1   Getting off to a shaky start: specificity in planning and feedforward control during

2   sensorimotor learning in autism spectrum disorder

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Abstract

Whilst autistic individuals develop new internal action models during sensorimotor learning, the acquired movements are executed less accurately and with greater variability. Such movement profiles are related to differences in sensorimotor integration and/or altered feedforward/feedback sensorimotor control. We investigated the processes underlying sensorimotor learning in autism by quantifying accuracy and variability, relative timing, and feedforward and feedback control. Although autistic individuals demonstrated significant sensorimotor learning across trials, which was facilitated by processing knowledge-of-results feedback, motor execution was less accurate than non-autistic individuals. Kinematic analysis indicated that autistic individuals showed significantly greater spatial variability at peak acceleration, but comparable spatial variability at peak velocity. These kinematic markers suggest that autistic movement profiles are driven by specific differences in sensorimotor control processes (i.e., internal action models) associated with planning and regulating the forces required to execute the movement. The reduction of variability at peak velocity indicates intact early feedback-based sensorimotor control in autism. Understanding how feedforward and feedback-based control processes operate provides an opportunity to explore how these control processes influence the acquisition of socio-motor actions in autism.

Lay Summary: Autistic adults successfully learned a new movement skill by physically practising it, and using feedback about how well they had done to become more accurate. When looking at the movements in detail, autistic adults were more variable than non-autistic adults when planning (e.g., how much force to use), and performing, the movement. These differences impact how autistic individuals learn different types of movement skills, which might influence how other behaviours (e.g., imitation) are acquired that support social interaction.

Key words: sensorimotor learning, feedforward and feedback motor control, autism

Introduction

Autism Spectrum Disorder (henceforth 'autism') is a neurodevelopmental condition characterised by restricted and repetitive patterns of behaviour, and an impaired ability to communicate and interact with others (American Psychiatric Association, 2013). Although not part of the formal diagnostic criteria, autistic individuals often show atypical sensorimotor behaviour (Fournier, Hass, Naik, Lodha, & Cauraugh, 2010; Gowen & Hamilton, 2013). For example, there are reports of greater clumsiness during gait (Calhoun, Longworth, & Chester, 2011; Rinehart, Tonge, et al., 2006), atypical motor coordination (Green et al., 2002), planning (Glazebrook, Elliott, & Szatmari, 2008), postural instability (Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer, 1998) and generally worse performance on standardised tests of motor function (Green et al., 2009). The sensorimotor basis of these motor difficulties may explain why autistic individuals experience difficulty in praxis (Dewey, Cantell, & Crawford, 2007) and acquiring new sensorimotor skills important for social interaction.

Novel sensorimotor behaviours are generally acquired via trial-and-error learning, where internal action models are developed by representing associations between descending motor commands (efferent outflow) that drive a limb towards a specified movement goal, the sensory consequences (reafferent inflow from vision and proprioception) of limb movement (Wolpert, Ghahramani, & Jordan, 1995), and parameters of the external world (height of a basketball hoop). Following learning, internal action models form part of a mechanism that underpins sensorimotor planning and feedforward control, as well as regulating online movement control and sensorimotor adaptation by processing and comparing incoming feedback (vision and proprioception) with that predicted by the action model. Research suggests that the development of internal action models is functional in autistic individuals (Gidley Larson, Bastian, Donchin, Shadmehr, & Mostofsky, 2008; Haswell, Izawa, Dowell, Mostofsky, & Shadmehr, 2009; Hayes et al., 2018; Izawa et al., 2012; Müller, Cauich, Rubio, Mizuno, & Courchesne, 2004). For example, when beginning to

1   learn a motor aiming task both autistic and neurotypical groups were influenced by prisms

2   that perturbed the visuomotor relationship between their body and the target location (Gidley

3   Larson et al., 2008). Both groups then demonstrated sensorimotor adaptation to the prisms

4   over training by becoming more accurate at achieving the task goal. Adaptation indicated

5   performers successfully compared the expected consequences (efference copy) of an

6   executed movement on *trial n* against the actual sensory (reafference; visual and

7   proprioceptive) feedback, and subsequently made corrective adjustments when planning

8   *trial n+1* (Wolpert, Diedrichsen, & Flanagan, 2011). When the prisms were removed in a

9   post-test, both groups showed after-effects where outcome performance was skewed (target

10   accuracy decreased) in the opposite direction to the perturbation. Corrective and adaptive

11   processes, plus the occurrence of after-effects, would not be expected if the sensorimotor

12   processes underpinning internal action model formation were deficient in autism.

13   There is neuropsychological (Allen, Müller, & Courchesne, 2004; Courchesne, Press,

14   & Yeung-Courchesne, 1993; Müller et al., 2004; Müller, Kleinhans, Kemmotsu, Pierce, &

15   Courchesne, 2003; Sharer et al., 2015; Travers, Kana, Klinger, Klein, & Klinger, 2015) and

16   behavioural (Ament et al., 2015; Fournier et al., 2010; Gowen & Hamilton, 2013; Haswell et

17   al., 2009; Mostofsky, Goldberg, Landa, & Denckla, 2000) evidence indicating atypical

18   sensorimotor integration during the formation of action models in autism, which can

19   influence how movements are planned and executed. For example, although autistic

20   volunteers developed action models for a novel visuomotor sequence timing task (Hayes et

21   al., 2018), the duration of executed movements was less accurate and more variable than

22   those performed by neurotypical participants. Slower movements were evident both with

23   knowledge-of-results feedback (acquisition phase), and without (retention test).

24   Similar findings have been reported for a single-segment aiming task, with autistic

25   volunteers taking up to 50% longer than neurotypical individuals to reach the target

26   (Glazebrook, Elliott, & Lyons, 2006). Interestingly, autistic volunteers in this study also

27   showed greater variability in the spatial position of peak acceleration. Increased variability in

this kinematic marker is reflective of sensorimotor control processes associated with the planning and control of muscular forces required to generate (i.e., an inverse model, see Wolpert & Kawato, 1998) and update (i.e., feedforward control; see Desmurget & Grafton, 2000; Wolpert & Flanagan, 2010) the motor command for goal-directed movement (Glazebrook et al., 2006; Hughes, 1996; Mari, Castiello, Marks, Marraffa, & Prior, 2003; Rinehart, Bradshaw, Brereton, & Tonge, 2001). Specifically, variabilty in planning a motor command leads to less efficient initial motor execution (Elliott et al., 2010), whereas the efficacy of the internal forward model (i.e., efference copy) impacts upon early movement execution during the processing and integration of expected and actual sensorimotor information (Glazebrook et al., 2006; Mosconi et al., 2015; Schmitz, Martineau, Barthélémy, & Assaiante, 2003). Notably, however, the autistic group were comparable to the non-autistic group in terms of the overall structuring of the movement (i.e., proportional time after peak velocity and displacement at peak velocity) and in the processing of visual information for online movement control.

In the present study we investigated the underlying sensorimotor control processes that operate while volunteers (autistic and neurotypical) learned a visuomotor sequence timing task (VSTT). The VSTT required volunteers to move a stylus on a graphics tablet through a 3-segment movement sequence with a timing goal of 1700 ms. The VSTT was selected because it is a goal-directed action that has successfully been shown (Hayes et al., 2018) to quantify sensorimotor learning in autistic volunteers using outcome accuracy and variability error scores. Importantly, with a 1700ms timing goal the duration of each segment (see results below) within the sequence is long enough for participants to make online sensorimotor corrections (see Schmidt et al., 1979). Therefore, as well as facilitating the quantification of outcome-based dependent variables, the 1700ms VSTT allows us to extend our understanding of how the underlying sensorimotor control processes operate during acquisition. One additional benefit of using the VSTT is that participants learn a self-selected, rather than an experimenter-imposed, 3-segment relative timing pattern (Heuer &

1   Schmidt, 1988; Schmidt, 1985). Therefore, using detailed movement analysis it is possible to

2   measure specific kinematic markers (Khan et al., 2006) during the acquisition of a self-

3   selected relative timing task that requires sensorimotor planning and feedforward control

4   across a number of movement segments.

5   Based on our previous study (Hayes et al., 2018), we expected to find that autistic

6   volunteers learn the VSTT timing goal by reducing timing error and variability through trial-

7   and-error learning that involved processing knowledge-of-results. Despite such learning, we

8   still expected timing error to be greater, and more variable, in autistic volunteers than a

9   neurotypical control group during both acquisition and retention (Glazebrook et al., 2006;

10   Hayes et al., 2018). Extrapolating from work on single-segment manual aiming (Glazebrook

11   et al., 2006), we expected both groups to execute comparable relative timing patterns. In

12   terms of motor control, if the expected differences in timing accuracy (longer movement

13   times) and variability are associated with the specificity of the underlying autistic

14   sensorimotor planning and feedforward control processes, we expected greater variability in

15   the spatial position of peak acceleration in the autism group compared to the neurotypical

16   group. Finally, given that autistic individuals show intact visual online motor control

17   (Glazebrook et al., 2006; Mosconi et al., 2015), we expected no difference between the

18   groups in variability in the spatial position of peak velocity.

19   **Method**

20   *Volunteers*

21   Volunteers were recruited from an autistic society, and the host university, and

22   provided with a participant information sheet to read, followed by an opportunity to ask

23   questions to clarify the experimental procedures, and then a time period to consider whether

24   they consent to participate in the study. Following this process, 26 neurotypical (25 male; 1

25   female), and 26 autistic (25 male; 1 female) volunteers participated. All participants were

26   right-handed and indicated this via self-report following a standard set of pre-experimental

1   questions ("which hand do you write with"; "which hand do you throw with"; which hand do

2   you use to brush your teeth"). Furthermore, participants were screened via self-report for the

3   following exclusion criteria: dyspraxia, dyslexia, epilepsy and other neurological or

4   psychiatric conditions. Autistic participants had a diagnosis of autism, Asperger's syndrome,

5   or autism spectrum disorder by an independent clinician. Diagnosis was confirmed by a

6   researcher trained (with research-reliability status) in the administration of module 4 of the

7   Autism Diagnostic Observation Schedule 2 (ADOS-2) (Lord et al., 2012). Autistic participants

8   met the threshold for autism spectrum disorder on the ADOS-2 total classification score, and

9   on the communication, and social interaction subscales. Groups were matched for age, as

10 well as full-scale, verbal, and performance IQ, which was measured using the Wechsler

11 Abbreviated Scale of Intelligence (WASI) (Wechsler, 1999). Participant characteristics are

12 presented in *Table 1*. In addition to the autistic volunteers who participated in the study, we

13 also engaged with a group (n = 6; 1 female; 5 male) of autistic advocates who helped to

14 develop the methods via a participatory research process (Fletcher-Watson et al., 2019,

15 Nicolaidis et al., 2011). During engagement, advocates offered their opinion on the to-be-

16 used apparatus, number of trials, task instructions, how the participant information sheets

17 were constructed, and the research question on sensorimotor learning. Feedback from the

18 participatory engagement process was used to refine the methods. Interestingly, there was

19 consensus from the autistic advocates indicating from their own experience that

20 understanding sensorimotor processing in autism was an important yet under-addressed

21 area of research (Robledo et al., 2012). Finally, the experiment was designed in accordance

22 with the 1964 declaration of Helsinki and received full approval by the host University

23 Research Ethics Committee.

24

25   Insert Table 1 about here.

26

1   *Apparatus*

2   Participants sat at a table in front of a 21-inch CRT monitor (Iiyama Vision Master

3   505) located at a viewing distance of approximately 900 mm. The CRT monitor had a

4   resolution of 1280 x 1024 pixels, and a refresh rate of 85 Hz. The monitor was connected to

5   a desktop PC (Dell Optiplex GX280), which received input from a hand-held stylus as it

6   moved on a graphics tablet (Wacom Intuos Pro XL; Figure 1). Experimental stimuli were

7   presented on the CRT monitor using the COGENT toolbox (developed by John Romaya at

8   the Laboratory of Neurobiology at the Wellcome Department of Imaging Neuroscience)

9   implemented in MATLAB (Mathworks Inc.)

10

11 *Procedure*

12 All participants first performed a familiarisation period where they sat in front of the

13 CRT monitor (Figure 1) and received a visual demonstration, plus verbal instructions, of the

14 VSTT. The VSTT required a participant to move the cursor horizontally rightwards so that it

15 was located in the middle target (segment 1), followed by a leftwards reversal to locate the

16 cursor in the start circle (segment 2), and finally a rightwards reversal to move the cursor

17 through the middle target and then stop in the right-hand end target (segment 3). Once

18 participants confirmed they understood how to complete the VSTT, they were informed the

19 goal of the task was to perform the 3-segment movement in a timing goal of 1700 ms

20 exactly. All participants were informed about, and confirmed that they understood, the

21 millisecond unit. In the acquisition period participants performed 30 trials of the VSTT using

22 their preferred arm. To ensure participants performed the correct spatial dimensions of the

23 movement sequence, the stimulus generation routine presented an error message on the

24 monitor if the cursor did not pass through each target in the correct sequence order (NB. no

25 error trials were recorded). To facilitate sensorimotor adaptation in the acquisition phase,

26 terminal feedback in the form of knowledge-of-results was presented on the monitor

27 following each trial (e.g., Too Fast or Too Slow by 350 ms). All participants were informed

1 and confirmed that they understood how knowledge-of-results after trial *n* could be used to

2 modify trial *n + 1.* Following the acquisition period, six retention trials without knowledge-of-

3 results were completed to assess sensorimotor learning.

4

5   Insert Figure 1 around here.

6

7 *Data Reduction*

8   Using a custom MATLAB routine we identified the start and end of each 3-segment

9 movement sequence from the x-axis position data. The start was defined as the moment the

10 centre of the cursor moved beyond the perimeter of the start-target, and the end equated to

11 when the centre of the cursor moved within the perimeter of the end-target. Using these

12 points, we extracted the time-series position data for each acquisition and retention test trial.

13 The position data for each trial were processed using a low-pass 4th order autoregressive

14 filter with an 8 Hz cut-off, and then differentiated using a 2-point central difference algorithm

15 to obtain velocity and acceleration. For each trial, the end of the movement made in

16 segment 1 and 2 was identified by searching for a zero-crossing in the velocity data that was

17 associated with a change in movement direction (i.e., reversal).

18   Having identified the start and end of a trial, as well the individual segments within

19 the sequence, we extracted four dependent variables. *Total error* is an outcome error

20 measure that reflects accuracy and consistency of achieving the 1700 ms timing goal

21 (Schmidt, Lee, Winstein, Wulf, & Zelaznik, 2018). It is calculated as $\sqrt{CE^2 + VE^2}$, where

22 constant error (CE) is a measure reflecting the average signed deviation (e.g., plus or

23 minus) between a movement time on *trial n* (e.g., 1900 ms) and the criterion timing goal that

24 is 1700 ms (e.g., a movement time of 1900 ms would lead to +200 ms), and variable error

25 (VE) quantifies variability in the responses across a set number of trials (e.g., 6 trials, see

26 the data analysis section below) around the average CE for the same 6 trials. To quantify

27 *relative timing* (i.e., a measure of how the 3 segments are proportionally expressed relative

                                                                                            10

to the total movement time; Schmidt, 1975), each segment within the 3-segment sequence was expressed as a percentage of the overall movement time. For example, if on *trial n* a participant performs the VSTT in a total movement time of 1800 ms, and the segment movement times are 300, 500 and 1000 ms respectively, the *relative timing* structure would be 17%, 28%, and 55%. To quantify sensorimotor control, we extracted *spatial variability at the position of peak acceleration (sdPA)*, and *peak velocity (sdPV)*. The variability in distance travelled at *peak acceleration* is reflective of the effectiveness of planning the correct specification of muscular forces, and early sensorimotor corrections based on the comparison of expected, and actual, efference, plus early sensorimotor (proprioception; vision) afference (see Elliott et al., 2010).

Data Analysis

To examine changes in motor adaptation across acquisition, mean *total error* was calculated from the first and last six of the 30 acquisition trials. Data were submitted to a 2 Group (autism; neurotypical) x 2 Phase (early; late) mixed design ANOVA. To quantify how the three individual movement segments were learned, mean *relative timing*, *sdPA*, and *sdPV* were calculated from the first and last six trials of acquisition. These data were submitted to separate 2 Group (autism; neurotypical) x 2 Phase (early; late) x 3 Segment (one; two; three) mixed design ANOVAs.

To assess sensorimotor learning in the retention test, mean *total error* was calculated for the six retention trials and submitted to a 2 Group (autism; neurotypical) one-way ANOVA. For *relative timing*, *sdPA* and *sdPV,* means from the six retention trials were submitted to separate 2 Group (autism; neurotypical) x 3 Segment (1, 2, 3) mixed design ANOVAs.

To establish whether knowledge-of-results feedback provided on a trial was used to modify total movement time on the next trial, we calculated the difference in movement time performed on *trial n* and the target movement time (1700 ms). The resulting value provides the directional (+/-) error on that trial attempt, and when presented as knowledge-of-results it

1 provides the direction (+ or -) and magnitude of the correction in ms to be made on *trial n+1*.

2 Next, we calculated the signed (+ or -) magnitude of the correction made on *trial n+1* by

3 subtracting the movement time performed on *trial n* from the movement time performed on

4 *trial n+1*. We then correlated the two measures for the block of 6 trials performed by each

5 participant during the early and late phases of acquisition. Within each group, a strong

6 negative correlation would suggest participants used knowledge-of-results feedback to adapt

7 motor performance on a trial-to-trial basis (Blandin & Proteau, 2000). Following Fisher's R to

8 Z transformation, correlation scores were analysed using a 2 Group (autism; neurotypical) x

9 2 Phase (early; late) mixed design ANOVA.

10 To establish whether the degree of sensorimotor learning measured in the retention

11 test is related to the magnitude of sensorimotor adaptation across acquisition, we first

12 computed the *percentage change* (%Δ) between the mean *total error* in the first six (early)

13 and last six (late) acquisition trials: %Δ = ((late $\overline{x}$ - early $\overline{x}$) / early $\overline{x}$)*100. We then performed

14 separate group correlation analyses on the *percentage change scores* (%Δ) against the

15 mean *total error* scores in the retention test.

16 Significant main and/or interaction effects were decomposed using Fisher LSD post-

17 hoc procedure, with alpha set at $p < 0.05$. Partial eta squared (η_p^2) was used to express the

18 size of each effect. ANOVAs that included three levels of segment as a within-subject factor

19 were checked for violation of sphericity using Mauchly's Sphericity Test and corrected where

20 necessary with Greenhouse-Geisser (i.e., $p < 0.05$). Additionally, a Bayesian approach

21 (implemented in JASP; JASP Team, 2019) was used to evaluate evidence for the alternative

22 hypothesis compared to the null hypothesis, where stronger evidence for accepting an

23 alternative hypothesis is related to the magnitude of the Bayes factor (BF) value (Jarosz &

24 Wiley, 2014; Jeffreys, 1961). For example, a value below 1 indicates no evidence; a value

25 between 1 and 3 provides anecdotal evidence; a value greater than 3 provides moderate

1   evidence; and a value greater than 10 provides strong evidence (see Lee & Wagenmakers,

2   2014).

3

4   **Results**

5   <u>Acquisition</u>

6   Group mean *total error* is illustrated in Figure 2. ANOVA revealed no significant

7   group x phase interaction [$F(1, 50) = 1.30$, $p = 0.26$, $\eta_p^2 = 0.025$, BF = 1.81], but significant

8   main effects were observed for group [$F(1, 50) = 7.82$, $p = 0.007$, $\eta_p^2 = 0.135$, BF = 4.117 x

9   10^{11}] and phase [$F(1, 50) = 88.05$, $p < 0.001$, $\eta_p^2 = 0.638$, BF = 5.31]. Although the autism

10 group demonstrated greater *total error* compared to the neurotypical group, both groups

11 demonstrated similar reductions in *total error* from early acquisition (Autism: 1347.66 ±

12 691.64 ms; Neurotypical: 969.90 ± 479.08 ms) to late acquisition (Autism: 531.67 ± 322.35

13 ms; Neurotypical: 330.91 ± 238.61 ms).

14

15   Insert Figure 2 around here.

16

17   For *relative timing*, ANOVA revealed no significant main effect of group [$F(1, 50) =$

18 1.85, $p = 0.18$, $\eta_p^2 = 0.036$, BF = 1.11], but there was a significant main effect for segment [F

19 $(2, 100) = 272.95$, $p < 0.001$, $\eta_p^2 = 0.845$, BF = ∞]. Post hoc analyses indicated a difference

20 ($p = 0.04$) between segment 1 (30 ± 3 %) compared to segment 2 (29 ± 3 %) and segment 3

21 (41 ± 3 %), as well as segment 2 compared to segment 3. As illustrated in Figure 3 and

22 Table 2, a group x segment interaction [$F(2, 100) = 3.35$, $p = 0.04$, $\eta_p^2 = 0.063$, BF = 5.35]

23 indicated that both groups spent similar relative time executing the movement in segment 1

24 (mean group difference = 0.7 units) and segment 2 (mean group difference = 0.9 units).

25 However, the autism group exhibited significantly longer relative time in segment 3 than the

26 control group (mean difference = 1.7 units).

27

1   Insert Figure 3 and Table 2 around here.

2

3

4   To supplement the discrete *relative timing* data, normalised group mean movement

5   profiles in the x and y axis, along with group mean within-participant standard deviation,

6   were calculated and are plotted in Figure 4. As illustrated, within-participant standard

7   deviation in the x-axis for the autism group (red shaded area) is greater in segments 2 and 3

8   in the late acquisition and retention phases compared to the neurotypical group (blue

9   shaded area). Within-participant standard deviation is lower overall in the y-axis and similar

10   for both groups.

11

12   Insert Figure 4 around here.

13

14   Group mean *sdPA* is illustrated in Figure 5a and Table 2. ANOVA revealed

15   significant main effects of group [$F_{(1, 50)}$ = 4.792, p = 0.03, η_p^2 = 0.087, BF = 0.33],

16   segment [$F_{(1.47, 73.36)}$ = 121.29, $p < 0.001$, η_p^2 = 0.708, BF = 1.608 x 10^{15}], and phase [F

17   $_{(1, 50)}$ = 20.91, $p < 0.001$, η_p^2 = 0.295, BF = 7.277 x 10^7]. *sdPA* was greater in the autism

18   group (10.27± 8.78 mm) compared to neurotypical group (8.78± 6.40 mm), and was

19   significantly (*ps < 0.05*) greater in segment 2 (17.19 ± 6.21 mm) and 3 (6.20 ± 2.62 mm)

20   than segment one (5.19 ± 3.36 mm). There was also a significant segment x phase

21   interaction [$F_{(1.40, 69.95)}$ = 20.04, $p < 0.001$, η_p^2 = 0.286, BF = 443028.67], which indicated

22   that *sdPA* decreased by 7.79 mm ($p < 0.001$) from early to late acquisition in segment 2,

23   whereas there was no significant changes in segment 1 (p = 0.14) or 3 (p = 0.44).

24

25   Insert Figure 5 around here.

26

14

1   Group mean *sdPV* is illustrated in Figure 5b and Table 2. ANOVA revealed no

2 significant main effect of group [$F_{(1, 50)} = 1.587$, $p = 0.21$, $\eta_p^2 = 0.031$, BF = 0.15], or any

3 significant 2-way or 3-way interactions (*ps > 0.05*). There was, however, a significant main

4 effect of phase [$F_{(1, 50)} = 4.23$, $p = 0.045$, $\eta_p^2 = 0.078$, BF = 0.42] where *sdPV* decreased

5 by 2.19 mm from the early to late phase of acquisition. There was also a significant main

6 effect of segment [$F_{(1.15, 57.31)} = 51.43$, $p < 0.001$, $\eta_p^2 = 0.507$, BF = 3.217×10^{15}]. *sdPV*

7 was greater in segment 1 (10.77 ± 2.99 mm) compared to segment 2 (7.84 ± 2.17 mm) ($p <$

8 0.001), and also greater in segment 3 (21.23 ± 12.18 mm) ($p < 0.001$).

9   Large negative correlations for the autism [early: $r = -0.8$; late: $r = -0.7$] and

10 neurotypical [early: $r = -0.8$; late: $r = -0.7$] groups during early and late acquisition blocks

11 indicated strong relationships between the magnitude and direction of knowledge-of-results

12 feedback on trial *n,* and the resulting error correction on trial *n+1*. Follow-up ANOVAs on the

13 transformed correlation coefficients revealed no significant effects of group [$F_{(1, 50)} = 0.51$

14 $p = 0.48$, $\eta_p^2 = 0.010$, BF = 0.23], and phase [$F_{(1, 50)} = 3.66$ $p = 0.06$, $\eta_p^2 = 0.068$, BF =

15 1.31], or phase x group interaction [$F_{(1, 50)} = 0.06$ $p = 0.80$, $\eta_p^2 = 0.001$, BF = 0.16].

16 Therefore, there was no difference in the trial-to-trial error correction process used by both

17 groups during sensorimotor adaption in the acquisition trials.

18

19 Retention

20   A significant main effect of group [$F_{(1, 50)} = 12.77$, $p = 0.001$, $\eta_p^2 = 0.203$, BF =

21 37.76] for *total error* revealed the neurotypical group had a total error score that was 312 ms

22 lower than the autism group when performing the timing goal without knowledge-of-results.

23   For relative timing, there was a significant main effect for segment [$F_{(1.47, 73.44)} =$

24 174.06, $p < 0.001$, $\eta_p^2 = 0.777$, BF = ∞], but no main effect for group [$F_{(1, 50)} = 0.99$, $p =$

25 0.33, $\eta_p^2 = 0.019$, BF = 0.32] or a group x segment interaction [$F_{(2, 100)} = 2.20$, $p = 0.12$, η_p^2

26 = 0.042, BF = 0.94]. As illustrated in *Figure 3*, the autism group (Segment 1: 30 ± 3 %;

27 Segment 2: 29 ± 3 %; Segment 3: 41 ± 4 %) executed the three-segment movement

15

1 sequence with comparable *relative timing* as the neurotypical group (Segment 1: 30 ± 2 %;

2 Segment 2: 30 ± 2 %; Segment 3: 40 ± 3 %).

3   For *sdPA,* there were significant main effects for segment [$F(1.31, 65.54) = 58.83$, p

4 < 0.001, $\eta_p^2 = 0.541$, BF = ∞] and group [$F(1, 50) = 6.06$, $p = 0.02$, $\eta_p^2 = 0.108$, BF = 11.36],

5 plus a significant group x segment interaction [$F(2, 100) = 5.23$, $p = 0.007$, $\eta_p^2 = 0.095$, BF =

6 24.85]. As illustrated in Figure 5a, *sdPA* was greater (both *ps* < 0.001) in segment 2 (14.39 ±

7 8.6 mm) compared to segment 1 (3.85 ± 2.22 mm) and 3 (6.55 ± 3.19 mm). The biggest

8 difference in *sdPA* between the autism and neurotypical groups occurred in segment 2 ($p <$

9 0.001; Autism: 17.32 ± 8.97 mm; Neurotypical: 11.45 ± 7.25 mm).

10   A significant main effect of segment [$F(1.19, 59.27) = 28.65$ $p < 0.001$, $\eta_p^2 = 0.364$

11 BF = 6.967 x10^8] indicated *sdPV* was greater in segment 1 (9.55 ± 4.10 mm) compared to

12 segment 2 (7.57 ± 2.96 mm) ($p = 0.006$], and even greater still in segment 3 (19.27 ± 14.56

13 mm) (*ps* < 0.001). Unlike *sdPA,* there was no significant main effect of group [$F(1, 50) =$

14 0.54, $p = 0.82$, $\eta_p^2 = 0.001$, BF = 0.18] or group x segment interaction [$F(2, 100) = 0.97$, $p =$

15 0.38, $\eta_p^2 = 0.019$, BF = 0.17]. *sdPV* did not differ between the autism and neurotypical

16 groups across the 3 segments.

17   The correlation analyses between the *percentage change* (%Δ) in *total* error from

18 early to late acquisition and *total error* scores in the retention test, indicated significant

19 relationships for the autism ($r = 0.4$, $p = 0.04$; Fig. 6a) and neurotypical ($r = 0.6$, $p = 0.002$;

20 Fig. 6b) groups. As illustrated in Figures 6a and 6b, participants who demonstrated the

21 highest (or lowest) magnitude of sensorimotor adaptation across the acquisition phases (see

22 *X* axis) exhibited the lowest (or highest) *total error* (see *Y* axis) when performing the 3-

23 segment movement sequence in the retention test.

24

25   Insert Figure 6 around here.

26

27 **Discussion**

16

1   We quantified sensorimotor learning of a visuomotor sequence timing task (VSTT)

2   that required a self-selected relative timing pattern (Heuer & Schmidt, 1988; Schmidt, 1985)

3   to be performed in order to achieve an experimenter-imposed overall timing goal (see Hayes

4   et al., 2018). In addition to using measures of overall temporal accuracy and variability (i.e.,

5   total error) and relative timing of the individual movement segments, we examined specific

6   kinematic variables (Khan et al., 2006) that reflect the underlying sensorimotor control

7   processes (Wolpert et al., 1995). We found that the autism and neurotypical groups

8   significantly reduced Total Error when executing the VSTT as a function of trial-and-error

9   learning. Additional analyses demonstrated a relationship between performance accuracy

10   (total movement error) in the retention test, and adaptation across the acquisition phase.

11   The implication is that these sensorimotor adaptation effects are in part based on the

12   processing of knowledge-of-results feedback provided on each trial (Bilodeau, Bilodeau, &

13   Schumsky, 1959). While both groups showed comparable magnitudes of adaptation (autism

14   = 61 %Δ; neurotypical = 66 %Δ), the autism group exhibited slower movements across

15   acquisition (by 289 ms) and retention (by 312 ms). These Total Error effects are consistent

16   with previous work (Hayes et al., 2018) that examined sensorimotor learning in autism using

17   the same VSTT.

18   Analyses of relative timing indicated that both groups executed the movement

19   sequence with comparable timing structures in segments 1 and 2. Although the timing

20   structure for segment 3 was proportionally longer (by 1.7 units; which equals 476 ms in

21   movement time, see Table 2) for the autism group in acquisition, it was comparable in

22   retention. In general, this indicates the sensorimotor processes underlying the acquisition of

23   a self-selected (Heuer & Schmidt, 1988) relative timing structure is comparable to the

24   neurotypical group. The additional movement time (476 ms) exhibited in segment 3 could be

25   related to the specific task demands. For instance, the amplitude of this segment is twice as

26   large as segments 1 and 2, and required volunteers to visually guide the cursor through the

27   central sequence target in order to physically end the movement by stopping the cursor

1   accurately in the final target. The additional planning (increased force requirements for larger

2   amplitude movement; see Schmidt et al., 1979) and accuracy constraints could have

3   differentially impacted upon the noisier autistic sensorimotor system (Glazebrook et al.,

4   2006), and ineffective movement planning processes (Glazebrook, Gonzalez, Hansen, &

5   Elliott, 2009; Rinehart, Bellgrove, et al., 2006), such that motor behaviour was slower and

6   more variable (see Figure 4), in segment 3.

7   Kinematic analysis indicated the autism group exhibited greater spatial variability at

8   peak acceleration (*sdPA)*, but comparable spatial variability at peak velocity (*sdPV)*. These

9   findings suggest the differences observed in total error (accuracy and variability), as well as

10 relative timing, are underpinned by the efficacy of the sensorimotor control processes

11 associated with planning and feedforward control, but not the use of visual online feedback.

12 During goal-directed aiming, as in the VSTT, an initial sensorimotor plan is formed from an

13 inverse model (Wolpert & Kawato, 1998) that receives input regarding state-estimation and

14 *prior* experience (past learning). Once generated, the sensorimotor plan forms motor

15 commands that drive motor execution, from which an efference copy (Von Holst, 1954) is

16 formed for early feedforward motor corrections before sensorimotor feedback is processed.

17 Additionally a forward model is also created for predicting the expected sensorimotor

18 consequences needed for controlling movements (Desmurget & Grafton, 2000; Wolpert et

19 al., 1995; Wolpert & Kawato, 1998). During the initial stage of motor execution, *sdPA* reflects

20 the efficacy in processing activity associated with the specification of muscular forces

21 required to initiate limb movement, and the subsequent modification of force output via

22 feedforward control (Elliott et al., 2010). During this stage, (predicted) expected sensory

23 consequences and the sensory consequences (i.e., reafference) that are generated from the

24 motor command (Desmurget & Grafton, 2000) are compared, with any discrepancy forming

25 the basis of sensorimotor adjustments. We suggest that greater *sdPA* in the autism group is

26 related to ineffective sensorimotor planning based on state-estimation, specification of

27 muscular forces, inverse model development, and/or predictive feedforward control. This is

1   consistent with data also showing sensorimotor planning inefficiencies and feedforward

2   control in upper-limb manual aiming (Glazebrook et al., 2006) and finger force production

3   (Mosconi et al., 2015; Schmitz et al., 2003).

4   As well as replicating the aforementioned effects during sensorimotor learning, our

5   findings are novel in the respect that the autism group adapt the magnitude of *sdPA* via a

6   short period of practice. Previous data from tactile sensory perception protocols showing that

7   autistic volunteers perceive the numbness illusion (Guerra et al., 2017), and attenuate their

8   rating of tickliness (Blakemore et al., 2006) during self-produced movements, suggests that

9   the feedforward predictive mechanism functions typically. Our adaptation effect indicates

10   that sensorimotor planning processes and/or feedforward control, which although generally

11   is less effective in autism, are receptive to sensorimotor training. In addition, while there was

12   no significant difference in variability between the autism and neurotypical groups at *sdPV*,

13   both groups significantly reduced variability at this kinematic landmark as a function of

14   practice. This adaptation effect is indicative of functional sensorimotor control based on

15   reducing the difference between the perceived sensory consequences (visual and

16   proprioceptive feedback) of the executed action, and the expected sensorimotor

17   consequences specified by the forward model (Elliott et al., 2010). Moreover, visual

18   feedback-based control processes that operate to minimise the difference between actual

19   and intended limb position as the movement trajectory unfolds would also have contributed

20   to the reduction in variability at *sdPV* (Elliott et al., 2010; Mosconi et al., 2015; Saunders &

21   Knill, 2005). This feedback-based processing adaptation might have been engaged to help

22   offset the planning issues related to the specification of muscular forces in autism.

23   Whilst we do not report neurobehavioral data, one can speculate that the specificity

24   in feedforward and feedback based differences might (in part) be related to the cerebellum

25   and basal ganglia (Doyon et al., 2009; Shadmehr & Krakauer, 2008). fMRI data (Mostofsky

26   et al., 2009) collected whilst executing a motor control task (i.e., the PANESS task) showed

27   that autistic individuals exhibited decreased cerebellar activity, and increased pre-motor

1   cortex activity, compared to controls (see also Wang et al., 2019). This is perhaps not

2   surprising given the well-reported structural (e.g., lower Purkinje cell count, Ritvo et al.,

3   1986; hypoplasia, Courchesne, Yeung-Courchesne, Hesselink, & Jernigan, 1988) and

4   functional (e.g., greater spatial extent and magnitude of activation in ipsilateral anterior

5   cerebellum; Allen, Muller, & Courchesne, 2004) differences found in the autistic cerebellum

6   (see Amaral, Schumann & Nordahl, 2008; Oldehinkel et al., 2019). Similarly,

7   neurobehavioral correlation findings have indicated that structural differences (i.e., surface

8   deformation of the right posterior putamen) in the basal ganglia predict poorer motor skill

9   performance in autistic individuals (Qiu et al., 2010). Structural differences between the

10   autistic and neurotypical cerebellum could have impacted upon motor control (i.e., timing;

11   coordination) and supervised learning, with a particular emphasis on predicting the

12   sensorimotor consequences of an intended motor command based on the outcome of error

13   signal encoding during learning (Doya, 2000). Likewise, a difference in the basal ganglia

14   could influence reinforcement-based learning (e.g., external KR in the present study), where

15   processes evaluate the cost/benefit of executing an intended motor command in relation to

16   achieving the intended motor goal (Shadmehr & Krakauer, 2008).

17   Across a number of studies from our research group, we have shown autistic

18   individuals exhibit sensorimotor learning in a VSTT (see also Hayes et al., 2018), as well as

19   imitation learning of the temporal characteristics of the modelled movement (Hayes et al.,

20   2016). The fact that learning occurred in both protocols supports and extends an associative

21   framework (Heyes, 2001) perspective of imitation, where the underlying perception-action

22   processes that control imitation are intact in autism (Bird, Leighton, Press, & Heyes, 2007;

23   Sowden et al., 2016), and are modulated by sensorimotor experience (Heyes, Bird,

24   Johnson, & Haggard, 2005). Importantly, however, in both sensorimotor and imitation

25   learning, we have found movement planning and execution differences between autistic and

26   neurotypical controls. Others have also reported autism specific movement (i.e., increased

27   variability in jerk) characteristics (Cook, 2016; Cook, Blakemore, & Press, 2013), which

1   seem to influence the visual perception of observed actions (Brewer et al., 2016; Cook,

2   Blakemore, & Press, 2013; Edey, Cook, Brewer, Johnson, Bird & Press, 2016). The

3   implication is that a difference in autistic motor control could influence part of a predictive

4   system that underpins social interaction in autism.

5   In summary, although we found evidence of intact sensorimotor learning of a novel

6   VSTT in autism, the executed movements were slower. The autism group also exhibited

7   greater *sdPA* in each movement segment, which indicated less effective sensorimotor

8   control processes. Importantly, however, the magnitude of *sdPA* was reduced across

9   acquisition, indicating that these sensorimotor control processes were adapted via trial-to-

10   trial sensorimotor learning. Moreover, *sdPV* in the autism group was comparable to the

11   neurotypical control group, and showed a similar degree of adaptation across learning. The

12   implication is that visual feedback-based sensorimotor control processes are intact in

13   autism. Understanding the differential roles that feedforward and feedback-based control

14   processes play during sensorimotor learning will offer an opportunity to explore how similar

15   control processes influence socio-motor actions in autism.

16

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1   Tables

2   Table 1. Characteristics of autism and neurotypical participants.

	Autism ($n = 26$)		Neurotypical ($n = 26$)		t test p value
	Mean (SD)	Range	Mean (SD)	Range	
Chronological age in years	25 (7)	18-44	25 (7)	18-45	$p = 0.845$
Full scale IQ	107 (9)	91-125	109 (8)	94-123	$p = 0.396$
Verbal IQ	106 (11)	88-130	109 (8)	96-125	$p = 0.214$
Performance IQ	106 (11)	82-128	107 (12)	82-128	$p = 0.891$
Gender	25M : 1F		25M : 1F		

3

4

1 Table 2. Mean (SD) movement time (ms), relative timing (%), sdPA (mm) and sdPV (mm)

2 data presented as a function of group and phase.

		Autism			Neurotypical		
		Segment 1	Segment 2	Segment 3	Segment 1	Segment 2	Segment 3
Dependent Variable	Phase	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Movement Time (ms)	Early	775 (155)	887 (210)	1273 (364)	707 (101)	784 (159)	1018 (195)
	Late	650 (108)	620 (107)	875 (182)	598 (76)	588 (58)	794 (143)
	Retention	671 (128)	665 (105)	958 (250)	606 (70)	591 (59)	794 (122)
Relative Timing (%)	Early	27 (3)	30 (3)	43 (4)	29 (3)	31 (3)	40 (3)
	Late	30 (2)	29 (3)	41 (3)	30 (2)	30 (2)	40 (3)
	Retention	30 (3)	29 (3)	41 (4)	30 (2)	30 (2)	40 (3)
sdPA (mm)	Early	7.10 (6.08)	21.88 (8.65)	5.79 (3.37)	4.76 (2.92)	20.29 (8.41)	5.84 (2.31)
	Late	5.30 (3.32)	14.27 (8.13)	7.26 (4.66)	3.60 (2.44)	12.31 (8.14)	5.91 (2.57)
	Retention	4.13 (2.27)	17.32 (8.97)	6.52 (3.35)	3.57 (2.19)	11.45 (7.25)	6.58 (3.19)
sdPV (mm)	Early	12.44 (3.97)	8.88 (4.17)	24.13 (18.48)	12.01 (4.09)	8.07 (2.89)	20.72 (14.51)
	Late	10.06 (5.42)	7.66 (3.04)	21.29 (15.05)	8.60 (4.48)	6.73 (2.46)	18.79 (13.48)
	Retention	10.47 (4.53)	7.47 (3.09)	17.90 (14.19)	8.62 (3.46)	7.67 (2.89)	20.65 (15.07)

3

4

1   Figure Legends

2   Figure 1. A schematic representation of the movement sequence timing task that has a

3   timing goal of 1700 ms. The sequence was presented as three green targets (diameter = 12

4   mm) and is depicted by the arrows in Segment 1 (start target to centre target), Segment 2

5   (centre target to start target), and Segment 3 (start target to end target). The target positions

6   had an equidistant extent of 100 mm between the centre of each target. The white circle

7   depicts the cursor (diameter = 6 mm) and represents the motion of the hand-held stylus

8   drawn on the monitor. Feedback on the CRT monitor represents knowledge-of-results
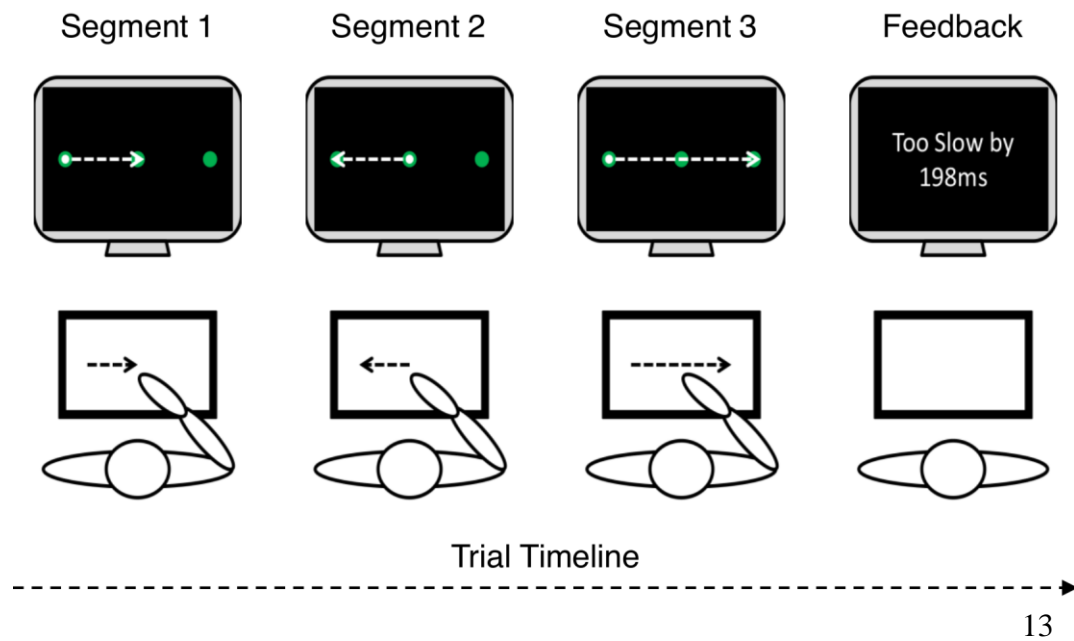
9   provided to the participant in ms.

10

11   Figure 2. Mean total error as function of group and phase. Error bars represent standard
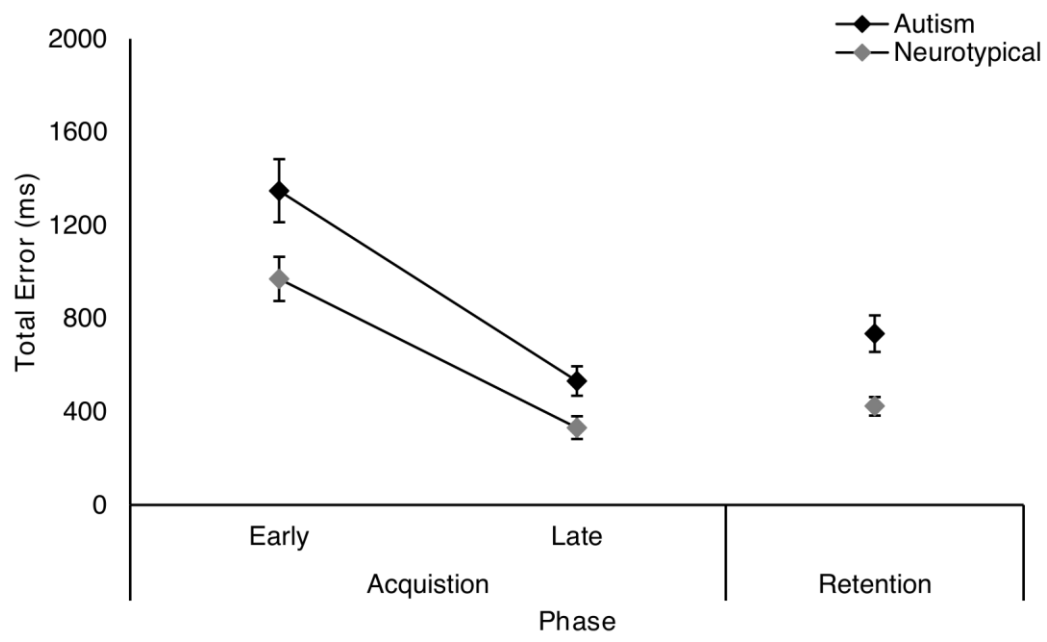
12   error of the mean.

13

14   Figure 3. Relative timing as a function of group, segment and phase. Error bars represent

15   standard error of the mean.

16

17   Figure 4. Normalised spatio-temporal movement trajectories and standard deviation (shaded

18   areas) for the autism (red line) and neurotypical (blue line) groups in the x- and y- axis in

19   early (x-: A; y-: B), late (x-: C; y-: D), and retention (x-: E; y-: F). All experimental trials from

20   the early, late and retention phases were resampled to 150 time points. Participant mean

21   positions were calculated for each time point, which were then averaged across groups to

22   create normalised group mean movement trajectories. In segment 1, participants moved

23   from the start target (x- position: -100mm) to the centre target (x- position: 0mm). Segment 2

24   consisted of a reversal from the centre target back to the start target. Segment 3 consisted

25   of a second reversal from the start target to the end target (x- position: 100mm).
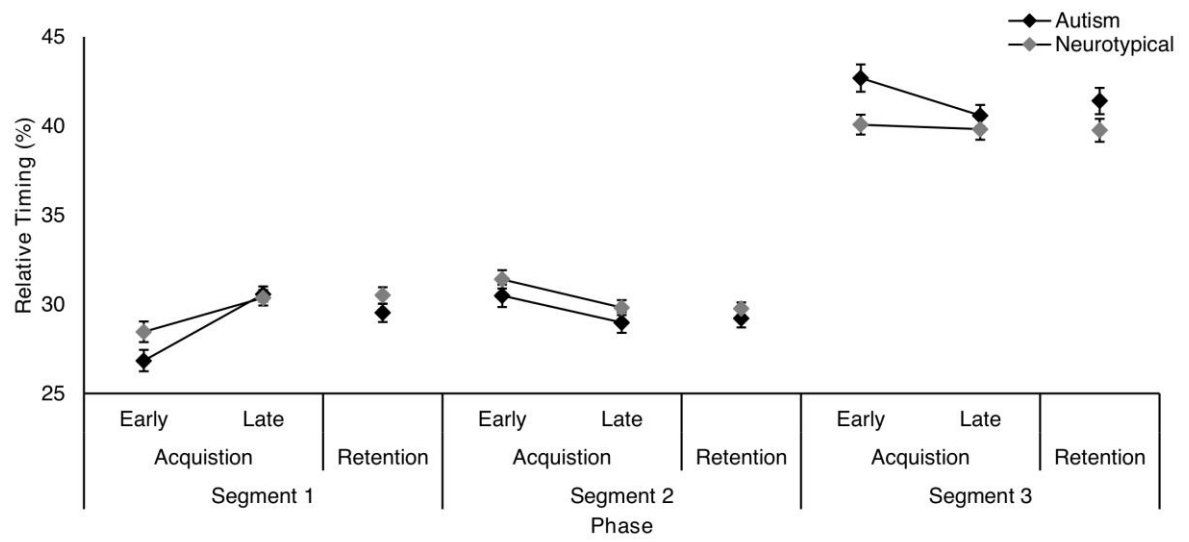
1 Figure 5. (A) Mean spatial variability at peak acceleration as a function of group, segment

2 and phase. (B) Mean spatial variability at peak velocity as a function of group, segment and

3 phase. Error bars represent standard error of the mean.

4

5 Figure 6. Relationship between percentage change from early to late acquisition and total

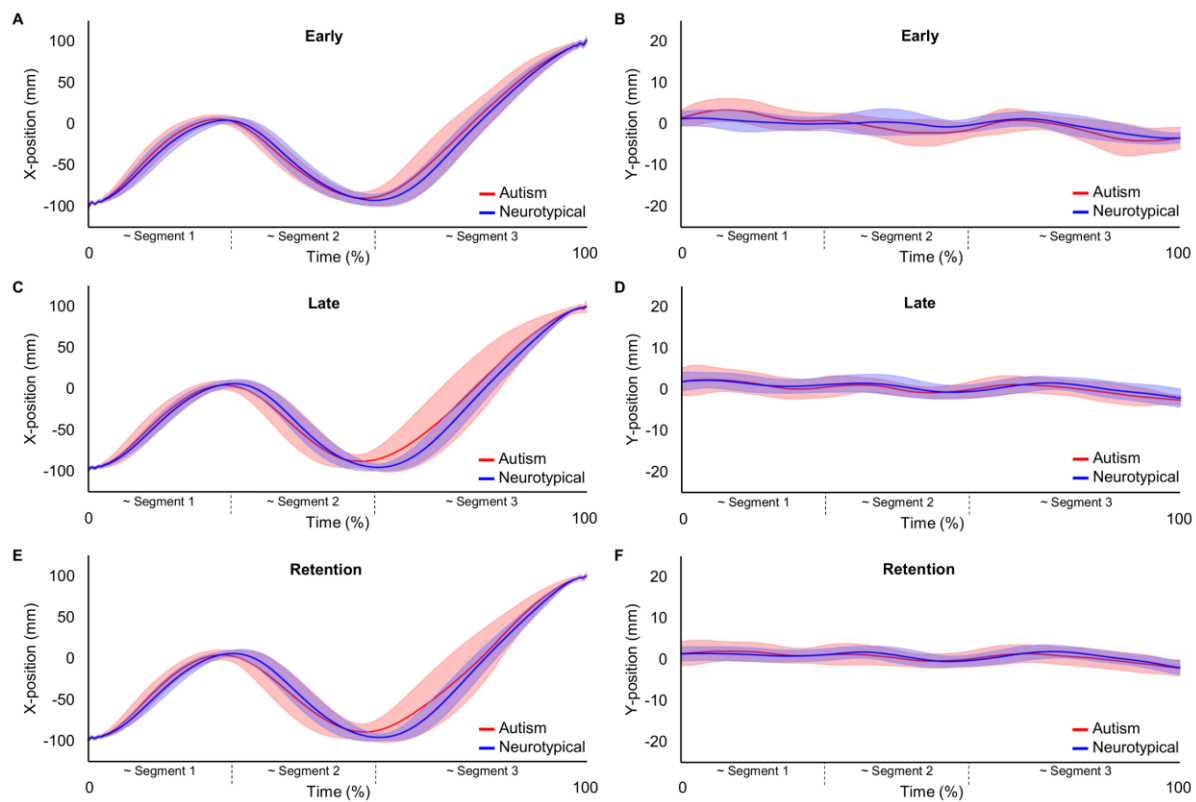6 error in the retention test for both the autism (A) and neurotypical (B) groups.

7
8

Segment 1   Segment 2   Segment 3   Feedback

Too Slow by
198ms

Trial Timeline

13

1
2
3

A

B

1