Atypical biological motion kinematics are represented by complementary lower-level and top-down processes during imitation learning

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ABSTRACT

Learning a novel movement requires a new set of kinematics to be represented by the sensorimotor system. This is often accomplished through imitation learning where lower-level sensorimotor processes are suggested to represent the biological motion kinematics associated with an observed movement. Top-down factors have the potential to influence this process based on the social context, attention and salience, and the goal of the movement. In order to further examine the potential interaction between lower-level and top-down processes in imitation learning, the aim of this study was to systematically control the mediating effects during an imitation of biological motion protocol. In this protocol, we used non-human agent models that displayed different novel atypical biological motion kinematics, as well as a control model that displayed constant velocity. Importantly the three models had the same movement amplitude and movement time. Also, the motion kinematics were displayed in the presence, or absence, of end-state-targets. Kinematic analyses showed atypical biological motion kinematics were imitated, and that this performance was different from the constant velocity control condition. Although the imitation of atypical biological motion kinematics was not modulated by the end-state-targets, movement time was more accurate in the absence, compared to the presence, of an end-state-target. The fact that end-state targets modulated movement time accuracy, but not biological motion kinematics, indicates imitation learning involves top-down attentional, and lower-level sensorimotor systems, which operate as complementary processes mediated by the environmental context.

Keywords: Imitation; Biological motion kinematics; Lower-level processes; Top-down attentional modulation

PsycINFO classification: 2300, 2323, 2330

1. Introduction

Imitation is a powerful mechanism that supports human interaction. In familiar social settings, imitation involves the automatic activation of a motor response triggered by observing a similar motor action (Chartrand & Bargh, 1999; Heyes, 2001, 2011; Heyes, Bird, Johnson, & Haggard, 2005). For example, individuals execute faster pre-specified movements (e.g., finger tapping) when observing biologically compatible (finger tapping), compared to incompatible (finger lifting), movements (Brass, Bekkering, & Prinz, 2001; Stürmer, Aschersleben, & Prinz, 2000). The shorter motor reaction times occur independent of task instructions, which suggests involvement of automatic sensorimotor processes linking perception and action (Brass & Heyes, 2005; Prinz, 1997).

To understand if the automatic sensorimotor effects are developed through experience, and linked to a general mechanism incorporating processes associated with perception, action and attention (Leighton, Bird, & Heyes, 2010), studies have examined automatic imitation following correlated sensorimotor training (Bird, Brindley, Leighton, & Heyes, 2007; Catmur, Mars, Rushworth, & Heyes, 2011; Catmur, Walsh, & Heyes, 2007, 2009; Cavallo, Heyes, Becchio, Bird, & Catmur, 2013; Heyes, et al., 2005). For example, individuals performed a countermirror protocol that required compatible or incompatible sensorimotor training (Catmur, et al., 2007). During compatible training, individuals executed index-finger movements, whilst simultaneously observing index-finger movements. During incompatible training, individuals executed index-finger movements, whilst simultaneously observing little-finger movements. After incompatible training, TMS-induced motor evoked potentials recorded from the little finger abductor muscle were greater during observation of index-finger movement compared to a little-finger movement. These findings demonstrate the sensorimotor system was reconfigured during correlated sensorimotor training, and thus indicate imitation is associated with a general mechanism involving lower-level visuomotor processes that represent biological motion, as opposed to a specialised mechanism that mediates (Meltzoff & Moore, 1997) the translation of visual information into a motor action.

Of primary interest to the present study is the suggestion that similar sensorimotor processes operate during automatic imitation and imitation learning (Brass & Heyes, 2005; Buccino et al., 2004; Heyes, 2011; Iacoboni, 2009). Like the countermirror principle, imitation learning often requires the sensorimotor system to represent a novel biological motion across consecutive imitation trials. Although there is strong evidence that biological motion is processed during automatic imitation (Brass, Bekkering, Wohlschlaeger, & Prinz, 2000; Heyes, et al., 2005; Press & Heyes, 2008) and interpersonal observation-execution imitation tasks (Kilner, Paulignan, & Blakemore, 2003), support from imitation learning studies has typically been based on protocols that manipulated the speed of the imitated movement (Bisio, Stucchi, Jacono, Fadiga, & Pozzo, 2010; Hayes, Timmis, & Bennett, 2009; Wild, Poliakoff, Jerrison, & Gowen, 2010).

Although participants have been shown (Wild, et al., 2010) to imitate different movement speeds (e.g., slow, medium, and fast upper-limb aiming movements), it is notable that the observed stimulus was representative of typical aiming movements. Thus, it remains possible that imitation was limited to recognizing differences in movement speed between observations, as opposed to representing the underlying biological motion kinematics. In this case, the feedforward contribution to motor execution could have been associated with an individual recruiting and rescaling a preexisting motor representation of a familiar and meaningful aiming movement (Hayes, Roberts, Elliott, & Bennett, 2014; Hayes, et al., 2009). This would imply imitation was based on higher-order semantic processes (Rumiati, Papeo, & Corradi-Dell'Acqua, 2010; Rumiati et al., 2005), as opposed to lower-level sensorimotor processes representing the observed biological kinematics.

In the current study, we adopted a novel protocol that enabled us to directly examine biological motion processing during imitation learning. In addition to displaying a constant velocity control model, we manipulated the structure of two experimental models so that peak velocity in the aiming movements no longer occurred at the typical mid-point (40-60% of the total time) of the trajectory (Elliott, Helsen, & Chua, 2001). With such stimuli, imitation can be quantified according to timing and magnitude of velocity, which in combination would

not reflect the kinematics of typical aiming movements (Hayes, et al., 2014). Imitation in this context is not solved by merely recruiting an existing sensorimotor representation associated with a typical upper-limb aiming movement and rescaling (Schmidt, 1975) the representation to meet the goal movement time of 1700 ms. Instead, because the novel atypical biological motion profiles are unlikely to be represented in the sensorimotor repertoire of our participants (Hayes, et al., 2014), imitation requires the specific velocity profile to be represented. Following this logic, we compared imitation learning of two different biological motion models, in which percentage-time-to-peak-velocity occurred at 17% or 26% of the total movement time (henceforth atypical17 and atypical26), and thus earlier than normally expected when aiming to a target. By maintaining equal movement time and amplitude, magnitude of peak velocity also differed between the biological motion models (atypical 17 = 0.37 mm/ms; atypical 26 = 0.24 mm/ms). Finally, given that the lower-level processes that code biological motion kinematics are modulated by various top-down processes (Bekkering, Wohlschlaeger, & Gattis, 2000; Heyes & Bird, 2007; Leighton, et al., 2010; Rumiati, et al., 2005; Southgate & Hamilton, 2008; Wang & Hamilton, 2012), we displayed motion stimuli as a non-human agent (a white dot) to control social context, and in the presence or absence of end-state-targets. The latter manipulation is important because previous work (Hayes, Hodges, Huys, & Williams, 2007; Wild, et al., 2010) has shown that the imitation of biological motion is attenuated in the presence of an end-state-target. In this context, the end-target provides a salient task-relevant (Leighton, et al., 2010) environmental visual cue that modulates attention so that this feature (target attainment) is prioritized and represented during imitation. The removal of end-state-targets in half of the present experimental trials enabled us to develop a protocol that examined biological motion kinematics during true imitation (Cook & Bird, 2012; Vivanti & Hamilton, 2014).

With a behaviorally realizable but atypical biological motion (i.e., atypical17; atypical26), represented as a non-human agent, it was expected that participants would imitate in accord with the observed biological kinematics (Hayes, et al., 2014) and thus produce movements scaled to both timing and magnitude of peak velocity. Because of the

constraints on human movement imposed by the neuro-muscular system (Abend, Bizzi, & Morasso, 1982), we did not expect participants to move with constant velocity having observed the constant velocity stimulus, or to execute a kinematic profile that resembled the atypical motion kinematics. Rather, we anticipated participants would recruit a pre-existing motor response and thus exhibit time of peak velocity that was similar to typical aiming movements. Finally, it was anticipated that imitation of atypical biological motion would be more accurate in the absence, compared to presence, of end-state-targets. In the absence of end-state targets, there should be minimal contribution from top-down attentional processes, thus encouraging participants to focus on representing the characteristics of lower-level visual stimuli during imitation learning.

2. Materials and methods

2.1. Participants

Data were recorded from twenty participants (aged range 18 - 21 years) who volunteered for the study. All participants had normal or corrected-to-normal vision and gave written informed consent. The experiment was designed in accordance with the Declaration of Helsinki and was approved by the ethics committee of the host University.

2.2. Apparatus and Procedures

The apparatus consisted of a PC (Dell Optiplex GX280), a 21-in CRT computer monitor (IIyama Vision Master 505), and a graphics tablet with a hand-held stylus (WACOM Intuos 3). The CRT monitor operated with a spatial resolution of 1280 x 1024, and a refresh rate of 85 Hz. Visual stimuli was generated via MATLAB (The Mathworks, Inc), using Cogent 2000 toolbox (www.vislab.ucl.ac.uk/cogent.php).

Participants were required to observe and imitate the movement of a model (a white cursor, diameter = 8mm) presented on the 21-inch CRT monitor. The model displayed a single horizontal trajectory that originated from a home-target positioned on the left-hand side of the screen. The amplitude of the movement was 200 mm, with a movement time of

1700 ms, and ended on the right-hand side of the monitor. For the end-state-target condition, two red circles representing home-target and the end-state-target (diameter = 16 mm) were positioned at center-left (home) and center-right (end-state) of the monitor (Figure 1A). To examine imitation of biological motion, three models were created: atypical (atypical17; atypical26) or constant velocity (Figure 2). The atypical models displayed a velocity profile that was positively skewed so that peak occurred at 17% or 26% of movement time, and with a magnitude of 0.37 mm/ms and 0.24 mm/ms, respectively. The models were created by a human volunteer who practiced the two atypical goal-directed aiming movements using a hand-held stylus on a graphics tablet until a white cursor, which represented the stylus, moved from a left-hand home-target to a right-hand end-state-target in a movement time of 1700 ms. The displacement time-series data recorded from a successful practice trial for each model was selected to create the models. The method of using a human to generate the models was critical because it ensured the kinematics of the movement was biological in origin, and thus the movement was achievable. The model displaying constant velocity was created according to the amplitude (200 mm) and time (1700 ms) constraints associated with the task. The model displayed the exact movement time, but with a constant velocity trajectory that had no deviations in the perpendicular axis.

Prior to the experimental trials, all participants completed a familiarization period that replicated the conditions of the imitation task. Participants sat on a chair in front of a CRT monitor and held the stylus in their preferred hand. The participants performed four familiarization trials; 2 trials representing the end-state-target condition (see Figure 1A) performed in the imitation task, and 2 trials representing the no-end-state-target condition (see Figure 1B) performed in the imitation task. Each trial commenced with the model being positioned in the center of the home-target. The participants observed the model display a movement from the home-target to an end-target (end-state-target condition), or end space (no-end-state-target condition), with a constant velocity trajectory and a movement time of 1700 ms. A constant velocity trajectory was used to ensure construct validity by preventing participants from experiencing biological motion before the imitation trials. Participants were

not informed about the agency of the model or duration of the movement time. Following observation of the model, participants moved the cursor from the center of the monitor to the center of the home-target, and clicked the lower-button on the stylus. In an end-state-target condition, the two targets remained on the screen as the participant imitated the model. In a no-end-state-target condition, the two targets were removed before a participant imitated the model. To finish imitation, participants clicked the lower-button on the stylus a second time once the cursor was located in the end-state-target, or end-space in the no-end-state-target condition. After familiarization, all participants confirmed they understood the model, the end-state-target and no-end-state-target conditions, the instruction to imitate, and the sensorimotor association between the stylus on a graphics tablet, and the corresponding movement of cursor on the monitor.

The imitation task comprised 14 blocks of 6 trials (84 trials). A block contained each of the 6 combinations of target (end-state-target, no-end-state-target) and velocity model (atypical17, atypical26, constant) presented in random order. A trial commenced with an observation phase where the home-target (red) was displayed on the monitor for 1000 ms, before disappearing for 1000 ms, and being replaced by a model positioned in the same location. Depending on the trial type, the model moved to an end-state-target (Figure 1A) or end-space in the no-end-state-target (Figure 1B) condition, with one of three velocity models. After observing the model, participants imitated the movement as per the instructions given in the familiarization period.

2.3. Statistical analysis

To quantify imitation performance, and imitation of atypical biological motion, we extracted movement kinematics exhibited by the participants on each trial. The start of movement was defined as the time the center of the cursor moved beyond the perimeter of the home-target, and the end was calculated when the participant clicked the lower-button on the stylus. For each imitation attempt, the 2-dimensional displacement data were filtered using a low-pass (8 Hz) autoregressive filter. These data were differentiated using a central

difference algorithm to obtain velocity. A MATLAB routine extracted the primary movement occurring in the x-axis and identified the following dependent variables: movement time, peak velocity, and percentage-time-to-peak-velocity (i.e., time to peak velocity / movement time) x 100). The two velocity variables were chosen for analysis because they most reflected the difference between the two atypical biological motion models. Intra-participant means from the 14 trials per condition were calculated for each dependent variable and submitted to separate Model (atypical17; atypical26; constant velocity) x Target (end-state-target; no-end-state-target) repeated measures ANOVAs. Alpha was set at $p < 0.05$, follow-up testing used the Tukey post-hoc procedure, and partial eta squared (η_p^2) expressed the size of the effect.

Insert Figure 1 and 2 about Here

3. Results

3.1. Movement time

As illustrated in Figure 3, the presence of an end-state-target [$F(1, 19) = 36.61$, $p < 0.05$, $\eta_p^2 = 0.49$] modulated movement time, with significantly shorter and more accurate movement times imitated in the absence (M = 2156 ms), compared to the presence (M = 2294 ms), of an end-state-target. Although there was no significant difference in movement times when imitating the atypical17 (M = 2121 ms) and atypical26 (M = 2191 ms) models, the main effect [$F(2, 38) = 17.90$, $p < 0.05$, $\eta_p^2 = 0.66$] indicated these two movement times were significantly shorter (*ps* < 0.05) and more accurate than imitating the constant velocity (M = 2362 ms) model. The interaction concerning model and target [$F(2, 38) = 3.51$, $p < 0.05$, $\eta_p^2 = 0.16$] indicated that significantly shorter and more accurate movement times were performed in the no-end-state-target compared to the end-state target condition (*ps* < 0.05) when viewing atypical17 and atypical26 models. This effect was not significant when imitating constant velocity.

Insert Figure 3 about Here

3.2. Peak velocity

An effect of Model [$F(2, 38) = 59.56$, $p < 0.05$, $\eta_p^2 = 0.76$] indicated the magnitude of peak velocity was significantly greater when imitating the atypical17 model (M = 0.24 mm/ms) compared to the atypical26 (M = 0.19 mm/ms) and constant velocity (M = 0.15 mm/ms) models. Moreover, the magnitude of peak velocity was significantly ($p < 0.05$) greater when imitating the atypical26 compared to the constant velocity model. As illustrated in left-hand and center portions of Figure 4, the magnitude of peak velocity executed by the participants in the atypical17 and atypical26 conditions (grey bars) was scaled (i.e., more similar) to peak velocity displayed by the model (black bar). However, peak velocity was not modulated by the presence or absence of an end-state-target [$F(1, 19) = 1.48$, $p > 0.05$, $\eta_p^2 = 0.07$], irrespective of how it was combined with the model stimulus [$F(2, 38) = 1.54$, $p > 0.05$, $\eta_p^2 = 0.17$].

Insert Figure 4 about Here

3.3. Percentage-of-time-to-peak-velocity

An effect of Model [$F(2, 38) = 68.99$, $p < 0.05$, $\eta_p^2 = 0.78$] indicated peak velocity occurred significantly earlier in the movement when imitating the atypical17 model (M = 22%) compared to both the atypical26 (M = 29%) and constant velocity (M = 38%) models (*ps* < 0.05). As illustrated in Figure 5, the grey bars indicate the temporal occurrence of peak velocity in the atypical17 and atypical26 conditions was scaled to peak velocity displayed by the model (black bar). This effect can also be seen from an exemplar velocity trace in Figure 6. When imitating the atypical17 (dark grey trace) model, peak velocity occurred significantly earlier in the movement than the atypical26 (light grey trace) model. When imitating the

constant velocity model, peak velocity occurred toward the midpoint of the movement (black trace). Although there was no main effect for Target [$F(1, 19) = 1.58$, $p > 0.05$, $\eta_p^2 = 0.08$], there was an interaction concerning Model and Target [$F(2, 38) = 11.40$, $p < 0.05$, $\eta_p^2 = 0.35$]. Percentage-of-time-to-peak-velocity occurred earlier in the movement in the end-state-target condition compared to the no-end-state-target condition when imitating the atypical17 and atypical26 models (*ps* < 0.05). This effect was reversed when imitating constant velocity model.

Insert Figure 5 and 6 about Here

4. Discussion

We examined the representation of biological motion kinematics during imitation learning using a novel protocol that systematically manipulated the structure of a model's kinematic profile. The percentage-time-to-peak-velocity data supported our expectations by indicating peak velocity occurred significantly earlier in the movement after imitating both the atypical17 and atypical26 models. Moreover, while movement time was similar in these conditions, the magnitude of peak velocity also differed in accord with the atypical biological motion models. Imitation of both atypical17 and atypical26 models was confirmed by the data showing participants exhibited peak velocity significantly later (38%) in the movement in the constant velocity control condition. Moreover, and as displayed in Figure 6 (black traces in A and B), the exemplar velocity profile(s) illustrates a relatively flat, and stable, trajectory that contains a number of discontinuities. The fact the velocity profile was not bell-shaped suggests participants attempted to imitate the constant velocity model, rather than recruiting a movement trajectory based on internal (pre-existing motor priors) and external (amplitude and speed of movement) constraints of the task. Moreover, the low peak, and discontinuities could be the result of error minimization using visual feedback (Elliott, et al., 2001), and/or sensorimotor noise associated with anatomical and physiological constraints of the motor system (Abend et al. 1982).

As expected, the findings also showed that imitation learning was modulated by the presence or absence of end-state-targets. Having observed the two atypical biological models in the absence of end-state-targets, participants exhibited shorter movement times, which were more accurate (M = 2156 ms) compared to when end-state-targets were present (M = 2294 ms). As suggested previously (Wild, et al., 2010), this effect was unlikely to be associated with differences in movement amplitude, which was 6 mm shorter when end-state-targets were absent[1]. Neither was it a function of greater average acceleration, which was less in the absence of end-state targets (i.e., similar peak velocity but achieved later). Although not measured in the present experiment, an explanation for the less accurate imitation of movement time in the presence of end-state-targets is that participants paid more attention (Leighton, et al., 2010) to target attainment and thus were more goal-directed during movement execution. As a consequence, it is likely they focused more on aiming to position the cursor in the end-target, which resulted in proportionately more time after peak velocity in the deceleration phase (Elliott, Hansen, Mendoza, & Tremblay, 2004).

The specificity of the aforementioned goal-directed imitation effect is important from a theoretical position because the decrease in movement time accuracy in the end-state-target condition did not lead to a concomitant decrease in the imitation of atypical biological motion kinematics. Also, there was an interaction between the biological nature of observed stimulus (biological motion versus constant velocity) and end-state-target condition. For instance, participants exhibited more accurate movement time in the absence of end-state-targets when observing biological motion but not constant velocity. This effect is somewhat consistent with the suggestion that multiple goals (kinematics; end-state-target-goal), as well as other salient factors in the environment (Leighton, et al., 2010), are represented when imitating different movements (Bekkering, et al., 2000; Hamilton, 2008). Unlike previous work that typically demonstrated an action-goal (to grasp an ear) was prioritized (hierarchal goal representation) at the expense of biological kinematics (Bekkering, et al., 2000; Hamilton, Brindley, & Frith, 2007; Hayes, Hodges, Scott, Horn, & Williams, 2007; Wohlschlager, Gattis, & Bekkering, 2003), we showed the attainment of an end-state-target

goal did not affect the representation of biological kinematics. Our findings build upon the aforementioned effects by indicating top-down and lower-level processes operate within an embedded system that is less hierarchal, and perhaps more complementary (Buxbaum & Kalénine, 2010; de Lange, Spronk, Willems, Toni, & Bekkering, 2008; Heyes, 2011), with the contribution of these processes modulated by the nature of task context. When the biological movement kinematics are novel, as per our atypical biological motion, both processes operate to represent movement kinematics and the end-state-target goal.

To minimize the potential modulation of biological motion processing by top-down factors associated with goal coding (Bekkering, et al., 2000), attention/salience (Leighton, et al., 2010), teleological reasoning (Csibra & Gergely, 2007) and social modulation (Wang & Hamilton, 2012), the atypical biological models were observed as non-human agents in the absence of end-state-targets. The finding of temporal correspondence (Gangitano, Mottaghy, & Pascual-Leone, 2001) between observed (atypical17; atypical26) and imitated movement kinematics is therefore consistent with biological motion being processed through lower-level visuomotor processes operating in the human mirror-mechanism (Brass & Heyes, 2005; Casile et al., 2010; Dayan et al., 2007; Press, Cook, Blakemore, & Kilner, 2011). Detection of biological motion is suggested to occur in a neural substrate associated with the posterior superior temporal sulcus (Allison, Puce, & McCarthy, 2000), while coding the kinematic properties of an observed action (Hamilton, 2008; Iacoboni, 2009) is suggested to occur in the fronto-parietal mirror-system (Di Dio et al., 2013; Press, et al., 2011). Within the fronto-parietal mirror mechanism, the premotor region has been associated with coding the temporal features of visual information through analysis of motor evoked potentials during different phases of a grasping action (Gangitano, et al., 2001). Moreover, evidence that certain phases of movement are reflected in time-synchronized neural activation (e.g., greatest activation during display of maximal grip aperture), has been suggested to indicate online visual processing during observation of biological motion. We concur with this reasoning and suggest the finding of temporal correspondence between the model and imitation of atypical biological motion was in part based on the online visual

processing of such motion during each observation trial. Such findings of continual matching of action-execution with action-observation is consistent with our previous work on biological motion coding during observational practice (Hayes, et al., 2014).

In summary, the findings in the present experiment showed atypical biological motion kinematics was represented during imitation learning, both in the presence and absence of end-state targets. Imitation of biological motion kinematics involves top-down attentional and lower-level visuomotor systems, which operate as complementary processes.

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Footnote

[1]We conducted additional analyses to determine if movement time was correlated with movement amplitude. Separate within-participant correlations were run on these two dependent variables for end-state-target and no-end-state-target conditions. For each participant we ran a correlation on movement time and movement amplitude from 42 trials (i.e., 14 trials and 3 velocity models).The logic is that a positive correlation would occur if longer movement times were associated with longer movement amplitudes, and vice versa. The group mean *r* value for the end-state-target condition was 0.27 ± 0.27, and 0.30 ± 0.2 for the no-end-state-target condition. Furthermore, of the 20 participants, 9 had a significant *r* value in the end-state-target condition, and 12 had a significant *r* value no-end-state-target condition. Only 8 of the participants exhibited a significant *r* value in both the end-state-target condition and no-end-state-target condition. In addition, the mean r^2 for the end-state-target condition was 0.14 ± 0.18 and 0.15 ± 0.14 for the no-end-state-target condition, and the coefficient of determination was less than 0.5 for all participants. These analyses indicate no clear trend across participants for a relationship between movement time and amplitude.

Figure Legend

Fig.1. A visual representation depicting a single trial in the end-state-target-condition (A) and no-end-state-target condition (B). The apparatus outlined in Panel A and B is a CRT monitor and a graphics tablet. The trial timeline arrows at the bottom of the figure indicate the Observation Phase and Imitation Phase. During the Observation Phase, the non-human agent model is positioned in the left-hand home target (A) and left-hand space (B). The model (atypical17 or atypical26 or constant velocity) displays a horizontal movement of 200 mm from the left-hand home target to an end-state-target (A) or end-space in the no-end-state-target-condition. The model has a movement time of 1700 ms. The Imitation Phase commences with the white cursor positioned in left-hand home target (A) or left-hand space (B). A participant imitates the observed model by controlling a stylus on the graphics tablet.

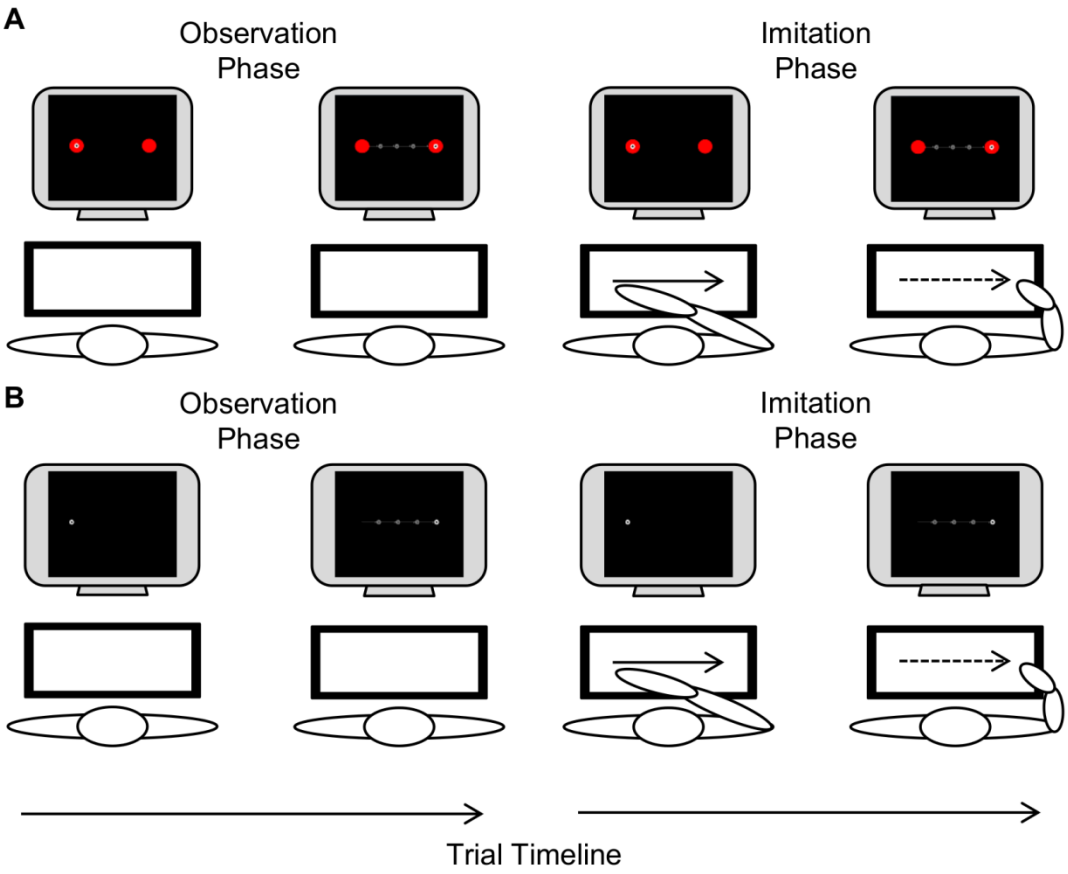
Fig.2. The velocity profiles for atypical17 model (light grey trace; peak), atypical26 model (dark grey trace), and constant velocity control model (black trace).

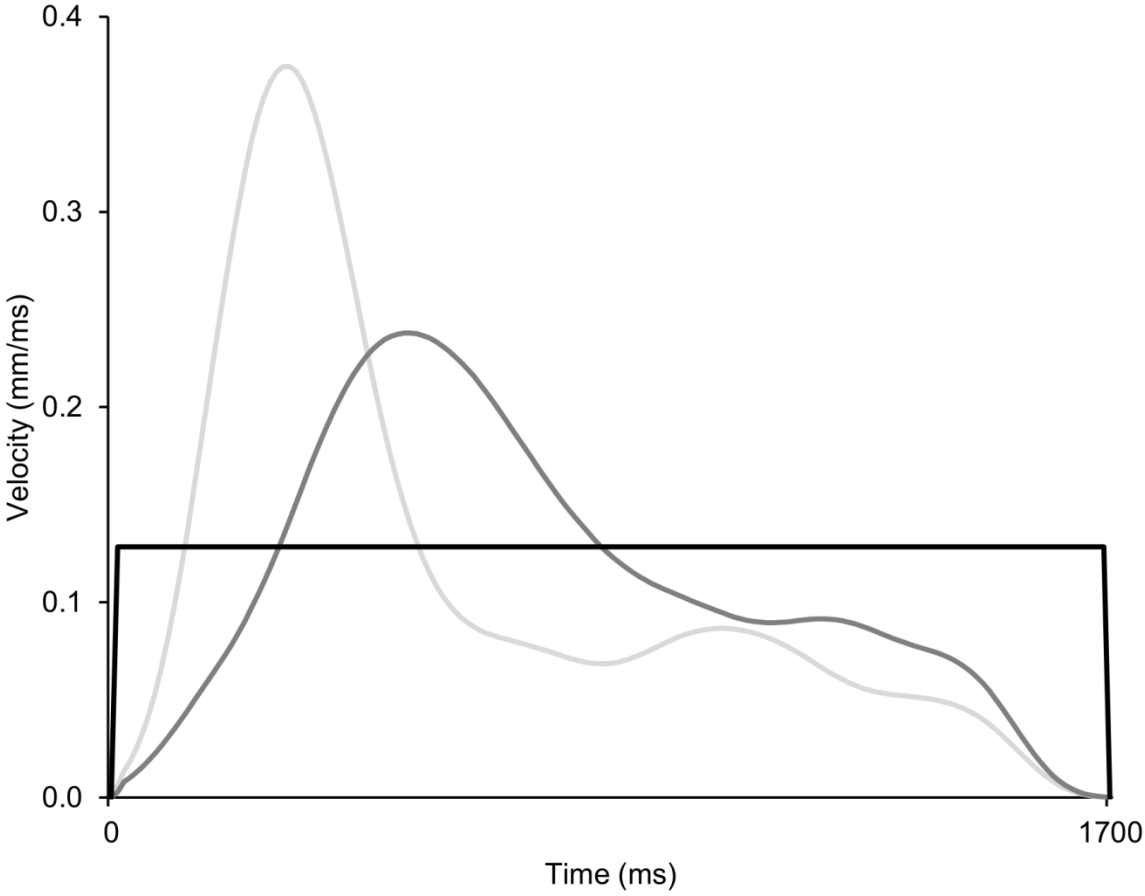
Fig.3. Mean movement time data (ms) as a function of model (atypical17, atypical26 and constant velocity) and target condition (light grey = end-state-target; dark grey bar = no-end-state-target). The criterion model data for atypical17 and atypical26 is represented in the black bars. Error bars (±) display the standard error mean.

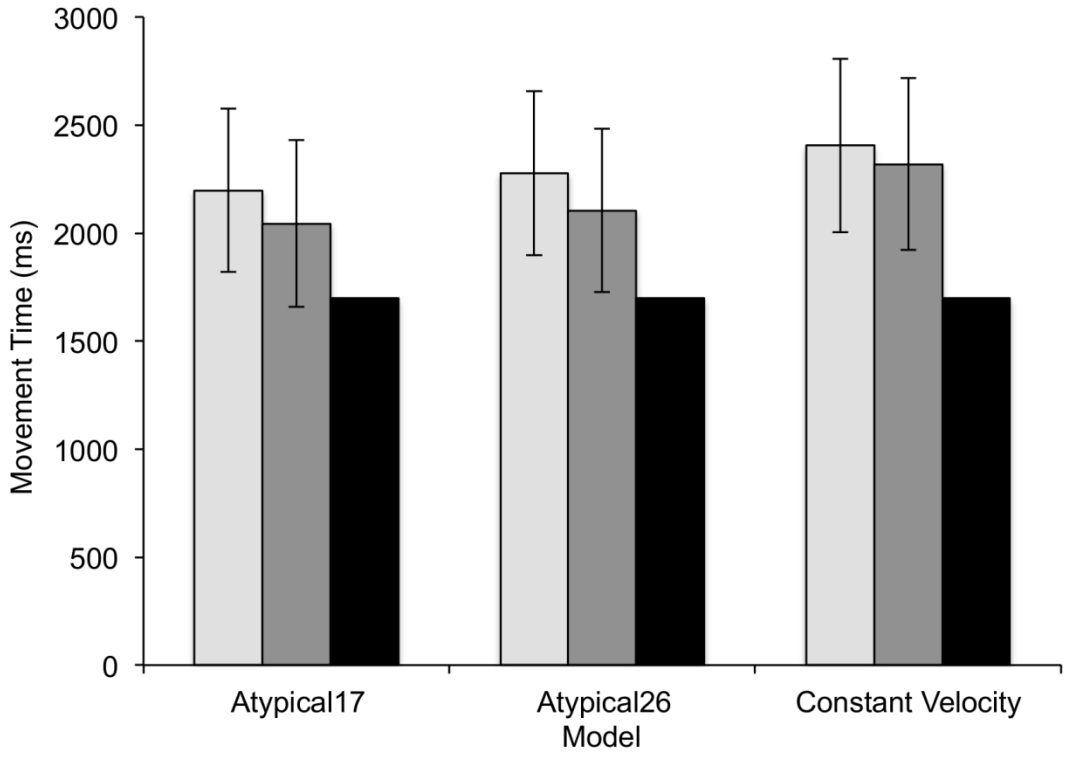
Fig.4. Mean peak velocity data (mm/ms) as a function of model and target condition. The target conditions are displayed in the light grey bar (end-state-target) and dark grey bar (no-end-state-target). The criterion model data for atypical17 and atypical26 is represented in the black bars. Error bars (±) display the standard error mean.

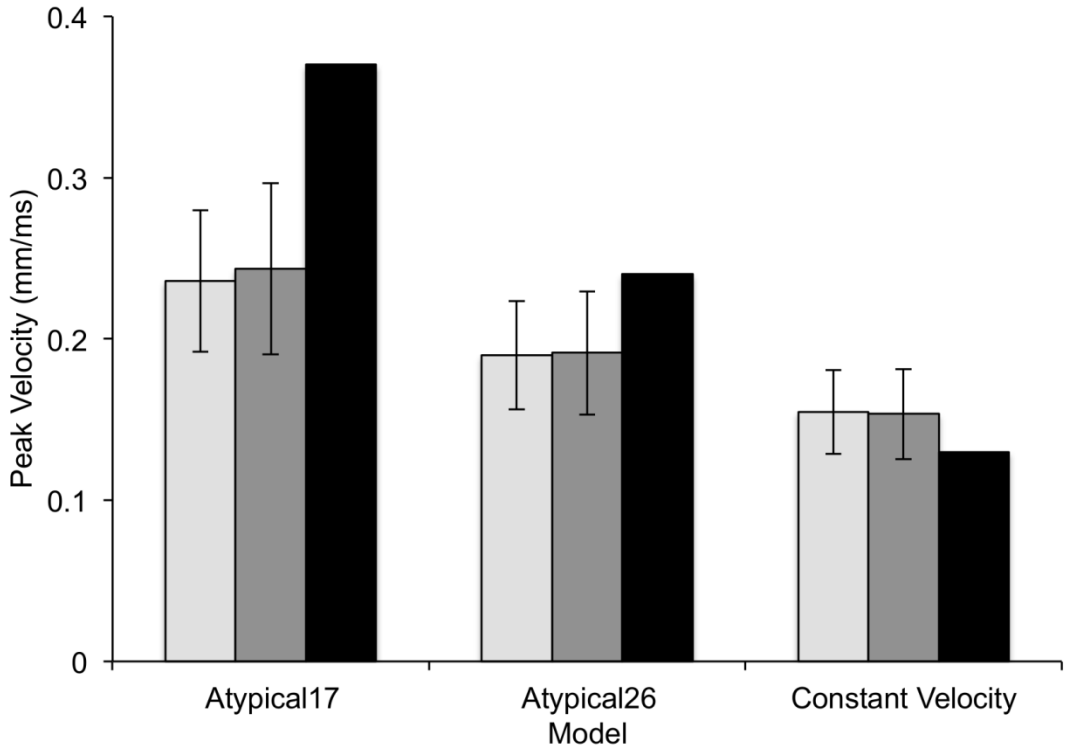
Fig.5. Mean percentage-time-to-peak-velocity (%) as a function of model and target condition. The target conditions are displayed in the light grey bar (end-state-target) and dark grey bar (no-end-state-target). The criterion model data for atypical17 and atypical26 is represented in the black bars. Error bars (±) display the standard error mean.

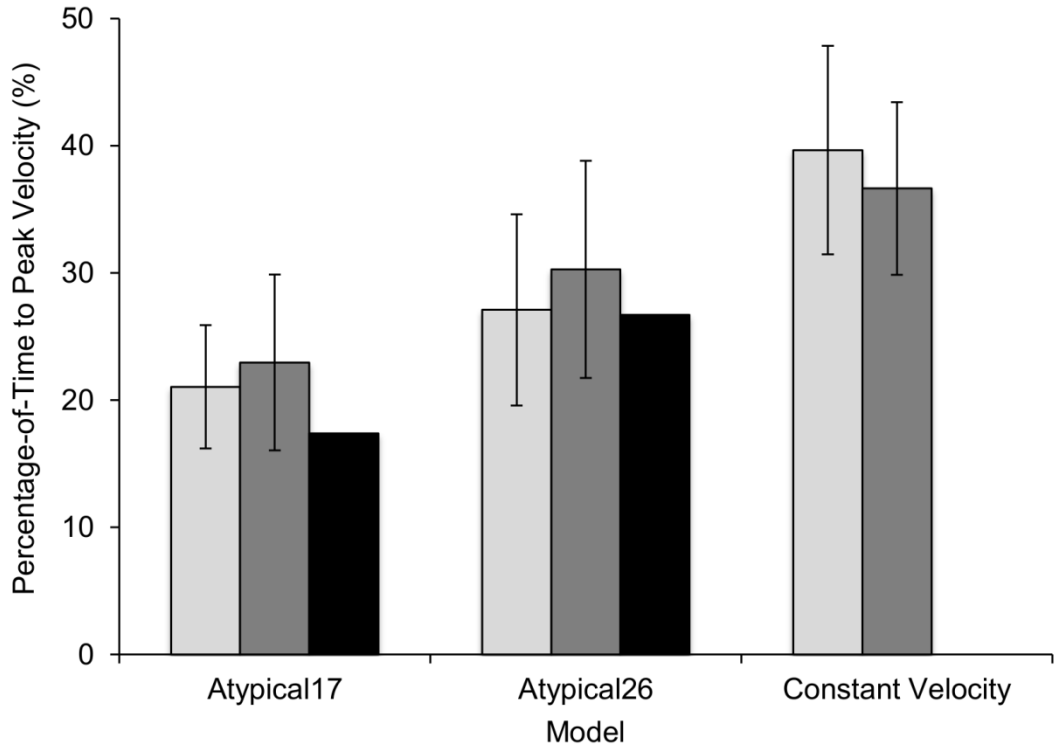
Fig.6. The velocity profiles are exemplar data from a representative participant imitating atypical17 model (light grey trace; peak), atypical26 model (dark grey trace), and the constant velocity control model (black trace) in the no-end-state-target (A) and end-state-target (B) conditions. The 1700 ms marker displayed on the x axis indicates the total movement time displayed by the three models.

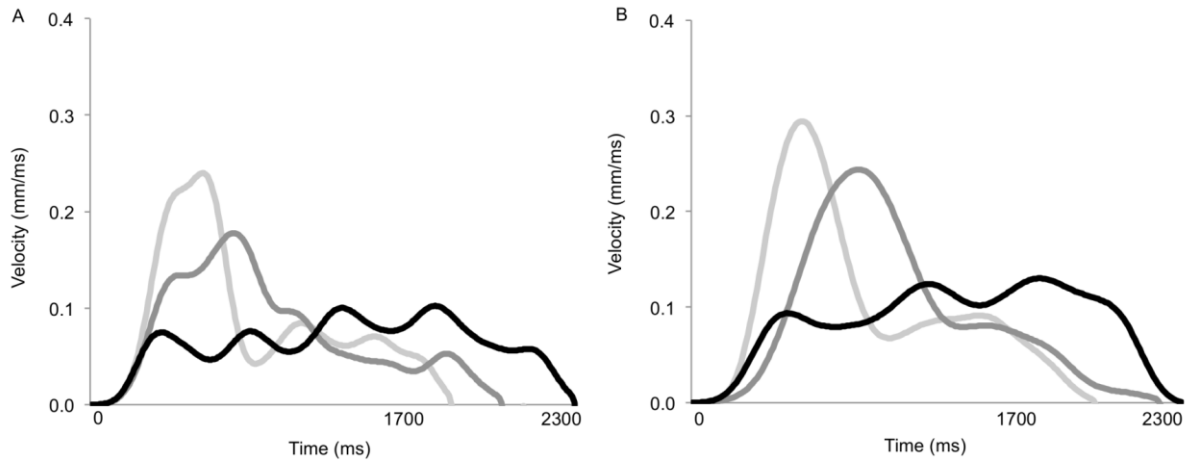

A
Observation Phase
Imitation Phase
B
Observation Phase
Imitation Phase
Trial Timeline


0.4
0.3
0.2
0.1
0.0
Velocity (mm/ms)
0
1700
Time (ms)


3000
2500
2000
1500
1000
500
0
Movement Time (ms)
Atypical17
Atypical26
Model
Constant Velocity


0.4
0.3
0.2
0.1
0
Peak Velocity (mm/ms)
Atypical17
Atypical26
Model
Constant Velocity


Percentage-of-Time to Peak Velocity (%)
50
40
30
20
10
0
Atypical17
Atypical26
Constant Velocity
Model


A
0.4
0.3
0.2
0.1
0.0
Velocity (mm/ms)
0
1700
2300
Time (ms)
B
0.4
0.3
0.2
0.1
0.0
Velocity (mm/ms)
0
1700
2300
Time (ms)