Abstract

Neurocognitive, Motor, and Social Abilities in Autism Spectrum Disorder:

An EEG Mobile Brain-Body Imaging Study

Purpose. Rising theories describe an interwoven connection between motor and social skills. Given high prevalence of motor and social deficits in autism spectrum disorder (ASD), it is important to examine underlying processes of natural movement, such as walking. No known study has examined social ability in relation to neural processing while walking in ASD. The goal was to investigate relationships among cognitive, motor, and social abilities in ASD and typically developing (TD) individuals utilizing electroencephalography (EEG) mobile brain-body imaging (MoBI).

Methods. ASD (n=20) and TD (n=18) adolescents participated in the cross-sectional study. Using EEG-MoBI, participants completed a Go/No-Go task while walking or standing. Separately, they completed a standardized motor assessment and Stroop test. Relationships among the following were examined: N2/P3, inhibition (Go/No-Go, Stroop), motor ability, gait variability, social ability, and autism severity.

Results. Regarding Aim 1, motor ability did not predict social ability in regression analyses; however, better motor ability was associated with better social ability in correlation analyses. Increased stride time variability was associated with social cognitive deficits. For Aim 2, no differences in Stroop were found based on motor ability. For Aim 3, for ASD, a trend demonstrated greater social deficits related to greater N2 (more negative) while walking. For TD, greater social deficits were related to greater N2, in walking and standing. For P3 in ASD, a trend demonstrated greater social deficits related to reduced P3 while walking and

standing; for TD, no links were found. TD exhibited greater N2/P3 in standing versus walking; ASD did not. Increased P3 while standing was related to lower autism severity. For Exploratory Aim 1, a trend demonstrated increased social deficits associated with worse manual dexterity in ASD; in TD the opposite was seen. Better manual dexterity was related to lower autism severity. Better balance was related to better social ability. For Exploratory Aim 2, weakened P3 while standing, not walking, was related to greater autism severity. N2 was not related to autism severity.

Conclusions. Findings generally supported the idea of a connection between social and motor skills. Increased stride time variability, and poorer balance and manual dexterity were related to greater social deficits. Poorer manual dexterity and weakened P3 were related to greater autism severity. TD, but not ASD, exhibited greater N2/P3 while standing than walking, suggesting differential neural processing. There are implications for screening, intervention, and education.

Neurocognitive, Motor, and Social Abilities in Autism Spectrum Disorder:

An EEG Mobile Brain-Body Imaging Study

by

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Submitted in partial fulfilment of the requirements

for the degree of

Doctor of Philosophy

in the

Ferkauf Graduate School of Psychology, Yeshiva University

Albert Einstein College of Medicine

New York

June 2023

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Acknowledgements

This endeavor would not have been possible without my chairperson, Dr. Andrea Weinberger, for her feedback, support, and patience over the past six years. I am additionally indebted to Drs. Pierfilippo DeSanctis and Sophie Molholm for their guidance and expertise. Special thanks to Drs. Christine Lee and Vance Zemon, who assisted, uplifted, and encouraged me. This dissertation would not have been possible without the participants and participants' families, who impacted and inspired me.

Many thanks to members of the Albert Einstein College of Medicine Cognitive Neurophysiology Lab and the Ferkauf Graduate School Smoking and Nicotine Dependence Research Lab, who helped in many aspects of completing projects. Thank you to the cohort, program, and clinical mates who have became trusted friends. I would also like to thank Albert Einstein College of Medicine, National Institutes of Health, and American Psychological Association for financial support for varied projects over the years.

Last, but not least: thank you to my family, partner, fur babies, and friends. Thank you for your unconditional support and love.

Dedication

For Nena B. Narvaez & Esteban Nicolas Cruz

thank you. gracias.

salamat. your tireless adventures

have endlessly inspired mine;

especially this one.

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Chapter I: Introduction

Overview of Autism Spectrum Disorder (ASD)

Autism spectrum disorder (ASD) is a neurodevelopmental disorder that is primarily characterized by abnormalities in social communication and behavior (APA, 2013). ASD affects individuals across all racial, ethnic, and socioeconomic groups. Based on the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM–5), diagnostic criteria for ASD include deficits in social communication and social interaction, as well as the presence of restricted, repetitive patterns of behavior, interests, or activities (APA, 2013). These symptoms typically persist throughout an individual's life. By the age of two, a diagnosis of ASD can be considered reliable if made by an experienced professional (Lord et al., 2006). However, most diagnoses are often made when the individual is older, which means that intervention and prevention services are delayed. Minshew and colleagues (2004) have suggested that research focused on the motor symptoms (e.g., abnormal gait), rather than on core psychiatric symptoms such as social deficit, has a greater potential to clarify the neurobiological basis of ASD, and thereby improve subsequent diagnosis and treatment (Minshew, Sung, Jones, & Furman, 2004).

Motor Ability in ASD

Recently, there has been increasing interest in examining motor development in ASD. Growing evidence suggests that motor deficits among young individuals precede and exacerbate the social deficits that are diagnostic of ASD; thus, certain motor signs may serve as biomarkers for ASD (Fournier, Hass, Naik, Lodha, & Cauraugh, 2010; Gandotra, 2020;

Harris, 2017; Hirjak et al., 2018; MacDonald, Lord, & Ulrich, 2014). Compared to social communicative deficits, there are fewer studies on motor behavior in ASD, despite "clumsy" gait in ASD reported as far back as 1943 (Kanner, 1968). Further, up to 80% of individuals with ASD exhibit motor deficits (Fournier et al., 2010; Green et al., 2009). Milestones of gross motor development – such as lying, sitting, crawling, and walking – are among the earliest identifiable clinical abnormalities in ASD (Esposito & Venuti, 2008; Travers et al., 2017). In a meta-analysis that included 41 studies, ASD participants performed significantly worse than typically developing (TD) participants in motor coordination, arm movement, gait, and postural stability tasks (Fournier et al., 2010).

Fine and Gross Motor Ability

Fine motor ability refers to the ability to coordinate small muscles such as fingers and hands to make precise movements (e.g., grasping, drawing), whereas gross motor ability refers to the ability to perform whole-body movement (e.g., walking, crawling, running) that use large muscles such as the torso, arms, and legs (Gonzalez, Alvarez, & Nelson, 2019). ASD children have been observed to have developmental delays in both fine and gross motor skills (Liu & Breslin, 2013). However, Whyatt and Craig (2011) have suggested that children with ASD are more likely to experience motor impairments with more complex tasks, specifically the ability to balance, compared to TD children.

Numerous researchers have utilized a standardized neuropsychological battery, the Movement Assessment Battery for Children (M-ABC 2) (Henderson, Sugden, & Barnett, 2007), to assess fine and gross motor functioning in younger individuals ages three through 16 years old. The M-ABC 2 measures three motor domains: manual dexterity, aiming & catching, and balance, and also provides a total score. In one study, Green and colleagues

(2009) used the M-ABC 2 to assess the frequency of movement impairments among school-aged children with ASD. The sample consisted of 101 ASD children ages 10 through 14 years old who had autism or broader ASD (i.e., Asperger's syndrome) and a wide IQ range (i.e., they included children with intellectual disability; IQ <70). Results demonstrated that 79% of ASD children exhibited clear movement impairments (<5th percentile), and an additional 10% of ASD children had borderline movement issues (5-15th percentile). The proportion of children with movement dysfunction was similar between the ASD group and broader ASD group. ASD children exhibited greater movement impairment compared to the broader ASD children. There was also a main effect of IQ, with lower IQ children exhibiting more movement impairment than higher IQ children. Further analyses demonstrated that across all ASD children, participants had the most difficulty with the timed manual dexterity pegboard activity and the static board balance task compared to all other tasks (Green et al., 2009).

Also using the M-ABC 2, Ament and colleagues (2015) evaluated motor functioning in children ages eight through 13 years with ASD (n = 56) and ADHD (n = 63), as well as TD children (n = 81). The three groups had comparable IQ scores as determined by Wechsler tests. Results demonstrated that children with ASD and ADHD performed more poorly than TD children, suggesting decreased motor ability. When broken down by items, the pegboard task, catching, and dynamic balance tasks were significantly associated with having either diagnosis. When comparing ASD with ADHD at the sub-scale level, ASD performed significantly more poorly on the aiming & catching as well as the balance subscales. At the item level, catching and static balance distinguished the two groups, with poorer performance among children with ASD compared to children with ADHD (Ament et al., 2015). Overall,

these studies demonstrate that children with ASD typically present with abnormal motor functioning, particularly in regard to gross motor ability.

Gait

Movement such as walking was once considered an activity that required minimal cognitive input; however, the role of high-level cognitive processes to maintain balance and adjust gait while navigating a complex environment has become increasingly recognized (Gwin, Gramann, Makeig, & Ferris, 2011; Holtzer, Verghese, Xue, & Lipton, 2006; Ijmker & Lamoth, 2012; Weiss, Herman, Giladi, & Hausdorff, 2015; Yogev-Seligmann, Hausdorff, & Giladi, 2008). Gait instability is considered a gauge of motor balance. For instance, to feel more balanced when walking, one may increase their step width (i.e., distance between the heels of the two feet) to obtain a wider base of support (Kindregan, Gallagher, & Gormley, 2015). Gait research in ASD has mostly focused on spatiotemporal gait measures such as stride length and stride time, with inconsistent results (Gong et al., 2020; Manicolo, Brotzmann, Hagmann-von Arx, Grob, & Weber, 2019; Rinehart, Tonge, Bradshaw, et al., 2006). A more consistent picture has begun to emerge when investigating gait variability (i.e., stride-to-stride fluctuations), which can also represent the regularity and "smoothness" of gait (Hausdorff, 2005; Manicolo et al., 2019). A fine-grained analysis on gait parameters may provide novel insights into motor functions in ASD.

Thus far, few researchers have investigated gait in individuals with ASD. In one retrospective study, Esposito and Venuti (2008) analyzed gait in toddlers to examine whether movement could serve as an early indicator of ASD. Using home videotapes that were provided by the toddlers' families, the researchers examined gait on three axes: foot movements, arm movements, and global movements. The sample included male toddlers (*M*

age = 20 months) in three groups: those with a future ASD diagnosis (n = 16), TD (n = 16), and those with a future mental retardation status (n = 10) (Esposito & Venuti, 2008). The team coded 210 minutes of home videos, with five minutes of video per participant. Researchers who were blind to participants' diagnoses only coded scenes in which the child was walking or where the whole body was visible, and subsequently assembled a five-minute video for each child.

Children who received a diagnosis of ASD in the future scored significantly higher (representing more atypical walking patterns), than both children who did not receive any future diagnosis, as well as children who received a future diagnosis of mental retardation, for both total scores and foot movements (Esposito & Venuti, 2008). Of the three subtests included for foot movement, heel-toe pattern (i.e., heel touches surface first followed by rolling to the ball, and lastly the toes) also discriminated significantly among groups. There were significant differences in all five subtests of walking-related arm movement axis across groups, with ASD exhibiting the most atypical walking patterns. For global movements, stereotyped or repetitive movements significantly distinguished the groups. This study highlights the potential diagnostic utility of assessing movement, specifically gait, in ASD.

Rinehart and colleagues (2006) explored whether gait characteristics of young ASD children were consistent with clinical characteristics of cerebellar ataxia, a degenerative disease characterized by gait imbalance stemming from cerebellar damage. ASD and TD children walked at their preferred walking speed on an electronic walkway, the GAITRite Walkway. In the tandem condition, they followed a white line, placing one foot in front of the other. Physiotherapists qualitatively rated specific aspects of movement and postural control, such as coordination, smoothness, consistency, and head and trunk posture. The

authors did not find significant differences in velocity, cadence, stride length, double support, and heel-to-heel base of support. However, on the walkway, ASD children exhibited greater width range, indicating poorer ability to walk straight. ASD children also demonstrated higher variability and irregularity in the preferred walking condition in gait velocity, stride time, and stride length. No gait differences were seen in the tandem condition (Rinehart, Tonge, Iansek, et al., 2006). This study highlights that motor abnormalities indicative of cerebellar ataxia can be recognized in ASD, and provides additional evidence that motor function may be useful as a clinical screening tool for ASD.

In a recent study, Manicolo and colleagues (2019) examined the relationship between gait and motor milestones within ASD (n = 32) and TD (n = 36) children (*M* age = 9.2 years). They hypothesized that ASD children would exhibit a more unstable and irregular walking pattern compared to TD children. They also explored whether gait variability measures were associated with motor milestones and motor skills. Gait parameters were assessed using the GAITRite walkway, and motor domains were assessed with the M-ABC 2. Motor milestones were self-reported by parents. Similar to previous studies, Manicolo and colleagues (2019) found that ASD children performed significantly worse than TD children on all three M-ABC 2 subscales. While the groups performed similarly in stride velocity, stride time, and stride length, ASD children exhibited higher gait variability compared to the TD children, reflecting a more abnormal walking pattern. Further, in ASD children, the M-ABC 2 total score was significantly associated with all measures of gait variability (i.e., variation in stride velocity, stride time). In other words, better motor skills were associated with lower gait variability in ASD children; notably, however, this relationship was not seen in TD children

(Manicolo et al., 2019). This lends support to the notion of potentially differing processes underlying walking for ASD versus TD individuals.

Lum and colleagues (2020) recently conducted a meta-analysis to examine gait parameters in children and adults, comprised of ASD as well as TD individuals. A total of 18 studies were included in the review with a total sample size of 561 individuals (n = 283 ASD; n = 278 TD). Methodological quality of each study was assessed using a modified version of the Newcastle-Ottawa Quality Assessment Scale, and factors such as IQ, height, and weight were considered when the information was provided for a specific study. Of note, a range of apparatuses were utilized across studies. Results of the analyses demonstrated that relative to TD individuals, ASD individuals exhibited wider step width, slower walking speed, longer gait cycle, longer stance time, and longer step time (Lum et al., 2020). Further, having ASD was associated with greater intra-individual variability on stride length, stride time, and walking speed. Overall, the authors concluded that given the results, there is evidence that ASD is associated with abnormal gait, and that certain gait parameters may serve as a useful biomarker for ASD.

Taken together, these studies demonstrate patterns of irregular gait variability among ASD individuals compared to TD individuals. While ASD and TD individuals perform similarly in terms of velocity and stride time, data indicate significantly greater gait variability and wider base of support needed for ASD individuals, representing a less steady walking pattern in this group compared to TD individuals.

Social Ability and Motor/Gait Abilities

Given that social deficits are widely prevalent in ASD (Lord, Cook, Leventhal, & Amaral, 2000), another important area to examine in ASD is social ability and social

responsiveness, or the ability to engage in appropriate social interactions and reciprocity (Constantino et al., 2003). Neuroanatomical and developmental perspectives both support the idea of a critical relationship between motor and social skills. Neuroanatomically, there is neural overlap, or shared brain regions and circuity, involved in social processing and motor functions (Carta, Chen, Schott, Dorizan, & Khodakhah, 2019). One region that has been consistently implicated to have abnormal neural processing in ASD is the cerebellum, based on postmortem and brain imaging studies (Ohara, Kanejima, Kitamura, & Izawa, 2019). The cerebellum serves multiple functions, including those that relate to both motor and social development. The cerebellum plays important roles in motor coordination, control, and balance (Ataullah & Naqvi, 2022), and has also been implicated in social cognition and executive functioning (Van Overwalle, Baetens, Marien, & Vandekerckhove, 2014; Van Overwalle et al., 2020). Additionally, Casartelli et al. (2016) argues that the circuits associated with motor ability may be connected to or trigger abilities in understanding others, which would contribute to social dysfunction in ASD (Casartelli & Molteni, 2014; Casartelli, Molteni, & Ronconi, 2016).

Developmentally, the learning and development of social and motor skills are intricately interwoven, especially during an individual's early years. Motor ability is an important tool for learning appropriate social skills (Gibson, 1988). As described in a review on the impact of motor development and social cognition, developing motor skill can affect the number and types of opportunities that young children have to interact with others, and consequently, development of social relationships (Leonard & Hill, 2014). For instance, infants who were independent walkers interacted more with their mothers compared to infants who crawled, suggesting that motor development may change a child's social

interactive environment (Campos et al., 2000; Leonard & Hill, 2014). Additionally, movement is also large part of playing, and if a child experiences motor delays or deficits, the social opportunities and knowledge gained during play will thus also be limited (Ohara et al., 2019). Abnormal movement also appears to result in atypical interpretation of others' movements (Cook, 2016). Taken together, understanding the link between motor ability and social skills is especially crucial in ASD where both features may be affected.

Though most studies have explored motor and social abnormalities in ASD separately, several researchers have started to investigate the idea that motor mechanisms may be related to social functioning. It has been argued that "motor cognition" is an intermediate phenotype (i.e., trait that is reasonably heritable) for ASD (Casartelli et al., 2016; Gallese, 2013). To this end, researchers have found links between motor and social communication skills; better motor skills are typically associated with better social ability (Dziuk et al., 2007; Mostofsky & Ewen, 2011). Further, motor abnormalities may be a biomarker of, and contributor to, social dysfunction in ASD (Gallese, 2013).

In one study, Bradshaw and colleagues (2018) examined associations between motor and social communication skills in 12-month-old infant siblings at high-risk for autism due to having an older sibling with autism (n = 86) versus low-risk controls (n = 113). Individuals were classified as pre-walkers, standers, or walkers. High-risk walkers had higher social-communication scores compared to high-risk pre-walkers (cognitive function did not differ between the groups). In contrast, a relationship between walking stage and social-communication was not found for the low-risk group (Bradshaw et al., 2018). The integrity of functional connections between motor cortex and other regions has also been implicated in poorer social communication in ASD. Analyzing resting state fMRI from children ages 8-12

years old with and without autism, another group of researchers found that functional connectivity between visual and motor regions was impaired, and that poorer functional connectivity was associated with poorer social communication skills, and suggested in turn that this impaired communication interfered with both motor and social skill acquisition (Nebel et al., 2016).

Several meta-analyses and reviews have been conducted to examine the relationship between social ability and motor skills in ASD. In a meta-analysis that included 890 infants (ages 6 through 43 months), West and colleagues (2019) found that within infants with ASD, better fine and gross motor ability motor ability were related to better social communicative ability. A stronger relationship was observed between fine motor skills and social/language ability, to relative gross motor skills. The suggested pathway of the social-motor relationship is through the links between action, perception, and cognition (West, 2019). West et al. (2019) described that every motor action carries visual, tactile, and proprioceptive information, which are materials used for gathering data - including social information - about the world. As one example, social stimuli, such as mutual gaze, happen more in positions where one is standing or walking, relative to other postures (Franchak, Kretch, & Adolph, 2018; West, 2019). In another study, Ohara and colleagues (2019) also investigated the links between social and motor abilities in ASD. In a systematic review of 16 studies within ASD, they found that more than half of the studies (12) reported associations between social and motor abilities (Ohara et al., 2019). The gross motor ability of object control skills was most associated with socials skills, whereas the fine motor ability of manual dexterity was most associated with socials skills.

At this time, there is only one known study that examined gait parameters in relation to social severity in ASD. Gong and colleagues (2020) investigated whether abnormal coordination during gait among children with ASD would correlate with core symptoms, such as social deficits. The sample included ASD children who were divided into high functioning (n = 46) and low functioning (n = 12), as well as TD children (n = 28) who were age and FSIQ-matched with the high functioning children. To measure gait, children were instructed to walk barefoot on a carpet that held a plantar pressure mat with sensors (RsScan, Inc.) in the middle. Using the mat, Gong et al. (2020) examined foot-rolling patterns and pressure, gait, and gait variability. To measure autistic traits and social ability, Gong et al. (2020) used the Social Responsiveness Scale (SRS) (Constantino et al., 2003). The SRS measures social responsiveness and social deficits, and is associated with ASD diagnosis. Regarding results, they found that relative to the control group, both ASD groups exhibited flat-footed pattern, increased left–right asymmetry, and larger step-to-step gait variability. Notably, after controlling for IQ, across the full sample, larger gait variability was associated with increased social deficits (Gong et al., 2020).

Overall, there is strong evidence of shared social and motor circuity in general as well as in ASD in particular (Bradshaw et al., 2018; Dziuk et al., 2007; Leonard & Hill, 2014; MacDonald, Lord, & Ulrich, 2013; Mostofsky & Ewen, 2011; Nebel et al., 2016; Ohara et al., 2019; West, 2019). Further, a fair share of recent studies has demonstrated that gait variability is a particular motor weakness among ASD individuals, with nascent research exhibiting a link between gait variability and core autism features such as social deficits. More studies probing the shared neural bases of motor/gait ability as well as social ability are

needed to better understand the interrelationship between these processes in ASD, where both domains are vulnerable.

Neurocognitive Functioning in ASD

Neurocognition is an inherent and critical part of all behavioral functions, including motor, gait, and social abilities. Neurocognition refers to cognitive ability that has been reliably linked to particular brain regions, neural pathways, or cortical networks within the brain. The neurocognitive domain of executive functioning refers to skills required to complete complex, goal-directed actions, adapt to a range of environmental demands, and to extemporize or adjust to task demands when unexpected events arise (Chan, Shum, Toulopoulou, & Chen, 2008; Miyake et al., 2000). Executive functioning has been linked to the frontal lobes, particularly to areas within the prefrontal cortex. Of note, executive functioning is a heterogeneous construct, with diverse aspects of EF linked to varying regions within the prefrontal cortex (Zelazo, 2002).

Within ASD, deficits in executive functioning, or executive dysfunction, are widely prevalent (Hill, 2004; Hughes, Russell, & Robbins, 1994). Executive dysfunction may present as disorganization, perseveration, slower information processing, and inability to inhibit inappropriate behaviors, all or most of which are commonly seen in ASD (Hill, 2004). Further, researchers have surmised that executive dysfunction is linked to repetitive and restricted behaviors, a core characteristic of ASD, due to an inability to generate new behaviors or shift set (Kenworthy, Black, Harrison, della Rosa, & Wallace, 2009). Having increased types of cognitive deficits have also been associated with higher symptom severity in ASD (Brunsdon et al., 2015), and executive dysfunction has also been related to social-

communication impairments (Christ, Holt, White, & Green, 2007; McEvoy, Rogers, & Pennington, 1993).

In regard to the relationship between neurocognition and motor abilities, in TD populations, motor performance as a toddler was found to be predictive of future cognitive performance, when individuals were school-aged (Piek, Dawson, Smith, & Gasson, 2008). Gait has been investigated in older adult populations, where worse cognitive abilities across domains were linked with gait abnormality (Hamacher, Herold, Wiegel, Hamacher, & Schega, 2015; Sheridan, Solomont, Kowall, & Hausdorff, 2003; Yogev-Seligmann et al., 2008). However, few studies have investigated neurocognitive and motor abilities in ASD, and no known studies have examined the relationship between neurocognitive ability and gait in a sample of individuals with ASD.

Inhibitory Control

Inhibitory control is a type of executive function that refers to the ability to inhibit or refrain from making an habitual or dominant behavioral response to stimuli, in order to select a more suitable behavior that is consistent with completing goals (Diamond, 2013). Repetitive and stereotyped behaviors prevalent in ASD are thought to be highly suggestive of inhibitory difficulties (Adams & Jarrold, 2009). Indeed, inhibitory errors in individuals with ASD have been linked to certain repetitive behaviors, such as compulsions (Adams & Jarrold, 2009; Mosconi et al., 2009). A recent meta-analysis that included 41 studies demonstrated that ASD individuals (n = 1,091) had increased difficulties in prepotent response inhibition compared to TD individuals (n = 1,306) (Geurts, van den Bergh, & Ruzzano, 2014).

A common inhibitory control test is the continuous performance test. There are many published forms of this test that are used in research as well commercially. The common attribute among the tests is that they involve sequential presentation of stimuli over an extended period of time. One form of a continuous performance test is the stop-signal task, also known as the Go/No-Go task (Lipszyc & Schachar, 2010). During the Go/No-Go task, individuals are instructed to perform a primary task, such as pressing a button if they see a specific visual stimulus that is displayed for a majority of the trials. On a minority portion of trials (typically ~25%), participants are instructed to withhold the button press when they see a different visual stimulus. For instance, an individual is instructed to click the button press when there is an "O" on the screen ("Go" trial), and withhold clicking the button when there is an "X" on the screen ("No Go" trial). The "O" is displayed on the screen for a majority of the time, while the "X" is displayed for a shorter period of time. Go/No-Go performance can be evaluated a number of ways, including reaction time (time to response), number of hits (correctly responding with button press), correct rejections (correctly inhibiting the response), and false positives (pressing button when they should have inhibited the response).

Many forms of the Go/No-Go task have been studied across clinical populations, including ASD. In a recent study using an online Go/No-Go task (n = 201 ASD; n = 240 TD), a higher rate of false alarms and false positives were associated with having autism as well as common autism traits and symptomology (Uzefovsky, Allison, Smith, & Baron-Cohen, 2016). ASD studies also have utilized Go/No-Go tasks to examine neural indices, such as to trigger event-related potentials or measure activity from blood flow in the brain (Casey et al., 1997; Hoyland et al., 2017). The next section below will discuss more studies examining Go/No-Go performance and neural substrates in ASD.

Another widely-used test to measure inhibitory control is the Stroop Color and Word Test (Stroop, 1935). Similar to the Go/No-Go task, the Stroop test measures the ability to inhibit a prepotent response. In the Stroop test, the participant is instructed to name the ink color that a word is printed in, and ignore the color that the word says (i.e., when the word “blue” is printed in green ink, the correct response is "green"). The difficulty that many individuals have in inhibiting the more automated process of reading the word is called “the Stroop effect”. Since its development in 1935, many different forms of the Stroop test have been created, with diverse scoring methods. Findings of the Stroop task in ASD samples have been mixed, with some studies pointing to similar performance between ASD and TD groups (Goldberg et al., 2005; Hill, 2004), while other studies demonstrated worse performance in ASD groups compared to TD groups (Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Robinson, Goddard, Dritschel, Wisley, & Howlin, 2009). Notably, word reading ability should be accounted for given weaker verbal comprehension in ASD (Adams & Jarrold, 2009).

Overall, inhibition appears to be an area of difficulty for ASD, which has been consistently demonstrated through varying test forms such as the Go/No-Go task and the Stroop test. No known study has explored potential relationships in inhibition, ASD diagnosis, and motor ability. Doing so may provide insight into how motor ability may relate to neurocognitive inhibition in ASD.

Neural Indices (N2/P3) in ASD

As one method of investigating neurocognition using the Go/No-Go task, electroencephalography (EEG) is an electrophysiological tool that allows for the measurement of brain activity in the form of electrical brain waves. Event-related brain

potentials (ERPs) reflect the average brain responses based on the presentation of a stimulus, such as a visual or an auditory stimulus. EEG and ERPs provide an essential bridge in examining the relationship between brain structure and function and behavioral performance (Polich, 1993). Lower EEG power (i.e., frequency) in frontal polar, temporal, and parietal brain regions has been associated with higher risk for autism symptoms, such as socioemotional difficulties (Brito et al., 2019). Regarding ERPs, the ERPs N2 and P3 are consistently elicited in inhibitory tasks such as Go/No-Go paradigms, which are considered by most researchers to measure inhibitory control (see previous section). The cortical region considered to be responsible for generating the N2 is the anterior cingulate cortex, which is located in the prefrontal cortex (Van Veen & Carter, 2002).

N2

The ERP N2 is a negative deflection that is elicited 200 - 450 milliseconds after the onset of a stimulus, and is typically observed at fronto-central scalp sites. Of note, some debate exists as to whether the N2 more specifically represents conflict monitoring (i.e., detecting information discrepancies) rather than inhibition (Donkers & van Boxtel, 2004; Geraldo, Azeredo, Pasion, Dores, & Barbosa, 2019), or that the N2 reflects neither while the P3 is a more accurate reflection of inhibition (Smith, Johnstone, & Barry, 2007). In brief, according to conflict monitoring theory, individuals who exhibit more difficulty with control have decreased ability to process target stimuli, or deal with competing responses from task-relevant and task-irrelevant information; therefore, discrepancies between errors and correct trials in Go/No-Go tasks are expected, which results in neural differences that can be observed through ERP amplitudes (Larson, 2011; Lo, 2018). While the nuances of the diverse but overlapping constructs of N2 and P3 are yet to be precisely agreed upon (i.e.,

whether they represent conflict monitoring or inhibition), there is consensus that N2 and P3 both reflect effortful cognitive processing.

Larger (i.e., more negative) N2 amplitudes represent more effortful neural processing in the context of conflicting information (Hoyniak, 2017). N2 amplitudes tend to be larger (i.e., more negative) on successful No-Go trials than on Go trials , as well as during trials in which discriminability between No-Go and Go stimuli is challenging (Cragg, Fox, Nation, Reid, & Anderson, 2009; Folstein & Van Petten, 2008; Jodo & Kayama, 1992). Of note, there is some discrepancy regarding whether larger N2 amplitude represents inefficiency, or whether larger N2 represents having better inhibitory control. Across the general population, children with stronger conflict monitoring skills, demonstrated reduced (i.e., more positive) N2 amplitude as well as shorter N2 latencies, relative to children with weaker conflict monitoring (Lamm, Zelazo, & Lewis, 2006; Rueda, Rothbart, McCandliss, Saccomanno, & Posner, 2005; Todd, Lewis, Meusel, & Zelazo, 2008).

Based on a meta-analysis across clinical populations (e.g., ADHD, anxiety disorders), children with ADHD exhibited larger amplitudes compared to TD children in several studies; however, reduced (i.e., more positive) amplitudes was most frequently been observed in clinical children relative to TD children (Lo, 2018). In other studies using inhibition tasks, reduced N2 amplitude was observed within juvenile offenders (Vila-Ballo, Hdez-Lafuente, Rostan, Cunillera, & Rodriguez-Fornells, 2014), depressed individuals (Kaiser et al., 2003), individuals with bipolar disorder (Morsel et al., 2017), adult smokers (Luijten, Littel, & Franken, 2011), and individuals with amnestic mild cognitive impairment (Cid-Fernandez, Lindin, & Diaz, 2014). On the other hand, also using inhibition tasks, larger N2 amplitude was seen in children with concussions (Moore et al., 2015) and depressed individuals with

binge drinking symptoms (Connell, Danzo, & Dawson, 2018). Overall, there are varying findings regarding observations of enlarged or reduced N2 amplitude in clinically diverse populations relative to healthy controls.

P3

The ERP P3 is positive, and is elicited 300-600 milliseconds after stimulus onset, typically observed at frontal-central and medial scalp sites. Similar to N2, P3 is also considered to be an index of executive functioning. More specifically, P3 has been theorized to reflect attentional capacity, information processing, orienting, and processing novelty (Comerchero & Polich, 1999; Cui, Wang, Liu, & Zhang, 2017). A more updated theory of P3 breaks P3 down even further, into P3a (originating from frontal brain regions and reflects attention) versus P3b (originating from temporal and parietal brain regions and reflects attention, memory processing, and decision making) (Polich, 2007).

Generally, P3 amplitude decreases when less attentional and other cognitive-related neurons are aroused. Further, reduced P3 amplitude is observed during tasks deemed to be more challenging, reflecting that more cognitive resources are being utilized (Cui et al., 2017; Polich, 1987). Consistent with this, larger P3 amplitude is associated with the effective recruitment of cognitive resources, or better cognitive abilities (Geraldo et al., 2019; Larson, 2011). In regard to Go/No-Go tasks, in TD individuals larger (i.e., more positive) P3 amplitude is typically observed on No-Go trials relative to Go trials (Bokura, Yamaguchi, & Kobayashi, 2001; Eimer, 1993; Kopp, Mattler, Goertz, & Rist, 1996).

In clinical populations during inhibition tasks, reduced P3 amplitude was observed within individuals who smoke (Evans, Park, Maxfield, & Drobes, 2009; Yin et al., 2016), as well as in children with learning disabilities, however significance dissipated once IQ was accounted for (Buchmann, Gierow, Reis, & Haessler, 2011). On the other hand, also during

inhibition tasks, larger P3 amplitude was observed within individuals with bipolar disorder (Morsel et al., 2017). P3 amplitudes did not differ between individuals with amnestic mild cognitive impairment and healthy controls (Cid-Fernandez et al., 2014). P3 amplitudes were also similar between children with ADHD and controls (Buchmann et al., 2011). Overall, similar to N2, there are varying findings regarding observations of increased or reduced P3 amplitude in clinically diverse populations relative to healthy controls.

N2/P3 in ASD

In ASD, an assortment of studies have examined N2/P3, eliciting the ERPs using an array of tasks. Several studies have specifically utilized the Go/No-Go Task. Within ASD, inefficiency in behavioral responses (i.e., slower reaction time or more errors) from Go/No-Go tasks have been linked to lower N2 but not P3 amplitudes (Magnuson et al., 2019). Recent studies within ASD groups have found contrasting N2/P3 results elicited by Go/No-Go tasks (Cox et al., 2015; Hoyland et al., 2017; Kim, Grammer, Benrey, Morrison, & Lord, 2018).

For instance, Hoyland and colleagues (2017) investigated N2 and P3 associated with cognitive control measures, using a Go/No-Go paradigm, within ASD (n = 49) and TD individuals (n = 49) ages 12 through 21 years old. The Go/No-Go paradigm included neutral pictures and pictures of emotional faces, and participants were given instructions as to which pictures they had to press a button for (“Go”) and which pictures to refrain from pressing for (“No Go”). The main goal of the study was to compare differences between ASD and TD in the amplitudes of these ERPs, elicited by the Go/No-Go task: P3, contingent negative variation (CNV; a slow negative potential elicited in the time between the cue and the stimulus), N2 Go and No-Go, P3 Go, and P3 No-Go. No group differences were observed

between ASD and TD, however age-related differences in relation to the N2 No-Go were found (Hoyland et al., 2017). Notably, 17 ASD participants with comorbid ADHD were included; when these participants were excluded from analyses, they found that the N2 No-Go effect was significantly increased, compared to when ASD individuals were included in analyses. Regarding P3, similar amplitudes in the ASD and TD groups were observed.

In another study, Cox and colleagues (2015) examined neural responses to social and non-social reward anticipation in 35 TD young adults, specifically examining modulation of reward sensitivity by level of autistic traits. To evoke neural responses, they used a modified version of a cued incentive-delay ERP task with three incentive conditions: a social reward (video feedback from an observer), non-social reward (candy) and a non-reward (video of a grey shape). Participants were instructed to click a response as quickly and as accurately as possible upon seeing a specified target on the screen. Notably, they measured autistic traits using the SRS (Constantino et al., 2003). As the authors hypothesized, higher expressions of autistic traits were associated with a weakened P3 response to the anticipation of social, but not non-social (candy), rewards among the non-clinical, young adult sample (Cox et al., 2015).

Due to inconsistencies seen in P3 across research studies, Cui and colleagues (2016) conducted a meta-analysis to investigate P3a and P3b amplitude and latency in ASD relative to TD groups. Thirty-two studies were included in the analysis, with a total of 407 ASD individuals and 457 TD individuals. Relative to the TD group, significantly reduced P3b amplitude was observed, while P3a amplitude, P3a latency, and P3b latency were similar between the ASD and TD groups (Cui et al., 2017). The authors therefore theorize that memory processing and decision making differs in ASD relative to TD groups.

To summarize, while reduced N2/P3 responses are typically elicited during inhibition No-Go trials in the general population, the N2 and P3 inhibitory responses produced by Go/No-Go tasks is less clear for ASD groups. There are few studies that have specifically used the Go/No-Go to examine N2/P3 neural processing in ASD groups. Furthermore, no known studies have investigated social ability in relation to N2/P3 neural indices, nor gait parameters, within ASD individuals.

Mobile Brain Body Imaging (MoBI)

The EEG-based mobile brain-body imaging (MoBI) system is an integrative, multimethod system that examines human brain activity, motor behavior, and other physiological data associated with cognitive processes that involve active behavior (Jungnickel, 2018). MoBI allows for the recording of synchronized high-density electrophysiological brain activity in concert with 3D motion-capture kinematic data, while individuals walk on a treadmill. The present lab has previously utilized the MoBI system within healthy subjects, older adults, and individuals with multiple sclerosis (De Sanctis, Butler, Malcolm, & Foxe, 2014; De Sanctis et al., 2020; De Sanctis et al., 2021; Malcolm, Foxe, Butler, & De Sanctis, 2015; Malcolm, Foxe, Butler, Molholm, & De Sanctis, 2018).

In the parent study for the current study (see Methods section), there were multiple conditions of task type and flow type. Regarding the task, the task could be done as a single-task (walking only) or dual-task (walking while performing Go/No-Go task). As for flow type, the parent study investigated sensorimotor processes with optical flow, in which a large-scale visual display was projected in front of them, consisting of 100 randomly placed white dots emanating outward from a central focus of expansion onto a wall. The flow

condition was either with optical flow condition or a no flow condition (white dots were static on the screen).

Of note, for the purposes of the current study, gait performance during the static *(no flow), no-task (not performing the Go/No-Go Task)* conditions were utilized; hence, any effect of optical flow and performing the Go/No-Go task would not be factors. Further, only the N2 and P3 ERPs elicited from the correct rejections (No-Go) from the Go/No-Go task, during the *static (no optical flow)* were analyzed. More information is described in the Research Design section.

Significance & Innovation

Given the high prevalence of motor dysfunction in the ASD population, it is important to further examine the underlying neural processes of natural movement such as walking. Additionally, the links between motor ability, social ability, neurocognition, and underlying neural indices should be clarified. Although there has been a growing momentum in examining motor and social behaviors in ASD in recent decades, no known study has investigated social responsiveness in relation to neural processing while walking and standing, in ASD versus TD. Further, no published studies have used the EEG/MoBI design to investigate movement and neural indices within ASD populations.

The goal of this study was to integrate neurophysiological data using MoBI, and neuropsychological data with standardized movement and clinical measures to better understand motor, gait, and social ability in autism. This work is expected to illuminate the relationships between motor ability, social behavior, and neural functioning in ASD, thus potentially paving the way for more efficacious and personalized treatment and intervention options for motor, social, and cognitive deficits in ASD.

Specific Aims

Specific Aim 1: To investigate whether motor ability is associated with social ability in ASD and TD individuals.

Hypothesis 1a: We predicted that gross motor impairment will be associated with poorer social ability for the ASD group, but not the TD group.

Hypothesis 1b: We predicted that greater gait stride time variability will be associated with poorer social ability for the ASD group, but not the TD group.

Hypothesis 1c: We predicted that greater gait step width variability will be associated with poorer social ability for the ASD group, but not the TD group.

Specific Aim 2: To determine whether there is an effect of ASD diagnosis (ASD versus TD) and motor ability on inhibitory control.

Hypothesis 2: We predicted that there will be an interaction between ASD diagnosis and motor ability on inhibitory control, such that ASD individuals with low motor ability will demonstrate poorer inhibitory control, compared to ASD individuals with high motor control, TD individuals with low motor ability, and TD individuals with high motor ability.

Specific Aim 3: To examine neural substrates of inhibitory control while walking and standing in relation to social ability in ASD and TD.

Hypothesis 3a: We predicted that as the number of social deficits increases, N2 amplitude (evoked from an inhibition task while standing) is expected to be reduced (i.e., more positive) for the ASD group, but not the TD group.

Hypothesis 3b: We predicted that as the number of social deficits increases, N2 amplitude (evoked from an inhibition task while walking) is expected to be reduced (i.e., more positive) for the ASD group, but not the TD group.

Hypothesis 3c: We predicted that as the number of social deficits increases, P3 amplitude (evoked from an inhibition task while standing) is expected to be reduced (i.e., more negative) for the ASD group, but not the TD group.

Hypothesis 3d: We predicted that as the number of social deficits increases, P3 amplitude (evoked from an inhibition task while walking) is expected to be reduced (i.e., more negative) for the ASD group, but not the TD group.

To clarify further, positive correlations are predicted for Aims 3a and 3b, while negative correlations are predicted for Aims 3c and 3d.

Exploratory Aim 1: To examine whether specific motor domains (aiming & catching, balance, and motor dexterity) are related to social ability among ASD individuals and, separately, TD individuals.

Exploratory Aim 2: To examine whether N2/P3 indices are related to clinically rated levels of autism severity.

Chapter II: Research Design & Methods

The study was a secondary analysis from a primary study titled "The Neurophysiological Underpinnings of Sensory-Motor Dysfunctions in Autism Spectrum Disorder" that recruited ASD and TD adolescents and young adults who were matched on gender, age, and IQ. The parent study procedures were approved by the Albert Einstein College of Medicine Institutional Review Board (IRB #2016-6736; PI: Pierfilippo De Sanctis, PhD). The current study procedures were approved by the Yeshiva University, Ferkauf Western IRB (IRB Study #1315824; Tracking #20213660; PI: Lisa N. Cruz, MA).

Participants

Participants were originally recruited by the Human Clinical Phenotyping Core (HCP) at the Albert Einstein College of Medicine. The HCP is part of the National Institute of Child Health and Human Development (NICHD) funded Intellectual and Developmental Disabilities Research Center at the Albert Einstein College of Medicine (AECOM). The HCP maintains an extensive research-participant database, which currently contains more than 1,700 people who have agreed to be contacted for future studies. There is also a database for TD participants who were contacted for the study. Along with the HCP databases, subjects were recruited by community outreach, announcements on social media, listservs, recruitment events, and referrals from clinical partners. Individuals were screened (see below for eligibility criteria). Care was taken to obtain informed consent from the participant or parent/guardian (depending on legal age) to ensure that HIPAA, state law, and professional ethical standards were met. Licensed clinical neuropsychologists administered and

supervised the clinical and neuropsychological aspects of the project. Graduate research assistants (RA) and full-time RAs supported the recruiting, screening, obtaining of informed consent from participants or parents/guardians, collecting demographic information, distributing self-report questionnaires, and administering clinical and neuropsychological assessments. All RAs were trained and assessed for competency on providing the measures in a professional, standardized, and ethical manner.

Inclusion criteria for the parent study: *All participants*: ages 13-25 years (given that autism in adolescence was of interest); IQ > 70 based on Wechsler tests. *ASD subjects*: diagnosis obtained by a trained HCP clinical psychologist using the Autism Diagnostic Interview-R (ADI-R) (Lord, Rutter, & Le Couteur, 1994), Autism Diagnostic Observation Schedule (ADOS) (Lord, Risi, et al., 2000), and clinical judgment.

Exclusion criteria for the parent study: *TD subjects:* history of psychiatric, educational, or other developmental issues as reported on questionnaires.

There were no additional inclusion nor exclusion criteria for the current study.

Study Procedures

Participants were recruited by RAs who searched for potentially eligible subjects using the HCP database (mentioned above in the Participants section). Prior to coming in, all participants completed a web-based, secure survey (RedCap) to screen for general and mental health. RAs obtained informed consent from either the parent/guardian (if under age 18) or the participant him or herself. Participants came in for two sessions for the present study. The first session was to perform the EEG MoBI session. The EEG MoBI session lasted approximately 3.5 hours, with half an hour for capping, 1.5 hours for data collection, and 1.5 hours for total rest periods. The second session was to complete neuropsychological

testing, which lasted from 1 to 3 hours. Of note, the timing was partially dependent upon which data had already been collected through participation in other lab studies (i.e., if IQ testing had already been recently obtained through another HCP study, the data from the prior study were used and IQ testing was not done again for the present study). Participants were compensated at the rate of $15 per hour for time and travel expenses. See Figure 1 for summary of sessions.

Measures

The following lists measures that were collected in the parent study and will be used for the present analysis. The full collection of parent study measures is not listed here. Of note is that for one measure, the Social Responsiveness Scale, either the participant (if $\geq$ 19 years old) or the participants' parent or guardian provided information. For all other measures, the participant provided information or completed the assessment themselves.

Demographics. Basic demographic information was collected via self-report from the participant. Data collected included age, gender (male, female), race (White, Black/African American, Asian, Multi-racial, Unknown), and ethnicity (Hispanic, Non-Hispanic).

Diagnosis and autism severity. Two instruments were used for confirming ASD diagnosis. The Autism Diagnostic Interview-R (ADI-R) is a widely used standardized interview that aids in distinguishing autism from other developmental disorders in individuals ages two and older (Lord et al., 1994). The ADI-R provides categorical scores for three domains: language/communication, reciprocal social interactions, and repetitive behaviors/interests. The Autism Diagnostic Observation Schedule (ADOS) was also used, which is a standardized test used to diagnose ASD in individuals 12 months and older (Lord, Risi, et al., 2000).

Further, there is an autism severity score as part of the ADOS, which has been demonstrated to be useful to account for differences in verbal development across the various ADOS versions (Shumway et al., 2012). The ADOS severity score, a continuous score, was utilized for Exploratory Aim 2. Lower ADOS severity score represents less severe autism symptoms, while higher ADOS severity scores represents more severe autism symptoms.

IQ. The Wechsler tests are standardized assessments that provide a full-scale IQ (FSIQ) score that represent general intellectual ability (Wechsler, 2014). IQ was determined through one of three Wechsler tests: Wechsler Intelligence Scale for Children-5th Edition (WISC-V) (Wechsler, 2014), Wechsler Adult Intelligence Scale-4th Edition (WAIS-IV), or Wechsler Abbreviated Scale of Intelligence-2nd Edition (WASI-II). The WISC-V was used for individuals younger than 17 years old, while the WAIS-IV or WASI-II was used for individuals $\geq$ 17 years old. Whether the WAIS-IV or WASI-II was used was dependent on which test (if either) had been administered to a participant in a previous HCP study within the past two years. If the participant did not have any Wechsler testing done in the HCP in the previous two years, the WASI-II was administered.

Gross motor ability. The Movement Assessment Battery for Children (M-ABC 2) is a well-known standardized test for detecting movement difficulty in individuals up to age 17, and allows for the measurement of gross and fine motor skills (Henderson et al., 2007). The M-ABC 2 is comprised of three domains: manual dexterity, aiming & catching, and balance. The total score is comprised of the three domains, which represents an individual's overall motor ability. Higher scores represent better motor abilities. The total and domain scores can be translated into age-normative scores. According to the manual's scoring system, the standardized scores can then be converted to percentile ranks and then interpreted in terms of

percentile equivalent: difficulty (at or below the 5th percentile), at risk (between the 6th and 15th percentile), and typical (above the 15th percentile) (Henderson et al., 2007). In a sample of children with developmental coordination disorder, the internal consistency for the M-ABC 2 was $\alpha = 0.90$, and test–retest reliability for the total score was considered excellent, with an intraclass correlation coefficient of 0.97 (Wuang, Su, & Su, 2012). Of note, as some participants in the current study were older than 17 years old, and therefore normative scores were unavailable for these individuals, the raw scores (as opposed to normative scores) were used for analyses.

Social ability. The Social Responsiveness Scale-2nd edition (SRS-2) is a 65-item survey that identifies the presence and severity of social deficits in individuals ages 2.5 years through 18 years old (Constantino et al., 2003). Higher scores indicate higher rates of social impairment. The SRS-2 utilizes a Likert scale of 1 through 4 (1 = not true, 4 = almost always true). In a sample of child psychiatric patients, SRS scores were highly positively associated with ADI scores (0.70), and not related to IQ (Constantino et al., 2003). The SRS-2 provides scores across subscales: social awareness, social cognition, social communication, and social motivation, restricted interests & repetitive behavior, as well as an overall total score. SRS-2 raw scores can be converted to normative scores. A T-score of < 59 is considered within the normal range, 60–75 is considered mild to moderate, and a $T > 75$ indicates severe impairment.

For the current study, two SRS-2 forms were used, the SRS-2 Adult Self Report form and the SRS-2 Parent Report. The SRS-2 Adult Self Report form was given to participants ages 19 years and older, while the SRS-2 Parent form was filled out by a parent or guardian for participant younger than 19 years old. The raw SRS total score is a continuous variable

that was used for analyses (Aims 1 & 3, Exploratory Aim 1). The raw SRS domain scores were used for additional exploratory analyses.

Inhibitory control. Inhibitory control was assessed using the Delis-Kaplan Executive Function System (D-KEFS) Color-Word Interference Test (CWIT), which measures inhibitory control and cognitive flexibility (Delis, 2001). The CWIT is based on the original Stroop test (Stroop, 1935), of which its variations have been widely used since its initial development in 1935. Abnormal Stroop scores have been found to be sensitive to damage in the frontal lobe (Demakis, 2004; Golden, 1976), the brain region considered to play a critical role in executive functioning, including inhibitory control.

In the D-KEFS CWIT, there are a total of four different conditions: color naming, word reading, inhibition, and inhibition/switching. For the current study, performance on the inhibition condition was analyzed. The D-KEFS CWIT inhibition condition is a Stroop task, in which the participant is presented with a page containing the words "red," "green," and "blue" printed *incongruently* in red, green, or blue ink. For example, the word "red" is printed in blue ink. The participant is instructed to say the ink color as quickly as possible without making mistakes (i.e., reading the word instead). The D-KEFS Stroop/CWIT inhibition score is the time in seconds that it takes an individual to finish the task. Using the manual's normative data based on age, the inhibition score was converted into a continuous, standardized subscale score that was used for analyses (Aim 2). Higher D-KEFS Stroop/CWIT scores reflect better inhibitory control abilities.

Gait. Gait was quantified using the OptiTrack R2 system, which is an optical 3D motion analysis system that is currently the largest motion capture provider in the world (Carse, Meadows, Bowers, & Rowe, 2013). OptiTrack consists of a 9-camera setup that

electronically connects to Arena v1.5 software and has the ability to track many different types of subjects' movements. Data were collected at 100 Hz. Each participant wore six reflective markers: three markers were placed on each foot, over the participants' shoes (on the calcanei and the second and fifth distal metatarsals).

The measurement of gait has been done in multiple sclerosis and aging populations (Malcolm et al., 2015; Malcolm et al., 2018). Based on previous studies (De Sanctis et al., 2014; Malcolm et al., 2018), the following quantitative gait markers were chosen which have found to be sensitive measures to characterize gait stability in virtual reality environments: stride time and step width variability. Heel strikes were computed from the heel marker trajectory, using an automated peak-picking function (MATLAB custom scripts) and confirmed by manual inspection, to identify the point where the heel marker was at the most anterior point in the anterior-posterior direction (Kang & Dingwell, 2008). Individual strides were defined as consecutive heel strikes of the same foot. Stride time was defined as the time between consecutive heel strikes of the same foot (Alton, Baldey, Caplan, & Morrissey, 1998). Step width was computed as the lateral distance between the two heel markers at the time of right heel strike (Kang & Dingwell, 2008). The means and standard deviations of each of these measures were calculated over each block of each condition, for every participant. Automatic peak detection software (custom MATLAB scripts) were used to quantify gait parameters, providing a continuous variable that will be used for analyses (Aim 1).

Treadmill stimuli and task. While walking on the treadmill, participants performed a Go/No-Go task. Either an "X" or an "O" was projected onto the middle of the screen in front of them, and participants were instructed to click a wireless computer mouse button with

their right hand each time an “O” appeared (“Go” trials), but to withhold the click if an “X” appeared (“No-Go” trials). Images were presented using Presentation software version 21.1 (Neurobehavioral Systems, Albany, CA, USA) and projected centrally (InFocus XS1 DLP, 1024 x 768 pxl) onto a black wall approximately 1.5 meters in front of the treadmill (LifeFitness TR-9000). The duration of each image was 400 ms with a random stimulus-onset-asynchrony ranging from 200 to 400 ms. On average, images subtended 28° horizontally by 28° vertically. Participants were encouraged to perform the Go/No-Go task quickly and accurately. Each block consisted of 180 trials. The probability of Go and No-Go trials were 80% and 20%, respectively. To become familiar with the protocol, a practice block was provided prior to beginning the experiment. While walking on the treadmill, participants wore a custom-designed safety harness at all times (see Figure 2 in for an illustration of the harness, originally presented in (De Sanctis et al., 2014). Prior to walking on the treadmill, participants determined their preferred treadmill walking speed, and the treadmill was set at this chosen speed for the duration of the treadmill tasks.

The maximum number of blocks was 15, with each block roughly 4 minutes long. The original study included an optical flow condition, where participants encountered periodic visual perturbations consisting of mediolateral fluctuations in optical flow, introducing conflict between visual and proprioceptive information while walking. Experimental conditions were presented in a pseudorandom order: 3 blocks of a Go/No-Go response inhibition task were performed while standing, without optical flow; 6 blocks of the Go/No-Go task were performed while walking on the treadmill (with 3 blocks with optical flow, and 3 blocks without optical flow); and 6 blocks where no task was performed while walking on the treadmill (with 3 blocks with optical flow, and 3 blocks without optical flow).

Notably, for the current study, only the blocks without optical flow were analyzed. See Figure 3 for a summary of the parent study, highlighting the specific conditions that were analyzed in the current study.

EEG and ERP recording. Electroencephalography (EEG) is a brain imaging tool that tracks and records electrical of the brain in the form of brainwaves. EEG from 64 channels was recorded with the BioSemi ActiveTwo system (BioSemi, Amsterdam, the Netherlands), which has been used by many researchers (Bakardjian, Tanaka, & Cichocki, 2011; Praamstra, Boutsen, & Humphreys, 2005). The RAs measured the subjects' head, and the subject was fit with a small, medium, or large 64-electrode EEG cap. Electrode-skin contact was improved with gel that is easily washable with soap and water. The RAs attached the electrode wires onto the cap. This set-up time took roughly 30 minutes. RAs frequently checked in with participants to ensure they were comfortable.

Preprocessing and analysis were performed using custom MATLAB scripts and EEGLAB (Delorme & Makeig, 2004). Continuous raw data were re-referenced to CPz, and filtered from 0.25Hz highpass (filter order 3380) and 40Hz lowpass (filter order 338) calculated with pop_firwsord to remove low frequency drift and high frequency noise. Blocks across conditions (i.e., standing or walking) were appended. Noisy channels were automatically removed by detecting channels with flat lines (>8 seconds), correlation between neighboring channels < 0.4, and values of line noise exceeding signal by eight standard deviations. Additionally, through visual inspection of the data, channels were excluded if artefacts were present over extended periods of time (~50 seconds). Remaining channels were visually inspected for prominent artifacts. Individual subject data were decomposed using an Independent Component Analysis (Jung et al., 2001). Components

identified as eye movement activity were removed, and epochs automatically identified as artifactual based on spectrum thresholding were excluded. Excluded channels were reinserted using a spherical interpolation. Epochs for correct trials were then computed time-locked to the onset of stimulus presentation, with an 800ms post-stimulus period and a 50ms pre-stimulus baseline for Go trials during which the participant successfully responded (Hit trials) and No-Go trials during which the participant successfully withheld a response (Correct Rejection trials [CRs]). Incorrect trials were excluded from the analysis.

N2 and P3. The N2 and P3 ERP components have been well characterized in previous studies, and have demonstrated links with successful response inhibition in Go/No-Go paradigms (Bokura et al., 2001; Donkers & van Boxtel, 2004; Eimer, 1993; Garavan, Ross, Murphy, Roche, & Stein, 2002; Katz et al., 2010; Morie et al., 2014). As N2/P3 have been shown to produce greatest amplitudes over fronto-central scalp sites, the midline site of electrode FCz was selected to represent the N2/P3 components.

For N2, the parent study analyzed N2 peak-to-peak amplitude scores, as opposed to amplitude due to a high prevalence of negative numbers. Therefore, the N2 peak-to-peak amplitude was utilized for the current study analyses as well. For P3, the amplitude was used, which was measured by calculating the average amplitude across the corresponding time period of interest. This method has been deemed to provide a more comprehensive account of the component across the entire time window (Luck, 2004).

MATLAB custom scripts output N2 and P3 amplitudes as continuous variables, which were used for analyses (Aim 3, Exploratory Aim 2). Of note, a full-time technician employed by AECOM CNL performed the majority of the MATLAB analyses. For the

present study, the relevant data was extracted from the AECOM dataset and subsequently used to perform the SPSS analyses for the current study.

See Figure 4 for a summary schematic of the constructs examined, along with the measures utilized to assess the constructs.

Ethics

The procedures and assessments for this proposal involved minimal risk to participants. Procedures were in place to deal with medical, stress and psychiatric issues that may have arisen in the course of the evaluations (i.e., RAs would reach out to administration and/or PIs for further guidance). Risks associated with gait assessments and the MoBI protocol are similar to those involved in everyday standing, walking, maintenance of balance and activities of daily living. Participants may have experienced fatigue while walking. RAs closely monitored participants for signs of weariness, loss of balance or other physical discomforts, in which case they immediately initiated a break or ceased testing as needed.

EEG recordings used approved materials and equipment. No physical, social, or legal consequences stemming from study participation were expected. This study entailed the recording of EEG from the scalp employing standard sensors and amplification methods, a procedure that is well standardized and in use clinically. Participants may have become somewhat bored, irritated, or frustrated with the repetitiveness of the tasks. Anxiety about the testing procedures and recording procedures were assessed during the informed consent procedures and throughout the experiment, and addressed if need be. The emotional state of subjects was closely monitored by research staff, and they were reminded of their right to withdraw at any time with no negative consequences. The recording equipment used in these studies meets the current design criteria for subject safety, including isolation from potential

electrical hazards. Regular preventative maintenance and careful attention to recording procedures further minimize the already insignificant risk of electrophysiological recording. The visual stimuli were carefully calibrated and were presented at a level that prevents the occurrence of peripheral or central trauma due to excessive intensity.

Participants were assigned code numbers to maintain confidentiality. Lists of codes were kept in a locked file cabinet and were maintained separately from coded materials. Consent documents, which were the only identifying documents, were kept in a locked file cabinet accessible only to research staff away from all other materials. All computer files identify subjects with a code. No names or other identifying information are kept in computer files. Electrodes, caps, furniture, experimental response devices, and anything else participants came in contact were disinfected after each use.

Data Analyses

The sample size of n = 18 TD and n = 20 ASD was determined by the parent study, based on similarly designed studies that were able to detect significant effects (Malcolm et al., 2015). MATLAB was used to calculate specified variables, and IBM SPSS Version 27 was used to run all analyses (**IBM**, 2020).

Analyses:

Aim 1: To investigate whether motor ability is associated with social ability in ASD and TD individuals.

Gross motor ability was measured using the M-ABC 2 total raw score, a continuous score. Gait variability was measured using the OptiTrack system, and calculated using custom MATLAB scripts. The scores for gait stride time and step width variabilities were continuous measures. Of note, for the gait variability scores (i.e., stride time and step width

variabilities), performance during the *static (no optical flow), no-task (not performing the Go/No-Go Task)* conditions were utilized, hence any effect of optical flow and performing the inhibitory control task would not be factors. Of note, one participant's scores for gait stride time variability was found to be an outlier and therefore excluded. Social ability was represented by the SRS total score, a continuous score.

For each part of Aim 1, to assess the relationship between group and the type of motor ability, a categorical by continuous interaction in linear regression was performed. An interaction term was created, with group by the motor variable (i.e., gross motor ability, gait step width variability, and gait step width variability). Group was the categorical variable, with TD coded as "0", and ASD as "1" (i.e., ASD was the "dummy" variable). For each motor score, the score was mean centered in order for the average to be zero. This was computed by subtracting each score from the mean score. Further, to control for IQ and age, FSIQ and age were introduced as covariates. The motor ability variables were the independent variables (i.e., gross motor ability, gait step width variability, and gait step width variability), and social responsiveness was the dependent variable.

1a. The relationship between gross motor ability and social ability was tested using a linear regression analysis.

1b. The relationship between gait stride time variability and social ability was tested using a linear regression analysis.

1c. The relationship between gait step width variability and social ability was tested using a linear regression analysis.

Of note, when checking assumptions for Aim 1a, gross motor ability (M-ABC 2) was found to be significantly positively associated with IQ. Therefore, the decision was made to

remove IQ from the model for Aim 1a. All other assumptions for Aim1a were met. Regarding Aims 1b and 1c, assumptions for the use of linear regression analyses were met (i.e., independence of observations; linear relationships between dependent and independent variables; normally distributed residuals; no multicollinearity between independent variables; and homoscedasticity of residuals).

Aim 2: Due to methodological limitations (i.e., sample sizes too small in certain groups), the original aim was adjusted (see below for further information).

Original Aim - To determine whether there was an effect of ASD diagnosis and motor ability on inhibitory control.

Adjusted Aim - To examine whether there was a difference in inhibitory control based on motor ability, across TD and ASD groups.

Originally, a two-way ANOVA was planned to examine the effect of gross motor ability and diagnosis on inhibitory control. However, upon examination of the data, it was realized that within the TD group, no participants performed in the at-risk range (6^{th}-15^{th} percentile) based on the M-ABC 2 total score. For the ASD group, only one participant performed in the at-risk range, and one participant performed in the typical range (>15^{th} percentile). Given the limited sample sizes within the TD (at-risk) and ASD (at-risk and typical) groups for the M-ABC 2 measure, a valid two-way ANOVA could not be performed. Based on methodological limitations and assumptions that need to be met to use ANOVA and other statistical analyses, the original Aim 2 could not be examined with the current sample.

Therefore, the adjusted aim was to examine whether there was a difference in inhibitory control based on motor ability, across the TD and ASD groups. For both TD and

ASD groups, the at-risk and typical M-ABC 2 scores were combined into one group: at-risk/typical. Participants were then categorized into one of two M-ABC 2 groups: 1) at-risk /typical ($\geq 6^{th}$ percentile); or 2) difficulty ($\leq 5^{th}$ percentile). A Mann Whitney U test was run to examine the adjusted aim of whether there was an effect of motor ability on inhibitory control (Stroop/CWIT). Of note, a Mann Whitney U was selected rather than independent samples t-test, due to the small sample sizes of groups and concerns with non-normal distributions.

For the adjusted aim, the independent variable was gross motor ability (as assessed by the M-ABC 2 categorized score). The dependent variable was the Stroop/CWIT inhibition score, a continuous score. The standardized Stroop/CWIT scores were used for analyses. The four assumptions for use of a Mann Whitney U test were met (i.e., continuous dependent variable, independent variable is categorical with two groups, independence of observations, and the distribution of scores for both M-ABC 2 groups had approximately similar shapes for Stroop/CWIT).

Lastly, a Fisher's exact test was run to test for whether there was an association between diagnostic group (ASD vs. TD) and motor group (at risk/typical motor ability, or motor difficulty).

Aim 3: To examine neural substrates of inhibitory control while walking and standing, in relation to social ability in ASD.

Neural indices of N2 and P3 were quantified by measuring the amplitude evoked from the correct rejection (No-Go) inhibition task performed while walking, and another for standing (see Measures). The amplitude from the fronto-central electrode site FCz was examined. Bivariate correlations were run to assess neural indices for standing and walking,

for N2 as well as for P3. As the assumption for linearity was not met to use Pearson's correlations, Spearman's rank order correlations were run instead.

Additionally, exploratory analyses using paired samples t-tests were conducted to examine whether there was a significant difference in N2/P3 amplitude when standing versus walking for the ASD and TD groups, separately.

Exploratory Aim 1: To examine whether specific motor domains (aiming and catching, balance, and motor dexterity) are related to social ability among ASD and TD groups.

The M-ABC 2 domain scores were used to measure aiming and catching, balance, and motor dexterity. The relationship between each motor domain and social responsiveness were analyzed using three correlation analyses. When examining the assumptions for the use of Pearson's correlations, the assumption of linearity was not met for manual dexterity within the ASD group; therefore, Spearman's correlations was used to assess manual dexterity in ASD. All other correlations were Pearson's correlations.

Exploratory Aim 2: To examine neural substrates of inhibitory control while walking and standing, in relation to autism severity level in ASD.

Similar to Aim 3 (above), neural indices of N2 and P3 were quantified with the amplitude evoked from the inhibition task performed while walking and standing. The central electrode site FCz was examined. The relationships between neural indices and autism severity levels were tested using Spearman's rank correlation analyses, given that the assumption of linearity was not met for the use of Pearson's correlations.

Additional Analyses: To examine relationships among motor ability, social ability, autism severity, and neurocognition across the full sample of ASD and TD individuals.

The overall goal of the current study was to better understand the relationships amongst motor ability, social ability, neurocognition, and underlying neural indices. Therefore, to further supplement the findings, exploratory correlational analyses were conducted using the combined sample of ASD and TD individuals, thereby allowing for more statistical power to examine constructs irrespective of diagnoses. Additional rationale for these added analyses was for replication purposes, as replication of previous study findings is a vital way to bring confidence to scientific results.

Spearman's rank correlations were run, given the high number of variables being examined and relatively small sample size. (Of note, Pearson's correlations were considered to run instead of Spearman's, and final analyses demonstrated that results of both types of correlations were overall similar. See Appendix A for results of Pearson's correlations.) The variables examined were: motor (stride time variability, step width variability, M-ABC 2 total and the three domains of aiming & catching, balance, and manual dexterity); clinical (ADOS severity score, SRS-2 total, and the domains of social awareness, social cognition, social communication, and social motivation); and neurocognitive (DKEFS Stroop/CWIT inhibition, N2 while standing and walking, and P3 while standing and walking), across the full sample of ASD and TD individuals.

Chapter III: Results

Sample Recruitment

As mentioned, the current study is a secondary analysis based on a primary, parental study at the Albert Einstein College of Medicine. Data were analyzed from the same dataset, which consisted of 38 participants, with 18 ASD individuals and 20 TD individuals.

Sample Characteristics

Table 1 presents the demographic, cognitive, and clinical information for study participants broken down by ASD and TD classification. ASD and TD individuals were similar in age, gender, handedness, race, and ethnicity (*p*s > .05). Despite all ASD participants having to meet an inclusion criterion regarding IQ, full-scale IQ scores were significantly higher for TD individuals compared to ASD individuals ($U = 51.5$, $p < .001$). The same pattern of TD individuals performing better was seen within the domains that comprise the FSIQ, which are verbal abilities ($U = 70.5$, $p = .006$) and perceptual abilities ($U = 81.5$, $p = .018$). TD individuals also performed better on the Stroop/color word-interference task, relative to ASD individuals ($U = 34.0$, $p = .009$).

Regarding neural ERP indices, only P3 (during the standing task) was found to be statistically different between the two groups (see Table 1). More specifically, TD individuals exhibited higher P3 amplitude relative to ASD individuals, ($U = 101.0$, $p = .020$). As for social deficits, ASD individuals demonstrated greater social deficits relative to TD individuals ($U = 184.0$, $p < .001$), as expected given the criteria for a diagnosis of ASD.

In terms of gross motor ability based on the M-ABC 2, TD individuals exhibited better performance than ASD individuals on all three domains (aiming and catching, balance, manual dexterity) as well as the total score (see Table 1). Regarding gait parameters, both stride time variability and step width variability (during the no flow, no task condition) were statistically different between the two groups. That is, ASD individuals exhibited greater stride time variability as well as step width variability, relative to TD individuals.

Aim 1: Motor Ability and Social Ability

See Table 2 for information regarding the results and analyses Aims 1a, 1b, and 1c.

Aim 1a. A multiple regression was run to assess the relationship between gross motor ability (as assessed using the M-ABC 2), group (ASD versus TD), and the interaction between gross motor ability and group on social responsiveness, while accounting for age. Results of the analysis revealed that the interaction term (gross motor ability x group) was not significant (t = -1.42, p = .17), indicating that data did not support the hypothesis that greater gross motor impairment would be associated with poorer social responsiveness for the ASD group, but not the TD group.

There was a collective significant effect of the model, ($F(4,18) = 6.84$, $p = 0.002$, $R^2 = 0.60$). The individual predictors were examined further, and group was found to be a significant predictor in the model (t = 3.53, p = .002). In other words, the ASD group was significantly predicted to have worse social ability than the TD group, which is unsurprising given ASD diagnostic criteria. Gross motor ability, the interaction between gross motor ability and group and age were not significant predictors of the model ($ps > .05$).

Aim 1b. A multiple regression was run to test the association between motor ability (as assessed using stride time variability), group (ASD versus TD), and the interaction

between stride time variability and group on social responsiveness, while accounting for age and FSIQ. Results of the analysis revealed that the interaction term (stride time variability x group) was not significant (t = -0.59, p = .56), indicating that data did not support the hypothesis that greater gait stride time variability would be associated with poorer social responsiveness for the ASD group, but not the TD group.

There was a collective significant effect of the linear regression, (F(5,21) = 6.69, R^2 = 0.61, p < .001). The individual predictors were examined further, and group was found to be a significant predictor in the model (t = 3.42, p = .003). That is, the ASD group was significantly predicted to have worse social ability relative to the TD group, as expected given diagnostic criteria for ASD. Stride time variability, the interaction between stride time variability and group, age, and FSIQ were not significant predictors of the model (ps > .05).

Aim 1c. A multiple regression was run to test the association between motor ability (as assessed using step width variability), group (ASD versus TD), and the interaction between the step width variability and group on social responsiveness, while accounting for age and FSIQ. Results of the analysis revealed that the interaction term (step width variability and group) was not significant (t = 0.51, p = .61), indicating that data did not support the hypothesis that greater gait step width variability is associated with poorer social responsiveness for the ASD group, but not the TD group.

There was a collective significant effect of the linear regression, (F(5,21) = 7.11, p < .001, R^2 = 0.63). The individual predictors were examined further, and indicated that group (t = 4.82, p < .001) was a significant predictor in the model. Similar to Aims 1a and 1b, the ASD group was significantly predicted to have worse social ability relative to the TD group.

Step width variability, the interaction between step width variability and group, age, and FSIQ were not significant predictors of the model (*ps* > .05).

In summary, based on the results of the regression analyses of Aims 1a, 1b, and 1c, motor ability variables (i.e., gross motor ability, stride time variability, step width variability) were not significant predictors of social responsiveness.

Aim 2: ASD Diagnosis and Motor Ability on Inhibitory Control

A Mann Whitney U test was run to examine whether there was a difference in inhibitory control (as assessed by the Stroop/CWIT) based on motor ability (either at risk/typical motor performance, or difficulty with performance based on M-ABC 2), across TD and ASD groups. Contrary to the prediction, inhibitory control was not statistically different between individuals with at/risk typical motor performance (median = 9.50) and individuals who had difficulty with motor performance (median = 10.0; $U = 63.0$, $p = .87$) (see Table 3). A Spearman's correlation was also run to examine whether inhibitory control (as assessed by the Stroop/CWIT) was significantly associated with motor ability (based on the continuous total M-ABC 2 score) across the full sample of TD and ASD individuals. Results indicated that inhibitory control and motor ability were not significantly associated, ($r_s(20) = .22$, $p = .32$).

Additionally, a Fisher's exact test was run to for association between diagnostic group (ASD vs. TD) and motor group (at risk/typical motor ability, or motor difficulty; see Figure 5). There was a statistically significant association between diagnostic group and motor group based on Fisher's test, $p < .001$. That is, for ASD individuals, 14 (87.5%) met criteria for having motor difficulties, while only 2 (12.5%) met criteria for at risk/typical motor performance, based on the M-ABC 2 total score. For TD individuals, the opposite

profile was depicted. More specifically, 12 (85.7%) TD individuals met criteria for at risk/typical motor performance, while only 2 (14.3%) TD individuals met criteria for having motor difficulties, based on the M-ABC 2 total score. Overall, data indicated that ASD individuals were more likely than TD individuals to have motor difficulties, while TD individuals were more likely than ASD individuals to have at risk/typical motor performance.

Aim 3: N2/P3 and Social Ability

See Table 4 for more information regarding analyses for Aim 3. Spearman's rho correlations were run to assess relationships between neural indices of inhibitory control (N2/P3) while walking and standing, in relation to social responsiveness in ASD (as assessed by SRS-2) (see Table 4). N2/P3 were elicited during the Go/No-Go task, and in regard to this aim, only the No-Go correct rejections were examined.

Regarding N2, contrary to the hypothesis that increased social deficits would be positively associated with reduced (i.e., more positive) N2 amplitude in ASD, no statistically significant relationships were found between N2 and social responsiveness in either the standing or the walking condition (ps > .05). Rather, although not significant, moderate negative correlations (i.e., in the -.30 range) were found between N2 and social responsiveness, while walking for both the ASD ($r_s(15) = -.33$, $p = .19$) and TD groups ($r_s(9) = -.30$, $p = .37$). This finding represents a trend that, while walking, greater social deficits are associated with greater (i.e., more negative amplitude) N2 neural processing for both ASD and TD individuals. In regard to N2 while standing, no relationship was found between social ability and N2 amplitude for ASD. However, for TD while standing, a moderate negative correlation was found between N2 and social responsiveness ($r_s(9) = -.46$, $p = .15$), indicating that greater social deficits are associated with greater (i.e., more negative amplitude) N2

neural processing when standing. Of note are the limited sample sizes particularly for the TD group.

Regarding P3, it had been hypothesized that increased social deficits would be negatively associated with reduced (i.e., more negative) P3 amplitude for the ASD group. While no significant relationships were found between P3 amplitude and social responsiveness in either the standing or the walking condition (*p*s > .05), the trends were in the predicted direction for the ASD group. That is, although not significant, moderate negative correlations were found, indicating greater social deficits were related to reduced (i.e., more negative amplitude) P3 neural processing for ASD, while both walking ($r_s(15) = -.38$, $p = .13$) and standing ($r_s(15) = -.34$, $p = .18$). In terms of P3 within the TD group, no significant associations or trends were found between P3 and social ability while walking or standing (*ps* > .05).

The correlational analyses revealed several statistically significant, additional findings (see Table 4). Within ASD individuals: N2 while standing was significantly positively associated with N2 while walking; P3 while walking was significantly positively associated with N2 while walking; and P3 while standing was significantly positively associated with P3 while walking. Within TD individuals: N2 while standing was significantly positively associated with N2 while walking; and P3 while standing was significantly positively associated with P3 while walking for TD individuals. Additionally, within TD, a moderate negative correlation was observed for P3 while standing with P3 while walking.

Notably, upon further examination of the data, it was observed that for TD individuals, neural data from while walking and while standing appeared to be different,

while data from while walking and while standing appeared to be similar for ASD. Therefore, additional exploratory analyses were conducted to examine whether there was a significant difference in N2/P3 amplitude when walking versus standing for the ASD and TD groups, separately (see Table 5 and Figure 6). For TD individuals, N2 while walking elicited a significantly reduced amplitude compared to N2 elicited while standing, (M = 1.61, 95% CI [0.24, 2.98], $t(17) = 2.47$, $p = .024$), reflecting increased neural processing while standing relative to walking. For the ASD group on the other hand, there was no significant difference in N2 when walking versus standing ($p > .05$).

Regarding P3, the same pattern was depicted. That is, for the TD group, P3 while walking elicited a significantly reduced amplitude compared to P3 elicited while standing, (M = -1.94, 95% CI [-3.41, -0.467], $t(17) = -2.78$, $p = .013$). In other words, similar to N2, for TD individuals increased neural processing was utilized when standing relative to walking. For the ASD group, there was no significant difference in P3 when walking versus standing ($p > .05$).

In summary, in regard to N2, a trend demonstrated that while walking, greater social deficits were associated with greater N2 neural processing for both ASD and TD individuals. For TD individuals, a trend also demonstrated that greater social deficits were associated with greater N2 neural processing when standing; this was not observed with ASD individuals. In regard to P3, for ASD individuals, a trend demonstrated that greater social deficits were related to reduced P3 neural processing for ASD, while both walking and standing. For TD individuals, no significant associations or trends were found between P3 and social ability while walking or standing. Lastly, additional exploratory analyses revealed that TD individuals exhibited greater N2/P3 neural processing when standing relative to

walking; for ASD individuals, N2/P3 neural processing was similar when standing and walking.

Exploratory Aim 1: Motor Domains and Social Ability

Bivariate correlations were used to assess the relationships between the three domains of gross motor ability (M-ABC 2 manual dexterity, aiming & catching, and balance) and social ability (SRS-2) of ASD and TD individuals, separately. See Table 6 for information regarding analyses for Exploratory Aim 1.

Within ASD, no significant relationships were found between social ability and any of the three motor domains of manual dexterity, aiming & catching, and balance (ps > .05). However, although not significant, a moderate negative correlation was found between manual dexterity and social ability, ($r_s(12) = -.42$, $p = .14$) in ASD. In other words, for ASD individuals, a greater number of social deficits was associated with worse manual dexterity. Interestingly, a significant and opposite pattern was observed in the TD group; social ability was significantly positively associated with manual dexterity, ($r(9) = .70$, $p = .02$). That is, for TD individuals, a greater number of social deficits was associated with better manual dexterity. See Figure 7 for a visual representation.

Exploratory Aim 2: N2/P3 and Autism Severity

Spearman's rho correlations were used to assess the relationships between neural substrates of inhibitory control (N2/P3) while walking and standing, in relation to autism severity level (as assessed by the ADOS severity scores) in ASD. See Table 7 for information regarding analyses for Exploratory Aim 2.

P3 while standing was significantly negatively correlated with autism severity level, ($r_s(10) = -.70$, $p = .011$). In other words, reduced P3 response (i.e., more negative amplitude)

was associated with more severe autism symptoms while standing in ASD individuals. P3 was not associated with autism severity level while walking ($p > .05$). N2 was not associated with autism severity level while walking or standing ($ps > 0.05$).

The correlational analyses revealed several statistically significant, tangential findings (see Table 7). N2 while standing was positively associated with N2 while walking, and P3 while standing was positively associated with P3 while walking, in ASD ($ps < .01$).

Additional Analyses: Motor, Social, and Neurocognition

Spearman's correlations were run to examine relationships among the motor (stride time variability, step width variability, M-ABC 2 total and the three domains of aiming & catching, balance, and manual dexterity); social (SRS-2 total, and the domains of social awareness, social cognition, social communication, and social motivation); and neurocognitive (DKEFS Stroop/CWIT inhibition, N2 while standing and walking, and P3 while standing and walking) variables, across the full sample. For the ASD group, ADOS severity was examined as well. The complete results are listed in Table 8, and select results are described below. As previously mentioned, the results of Pearson's correlations were overall similar, and are provided in Appendix A.

In terms of gait, stride time variability was positively associated with social ability (SRS-2 total), particularly with social cognition (SRS-2 social cognition). In other words, greater stride time variability was related to greater social deficits, particularly poorer social cognition. Step width variability was negatively associated with gross motor ability (M-ABC 2 total); that is, greater step width variability was related to worse motor ability. Step width variability was also negatively associated with P3 amplitude while walking as well as standing; or, greater step width variability was related to decreased P3 neural processing.

Regarding motor ability (as assessed with the M-ABC 2), although motor ability was not a significant predictor of social ability in the regression models described above for Aim 1a, exploratory correlational analyses found that motor ability was significantly correlated with several social and clinical measures. Motor ability (M-ABC 2 total) was negatively correlated with social ability (SRS-2 total) as well as autism severity (ADOS severity). That is, better motor ability was related to fewer social deficits as well as lower autism severity. Of note, results for the discrepancy between the regression and correlation analyses may be due to group effects (i.e., differences in data by group) being accounted for within the regression but not in the correlation analyses. Motor ability (M-ABC 2 total) was also positively correlated with P3 while standing; or, better motor ability was related to increased P3 neural processing while standing.

In regard to the three motor domains (M-ABC 2 domains), aiming and catching was not associated with any social and clinical measures, while balance and manual dexterity were. More specifically, balance was negatively correlated with social ability (SRS-2 total), particularly social cognition and social communication. In other words, better balance was related to better social cognition and social communication. Manual dexterity was negatively correlated with autism severity; or, better manual dexterity was related to decreased autism severity. Manual dexterity was also positively correlated with P3 amplitude while standing; or, better manual dexterity was related to increased P3 neural processing while standing.

In terms of neurocognition, inhibition (Stroop/CWIT) was negatively correlated with social ability (SRS-2 total). That is, better inhibitory control was related to fewer social deficits. Regarding neural processing, P3 while standing was negatively correlated with autism severity; or, increased P3 neural processing while standing was related to decreased

autism severity. A trend demonstrated that P3 while standing was positively correlated with inhibition (Stroop/CWIT); or, increased P3 neural processing while standing was related to better inhibitory control. N2 while walking was negatively correlated with inhibition (Stroop/CWIT); or, increased N2 neural processing (i.e., more negative) while walking was related to better inhibitory control.

Chapter IV: Discussion

Summary

Individuals with ASD exhibit high prevalence of motor abnormalities in addition to social deficits, and there have been increasing theories regarding the interwoven relationship between motor and social development in ASD. The current study examined the relationships among motor, social, and cognitive abilities in ASD, with the integration of neurophysiological data and neuropsychological data with standardized movement and clinical measures. The study was the first known to investigate social ability in relation to neural processes while walking and standing in ASD versus TD using an EEG MoBI paradigm. Overall, the present study investigated critical relationships between motor ability, social ability, inhibition, and underlying neural indices.

Aim 1: Motor Ability and Social Ability

The first aim of the study was to investigate whether various forms of motor ability were associated with social ability in ASD and TD individuals. The three types of motor ability examined were: gross motor ability (as assessed with the M-ABC 2), stride time variability, and step width variability.

Based on the regression analyses of Aim 1a, gross motor ability was not significantly associated with social responsiveness, in either the ASD or TD group. Although, it should be noted that there were significant relationships when exploratory correlational analyses were conducted using the full sample; that is, better gross motor ability was related to lower autism severity as well as fewer social deficits, particularly in the social cognitive domain. There are

several potential explanations for the perceived discrepancy between the regression and correlation analyses. For instance, there may be group effects that are accounted for within the regression but not in the correlation analyses. Additionally, there are inherent differences in regression and correlation. Based on the results, gross motor ability was unable to predict social ability (regression), however when directionality of the variables is removed, gross motor ability and social ability have a relationship (correlation).

In regard to similar previous research, Dyck and colleagues (2007) found that worse motor ability was related to higher severity of social impairment in ASD, which aligns with the current study's correlation analyses, but not with the regression analyses. There were several methodological differences between the current study and the Dyck (2007) study. For instance, Dyck (2007) measured motor ability with the McCarron Assessment of Neuromuscular Development, rather than the M-ABC 2 (Dyck, 2007). The McCarron Assessment measures fine motor skills and heel-to-toe walking, similar to the M-ABC 2, however it does not incorporate aiming and catching, nor balance, which the M-ABC 2 incorporates. Similarly, social ability in the Dyck (2007) study was measured using a combination of several measures, none of which included the SRS-2. Additionally, the age range of participants in the Dyck (2007) sample was younger, with children ages 4 through 13 years old, indicating potential developmental differences of individuals in the Dyck (2007) versus the current study.

Similar to Aim 1a, based on the regression models, neither stride time variability or step width variability (Aims 1a and 1b, respectively) were associated with social ability in either the ASD or TD group. However, based on the exploratory correlational analyses using the full sample, increased stride time variability was associated with increased social deficits,

particularly in the social cognitive domain. Step width variability, on the other hand, was not associated with any of the social domains. Differences in stride time variability and step width variability are not unexpected, given that based on research on older adults, the two constructs are not homogenous, with sensory impairment being related to step width variability and central nervous system dysfunction potentially more related to stance time variability (Brach, Studenski, Perera, VanSwearingen, & Newman, 2008).

Only one known study specifically examined gait variability parameters in relation to social ability in ASD. Gong and colleagues (2020) found a significant association between gait abnormalities and social ability among ASD individuals, such that increased gait variability was related to greater autism severity, or increased social deficits. Of note, Gong et al. (2020) measured gait parameters differently than the current study (i.e., gait asymmetry, step-to-step variance), and also utilized different technology (i.e., plantar pressure mat from RsScan, Inc) that did not incorporate 3D kinematic measurements. As of yet, there is no standardized way to measure aspects of gait. Another caveat is that the age range of participants in the Gong et al. (2020) study was four through seven years old, thus considerably younger than the current study's participants who were ages 13 through 25. Though, similar to the current study, Gong et al. (2020) used the SRS to measure social ability.

Future research on motor and gait abilities in relation to social ability in ASD can be improved in several ways. For instance, most of the studies that have examined motor and gait abilities in ASD largely included high functioning individuals. While it is generally challenging to recruit low functioning ASD individuals given the complex requirements of the study tasks, future studies may consider simpler methods and design that allow low

functioning ASD individuals to participate, which would further enrich the knowledge base. Additionally, future research may also shed more light on potential differences in stride time variability and step width variability in ASD.

Aim 2: ASD Diagnosis and Motor Ability on Inhibitory Control

The second aim was to determine whether there was an effect of ASD diagnosis (ASD versus TD) and motor ability on inhibitory control. Originally, it was hypothesized that there would be an interaction between ASD diagnosis and motor ability on inhibitory control (i.e., ASD individuals with low motor ability would demonstrate poorer inhibitory control, compared to ASD individuals with high motor control, TD individuals with low motor ability, and TD individuals with high motor ability). Unfortunately, due to small sizes and methodological limitations, the original statistical analyses could not be performed. Therefore, the sample was pooled and analyses were run to examine whether there was a difference in inhibition (Stroop/CWIT) based on motor ability (at risk/typical motor performance, or difficulty based on M-ABC 2). Data did not demonstrate differences in inhibition based on motor ability. Further, inhibition and motor ability were not associated. There was an association between diagnostic group and motor group. That is, ASD individuals were more likely to have motor difficulties relative to TD, which aligns well with existing research (Fournier et al., 2010).

Regarding inhibition, previous studies have demonstrated that inhibition appears to be an area of difficulty for ASD individuals (Corbett et al., 2009; Robinson et al., 2009). Further, errors in inhibition have been linked to the core ASD symptom of repetitive and stereotyped behaviors (Adams & Jarrold, 2009; Mosconi et al., 2009). To the author's knowledge, the current study is the first to examine relationships between inhibition, ASD

diagnosis, and motor ability. While the hypothesis of a relationship between inhibition and motor ability was not supported by the data, given the high prevalence in motor abnormality and inhibitory errors in ASD, future research should consider examining other ways to assess motor and inhibition within ASD. Part of the rationale is due to many inhibition tasks that inherently involve motor ability. As stated by Pratt and colleagues (2014), as inhibition is frequently examined with tasks that involve button presses or other motor methods, it is critical to assess the extent to which any difficulties are associated with producing the responses, affects inhibition. Though, in one study of children with development coordination disorder, motor load did not affect performance on the Stroop task (Pratt, Leonard, Adeyinka, & Hill, 2014).

Related to Aim 2, additional exploratory analyses revealed associations between inhibition (as assessed with Stroop/CWIT) with social ability and autism severity. Better inhibitory control was related to better social ability, which is consistent with previous research. For instance, inhibition is closely related to social cognitive abilities especially in school-age children, such that inhibiting and regulating one's behavior allows children to focus more on what teachers' and parents' instructions (Anderson, 2002). Additionally, findings on theory of mind (understanding others' mental states), mirror neuron system (neural circuit of interconnected brain regions that process perceiving and executing motor actions), and imitative behavior based on cognitive psychology and neuroscience research is also relevant (Baron-Cohen, 1991; Keysers & Gazzola, 2010). Spengler and colleagues (2010) examined the relationship between the inhibition of motor behaviors and social cognitive tasks (i.e., perspective-taking) in 28 patients with focal lesions (e.g., from traumatic brain injury, tumors). In the imitation-inhibition task, participants lifted a finger in response

to seeing numbers on a screen, while watching videos of other individuals perform the hand sequences. In the congruent task, participants lifted the corresponding finger; in the incongruent task, they lifted the non-corresponding finger (i.e., inhibit the motor behavior). A computerized version of the Stroop was also administered to measure inhibition.

Spengler and colleagues (2010) found that, at both the functional and neural levels, the inhibition of motor behavior overlapped with social cognitive abilities (Spengler, von Cramon, & Brass, 2010). The functions were associated with each other, and imaging demonstrated that areas of interest were frontal and tempero-parietal-junctions brain areas. Interestingly, they did not find any associations with inhibition from the Stroop. This may be because of clinical and social baseline differences in the current study and Spengler et al. (2010). The current study included individuals with ASD with inherently higher social deficits compared to the Spengler study. Additionally, imitation-inhibition and inhibitory control are separate entities, with the former likely having more overlap with ASD symptomology and social cognition, relative to inhibition. There may also be developmental effects, in that, as stated previously, inhibition appears to be tied to social cognition in early development relative to when individuals are older. Thus, future research should control for developmental factors such as age that may play a role in the relationship between inhibition, social deficits, and autism severity.

Aim 3: N2/P3 and Social Ability

The third aim of the study was to examine relationships between neural indices of inhibitory control (N2/P3) while walking and standing in relation to social ability in ASD and TD (as assessed by the SRS-2). Contrary to predictions, no significant relationships were found between N2/P3 in relation to social ability in ASD. Further, the relationship between

N2 and social ability was in the opposite direction than predicted. That is, for N2 in ASD, while not significant, a trend demonstrated that greater social deficits were related to greater N2 neural processing (i.e., more negative amplitude) while walking, but not while standing. For TD, greater social deficits were significantly related to greater N2 neural processing, while both walking and standing. Regarding P3 in ASD, while not significant, the relationship with social ability was in the predicted direction. That is, a trend demonstrated greater social deficits were related to reduced P3 neural processing for walking and standing. For TD, no trends between P3 and social ability while walking or standing were found. In additional exploratory analyses, increased P3 neural processing while standing was related to lower autism severity (based on ADOS). Taken together, the data only partially supported hypotheses that the relationship between social ability and N2/P3 processing would differ between ASD and TD groups, and there was more support for P3.

There are several potential explanations for why the findings did not fully support predictions. For one, the literature is mixed regarding what greater N2 amplitude represents (Donkers & van Boxtel, 2004; Geraldo et al., 2019; Smith et al., 2007). That is, there is debate on whether greater N2 amplitude elicited from inhibition tasks reflects more inefficiency, or whether larger N2 represents having better inhibitory control. Studies on clinical populations have varied, such that children with ADHD exhibited larger N2 amplitudes, while reduced N2 amplitudes were observed in individuals with bipolar disorder (Morsel et al., 2017), relative to TD individuals. Of note, additional exploratory analyses of the current study revealed that greater N2 neural processing (i.e., more negative amplitude) was associated with better performance on the Stroop/CWIT task across the full sample. This suggests that, at least in the current sample, greater N2 amplitude (i.e., more negative) was

related to better inhibitory control. Regarding P3, though some clinical findings are also mixed, there is general consensus that larger P3 amplitude is related to the effective recruitment of cognitive resources, or better cognitive abilities (Geraldo et al., 2019; Larson, 2011). Aligning with the latter notion, additional exploratory analyses revealed that greater P3 neural processing was related to better inhibitory ability on the Stroop/CWIT task across the full sample.

Additionally, results may differ from previous literature because the current study only examined N2/P3 response in one electrode site, FCz, which is a mid-frontal cortical area. It is possible that examination of additional sites would yield diverse results. For instance, for N2, a previous study that examined healthy subjects found that correct rejection No-Go source activity was lateralized more towards the right hemisphere (Bokura et al., 2001). In the same study by Bokura et al. (2001), for P3, correct rejection No-Go activity was localized to the left orbitofrontal cortex. Of note, Bokura et al. (2001) examined only healthy subjects who were older than the present sample, with a mean age of 32 years (range of 23 to 42 years). In another study, siblings of individuals with autism displayed a reduced (less negative) N2 amplitude and shorter N2 latency, which was more clear at site Fz relative to FCz (Mohapatra, 2011). They did not observe reduced N2 amplitude and shorter N2 latency in siblings of individuals who did not have autism. Taking the two study findings together, it is possible that examining additional electrode sites may have yielded diverse results regarding the association between N2/P3 and social ability.

Cox and colleagues (2015) found that reduced P3 amplitude was associated with higher expression of autism traits as measured by the SRS, when P3 was elicited by social rewards. Individuals with high autistic traits demonstrated attenuated P3 amplitude to social

incentives relative to individuals with low levels of autistic (Cox et al., 2015). Notable here is that P3 was specifically elicited by social feedback (i.e., receiving video feedback about one's performance), whereas in the current study, P3 was elicited by performing an inhibition Go/No-Go task. The contrasting findings may thus be due to divergence in the methods of eliciting P3. P3 is a complex brain component that provides information about an array of cognitive and mental functions, that is triggered by an individual's reaction.

Interestingly, further analyses revealed that in the TD group, neural differences in N2/P3 were observed in standing versus walking. More specifically, for both N2 and P3, greater amplitude was observed while standing relative to walking in TD. Greater N2 amplitude was seen while standing than in walking; greater P3 amplitude was seen while standing than in walking. For ASD, on the other hand, N2/P3 responses were similar while standing and walking. The pattern is somewhat similar to previous research on multiple sclerosis. De Sanctis et al. (2020) utilized EEG MoBI to examine dual-task costs in individuals with multiple sclerosis and healthy controls while they performed a Go/No-Go task while sitting (i.e., single task) or walking (i.e., dual task). In terms of ERP results, for healthy controls, the N2 differentiation (between correct rejections and hits) was significantly reduced during walking relative to sitting (De Sanctis et al., 2020). However, for individuals with multiple sclerosis, the N2 differentiation in walking relative to sitting was not significant. Overall, the results suggested aberrant ERP brain activity in multiple sclerosis compared to healthy controls. Similar to De Sanctis et al. (2020), the current study demonstrated divergent neural functioning in walking relative to standing in the clinical group between the ASD group and TD group. Both studies thus demonstrated differences in neural processing in clinical groups relative to healthy controls.

As the current study was the first to investigate neural processing in walking versus standing in ASD, and found that neural processing diverged from TD, future research would be needed to replicate the finding and better understand potential salience of the difference.

Exploratory Aim 1: Motor Domains and Social Ability

The goal of Exploratory Aim 1 was to examine the relationships between three domains of gross motor ability (M-ABC 2 manual dexterity, aiming & catching, and balance) and social ability of ASD and TD individuals, separately. In the ASD group, there were no significant relationships between social ability and any of the three domains of manual dexterity, aiming & catching, and balance. However, while not significant, a trend demonstrated that increased social deficits were associated with poorer manual dexterity in ASD. In the TD group, on the other hand and surprisingly, increased social deficits were significantly associated with better manual dexterity. Additional exploratory analyses revealed that better manual dexterity was related to lower autism severity. Further, across the full sample, better balance was related to better social cognition and social communication.

The contrasting relationship between manual dexterity and social ability in ASD versus TD is notable given previous research in this area. Along with a balance task, ASD children exhibited the most difficulty with a manual dexterity pegboard activity compared to all other tasks using the M-ABC 2 (Green et al., 2009). Further, in a systematic review of 16 studies, among fine motor skills, manual dexterity was found to be the aspect most related to social ability within ASD individuals (Ohara et al., 2019). The current study's trend in ASD that worse manual dexterity is related to worse social ability in ASD is thus overall consistent with prior research (Lidstone, 2020; West, 2019), lending support to previous theories regarding the importance of manual dexterity in ASD.

The contrasting finding that in the TD group, better social ability was associated with better manual dexterity, was unexpected. The finding points to the possibility that the development of motor and social skills is distinctive in the general population, while a different process of developing motor and social skills occurs in ASD. Previous studies have largely investigated the relationship between motor and social skills in ASD or other clinical populations, however future studies may wish to specifically include control groups, to compare the potentially distinct development processes that occurs relative to the general population.

Neuroanatomical and developmental perspectives may offer mechanisms in which manual dexterity is affected within people with ASD, and is related to social ability. Firstly, from a neuroanatomical perspective, there is underlying shared neuroanatomical circuity in both social and motor development, such as the cerebellum which has been consistently implicated to be a brain region of abnormal processing in ASD (Ohara et al., 2019). The cerebellum has been implicated in both motor and social skills (Ataullah & Naqvi, 2022; Van Overwalle et al., 2020). More specifically regarding manual dexterity, in an fMRI study, gray matter volume in the cerebellar right lobule VI area predicted manual dexterity in a pegboard task within TD adolescents (Kuhn et al., 2012). Further, the finding that balance was related to social ability, particularly social cognition and social communication, also fits in given the cerebellum's strong role in balance. Taken together, there is neuroanatomical evidence that the systems involved in manual dexterity, balance, and social ability may function differently in ASD relative to TD. Secondly, from a developmental perspective, motor and social abilities are considered to be inextricably interwoven, especially in early development.

Adequate motor skills allows young children to interact with their environment, which therefore helps to inform the child about the world around them (Leonard & Hill, 2014).

Future research may aim to investigate more particularly how manual dexterity and balance are related to social cognition and social communication, potentially integrating the use of brain imaging methods such as fMRI to examine regions such as the cerebellum.

Exploratory Aim 2: N2/P3 and Autism Severity

The goal of Exploratory Aim 2 was to examine the relationships between neural substrates of inhibitory control (N2/P3) while walking and standing, in relation to autism severity level in ASD (as assessed by the ADOS severity score). Interestingly, only weakened P3 response while standing was associated with more severe autism symptoms. P3 elicited while walking was not associated with autism severity level. N2 elicited during standing or walking was not associated with autism severity level.

Related to this aim, additional exploratory analyses across the full sample revealed associations between N2/P3 neural processing and inhibitory control. More specifically for N2, increased N2 neural processing (i.e., more negative) while walking was related to better inhibitory control; no relationship was seen between N2 while standing and inhibitory control. Regarding P3, there was a trend for increased P3 neural processing while standing related to better inhibitory control; no relationship was seen between P3 while walking and inhibitory control. Thus, in this sample, utilizing more N2 neural resources while walking, and P3 neural resources while standing, was related to better inhibitory control.

It was surprising and unclear why N2 while walking, and P3 but with standing, were related to inhibitory control. Additionally, weakened P3 while standing, but not walking, was related to autism severity. As mentioned, N2 was not related to autism severity, while

standing or walking. It may be that, although N2 and P3 are related and overlapping constructs, they are separate entities that are differentially elicited during the intersection of cognitive (inhibition) and diverse motoric (standing vs. walking) activities. To this end, future research is needed to ascertain whether the current study findings can be replicated, and what this may mean for individuals with ASD.

Clinical Implications

Individuals with ASD exhibit difficulties in motor domains and gait parameters, and there is considerable research that various motor abilities are linked with social abilities. As such, there are critical implications for ASD individuals, as well as their parents, guardians, educators, and their health care team. Based on the findings of the current study as well as previous research, there are important clinical implications for detection and screening tools, therapeutic interventions, and education and outreach for the general public.

Early detection of ASD using motor and gait measures as a screening tool is one area that could potentially be improved. While objective measures such as the M-ABC 2 could be utilized, undergoing a full M-ABC 2 may be burdensome and inappropriate for very young children. Future teams may wish to develop a simpler and more feasible tool that is also standardized and objective in measuring motor skills in young individuals. As of yet, there is no gold standard for objectively measuring motor domains in young children. Alternatively, a motor or gait survey or questionnaire may be created that can be used by parents and/or clinicians. The Developmental Coordination Questionnaire has demonstrated excellent sensitivity to detecting motor problems with ASD children and adolescents in Belgium (Van Damme, Vancampfort, Thoen, Sanchez, & Van Biesen, 2022), however more research is needed to clarify whether the results can be replicated and appropriate for ASD children in

other cultures. In brief, some studies have found there are differences by culture, in the perception of “abnormal” versus “normal” factors (Mandell et al., 2009), which may impact whether there is detection and recognition of motor problems. In terms of gait, there is even less standardization for measurement, especially for examining the types of variability that occur in gait. Overall, there is room for improvement in screening for motor and gait abnormalities in young children who potentially have ASD.

New interventions may be developed that help to improve motor abilities for individuals with ASD, which may thereby potentially improve social abilities if interventions are delivered at a young age. Of note, more clinical and intervention research is still needed for this nascent idea. In particular based on the study findings, manual dexterity, balance, and gait variability are specific areas that may be targeted. For instance, a possible therapy may involve assisting the individual with safe exercises that help them to develop improved manual dexterity (e.g., spend time in holding different objects). Similarly, for balance and gait, an intervention may focus on practicing to walk more steadily and assuredly.

More education and outreach can be provided to the wider public in terms of recognizing the salience of motor and gait abnormalities in ASD. While social skills are commonly known by lay people to be deficient in ASD, the general public is unaware that motor is also a critical area in ASD. It is important to educate the community at large because difficulty with reaching motor milestones is potentially the first indication that an individual may have ASD. The earlier a parent or guardian detects motor abnormalities, ideally, the earlier the family will be able to obtain a diagnosis. Earlier recognition of ASD can thus lead to earlier intervention, and earlier intervention in ASD is linked to better outcomes particularly in the domain of social communication (Fuller & Kaiser, 2020).

Limitations

There are a number of limitations of the study. With the cross-sectional design, the results do not allow inference of causal associations. The limited sample size is also a concern, which may underpower the results. However, there are inherent challenges in the recruitment of ASD individuals who are able to engage in studies that involve complex sensory-motor stimuli and a moderate length of time. The sample was thus comprised of high functioning ASD individuals, and the findings may not be representative of all individuals with ASD (i.e., not lower functioning ASD). Despite the limitation, the current study was able to identify an array of racially and ethnically diverse ASD individuals who successfully completed the study. Individuals resided in the same geographic location, which limits geographic generalizability; however, again, the racial and ethnic diversity of individuals in the Bronx and the greater New York City area represent a strength.

Several aspects of assessment were limited. As mentioned, the M-ABC 2 manual does not provide norms for individuals over the age of 17 years old. Therefore, the raw scores (as opposed to standardized scores), were used for analyses. Regarding the SRS-2, two forms were used (self-report or filled out by a parent/guardian), which is limiting because a participant and parent may have discrepant ratings. It is unclear whether SRS-2 self-report and parent/guardian are generally concordant, as research examining the association was not readily found. However, there are inherent limitations overall to survey measures that are thought to be subjective, relative to data considered to be more objective (i.e., ratings by a trained clinician). Notably, to supplement the weaknesses surrounding the use of the SRS-2,

the current study also examined autism severity, which was based on the clinician-rated ADOS. Furthermore, the majority of the ADOS evaluations were completed by the same clinician, which minimizes potential inter-rater discordance. Despite the drawbacks surrounding the use of the SRS-2 in the study, examining the SRS-2 data of the current sample was deemed worthwhile given that a large number of researchers and clinicians have previously used the SRS-2 instrument. It was considered important to analyze how the present sample data fit within the larger literature base, and ultimately help to better understand social ability and domains of individuals with ASD.

The timing of the sessions and procedures varied for participants. That is, some individuals had previously participated in CNL studies recently and therefore had already been administered Wechsler tests. In that case, the FSIQ was done at an earlier time point relative to participants who were participating in a CNL study for the first time. However, FSIQ scores are generally stable across one's lifetime for TD individuals, and FSIQ and index scores appear to be stable in the long term for children with ASD ages 11 years and older as well (Okada et al., 2022).

Lastly, the trends mentioned in the current study should be interpreted with caution. Given the limited sample size and other limitations previously mentioned, more research is needed prior to making strong claims regarding the trends demonstrated in this study.

Conclusions

The goal of the current study was to examine critical relationships between motor ability, social ability, inhibition, and underlying neural indices. Overall, the study findings generally bolstered previous research, lending additional support for the idea that there is a notable relationship between motor and social abilities. Increased stride time variability,

decreased balance, and decreased manual dexterity were related to greater social deficits, particularly in the social cognitive domain.

This study was novel in that it was the first known to investigate social ability in relation to neural processes while walking and standing in ASD versus TD, utilizing an EEG MoBI paradigm. In this regard, TD individuals exhibited greater N2/P3 amplitude while standing than walking, which was not seen in ASD. The incongruity points to differential neural processes in ASD and TD, and provides a starting point for future research to further investigate. Additionally, decreased P3 response while standing was associated with more severe autism symptoms, but not while walking.

Despite several limitations of the study, the study has built upon previous research, and paves the way for future research to further examine walking, neural processes, and social deficits in ASD. There are clinical implications for adding motor skills in ASD screening tools, developing therapeutic interventions for ASD, and providing education to the general public surrounding motor and gait aberrances in ASD.

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Tables

Table 1.

Demographic, Cognitive, Clinical, and Motor Characteristics in TD vs. ASD

	TD M (SD) or %	ASD M (SD) or %	Test Statistic	*P*	Mean Rank [a] TD | ASD
DEMOGRAPHIC CHARACTERISTICS	n = 18	n = 20			
Age (Years) [a]	17.19 (2.58)	16.75 (2.73)	165.0	.68	20.33 | 18.75
Gender (% Male) [b]	61%	85%	--	.09	--
Handedness (% Right) [c]	89%	70%	4.61	.10	--
Race [c]	% (n)	% (n)	5.97	.20	--
White	55.6% (10)	35.0% (7)	--	--	--
Black/African American	22.2% (4)	40.0% (8)	--	--	--
Asian	5.6% (1)	0.0% (0)	--	--	--
Multi-racial	16.7% (3)	10.0% (2)	--	--	--
Unknown	0.0% (0)	15.0% (3)	--	--	--
Ethnicity (% Hispanic)	27.8% (5)	40.0% (8)	--	.43	--
COGNITIVE CHARACTERISTICS					
IQ	n = 14	n = 16			
FSIQ [a]	107.50 (9.60)	94.20 (14.21)	51.5	<.001**	25.28 | 13.08
Verbal [a]	108.93 (12.09)	95.78 (17.73)	70.5	.006**	23.09 | 13.71
Perceptual [a]	108.50 (10.95)	99.47 (15.56)	81.5	.018*	22.41 | 14.29
Stroop/CWIT Inhibition [a]	n = 13	n = 13			
	11.54 (0.72)	8.08 (0.84)	34.0	.009**	17.38 | 9.62
ERP (Go/No-Go Task)	n=18	n=20			
N2, Walking [d]	-6.24 (3.57)	-4.45 (3.23)	225.0	.19	17.00 | 21.75
N2, Standing [d]	-7.85 (5.23)	-4.81 (5.02)	237.0	.09	16.33 | 22.35
P3, Walking [d]	4.97 (4.28)	3.71 (4.25)	156.0	.49	20.83 | 18.30
P3, Standing [d]	6.90 (4.13)	3.63 (3.42)	101.0	.02*	23.89 | 15.55
CLINICAL CHARACTERISTICS					
Social Responsiveness (SRS-2) [a]	n = 11	n = 17			
	23.36 (13.17)	82.17 (31.01)	184.0	<.001**	6.27 | 19.82
Autism Severity (ADOS)	--	n = 12 10.00 (4.24)		--	--
MOTOR CHARACTERISTICS					
Movement (M-ABC 2) [a]	n = 14	n = 16			
Overall Movement	70.07 (11.61)	46.25 (12.74)	23.0	<.001**	21.86 | 9.94
Aiming & Catching	20.07 (4.78)	14.93 (5.45)	58.0	.025*	19.36 | 12.13
Balance	27.50 (4.97)	16.93 (5.92)	18.0	<.001**	22.21 | 9.63
Manual Dexterity	22.92 (6.76)	14.37 (8.72)	48.0	.007**	20.07 | 11.50
Gait [a]	n = 18	n = 18			
Stride Time Variability [e]	33.90 (10.30)	58.03 (31.66)	240.0	.013*	14.17 | 22.83
Step Time Variability [e]	15.56 (4.21)	20.86 (7.25)	246.0	.007**	13.83 | 23.17

Key. TD = typically developing; ASD = autism spectrum disorder; M = mean; SD = standard deviation; FSIQ = Full Scale IQ, based on Wechsler tests; CWIT = Color Word Interference Test; M-ABC 2 = Movement Assessment Battery for Children-2; SRS = Social Responsiveness Scale-2; ADOS = Autism Diagnostic Observation Schedule

[a] Mann-Whitney *U*
[b] Fisher's exact test
[c] Chi square
[d] No flow (no optical flow), No-Go (correct rejection) trial
[e] No flow (no optical flow), no task (only walking) condition

*$p < .05$, **$p < .01$

Table 2.
Regression Models of Motor Abilities as Predictors for Social Responsiveness (Aims 1a-c)

	Predicting Social Responsiveness (SRS-2)							
	Model with Gross Motor Ability (M-ABC 2)					R^2	F	*P* of Model
Predictors	B	SE	t	Sig. of Predictors	95% CI	.60	6.84	.002**
Constant	20.29	47.19	.43	.67	[-78.86, 119.44]			
Group [a]	56.09	15.88	.68	.002**	[22.73, 89.45]			
M-ABC 2	.50	.74	.21	.51	[-1.05, 2.05]			
Group x M-ABC 2	-1.33	.94	-.39	.17	[-3.31, 0.64]			
Age	-.174	2.65	-.01	.95	[-5.74, 5.39]			
Model with Stride Time Variability (STV)								
Predictors	B	SE	t	Sig. of Predictors	95% CI	R^2	F	*P* of Model
Constant	43.95	53.30	.82	.41	[-66.89, 154.79]	.61	6.69	<.001**
Group [a]	55.19	16.12	3.42	.003**	[21.65, 88.73]			
STV	.56	.97	.58	.56	[-1.45, 2.58]			
Group x STV	-.58	.99	-.59	.56	[-2.65, 1.48]			
Age	-2.00	2.49	-.80	.43	[-7.17, 3.17]			
FSIQ	.19	.48	.41	.68	[-.80, 1.19]			
Model with Step Width Variability (SWV)								
Predictors	B	SE	t	Sig. of Predictors	95% CI	R^2	F	*P* of Model
Constant	22.66	48.26	.47	.64	[-77.71, 123.04]	.63	7.11	<.001**
Group [a]	59.97	12.44	4.82	<.001**	[34.09, 85.84]			
SWV	-.91	2.97	-.31	.97	[-6.28, 6.10]			
Group x SWV	1.70	3.33	.51	.61	[-5.22, 8.64]			
Age	-.97	2.41	-.40	.69	[-5.99, 4.05]			
FSIQ	.16	.46	.36	.71	[-.79, 1.12]			

Key. SRS = Social Responsiveness Scale-2; M-ABC 2 = Movement Assessment Battery for Children-2; SE = standard error; Sig = significance; CI = confidence interval; FSIQ = Full-Scale IQ; STV = stride time variability; SWV = step width variability

[a] Group = ASD versus TD, with ASD values shown in the table

*$p < .05$, **$p < .01$

Table 3.
Examining Inhibition Across Motor Category (Aim 2)

Examining Inhibition (CWIT) Across Motor Category (M-ABC 2)					
M-ABC 2 Category	n	Mean Rank	*U* [a]	Std. Error	*P*
Motor Difficulties [b]	12	11.8	63.0	15.08	.87
At Risk/Typical Motor Abilities [c]	10	11.2			

Key. CWIT = Color-Word Interference Test; M-ABC 2 = Movement Assessment Battery for Children-2

[a] Mann Whitney U test examining inhibitory control (as assessed by the Stroop/CWIT) based on motor ability (categorized M-ABC 2)
[b] M-ABC 2 total score $\leq 5^{th}$ percentile
[c] M-ABC 2 total score $\geq 6^{th}$ percentile

*$p < .05$, **$p < .01$

Table 4.
Correlations Examining N2/P3 and Social Responsiveness while Walking and Standing in ASD vs. TD (Aim 3)

			ASD		
Variable	1	2	3	4	n
1. SRS-2	--				17
2. N2, Walking[a]	-.33	--			20
3. N2, Standing[a]	.00	.66**	--		20
4. P3, Walking[a]	-.38	.46*	.25	--	20
5. P3, Standing[a]	-.34	.26	.72	.49*	20
			TD		
Variable	1	2	3	4	n
1. SRS-2	--				11
2. N2, Walking[a]	-.30	--			18
3. N2, Standing[a]	-.46	.75**	--		18
4. P3, Walking[a]	.26	-.29	-.23	--	18
5. P3, Standing[a]	.09	-.35	-.32	.74**	18

Key. ASD = autism spectrum disorder; TD = typically developing; SRS-2 = Social Responsiveness Scale-2

[a] No flow (no optical flow), No-Go (correct rejection) trials

*$p < .05$, **$p < .01$

Table 5.
Paired Samples T-test Examining N2/P3 in Walking vs. Standing, in ASD vs. TD

		ASD							
		Mean of the Difference	SD	Std. Error Mean	95% CI Lower	95% CI Upper	t	df	*P*[a]
N2	Walking - Standing[b]	.36	3.35	.75	-1.20	1.93	.48	19	.63
P3	Walking - Standing[b]	.08	4.30	.96	-1.93	2.09	.08	19	.93
		TD							
		Mean of the Difference	SD	Std. Error Mean	95% CI Lower	95% CI Upper	t	df	*P*
N2	Walking - Standing[b]	1.61	2.76	.65	.24	2.98	2.47	17	.024*
P3	Walking - Standing[b]	-1.94	2.96	.69	-3.41	-0.47	-2.78	17	.013*

Key. ASD = autism spectrum disorder; TD = typically developing; SD = standard deviation; CI = confidence interval

[a] Paired samples t-test
[b] No flow (no optical flow), No-Go (correct rejection) trials

*$p < .05$, **$p < .01$

Table 6.
Correlations Examining Motor Ability Domains and Social Ability in ASD vs. TD (Exploratory Aim 1)

		ASD		
Variable [a]	1	2	3	n
1. SRS-2	--			14
2. Manual Dexterity [b]	-0.42	--		16
3. Aiming & Catching	0.13	0.05	--	16
4. Balance	-0.22	0.19	-0.05	16
		TD		
Variable	1	2	3	n
1. SRS-2	--			11
2. Manual Dexterity	0.70*	--		14
3. Aiming & Catching	-0.06	0.33	--	14
4. Balance	-0.13	-0.09	0.35	14

Key. ASD = autism spectrum disorder; TD = typically developing; SRS-2 = SRS = Social Responsiveness Scale-2

[a] Pearson's correlations, with the exception of Manual Dexterity for ASD
[b] Spearman's rho correlation

*$p < .05$, **$p < .01$

Table 7.
Correlations Examining N2/P3 and Autism Severity Level while Walking and Standing (Exploratory Aim 2)

	Within the ASD Group				
Variable	1	2	3	4	n
1. ADOS Severity	--				12
2. N2, Walking[a]	-0.19	--			12
3. N2, Standing[a]	0.03	0.70**	--		12
4. P3, Walking[a]	-0.31	0.00	-0.05	--	12
5. P3, Standing[a]	-0.70*	-0.20	-0.26	0.63**	12

Key. ASD = autism spectrum disorder; ADOS = Autism Diagnostic Observation Schedule severity score

*$p < .05$, **$p < .01$

[a] No flow (no optical flow), No-Go (correct rejection) trials

Table 8.
Spearman's Correlations Examining Motor, Social, and Neurocognitive Variables Across the Full Sample

			1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1	Stride Time Variability	Corr. Coeff.	---															
		P																
		N	36															
2	Step Width Variability	Corr. Coeff.	.33	---														
		P	.05															
		N	36	36														
3	M-ABC 2 Total	Corr. Coeff.	-.384*	-.605**	---													
		P	.04	.001														
		N	28	28	30													
4	M-ABC 2: Aiming & Catching	Corr. Coeff.	-.3	-.499**	.669**	---												
		P	.14	.007	.000													
		N	28	28	30	30												
5	M-ABC 2: Balance	Corr. Coeff.	-.456*	-.521**	.778**	.32	---											
		P	.02	.004	.000	.08												
		N	28	28	30	30	30											
6	M-ABC 2: Manual Dexterity	Corr. Coeff.	-.2	-.36	.809**	.387*	.444*	---										
		P	.4	.06	.000	.03	.01											
		N	28	28	30	30	30	30										
7	SRS-2 Total	Corr. Coeff.	.432*	.33	-.511*	-0.3	-.620**	-.33	---									
		P	.02	.09	.01	.17	.002	.12										
		N	27	27	23	23	23	23	28									

8	SRS-2 Total: Social Awareness	Corr. Coeff.	.38	.24	-.19	-.2	-.32	-.06	.739**	---						
		P	.05	.23	.39	.52	.15	.8	.000							
		N	26	26	22	22	22	22	27	27						
9	SRS-2: Social Cognition	Corr. Coeff.	.438*	.15	-.430*	-.2	-.532*	-.31	.860**	.724**	---					
		P	.03	.46	.05	.32	.011	.16	.000	.000						
		N	26	26	22	22	22	22	27	27	27					
10	SRS-2: Social Communication	Corr. Coeff.	.31	.23	-.42	-.2	-.599**	-.19	.958**	.769**	.865**	---				
		P	.12	.25	.05	.37	.003	.4	.000	.000	.000					
		N	26	26	22	22	22	22	27	27	27	27				
11	SRS-2: Social Motivation	Corr. Coeff.	.15	.14	-.21	.01	-.445*	-.06	.813**	.589**	.693**	.875**	---			
		P	.47	.49	.35	.95	.04	.8	.000	.000	.000	.000				
		N	26	26	22	22	22	22	27	27	27	27	27			
12	ADOS Severity	Corr. Coeff.	.35	.52	-.6	-.3	-.39	-.603*	.54	.49	.58	.56	.24	---		
		P	.32	.12	.05	.41	.24	.05	.08	.12	.06	.07	.48			
		N	10	10	11	11	11	11	11	11	11	11	11	12		
13	Stroop/CWIT Inhibition	Corr. Coeff.	-.1	-.06	.22	.1	.23	.19	-.19	-.471*	-.36	-.26	-.2	-.02	---	
		P	.64	.78	.32	.66	.3	.39	.42	.04	.13	.28	.44	.97		
		N	25	25	22	22	22	22	20	19	19	19	19	8	26	
14	N2 - Standing	Corr. Coeff.	-.1	.29	-.34	-0.3	-.19	-.19	.22	.05	.18	.21	.11	.03	-.31	---
		P	.48	.09	.07	.08	.32	.31	.27	.82	.36	.29	.59	.93	.12	
		N	36	36	30	30	30	30	28	27	27	27	27	12	26	38
15	N2 - Walking	Corr. Coeff.	-.2	.24	-.18	-.3	-.13	.08	.02	.18	.05	.12	-0	-.19	-.417*	.702**
		P	.3	.17	.34	.13	.5	.69	.92	.38	.81	.54	.95	.56	.03	.000

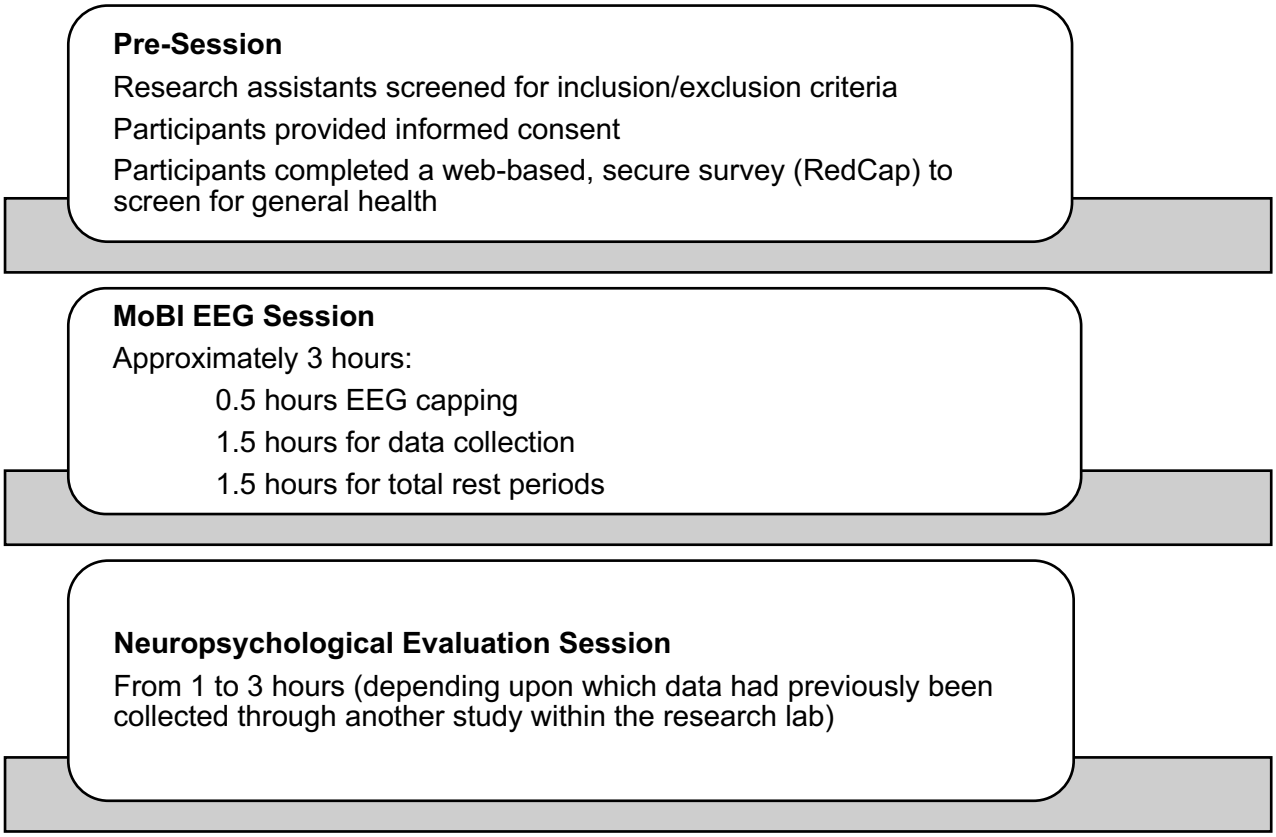
		N	36	36	30	30	30	30	28	27	27	27	27	12	26	38	38	
16	P3 - Standing	Corr. Coeff.	-.2	-.399*	.384*	.08	.36	.415*	-.27	-.21	-.24	-.2	.07	-.70*	.38	-.26	-0	---
		P	.18	.016	.04	.67	.05	.023	.17	.29	.22	.32	.71	.01	.06	.11	0.2	
		N	36	36	30	30	30	30	28	27	27	27	27	12	26	38	38	38
17	P3 - Walking	Corr. Coeff.	-.3	-.345*	.09	-.1	.15	.14	-.18	-.12	-.04	-.16	-0	-.31	.15	-.05	0	.629**
		P	.14	.039	.65	.7	.43	.46	.35	.56	.84	.43	.94	.32	.47	.75	1	.000
		N	36	36	30	30	30	30	28	27	27	27	27	12	26	38	38	38

Key. M-ABC 2 = Movement Assessment Battery for Children-2; SRS = Social Responsiveness Scale-2; ADOS = Autism Diagnostic Observation Schedule; CWIT = Color Word Interference Test

*$p < .05$, **$p < .01$

Figures

Figure 1.
Summary of Study Sessions



Key. MoBI = mobile brain body imaging; EEG = electroencephalography

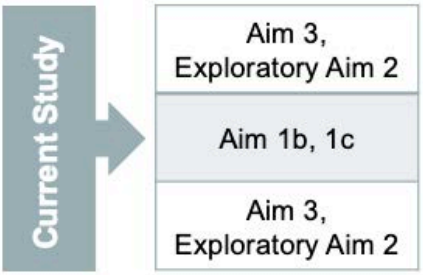
Figure 2.
EEG MoBI Setup

Note. Photograph demonstrates a sample participant wearing the EEG cap and pressure sensors, hooked onto the safety harness, while walking on the treadmill. Across from the participant is a screen on which the Go/No-Go task is displayed. Used with permission by De Sanctis and colleagues (De Sanctis et al., 2014).

Figure 3.
Blocks Examined in the Parent Study and the Current Study

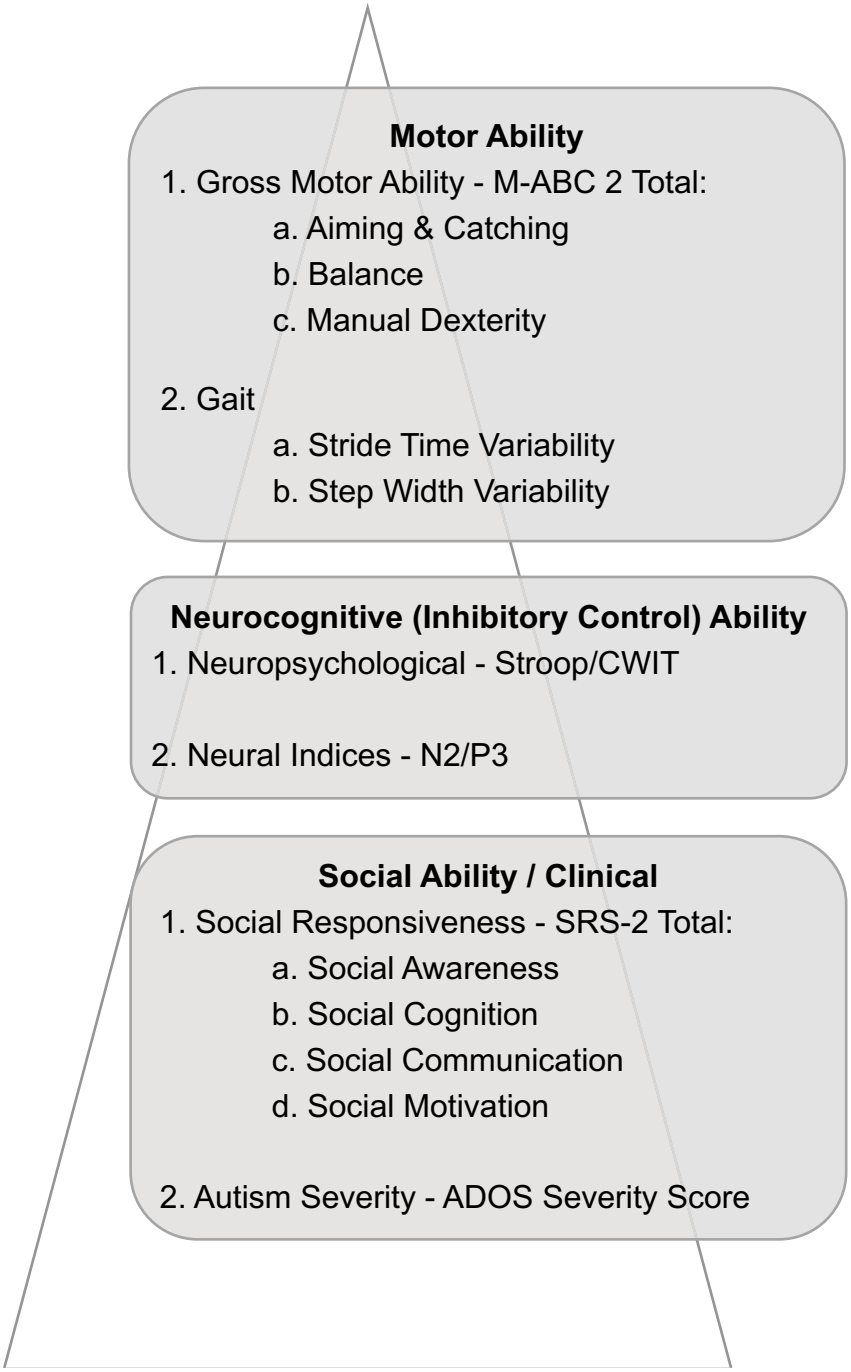
Parent Study

Walk or Stand	Flow or No Flow	Go/No-Go Task or No Task	# of Blocks
Walk	Flow	No Task	3
Walk	Flow	Task	3
Walk	**No Flow**	**Task**	**3**
Walk	**No Flow**	**No Task**	**3**
Stand	**No Flow**	**Task**	**3**



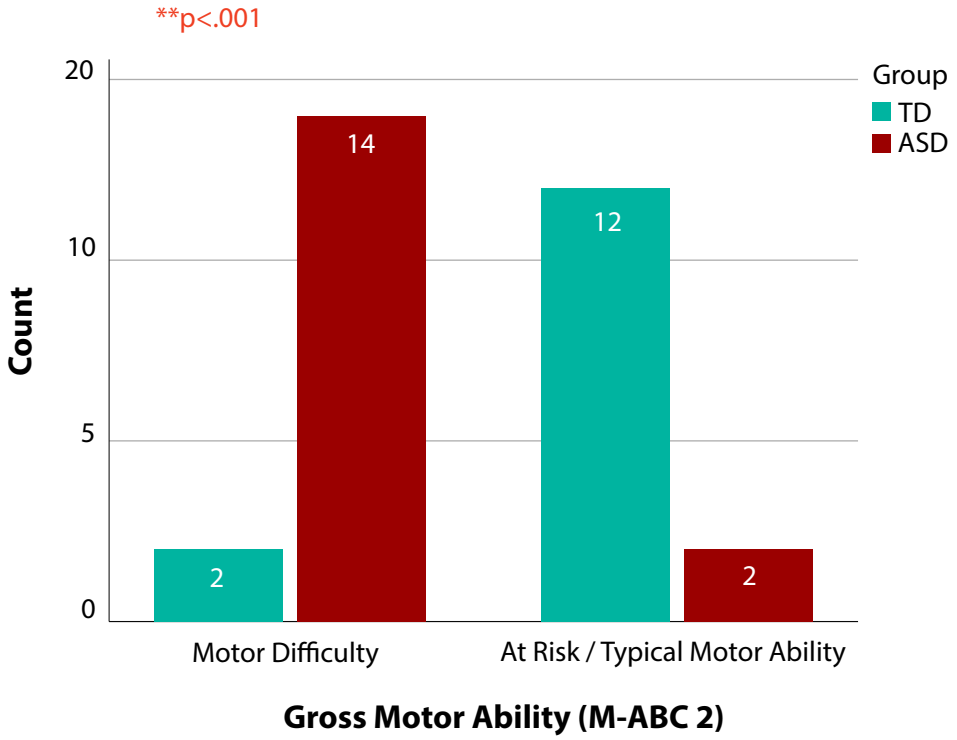
Note. Figure demonstrates the blocks completed in the parent study (left), and which of those blocks were completed in the current study along with aims (right).

Figure 4.
Summary of Constructs Examined and Measures Utilized



Key. M-ABC 2 = Movement Assessment Battery for Children-2; CWIT = Color-Word Interference Test; SRS = Social Responsiveness Scale-2; ADOS = Autism Diagnostic Observation Schedule

Figure 5.
Breakdown of Categorized Gross Motor Ability in ASD vs. TD

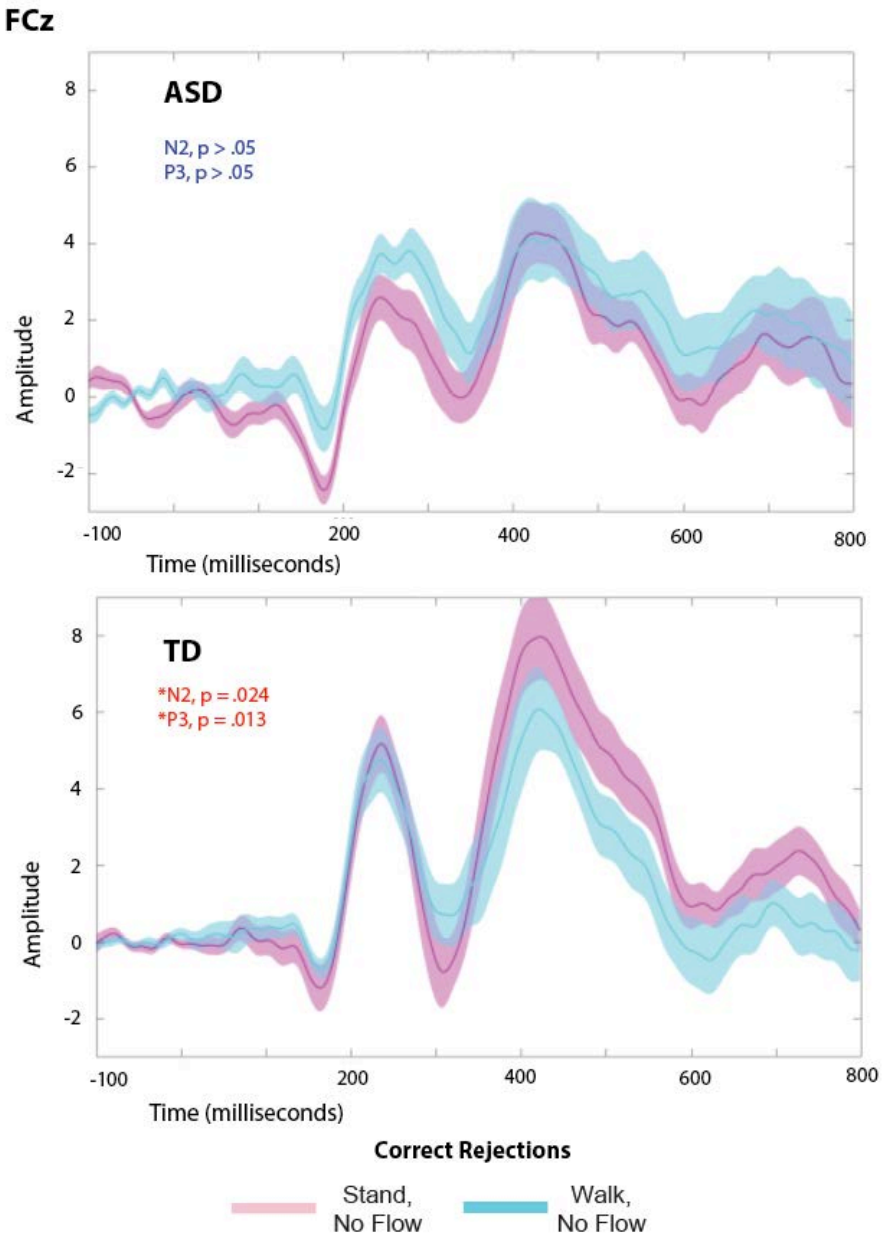


Note. M-ABC 2 = Movement Assessment Battery for Children-2; ASD = autism spectrum disorder; TD = typically developing

[a] M-ABC 2 total score $\leq 5^{th}$ percentile
[b] M-ABC 2 total score $\geq 6^{th}$ percentile
[c] Fisher's exact test, testing for association between diagnostic group (ASD vs. TD) and motor group (at risk/typical motor ability, or motor difficulty); Mann Whitney U test

*$p < 0.05$, **$p<0.01$

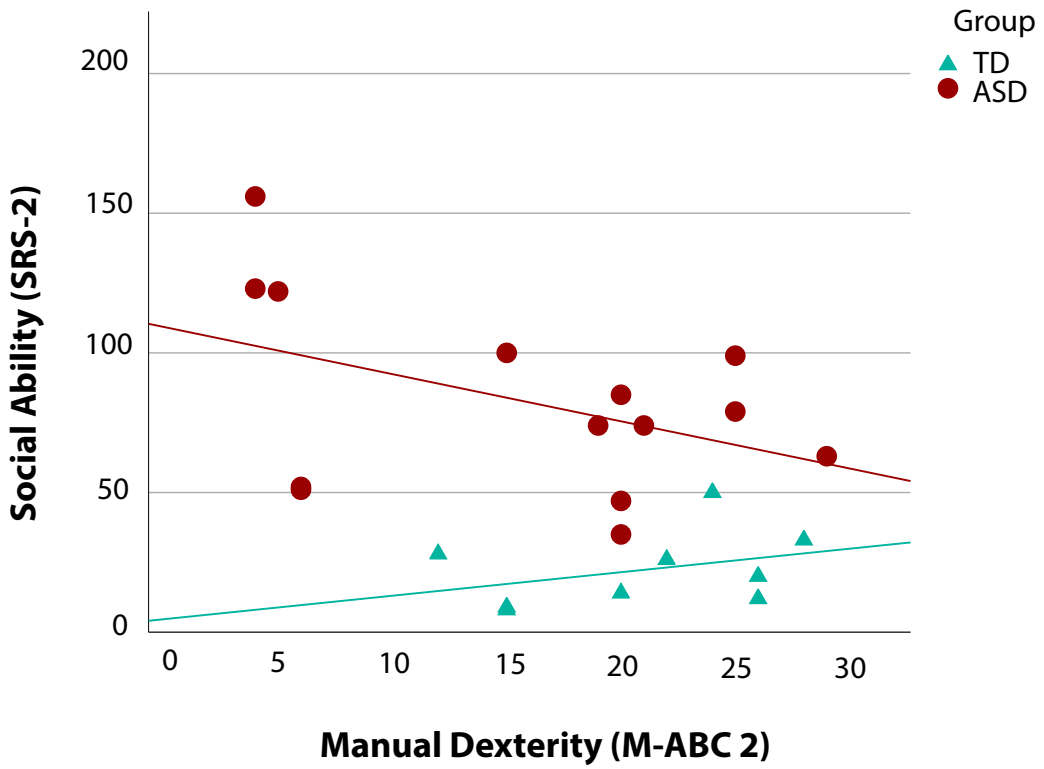
Figure 6.
N2/P3 Amplitudes Elicited from Correct Rejections While Standing vs. Walking in ASD vs. TD (Aim 3)



Note. In ASD, no differences in N2/P3 amplitude were found between the standing and walking conditions. In TD, differences in both N2 and P3 were found in the standing versus walking conditions. That is, significantly greater N2/P3 neural processing was exhibited while standing relative to walking.

Key. FCz = fronto-central electrode channel; ASD = autism spectrum disorder; TD = typically developing

Figure 7.
Scatterplot of the Relationship Between Social Ability and Manual Dexterity in ASD vs. TD (Exploratory Aim 1)



Note. For TD, a greater number of social deficits was significantly associated with better manual dexterity. On the other hand, for ASD (though not significant), a greater number of social deficits was associated with worse manual dexterity.

Key. ASD = autism spectrum disorder; TD = typically developing; SRS-2 = SRS = Social Responsiveness Scale-2; M-ABC 2 = Movement Assessment Battery for Children-2

Appendix

Appendix A

Pearson's Correlations Examining Motor, Social, and Neurocognitive Variables Across the Full Sample

			1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1	Stride Time Variability	Corr. Coeff.	--															
		P																
		N	36															
2	Step Width Variability	Corr. Coeff.	0.20	--														
		P	0.24															
		N	36	36														
3	M-ABC 2 Total	Corr. Coeff.	-.393*	-.521**	--													
		P	0.04	0.005														
		N	28	28	30													
4	M-ABC 2: Aiming & Catching	Corr. Coeff.	-0.30	-0.33	.691**	--												
		P	0.11	0.09	0.000													
		N	28	28	30	30												
5	M-ABC 2: Balance	Corr. Coeff.	-.573**	-.404*	.778**	.391*	--											
		P	0.001	0.03	0.000	0.03												
		N	28	28	30	30	30											
6	M-ABC 2: Manual Dexterity	Corr. Coeff.	-0.10	-.456*	.810**	0.34	.398*	--										

		P	0.61	0.01	0.000	0.06	0.03							
		N	28	28	30	30	30	30						
7	SRS-2 Total	Corr. Coeff.	0.35	.400*	-.541**	-0.23	-.559**	-.447*	--					
		P	0.08	0.04	0.01	0.30	0.006	0.03						
		N	27	27	23	23	23	23	28					
8	SRS-2 Total: Social Awareness	Corr. Coeff.	0.29	0.24	-0.22	-0.12	-0.31	-0.08	.751**	--				
		P	0.15	0.24	0.33	0.61	0.15	0.72	0.000					
		N	26	26	22	22	22	22	27	27				
9	SRS-2: Social Cognition	Corr. Coeff.	0.32	0.22	-.447*	-0.19	-.491*	-0.34	.862**	.736**	--			
		P	0.11	0.29	0.04	0.39	0.02	0.12	0.000	0.000				
		N	26	26	22	22	22	22	27	27	27			
10	SRS-2: Social Communication	Corr. Coeff.	0.28	0.30	-.461*	-0.18	-.567**	-0.30	.967**	.771**	.859**	--		
		P	0.17	0.13	0.03	0.43	0.006	0.18	0.000	0.000	0.000			
		N	26	26	22	22	22	22	27	27	27	27		
11	SRS-2: Social Motivation	Corr. Coeff.	0.08	0.19	-0.22	0.04	-0.41	-0.11	.825**	.590**	.697**	.868**	--	
		P	0.69	0.36	0.32	0.86	0.06	0.64	0.000	0.001	0.000	0.000		
		N	26	26	22	22	22	22	27	27	27	27	27	
12	ADOS Severity	Corr. Coeff.	0.11	0.56	-.702*	-0.39	-0.27	-.657*	.676*	0.48	.650*	.625*	0.22	--
		P	0.77	0.09	0.02	0.23	0.42	0.03	0.02	0.13	0.03	0.04	0.52	
		N	10	10	11	11	11	11	11	11	11	11	11	12
13	Stroop/CWIT Inhibition	Corr. Coeff.	-0.12	-0.08	0.24	0.17	0.21	0.17	-0.21	-.489*	-0.41	-0.28	-0.20	-0.22	--
		P	0.57	0.71	0.29	0.45	0.35	0.46	0.37	0.03	0.08	0.24	0.41	0.60	

		N	25	25	22	22	22	22	20	19	19	19	19	8	26			
14	N2 - Standing	Corr. Coeff.	-0.20	0.33	-0.30	-0.30	-0.17	-0.21	0.18	0.03	0.21	0.16	0.03	0.18	-0.36	--		
		P	0.23	0.05	0.11	0.10	0.36	0.27	0.36	0.87	0.30	0.41	0.88	0.57	0.07			
		N	36	36	30	30	30	30	28	27	27	27	27	12	26	38		
15	N2 - Walking	Corr. Coeff.	-0.17	0.31	-0.18	-0.31	-0.16	0.03	0.04	0.11	0.07	0.09	-0.02	-0.13	-0.34	.825**	--	
		P	0.33	0.07	0.35	0.10	0.40	0.86	0.83	0.58	0.73	0.64	0.93	0.69	0.09	0.000		
		N	36	36	30	30	30	30	28	27	27	27	27	12	26	38	38	
16	P3 - Standing	Corr. Coeff.	-0.12	-.398*	.460*	0.12	.373*	.457*	-0.28	-0.24	-0.23	-0.26	0.01	-0.29	.388*	-0.22	-0.21	--
		P	0.50	0.02	0.01	0.52	0.04	0.01	0.15	0.24	0.24	0.20	0.95	0.36	0.05	0.18	0.20	
		N	36	36	30	30	30	30	28	27	27	27	27	12	26	38	38	38
17	P3 - Walking	Corr. Coeff.	-0.14	-.355*	0.14	-0.06	0.05	0.26	-0.23	-0.18	-0.13	-0.24	-0.05	-0.35	0.13	0.08	0.11	.58**
		P	0.43	0.03	0.45	0.76	0.79	0.17	0.24	0.38	0.51	0.23	0.81	0.26	0.53	0.64	0.50	0.000
		N	36	36	30	30	30	30	28	27	27	27	27	12	26	38	38	38

Key. M-ABC 2 = Movement Assessment Battery for Children-2; SRS = Social Responsiveness Scale-2; ADOS = Autism Diagnostic Observation Schedule; CWIT = Color Word Interference Test

*$p < .05$, **$p < .01$