

2020-01-01

A Comparative Assessment Of Discrete And Reciprocal Design In Children With Autism Spectrum Disorder

Jallycia Rene Pearson
University of Texas at El Paso

Follow this and additional works at: https://scholarworks.utep.edu/open_etd



Part of the [Kinesiology Commons](#)

Recommended Citation

Pearson, Jallycia Rene, "A Comparative Assessment Of Discrete And Reciprocal Design In Children With Autism Spectrum Disorder" (2020). *Open Access Theses & Dissertations*. 3017.
https://scholarworks.utep.edu/open_etd/3017

This is brought to you for free and open access by ScholarWorks@UTEP. It has been accepted for inclusion in Open Access Theses & Dissertations by an authorized administrator of ScholarWorks@UTEP. For more information, please contact lweber@utep.edu.

A COMPARATIVE ASSESSEMENT OF DISCRETE AND RECIPROCAL DESIGN IN
CHILDREN WITH AUTISM SPECTRUM DISORDER

JALLYCIA RENE PEARSON

Master's Program in Kinesiology

APPROVED:

Jason B. Boyle, Ph.D., Chair

Rhonda Manning, PT, DPT, PCS.

Jeffrey Eggleston, Ph.D.

Christopher Aiken, Ph.D.

Stephen L. Crites, Jr., Ph.D.
Dean of the Graduate School

Copyright ©

by

Jalylcia Rene Pearson

2020

Dedication

This work of many to come is dedicated to all the children diagnosed with Autism Spectrum

Disorder and to my family who have always supported me in my endeavors.

A COMPARATIVE ASSESSEMENT OF DISCRETE AND RECIPROCAL DESIGN IN
CHILDREN WITH AUTISM SPECTRUM DISORDER

by

JALLYCIA RENE PEARSON, BS

THESIS

Presented to the Faculty of the Graduate School of

The University of Texas at El Paso

in Partial Fulfillment

of the Requirements

for the Degree of

MASTER OF SCIENCE

Department of Kinesiology

THE UNIVERSITY OF TEXAS AT EL PASO

May 2020

Acknowledgement

This work would not have been possible without the help and constant support of Dr. Jason B, Boyle. Thank you for your patience and guidance through this journey of working with you

Abstract

Although typically diagnosed through social impairment and repetitive stereotypical behavior, recent work in the last decade has shown motor irregularities across the autism spectrum. With regard to upper extremity coordination, studies have agreed that children on the Autism spectrum present overall decreased performance compared to their neurotypical peers but fail to find commonality on the locus of this error. For example, studies have highlighted reaction time, arm trajectory as well as corrective sub-movements as areas in need of improvement. One possible reason for the conflicting results could be related to the nature of the task employed, e.g. discrete vs reciprocal tasks. Studies have shown that, although simple in comparison, the kinematic composition of goal directed movement under these two conditions varies greatly in the demand of the processing load. Given this, the purpose of the following thesis will be to compare the kinematic composition of discrete and reciprocal aiming in children diagnosed with high functioning autism to their neurotypical peers. Both populations of children (6-12 yrs. old) will be asked to perform a mixture of single (discrete) and continuous (reciprocal) movements between defined target areas. Target distance and width manipulations will provide three index of difficulty values (3, 4, &5) for kinematic assessments. Variables of interest will be: Total movement time, Movement time, peak velocity, % time to peak velocity and end point accuracy.

Table of Contents

Acknowledgement	v
Abstract	vi
Table of Contents	vii
List of Tables	ix
List of Figures	x
Chapter1 Autism Spectrum Disorder	1
1.1 Autism Spectrum Disorder	1
1.1.1 Prevalence	2
1.1.2 Diagnosis	3
1.2 Early Motor Impairment	4
1.2.1 Motor Imitation Impairment	5
1.2.2 Gross and Fine Motor Impairment.....	6
1.2.3 Motor Control Impairment.....	7
1.3 Brain Mechanism	8
1.3.1 Cerebellum.....	9
1.3.2 Basal Ganglia.....	10
Chapter 2 Goal Directed Movements	12
2.1 Goal Directed Movements	12
2.2 Fitts Law	12
2.2.1 Discrete & Reciprocal Paradigm	14
Chapter 3 Movement Structure of Children with ASD	15
3.1 Motor Learning and Control	15
3.1.2 Motor Planning Impairments	15
3.1.3 Motor Execution Impairment	17
3.1.4 Motor Planning and Execution Utilizing Fitts	19
Chapter 4 Methods	22
4.1 Purpose.....	22
4.2 Method	23

4.2.1 Participants.....	23
4.2.2 Apparatus.....	23
4.2.3 Procedure	24
4.2.4 Measures & Data Analysis.....	25
Kinematic Data	25
Chapter 5 Results	26
5.1 Pearson's Correlation	26
5.2 Repeated Mixed Model	28
Total Time.....	28
Peak Velocity	31
Number of Movements	33
Normalized Jerk	35
Time to Total Peak Velocity	35
5.3 One Way ANOVA (Welch T-Test)	35
Peak Velocity	
Chapter 6 Discussion & Conclusion.....	35
List of References	42
Vita.....	52

List of Tables

Table 1 Breakdown of Index of difficulty	23
Table 2. Reciprocal Task. Mean Average of Peak Velocity.....	35
Table 3. Discrete 1 Task. Mean Average of Peak Velocity	35

List of Figures

Figure 1 Experiment set up.....	23
Figure 2 Scatterplot of Index of Difficulty with Movement time of all ASD participants.....	26
Figure 3 Scatterplot of Index of Difficulty with Movement time of all neurotypical participants...	26
Figure 4. Scatterplot of Index of Difficulty with Movement time of all ASD participants.....	27
Figure 5. Scatterplot of Index of Difficulty with Movement Time of all neurotypical participants.	28
Figure 6. Mean Movement time of all participants (ASD group and NT group).....	28
Figure 7 Mean Total Time of width, middle of discrete and reciprocal for All Participants (ASD group and NT group)	30
Figure 8 Mean Peak Velocity of All Participants (ASD group and NT group).....	31
Figure 9 Mean Peak Velocity of width, middle of discrete and reciprocal for All Participants(ASDgroupandNTgroup).....	32
Figure 10. Mean Number of Movements of All Participants (ASD group and NT group).....	33
Figure 11 Mean number of Movements of width, middle of discrete and reciprocal for All Participants (ASD group and NT group).....	34

Chapter 1: Autism Spectrum Disorder

1.1 Autism Spectrum Disorder (ASD)

Autism spectrum disorder (ASD) is a clinically and etiologically heterogeneous neurodevelopmental disorder characterized by early on-set deficits in social communication and interaction, and the presence of stereotyped or repetitive behaviors with restricted interest (Won, Mah, & Kim, 2013; Tick, Bolton, Happé, Rutter, & Rijdsdijk, 2016; Myers, Voigt, Colligan, Weaver, Storlie, Katusic, Port, 2019). According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) these symptoms must be present in the early developmental period ,however, may not become fully manifested until social demands exceed limited capacities or may be masked by learned strategies in life. ASD is known as a wide-spectrum with variability within the severity of the symptoms; thus, no two diagnoses are the same (Paquet, Olliac, Golse, & Vaivre-Douret, 2016).

The causation of ASD is the subject of intense investigation, with researchers implying various environmental factors and genetic susceptibilities for the increased risk of ASD (Landrigan, 2010; Kreiser & White, 2014). ASD is characterized as heterogeneity with estimated 300-1000 genes that are targeted which are due to variety of genetic factors such as gene mutations, gene deletions, copy number variants (CNVs) and other genetic anomalies (Landrigan, 2010; Packer, 2016). Although, genetics contribute to ASD there are discrepancies in explaining certain clinical and epidemiological aspects of ASD that led to the possibility of environmental exposure to play a role in the causation (Landrigan, 2010; Daniels, 2006; Santangelo & Tsatsanis, 2005). There is support by many researchers that there is a need for understanding of the exquisite vulnerability of the developing human brain to toxic exposures in the environment such as lead; methylmercury; polychlorinated biphenyls (PCBs); arsenic, manganese; organophosphate

insecticides; (DDT); and ethyl alcohol (Landrigan, 2010; Sealey, Hughes, Sriskanda, Guest, Gibson, Johnson-Williams, Pace & Bagasra,2016). Strong evidence supports the idea that various genetic factors alone or in combination with environmental factors could be the eventual causation of abnormalities in brain formation inducing ASD behavior (Bailey et al., 1995; Bolton et al., 1994; Wing and Potter, 2002).

1.1.1 Prevalence

The global prevalence of autism has increased twentyfold to thirtyfold since the earliest epidemiologic studies were conducted in the late 1960s and early 1970s (Baio, 2014). The number of diagnoses of ASD has increased throughout the world due to the following diagnostic criteria over time, new assessment instruments, inaccurate diagnoses, and utilizing different research methodologies to identify prevalence estimates (Fombonne, 2009; Matson & Kozlowski, 2011). This increase is seen especially in developed countries compared to developing countries due to the awareness and proper diagnosis (Adak& Halder, 2017). According to the Center of Disease Control’s Autism and Developmental Disabilities Monitoring Network, the most recent prevalence about 1 in 54 children have been identified with autism spectrum disorder (ASD).

Within the United States, there are more individuals than ever before being classified as having ASD (Baio, Wiggins, Christensen, Maenner, Daniels, Warren, Kurzius-Spencer, Zahorodny, Rosenberg, White & Durkin, 2018). ASD has emerged as a major focus of public health concern in the U.S as an increasing number of families seek educational, social, and health care services to deal with its widespread impact (Lyll et al. 2017). In 2010, ASD accounted for 7.7 million disability adjusted life and was the leading mental cause of disability in children under five in terms of years lived with disability (Baxter, Brugha, Erskine, Scheurer, Vos & Scott, 2015; Modabbernia, Velthorst & Reichenb). It has been notes that the annual total costs associated with

ASD in the United States have been estimated to approach \$250 billion, with lifetime individual ASD-associated costs in the \$1.5 to \$2.5 million range (estimates in 2012 US dollars) (Buescher, Cidav, Knapp, & Mandell, 2014). In the year 2015, the ASD economic burden estimated at 286 billion, including the direct and indirect costs; this amount is expected to increase every year, estimated \$461 billion for the year 2025 (Leigh & Du, 2015).

1.1.2 Diagnosis

There is no specific biochemical indicator or distinct neuroanatomical abnormality that defines autism, and diagnosis is based on clinical and behavioral assessment (Landrigan, 2010). The early assumptions of an autism diagnosis based on observation of overt signs of motor impairments and the apparent grace and skill in spontaneous movements in many autistic children (Jones & Prior, 1985). Even so, there is some evidence that when actually tested, autistic children do not show motor development consistent with their chronological age level (DeMyer, Hingtgen & Jackson, 1981). A longitudinal study on infants with older siblings with ASD described the first signs of autism as unusual motor movements, unusual response to sensory stimuli and unusual visual preoccupations emerging between 9 and 12 months of age; while between 12 to 24 months, disturbance in temperament and regulations of activity, mood, and sleep emerged along with intellectual disability and social and communication disturbances (Zhang & Roeyers, 2019; Rogers, 2009). Also, a meta-analysis focused on motor impairment in ASD found that motor deficits were clearly present in both upper and lower extremities in children with ASD. (Fournier, Hass, Nalk, Lodha, & Cauraugh, 2010).

Although ASD is not associated with severe motor disturbance, motor deficits including alterations in motor milestone development are reported (Teitelbaum, Teitelbaum, Nye, Fryman, Maurer, 1998). Through standardized motor tests used to assess movement profiles, known as

Henderson test of Motor Impairment, the Bruininks-Oseretsky Test of Motor Proficiency and Movement Assessment Battery for Children have reported inconsistent results that inherent difficulties in characterizing the movement profile of ASD individuals (Papadopoulos, McGinley, Tonge, Brashaw, Saunders, & Rinehart, 2012). The kinematic methods of the ASD profile give insight into a disrupted motor function that aids the hypotheses of underlying brain dysfunction and cerebellar dysfunction (Papadopoulos, McGinley, Tonge, Brashaw, Saunders, & Rinehart, 2012).

1.2 Early Motor Impairment

The motor development of toddlers and preschool age children with ASD has emerged as an area of interest due to the increased need for early diagnosis and the increasing evidence that children with ASD exhibit atypical motor characteristics (Lloyd, MacDonald & Lord, 2013). The initial observations by Kanner (1943) referred to unusual motor characteristics (Paquet et al., 2016). Kanner observed several children in group of 11 and described ASD primarily as severe impairment in social-emotional and communication ability; also commented on several aspects of motor development: motor milestones were generally within normal limits and fine motor coordination was “very skillful”, although some patients had gross motor deficits (Sacrey, Germani, Bryson, & Zwaigenbaum 2014; Paquet et al., 2016). In a prospective study, with a sample of children 3-6 months of age Baht and colleagues (2012) investigated gross motor development of 24 infants with siblings with ASD and 24 infant low risk for autism using the Alberta Infant Motor scale (AIMS; Piper & Darrah, 1994) and the Mullen Scales of early learning (MSEL; Mullen, 1995). Resulted in significantly more infants with siblings with ASD showed motor delay at 3 to 6 months than the low risk infants. These majority of the infants with siblings with ASD showed both early motor delays and later communication delays (Bhat, Galloway, &

Landa, 2012). Another prospective study of infant, 71 High Risk infants defined by presence of ASD in older sibling, compared to 25 low risk infants with MSEL (Mullen, 1995), ADOs Toddler Module (ADOS-T; Lord et al 2005) and ADOS- G (Lord et al 2000) (Rowberry, Macari, Chen, Campbell, Leventhal, Weitzman & Chawarska, 2015). There was significant difference in motor delay between the groups at 12-months-old infants later diagnosed with ASD, and at 24 months-old. The imitation construct was highest in infants later diagnosed with ASD that taps into early emerging motor, vocal and social imitation skills. These results suggested that 12 months HR-infants later diagnosed with ASD are more atypical with regard to their social and communicative skills (Rowberry et al, 2015)

1.2.1 Motor Imitation

For the most part the use of motor skill performance in an ASD diagnosis is embedded in gestures, stereotypies, and imitation (Lord et al., 2000; Luyster et al., 2009). One of the commonly affected areas in children with ASD is the ability to imitate the actions of others, particularly when requested to do so. (Shih, Shen, Ottl, Keehn, Gaffrey, Muller & 2010; Hobson & Lee, 1999; Rodgers, Bennetto, McEvoy & Pennington, 1996; Vivanti, Nadig, Ozonoff & Rodgers, 2008). This is known as motor imitation which is a complex developmental phenomenon that is essential for learning new behaviors and transferring cultural knowledge between individuals (Whiten, 2009).

During infancy, this emerges in early development and plays a critical role in development of cognitive and social skills and associated with development of language (Meltzoff & Moore, 1997; Brooke & Schreibman, 2006). From a social perspective, infants are able to detect that others “like me” that lead to later understanding of others’ intentional behavior and the development of theory of the mind (Meltzoff & Gopnik, 1993; Meltzoff & Moore, 1999). While

from a cognitive perspective, imitation is described as a precursor for symbolic functioning, as well as a learning strategy through which infants acquire and master new behavior (Piaget, 1962; Meltzoff & Moore 1983; McDaffie, Turner, Stone, Yoder, Wolery, & Ulman, 2006). In this respect, imitation allows for development of social relationships by enhancing interpersonal bonding by signaling and generating feelings like affiliation with others. (Chartrand and Bargh 1999; Lakin and Chartrand 2003, Van Baren et al. 2009; Wild, Poliakoff,, Jerrison & Gowen, 2012).

1.2.2 Fine and Gross Motor Impairments

Within most motor skills studies individuals with ASD frequently have motor disorders that can be classified as gross or fine in nature (Downey & Rapport, 2012; Fournier et al., 2010). Fine motor skills are small movements that are produced by the body's small muscle groups such as the hands, fingers, toes, wrists, and other small muscles (Schmidt & Lee, 2011). While gross motor skills use large muscle groups such as the arms, legs, and trunk to move that body and engage in activities (Schmidt & Lee, 2011). Provost et al (2007) administered two batteries of Bayley and Peabody scales tests to evaluate motor development in young children with ASD, children with developmental delays and children without delays, and they observed motor disturbances (gross motor delay, fine more delay or both) in more than 60% of children with ASD. (Provost, Lopez, & Heimerl, 2007). While in cross-sectional study of 172 young children ranging from 14-36 months with ASD fine and gross motor skill deficits became significantly worse within a short chronological timeframe (6–18 months) (Lloyd et al., 2013). Also, confirmed that the same children assessed approximately one-year apart displayed significantly motor skill deficits as they aged (Lloyd et al., 2013). Various researchers have noted differences of gross and fine motor skills in school-aged children with ASD; including reduced stride lengths and increased stance times (Vilensky, Dama-sio, & Maurer,1981), g manual dexterity, ball skills, and balance (Green, Baird,

Barnett, Henderson, Huber & Henderson, 2002; Manjiviona & Prior,1995) locomotor and object control(Berkeley, Zittel, Pitney, & Nichols,2001), and reach to grasp tasks that included movement execution and planning (Mari, Castiello, Marks, Marraffa, & Prior, 2003). However, Mayer and Calhoun (2003) reported that 66% of their sample of 3-15-year-old children with autism had normal motor milestones for walking independently; however, these were collected by retrospective parent report rather than by standardized testing (Mayes & Calhoun, 2003). Even though research consistently found that infants and children with ASD experience both gross and fine motor delays, and/or a-typical motor patterns there are some discrepancies due to the wide variability among this disorder.

1.2.3 Motor Control Impairments

Motor control is the exploring the physical and physiological process on how the central nervous system (CNS) produces purposeful, coordinated movements in its interaction with the rest of the body and with its environment (Latash, Levin,Scholz & Schöner 2010). To further explain, planning process are responsible for selecting the appropriate motor program from intended action conversely supporting movement execution through monitoring discrepancies and generating corrections (Forti, Valli, Perego, Nobile, Crippa & Molteni, 2011). Motor deficits in individuals with ASD have shown to alter motor planning and execution of movements (Dowd, McGinley, Taffe & Rinehart, 2012; Rinehart et al., 2006). Forti et al (2011) examined preschooler diagnosed with ASD performing a reach and ball drop task. They analyzed the upper limb movement kinematics of preschooler's performance and compared those results to the normal developing preschoolers. By examining the kinematics, there was no significant difference within the primary movement (planning-based) however there was a significant difference in the corrective submovement (control based) suggesting disruption in the planning-control integration

Conversely, Dowd et al. (2012) examined children diagnosed with ASD between the ages of 3 – 7 years performing a point to point aiming task. They analyzed the upper limb movement kinematics of the children’s performance and compared them with normal developing children. Between the two groups, there was a significant difference in movement preparation suggesting motor planning impairments. This outlines how deficits in movement preparation and execution could lead to many behaviors exhibited by individuals with autism (Leary & Hill, 1996).

Research demonstrates deficit in wide range of motor skill, including fine and gross motor coordination, performance of skilled gestures, and imitation, and subtle neurological signs within children with autism spectrum disorder (ASD) (Hilton, Zhang, Whilte, Klohr & Constantino, 2012). A meta- analysis concluded ASD is associated with significant and widespread alterations in motor performance, suggesting that motor deficits are potential core symptom of ASD (Fournier, Hass, Naik, Lodha, & Cauraugh, 2010). These characteristics are often downplayed while the impact is significant on relationships with others and in other skills as adaptability to the environment or cognitive tasks (Paquet, Olliac, Bouvard, Golse, & Vaivre-Douret, 2016).

1.3 Brain Mechanism

Autism Spectrum Disorder has a prevailing theory that the abnormalities in the neuronal system and social brain network cause disturbances within the neurobiological mechanisms (Lee, Kyeong, Kim & Cheon, 2016; Minshew & Keller 2010; Nebel Joel, Muschelli, Barber, Caffo, Pekar, & Mostofsky, 2014). There are multiple studies that have found reduced functional connectivity within the brain regions, major networks (functional integration), and altered connectivity in different networks (functional segregation) with individuals with ASD (Rudie, Brown, Beck-Pancer, Hernandez, Dennis, Thompson, Bookheimer, & Dapretto, 2013). The structural and functional abnormalities within the brain network is reflected by the difficulties with

motor execution; with evidence of a positive correlation with motor disability and the abnormality in the development of frontal white matter, specifically left motor and premotor white matter (Nagae, Zarnow, Blaskey, Dell, Khan, Qasmieh, Levy & Roberts, 2012; Schmitz, Rubia, Daly, Smith, Williams, & Murphy, 2006). There are other reports of abnormalities in the brain anatomy within this disorder, in particular, frontal, limbic, basal ganglia, parietal, and cerebellar regions (Schmitz, Rubia, Daly, Smith, Williams, & Murphy, 2006; Bauman & Kemper, 2005).

1.3.1 Cerebellum

The cerebellum has been highlighted due to the relative frequency of cerebellar anatomic abnormalities found in individual with ASD (Marko, Crocetti, Hulst, Donchin, Shadmehr, & Mostofsky, 2015). These abnormalities within the cerebellum vary from reduced Purkinje cell density, abnormal size and number of Purkinje cells, smaller cerebellar vermis or enlarged cerebellar hemisphere volumes. (Ritvo et al., 1986; Whitney et al., 2008; Bauman & Kemper, 2005). The cerebellum is known to connect with many cortical and subcortical structures in the cerebral hemisphere, and acts as a modulator for many cognitive, language, motor, sensory and emotional functions associated with these regions (Schmahmann & Pandya, 1997). The cerebellum monitors the position of the trunk and limbs in space, information from the premotor cortex about the programmed (intended) movements and from the ascending tracts about actual movement. (Nayate, Bradshaw & Rinehart, 2005).

Within a healthy, typically developing (TD) individual's cerebellum, is thought to be involved in constructing internal models of surrounding environment and one's position in space, including kinematic state. While in an individual with subtle cerebellar abnormalities, this may affect refinement or information that would impair temporal order of events and precision in movements (Cerminara, Apps & Marple-Horvat, 2009; Paulin, 2005; Paulin & Hoffman, 2011;

Cullen et al., 2011; Leggio et al., 2008; Lawson, Rees & Friston 2014). During goal directed movements, the internal model of control of movement suggest the requirements of anticipated movements are predicted and subsequently encoded by the cerebellum (Corben et al., 2011; Ito, 2008). Also, it has been noted that the cerebellum in conjunction with the cerebral cortex, acts as a “comparator”, detecting discrepancies between the predicted and actual movement via visual and proprioceptive feedback, that allows for altering subsequent movement (Corben et al.,2011; Ito, 2008). Given that there are many of the functions of the cerebellum and connections with the cortical and subcortical regions of the cerebral hemisphere, it is likely the abnormalities of the cerebellum significantly contribute motor development displayed by this disorder

1.3.2 Basal Ganglia

Furthermore, there is increasing evidence that associates different areas of the basal ganglia with that of ASD impairments in development of social skills, communicative skills, motor skills and repetitive, and stereotyped behavior (RSB) that encompasses a broad range of symptom such as motor mannerisms, unusual preoccupations and interests, extreme rigidity and insistence on sameness (Estes, Shae, Sparks, Friedman, Giedd, Dawson, Bryan, & Dager, 2011; Qui, Adler, Crocetti, Miller & Mostofsky, 2010). The basal ganglia consists of large subcortical nuclear masses, which is comprised of the caudate nucleus, the nucleus accumbens, the putamen, and the globus pallidus, are involved in psychomotor behavior that optimize control of actions based on learned response-reward associations, and contribute to acquisition and subsequent coordination or motor, cognitive, and social-emotional control (Ring & Serra-Mestres, 2002; Qui, Adler, Crocetti, Miller & Mostofsky, 2010; Ring & Serra-Mestres, 2002). The basal ganglia are known to cause skilled movement disorders in Parkinson and Huntington’s patients, that during a handwriting experiment, these patients had difficulty in preparing movements, and therefore, the

movements were laborious rather than smooth and effortless. This finding suggests the basal ganglia has a role in scheduling submovement which allows for automatic execution of serially ordered complex movements (Phillips, Bradshaw, Iansek, & Chiu, 1993). While analyzing and comparing the basal ganglia between neural typical children and children with ASD, there was no volume difference however there were localized shape differences right caudate, putamen and globus pallidus (Qui, Adler, Crocetti, Miller & Mostofsky, 2010). The putamen function is sensory guided for goal directed movements and sequencing skilled movements while the caudate is helps executed appropriate social behaviors and emotional control (Qui, Adler, Crocetti, Miller & Mostofsky, 2010). Given that many of the functions of basal ganglia, it is likely the abnormalities impact motor development displayed by this disorder.

However, even though various motor coordination and programming deficits have been recognized, ASD is not a syndrome that is perceived with obvious motor impairments (Ming, Brimacombe & Wagner, 2007). Consequently, the delayed onset of motor capabilities and coordination deficits with a distinct association to ASD are of fundamental importance. The following section will have a more detailed description of the mathematical equation used to analyze motor planning as well as execution. As well as the evaluation and the relevance to ASD.

Chapter 2: Goal Directed Movement

2.1 Goal Directed Movements

A goal directed movement is a type of autonomous movement in which the agent contingently directs its movement toward (or away from) another object, state, or location (Opfer, 2002). R.S Woodworth's studied goal directed movement characteristics through an aiming procedure in which participants made horizontal sliding movements between two fixed distances or matched their amplitude of movement to the previous attempt. Thus, examining spatial accuracy and consistency of the movement endpoints as well as the spatial and temporal trajectories. He concluded that goal directed movements are composed of two distinct phases, an initial impulse phase and a current control phase (Woodworth, 1899; Elliot, Helsen,& Chua ,2001) The initial projection phase dependent on planning processes design to bring the limb to vicinity of the target. Then once in the region of the target, the limb comes under current or feedback- based processes; this is known as the second "homing" phase, visual and proprioceptive information about the relative position of the limb and target to make adjustments to the movement trajectory (Woodworth, 1899; Elliot, Helsen,& Chua ,2001). Through Woodworth work, he came with the fundamental property of human motor behavior which is the tradeoff between directed movements (Woodworth, 1899).

2.2 Fitts Law

Fitts provided the first quantitative description of the relationship of speed and accuracy in self-paced, cyclic tapping movement and discrete aiming movement (Fitts, 1954; Fitts & Peterson 1964).). Fitts described a linear relationship between the effects of index of difficulty (ID) has on movement time (MT) (Fitts, 1954; Fitts & Peterson 1964). This "speed accuracy tradeoff" implies that one has to adapt to this double constraint in all situations, either by accepting

an increase in time spent during a movement, or by accepting to achieve a lesser degree of terminal accuracy (Fitts, 1954; Fitts & Peterson 1964; Bootsma, Fernandez & Mottet, 2004). Fitts (1954) formulated this linear relationship by this following the equation: $MT = a + b [\log_2 (2A/W)]$ where MT is the movement time between the two targets, and a and b are empirically derived constants. The ID is represented by the term $[\log_2 (2A/W)]$, where W is the width of each target in the plane of movement and A is the distance between the centers of each target known as the amplitude (Smits-Engelsman, Van Galen & Duysens, 2002). Through this relationship, Fitts reduced the complex relation between environmental constraints and motor control to a one-dimensional problem relating two scalar variables that captures the essence of goal directedness (Huys, Fernandez, Bootsma, & Jirsa, 2010).

There have been efforts to explain the mechanism of the speed-accuracy trade-off. Meyer et al. (1988) suggested that goal directed movements consist of an initial and a secondary phase, each operating under different control processes. The initial impulse “primary movement” is thought to pre-planned to end of the location of the target; While the secondary phase known as the error correction phase is based off feedback information using visual and proprioceptive feedback (Meyer, Abrams, Kornblum, Wright & Smith; Elliott, Helsen & Chua, 2001; Chua & Elliott, 1993). Meyer and colleagues proposed that there is a compromise between initial movement speed, in which faster movements are more likely to induce spatial errors, and corrective sub movement, which may increase accuracy by decreasing limb velocity (Meyer, Abrams, Kornblum, Wright & Smith, 1988; Forti, Valli, Perego, Nobile, Crippa & Molteni, 2011). Along the same lines, Glover’s theory proposed that the action goal and target characteristics influence the appropriate motor program selected by the planning processes and that control processes support movement execution, by monitoring discrepancies among the motor plan, actual

movement and the target, as well as by quickly generating correction for spatial errors (Glover, 2004; Forti, Valli, Perego, Nobile, Crippa & Molteni, 2011).

2.2.1 Reciprocal and Discrete Paradigm

Fitts' law has been shown to hold when rhythmically pointing at two targets, as in experiment one of Fitts' original paper (Fitts, 1954) and when moving a pointer to a target and stopping there (Fitts & Peterson, 1964). However, there is one important empirical finding when comparing the discrete and reciprocal case of the paradigm is that the slope of Fitts' law differs, being steeper in the reciprocal case (Guiard, 1997). Researchers have shown when comparing discrete and repetitive point to point movements for the same IDS kinematics (time to peak velocity, MT) differs within the first and last half cycles of motion in a repetitive aiming action and discrete point to point aiming actions (Mourik and Beek 2004; Buchanan, Jin-Hoon & Shea, 2006). These observations can hardly be accounted for using informational logic behind Fitts Law; because all the components of the interaction are exactly the same, the difference is to be sought in the way movement execution is controlled by the nervous system (Bootsma, Fernandez & Mottet, 2004).

Many motor activities (walking and tapping) are cyclic and have been proposed that these movement use some type of neural oscillator (Smits-Engelsman et al., 2002, Zehr and Duysens, 2004). These movements differ from activities such as aiming and pointing known as discrete task. Even though both these tasks comply with Fitts law (Fitts 1954; Fitts & Peterson 1964), cyclic movements are executed more in a ballistic fashion than discrete movements, this would indicate that these movements are more preplanned and rely on feedback controlled corrections (Smits-Engelsman, Sugden, & Duysens, 2006).

Chapter 3: Movement Structure of Children with ASD

3.1 Motor Learning & Control

In terms of understanding movement behavior, a distinction between movement skill and motor ability is crucial (Staples & Reid, 2010). A movement skill consists of a goal-directed movement, specific purpose such as throwing a ball which can be described according to a final outcome or movement pattern used (Burton & Miller 1998; Magill, 1998). These skills considered “fundamental” are assumed to be the basis of more advanced or sport specific skills (Burton & Miller 1998; Staples & Reid, 2010) Contrarily, motor abilities refer to the capacity to perform a movement skill (Magill, 1998). Motor abilities are not directly observed rather inferred from the performance of movement skills such balance or coordination (Magill, 1998). Therefore, good motor skills are important for a range of everyday abilities.

Although motor impairments in Autism Spectrum Disorder are not considered to a core feature, there is an increasing acknowledge that can significantly impact on the quality of life and social development (Gowen & Hamilton, 2012). Research has consistently associated people with ASD with motor impairments and poor movement skills, thus indicating poor motor abilities as well (see review Sacrey et al., 2014; Staples & Reid, 2010). In the following sections movement behavior of upper limb goal directed movements in people with ASD is going to be further explained.

3.2 Motor Planning Impairments

Motor planning is the process of converting a current state (my hand is by my side) and a desired state (my hand should be on the mug) into a sequence of motor commands (Gowen & Hamilton, 2012). Planning often begins before a movement is initiated, but the inverse model continues to control action and correct error during execution (Gowen & Hamilton, 2012). The

simplest way to assess motor planning is study reaction time before a movement is performed, which provides a basic measure of time take to formulate a motor plan (Gowen & Hamilton, 2012).

Rinehart et al (2001) examined adolescents with ASD, with HFA and who are NT between the age 10-18 years old, who performed rapid reciprocal aiming movements to two targets on a response board. The task required movements that involved only the four pairs of buttons; they were required to move between a start position at the bottom center of horizontal digitizing tablet to a target either top left or right in response to an illuminated light. The children showed anomalies in movement preparation, their pattern of anticipation was not reflecting in their preparation. These findings are comparable with those of Hughes (1996) who presented that autism is associated with an atypical planning and anticipation of a motor response. The ASD group, deficit in movement preparation at the point where movement preparation should be optimal implies a disturbance of the AC/supplementary motor area (SMA) circuitry (Rinehart, Bradshaw, Brereton & Tonge, 2001). This is suggesting that this disturbance would result in difficulty internally initiating or generating a motor program (Pantelis & Brewer, 1996; Rinehart, Bradshaw, Brereton & Tonge, 2001).

Similar to the previous study, Rinehart et al (2006) examined the kinematic movements of children diagnosed with ASD and the impact of an executive load on movement kinematics by including expectancy and inhibitory components. The task included a digitized tablet, where the targets were positioned in the top left- and right-hand corners and the start position positioned at the bottom, center of the table. The kinematic task comprised of three different levels that involved a response to the illumination of the left or right led target. In all three conditions, the participants with ASD displayed a clear motor preparation deficit compared to the control. Level 3 condition showed similar results from the previous that regardless if the movement was predictable or

unpredictable; This was seen in the previous study suggesting a lack of anticipation for individuals with ASD (Rinehart, Bradshaw, Brereton & Tonge, 2001)

Dowd et al. (2012) reported similar results in children with ASD between the ages 3 – 7 years when they performed a point-to-point aimed task. They analyzed the time kinematics of the children's performance and found that the ASD group displayed substantial variability in their movement preparation compared to the NT group, suggesting motor planning impairments. They found that the preparation phase was significantly different with increased variability in the time taken to prepare simple point to point movement (Dowd, McGinley, Taffe, & Rinehart ,2012). However, with the visual distractor present there was no differences in the motor execution or preparation phase among the children with ASD, suggesting that alternative motor plans are not taken due to a deficit in visual perceptual integration (Dowd, McGinley, Taffe, & Rinehart ,2012).

3.3 Motor Execution Impairments

Mari et al. (2003) reported that children with ASD between the ages of 7-13 years old did not display problems with their motor planning in a reaching or grasping but displayed impairments in their homing- in phase. Children were instructed to reach and grasp either a large or small cube that was positioned 18cm or 28cm in front of them. There were significant kinematic differences among the low ability group and high ability compared to the average group. The low ability group displayed bradykinesic motor pattern with longer movement duration, longer deceleration time, longer amplitude peak velocity, and time of maximum grip aperture. While the high ability group children reach for objects more rapidly than neural typical children of the same age. Therefore, suggesting children with ASD developed a movement strategy but unable to regulate and process the concurrent visual online feedback during the execution of the movement (Mari, Castiello, Marks, Marraffa & Prior 2003).

Similarly, Forti et al. (2011) investigated the planning and control processes of 12 preschool children with ASD. The purpose was to examine the children's ability to transport an object from one location to another and drop into a hole, actions resembled a reaching movement requiring the hand to move from set location to a target location. There were no significant variations among the groups in the initial movement phases or the accuracy, however, children with ASD showed higher velocities, additional corrective sub-movements, and motor slowness in the homing phase. The additional sub-movements implied that children with ASD performed the homing-in phase with a less efficient structure, even with achieving similar levels of accuracy compared to the control group. The presence of additional sub-movements in the secondary phase of the movement task suggested that children with ASD have feedback processing impairments and/or lack the capacity to create a complete motor plan prior to executing the movement (Forti, Valli, Perego, Nobile, Crippa & Molteni, 2011).

Lastly, Stoit et al. (2013) suggested that motor deficits in ASD originate in the functional mechanism that support movement execution. They investigated 31 children and adolescent in a two-choice reach and grasp paradigm in which participants received cuing information indicating either the object location or the required manner of grasping. There were no significant variation in the reaction times and performances however the ASD group movement times were significantly delayed in comparison with the controls. The movement time delays were a reflection on the execution phase; Suggesting there is deficit in action chaining derived from impairments in functional mechanism that support feedforward model (Stoit, Van Schie, Slaats-Willemsse & Buitelaar, 2013).

3.4 Motor Planning and Execution Analysis Utilizing Fitts Paradigm

Researchers have shown that within ASD, there are delays and deficit in motor functioning that impact their lives. However, there are not many studies that utilize Fitts Law, a simple manual aiming movement, to gain a better understanding what is different about their movements from typical developing individuals (Glazebrook, Elliot, & Lyons, 2006). Fitts law is known as one of the most lawful and robust relations in human movement sciences, linking the time required to complete a pointing movement to the difficulty of the task as defined by the amplitude and precision constraints imposed (Bootsma, Fernandez & Mottet, 2004). Through these limited studies, there are discrepancies that are seen in the online control.

Glazebrook, Elliot, and Lyons (2006) examined young adults with ASD and young adults who are NT between the ages of 20 – 30 years, who performed rapid discrete aiming movements to one of two targets. The authors investigated Fitts law by using different levels of movement difficulty by modifying both target size and distance. When compared to the NT group, participants with ASD displayed more spatial and temporal variability in the first phase of the movement, indicated by the movement time at the different indices of difficulty, but were able to maintain end-point accuracy. This was seen when the participants with ASD had reached half the accelerations even for longer movements were the target was further away, those accelerations were not the same degree as neural typical young adult group. They required more time to prepare their movement, this was seen in the slower reaction time and movement times. These young adults with autism have are atypical in motor skills and performance in a simple straightforward manual movement with reduced spatial variability by decreasing their initial impulses during the control phase (Glazebrook, Elliot, & Lyons, 2006).

In a follow up experiment, Glazebrook et al. (2008) examined young adults with ASD between the ages of 17 – 30 years. The authors used two experiments to investigate planning manual aiming movements when advance information is direct and required strategic planning. The protocol was similar to the one used by Glazebrook et al. (2006), but in this experiment participants chose their start position between the two targets anticipating the light signal for either the left or right target. Participants with ASD compared to participant without autism, would consistently select the midpoint as their starting position and displayed longer reaction time and movement time. The autism group planned their movement when the instruction were direct (Glazebrook, Elliott & Szatmari, 2008).

A novel experiment conducted by Papadopoulos and colleagues (2012) investigated repetitive Fitts aiming task in upper limb motor function of high functioning autism and Asperger's disorder children compared to neural typical children. The children between the age of 7 and 12 years were instructed with a stylus to draw 10 continuous straight lines between two targets with 4 IDs. The results showed there were no significant difference in movement time among the three groups. However, the high functioning autistic children made the same amount of errors in two conditions of equal difficulty, suggesting the autistic children approach motor task may differ from Asperger's and neural typical children. Also, the high functioning autism individuals' results seem to better follow ID associated with Fitts' Law compared to other two groups. Overall, the study showed that the children with high functioning autism produced more scattered movements within their endpoints (Papadopoulos, McGinley, Tonge, Bradshaw, Saunders, & Rinehart, 2012). Similar findings of Forti et al. (2011), participants with ASD showed more variability of movement during the homing-in phase of the task compared to the NT group. The difficulty to plan subsequent movements or lack of feed-forward control could be explained by an underlying

cerebellar disturbance, which could explain the difficulties in planning and regulating movements during the homing-in phase of a goal directed movement (Mosconi, Mohanty, Greene, Cook, Vaillancourt & Sweeney, 2015).

Chapter 4: Methods

4.1 Purpose

There is a gap in the literature of overall movement construction of children with ASD within Fitts Law. Previous studies analyzing the movement kinematics of people with ASD have inferred that they suffer disturbances in the overall structure of their movements suggesting impairments in both cognitive processes, motor planning (Glazebrook et al., 2006; Glazebrook et al., 2008) and motor control (Mari et al., 2003; Papadopoulos et al., 2012; Rosenbaum, 1991).

Therefore, our study had several goals. First, we wanted to further the understanding of the kinematic components of goal directed upper limb movement in children diagnosed with ASD children. Specifically, we wanted to determine where in the construction of their movements were deficits in motor control compared to children who are neural typical. Second, we wanted to investigate the discrete and reciprocal task of ASD children limb movements. This purpose is warranted given the overwhelming lack of investigations of this nature. We were especially interested in determining the similarities and differences. This was considered especially important if participants with ASD show differences in movement kinematics compared to participants who are NT.

There were two hypotheses to investigate in this study. First, participants with ASD will exhibit motor deficits in goal directed aiming with discrete and reciprocal tasks. Specifically, these deficits will be seen in the secondary corrective phase of the movement or homing in on the target. Second, participants between the ASD group and NT group will exhibit kinematic differences among both tasks.

4.2 Method

4.2.1 Participants

Participants (N=17) were children aged 6-12 recruited from the local El Paso community. The population consisted of seven children who are Neural Typical (NT) as well as ten children diagnosed with high functioning Autism Spectrum Disorder (ASD). Due to the nature of the population, guardians were responsible for reading and signing the informed consent, and they were encouraged to take part in the instruction phase of the experiment. Guardians were also required to be present at all times. The guardians and participants were advised that if at any time the guardian or participant would like to leave the laboratory or terminate their participation in the experiment, they were free to do so at no penalty to them.

4.2.2 Apparatus

The participants sat comfortably at a small rectangular table in a height adjustable chair. A mouse, which was used to collect upper extremity data, was placed flat on the right side of the tabletop. A 42in mounted television screen displaying the task/performance feedback was wall mounted at eye level. The task (Figure 1 –right) was custom developed and displayed through Movable Software.

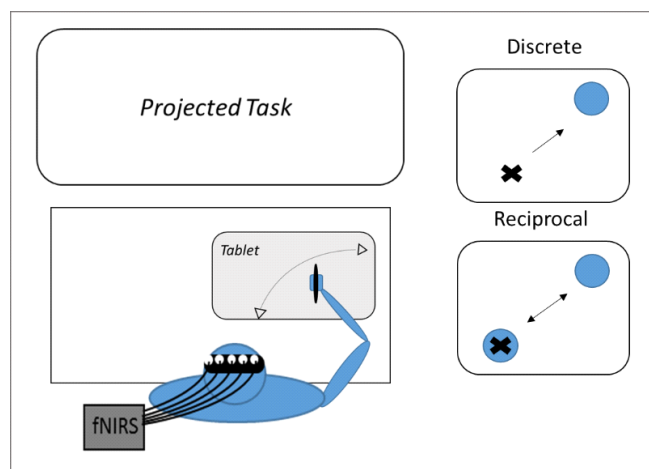


Figure 1: Experimental set up (left) with visual depiction of the 2 tasks (right)

4.2.3 Procedure

Following the classification of the participants as ASD or NT, subjects were randomly assigned to begin the experiment with either the Discrete or Reciprocal protocol. In all cases, a lab member would allow the child time to adjust to the apparatus as well as provide a general example of the expectations for the task.

In the Reciprocal condition, participants were seated with their feet firmly on the ground (or box), their right arm comfortably resting on the table with their right hand over the mouse. Participants were instructed to move their hand (mouse) to a position that aligns with the visual cursor (the mouse image) at the start position. This starting position was highlighted by a gray box labeled START HERE. Once prompted to go (e.g. the targets appeared), the participants were instructed to move the mouse in and out of two defined target areas (Blue rectangle) on the screen in front of them. These targets varied in size and distance (amplitude) between center for a total of 3 ID values of 3, 4 & 5. Table 1 provides a breakdown of all of the possible ID combinations as well as the total random presentations of each event. The children were given specific instructions to complete this task as fast and accurate as they could. In total for the Reciprocal, the participants completed 27 trials with 9 unique combinations per ID. A single trial lasted a total of 15 seconds with the middle 8 seconds of each trial subjected to analysis.

Everything remained the same for the Discrete task except once the participant positioned themselves in the START HERE box, a single target would appear. The participants were instructed to move as to that target and come to a stop as fast and accurate as possible. Similar to the Reciprocal design, a number of ID combinations were tested (see Table 1). A single trial lasted 12 seconds and the participants were asked to complete a total of 36 trials. The procedure took an

average of one hour; however, a 2-hour window was set aside to ensure the participants remained as comfortable as possible during the session.

Table 1: Break down of Index of difficulty

Reciprocal				Discrete			
ID	D (cm)	W (cm)	Trials	ID	D (cm)	W (cm)	Trials
3	16	4	4	3	16	4	4
	12	3	4		12	3	4
	8	2	4		8	2	4
4	16	2	4	4	16	2	4
	12	1.5	4		12	1.5	4
	8	1	4		8	1	4
5	16	1	4	5	16	1	4
	12	0.75	4		12	0.75	4
	8	0.5	4		8	0.5	4

Variables of interest were Duration (Total Time-TT), Peak Velocity (PVEL), Percent time to Peak Velocity (%TPV), Normalized Jerk (NJRK) and total number of Movements (MVMNT). A Pearson correlation was first run to examine the linear relationship between movement time and ID. Second, the dependent variables of TT, PKVEL, %TPV, NJRK and MVMNT were analyzed in separate Task (Reciprocal, Discrete) x Group (ASD, NT) X ID (3, 4, 5) linear mixed model analyses of variance (ANOVAs) with repeated measures on ID. Given the un-equal sample size, a Welch's T test was utilized to compare unique ID scenarios by Group (ASD vs NT). An alpha = .05 was used for all tests.

CHAPTER 5 Results

5.1 Pearson's Correlation

A Pearson correlation coefficient was computed to assess the relationship between Index of Difficulty and Movement Time. There was a significant correlation for the reciprocal tasks for both groups, $r=.250$, $n=90$, $p=.017$ and $r=.494$, $n=54$, $p=.000$. A scatterplot summarized in the results (Figure 1, Figure 2). Overall, within the reciprocal task, there was weak, positive correlation between the difficulty of task and participant movement. The increase in task difficulty correlated with the increase in movement time. Unexpectedly however, the correlation results for the discrete tasks at either group (ASD, NT) failed to reach significance, $r=.057$, $n=180$, $p=.448$ and $r=.111$, $n=126$, $p=.216$.

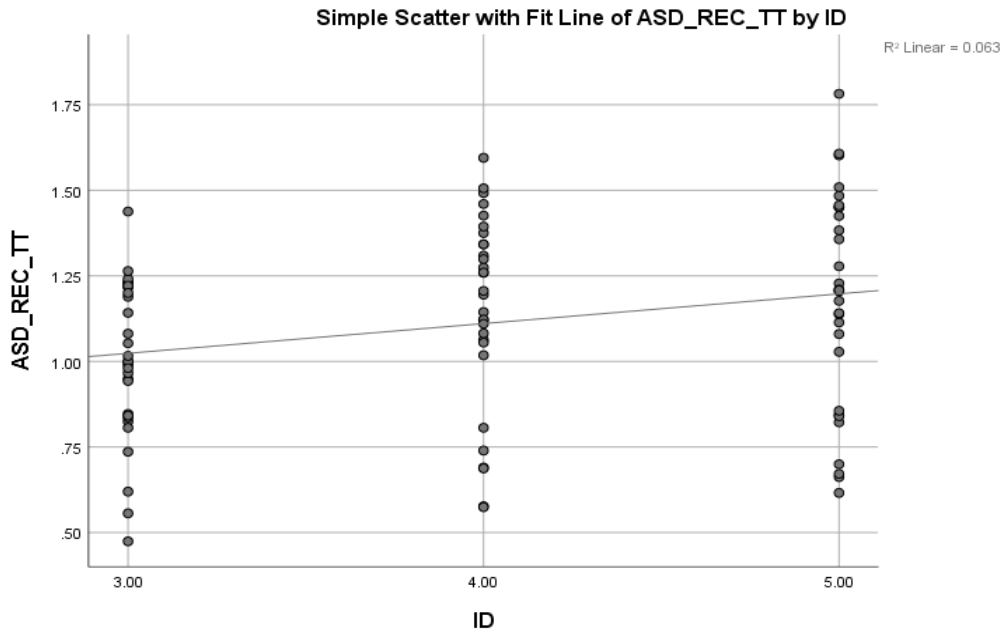


Figure 2. Scatterplot of Index of Difficulty with Movement time of all ASD participants (Reciprocal). ($R^2 = 0.063$)

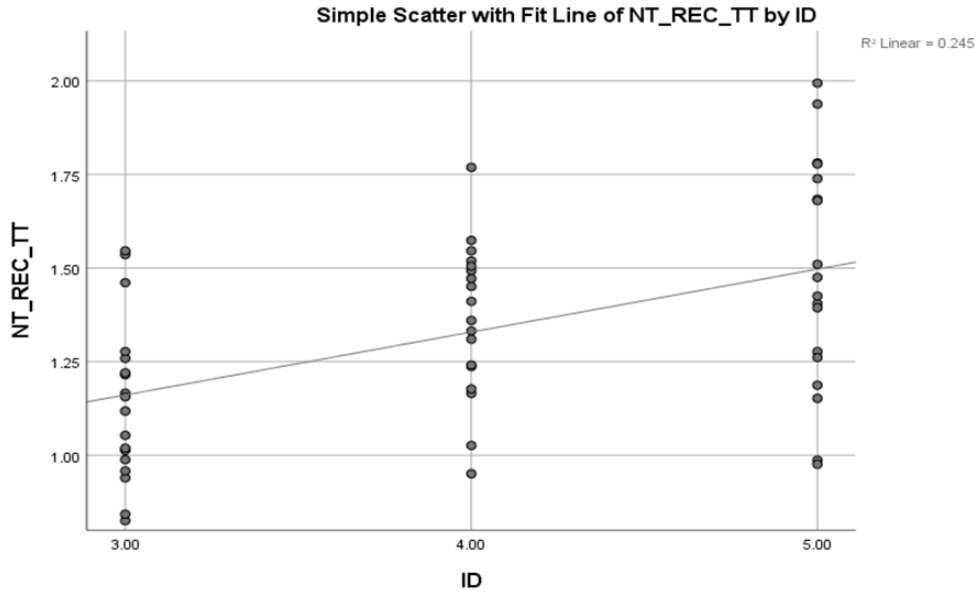


Figure 3. Scatterplot of Index of Difficulty with Movement time of all NT participants (Reciprocal). ($R^2 = 0.245$)

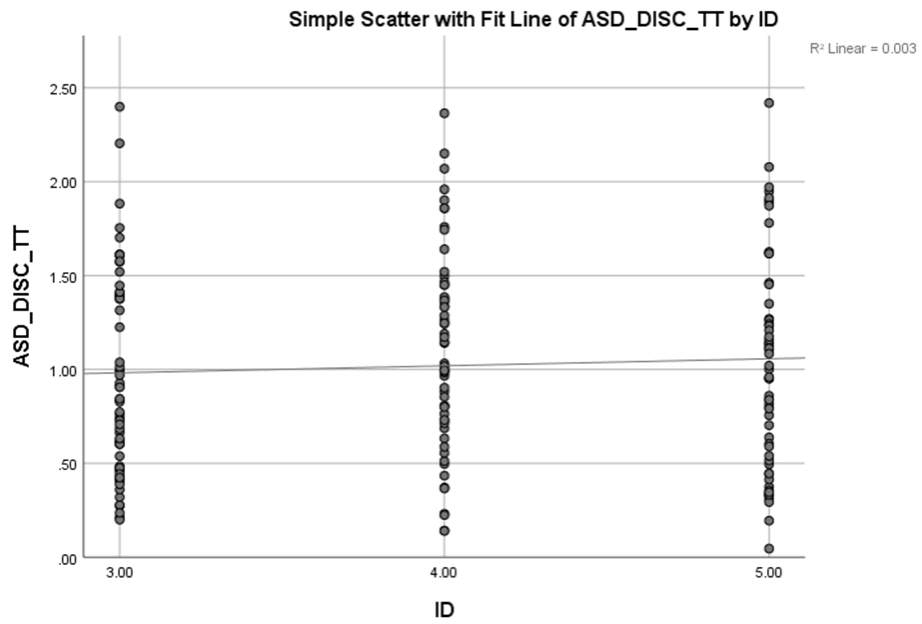


Figure 4. Scatterplot of Index of Difficulty with Movement time of all ASD participants (Discrete). ($R^2 = 0.003$)

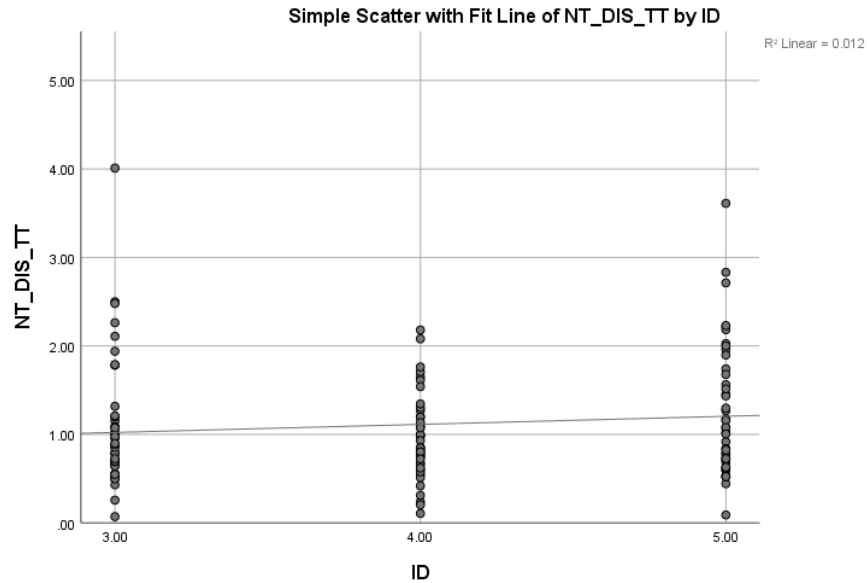


Figure 5. Scatterplot of Index of Difficulty with Movement time of all NT participants (Discrete). ($R^2 = 0.012$)

5.2 Repeated Mixed Model

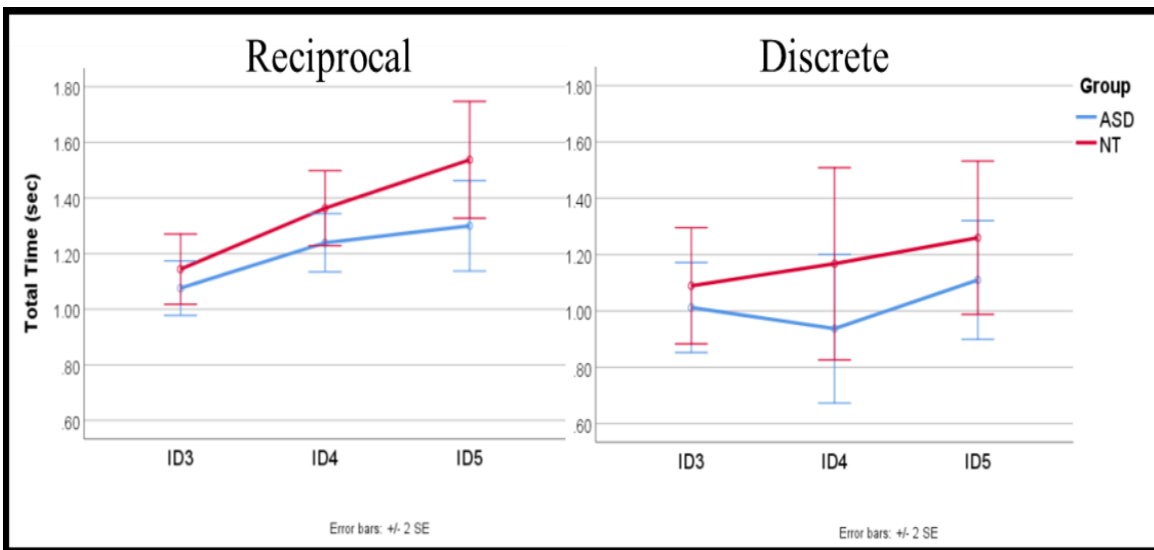


Figure 6. Mean Movement time of All Participants (ASD group and NT group)

Total Time (TT)

The analysis indicated a trend towards significance within subject effect for task, $F(2, 14) = 3.579$, $p=0.79$, with the slower TTs on the reciprocal task (mean \pm sd= $1.277 \pm .045$) compared to

the discrete task(mean \pm sd= 1.096 \pm .071). The analysis also indicated a within subject effect for ID, F (2, 28) =11.877, p=0.00, faster TTs between to ID3(mean \pm sd= 1.080 \pm .033)compared to ID4 (mean \pm sd= 1.177 \pm .058) and to ID4 compared to ID5(mean \pm sd= 1.302 \pm .038). The analysis indicated a between subject effects for Group, F (1, 14) =1119.249, p=0.00, with faster TTs for the ASD group (mean \pm sd= 1.112 \pm .043) compared to the NT group (mean \pm sd= 1.260 \pm .056).

The total time was affected by the distance manipulation and width manipulation of the two tasks, discrete and reciprocal. The analysis of the task based of distance (D) indicated within subject effect for task, reciprocal (mean \pm sd= 1.438 \pm .042) and discrete (mean \pm sd= 1.102 \pm .086); F (1,14), p=.005. Also demonstrated for the index of difficulty, ID3 (mean \pm sd= 1.143 \pm .097), ID4 (mean \pm sd= 1.232 \pm .050), ID5(mean \pm sd= 1.435 \pm .077); F(2, 5.28)=7.299, p=.003. The analysis of the task based off width (width) indicated within subject effect for index of difficulty, ID3 (mean \pm sd= .949 \pm .058), ID4 (mean \pm sd= 1.106 \pm .046), ID5(mean \pm sd= 1.239 \pm .099); F(2, 28)=5.803, p=.008

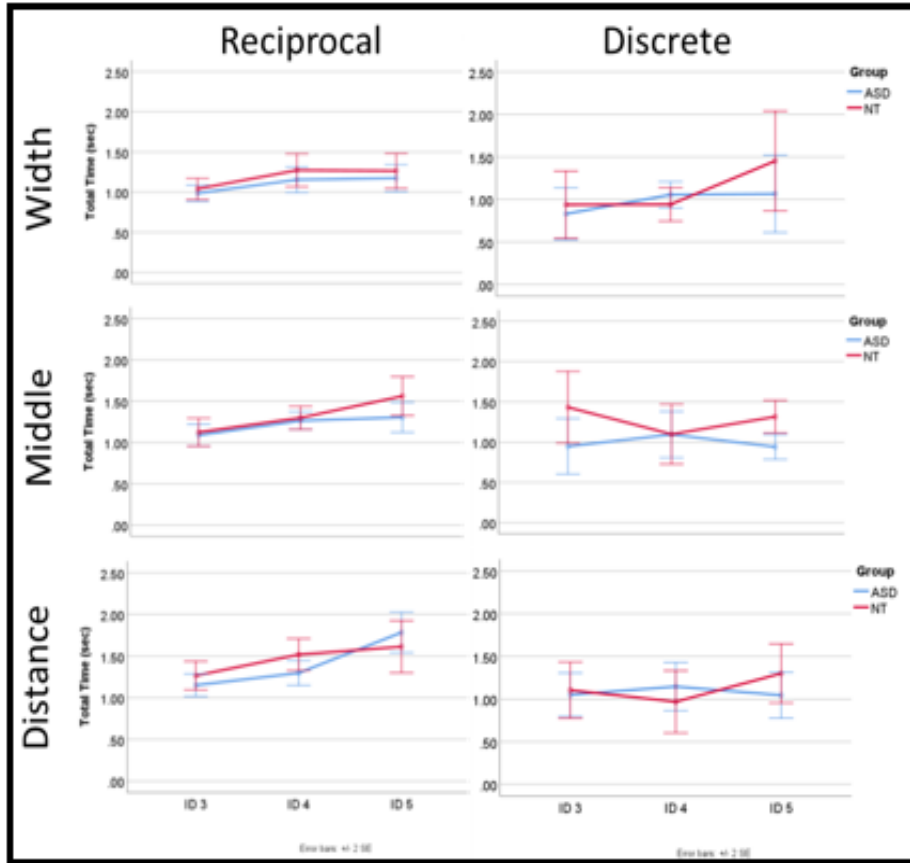


Figure 7 Mean Total Time of width, middle of discrete and reciprocal for All Participants (ASD group and NT group)

Peak velocity (PVEL)

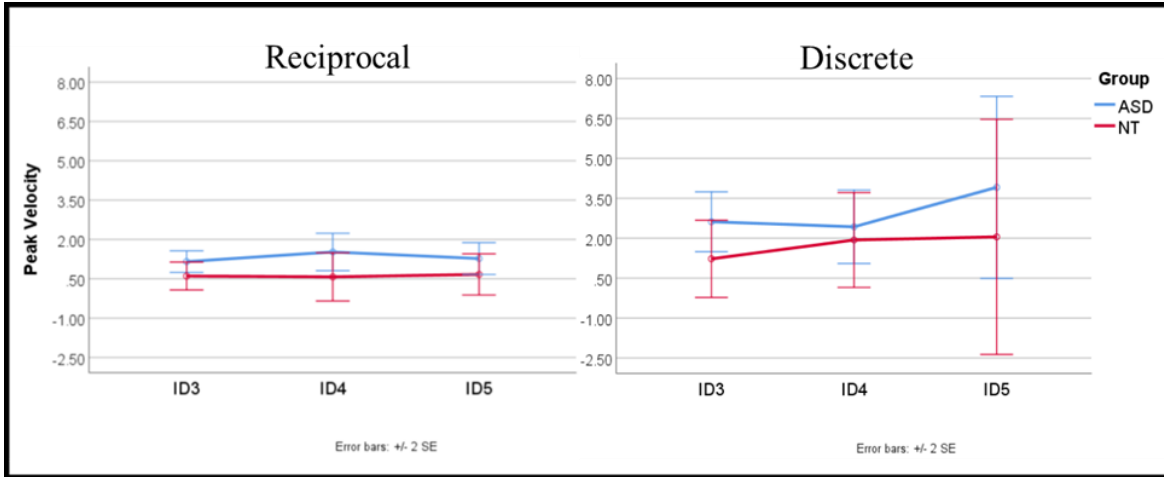


Figure 8. Mean Peak Velocity of All Participants (ASD group and NT group)

The analysis indicated within subject effects for Task, $F(1,14) = 6.384$, $p = 0.024$, with higher PVEL for the discrete task (mean \pm sd = $2.362 \pm .504$) compared to the reciprocal task (mean \pm sd = $.967 \pm .181$). The analysis also indicated trend towards significance between subject effects for Group, $F(1,14)$, $p = 0.08$, with faster PVEL for the ASD group (mean \pm sd = $1.176 \pm .409$) compared to the NT group (mean \pm sd = $2.122 \pm .317$). The analysis failed to detect any interaction for Index of Difficulty $p = .789$.

The peak velocity was affected by the distance manipulation and width manipulation of the two tasks, discrete and reciprocal. The analysis based of distance (Distance) and middle (middle) failed to indicate within subject effect. However, the analysis based of width indicated a trend towards significance for within subject effect for index of difficulty ID3 (mean \pm sd = $.396 \pm .547$), ID4 (mean \pm sd = $-.503 \pm .314$), ID5 (mean \pm sd = $1.340 \pm .718$); $F(2, .28) = 2.889$, $p = .072$. As well as, between subject effect of group with ASD (mean \pm sd = $1.210 \pm .403$) compared to NT (mean \pm sd = $-.388 \pm .043$); $F(2,14) p = .024$. The analysis of the task based off width (width) indicated within subject effect for index of difficulty, ID3 (mean \pm sd = $.949 \pm .058$), ID4 (mean \pm sd = $1.106 \pm .046$), ID5 (mean \pm sd = $1.239 \pm .099$); $F(2,28) = 5.803$, $p = .008$

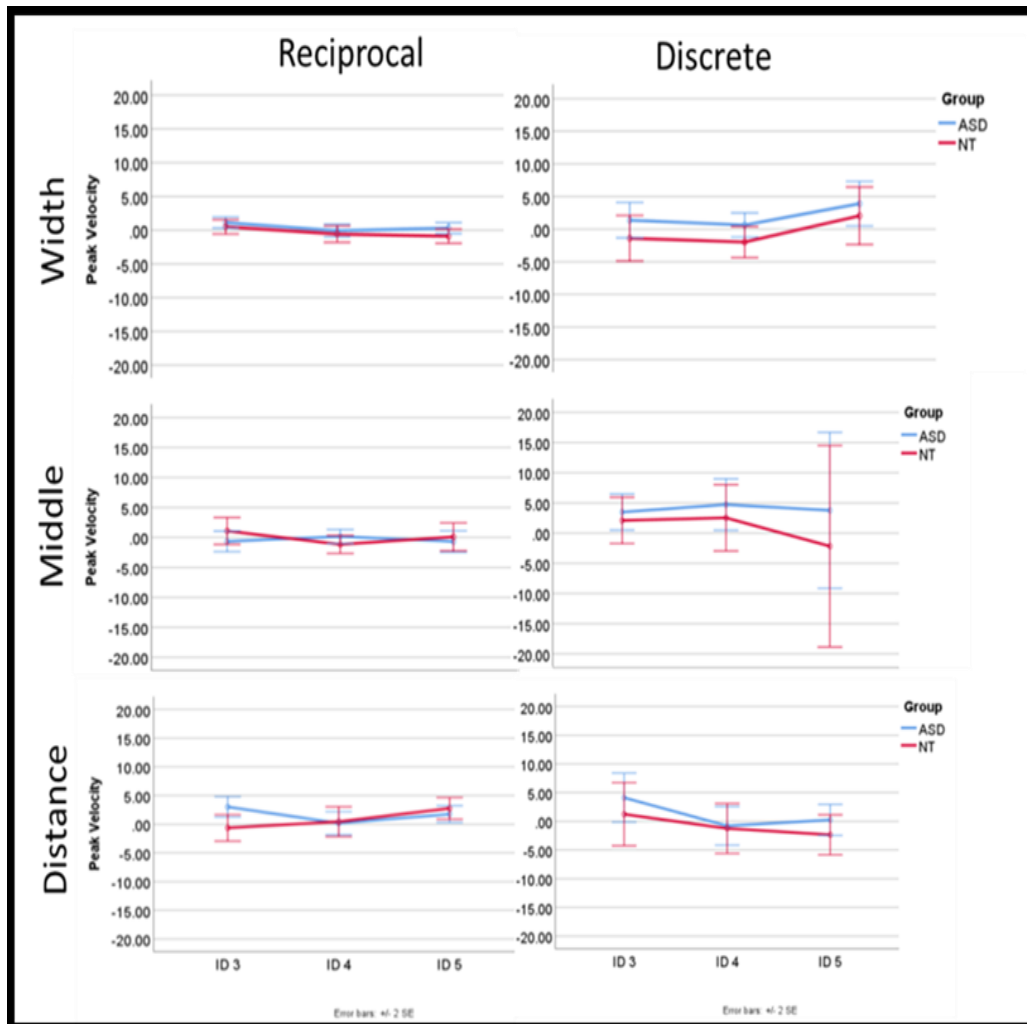


Figure 9. Mean Peak Velocity of width, middle of discrete and reciprocal for All Participants (ASD group and NT group)

Number of Movements (MVMTS)

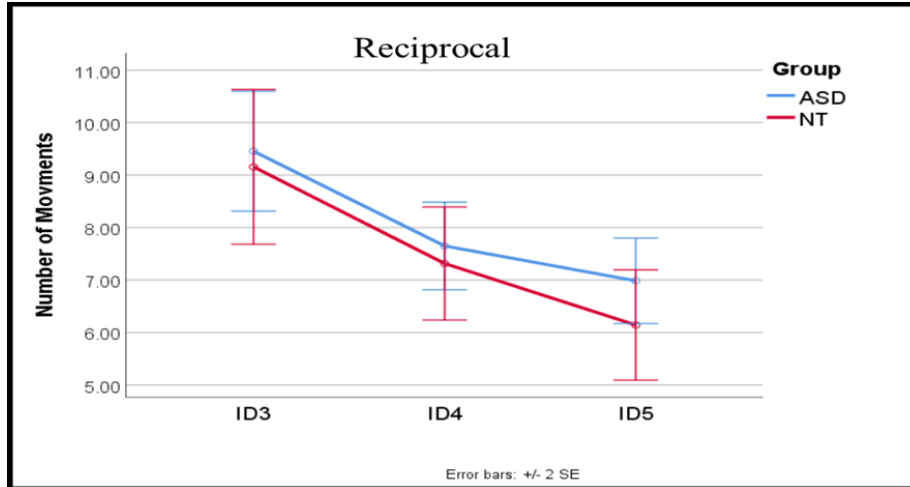


Figure 10 Mean Number of Movements of All Participants (ASD group and NT group)

The analysis indicated within subject effects for Index of Difficulty, $F(2, 28) = 48.536$ $p = 0.00$, fewer movements as the difficulty increased with ID3 (mean \pm sd= 9.305 ± 0.466), ID4 (mean \pm sd= 7.482 ± 0.341), ID5 (mean \pm sd= 6.564 ± 0.332); The analysis failed to detect between subject effects of group, $p = .491$.

The total number of movements was affected by the distance manipulation and width manipulation of the two tasks, discrete and reciprocal. The analysis of based of distance (Distance) indicated within subject effect with ID3 (mean \pm sd= 9.134 ± 0.608), ID4 (mean \pm sd= 7.366 ± 0.501), ID5 (mean \pm sd= 6.068 ± 0.372); $F(2, 28) = 16.846$, $p = .000$. The analysis of the based off middle (middle) indicated within subject effect for index of difficulty ID3 (mean \pm sd= 9.234 ± 0.549), ID4 (mean \pm sd= 7.001 ± 0.290), ID5 (mean \pm sd= 6.646 ± 0.355); $F(2, 28) = 21.344$, $p = .000$. The analysis of the based off width (width) indicated within subject effect for index of difficulty, ID3 (mean \pm sd= 9.540 ± 0.540), ID4 (mean \pm sd= 8.077 ± 0.674), ID5 (mean \pm sd= 4.977 ± 0.443); $F(2, 28) = 11.493$, $p = .000$.

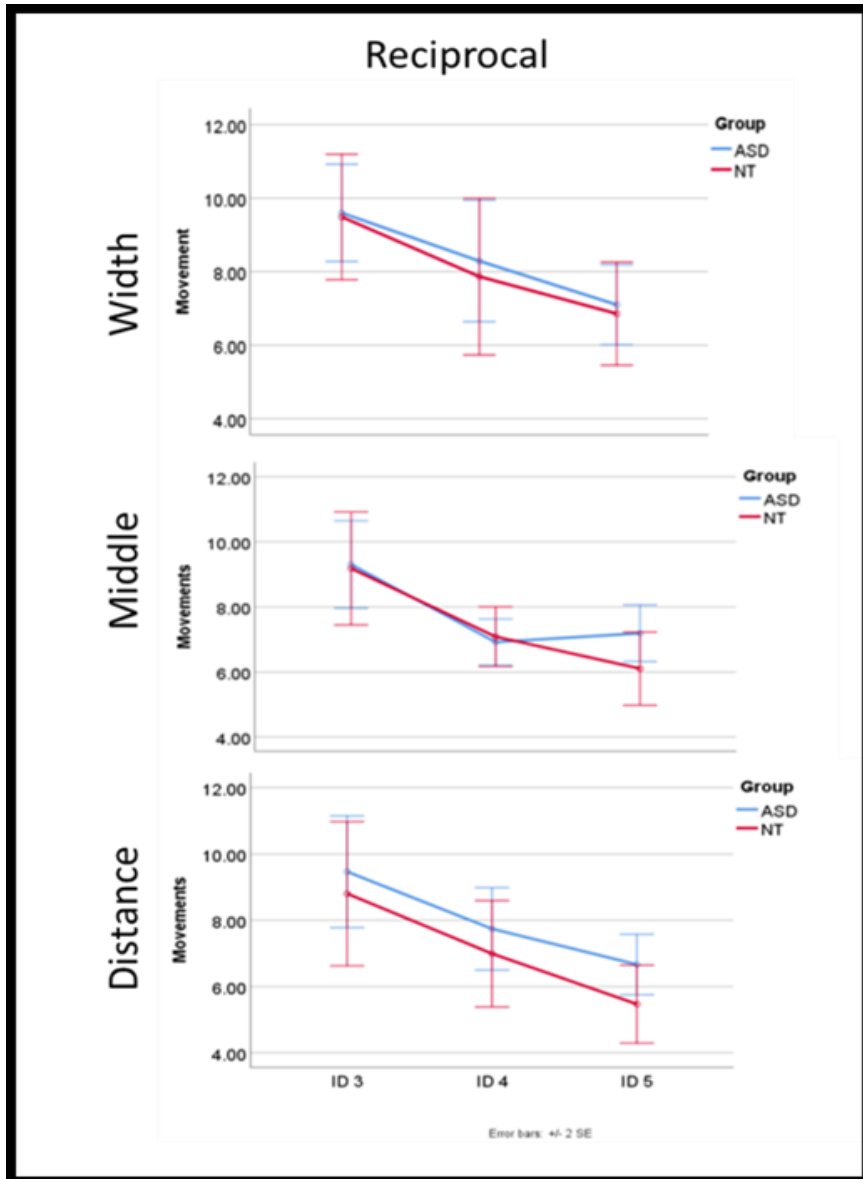


Figure 11. Mean number of Movements of width, middle of discrete and reciprocal for All Participants (ASD group and NT group)

Normalized Jerk

The analysis indicated no significant with in subject effects on Task ($p=.562$) and ID ($p=.797$). As well as no significant between subject effect on Group ($p=.764$)

Time to Total Peak Velocity

The analysis indicated no significant with in subject effects on Task ($p=.562$) and ID ($p=.797$). As well as no significant between subject effect on Group ($p=.764$)

5.3 One Way ANOVA (Welch T-Test)

Peak Velocity

Table 2. Reciprocal Task. Mean Average of Peak Velocity

Robust Tests of Equality of Means					
		Statistic ^a	df1	df2	Sig.
ID3	Welch	3.789	1	13.533	.073
ID4	Welch	4.359	1	10.285	.063
ID5	Welch	2.116	1	13.345	.169

a. Asymptotically F distributed.

Table 3. Discrete Task. Mean Average Peak Velocity

Robust Tests of Equality of Means					
		Statistic ^a	df1	df2	Sig.
ID3	Welch	3.768	1	13.070	.074
ID4	Welch	.116	1	14.710	.738
ID5	Welch	.961	1	9.886	.350

a. Asymptotically F distributed.

The analysis indicated a trend towards significance for both Fitts Tasks. The Reciprocal task displayed a trend at two Index of Difficulty at ID3 ($p=.073$) and ID4 ($p=.063$). While the Discrete task displayed a trend at ID3 ($p=.074$),

Chapter 6 Discussion & Conclusion

Discussion

The following thesis aimed to further the understanding of upper extremity coordination issues previously seen comparing children with ASD and NT. More specifically, this thesis was aimed at further investigating literature differences potentially being a result from methodology, for example, data extracted from a Reciprocal or Discrete task. Kinematic points of interest in this study were Total Time (TT), Peak Velocity (PV), Percent Time to Peak Velocity (%TPV), Normalized Jerk (NJRK), and Number of Movements (MVMT). By highlighting these values in a between and within subjects repeated design, it was the goal of this study to conclude an explanation as to why conflicting results appear in this small pool of studies. Based on the findings of this current data set, it would appear that this study has potentially made the discussion more puzzling than closer to solved.

To start, the results of the Pearson's correlation analysis revealed there was a significant positive correlation between index of difficulty of the task and the participants' movement time within the reciprocal task for both groups (Figure 2, Figure 3). These results indicated that the neurotypical group had a strong relationship between the two variables, even though it is a weak correlation ($r=.494$, $p=.000$). These two correlations support mathematical MT/ID slope relationship (Fitts, 1954). Surprisingly, the results for the discrete task for each group violates Fitts' Law with no correlation between TT and ID with ASD ($r=.057$, $p=.484$) and NT ($r=.111$, $p=.216$). These results were surprising and mark a major limitation to results discussed moving forward.

One potential explanation for this result is the age-range of the participants 6-12 years old support by Caeyenberghs et al (2009) study concluding age-related changes in the slope of the function that describes the relationship between task difficulty (ID) and MT. Another potential reason for this result is the constraint of accuracy was not upheld to the highest possible standard.

Further analysis of raw data points in the target zone are currently ongoing and will be completed by manuscript submission. Preliminary results appear to show a high amount of variability in select individuals and target accuracy. If the result appears to be high in error, a functional target width will be calculated for both Groups and re-assessed based upon the new lower ID values. Additionally, end point variability, even if too high to uphold Fitts Law, still provides a valuable descriptor of the motor behavior.

Smits-Engelsman et al (2006), reported that children's performance for both Fitts Tasks are similar to that of adults just slower speed. They examined the developmental effects on speed/accuracy trade-off of a group of children 6-10 years old performing both tasks. The slope of the speed accuracy trade-off was similar in the three age groups in the cyclic as compared to the discrete task, suggesting that children learn both tasks equally well in this age range. They also note a clear difference between the kinematics of discrete and cyclic movements with the cyclic movements were faster, higher index performance, fewer changes in the velocity and more ballistic. They found that children were about 40% faster in cyclic movements compared to discrete movements, even as young as 6 years of age showed relative superiority in performance. Therefore, the movement execution are different between the two tasks. The results from total time (Figure 8) and peak velocity (Figure 8), lean towards a possible difference of execution between the two tasks.

Lambert and Bard (2005) conducted a study on 6-10-year-old children, with two-dimensional discrete pointing task using a computer mouse. They investigated the motor performance and visuomanual control through Fitts law and showed younger children (6-8) had a stronger ID effect than the older children. This relates to the level of task and age which could also explained the results displayed within this study.

The group of children with ASD showed difference in their execution of the motor sequences. The duration (TT) of the motor sequence averaged across different levels of index of difficulty, were faster in the ASD group than in the NT group. The results of this particular study contradict the findings of the three other studies that used Fitts law speed-accuracy trade-off protocol to analyzed the kinematics of movement structure of individuals with ASD (Glazebrook et al, 2006; Glazebrook et al. 2008; Papadopolous et al., 2012). There was a slower movement time for the neurotypical children than for children with ASD with both the discrete task and reciprocal task, that contradicts the finds form Glazebrook et al (2006, 2008) and Papadopoulos et al (2012). Papadopoulos et al (2012), found no difference in movement time between the children with ASD to the NT group after performing a Reciprocal aiming task with a greater end-point variability in their movements around the target. In contrast, Glazebrook et al (2006, 2008) found that individuals with ASD had a longer movement time than that of NT when completing the Discrete aiming task with ASD maintaining accuracy at the end-point- of their movements. From those studies, it implies that individuals with ASD face impairments in their cognitive process regardless of the nature of the task (Discrete or Reciprocal). Even though the children with ASD within this study moved faster does not mean they were accurately hitting the target. For this study analyzing the end-point-variability of the two groups will give more insight of the movement performance deficits noted by previous studies.

It must be stressed that these preliminary results are tentative and warrant further investigation. This conclusion is not simply made on the fact that the results are contradictory to the reviewed literature, but also due to a number of outside factors and design limitations. As this manuscript is being written, our research lab is currently shut down for the foreseeable future do to the COVID-19 pandemic. Limitations to this study include: A wide age range of the participants

(6-12years), unbalanced populations, and a small sample size (n=16); factors that negatively affected statistical power due to the variability in performance that exists within the ASD population.

Another limitation to this study was the use of an adult size mouse. The original experimental proposal included the use of a large Wacom digital touch tablet for data collection. Unfortunately, this protocol was changed to the mouse use when the tablet received water damage from a roof leak. Use of the adult size mouse has the potential to cause a problem in the approach given the task requires movement from a stylus palmer/tripod grasp to a hand over mouse. In many of the cases, the children's small hands looked awkward given they also needed to push down the button as they moved. Furthermore, the click design in Movalyzer could have also created a constraint on the findings given holding down the left click and dragging the mouse is more complex than simply moving the mouse.

Also, the children showed little interest with the visual set up. This led to a Bunny to Carrot visual set up that followed the same procedure as the previous experiment however now the child was instructed on moving the cursor from the bunny to the carrot. At this point it is too early to tell if the switch has made an impact statistically, but from the perspective of the data collector it appeared to provide much more of an engaging task for the children compared to the previous set up.

List of References

- Acharya, S. S., Patnaik, S., & Nanda, S. B. (2018). Patients with Autism Spectrum Disorders: Strategy for Orthodontic Care. *Journal of Clinical & Diagnostic Research*, 12(7).
- Adak, B., & Halder, S. (2017). A review-based prevalence of autism spectrum disorder. *Indian Journal of Health & Wellbeing*, 8(8)
- Bailey, A., Le Couteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., & Rutter, M. (1995). Autism as a strongly genetic disorder: evidence from a British twin study. *Psychological medicine*, 25(1), 63-77.
- Baio, J. (2014). Prevalence of autism spectrum disorder among children aged 8 years—autism and developmental disabilities monitoring network, 11 sites, United States, 2010.
- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., Kurzius-Spencer, M., Zahorodny, W., Rosenberg, C.R., White, T. and Durkin, M.S & Durkin, M. S. (2018). Prevalence of autism spectrum disorder among children aged 8 years—autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(6), 1.
- Bauman, M., & Kemper, T. L. (1985). Histoanatomic observations of the brain in early infantile autism. *Neurology*, 35(6), 866-866
- Bauman, M. L., & Kemper, T. L. (2005). Neuroanatomic observations of the brain in autism: a review and future directions. *International journal of developmental neuroscience*, 23(2-3), 183-187.
- Baxter, A. J., Brugha, T. S., Erskine, H. E., Scheurer, R. W., Vos, T., & Scott, J. G. (2015). The epidemiology and global burden of autism spectrum disorders. *Psychological medicine*, 45(3), 601-613.
- Bhat, A. N., Galloway, J. C., & Landa, R. J. (2012). Relation between early motor delay and later communication delay in infants at risk for autism. *Infant Behavior and Development*, 35(4), 838-846.
- Beamish, D., Bhatti, S. A., MacKenzie, I. S., & Wu, J. (2006). Fifty years later: a neurodynamic explanation of Fitts' law. *Journal of the Royal Society Interface*, 3(10), 649-654.
- Bolton, P., Macdonald, H., Pickles, A., Rios, P., Goode, S., Crowson, M. Bailey, A., & Rutter, M. (1994). A case-control family history study of autism. *Journal of child Psychology and Psychiatry*, 35(5), 877-900.
- Bootsma, R. J., Fernandez, L., & Mottet, D. (2004). Behind Fitts' law: kinematic patterns in goal-directed movements. *International Journal of Human-Computer Studies*, 61(6), 811-821.

Broder-Fingert, S., Sheldrick, C. R., & Silverstein, M. (2018). The Value of State Differences in Autism When Compared to a National Prevalence Estimate. *Pediatrics*, *142*(6), e20182950.

Buchanan, J. J., Park, J. H., & Shea, C. H. (2006). Target width scaling in a repetitive aiming task: switching between cyclical and discrete units of action. *Experimental Brain Research*, *175*(4), 710-725.

Buescher, A. V., Cidav, Z., Knapp, M., & Mandell, D. S. (2014). Costs of autism spectrum disorders in the United Kingdom and the United States. *JAMA pediatrics*, *168*(8), 721-728.

Caeyenberghs, K., Wilson, P. H., Van Roon, D., Swinnen, S. P., & Smits-Engelsman, B. C. (2009). Increasing convergence between imagined and executed movement across development: evidence for the emergence of movement representations. *Developmental science*, *12*(3), 474-483.

Cerminara, N. L., Apps, R., & Marple-Horvat, D. E. (2009). An internal model of a moving visual target in the lateral cerebellum. *The Journal of physiology*, *587*(2), 429-442

Chawarska, K., Paul, R., Klin, A., Hannigen, S., Dichtel, L. E., & Volkmar, F. (2007). Parental recognition of developmental problems in toddlers with autism spectrum disorders. *Journal of autism and developmental disorders*, *37*(1), 62-72.

Chua, R., & Elliott, D. (1993). Visual regulation of manual aiming. *Human Movement Science*, *12*(4), 365-40

Courchesne, E., Saitoh, O., Yeung-Courchesne, R., Press, G. A., Lincoln, A. J., Haas, R. H., & Schreibman, L. (1994). Abnormality of cerebellar vermal lobules VI and VII in patients with infantile autism: identification of hypoplastic and hyperplastic subgroups with MR imaging. *American journal of roentgenology*, *162*(1), 123-130. N

Corben, L. A., Georgiou-Karistianis, N., Bradshaw, J. L., Hocking, D. R., Churchyard, A. J., & Delatycki, M. B. (2011). The Fitts task reveals impairments in planning and online control of movement in Friedreich ataxia: reduced cerebellar-cortico connectivity?. *Neuroscience*, *192*, 382-390.

Cullen, K. E., Brooks, J. X., Jamali, M., Carriot, J., & Massot, C. (2011). Internal models of self-motion: computations that suppress vestibular reafference in early vestibular processing. *Experimental brain research*, *210*(3-4), 377-388.

Daniels, J. L. (2006). Guest editorial: autism and the environment.

Davis, N. J., Cui, S., & Spence, C. (2008). The dynamics of reciprocal aiming with a steering wheel. *Experimental brain research*, *188*(1), 141-146.

- Dean, M., Wu, S. W., & Maloney, L. T. (2007). Trading off speed and accuracy in rapid, goal-directed movements. *Journal of Vision*, 7(5), 10-10.
- DeMyer, M. K., Hingtgen, J. N., & Jackson, R. K. (1981). Infantile autism reviewed: A decade of research. *Schizophrenia bulletin*, 7(3), 388-451
- Donnellan, A. M., Hill, D. A., & Leary, M. R. (2013). Rethinking autism: implications of sensory and movement differences for understanding and support. *Frontiers in integrative neuroscience*, 6, 124.
- Dowell, L. R., Mahone, E. M., & Mostofsky, S. H. (2009). Associations of postural knowledge and basic motor skill with dyspraxia in autism: implication for abnormalities in distributed connectivity and motor learning. *Neuropsychology*, 23(5), 563.
- Dowd, A. M., McGinley, J. L., Taffe, J. R., & Rinehart, N. J. (2012). Do planning and visual integration difficulties underpin motor dysfunction in autism? A kinematic study of young children with autism. *Journal of autism and developmental disorders*, 42(8), 1539-1548
- Dziuk, M. A., Larson, J. G., Apostu, A., Mahone, E. M., Denckla, M. B., & Mostofsky, S. H. (2007). Dyspraxia in autism: association with motor, social, and communicative deficits. *Developmental Medicine & Child Neurology*, 49(10), 734-739.
- Elliot, D., Helsen, W. F., & Chua, R. (2001). A century later: Woodworth's (1899) two component model of goal-directed aiming. *Psychological bulletin*, 127(3), 342.
- Estes, A., Munson, J., Dawson, G., Koehler, E., Zhou, X. H., & Abbott, R. (2009). Parenting stress and psychological functioning among mothers of preschool children with autism and developmental delay. *Autism*, 13(4), 375-387.
- Fabbri-Destro, M., Cattaneo, L., Boria, S., & Rizzolatti, G. (2009). Planning actions in autism. *Experimental brain research*, 192(3), 521-525.
- Fitts, P. M. (1954). The information capacity of the human motor system in controlling the amplitude of movement. *Journal of experimental psychology*, 47(6), 381.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric research*, 65(6), 591.
- Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: a synthesis and meta-analysis. *Journal of autism and developmental disorders*, 40(10), 1227-1240.
- Forti, S., Valli, A., Perego, P., Nobile, M., Crippa, A., & Molteni, M. (2011). Motor planning and control in autism. A kinematic analysis of preschool children. *Research in Autism Spectrum Disorders*, 5(2), 834-842

- Glazebrook, C. M., Elliott, D., & Lyons, J. (2006). A kinematic analysis of how young adults with and without autism plan and control goal-directed movements. *Motor control*, *10*(3), 244-264.
- Glazebrook, C. M., Elliott, D., & Szatmari, P. (2008). How do individuals with autism plan their movements? *Journal of Autism and Developmental Disorders*, *38*(1), 114-126.
- Glover, S. (2004). Separate visual representations in the planning and control of action. *Behavioral and brain sciences*, *27*(1), 3-24.
- Green, D., Baird, G., Barnett, A. L., Henderson, L., Huber, J., & Henderson, S. E. (2002). The severity and nature of motor impairment in Asperger's syndrome: a comparison with specific developmental disorder of motor function. *Journal of child psychology and psychiatry*, *43*(5), 655-668.
- Hassall, R. (2016). "Autism" or "Autism Spectrum Disorder": Does either represent a natural kind of psychological disorder? *History & Philosophy of Psychology*, *17*(1), 17–23. Retrieved from <http://0search.ebscohost.com.lib.utep.edu/login.aspx?direct=true&db=a9h&AN=119119081&site=ehost-live&scope=site>
- Herbert, M. R. (2010). Contributions of the environment and environmentally vulnerable physiology to autism spectrum disorders. *Current opinion in neurology*, *23*(2), 103-110.
- Hilton, C. L., Zhang, Y., Whilte, M. R., Klohr, C. L., & Constantino, J. (2012). Motor impairment in sibling pairs concordant and discordant for autism spectrum disorders. *Autism*, *16*(4), 430-441
- Hughes, C. (1996). Brief report: Planning problems in autism at the level of motor control. *Journal of autism and developmental disorders*, *26*(1), 99-107.
- Huys, R., Fernandez, L., Bootsma, R. J., & Jirsa, V. K. (2010). Fitts' law is not continuous in reciprocal aiming. *Proceedings of the Royal Society of London B: Biological Sciences*, *277*(1685), 1179-1184.
- Ito, M. (2008). Control of mental activities by internal models in the cerebellum. *Nature Reviews Neuroscience*, *9*(4), 304.
- Jansiewicz, E. M., Goldberg, M. C., Newschaffer, C. J., Denckla, M. B., Landa, R., & Mostofsky, S. H. (2006). Motor signs distinguish children with high functioning autism and Asperger's syndrome from controls. *Journal of autism and developmental disorders*, *36*(5), 613-621.
- Jones, V., & Prior, M. (1985). Motor imitation abilities and neurological signs in autistic children. *Journal of autism and developmental disorders*, *15*(1), 37-46.

Kogan, M. D., Vladutiu, C. J., Schieve, L. A., Ghandour, R. M., Blumberg, S. J., Zablotsky, B., Perrin, J.M, Shattuck, P, Kuhlthau, K. A, Harwood, R.B & Lu, M. C. The prevalence of parent-reported autism spectrum disorder among US children. *Pediatrics*, 142(6), e20174161.

Kovacs, A. J., Buchanan, J. J., & Shea, C. H. (2008). Perceptual influences on Fitts' law. *Experimental Brain Research*, 190(1), 99-103.

Kreiser, N. L., & White, S. W. (2014). ASD in females: are we overstating the gender difference in diagnosis?. *Clinical child and family psychology review*, 17(1), 67-84.

Lambert, J., & Bard, C. (2005). Acquisition of visuomanual skills and improvement of information processing capacities in 6-to 10-year-old children performing a 2D pointing task. *Neuroscience letters*, 377(1), 1-6.

Landrigan, P. J. (2010). What causes autism? Exploring the environmental contribution. *Current opinion in pediatrics*, 22(2), 219-225.

Latash, M. L., Levin, M. F., Scholz, J. P., & Schöner, G. (2010). Motor control theories and their applications. *Medicina*, 46(6), 382.

Lawson, R. P., Rees, G., & Friston, K. J. (2014). An aberrant precision account of autism. *Frontiers in human neuroscience*, 8, 302.

Leary, M. R., & Hill, D. A. (1996). Moving on: autism and movement disturbance. *Mental Retardation-Washington*, 34(1), 39-53.

Lee, J. M., Kyeong, S., Kim, E., & Cheon, K. A. (2016). Abnormalities of inter-and intra-hemispheric functional connectivity in autism spectrum disorders: a study using the autism brain imaging data exchange database. *Frontiers in neuroscience*, 10, 191.

Leggio, M. G., Tedesco, A. M., Chiricozzi, F. R., Clausi, S., Orsini, A., & Molinari, M. (2008). Cognitive sequencing impairment in patients with focal or atrophic cerebellar damage. *Brain*, 131(5), 1332-1343.

Leigh, J. P., & Du, J. (2015). Brief report: Forecasting the economic burden of autism in 2015 and 2025 in the United States. *Journal of autism and developmental disorders*, 45(12), 4135-4139.

Lloyd, M., MacDonald, M., & Lord, C. (2013). Motor skills of toddlers with autism spectrum disorders. *Autism*, 17(2), 133-146.

Lyall, K., Croen, L., Daniels, J., Fallin, M. D., Ladd-Acosta, C., Lee, B. K., Park, B.Y., Snyder, N.W., Schendel, D., Volk, H. & Windham, G.C. (2017). The changing epidemiology of autism spectrum disorders. *Annual review of public health*, 38, 81-102.

MacKenzie, I. Scott. "Fitts' law as a research and design tool in human-computer interaction." *Human-computer interaction*, 7(10), 91-139.

Marko, M. K., Crocetti, D., Hulst, T., Donchin, O., Shadmehr, R., & Mostofsky, S. H. (2015). Behavioural and neural basis of anomalous motor learning in children with autism. *Brain*, 138(3), 784-797.

Mari, M., Castiello, U., Marks, D., Marraffa, C., & Prior, M. (2003). The reach-to-grasp movement in children with autism spectrum disorder. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 358(1430), 393-403.

Matson, J. L., & Kozlowski, A. M. (2011). The increasing prevalence of autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(1), 418-425.

Mayes, S. D., & Calhoun, S. L. (2003). Ability profiles in children with autism: Influence of age and IQ. *Autism*, 7(1), 65-80.

Ming, X., Brimacombe, M., & Wagner, G. C. (2007). Prevalence of motor impairment in autism spectrum disorders. *Brain and Development*, 29(9), 565-570.

Minschew, N. J., & Keller, T. A. (2010). The nature of brain dysfunction in autism: functional brain imaging studies. *Current opinion in neurology*, 23(2), 124

Modabbernia, A., Velthorst, E., & Reichenberg, A. (2017). Environmental risk factors for autism: an evidence-based review of systematic reviews and meta-analyses. *Molecular autism*, 8(1), 13.

Mosconi, M. W., Mohanty, S., Greene, R. K., Cook, E. H., Vaillancourt, D. E., & Sweeney, J. A. (2015). Feedforward and feedback motor control abnormalities implicate cerebellar dysfunctions in autism spectrum disorder. *Journal of Neuroscience*, 35(5).

Meyer, D. E., Abrams, R. A., Kornblum, S., Wright, C. E., & Keith Smith, J. E. (1988). Optimality in human motor performance: ideal control of rapid aimed movements. *Psychological review*, 95(3), 340.

Myers, S. M., Voigt, R. G., Colligan, R. C., Weaver, A. L., Storlie, C. B., Katusic, S. K., ... Port, J. D. (2019). Autism Spectrum Disorder: Incidence and Time Trends Over Two Decades in a Population-Based Birth Cohort. *Journal of Autism & Developmental Disorders*, 49(4), 1455–1474. <https://0-doi-org.lib.utep.edu/10.1007/s10803-018-3834-0>

Nagae, L. M., Zarnow, D. M., Blaskey, L., Dell, J., Khan, S. Y., Qasmieh, S., ... & Roberts, T. P. L. (2012). Elevated mean diffusivity in the left hemisphere superior longitudinal fasciculus in autism spectrum disorders increases with more profound language impairment. *American Journal of Neuroradiology*, 33(9), 1720-1725.

- Nayate, A., Bradshaw, J. L., & Rinehart, N. J. (2005). Autism and Asperger's disorder: are they movement disorders involving the cerebellum and/or basal ganglia?. *Brain research bulletin*, 67(4), 327-334.
- Opfer, J. E. (2002). Identifying living and sentient kinds from dynamic information: The case of goal-directed versus aimless autonomous movement in conceptual change. *Cognition*, 86(2), 97-122.
- Packer, A. (2016). Neocortical neurogenesis and the etiology of autism spectrum disorder. *Neuroscience & Biobehavioral Reviews*, 64, 185-195.
- Papadopoulos, N., McGinley, J., Tonge, B. J., Bradshaw, J. L., Saunders, K., & Rinehart, N. J. (2012). An investigation of upper limb motor function in high functioning autism and Asperger's disorder using a repetitive Fitts' aiming task. *Research in Autism Spectrum Disorders*, 6(1), 286-292
- Paquet, A., Olliac, B., Bouvard, M. P., Golse, B., & Vaivre-Douret, L. (2016). The semiology of motor disorders in autism spectrum disorders as highlighted from a standardized neuro-psychomotor assessment. *Frontiers in psychology*, 7, 1292.
- Paquet, A., Olliac, B., Golse, B., & Vaivre-Douret, L. (2016). Current knowledge on motor disorders in children with autism spectrum disorder (ASD). *Child Neuropsychology*, 22(7), 763-794. <https://doi-org.lib.utep.edu/10.1080/09297049.2015.1085501>
- Paulin, M. G. (2005). Evolution of the cerebellum as a neuronal machine for Bayesian state estimation. *Journal of Neural Engineering*, 2(3), S219.
- Paulin, M. G., & Hoffman, L. F. (2011). Bayesian head state prediction: computing the dynamic prior with spiking neurons. In *Natural Computation (ICNC), 2011 Seventh International Conference on* (Vol. 1, pp. 445-449). IEEE.
- Phillips, J. G., Bradshaw, J. L., Iansek, R., & Chiu, E. (1993). Motor functions of the basal ganglia. *Psychological research*, 55(2), 175-181.
- Plamondon, R., & Alimi, A. M. (1997). Speed/accuracy trade-offs in target-directed movements. *Behavioral and brain sciences*, 20(2), 279-303.
- Provost, B., Lopez, B. R., & Heimerl, S. (2007). A comparison of motor delays in young children: autism spectrum disorder, developmental delay, and developmental concerns. *Journal of autism and developmental disorders*, 37(2), 321-328.
- Qiu, A., Adler, M., Crocetti, D., Miller, M. I., & Mostofsky, S. H. (2010). Basal ganglia shapes predict social, communication, and motor dysfunctions in boys with autism spectrum disorder. *Journal of the American Academy of child & adolescent Psychiatry*, 49(6), 539-551

Ritvo, E. R., Freeman, B. J., Scheibel, A. B., Duong, T., Robinson, H., Guthrie, D., & Ritvo, A. (1986). Lower Purkinje cell counts in the cerebella of four autistic subjects: Initial findings of the UCLA-NSAC research report. *The American journal of psychiatry*.

Ring, H. A., & Serra-Mestres, J. (2002). Neuropsychiatry of the basal ganglia. *Journal of Neurology, Neurosurgery & Psychiatry*, 72(1), 12-21.

Rinehart, N. J., Bellgrove, M. A., Tonge, B. J., Brereton, A. V., Howells-Rankin, D., & Bradshaw, J. L. (2006). An examination of movement kinematics in young people with high-functioning autism and Asperger's disorder: further evidence for a motor planning deficit. *Journal of autism and developmental disorders*, 36(6), 757-767.

Rinehart, N. J., Bradshaw, J. L., Brereton, A. V., & Tonge, B. J. (2001). Movement preparation in high-functioning autism and Asperger disorder: a serial choice reaction time task involving motor reprogramming. *Journal of autism and developmental disorders*, 31(1), 79-88.

Rogers, S. J. (2009). What are infant siblings teaching us about autism in infancy?. *Autism Research*, 2(3)

Rowberry, J., Macari, S., Chen, G., Campbell, D., Leventhal, J. M., Weitzman, C., & Chawarska, K. (2015). Screening for autism spectrum disorders in 12-month-old high-risk siblings by parental report. *Journal of autism and developmental disorders*, 45(1), 221-229.

Rudie, J. D., Brown, J. A., Beck-Pancer, D., Hernandez, L. M., Dennis, E. L., Thompson, P. M., ... & Dapretto, M. J. N. C. (2013). Altered functional and structural brain network organization in autism. *NeuroImage: clinical*, 2, 79-94.

Rutter, M., Andersen-Wood, L., Beckett, C., Bredenkamp, D., Castle, J., Groothues, C., Kreppner, J., Keaveney, L., Lord, C & O'Connor, T. G. (1999). Quasi-autistic patterns following severe early global privation. *The Journal of Child Psychology and Psychiatry and Allied Disciplines*, 40(4), 537-549.

Sacrey, L. A. R., Germani, T., Bryson, S. E., & Zwaigenbaum, L. (2014). Reaching and grasping in autism spectrum disorder: a review of recent literature. *Frontiers in Neurology*, 5, 6.

Santangelo, S. L., & Tsatsanis, K. (2005). What is known about autism. *American Journal of Pharmacogenomics*, 5(2), 71-92.

Schmahmann, J. D., & Pandyat, D. N. (1997). The cerebrotocerebellar system. In *International review of neurobiology* (Vol. 41, pp. 31-60). Academic Press.

Schmitz, N., Rubia, K., Daly, E., Smith, A., Williams, S., & Murphy, D. G. (2006). Neural correlates of executive function in autistic spectrum disorders. *Biological psychiatry*, 59(1), 7-16.

Sealey, L. A., Hughes, B. W., Sriskanda, A. N., Guest, J. R., Gibson, A. D., Johnson-Williams, L., & Bagasra, O. (2016). Environmental factors in the development of autism spectrum disorders. *Environment international*, 88, 288-298.

Silva, L. C., Teixeira, M. C., Ribeiro, E. L., & Paula, C. S. (2017). Impact of a provider training program on the treatment of children with autism spectrum disorder at psychosocial care units in Brazil. *Revista Brasileira de Psiquiatria*, (AHEAD), 0-0.

Shannon, C. E. (1949). *The Mathematical Theory of Communication*, by CE Shannon (and Recent Contributions to the Mathematical Theory of Communication), W. Weaver. University of Illinois Press.

Sheldrick, R. C., & Carter, A. S. (2018). State-Level Trends in the Prevalence of Autism Spectrum Disorder (ASD) from 2000 to 2012: A Reanalysis of Findings from the Autism and Developmental Disabilities Network. *Journal of Autism & Developmental Disorders*, 48(9), 3086–3092. <https://doi-org.lib.utep.edu/10.1007/s10803-018-3568-z>

Sleimen-Malkoun, R., Temprado, J. J., Huys, R., Jirsa, V., & Berton, E. (2012). Is Fitts' law continuous in discrete aiming?. *PLoS One*, 7(7).

Smits-Engelsman, B. C. M., Van Galen, G. P., & Duysens, J. (2002). The breakdown of Fitts' law in rapid, reciprocal aiming movements. *Experimental Brain Research*, 145(2), 222-230.

Smits-Engelsman, B. C., Sugden, D., & Duysens, J. (2006). Developmental trends in speed accuracy trade-off in 6–10-year-old children performing rapid reciprocal and discrete aiming movements. *Human movement science*, 25(1), 37-49.

Smyrnis, N., Evdokimidis, I., Constantinidis, T. S., & Kastrinakis, G. (2000). Speed-accuracy trade-off in the performance of pointing movements in different directions in two-dimensional space. *Experimental Brain Research*, 134(1), 21-31.

Staples, K. L., & Reid, G. (2010). Fundamental movement skills and autism spectrum disorders. *Journal of autism and developmental disorders*, 40(2), 209-217.

Stoit, A. M., van Schie, H. T., Slaats-Willemsse, D. I., & Buitelaar, J. K. (2013). Grasping motor impairments in autism: not action planning but movement execution is deficient. *Journal of autism and developmental disorders*, 43(12), 2793-2806.

Teitelbaum, P., Teitelbaum, O., Nye, J., Fryman, J., & Maurer, R. G. (1998). Movement analysis in infancy may be useful for early diagnosis of autism. *Proceedings of the National Academy of Sciences*, 95(23), 13982-13987.

Temprado, J. J., Sleimen-Malkoun, R., Lemaire, P., Rey-Robert, B., Retornaz, F., & Berton, E. (2013). Aging of sensorimotor processes: a systematic study in Fitts' task. *Experimental brain research*, 228(1), 105-116.

Tick, B., Bolton, P., Happé, F., Rutter, M., & Rijdsdijk, F. (2016). Heritability of autism spectrum disorders: a meta-analysis of twin studies. *Journal of Child Psychology and Psychiatry*, 57(5), 585-595.

Vilensky, J. A., Damasio, A. R., & Maurer, R. G. (1981). Gait disturbances in patients with autistic behavior: a preliminary study. *Archives of neurology*, 38(10), 646-649.

Whitney, E. R., Kemper, T. L., Bauman, M. L., Rosene, D. L., & Blatt, G. J. (2008). Cerebellar Purkinje cells are reduced in a subpopulation of autistic brains: a stereological experiment using calbindin-D28k. *The Cerebellum*, 7(3), 406-416.

Wing, L. (1990). What is autism? In *Autism* (pp. 1-24). Springer, Boston, MA.

Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: is the prevalence rising?. *Mental retardation and developmental disabilities research reviews*, 8(3), 151-161.

Wills, S., Cabanlit, M., Bennett, J., Ashwood, P., Amaral, D., & Van De Water, J. (2007). Autoantibodies in autism spectrum disorders (ASD). *Annals of the New York Academy of Sciences*, 1107(1), 79-91.

Won, Hyejung, Won Mah, and Eunjoon Kim. "Autism spectrum disorder causes, mechanisms, and treatments: focus on neuronal synapses." *Frontiers in molecular neuroscience* 6 (2013): 19.

Woodworth, R. S. (1899). Accuracy of voluntary movement. *The Psychological Review: Monograph Supplements*, 3(3), i

Zaal, F. T., & Thelen, E. (2005). The developmental roots of the speed-accuracy trade-off. *Journal of Experimental Psychology: Human Perception and Performance*, 31(6), 1266.

Zhang, F., & Roeyers, H. (2019). Exploring brain functions in autism spectrum disorder: A systematic review on functional near-infrared spectroscopy (fNIRS) studies. *International Journal of Psychophysiology*.

Zimmerli, L., Krewer, C., Gassert, R., Müller, F., Riener, R., & Lünenburger, L. (2012). Validation of a mechanism to balance exercise difficulty in robot-assisted upper-extremity rehabilitation after stroke. *Journal of neuroengineering and rehabilitation*, 9(1), 6.

Vita

Ms. Jallycia Pearson obtained a B.S in Kinesiology and will graduate in May 2020 with a M.S in Kinesiology from the University of Texas at El Paso. Throughout her education, Ms. Pearson attained the opportunity to work in the Virtual Reality and Motor Control Laboratory and be a part of many different research projects working with children with Autism Spectrum Disorder.

After graduation, Ms. Pearson will apply to the Physical Therapy Program in hopes to continue studying the movement of the human body through

Permanent address: 6909 Oveja Ave, El Paso, Texas, United States 79912

This thesis was typed by Jallycia R Pearson