MOTOR LEARNING IN CHILDREN WITH AN AUTISM SPECTRUM DISORDER

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by

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ABSTRACT

In addition to social communication difficulties, individuals with an autism spectrum disorder (ASD) frequently experience problems in other domains such as motor control and learning. The goal of the current study was to better understand ASD-related differences in motor learning. We isolated and examined the ballistic and corrective submovements associated with learning of a rapid aimed limb movement in a sample of children with ASD and a matched comparison group of typically developing children without ASD. The overall rate of motor learning did not differ significantly between groups. Analysis of movement subcomponents, however, revealed significant group differences. These findings support the hypothesis that, while motor learning per se is not impaired in ASD, individuals with ASD utilized different strategies in motor learning.
INTRODUCTION

Autism is a neurodevelopmental disorder associated with core deficiencies in social interaction, communication, and repetitive or stereotyped behaviors or interests (American Psychiatric Association, 2000). In addition to experiencing difficulties with social communications, individuals with an autism spectrum disorder (ASD) frequently experience problems in other domains of functioning such as motor control and learning (see Nayate, Bradshaw, & Rinehart, 2005 for review). ASD has long been associated with motor difficulties, with both Kanner (1943) and Asperger (Frith, 1991) describing atypical motor behavior and/or skill acquisition in their case study samples. Subsequent research has affirmed these earlier accounts and documented pervasive motor problems in children and adults with ASD.

Mounting evidence suggests that problems with motor control may be apparent at a very early age in infants and children with ASD. For example, based on analysis of videotapes of infants that would later be diagnosed with ASD, (P. Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer, 1998) found that problems with motor behaviors such as crawling and rolling over were evident as early as 4-6 months of age. Additional support comes from research by Young, Brewer, & Pattison 2003) in which parents of children with ASD were asked to retrospectively report if/when they noticed atypical behavior during early development. The study found that 28% of parents noted early problems with gross motor skills, and 41% noted early problems with fine motor skills. These difficulties with gross and fine motor control were evident on average by 12 and 17 months of age, respectively. Results from subsequent studies have affirmed this finding. In a prospective study of children at high and low risk for development of ASD,
Landa & Garrett-Mayer (2006) documented that difficulties in gross and fine motor ability may be evident as early as 14 months of age in children who are eventually diagnosed with ASD. Most recently, (Lloyd, Macdonald, & Lord, in press) reported impairments in gross and fine motor ability in a sample of early diagnosed (12-36 mos) toddlers with ASD.

Findings from additional studies suggest that some motor control difficulties persist into later childhood and eventual adulthood. Based on a review of medical charts for 154 children and adults with ASD, Ming, Brimacombe, & Wagner (2007) documented higher-than-typical incidence rate of hypotonia, motor apraxia, toe-walking, and gross motor delay in young children with ASD. In addition, although such deficits were more apparent in younger (2-6 years old) as compared to older (7-18 years old) children with ASD, the researchers found that the incidence rate of motor impairment in older children with ASD was still significantly higher than that observed in the general population. Consistent with this, other studies have also reported motor control and coordination problems in adolescents and adults with ASD (e.g., Rinehart et al., 2006; Beversdorf et al., 2001; Ghaziuddin & Butler, 1998; Nayate et al., 2005; Noterdaeme, Mildenberger, Minow, & Amorosa, 2002). Although the full etiologic basis for the observed motor difficulties remains unclear, recent research (e.g. Mostofsky et al., 2009) suggests that disruptions in neurocognitive processes related to motor learning contribute to the motor difficulties experienced by individuals with ASD.

Motor Learning

Motor learning refers to the process that, largely through practice and repetition, leads to the acquisition of motor skills (Savion-Lemieux, Bailey, & Penhune, 2009). This
consolidation process is marked by improvements in the speed and accuracy/precision with which the given motor movement (or sequence of movements) is performed. Over time with extensive repetition, movements may also become automated, no longer requiring overt attention control for their execution (Doyon et al., 2009; Schmidt, 1975).

As a framework for understanding motor learning, Woodworth (1899) and others have suggested that execution and learning of voluntary aimed movement may be best conceptualized as consisting of two distinct components: an initial ballistic component which brings the body part (e.g., the arm) into the general vicinity of the target position/location, and a corrective component whereby feedback from sensory systems (e.g., vision) is used to correct for any inaccuracies in the initial movement and ultimately bring the body part to rest in the target position/location. Subsequent research (e.g., Abrams & Pratt, 1993; Elliott et al., 2010; Pratt, Chasteen, & Abrams, 1994) has demonstrated that practice and learning of a motor movement is accompanied by, not only an improvement in overall movement speed and accuracy, but also an increase in the accuracy of the initial ballistic component and thus a decrease in the proportion of movement time that must be devoted to the feedback/corrective component.

From a neurophysiological standpoint, motor learning relies on a complex network of brain regions including aspects of the cerebellum, basal ganglia, primary and supplementary motor areas, parietal cortex, and thalamus (for review, see Hikosaka, Nakamura, Sakai, & Nakahara, 2002). Importantly, in much the same manner as the aforementioned movement components are dynamic and change with practice/repetition so too does the relative contribution of these brain areas. The cerebellum and its interconnections appear more active in the earlier stages as compared to the later stages...
of motor learning (for review, see Doyon et al., 2009). In contrast, the basal ganglia and supplementary motor areas demonstrate the opposite pattern of activation, with these areas showing relative greater activation for tasks that are already learned.

Differences in motor learning pathways in persons with ASD as compared to those without ASD include activation differences in the cerebellum (Mostofsky et al., 2009; Allen & Courchesne, 2003; Allen, Müller, & Courchesne, 2004), parietal regions of the cerebral cortex, (Allen & Courchesne, 2003) inferior parietal cortex, supplementary motor area, (Silk et al., 2006), the prefrontal cortex (Müller et al., 2003), and the thalamus (Müller et al., 1998). Structural differences have also been observed in the cerebellum, (Allen et al., 2004) and the striatum, specifically the caudate which has been shown to have increased volume in those with autism (Langen et al., 2009). In addition, Mostofsky et al. (2009) reported decreased functional connectivity (i.e., neural synchronization) among the motor activation regions of the brain for a group of children with ASD as compared to a group of age matched controls.

**Motor Control and Learning in ASD**

Past studies suggest that individuals with ASD are able to execute an aimed motor movement with accuracy comparable to that of typically developing individuals (e.g., Forti et al., 2011; Glazebrook, Gonzalez, Hansen, & Elliott, 2009). However, ASD is associated with increased latency and variability in the time needed to initiate and complete such movements (e.g., Forti et al., 2011; Glazebrook, Elliott, & Szatmari, 2008; Glazebrook, et al., 2009; Nazarali, Glazebrook, & Elliott, 2009). Recent research by Forti et al. (2011) provides additional insight into the mechanisms underlying these group-related differences. In this study, the researchers examined movement kinematics
in preschool children with and without ASD during a simple task requiring them to pick up a ball and drop it in target box. They found that slower movement times for the ASD group were associated with a disruption in the ability to transition from the ballistic component to the corrective component of the movement and a subsequent need for more corrective movements in order to attain the same accuracy as the non-ASD children.

Despite the previously described evidence of neurophysiologic disruption as well as atypical movement kinematics in ASD, behavioral studies have generally reported findings of intact motor learning in individuals with ASD. For example, Barnes et al. (2008) reported that both children with and without ASD showed improved performance (as reflected by decreased reaction time) with practice on a serial reaction time (SRT) task. SRT tasks measure response times over a period of trials where participants are required to push a button in response to a particular stimulus. Interestingly, they also found that, whereas performance of the non-ASD group appeared to level off, the ASD group showed prolonged improvements throughout the test. This contributed to a finding of a significantly steeper rate of improvement overall in the ASD group as compared to the non-ASD group. In studies of adolescents and young adults, Travers, Klinger, Mussey, & Klinger, (2010) and Müller et al. (2003) also failed to find behavioral evidence of ASD-related impairment in learning on a SRT task. Of note, the Müller et al. study used fMRI technology thus allowing for additional examination of the neurophysiological processes underlying task performance and learning. Within this context, they found that, despite intact behavioral performance, individuals with ASD showed atypical activation in the frontal and parietal brain regions during performance of the SRT task.
In another study, Gidley Larson & Mostofsky (2008) utilized a rotary pursuit task to examine motor learning in individuals with and without ASD. The rotary pursuit task, used to measure motor skill learning, involves individuals learning a complex movement sequence or task and also measures one’s ability to anticipate nuances in that task over a period of trials. In the present case, performance on the task is reflected by the amount of time the participant is able to successful keep a stylus on a target as it moved in specific patterns. Consistent with the aforementioned findings using a SRT task, Gidley Larson & Mostofsky found that their ASD and non-ASD groups showed similar performance improvements (i.e., increased time-on-target) with practice. Interestingly, whereas a change in the target’s pattern of movement (i.e., from a circle pattern to a square one) resulted in a marked decrease in performance in the non-ASD group, the change had significantly less effect on the performance of the ASD group. This finding is consistent with the hypothesis that the processes underlying motor learning in individuals with ASD may differ from those associated with motor learning in typically developing individuals.

The Current Study

The goal of the current study was to further advance our understanding of ASD-related differences in motor movement control and learning. To this end, we isolated and examined the ballistic and corrective submovements associated with performance of a rapid aimed limb movement in a sample of children with ASD and a demographically-matched group of typically developing children without ASD. Based on past studies of motor learning in ASD, we hypothesize that children with and without ASD will show similar rates of improvement in overall movement performance (as reflected by total movement time and endpoint accuracy) with practice. Consistent with potential ASD-
related differences in the mechanisms underlying motor learning, however, we also hypothesize that practice will affect the pattern of observed submovements differently for the ASD group as compared to the non-ASD group.

METHOD

Participants

A group of 28 individuals with ASD (26 male, 2 female) ranging in age from 8.1 to 22.8 years ($M = 13.7, SD = 4.01$) participated. These participants were patients receiving clinical services at the University of Missouri Thompson Center for Autism and Neurodevelopmental Disorders, an interdisciplinary academic medical center specializing in diagnosis and treatment of ASD. Diagnostic interviews, caregiver questionnaires, and observation focusing on DSM-IV criteria (American Psychiatric Association, 2000) were used for the diagnosis of ASD in these individuals. Twenty-one participants received both the Autism Diagnostic Interview-Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994) and the Social Responsiveness Scale (SRS; Constantino et al., 2003). Seven participants received only the SRS. Evaluations were conducted by a pediatrician and/or neuropsychologist; if there was disagreement, the results were discussed jointly to reach a consensus diagnosis. Individuals with severe cognitive impairment, learning disorders, major medical disorders unrelated to autism were excluded.

An age-matched comparison group of 49 typically developing individuals (39 male, 10 female) ranging in age from 8.2 to 22.1 years ($M = 13.4, SD = 3.17$) also participated. Control participants were recruited from the Columbia, Missouri community. Prior to enrollment, the parents of potential participants were asked to
complete an extensive questionnaire detailing past developmental and medical history. Individuals with significant medical and/or psychiatric history were excluded.

The brief version of the Leiter International Performance Scale-Revised (Leiter-R, Roid & L. Miller, 1997) was administered to estimate non-verbal intellectual ability. For individuals in the ASD group, standard scores ranged from 82 to 139, with a mean of 101.1 ($SD = 14.5$). For individuals in the control group, standard scores ranged from 82 to 129, with a mean of 106.8 ($SD = 13.1$). The aforementioned difference in IQ scores between the groups was not statistically significant, $t(74) = 1.75, p = .09, d = 0.41$.

**Procedure**

The present study was approved by the University of Missouri-Columbia Internal Review Board. The task was administered in a small, quiet room with sufficient overhead lighting.

The experimental apparatus, stimuli, and procedure at illustrated in Figure 1. Participants sat in front of a computer screen and were fitted with a glove on their right hand to monitor hand position (see details of motion tracking below). Hand position corresponded with cursor position on the computer screen. Direct view of the participant’s hand was obstructed thus necessitating his/her reliance on the cursor display for visual feedback of hand position.

The initial visual display consisted of a small red oval (i.e., the starting point; $0.8^\circ$ wide by $0.6^\circ$ high) located $17.6^\circ$ to the right of the center of the display, a small white rectangle (i.e., the target ending point; $0.9^\circ$ wide by $1.2^\circ$ high) located $6.4^\circ$ to the left of the center of the display, and the aforementioned cursor ($0.2^\circ$ wide by $1.2^\circ$ high) representing the lateral position of the participant’s hand.
The participant initiated the task on each trial by aligning his/her hand (and thereby the cursor) with the starting point on the right side of the display. After a series of four 50 ms, 400 Hz beeps presented at 250 ms intervals, the red starting point oval was replaced by a green cross (0.9° wide by 0.6° high) and a single 50 ms 1000 Hz beep was sounded thus signaling the participant to initiate the hand movement. The participant was then to move his/her hand as quickly as possible and align the cursor with the target ending point on the left side of the display. The total sampling time for each trial was 1500 ms. The location of the cursor on the screen was refreshed every 33 ms.

Prior to beginning the experimental trials, each participant engaged in a demonstration trial to ensure proper understanding of the movement task. To minimize the influence of the demonstration trial on subsequent trials, demonstration endpoint distances were different than trial endpoints. Typically, participants needed only one demonstration trial to understand the task. Demonstrations were limited and were used only to ensure proper understanding of the task. Following the demonstration trial, each participant completed 100 experimental trials with short breaks (<2 minutes) offered every 10 trials.

**Data Processing and Analysis**

Hand position was gathered using a general-purpose, magnetic position and orientation tracking system that provides six degrees-of-freedom, temporal resolution of 100 Hz. and static spatial resolution of 0.5 mm; an Ascension Flock-of-Birds motion tracking system (Ascension, Milton, VT). A previously established algorithm (Abrams & Pratt, 1993) was used to delineate the overall movement and its sub-components. In brief, the movement start was defined as the timepoint when hand velocity exceeded 2.7 cm/s
for a period of 50+ ms. The end was defined as the time point when the velocity of the hand fell below 2.7 cm/s for a period of 180+ ms. With regards to movement sub-components, the end of the initial ballistic movement was defined as the timepoint when one of three criteria were met: (1) the velocity changed from positive to negative; (2) the acceleration changed from positive to negative; or (3) a positive to negative transition occurred in the derivative of the acceleration when the acceleration was negative (an increase in breaking force).

RESULTS

Participants were excluded from analysis if they had an error rate greater than 25% of all trials. That is, if they had at least 75% of usable trials where they did not anticipate the “go” signal, then they were included in analysis. This resulted in exclusion of data from 10 participants (34%) from the ASD group and 16 participants (33%) from the non-ASD group.

Data were grouped into epochs of 20 trials each, and mean performance values (e.g., mean RT) for each “block” were entered into a 2 x 5 mixed model ANOVA with group (ASD and non-ASD) as the between-subjects factor and block (1-5) as the within-subject factor. In addition, simple linear regression analysis was used to calculate the learning slope, or rate of improvement, over the 5 blocks. The resulting values were analyzed using independent samples t tests.

Total Movement Duration

Analysis revealed a main effect of block, with total movement duration decreasing across blocks \( F(4, 196) = 2.56, \ p = .04, \ \eta^2 = .05 \). Total movement duration was not significantly different for the ASD group \( (M = 751 \text{ ms}) \) compared to the non-
ASD group (M = 723 ms) [F(1,49) = 1.07, p = .31, η² = .02]. The group by block interaction also failed to reach significance [F(4, 196) = 1.49, p = .21, η² = .03]. As can be seen in Figure 3, however, there was a non-significant trend towards a steeper learning slope for the non-ASD group (M = -18.6) compared to the ASD group (M = -2.1), [t(49) = 1.86, p = .07, d = .52].

**Ballistic Movement Component**

The proportion of movement time devoted to the ballistic submovement was computed and analyzed. As anticipated based on past research (e.g., Abrams & Pratt, 1993), a main effect of block was observed, with the proportion of time devoted to the ballistic component increasing with practice [F(4, 196) = 7.17, p < .001, η² = .12]. There was no main effect of group [F(1, 49) = 2.35, p = .13, η² = .05]; however, a significant group by block interaction was found [F(4, 196) = 5.12, p = .001, η² = .08]. As shown in Figure 4, the non-ASD group showed a clear learning slope (M = .018) that was significantly different than zero [t(31) = 7.07, p < .001, d = 2.54]. In contrast, the learning slope for the ASD group (M = .001) was significantly smaller than that of the non-ASD group [t(49) = 3.59, p = .001, d = .12] and did not differ from zero [t(18) < 1, p = .82, d = 111.25].

**Corrective Movement Component**

Results from the analysis of the corrective submovement mirrored those from the ballistic movement analysis and are illustrated in Figure 5. A main effect of block was found, with the corrective movement component decreased with practice [F(4, 196) = 5.27, p < .001, η² = .09]. There was no main effect of group [F(1, 49) = 2.96, p = .09, η² = .06], but the interaction between group and block was significant [F(4, 196) = 3.43, p =
As can be seen in Figure 5, the learning slope of the ASD group ($M = -0.001$) was effectively zero [$t(18) < 1, p = .78, d = 0.13$] and significantly different from that observed for the non-ASD group ($M = -0.017$) [$t(49) = 3.00, p = .004, d = .86$].

**Movement Accuracy**

Movement accuracy, as reflected by the absolute value of the difference between the movement endpoint and the target location, was also analyzed. Participants were generally quite accurate across all five blocks (Block 1 $M = .53$ cm; Block 5 $M = .45$ cm). Neither a main effect of block [$F(4, 196) = 1.89, p = .11, \eta^2 = .04$] nor group [$F(1, 49) = 1.65, p = .21, \eta^2 = .03$] was found. The interaction was also not significant [$F(4, 196) < 1, p = .86, \eta^2 = .01$].

**Age-Related Effects**

Additional regression analyses were conducted to determine if the group-related effects observed for the movement subcomponents differed as a function of age. Learning slope (rate of change) served as the dependent variable. Age was entered in the first step of the model followed by group in the second step. The interaction term (age x group) was entered in the third step.

Analysis of total movement duration failed to reveal evidence of an age-related interaction, $\Delta R^2 = .007, F(1, 47) < 1, p = .55$. Analysis of the movement subcomponents yielded similar results, with no evidence of an age-related interaction in either case [Ballistic: $\Delta R^2 = .001, F(1, 47) < 1, p = .83$; Corrective: $\Delta R^2 < .001, F(1, 47) < 1, p = .99$].

**Gender-Related Effects**


Whereas the present ASD sample (which includes only two females) was insufficient to allow for full examination of possible gender-related effects, a sub-analysis of data from only the male participants was conducted. Results were effectively identical to those found with the full sample. Significant group by block interactions continued to be observed for both the ballistic movement component \([F(4, 180) = 4.38, p = .001, \eta^2 = .08]\) and corrective movement component \([F(4, 180) = 3.43, p = .01, \eta^2 = .06]\). In both cases, the learning slope for the non-ASD group (ballistic \(M = .018\); corrective \(M = -.017\)) was also significantly greater than that of the ASD group (ballistic \(M = .001\); corrective \(M = -.001\)) \([t(45) = 3.41, p = .001, d = .99\) for ballistic and \(t(45) = 3.0, p = .01, d = .83\) for corrective].

**DISCUSSION**

Previous findings on motor learning in children with ASD have been mixed. Several studies have shown comparable rates of motor learning in children with and without ASD (Travers, Klinger, Mussey, & Klinger, 2010 and Müller et al., 2003). Others have documented group-related differences in learning rate and motor task-related activation (e.g. Forti et al., 2011) or differences in the processes underlying motor learning in ASD (e.g. Gidley Larson & Mostofsky, 2008). The present study was designed to improve upon these earlier studies by tracking subtle changes in movement kinematics over the course of a fairly long learning period (100 trials). We found that the ratio of ballistic to corrective subcomponents of the total movement effectively did not change in the ASD group across trials while it did in the non-ASD group. There was no main effect of group in the rate of overall learning, but the interaction effects in the ballistic and corrective submovements were significant.
The lack of significant differences in total duration of movement between groups (ASD vs. control) resembles what has been seen in previous studies regarding patterns of motor learning in Autism (e.g. Barnes et al., 2008). Individuals with autism did not show motor learning impairments generally, but the techniques and learning strategies used by persons with autism are nuanced as compared to those without autism. The motor learning task in the present study parcels ballistic and fine motor corrective movements into two separate and measurable aspects of the total movement. Thus a more detailed picture is present in the current study and represents its main contribution to the motor learning literature in autism research. The analysis of the ballistic vs. corrective proportion of movement across blocks reveals that those with autism utilize different strategies of motor learning than those without autism. As expected, the control group increased the amount of time spent in the ballistic portion as they gained practice in the task. The proportion ballistic for the ASD group did not increase as the control group did. In fact, the slope of the proportion ballistic across blocks approached zero, as mentioned above. The ASD group displayed a more rigid learning pattern and did not modify their strategy while the control group did.

A possible explanation of the differences seen in proportion ballistic and corrective submovements in those with ASD may relate to lack of a learning strategy. Those without ASD adapted as trials progressed and devoted more time the ballistic submovement. Those with ASD do not adapt at the same rate as the control group. They incorporate only one strategy in terms of ballistic vs. corrective movements and did not deviate from it significantly. The difference between groups in learning strategy may be
at least in part due to underlying neural differences in both structure and connectivity between groups.

The functional and structural neural differences found when comparing persons with autism with typically developing individuals have been well documented but the interconnecting substrates of the brain also show disturbance (Minshew & Keller, 2010). It is possible that differences in motor learning strategy are due not to deficiency in a specific region of the brain, but rather to deficiency in the connecting paths of the brain that link motor relevant structures. Impairments in paths such as the cortical-cerebellar and cortical-basil ganglia pathways (Gidley Larson & Mostofsky, 2008; Dziuk et al., 2007) may contribute to the spreading activation seen in autism during motor tasks where regions not normally associated with movement show activation. Children with autism rely more on their own proprioception than visual cues from the outside world during motor learning (see Mostofsky & Ewen, 2011 for review). Mostofsky and Ewen (2011) suspect the differences between short fiber connectivity (motor and proprioceptive) versus long fiber connectivity (motor and visual) may account for the overreliance of those with autism on proprioception for motor learning. One can see how a theory of connectivity impairment fits the literature of motor learning in autism, including the inability of those with ASD to change their ballistic vs. corrective movements as compared to controls. Loosely associated with the theory of spreading activation is the hypothesis that earlier developing regions of the brain, primarily sensorimotor regions, infringe on the regions of the brain generally associated with more complex functions (Müller, Kleinhaus, Kemmotsu, Pierce, & Courchense, 2003). The neural mechanisms underlying the differences seen in motor learning are not totally defined and more
investigation in this area is necessary and will continue to complement the behavioral motor research well.

Persons with ASD also show nuance in other forms of learning, specifically verbal learning. Previous research has found that children with ASD are able to use gaze cues and even break down phonologic components of language, yet show impairment in recognizing the social relevance of words and their social meaning, thus impairing verbal learning (Norbury, Griffiths, Nation, 2010). Although learning rates for gaze were similar, typically developing children improved rates of word learning where children with ASD did not. Norbury and colleagues (2010) attribute theoretical causes of such deficits to the neural underconnectivity theory as provided previously and by Belmonte and colleagues (2004). Underconnectivity may potentially account for failure of persons with ASD to transfer of learned information of various modalities into long term storage and may also impair executive functioning (Belmonte et al., 2004).

The current study is not without limitation. There was a subgroup of participants whose data had to be eliminated due to an error rate of over 25% of trials. It is possible that a certain sub-population within the broader ASD categorization is more prone to increased errors on this task. Most errors involved the participant anticipating the “go” signal and invalidating that particular trial. That is, they started the movement before they were prompted to. As is the concern in other lines of autism research, a concern with the present study may revolve around inadvertently excluding a portion of the ASD population such as those who are lower functioning. Another possible limitation also associated with the error rate is the design of the “go” signal. Participants were given a series of beeps to prompt them of the impending “go” signal each trial. This may have
increased the error rate and excluded participants more prone to impulsive or pre-emptive tendencies. A slightly different “go” signal consisting of just one signal may eliminate some of the error seen when 4 pre-emptive beeps are used. Finally, the ASD sample of the current study included only two females, which limits our ability to delineate the sex related differences in motor learning in autism.

Future directions for this line of research may lie in the utility of observing motor impairments in young children and using this data as a contributing factor in early diagnosis. Analyses of motor learning deficiencies in autism spectrum disorders have significant implications for the diagnosis of autism. Diagnosis of ASD is most prominently and proficiently performed, on average, at the age of 3.1 years (Mandell, Novak, & Zubritsky, 2005). It is widely recognized that earlier diagnosis is most beneficial when assessing the course of autism. It is possible that motor difficulties, broadly construed, are a useful tool for early detection of ASD. Motor deficiencies may represent the earliest signs of broader Autism expression later in life. Childhood represents a time of substantial learning but also a time during which the neural system itself is undergoing significant changes. A better understanding of these underlying processes will improve our ability to design and implement more effective intervention, teaching and training procedures. Similarly, evaluation of motor learning longitudinally may also provide beneficial insight into how motor learning changes in those with ASD and how the severity of ASD expression coincides with motor impairment across childhood and beyond.

The two most prominent paradigms used to study motor learning are the motor sequence learning paradigm and the motor adaptation paradigm. Under the motor
sequence learning paradigm, participants are typically asked to practice or repeat a series of movements and under the motor adaptation paradigm participants react to subtle changes in the path of a movement (see Doyon et al. (2009) for review). Another avenue of development in the motor learning literature in autism would be to incorporate motor adaptation in motor learning sequencing. For example, the current study could potentially be modified to include a modification of the learned task half way through the series of 100 trials. Insight on the ability of those with autism to adapt to modifications in the motor learning sequence could lend valuable information to the total picture of impairment in motor learning in autism and build upon the work of Gidley Larson & Mostofsky (2008) and others.

To summarize, the current study fits well into the framework of motor learning paradigms proposed by Pratt, Chasteen and Abrams (1994). For Pratt, Chasten and Abrams, young participants modified corrective movements over time but older participants did not. Differences were seen in the current study in the proportion ballistic vs. corrective between the ASD and control groups. The interesting findings in the current study stem from the differences in learning patterns between groups. While those with ASD did not exhibit motor learning impairments generally, they did fail to alter their movement strategy, incorporating the same proportion ballistic vs. corrective movements across trials. This finding fits in well into the current trends in neural connectivity and neural activation models in ASD. Further investigation in both the behavioral and neural mechanisms of motor learning in autism will advance our understanding of how motor learning differences may be useful in diagnosis, classification, and furthering our understanding of autism.
REFERENCES


Figure 1

*Illustration of the apparatus and procedure.*

![Illustration of the apparatus and procedure.](image)

Figure 2

*Illustration of breaking down of movement into sub-movements.*

![Illustration of breaking down of movement into sub-movements.](image)
Figure 3

*Graph of Total Movement Duration.*

![Graph of Total Movement Duration](image)

Figure 4

*Graph of Ballistic Movement Component.*

![Graph of Ballistic Movement Component](image)
Figure 5

*Graph of Corrective Movement Component.*