin and Frith (2005) reviewed the visual perception research in ASD, including processing of fine detail, global processing, and motion perception. The authors found that fine processing research was robust, but studies of global processing and motion processing were lacking. Specifically, the authors declare that motion perception is impaired in ASD, but that explanations in terms of magnocellular deficits are insufficient. Starting with earlier studies, we will illustrate the progression of vision research in ASD and where information is still needed, coming to how this study will address some of the issues brought up by Dakin and Frith.

Initial studies in the late 80s and early 90s have shown that ASD is associated with certain perceptual styles that include above-normal local processing abilities (Frith, 1989; Joliffe and Baron-Cohen, 1997; Shah and Frith, 1983, 1993). However, some researchers suspect that global and configural processing are compromised as a result of this superior local processing ability. Joliffe and Baron-Cohen (1997) demonstrated that individuals with ASD are able to detect static target shapes hidden within complex line designs more rapidly than typically-developing controls. It is suggested that typically-developing participants exhibit a more global processing style, analyzing images in their entirety rather than as elements that produce a whole image (Kaiser and Shiffrer, 2009).

Shah and Frith (1993) showed other examples of a local processing advantage in ASD by demonstrating that individuals with ASD showed superior performance on a

block design task, while Mottron et al (1999) showed superiority in individuals with ASD in the reproduction of impossible figures. Various other authors showed superior performance by participants with ASD in other local processing tasks, such as visual search (O'Riordan et al., 1998), the ability to learn confusing patterns (Plaisted et al., 1998) and performance on tasks with Navon figures that are incongruent across local and global analysis levels (Wang et al., 2007). Kaiser and Shiffrer (2009) suggest that while individuals with ASD have the ability to use global processing, their mind somehow defaults to a local processing level, which could support theories by Gepner et al (2002) that individuals with ASD can be "trained" to process stimuli more appropriately, as discussed later.

Most of these earlier studies of visual processing in ASD suggest that individuals rely heavily on low-level perceptual information to accomplish more complex cognitive tasks. This led Kaiser and Shiffrer (2009) to compare these studies to studies of local versus global motion processing. Wallach (1976) discusses the aperture problem in motion processing, in that the visual perception of a moving object or surface requires integration of information over disconnected regions of retinal space, and because of that, motion perception under real-world conditions is fundamentally global. If this is true, there may be inherent problems in motion perception in individuals with ASD. This information created a distinction between local and global motion perception that has divided researchers in terms of the types of motion that may be perceived differently in ASD. Local motion is one continual moving point, which is rare to witness in the real world, and global motion processing includes multiple points or contours moving

relative to one another, as in with coherent motion, biological motion, and pattern-reversal motion.

Gepner et al (1995) looked at optic flow in ASD and neurotypical controls by having participants stand on a force plate positioned near a large screen, upon which circular sinusoidal gratings were projected. When set in motion, they contracted and expanded at different speeds, and postural sway was recorded as participants closed their eyes or fixated on the center of the grating. In typical children, postural sway varied with perceived optic flow, whereas in ASD, optic flow had no significant impact on postural sway. This was the first study that focused on environmental motion as it related to motor responses in ASD, and results resonated across the ASD research community. Studies that followed, reviewed below, began to scale down the mechanisms of visual processing to their basic components in order to determine the most fundamental level at which motion deficits occur in ASD.

Studies that followed began to examine motion coherence thresholds in ASD, as well as the observance of biological motion. While earlier studies were behaviorally-focused, in the early 2000s people began to examine motion perception on an electrophysiological level.

Spencer et al (2000) first demonstrated that children with ASD had higher motion coherence thresholds than neurotypical children, by having children indicate which of three rectangular sub regions depicted dots that oscillated in opposite phase to dots in a comparison stimulus. Results indicated that thresholds were elevated by over 45% in children with ASD than in controls. Milne et al (2002) added evidence of elevated

motion coherence thresholds by using a behavioral method of studying deficits in motion processing via a random dot kinematogram (RDK). The RDK presents participants with a series of random dots moving in seemingly random patterns, with a small group of dots moving in unison in one particular direction. As the percentage of dots moving in unidirectional unison increases, so decreases the threshold for detecting them. The percentage at which a participant is able to detect the direction of motion of the coherent dots is considered their motion coherence threshold. The authors found that autistic participants had a higher threshold of detection; that is, they were unable to see the directional pattern until well after typically-developing participants, further suggesting magnocellular/dorsal stream impairment. Similar studies (Spencer et al, 2000) found ranges of 22-25% thresholds for ASD and 11-15% for neurotypicals. These studies, as discussed next, were soon followed by electrophysiological studies, but as discussed below, these studies were not without their faults.

McCleery et al (2007) studied abnormal magnocellular pathway processing in infants at risk for autism. In this study, infants with undeveloped or underdeveloped visual processing structures were studied based on their potential for autism to develop (based on the presence of another autistic individual within the family). However, as autism is a multifaceted disorder, there is no sure way of telling whether or not these participants will go on to develop autism. However, these studies are important in assessing the idea that particular dorsal stream dysfunctions can be inherent in early infancy, which can be instrumental in early diagnosis. While the authors claimed to be using electrophysiological methods, the authors actually used a forced-choice technique

with grating stimuli and studied eye-gaze behavior to judge stimulus location. It is difficult to generalize this type of behavior to motion perception deficits in ASD, as infants have underdeveloped magnocellular systems (Milne et al., 2007) and electrophysiological evidence was not collected, only psychophysical evidence.

Sutherland and Crewther (2010) studied motion perception in adults of normal intelligence who displayed some characteristics or traits associated with those of individuals diagnosed with ASD. They used low and high-contrast stimuli with EEG to compare groups scoring high versus those scoring low on the Autism Quotient Exam. High-scoring individuals showed magnocellular processing delays, which, according to the authors, implied that those participants have a decreased ability to benefit perceptually from feedback normally associated with the magnocellular advantage. The magnocellular advantage is a term referring to the high conduction speed of the magnocellular pathway that is integral in relaying information to other brain areas for integrative and global processing (Laycock et al., 2007).

Sutherland and Crewther believe that this could be applied to individuals with ASD; however, this is based on their belief that “everyone has autistic characteristics to a greater or lesser degree”. The authors fail to further explain the meaning behind this statement and why results could easily transfer to a population of individuals actually diagnosed with ASD. The intent of the present study was to apply the authors’ general principles to a population of individuals actually diagnosed with ASD and see if the results are similar. If higher-scoring adults on the AQE did indeed suffer delay in magnocellular processing, then their predictions would imply the same or worse delay

on a diagnosed population. Using children as opposed to adults, may, however, cause some discrepancies due to differences in brain development at different stages of life.

Many visual processing studies in ASD have shown that there are abnormalities with facial processing (which occurs in the fusiform face area, not primary visual cortex or the LGN), including configural face processing deficits, abnormal activation of the brain areas involved in social and emotional processing, and abnormal functioning of the amygdala and fusiform face area during face perception (Davies et al., 1994; Schultz, 2005; Lahaie et al., 2006) Hadjikhani et al., 2007). However, in several other studies exploring vision and perception in ASD (occurring in visual areas other than the FFA), items like visual acuity, spatial frequency, color vision and contrast sensitivity have shown controversial results. As far as visual processing abnormalities, motion processing still requires significantly more research. For example, Franklin et al (2008) tested color perception in children with ASD in two experiments. In the first experiment, the authors tested accuracy of color memory and search, and showed that children with ASD were significantly less accurate at color memory and search than typically developing children. In the second experiment, chromatic discrimination and categorical perception of color were tested using a target detection task, and children with ASD were less accurate than controls at detecting chromatic targets against chromatic backgrounds, however, they were equally as quick when target detection was accurate. The authors found that strength of categorical perception of color did not differ for the two groups.

Davis et al (2006), however, review psychophysical evidence in ASD and conclude that participants with ASD have normal color and form perception. De longe et al (2007)

assessed visual information processing in high-functioning individuals, using tasks for contrast sensitivity and form perception and found no deficits in contrast sensitivity for low or high spatial frequencies or for form perception between participants and typically-developing controls. Other evidence in color vision is lacking, especially with EEG, and while general research regarding color perception in ASD has stagnated, various researchers are looking into other stimuli and methods to examine new ways to study motion perception in ASD as it becomes clear that there are multiple components involved in motion processing.

Bach and Hoffman (2000) and Bach and Ullrich (1995) examined non-retinal mechanisms for motion detection by using an expanding and contracting dartboard. The purpose of using such a stimulus was to explore other types of motion, as pattern-reversal motion only represents one type of motion stimulus that occurs in the environment. According to them, a pattern-reversal stimulus triggers both form-processing and motion mechanisms that can be discriminated by latency. The dartboard is less contaminated by other motion responses. Currently, no visual processing studies have applied this stimulus to ASD. By using it in addition to pattern-reversal stimuli, we can explore components of motion processing that are not contaminated by other motion processes.

With the exception of the study by Sutherland and Crewther, many other studies of visual processing in autism have either, like Milne et al (2002), focused on behavioral methods of measurement, which lack the detail and physiological evidence underlying the behavior, or have been largely related to neuroimaging studies of social

communication and intention (Pelphrey et al., 2005). The implications for deficits in early processing are profound: because the visual system works together in a process of transmitting information and receiving feedback from other structures that involve memory and emotion. Because the magnocellular system is one of the first lines of communication, and the fastest of the systems in this chain, a deficit can have severe consequences for information processing. Thus, using electrophysiology, we directly measure responses from the brain as VEPs recorded from electrodes attached to the scalp at designated areas as reviewed by Kuba et al (2002). This type of data and the variety of stimuli that we will use can provide new insight into magnocellular functioning in young adults with ASD, which can help explain some of the symptomatology and lead to innovative new treatments and interventions. Dysfunction in magnocellular visual processing also has potentially profound implications for early diagnosis.

2. Methods

2.1 Participants

Eight male participants with ASD, six male neurotypical controls and five female neurotypical controls between the ages of 10 and 15 were tested. VEPs were recorded from three locations on the scalp, at Oz, Pz and Fz. Target participants carried a confirmed diagnosis of ASD, and severity was assessed via administration of the GARS2

and the ADOS. None of the participants carried a comorbid diagnosis of mental retardation and/or seizure disorder. Informed consent was received from parents and verbal assent was received from participants. A preference assessment was performed to determine enjoyable stimuli to present to participants while sensors were being attached and for any waiting periods between stimulus presentations. We allotted several sessions if necessary for habituating participants to the sensors, but all but one of the participants were able to adapt and perform the tasks within the first session. One participant with ASD was eliminated from the study because of extreme tactile sensitivities and an inability to adapt to the sensors after three habituation sessions.

2.2 Apparatus and Stimuli

We used two stimuli: a pattern-reversal black and white checkerboard (See figure 1), and an expanding and contracting black and white dartboard. (See figure 2), run on a Dell desktop computer. The checkerboard was presented at 4 different contrasts at each of two different check sizes (as per ISCEV standards, at 1 degree +/- 20 units per side and 0.25 degrees +/-20 units per side) with contrasts determined at 100%, 10%, 5% and 2.5%, as determined by a contrast sensitivity function during preliminary trials. Each reversal lasted 500 milliseconds and the viewing distance from the checkerboard was 56 centimeters.

Acquisition lengths for the checkerboard were 60 seconds, with participants staying as still as possible and fixating on a red cross in the center of each stimulus.

Acquisition length for the dartboards was 85 seconds with a viewing distance of 42 centimeters (Bach, 2007).

2.3 Procedure

Waveforms were recorded via electrodes plugged into a BIOPAC MP150 WSW-G Data Acquisition System GLP and input to a 21.5-inch Core i5/2.7GHz Apple IMAC computer using Aqknowledge software. Data were transferred into MATLAB 7.12.0 and EEGLAB v11.0.4.3b was used for further analysis. Artifact rejection was used implementing voltage thresholds between 0.4 and 1 Hz. We calculated individual average waveforms and then created grand averages for waves for each stimulus in each group (NT boys, ASD boys and NT females), and examined the P1 and the N1 peaks, with windows at 85-135 ms for P1 and 100-205 ms for N1 (Gruber et al, 2005; Luck, 2005). The windows were chosen based on the possibility that participants with ASD may require a larger window for possible delays or premature peaks.

We used a multivariate analysis of variance to examine the nature of each wave and any interactions between contrast, check size and diagnosis and measurements were taken for P1 and N1 peak amplitude, peak latency, fractional area latency and area amplitude. While peak amplitude and peak latency are generally common measures taken in EEG studies, fractional area latency and area amplitude are less common, but often show more robust findings otherwise missed by the first two measures. Fractional area latency provides a measure of latency by dividing the area under the curve into

specific fractions. Generally, one would find the midpoint of a component by finding the point that divides the area under the curve into two equal regions, which is a 50% area latency measure, and works well on large components or those that have been isolated by means of a difference wave (Luck, 2005).

Area amplitude is a calculation of the sum of the voltages at each time point within the measurement window, or the mean amplitude multiplied by the number of points in the measurement window. The goal of these techniques is to provide an accurate measurement of the size of the underlying ERP component with minimal distortion from noise and other overlapping components, again, providing more robust results that one could not see simply by measuring peak amplitude or peak latency alone (Luck, 2005).

3. Results

The multivariate analysis of variance revealed significant effects for three way interactions for the checkerboard stimuli between contrast, check size and diagnosis for ASD versus neurotypical (NT) male groups for P1 peak amplitude $F(3, 51)=3.906$, $p=0.014$ (see Figures 13 and 14), P1 area amplitude $F(3, 51)=5.735$, $p=0.002$ (see figures 15 and 16) and N1 fractional area latency $F(3, 18)=14.578$, $p=0.000$ (see Figures 17 and

18). This implies that there are differences in the way individuals with ASD respond to particular motion stimuli in their environment based on particular features of those stimuli, including size and salience.

For ASD versus NT males versus NT females, significant interactions were found between contrast, check size and diagnosis for N1 fractional area latency $F(6, 15)=7.403$, $p=0.001$ (see Figures 19 and 20) and P1 area amplitude $F(6, 48)=2.8$, $p=0.020$ (see Figures 21 and 22). Again, size and salience were affected by diagnosis, with individuals with ASD responding more dramatically to smaller check sizes and showing almost no change in response across contrasts for the larger check sizes (see Tables 13-16).

Main effects were found between ASD and NT males for N1 peak amplitude contrast $F(3, 51)=4.446$, $p=0.009$, N1 fractional area latency contrast $F(3, 18)=3.177$, $p=0.049$, size/contrast $F(3, 18)=15.184$, $p=0.001$ and N1 area amplitude contrast $F(3, 51)=4.048$, $p=0.012$ (see Tables 9-12).

For P1 in ASD and NT males, several main effects were found, including P1 peak amplitude check size $F(1, 17)=11.154$, $p=0.004$ and P1 contrast $F(3, 51)=3.906$, $p=0.014$. For P1 peak latency, main effects were found for check size $F(1, 17)=12.716$, $p=0.002$, and contrast $F(3, 51)=7.545$, $p=0.001$. For P1 fractional area latency main effects were found for check size $F(1, 6)=22.067$, $p=0.003$, contrast $F(3, 18)=20.86$, $p=0.001$ and size/contrast $F(3, 18)=3.856$, $p=0.027$. Area amplitude for P1 for this group showed main

effects of size F(1, 17)=17.061, p=0.001 and contrast F(3, 51)=10.426, p=0.000 (see Tables 1-5).

Main effects were also found for ASD versus NT males versus NT females for N1 peak amplitude contrast F(3, 48)=5.9, p=0.002, N1 fractional area latency size/contrast f(3, 15)=9.389, p=0.001 and N1 area amplitude contrast F(3, 48)=5.665, p=0.002.

For ASD versus NT males versus NT females, several main effects were also found for P1 (see Tables 5-8). For peak amplitude, main effects were found for check size F(1, 16)=14.335, p=0.002 and contrast F(3, 48)=26.354, p=0.001. For P1 peak latency, effects were seen for size F(1, 16)=7.803, p=0.013 and contrast F(3, 48)=8.728, p=0.000. For P1 fractional area latency, effects were found for check size F(1, 16)=23.425, p=0.005 and contrast F(3, 48)=19.907, p=0.001. Finally, main effects were also found for area amplitude for size F(1, 16)=17.821, p=0.001 and contrast F(3, 48)=11.73, p=0.001.

For the dartboard, no significant differences were found between ASD and both neurotypical groups (see Tables 17-20).

Results indicate that there are, indeed, differences in magnocellular processing in ASD. Particularly, there are some interactions between check size, checkerboard contrast and diagnosis, typically for area amplitude when comparing ASD males to NT males, and when comparing ASD males to NT males and also to NT females, with several main effects of size, contrast and size/contrast for N1 and P1.

Beginning with Figures 13 and 14, Figure 13 shows significant differences between ASD and NT male groups at the larger check size at the four different contrasts. It appears that individuals with ASD do not show as large an initial response to motion onset in terms of P1 peak amplitude, and additionally they appear to show a more gradual decrease in response with the smaller contrasts.

Figure 15 shows, however, that P1 area amplitude in ASD does not seem to differ much between the different contrasts for the larger check size, which is in keeping with the smaller responses to larger check size in P1 peak amplitude. According to the figure, NT males showed a larger response to 100% contrast and a significantly smaller response (more dramatic decrease) to the smaller contrasts.

For P1 area amplitude at the smaller check size (see Figure 16), again, a similar result occurs as did in P1 peak amplitude, in that the males with ASD show a larger response at 100% contrast. This is also consistent at the smaller check size, which larger responses in ASD at initial onset.

Concerning the N1 wave at the larger check size for fractional area latency (see Figure 17), the results appeared to be atypical, which could have been caused by noise or other distortional components. For the smaller check size (see Figure 18), responses between the two groups were similar, showing a slight increase in response to smaller contrasts for the NT group and a slight u-curve for the ASD group, implying that their fractional area latency responses were quite steady from one contrast to the other. This

evidence was not robust enough to draw any strong conclusions and significance could have been the result, again, of noise or other distortional components.

For comparisons between the three groups, N1 fractional area latency at the larger check size (see Figure 19) showed a larger response and steady (no real increase or decrease from 100% contrast to 2.5% contrast in NT males, and a slightly smaller response but also steady for the NT females. The ASD group showed similar results as with fractional area latency (as it was the same waveform) as the previous figures, and thus may also be attributed to data distortion. Because of this, fractional area latency may not actually be significant in this study. For the smaller check size (see Figure 20), as with the NT males, NT females showed almost identical results, implying no real differences in fractional area latency. This may be due to the fact that, as Luck (2005) claims, fractional area latency is a better measure for much larger waves, such as the P3 wave.

However, again, some very different results occurred for P1 area amplitude (see Figure 21) when compared across all three groups. The NT females had the largest response, decreasing dramatically as contrast decreased. For the smaller check size (see Figure 22), NT females tended to trend more closely with NT males, showing a smaller response than individuals with ASD and a less dramatic decreasing slope.

The results imply that individuals with ASD respond more drastically than neurotypicals to smaller, more local stimuli, and these responses decrease with decreasing salience. Individuals with ASD tend to show no changes in their perception of larger, more global stimuli regardless of salience.

4. Discussion

In summary, results indicate that individuals with ASD tend to respond more dramatically to smaller, more local stimuli in their environment and don't respond as dramatically to larger, more global features. Particularly, with changes in salience in larger, more global features, there is no change in perceptual response in individuals with ASD, whereas with the smaller, more local features, their perception of local features changes drastically with changes in salience.

Results regarding peak amplitude could imply that individuals with ASD don't tend to perceive differences in contrast as acutely as age-matched male NT controls and don't respond as powerfully to initial motion onset as controls. This could explain some of their symptomatology, including fixation with an inability to pull focus to other moving distractions. However, at the smaller check size (see Figure 14), individuals with ASD showed a larger response to motion onset and a more dramatic decrease in response than NT males. These results imply that there may be more complex brain activity underlying the visual perceptual process in ASD. The several main effects of size and contrast suggest that the type, size and contrast of moving stimuli in the environment are critical in terms of understanding exactly what types of stimuli in the visual world individuals with ASD do and do not perceive as robustly as NT individuals.

Results regarding area amplitude imply that generally, it is difficult for individuals with ASD to perceive salience differences in their environment with larger, more global stimuli, which could also explain their sometimes inability to change focus or pay

attention to changes in the environment with larger objects. The similar results with the smaller check size could explain underlying seizure disorders in ASD because individuals have larger responses to very robust stimuli and a more dramatic decrease, ending with a smaller reaction to the lower contrasts than NT males, who start off with a smaller response to 100% contrast. It appears that the smaller check size and different contrasts are interacting quite powerfully together, which begin to explain how individuals with ASD perceive their environment. Smaller items at larger contrasts evoke a larger response. Seizure disorders are comorbid with ASD (Milne et al., 2007) around 20-35% of the time. It may be useful to see if small item/large contrast items are more likely to contribute to seizure activity than other check sizes and contrasts in the future, which can provide more information about comorbid seizure disorders in ASD, why they happen (is it related to magnocellular processing?) and how to treat or prevent them.

In regards to the female results for P1 area amplitude, the larger response in females could be explained by the fact that the female brain tends to develop faster than the male brain (Kretschmann et al., 1979). We noticed that in individual waveforms, females across the age span all showed much cleaner waveforms and a generally higher P1 peak amplitude than NT males. For the smaller check size, again, implications across the results tend to show that individuals with ASD show a larger and more dramatic change in the response from the higher to lower contrasts at smaller check sizes and a smaller and steadier response across contrasts for the larger check size, which has important implications for their processing of specific visual information in their environment. It appears that more in-depth studies of visual acuity and

attention to size, contrast and detail using perceptual stimuli representative of the real environment could be the next step at determining exactly how these results apply to the processing of visual information based on size and contrast in the real world.

The magnocellular system is believed to be the first pathway for communication of visual information into the brain. Should there be deficits in this pathway that lead to dysfunctional processing of visual motion information, a domino-like effect might take place that effectively alters the way information is taken in and processed by subsequent pathways. By the time information arrives into higher-processing areas, it is inaccurate and can lead to problematic behavior and symptomatology as seen in many autistic individuals.

The implications of this information can be applied to early diagnosis and treatment. Magnocellular deficits have been shown to exist in early infancy in exams that were done with high-risk infants (those who have incidents of ASD running in the family). Deficits were seen in several high-risk infants compared to controls (McCleery et al., 2007). Experimental success will inspire the development of tests to detect these processing differences early. An early diagnosis can lead to earlier treatment interventions. It has been shown time and time again that early intervention is one of the most successful methods of treatment for individuals with autism, when combined with balanced educational, social, and medical programs. Gepner (2002) found that, when slowed down, video of facial expressions could be processed for emotional meaning by autistics. He implied that the quick motion of facial expressions might be difficult for individuals with ASD to comprehend. Using subsequently faster and faster

showings of the video may be a method of training that could help to eradicate the social deficits accompanying magnocellular deficiencies.

As the incidence of ASD diagnosis continues to rise, more and more parents are becoming concerned about the risks for their own children, especially considering that genetics is not the only factor that leads to an ASD diagnosis. The brain is highly adaptable, especially during childhood. Because ASD is not purely genetic, it may be exacerbated by abnormal sensory input from the environment. It is critical to understand how that sensory input is being processed. The improvement in quality of life that comes with treatment affects not only the individual, but family members of the individual as well, and any of them can attest to the time, cost and emotional difficulty of raising a child with autism. Possible outcomes can be innovative therapies that focus on breaking the cycle of impaired perception and learning to change the developmental cycle in ASD.

The next step is to use EEG to explore physiological correlates of perception of other types of motion, such as the coherent motion studied using the Random Dot Kinematogram by Milne and colleagues (2002), chromatic moving stimuli, biological motion, and even more pertinently, facial movements and motion as a result of change. Future research may want to move to the cellular level by examining eye and retinal tissue, including magnocellular neurons, to continue to delve into the more intricate layers that make up the visual processing system at a variety of levels.

The current study had several methodological advantages. Using Bach's (2005) dartboard was an innovative use of achromatic motion stimuli that added more

dimension to previous research looking at basic pattern-reversal motion perception in ASD. Most studies have looked at motion-onset, steady-state and pattern-reversal VEPs, but the expanding and contracting nature of the dartboard explores responses to motion of a different nature, as not all motion in the environment is of a pattern-reversal nature. Also, previous studies looking at VEPs in ASD have used inappropriate populations and generalized results to ASD (Sutherland and Crewther, 2010; McCleery et al, 2007). This includes populations of infants at risk for ASD and adults who tested on an autism quotient scale. Neither of the populations has confirmed diagnoses of ASD, and infants have an underdeveloped magnocellular system that does not respond typically to VEPs in the way that teenagers and adults do who tend to have a more highly-developed magnocellular system (Hickey, 1977).

Future improvements involve mostly recruitment, as there was a high level of difficulty in obtaining a large participant group, especially for neurotypical males and for females with ASD. A larger sample size for these two groups in particular could provide more robust results in the future. Lastly, the neurotypical male population of the current study showed atypical responses to the motion stimuli in their individual waveforms, which could have been for a number of reasons. The data collected may have simply been atypical, making it difficult to generalize this data to the rest of the population, or there may have been issues with equipment and acquisition of data during the trials. Some of the younger children, especially the ten-year-olds, had more difficulty sitting still and staying focused on the trials, whereas the older children tended to perform better sitting still for longer periods of time. These discrepancies can cause

skews in the data based on excess movement and noise. Additionally, there can be drastic changes in EEG response as it relates to age, in that these physiological responses change with neural development (Milne et al., 2005) It may be beneficial to focus on a slightly older population to eliminate this issue.

In the future, using the RDK from Milne and looking into EEG with other stimuli that have been used in motion research that are more advanced than basic motion-onset and steady-state VEPs can only add more depth to VEP research by looking at the fundamental level (basic brain activity) of items like coherent and biological motion in ASD, to compare to behavioral studies that have been done in the past.

It is always exceptional to be able to perform ASD studies with low-functioning individuals, but the time and effort required to habituate low-functioning individuals to wearing electrodes, sitting still and concentrating on stimuli for such a long period of time has been an underlying reason that little to no research in VEP acquisition has focused on low-functioning individuals. This is a key next step to acquiring more information on the range of the effects of such studies over the entire spectrum of ASD, instead of just the high-functioning individuals. However, Kaiser and Shiffrer (2009) have pointed out other difficulties involved in working with lower-functioning individuals with ASD, including not only their difficulty in completing tasks, but also discrepancies in IQ and the presence of comorbid disorders that complicate the implementation and interpretation of psychophysical performance.

Finally, while there were some issues with external noise in the data that may have altered our results, it is important to note that this study implemented several

new techniques, including using contrast sensitivity information of motion stimuli to test differences in contrast perception, and some patterns showed that individuals with ASD do not respond differently to different contrasts, which has huge implications to their perception of the visual world. While the contrast information was new and of note, the authors also implemented standardized, well-studied experimental procedures to assess low-level functioning in ASD, adding to the strength of the study.

Of the handful of electrophysiological studies done on populations similar to ASD, most have focused on basic electrophysiological measures such as peak amplitude and peak latency. This study incorporated other components of the EEG waveform, including fractional area latency and area amplitude. These measures can provide more information about a waveform that are missed when comparing results from only peak amplitude and peak latency. With only those two measures, information about the wave can be missed and results can seem to be insignificant when in fact, there are results that cannot be measured by amplitude and latency alone.

Lastly, the project was a collaborative effort across two research groups, the Cognitive and Brain Science (CBS) Department and the Applied Behavior Analysis (ABA) Department at the University of Nevada, Reno. This collaboration allowed the two groups to contribute their expertise: CBS with EEG and ABA with their knowledge of working with individuals with ASD, including the use of habituation techniques and training for participants.

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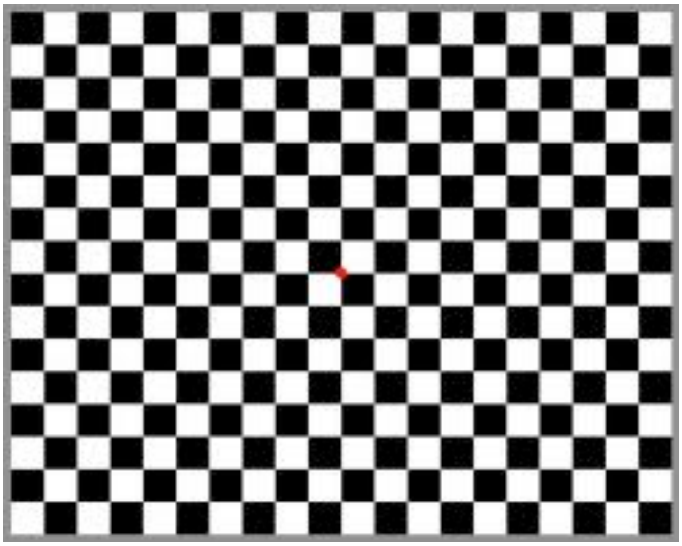
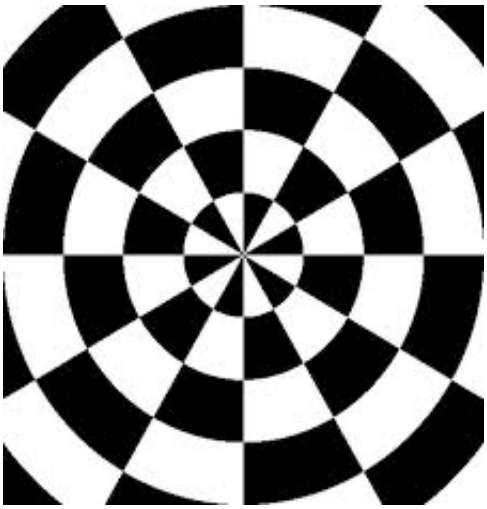
Figures

Figure 1: Pattern-reversal checkerboard stimulus



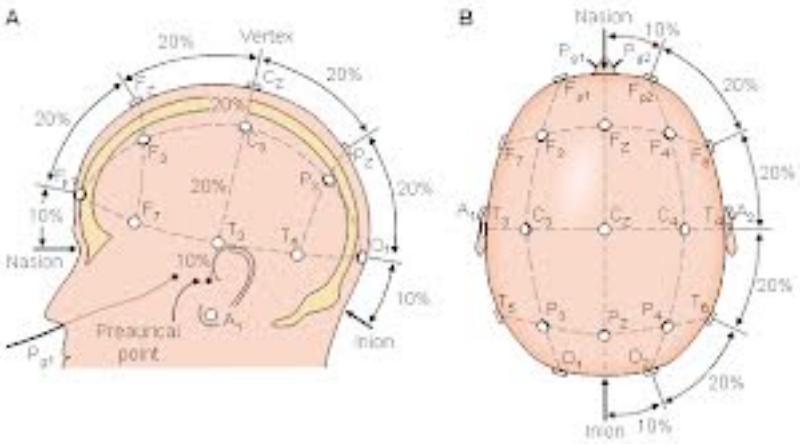
Stimulus 1: Reversing checkerboard pattern at 100% contrast, presented for 60 seconds, with a 500 ms reversal time.

Figure 2: Dartboard stimulus



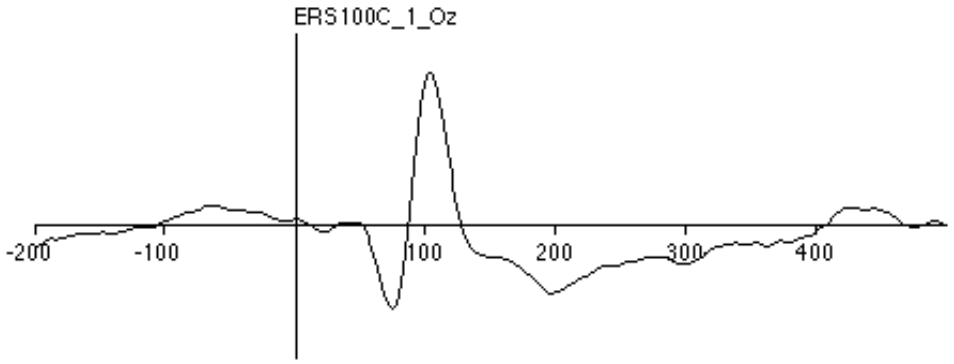
Stimulus 2: Expanding and contracting dartboard, presented for 85 seconds, with and inter-stimulus interval of 500 milliseconds between each expansion and contraction wherein the stimulus did not move.

Figure 3: Scalp placement of electrodes

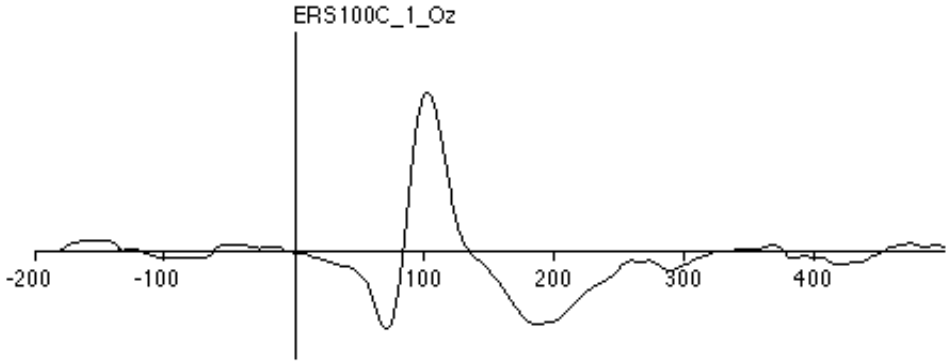


Electrodes were placed on the scalp at Oz, Pz, Cz and Fz, with Fz as ground and Cz as a reference.

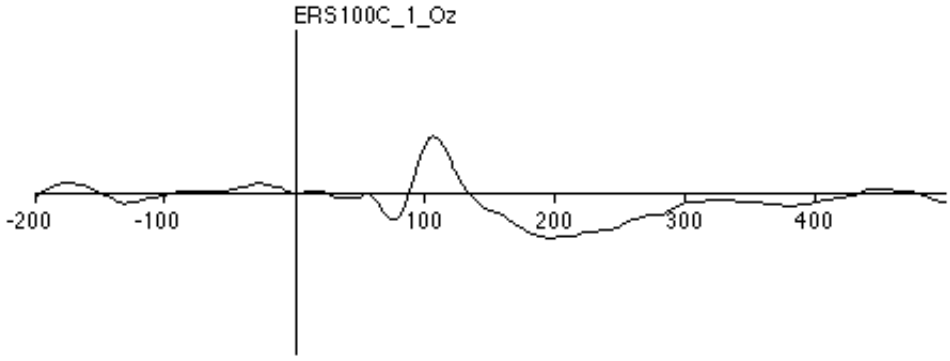
Figure 4: Checkerboard grand average waveforms at 100% contrast for 3 groups at check size 1 degree



4.1 ASD grand average waveform

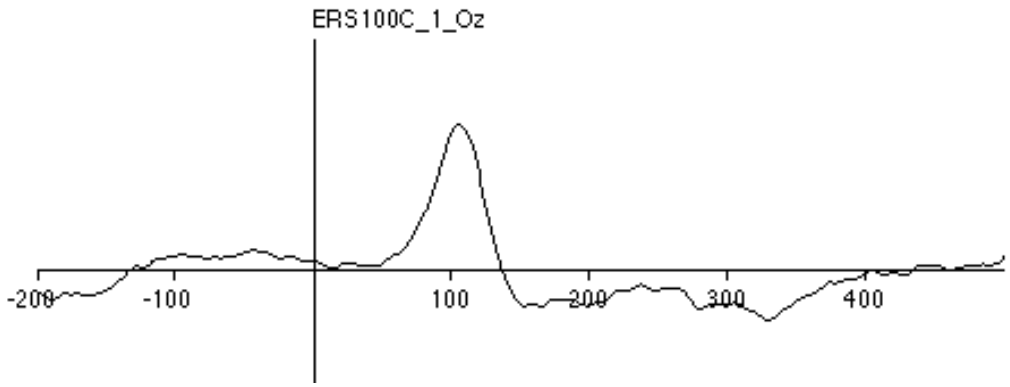


4.2 Neurotypical female grand average waveform

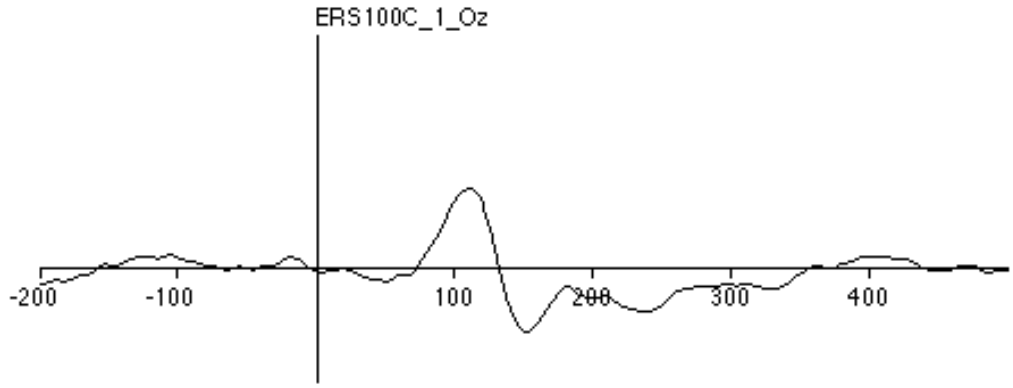


4.3 Neurotypical male grand average waveform

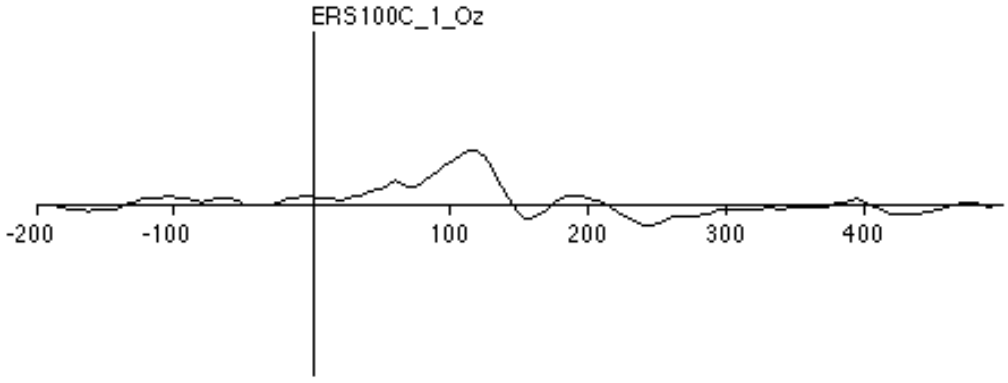
Figure 5: Checkerboard grand average waveforms at 10% contrast for 3 groups at check size 1 degree



5.1 ASD grand average waveform



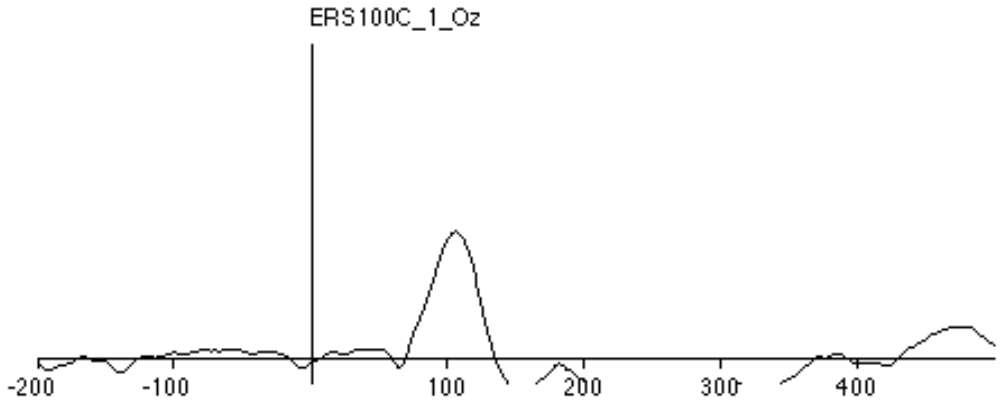
5.2 Neurotypical female grand average waveform



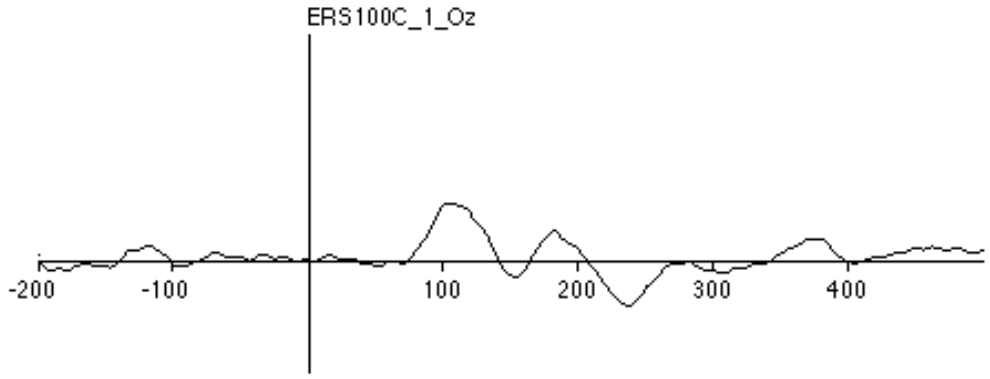
5.3 Neurotypical male grand average waveform

Figure 6: Checkerboard grand average waveforms at 5% contrast for 3 groups at check size 1 degree: P1 is more robust in ASD than in NT

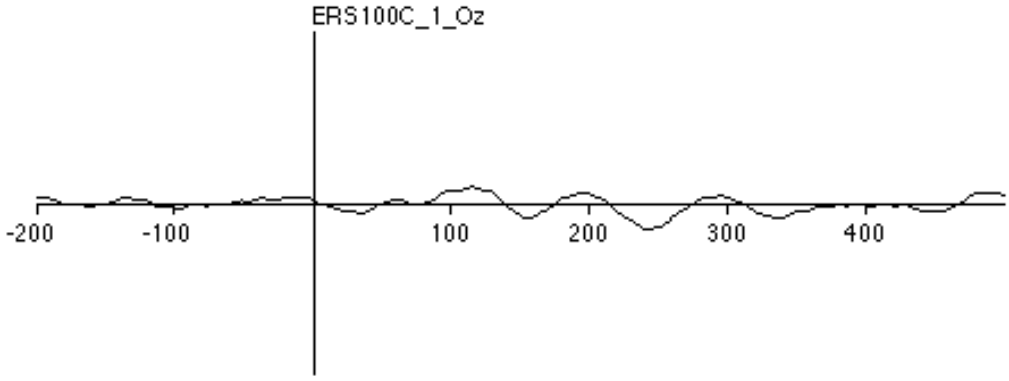
groups.



6.1 ASD grand average waveform:

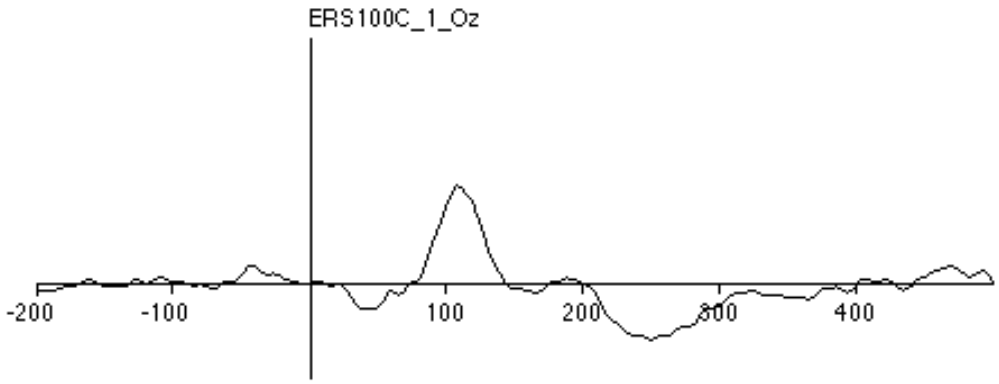


6.2 Neurotypical female grand average waveform

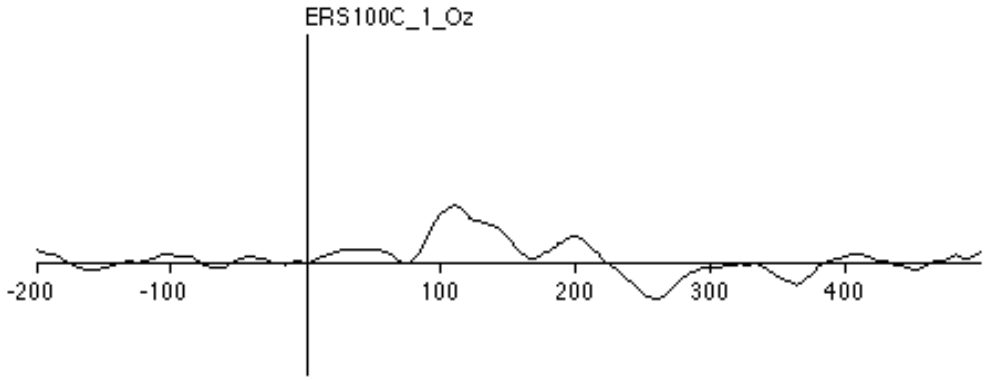


6.3 Neurotypical male grand average wave form

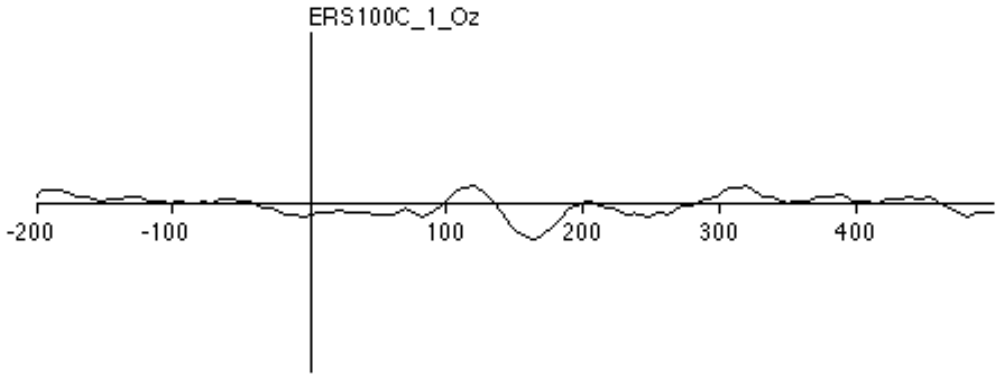
Figure 7: Checkerboard grand average waveforms at 2.5% contrast for 3 groups at check size 1 degree: Again, P1 is more robust in ASD than NT groups.



7.1 ASD grand average waveform

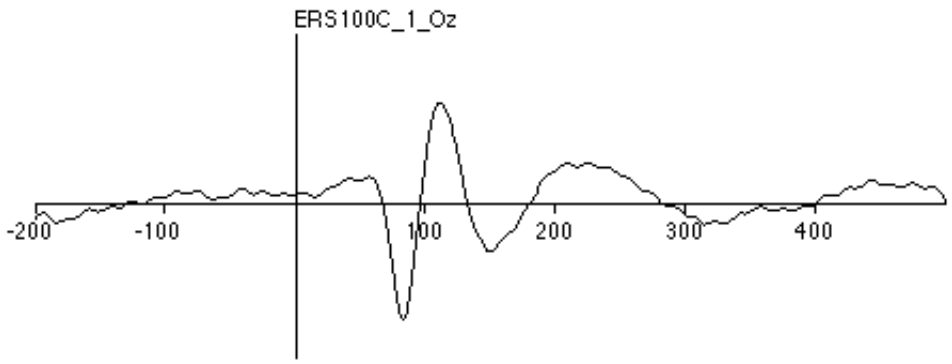


7.2 Neurotypical female grand average waveform

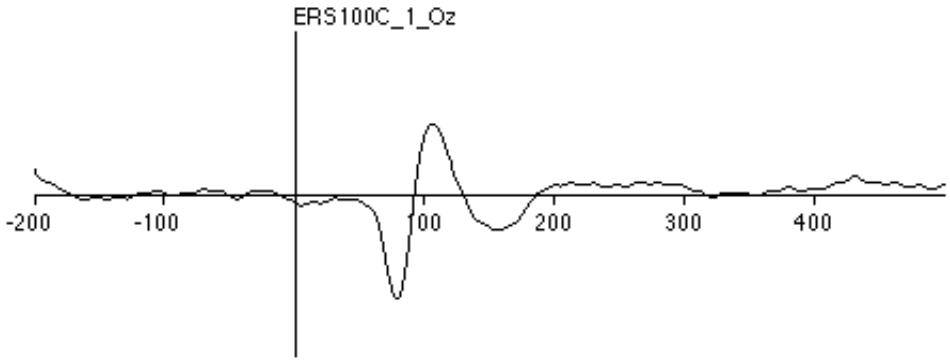


7.3 Neurotypical male grand average waveform

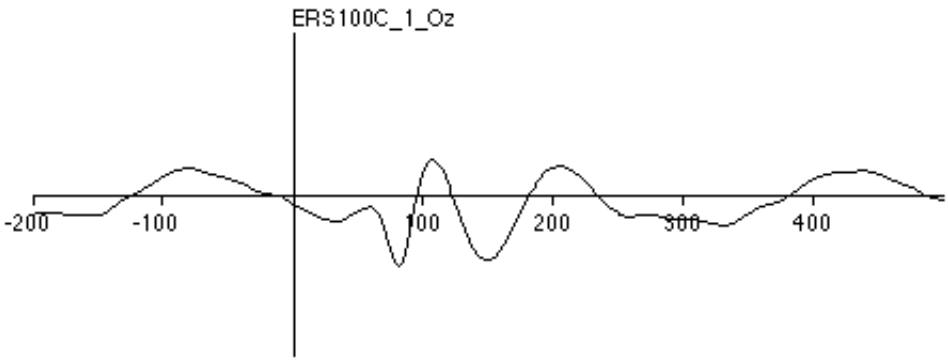
Figure 8: Checkerboard grand average waveforms at 100% contrast for 3 groups at check size 0.25 degrees.



8.1 ASD grand average waveform

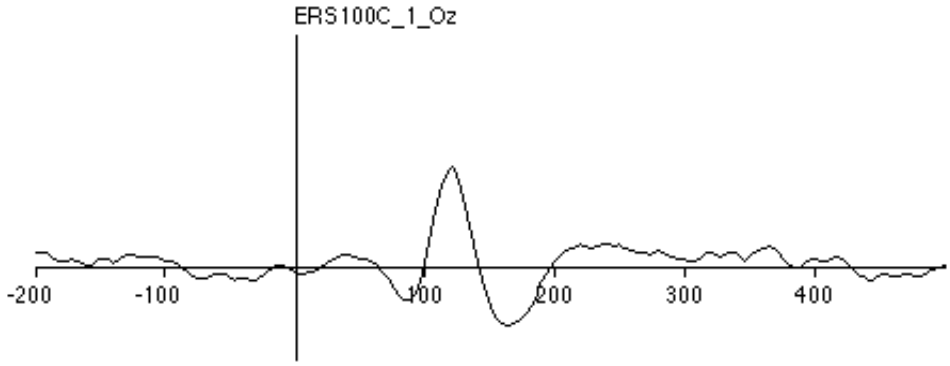


8.2 Neurotypical female grand average waveform

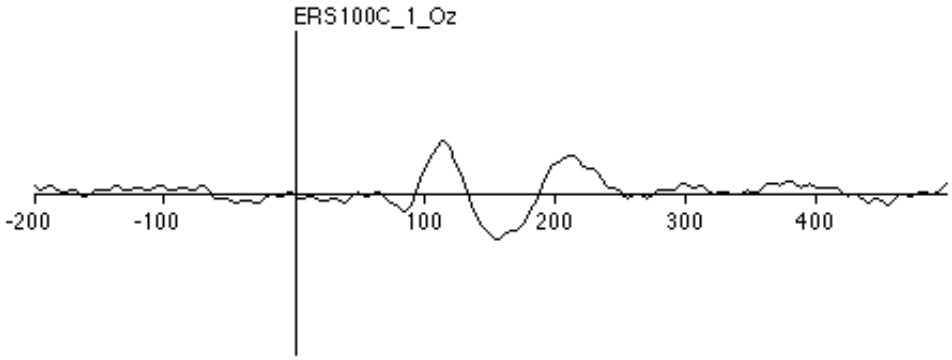


8.3 Neurotypical male grand average waveform

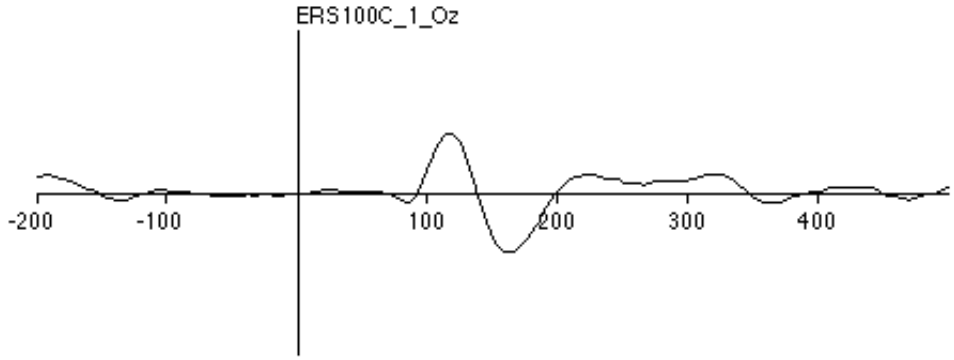
Figure 9: Checkerboard grand average waveforms at 10% contrast for 3 groups at check size 0.25 degrees: slightly larger ASD response than NT males and females.



9.1 ASD grand average waveform

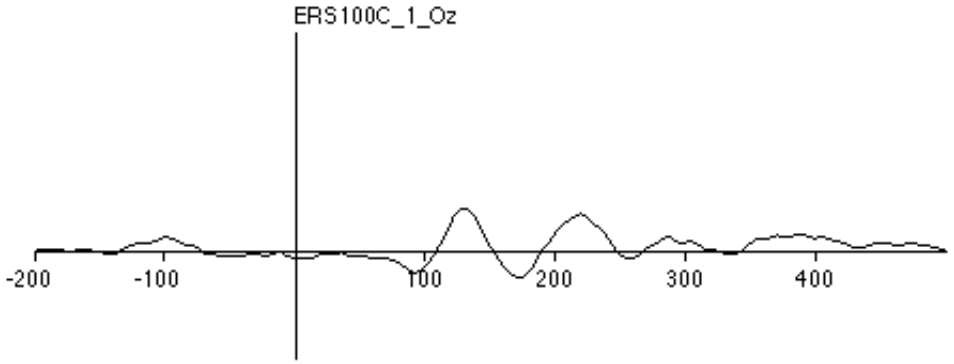


9.2 Neurotypical female grand average waveform

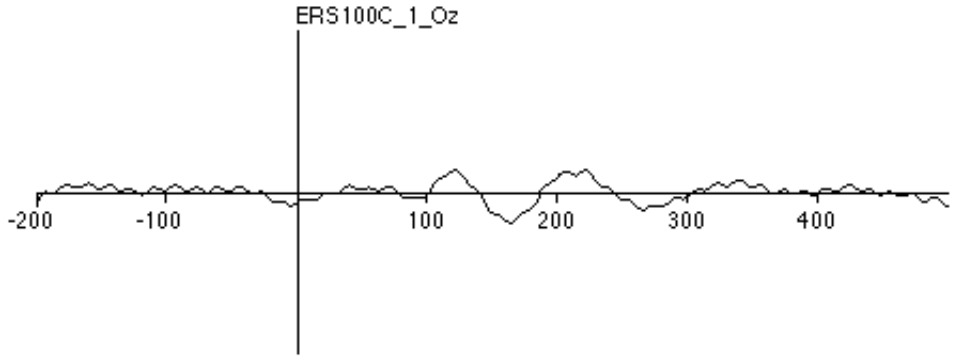


9.3 Neurotypical male grand average waveform

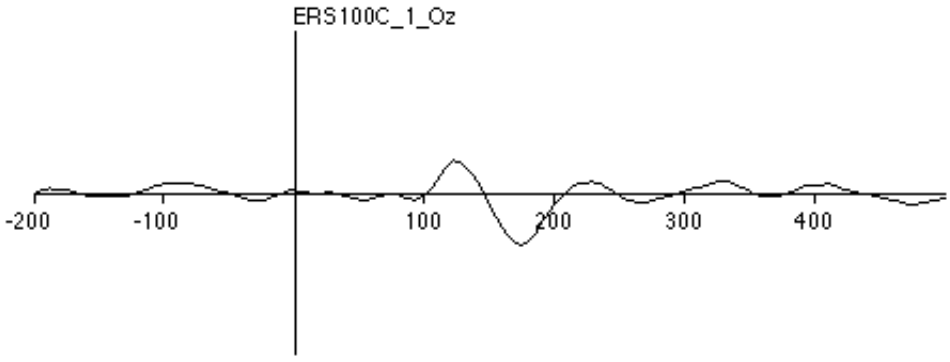
Figure 10: Checkerboard grand average waveforms at 5% contrast for 3 groups at check size 0.25 degrees: responses are similar across groups, with ASD showing a slightly smaller peak amplitude and more noise.



10.1 ASD grand average waveform

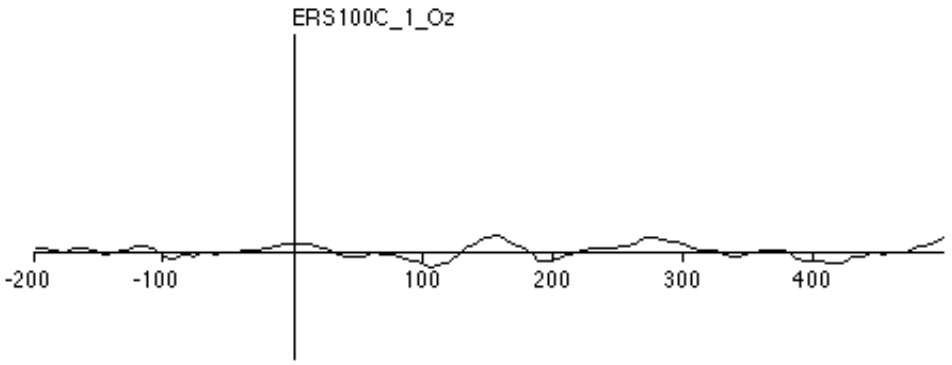


10.2 Neurotypical female grand average waveform

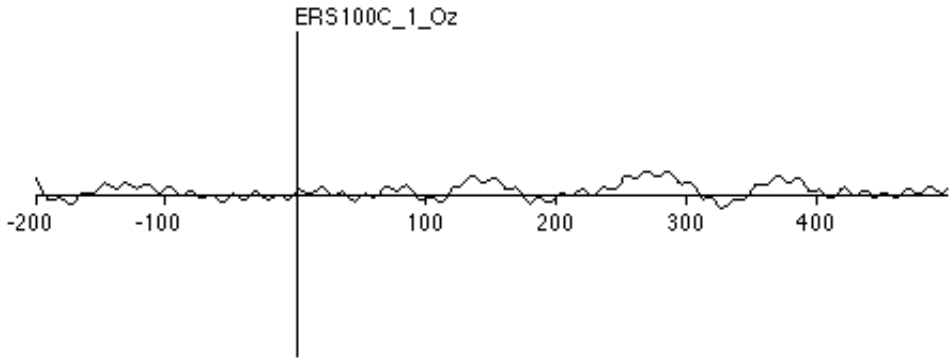


10.3 Neurotypical male grand average waveform

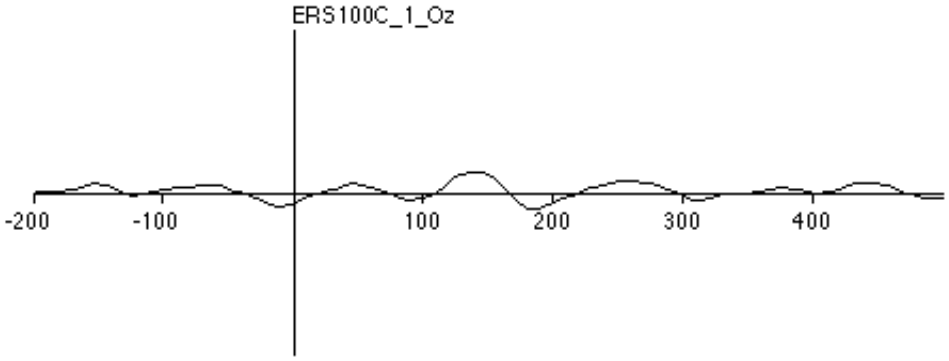
Figure 11: Checkerboard grand average waveforms at 2.5% contrast for 3 groups at check size 0.25 degrees: similar, almost flat responses without significant differences between groups and quite a lot of noise in the female NT group.



11.1 ASD grand average waveform

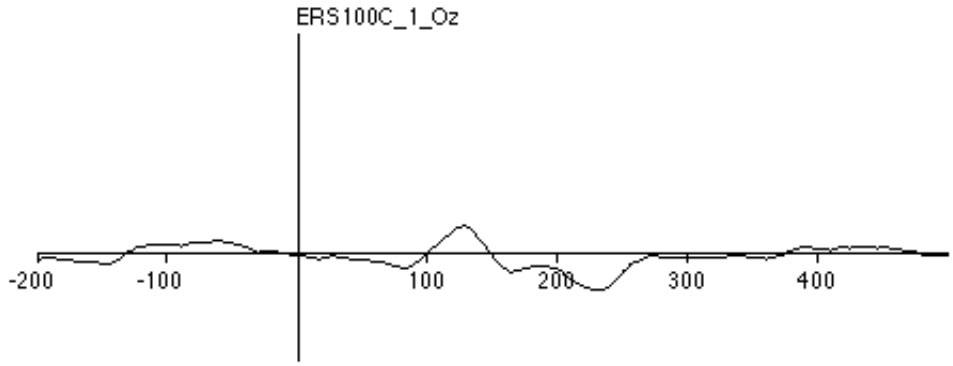


11.2 Neurotypical female grand average waveform

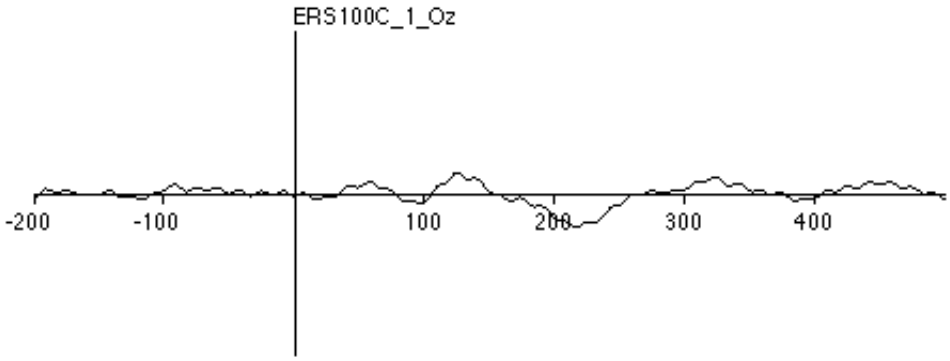


11.3 Neurotypical male grand average waveform

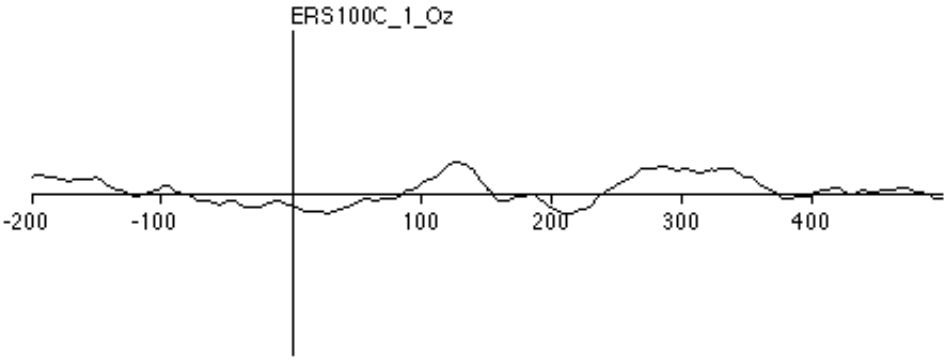
Figure 12: Dartboard grand average waveforms: no significant differences between groups, implying individuals with ASD do not process this type of motion differently.



12.1 ASD grand average waveform



12.2 Neurotypical female grand average waveform



12.3 Neurotypical male grand average waveform

Figure 13: P1 Peak Amplitude ASD versus neurotypical males at check size 1 degree

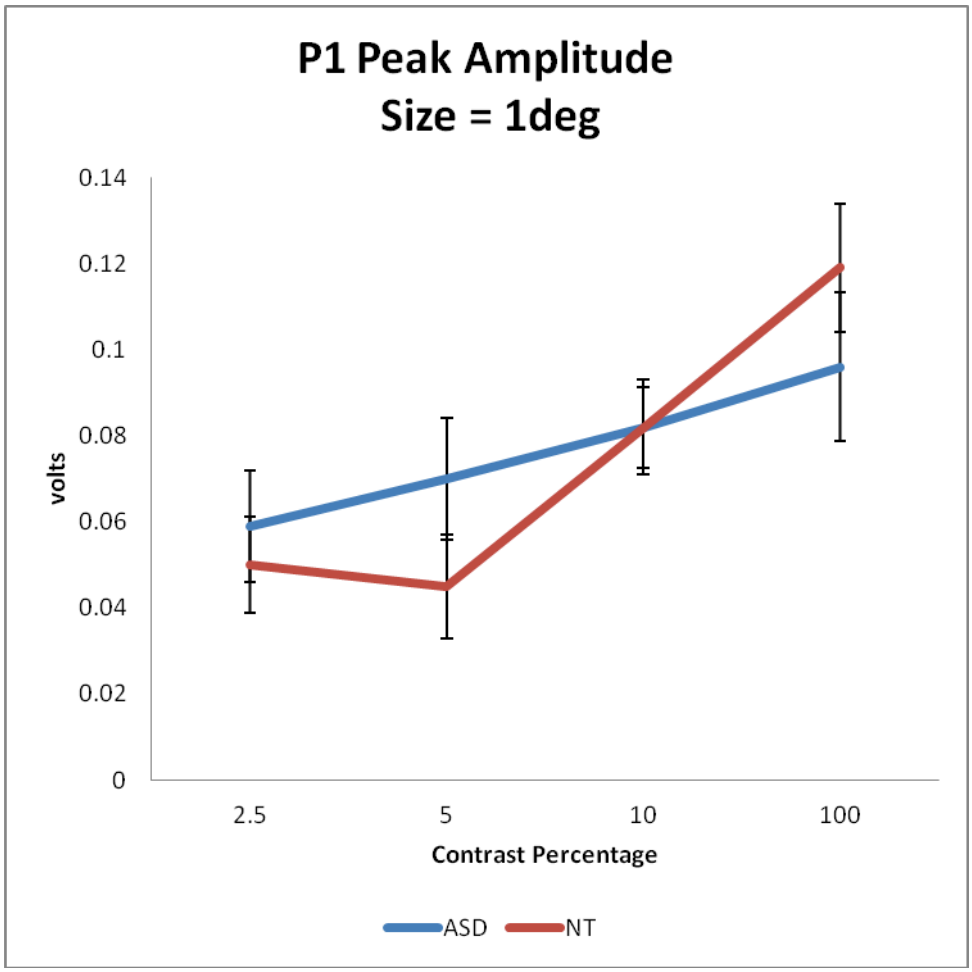


Figure 14: P1 Peak Amplitude ASD versus neurotypical males at check size 0.25 degrees

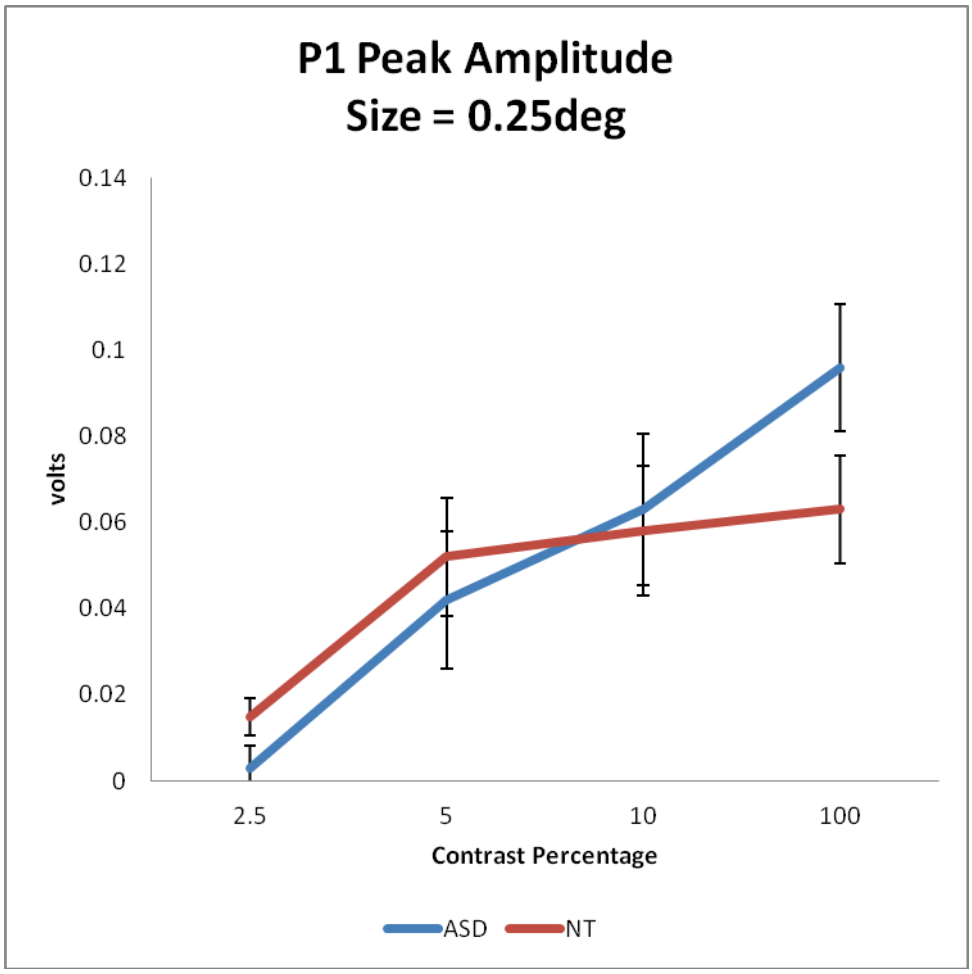


Figure 15: P1 Area Amplitude ASD versus neurotypical males at check size 1 degree

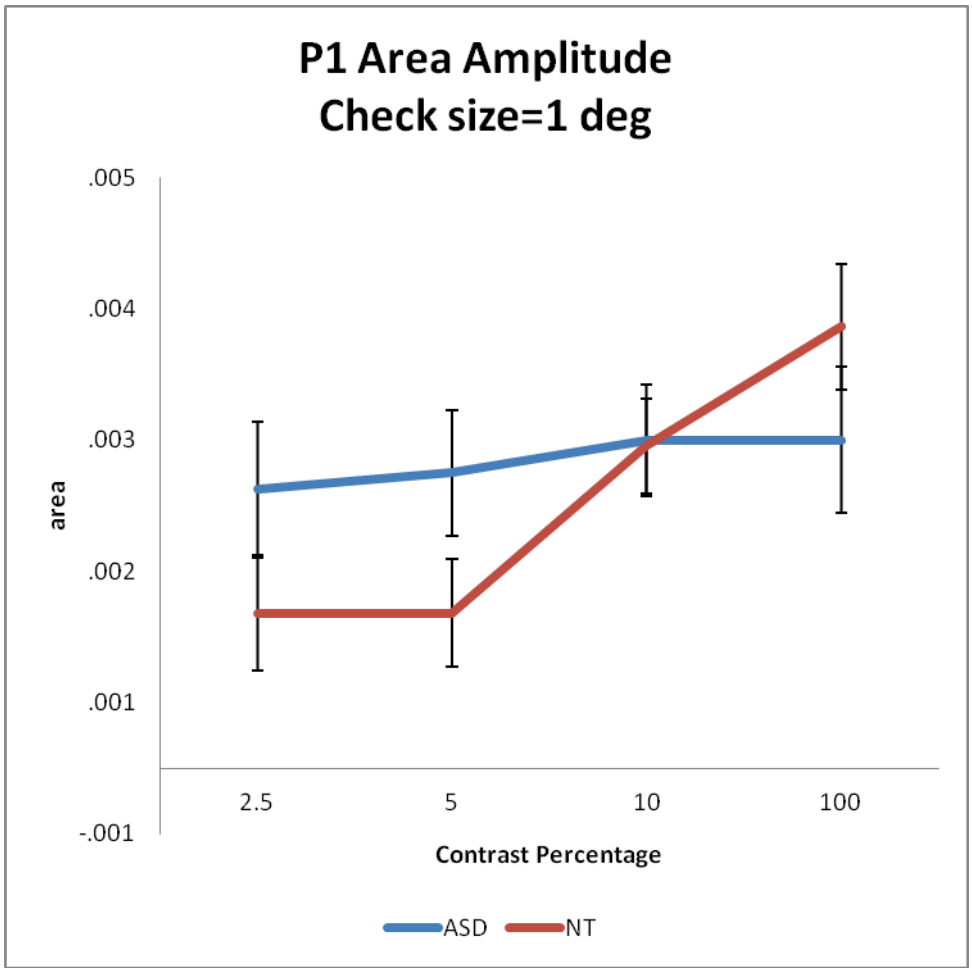


Figure 16: P1 Area Amplitude ASD versus neurotypical males at check size 0.25 degrees

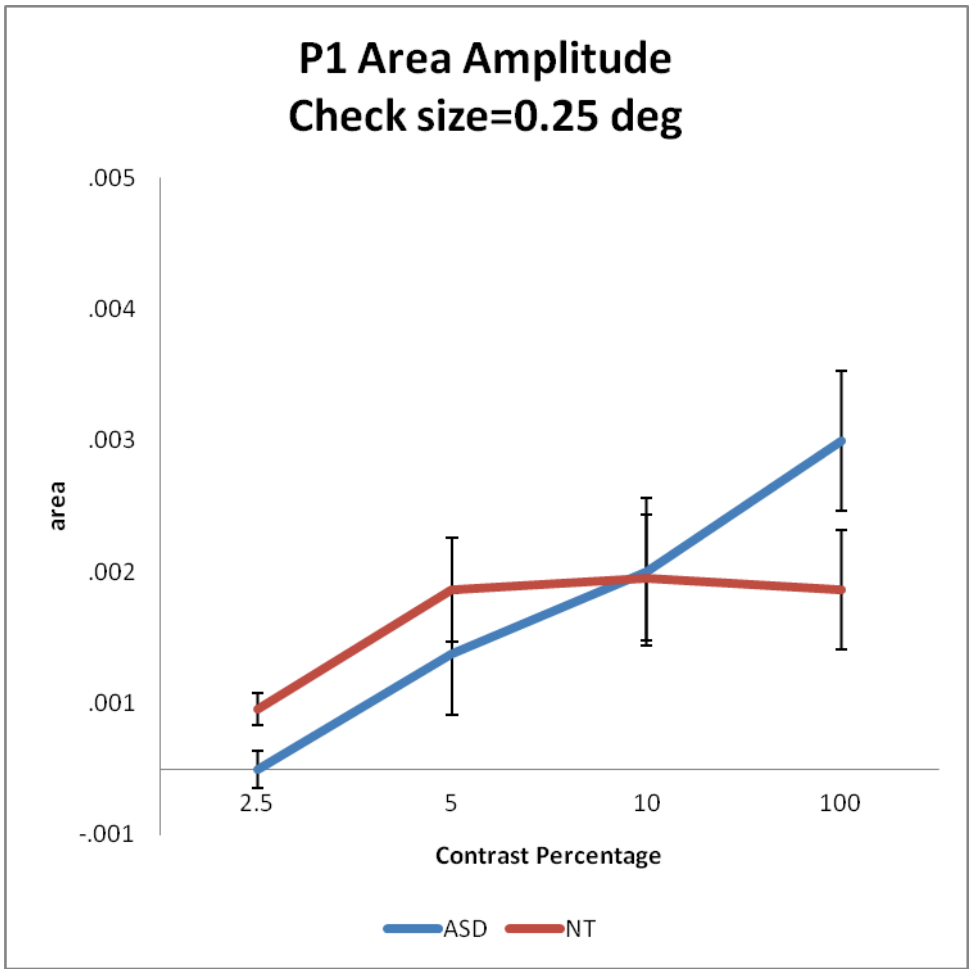


Figure 17: N1 Fractional Area Latency ASD versus neurotypical males at check size 1 degree

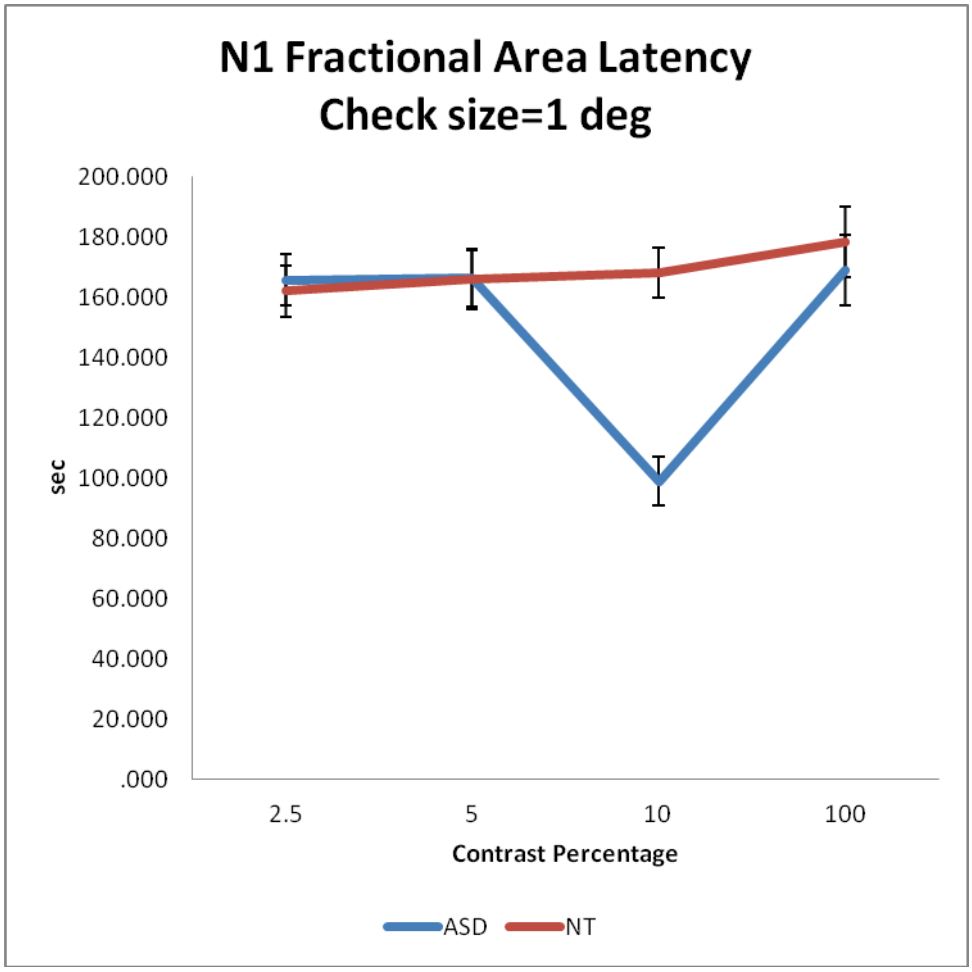


Figure 18: N1 Fractional Area Latency ASD versus neurotypical males at check size 0.25 degrees

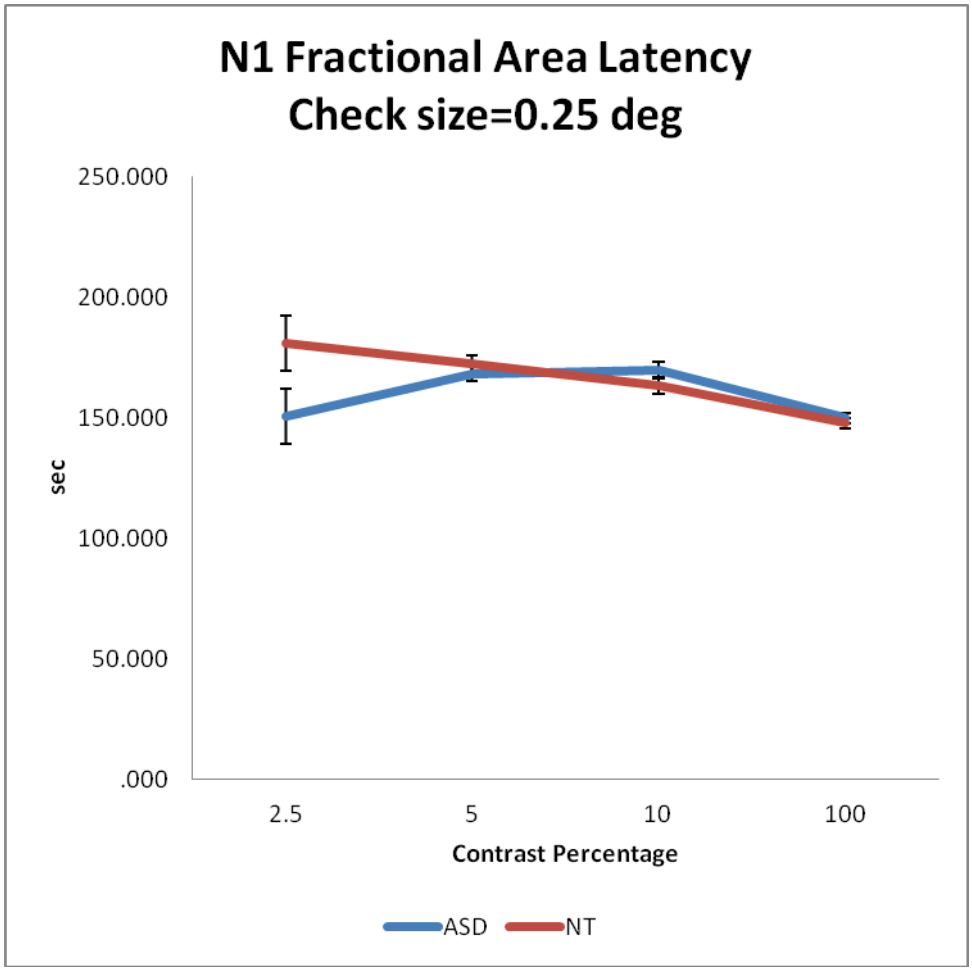


Figure 19: N1 Fractional Area Latency ASD versus neurotypical males versus neurotypical females at check size 1 degree

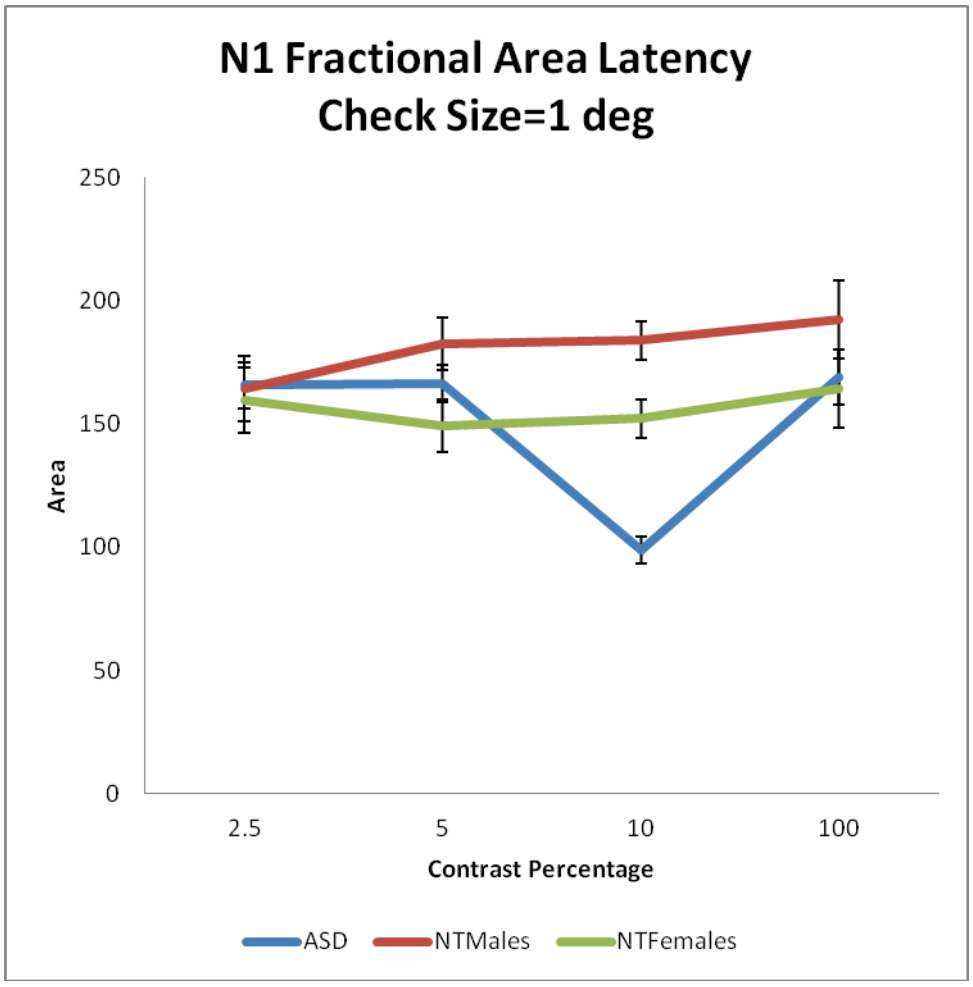


Figure 20: N1 Fractional Area Latency ASD versus neurotypical males versus neurotypical females at check size 0.25 degrees

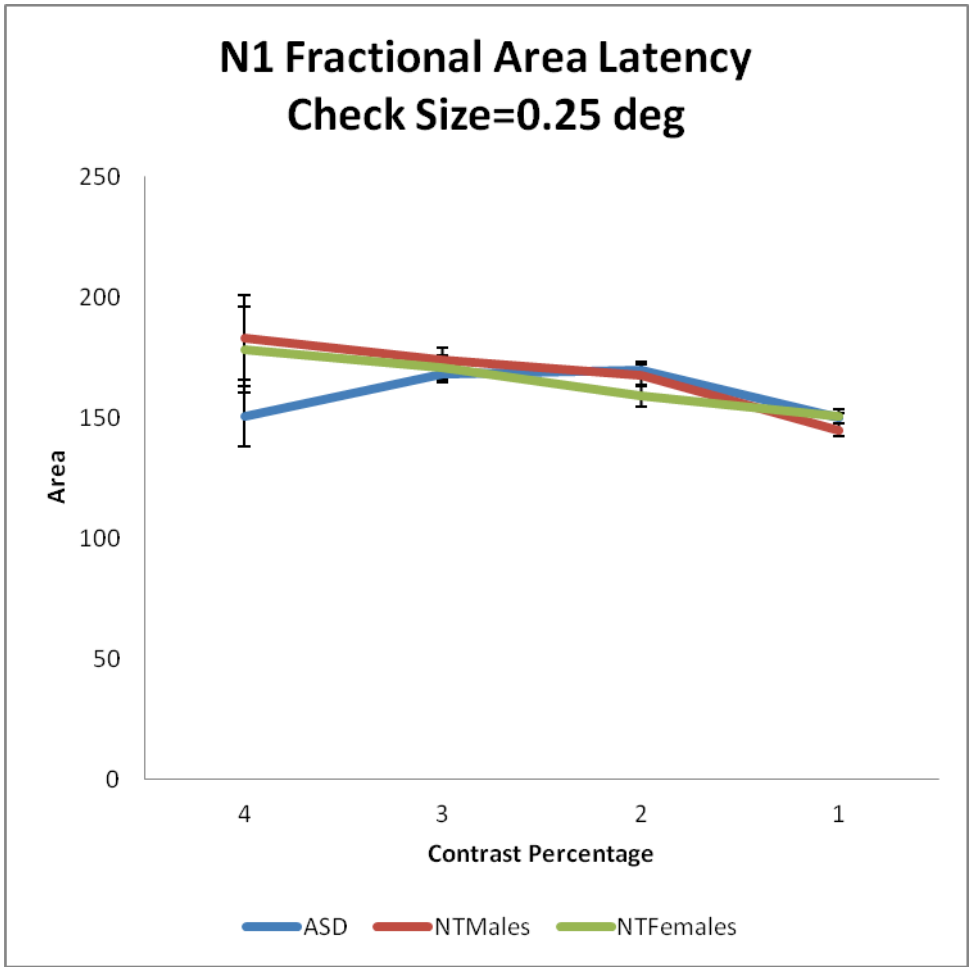


Figure 21: P1 Area Amplitude ASD versus neurotypical males versus neurotypical females at check size 1 degree

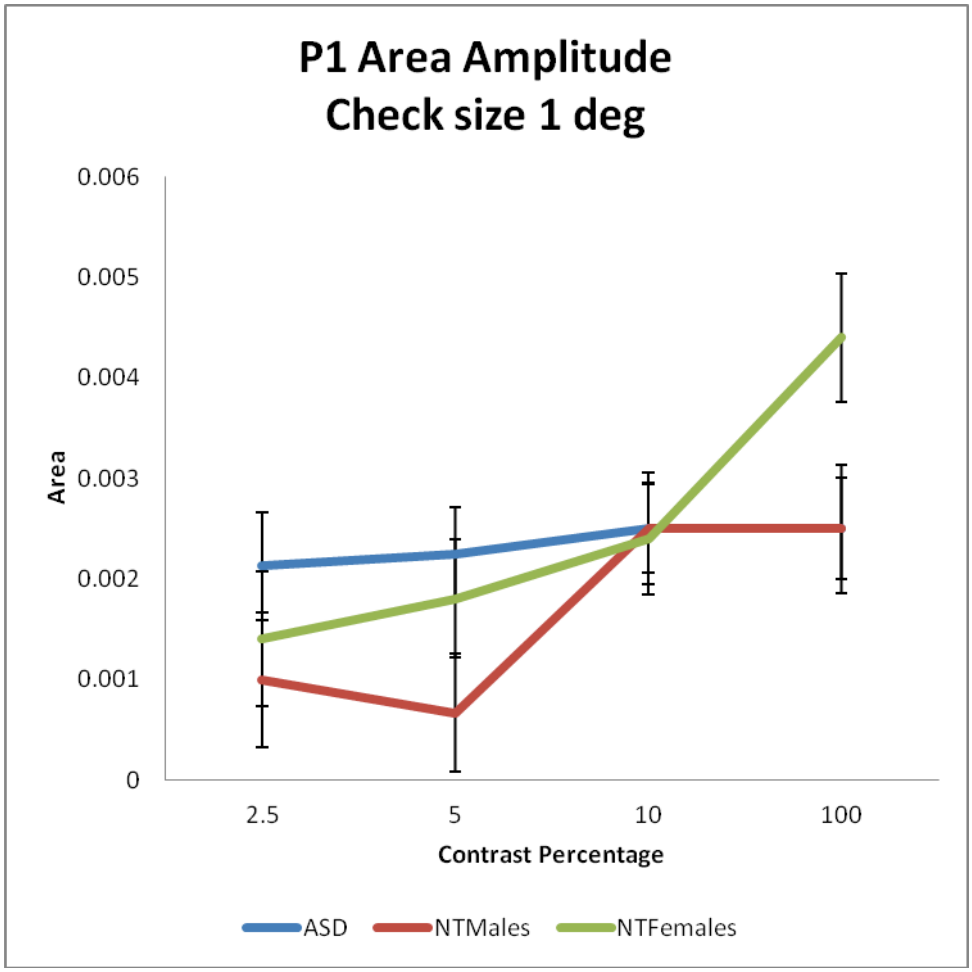


Figure 22: P1 Area Amplitude ASD versus neurotypical males versus neurotypical females at check size 0.25 degrees

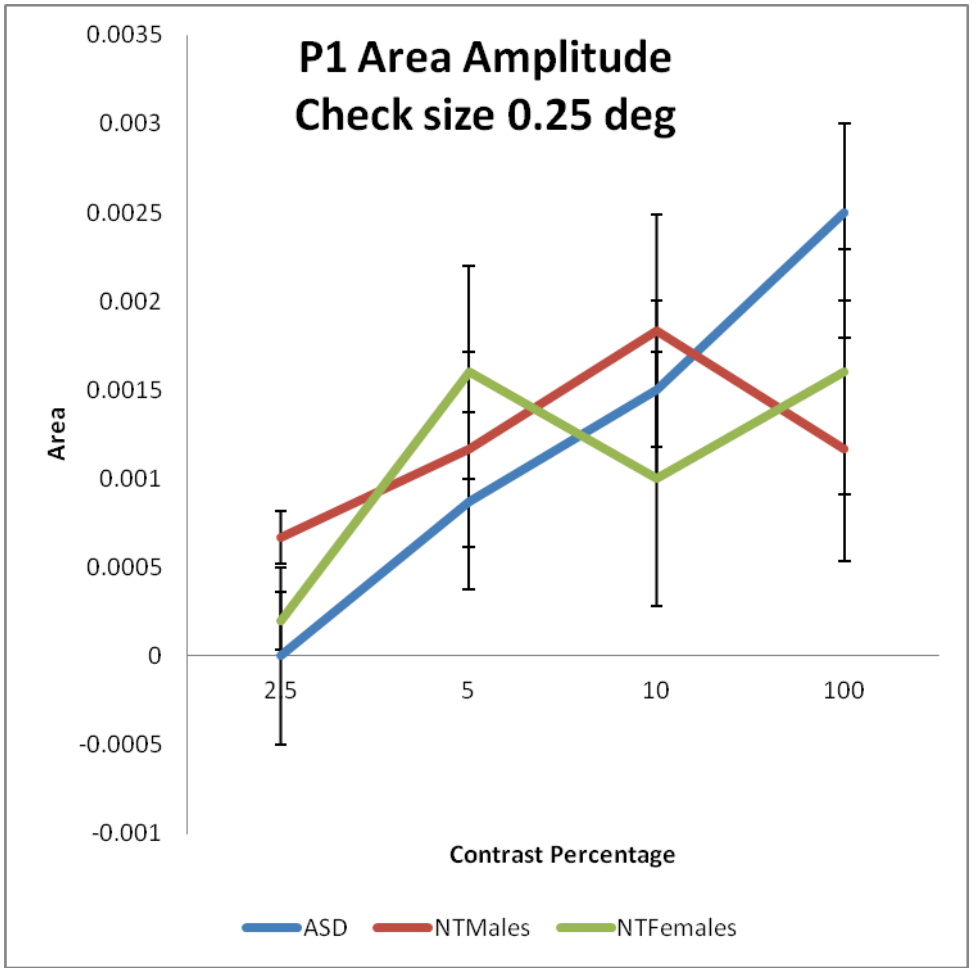


Table 1: P1 Checkerboards Peak Amplitude ASD v. NT

Criteria	**F value**	**df**	**P value**
Size	11.154	1	.004
Contrast	25.451	3	.001
Size x Diagnosis	.007	1	.934
Contrast x Diagnosis	.161	3	.922
Size x Contrast	2.119	3	.109
Size x Contrast x Diagnosis	3.906	3	.014

Table 2: P1 Checkerboards Peak Latency ASD v. NT

Criteria	**F value**	**df**	**P value**
Size	12.716	1	.002
Contrast	7.545	3	.001
Size x Diagnosis	4.272	1	.054
Contrast x Diagnosis	1.094	3	.060
Size x Contrast	.990	3	.405
Size x Contrast x Diagnosis	1.273	3	.293

Table 3: P1 Checkerboards Fractional Area Latency ASD v. NT

Criteria	**F value**	**df**	**P value**
Size	22.067	1	.003
Contrast	20.860	3	.001
Size x Diagnosis	3.067	1	.130
Contrast x Diagnosis	2.213	3	.122
Size x Contrast	3.856	3	.027
Size x Contrast x Diagnosis	1.189	3	.342

Table 4: P1 Checkerboards Area Amplitude ASD v. NT

Criteria	**F value**	**df**	**P value**
Size	17.061	1	.001
Contrast	10.426	3	.001
Size x Diagnosis	.240	1	.630
Contrast x Diagnosis	.074	3	.974
Size x Contrast	.969	3	.415
Size x Contrast x Diagnosis	5.735	3	.002

Table 5: P1 Checkerboards Peak Amplitude ASD males v. NT males v. NT females

Criteria	**F value**	**df**	**P value**
Size	14.335	1	.002
Contrast	26.354	3	.001
Size x Diagnosis	1.995	2	.168
Contrast x Diagnosis	1.041	6	.411
Size x Contrast	2.627	3	.061
Size x Contrast x Diagnosis	1.956	6	.091

Table 6: P1 Checkerboards Peak Latency ASD males v. NT males v. NT females

Criteria	**F value**	**df**	**P value**
Size	7.803	1	.013
Contrast	8.728	3	.001
Size x Diagnosis	2.035	2	.163
Contrast x Diagnosis	.838	6	.547
Size x Contrast	.548	3	.653
Size x Contrast x Diagnosis	.846	6	.541

Table 7: P1 Checkerboards Fractional Area Latency ASD males v. NT males v. NT females

Criteria	**F value**	**df**	**P value**
Size	23.425	1	.005
Contrast	19.907	3	.001
Contrast	2.148	2	.212
Contrast x Diagnosis	1.121	6	.396
Size x Contrast	2.651	3	.087
Size x Contrast x Diagnosis	.501	6	.798

Table 8: P1 Checkerboards Area Amplitude ASD males v. NT males v. NT females

Criteria	**F value**	**df**	**P value**
Size	17.821	1	.001
Contrast	11.730	3	.001
Size x Diagnosis	1.320	2	.295
Contrast x Diagnosis	1.117	6	.367
Size x Contrast	1.593	3	.203
Size x Contrast x Diagnosis	2.800	6	.020

Table 9: N1 Checkerboards Peak Amplitude ASD v. NT

Criteria	**F value**	**df**	**P value**
Size	.731	1	.405
Contrast	4.446	3	.008
Size x Diagnosis	.731	1	.401
Contrast x Diagnosis	.729	3	.540
Size x Contrast	.266	3	.849
Size x Contrast x Diagnosis	1.210	3	.316

Table 10: N1 Checkerboards Peak Latency ASD v. NT

Criteria	**F value**	**df**	**P value**
Size	.040	1	.844
Contrast	.087	3	.967
Size by Diagnosis	.478	1	.499
Contrast x Diagnosis	.176	3	.912
Size x Contrast	1.744	3	.170
Size x Contrast x Diagnosis	.959	3	.419

Table 11: N1 Checkerboards Fractional Area Latency ASD v. NT

Criteria	**F value**	**df**	**P value**
Size	.529	1	.494
Contrast	3.117	3	.049
Size x Diagnosis	1.528	1	.263
Contrast x Diagnosis	2.357	3	.166
Size x Contrast	15.184	3	.001
Size x Contrast x Diagnosis	14.587	3	.001

Table 12: N1 Checkerboards Area Amplitude ASD v. NT

Criteria	**F value**	**df**	**P value**
Size	.599	1	.450
Contrast	4.048	3	.012
Size x Diagnosis	1.005	1	.330
Contrast x Diagnosis	.793	3	.503
Size x Contrast	.400	3	.753
Size x Contrast x Diagnosis	1.502	3	.225

Table 13: N1 Checkerboards Peak Amplitude ASD males v. NT males v. NT females

Criteria	F value	df	P value
Size	1.110	1	.308
Contrast	5.900	1	.002
Size x Diagnosis	1.120	2	.351
Contrast x Diagnosis	.890	6	.509
Size x Contrast	.429	3	.733
Size x Contrast x Diagnosis	1.271	6	.289

Table 14: N1 Checkerboards Peak Latency ASD males v. NT males v. NT females

Criteria	F value	df	P value
Size	.001	1	.977
Contrast	.057	3	.982
Contrast	.225	2	.801
Contrast x Diagnosis	.916	6	.492
Size x Contrast	1.515	3	.222
Size x Contrast x Diagnosis	.581	6	.744

Table 15: N1 Checkerboards Fractional Area Latency ASD males v. NT males v. NT females

Criteria	F value	df	P value
Size	.128	1	.735
Contrast	1.444	3	.270
Contrast	2.870	2	.148
Contrast x Diagnosis	1.174	6	.370
Size x Contrast	9.389	3	.001
Size x Contrast x Diagnosis	7.403	6	.001

Table 16: N1 Checkerboards Area Amplitude ASD males v. NT males v. NT females

Criteria	F value	df	P value

Size	1.049	1	.321
Contrast	5.665	3	.002
Size x Diagnosis	.903	2	.425
Contrast x Diagnosis	1.121	6	.365
Size x Contrast	.522	3	.669
Size x Contrast x Diagnosis	1.162	6	.342

Table 17: P1 Dartboard Main Effect of Diagnosis (ASD v. NT)

Dependent Variable	**F value**	**df**	**P value**
Peak Amplitude	.000	1	.994
Peak Latency	.020	1	.002
Fractional Area Latency	.220	1	.647
Area Amplitude	.081	1	.701

Table 18: P1 Dartboard Main Effect of Group (Male NT, Female NT, Male ASD)

Dependent Variable	**F value**	**df**	**P value**
Peak Amplitude	1.023	2	.391
Peak Latency	2.172	2	.160
Fractional Area Latency	.889	2	.439
Area Amplitude	1.939	2	.190

Table 19: N1 Dartboard Main Effect of Diagnosis (ASD v. NT)

Dependent Variable	**F value**	**df**	**P value**
Peak Amplitude	.000	1	.994
Peak Latency	.020	1	.890
Fractional Area Latency	.220	1	.647
Area Amplitude	.000	1	.994

Table 20: N1 Dartboard Main Effect of Group (Male NT, Female NT, Male ASD)

Dependent Variable	**F value**	**df**	**P value**
Peak Amplitude	.067	2	.935
Peak Latency	.028	2	.973

Fractional Area Latency	.173	2	.843
Area Amplitude	.037	2	.964