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To cite this article: Cheryl M. Glazebrook, Kelsey Brown, Steven L. Prime, Steven R. Passmore & Jonathan J. Marotta (2020) Both reaching and grasping are impacted by temporarily induced paresthesia, Somatosensory & Motor Research, 37:2, 106-116, DOI: [10.1080/08990220.2020.1750359](https://doi.org/10.1080/08990220.2020.1750359)

To link to this article: <https://doi.org/10.1080/08990220.2020.1750359>



Published online: 20 Apr 2020.



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ARTICLE



Both reaching and grasping are impacted by temporarily induced paresthesia

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ABSTRACT

Along with visual feedback, somatosensory feedback provides the nervous system with information regarding movement performance. Somatosensory system damage disrupts the normal feedback process, which can lead to a pins and needles sensation, or paresthesia, and impaired movement control. The present study assessed the impact of temporarily induced median nerve paresthesia, in individuals with otherwise intact sensorimotor function, on goal-directed reaching and grasping movements. Healthy, right-handed participants performed reach and grasp movements to five wooden Efron shapes, of which three were selected for analysis. Participants performed the task without online visual feedback and in two somatosensory conditions: 1) normal; and 2) disrupted somatosensory feedback. Disrupted somatosensory feedback was induced temporarily using a Digitimer (DS7AH) constant current stimulator. Participants' movements to shapes 15 or 30 cm to the right of the hand's start position were recorded using a 3D motion analysis system at 300 Hz (Optotrak 3D Investigator). Analyses revealed no significant differences for reaction time. Main effects for paresthesia were observed for temporal and spatial aspects of the both the reach and grasp components of the movements. Although participants scaled their grip aperture to shape size under paresthesia, the movements were smaller and more variable. Overall participants behaved as though they perceived they were performing larger and faster movements than they actually were. We suggest the presence of temporally induced paresthesia affected online control by disrupting somatosensory feedback of the reach and grasp movements, ultimately leading to smaller forces and fewer corrective movements.

ARTICLE HISTORY

Received 8 November 2019
Accepted 30 March 2020

KEYWORDS

Goal-directed reaching;
motion analysis; motor
control; kinematic

Introduction

During everyday activities humans perform numerous reaching and grasping movements in order to interact with the world around them. Although vision is a primary source of sensory input that provides valuable information needed to plan, correct, and evaluate the success of reach and grasp actions (Carlton 1981; Elliott et al. 2017; Elliott et al. 1991; Elliott et al. 2001), the central nervous system combines sensory feedback from multiple sources to execute an appropriately timed, and accurately placed grasp (Desmurget and Grafton 2000; Khanafer and Cressman 2014). In addition to vision, the somatosensory system provides information about current limb position and movement (Balslev et al. 2007; Cluff et al. 2015; Ernst and Banks 2002; Lavrysen et al. 2018; Scott 2012; Wolpert et al. 1998). The somatosensory system includes a variety of sensations related to the body and provides another source of valuable sensory feedback for movement control. The contribution of somatosensory input to motor skill performance is included in a number of models of motor control and learning for both gross and fine motor skills (e.g., Elliott et al. 2010). Compared to visual input, somatosensory input also has the advantage that updates to ongoing limb movements can be made more rapidly (Cluff

et al. 2015; Desmurget and Grafton 2000; Scott 2016). While vision and somatosensation are both integral for limb control, we know relatively little about the specific impacts of distorted somatosensation on movement control. The contributions of vision for action have been studied using a wide range of visual illusions and distortions, whereas our understanding of how disrupted (as opposed to removed) somatosensory input affects action control is by comparison very limited (Aglioti et al. 1995; Christina 2017; Jackson et al. 1997; Marotta et al. 1998; Westwood and Goodale 2011). One exception is the use of tendon vibration to stimulate muscle spindles which has been found to degrade proprioception (Bock, Pipereit, and Mierau 2007; Capaday and Cooke 1981; Cordo et al. 1995; Goodman and Tremblay 2018; Lavrysen et al. 2018; Tidoni et al. 2015).

Reach and grasp movements are performed daily and are necessary for functional independence with most activities of daily living. Fine motor control is also becoming increasingly relevant when interacting with computers and other human-machine interfaces. Damage to the somatosensory system can occur from repetitive actions (e.g., carpal tunnel syndrome) or as a secondary complication related to diabetes, stroke or spinal cord injury (Mackay and Mensah 2004; Mathers and Loncar 2006). The prevalence of diabetes has

almost doubled since 1980 and carpal tunnel is the most common nerve entrapment in the upper limb (Atroshi et al. 1999; Mathers and Loncar 2006). As a consequence, a relatively large proportion of the population experiences paresthesia, a feeling of tingling and numbness. Paresthesia may be constant or intermittent and represents disruption as opposed to a complete loss of somatosensory input (Sharif-Alhoseini et al. 2012). Thus, a better understanding of the consequences of reaching and grasping with disrupted somatosensory input is both theoretically and practically relevant given the increasing prevalence of paresthesia (e.g., secondary to Type 2 Diabetes) and the growing reliance on fine motor skills in society (e.g., smartphone and remote operation interfaces).

Background

Models of limb control

The salience of vision has led to a focus on the visual control of goal-directed actions such as a reach to grasp action. Vision is considered to be the preferred modality for spatial information as it can directly code the location of objects. A number of researchers have examined the details around how both the disruption and removal of vision affect movement planning and control and have shown there are predictable patterns of behaviour that compensate for the lack of visual input (Hansen et al. 2006; Khan et al. 2006; Servos et al. 1992). One of the most consistent findings related to goal-directed reaching with and without vision is the concept of the worst-case scenario (Elliott and Allard 1985; Hansen et al. 2006; Zelaznik et al. 1983); if participants are unsure whether vision will be available after the “go” signal to initiate their action, they will prepare to move without vision – as in prepare for the worst-case scenario of not having vision. That is, reaching movements made without visual feedback (i.e., when vision is removed at movement onset—hereafter referred to as no vision movements) have been shown to have specific kinematic characteristics, including an earlier peak velocity compared to movement made with visual feedback. No vision movements have also been shown to be associated with greater movement variability (Rolheiser et al. 2006). During no vision reach-to-grasp movements, participants’ maximum grip apertures (i.e., the widest the grip gets during reach) tend to be larger and the time to maximum grip aperture tends to be longer during the reach compared to closed-loop movements (Hesse and Franz 2009; Bradshaw and Watt 2002; Prime and Marotta 2013). Larger maximum grip apertures appear to reflect participants allowing for a greater margin of error when visual feedback is not available. Researchers have also studied the time needed for visually-based corrective movements to be processed and when vision is most beneficial for corrective limb movements. For example, Tremblay and colleagues have demonstrated that visual feedback of the limb at or near peak velocity provides enough relevant visual input to improve limb control (Tremblay et al. 2017). Thus, corrections to initial limb trajectories can be made with limited amounts of sensory feedback.

Overall, there is a robust literature on when and how visual input affects online and offline control of actions (Khan et al. 2006). In addition to the well-acknowledged role of vision in motor control, current models of limb control also describe the contribution of somatosensory input (e.g., Elliott et al. 2010). More specifically, both behavioural and computational models of limb control refer to internal models where an internal model is considered to be a representation of both the current and desired limb position that is based on visual and/or somatosensory input (Wolpert et al. 1998; Todorov and Jordan 2002; and Scott 2012, 2016). Corrections to the limb trajectory are performed when visual and/or somatosensory feedback detects that the current limb position has deviated from the intended trajectory directed towards the action goal (e.g., the cup when reaching and grasping it). Visual information available prior to peak limb velocity is thought to inform visually based corrections that primarily occur during the limb-target phase of limb control (Elliott et al. 2017; Grierson and Elliott 2009; Tremblay et al. 2017). In contrast, proprioception contributes to early limb regulation by providing rapid updates to the limb’s current position (Grierson and Elliott 2008). Cutaneous input, on the other hand, plays a key role when interacting and manipulating objects (Ray et al. 2019).

Historically a common method for disrupting somatosensory input is tendon vibration. Tendon vibration is a technique that mechanically perturbs the muscle spindles, leading to varying degrees of illusions of movement that is specific to the tendon(s) being stimulated (Cordo et al. 1995; Lavrysen et al. 2018; Taylor et al. 2017). Besides illusions of movement, tendon vibration also leads to altered limb control. For example, during an upper extremity aiming task tendon vibration of the antagonist muscle was found to lead to target undershooting in trials without vision (Capaday and Cooke 1981). More recently, Goodman and Tremblay (2018) reported that dual tendon vibration (Bock et al. 2007) between movement trials altered movement control both with and without vision. The temporal characteristics of tendon vibration has also been found to modulate the changes in coordination. For example, Cordo et al. (1995) found that the frequency of tendon vibration led to different reaching characteristics. At higher frequencies (40 Hz) participants opened their hand earlier and before their elbow reached a target angle. In contrast, at lower frequencies (20 Hz) their hand opened *after* their elbow reached the target angle. In summary, tendon vibration has been used to mechanically disrupt somatosensory input leading to changes in movement perception and coordination (Capaday and Cooke 1981; Goodman and Tremblay 2018; Lavrysen et al. 2018; Taylor et al. 2017).

Reach and grasping

The action of reaching and grasping involves bringing the limb to a target object as well as preparing the hand to grasp the object. A widely-accepted view is that such reaching movements involve a two-stage process where first a movement plan is programmed based on comparing the

initial hand position to the target position and then motor commands are generated to execute the movement (Haffenden and Goodale 1998; Rossetti et al. 1995; Sober and Sabes 2005). Rossetti et al. (1995) showed evidence that movement planning in reaching-to-point actions are derived from integrating the hand position information from both vision and proprioception when programming the movement trajectory to the target. However, vision and proprioception appear to contribute differentially to different aspects of the motor plan. Proprioceptive information plays a larger role in planning the movement distance, whereas visual information plays a larger role in planning the movement direction (Lateiner and Sainburg 2003; Sainburg et al. 2003). Moreover, these cited studies show that the relative weighting of visual and proprioceptive information in planning and online control appears to depend on the degree to which vision is available (Balslev et al. 2007; Sarlegna and Sainburg 2009).

Much of the work on the visual control of manual prehension has focussed on the distinction between the first two components of a reaching and grasping movement – the control of the reaching arm and the control of the hand and fingers during grip formation. These components are not organised sequentially but instead unfold in parallel. Thus, as the hand moves towards the object, the fingers have already begun to open and the hand has begun to rotate in the appropriate direction (Jeannerod 1981, 1984, 1986). This temporal coordination may be just one aspect of a more fundamental interaction between these two components of prehension (Goodale and Servos 1996).

Taken together, researchers have shown that the relative weighting of sensory modalities changes with task demands, particularly if the salience of the dominant modality is reduced. Beyond the theoretical contributions of understanding how disrupted somatosensory input may be compensated for during reaching and grasping, there is also a large practical aspect to understanding how paresthesia affects the performance of reach and grasp actions. In order to look specifically at the impact of disrupted somatosensory input (without associated changes in motor pathways) we chose to temporarily induce paresthesia in young adults without any history of neurological injury or disease (Passmore et al. 2014; Zehr and Chua 2000). Temporarily induced paresthesia, using direct nerve stimulation, creates a feeling of paresthesia along the nerve stimulated that is similar to the feeling that accompanies a compressed nerve. Temporarily induced paresthesia has been shown to disrupt and enhance movement performance using continuous upper extremity tasks as well as tactile learning paradigms. However, the everyday task of reaching and grasping has not been studied. Thus, the present study examined the effects of temporarily induced paresthesia of the median nerve on goal-directed reaching and grasping movements. Based on current models of limb control (e.g., Elliott et al. 2010; Scott 2012), we predicted that temporarily induced paresthesia would negatively impact early online control and the ability to monitor limb and grasp position throughout the reach to grasp movement.

We chose the instruction “to move at a comfortable pace” because we wanted participants to move as they would for an activity of daily living, such as reaching and grasping a cup. Although the task instructions were to move comfortably and naturally, we predicted participants would increase their IT and MT compared to moving with intact somatosensory feedback in order to accommodate for the uncertainty introduced by the perturbed sensory feedback of limb position. We also predicted that the uncertainty of the limb and grasp positions would result in more cautious movement plans, leading to movements with smaller peak velocities (PVs), longer time to reach PV, and wider grip apertures.

We chose to remove visual feedback at the go signal because without visual feedback participants would be more reliant on proprioception to identify the current limb position and implement online corrective movements. We predicted that when paresthesia was present that online corrections to the limb position would be impeded by uncertainty in current limb position (due to the added stimulation of the median nerve) and any movement errors that remained uncorrected at movement end would result in more variability in the limb trajectory and grasp locations. Finally, the time around peak deceleration of a reaching movement was also analysed as it is thought to occur in the same time frame as the online corrective movements. We predicted peak deceleration would be greater (i.e., lower), and take longer to achieve, in the presence of paresthesia due to the uncertainty in limb position. With respect to the measures of grasp performance, we expected participants to use a wider maximum grip aperture that occurred earlier in the movement. We predicted participants would compensate for their uncertainty in finger and thumb position, caused by the induced paresthesia, by adopting a more conservative movement strategy that avoided a movement errors associated with a grip that was too small.

Method

Participants

Twelve right-handed participants (4 males, 8 females; ages 20–29, $M = 23$ years, $SD = 2.6$) from the University of Manitoba population participated in the present experiment. All of the participants self-reported having normal or corrected-to-normal vision and no history of numbness or tingling in the upper extremity. All participants were given information about the protocol before providing written informed consent. All procedures were approved by the Education and Nursing Research Ethics Board at the University of Manitoba and conducted in accordance with the Declaration of Helsinki (1964, 2013). Participants were provided a small honorarium for their participation in the study.

Apparatus

Participants sat on a height adjustable stool at a table. The table was covered by a black tablecloth to prevent glare

from the lights and to cover imperfections on the table that could be used as reference points. The start position for the reaching and grasping task was a 1 by 1 cm piece of VELCRO™ on the tablecloth that was lined up with each participant's midline. There were no other markings on the tablecloth.

The target object for each trial was one Efron shape placed 15 or 30 cm to the right of the home position. Five possible Efron shapes were presented individually as target objects on any given trial for the present study. The Efron shapes are rectangular wooden objects each with unique dimensions but the same surface area (Efron, 1969). The shapes were wooden blocks with lengths and widths as follows: (A) 15.2 × 4.2 cm, (B) 12.2 × 5.2 cm, (C) 10.2 × 6.2 cm, (D) 9.0 × 7.1 cm and (E) 8.0 × 8.0 cm. Efron shapes A, C and E were selected *a priori* as the targets for the present study. Efron shapes B and D were included in the testing procedure in order to prevent participants from simply estimating their grip apertures and to force them to attend to the shapes' sizes when planning and executing their movements. Movement amplitude was measured from the centre of the start position to the centre of the shape.

A three-dimensional motion capture system was used to record reach trajectories and grasping movements (Optotrak 3D Investigator, Northern Digital Inc., Waterloo, ON). Two Infra-red Emitting Diodes (IREDs) were secured at each of the following locations: the distal portion of the participant's right index finger, thumb, and radial styloid process (wrist). The markers were secured with medical tape and the cords were contained using an elastic hair band that was placed around the participant's forearm. Participant's movements were recorded during each trial at 300 Hz for 3 seconds. Visual occlusion spectacles (PLATO, Translucent Technologies, Toronto, Ontario) were used to control when the participants obtained visual information of the environment. A custom-made programme designed using E-Prime 2.0 (Psychology Software Tools) controlled and synchronised the initiation of the Optotrak recording and the visual occlusion spectacles becoming opaque.

Temporary paresthesia was induced using a Digitimer (DS7AH) Constant Current Stimulator on the median nerve. The stimulation was delivered transcutaneously using 30 mm disposable adhesive electrodes (Kendall™ Adhesive Snap Electrodes, Medi-Trace Mini, from King Medical, Ltd., King City, Ontario). Electrodes were placed over the distal aspect of the anterior forearm. The stimulator was active throughout the block of trials with induced paresthesia. Each pulse had a stimulus duration of 0.2 ms with an interstimulus interval of 10 ms and voltage edge of 0.2 V. The transcutaneous stimulation creates a sensation of pins and needles, which was reported by all participants.

In order to establish a consistent intensity level for individual participants the intensity level to reach the threshold for sensory, radiating, premotor, motor signs were recorded. The intensity at which participants first reported any sensation was defined as the sensory threshold. Radiating sensation was defined as when the participant reported the sensation travelling along the nerve (forearm). Premotor

threshold was defined as the most intense signal that could be delivered before motor contraction occurred. The premotor threshold was used to establish the appropriate intensity for the constant current stimulator for each participant.

A change in tactile perception was verified using a monofilament test for light touch on the palmar surface of the hand (Touch-Test™ Sensory Evaluator: Semmes-Weinstein Monofilaments). Immediately before participants performed the paresthesia condition monofilament testing was done to measure their baseline tactile perception in both the thumb and index finger. Once the appropriate intensity level for the transcutaneous stimulus was established then monofilament testing was repeated (see Table 1 for individual values). All of the monofilament testing was done while the participant's hand was prone in order to maintain a similar position to that of the grasping task.

Procedure

Participants completed two experimental sessions on two separate days: Paresthesia and No Paresthesia (normal somatosensory feedback) conditions. The order of the Paresthesia and No Paresthesia conditions were counter-balanced across participants so that half of the participants performed the Paresthesia condition first. An experimental session took 30-45 minutes per day and consisted of participants performing a total of 160 trials. Disrupted somatosensory feedback was induced temporarily using a Digitimer (DS7AH) constant current stimulator and caused a sensation of tingling and numbness throughout the index finger and thumb that was present throughout the block of trials in the paresthesia condition.

General experimental paradigm

At the beginning of each trial the experimenter placed one of the five Efron shapes (A, B, C, D, or E) at one of the two distances (15 or 30 cm) while the participant's vision was occluded with their right index finger and thumb on the start position. The order of the Efron shapes and distances were pseudo-randomly determined so that Efron shapes A, C and E were presented ten times at each location, while B and D were presented five times per location. As explained above, *a priori* Efron shapes A, C and E were selected for analysis.

Once the Efron shape was positioned the experimenter initiated the trial. The goggles opened for 1000 ms and then closed, which was the cue for the participant to begin their movement to the shape without vision. Participants were instructed to move at a comfortable pace, pick up the shape with their index finger and thumb gripping it widthwise, put the shape back down and return to the home position. Once the trial was complete, participants returned their finger to the home position and the goggles remained shut until the shapes were set up for the next trial.

Participants completed a total of 160 trials. A total of 80 trials were completed with and without paresthesia with only one shape being presented each trial. The order of the

Table 1. Tactile Perception using the Touch-Test™ Sensory Evaluator: Semmes-Weinstein Monofilaments.

Participant	Without Stimulus (Thumb)	Without Stimulus (Index)	With Stimulus (Thumb)	With Stimulus (Index)	Start With or Without Stimulus
P01	2.03	2.03	4.31	4.31	Without
P02	2.83	3.61	4.31	4.31	With
P03	2.83	2.83	3.61	3.61	Without
P04	2.83	2.83	2.83	2.83	With
P05	3.61	3.61	4.31	4.31	Without
P06	2.83	2.83	3.61	3.61	With
P07	2.83	2.83	4.31	3.61	Without
P08	2.83	2.83	3.61	3.61	With
P09	2.83	2.83	4.31	3.61	Without
P10	2.83	2.83	3.61	3.61	With
P11	2.83	3.61	4.56	6.65	Without
P12	2.83	2.83	3.61	3.61	With

Note. All values in millinewtons.

Efron shapes and distances were pseudo-randomly determined so that Efron shapes A, C and E were presented ten times at each location. Shapes B and D, which we refer to as distractors to prevent participants from ballparking their grasp size, were presented five times per location and not included in the data analysis.

Data analysis

All reach variables were derived from the wrist IRED. Reaching movement onset and offset were identified as the first frame that the limb velocity exceeded 50 mm/s or fell below 50 mm/s for a minimum of 15 frames respectively. Reaction time (RT) and Movement Time (MT) were calculated as the time required for movement planning and execution respectively. RT was defined as the duration of time from the go signal (i.e., vision was removed) until reaching movement onset. MT was defined as the time from movement initiation until movement termination. Other variables used to characterise the reach included: PV, time to peak velocity (ttPV), PD, time to peak deceleration (ttPD), and time after peak deceleration (taPD). Peak velocity and deceleration were calculated after differentiating the spatial displacement data from the primary axis.

Trial-trial variability of the reach trajectory in the primary axis of movement, as well as trial-trial variability of the grip aperture, were analysed as described above. To characterise intraparticipant trial-trial spatial variability in limb trajectory and grip aperture we identified the location of the wrist (limb trajectory), or finger and thumb to calculate grip aperture (i.e., resultant displacement of finger and thumb locations), at key standardised points throughout the movement: 20, 40, 60, 80, 100% of MT for the trial in question (Heath et al. 2011). Next, the within participant standard deviations of the wrist spatial location, as well as grip aperture, at each normalised time point was calculated. The logic for the trial-trial variability analysis is that any initial variability in limb position that is present early in the movement will continue to accumulate throughout the movement (Khan et al. 2003, 2006). If left uncorrected then the variability will be greatest at movement endpoint. However, if online control processes are engaged, then a significant decrease in trial-trial spatial variability is observed following the online correction. Thus, if the presence of paresthaesia impacts the movement preparation then we expected greater variability early in the

movement (20 and 40% of MT) whereas if the induced paresthaesia impedes the ability to engage in online control we expected greater variability at 80 and 100% of MT when compared to no paresthaesia (Heath et al. 2011; Khan et al. 2003, 2006).

Analyses of the grasp data were conducted on the index finger and thumb grasp positions along the horizontal axis of the shape. Dependent measures used to characterise the grasp component of the movement included index finger endpoint variability, maximum grip aperture (MGA), absolute time to maximum grip aperture (ttMGA) and the percentage of the movement time at maximum grip aperture (%MT at MGA). The maximum grip aperture (MGA) between the index finger and thumb during the reach component of the movement was identified as well as the absolute (ttMGA) and relative (%MT at MGA) taken to reach MGA. Finger endpoint variability calculated using the standard deviation in the location of finger at movement offset.

Individual trials were removed if the RT or MT exceeded ± 2.5 standard deviations of that participant's mean for that condition. Dependent variables were then analysed using a 2 Condition (Paresthaesia, No Paresthaesia) by 3 Shape (A, C, E) by 2 Distance (15, 30 cm) repeated measures ANOVA. Reaching trajectories were analysed using a 2 Condition (Paresthaesia, No Paresthaesia) by 3 Shape (A, C, E) by 2 Distance (15, 30 cm) by 5 MT proportion (20, 40, 60, 80, 100% of MT) repeated measures ANOVA. Post hoc analysis was performed on significant interactions using Tukey's Honestly Significant Difference.

Results

Performance measures

Initiation time

No significant main effects or interactions for IT were found ($F_s = 0.01$ to 2.04). The mean time to initiate movements without paresthaesia was 491 ms ($SD = 280$) and 535 ms ($SD = 339$) with paresthaesia.

Movement time

As expected the MT analysis revealed a significant main effect for distance, $F(1,11) = 31.65$, $p < 0.001$. Participants spent more time reaching to shapes that were 30 cm

($M = 816$ ms, $SD = 232$ ms) versus 15 cm ($M = 660$ ms, $SD = 167$ ms) away.

No other main effects or interactions were found ($F_s = 0.38$ to 1.9). The mean time to execute movements without paresthaesia was 724 ms ($SD = 222$) and 753 ms ($SD = 211$) with paresthaesia.

Kinematic measures

Peak velocity

There was a significant main effect for shape, $F(2,22) = 5.02$, $p < 0.02$ and distance, $F(1,11) = 83.13$, $p < 0.0001$. As expected, participants reached a higher peak velocity when reaching 30 cm ($M = 616$ mm/s, $SD = 172$ mm/s) compared to 15 cm ($M = 417$ mm/s, $SD = 108$ mm/s). Participants also reached a higher peak velocity when reaching to Shape A ($M = 528$ mm/s, $SD = 175$ mm/s) when compared to Shape E ($M = 503$ mm/s, $SD = 168$ mm/s). Shape C was not significantly different from either Shape A or E ($M = 512$ mm/s, $SD = 184$ mm/s).

Analysis of time to peak velocity revealed significant main effects for condition, $F(1,11) = 5.62$, $p < 0.04$ and distance, $F(1,11) = 92.83$, $p < 0.0001$. Participants took longer to reach peak velocity when experiencing paresthaesia ($M = 277$ ms, $SD = 67$ ms) versus no paresthaesia ($M = 260$ ms, $SD = 65$ ms). Participants also took longer to reach peak velocity when reaching 30 cm ($M = 291$ ms, $SD = 66$ ms) versus 15 cm ($M = 246$ ms, $SD = 59$ ms). None of the main effects or interactions including shape were significant.

Peak deceleration

There were significant main effects for Condition, $F(1,11) = 6.02$, $p < 0.04$, Shape $F(2,22) = 6.55$, $p < 0.01$, and Distance, $F(1,11) = 27.8$, $p < 0.001$. As expected, participants had higher deceleration when moving 30 cm ($M = 2752$ mm/s², $SD = 1015$ mm/s²) when compared to 15 cm ($M = 2130$ mm/s², $SD = 761$ mm/s²). The Condition by Shape interaction was also significant, $F(2,22) = 5.22$, $p < 0.02$. As illustrated in Figure 1, participants reached significantly lower peak decelerations when reaching to pick up Shapes A and C when paresthaesia was present. There was no significant difference between the two conditions for Shape E.

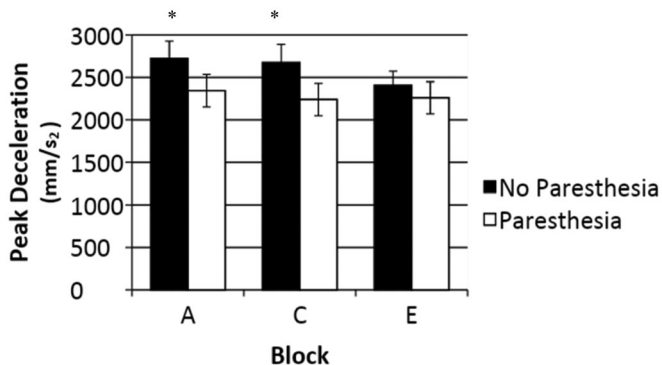


Figure 1. Peak deceleration (mm/s²) as a function of Condition and Shape. Movements performed to Shapes A and C had significantly lower peak decelerations when paresthaesia was present. There were no significant differences for Shape E.

Analysis of time to peak deceleration revealed significant main effect of condition, $F(1,11) = 16.26$, $p < 0.01$ and distance, $F(1,11) = 8.43$, $p < 0.02$. Overall, participants took longer to reach peak deceleration with paresthaesia ($M = 480$ ms, $SD = 181$ ms) compared to no paresthaesia ($M = 441$ ms, $SD = 156$ ms). Participants also took longer to reach peak deceleration when reaching 30 cm ($M = 502$ ms, $SD = 206$ ms) as opposed to 15 cm ($M = 418$ ms, $SD = 108$ ms).

Analysis of time after peak deceleration revealed a main effect for distance, $F(1,11) = 41.1$, $p \leq 0.0001$. Participants spent more time approaching the target when the shape was placed 30 cm away ($M = 316$ ms, $SD = 86$ ms) compared to when it was placed 15 cm away ($M = 244$ ms, $SD = 104$ ms). None of the main effects or interactions involving condition were significant (F_s ranged from 0.22 to 1.4).

Trial-trial variability

Analysis of the trial-trial variability throughout the reaching movement revealed main effects for Distance, $F(1,11) = 20.2$, $p < 0.001$, MT Proportion, $F(4,44) = 18.99$, $p < 0.0001$ and a Distance \times Location interaction, $F(4,44) = 8.84$, $p < 0.0001$. As illustrated in Figure 2, post hoc analysis of the Distance \times Location interaction indicated that reaches to shapes 30 cm away were significantly more variable early in the movement. However, the difference between the conditions was no longer significant by 80% of MT and remain not significant at 100% of MT. None of the main effects or interactions including Condition were significant (F -ratios 0.5 to 1.54).

Grasp measures

Maximum grip aperture

The analysis of the mean maximum grip aperture revealed significant main effects for condition, $F(1,11) = 5.84$, $p < 0.04$, shape, $F(2,22) = 53.2$, $p < 0.0001$ and distance, $F(1,11) = 15.56$, $p < 0.01$. Regardless of distance or shape, participants used a smaller grip aperture when experiencing paresthaesia ($M = 94$ mm, $SD = 12$ mm) versus without ($M = 97$ mm, $SD = 12$ mm). When MGA was collapsed across conditions and distances, the results revealed that participants scaled their grip aperture to each shape such that they reached a significantly wider grasp when reaching to shape E

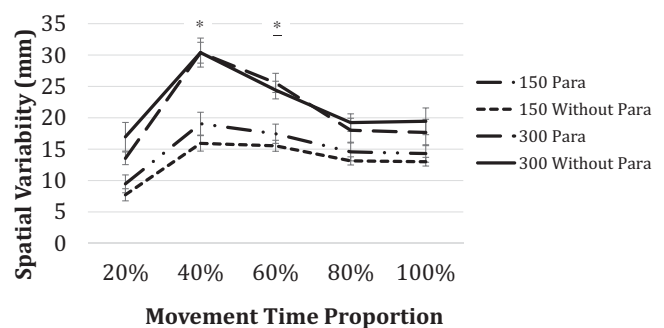


Figure 2. Mean trial-trial spatial variability (mm) in the position of the wrist as a function of Condition, Distance and Proportion of MT. Participants had significantly more variability at 40% and 60% of MT when reaching to shapes 30 cm away compared 15 cm away, regardless of whether paresthaesia was present or not.

($M = 104\text{mm}$, $SD = 8\text{mm}$) compared to shape C ($M = 97\text{mm}$, $SD = 9\text{mm}$) and shape A ($M = 86\text{mm}$, $SD = 12\text{mm}$). All MGA's were significantly different from one another. Participants also reached a wider MGA when reaching to shapes farther away (15 cm: $M = 94\text{mm}$, $SD = 12\text{mm}$; 30 cm: $M = 97\text{mm}$, $SD = 12\text{mm}$). Analysis of within participant standard deviation of MGA revealed no significant main effects or interactions (F ratios ranged from 0.53 to 2.8).

Time reach onset to MGA

Analysis of the time from reach onset until MGA revealed significant main effects for condition, $F(1,11) = 7.41$, $p < 0.02$, shape, $F(2,22) = 25.29$, $p < 0.0001$ and distance, $F(1,11) = 28.18$, $p < 0.001$. The shape and distance main effects were superseded by a significant shape by distance interaction, $F(2,22) = 6.75$, $p < 0.01$. As illustrated in Figure 2, time to MGA did not differ among the shapes at the near distance (150 mm), but did occur progressively later with increasing shape width (from shape A to E) when the shapes were placed at the far distance (300 mm). The observations of Figure 3 were confirmed by *post hoc* analysis that showed that at the near distance there were no significant differences between shapes while at the far distance there were significant differences between Shape A versus C and C versus E (both comparisons $p < 0.01$). Overall participants took longer to reach MGA when paresthaesia was present ($M = 606\text{ms}$, $SD = 224\text{ms}$) compared to when it was not present ($M = 536\text{ms}$, $SD = 230\text{ms}$).

Percent MT at MGA

Analysis revealed significant main effects for condition, $F(1,11) = 5.55$, $p < 0.04$, and shape, $F(2,22) = 10.06$, $p < 0.001$. Further *post hoc* analysis demonstrated that MGA occurred relatively later in the movement reaching to shape E, the shape that required the widest grasp (A: $M = 73.1\%$, $SD = 12.4\%$; C: $M = 75.9\%$, $SD = 9.7\%$; E: $M = 82.1\%$, $SD = 9.8\%$). Participants also reached MGA relatively later in the movement when reaching with paresthaesia ($M = 79.9\%$, $SD = 11.5\%$) versus without paresthaesia ($M = 74.2\%$, $SD = 10.4\%$).

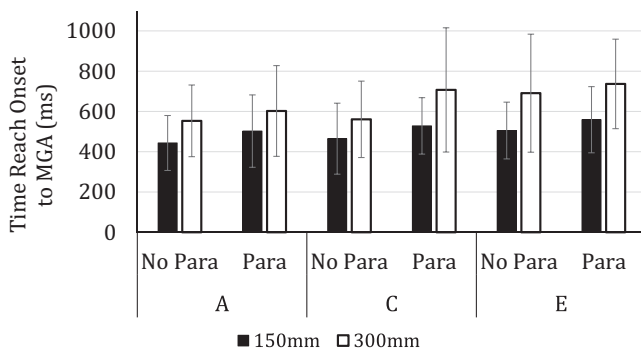


Figure 3. Absolute Time to Maximum Grip Aperture as a function of Condition, Shape and Distance. There was an overall main effect for condition, where Time to MGA occurred later when paresthaesia was present. Time to MGA also occurred significantly later as shape width increased (A < C < E), regardless of whether paresthaesia was present or not; however the difference was only significant when the shapes were located farther away (300 mm).

Finger endpoint variability

Analysis of the variability (Standard Deviation-SD) of the index finger endpoint revealed a main effect for condition, $F(1,11) = 6.62$, $p < 0.03$ and for distance, $F(1,11) = 7.23$, $p < 0.03$, where participants were more variable when reaching towards shapes 30 cm ($M = 29\text{mm}$, $SD = 6.7\text{mm}$) versus 15 cm away ($M = 18\text{mm}$, $SD = 4.1$). The endpoint of the finger was more variable with paresthaesia ($M = 28\text{mm}$, $SD = 7.9$) versus without ($M = 20\text{mm}$, $SD = 5.7\text{mm}$). No other main effects or interactions had a F -ratio above one.

Trial-trial variability grip aperture

Analysis of the trial-trial variability of grip aperture throughout the reaching movement revealed significant main effects for Shape, $F(2,22) = 11.67$, $P \leq 0.001$ and MT proportion, $F(4,44) = 9.99$, $p \leq 0.0001$. Both main effects were superseded by significant two-way interactions including shape by distance, $F(2,22) = 3.97$, $p \leq 0.04$, and condition by MT proportion, $F(4,44) = 3.38$, $p \leq 0.02$. Further analysis of the shape by distance interaction revealed that grip aperture was more variable throughout the movement when grasping shape E located 30 cm away ($M = 10.8\text{mm}$, $SD = 4.7$) compared to all other combinations of shape and distance (A-15: $M = 8.4\text{mm}$, $SD = 3.5$; A-30: $M = 8.9\text{mm}$, $SD = 3.6$; C-15: $M = 9.0\text{mm}$, $SD = 4.3$; C-30: $M = 8.9\text{mm}$, $SD = 4.6$; E-15: $M = 9.2\text{mm}$, $SD = 4.2$).

As illustrated in Figure 4, further analysis of the condition by proportion MT interaction revealed no differences in grip aperture variability early in the limb trajectory. However, at 80% and 100% of movement time participants exhibited significantly more variability in their grip aperture when experiencing paresthaesia.

Discussion

In the present study, we measured participants' non-visually guided reaching and grasping movements under two conditions, either with or without induced paresthaesia. While the presence of paresthaesia did not prevent participants from completing the reach and grasp task (i.e., participants successfully located and picked up the shape), the results

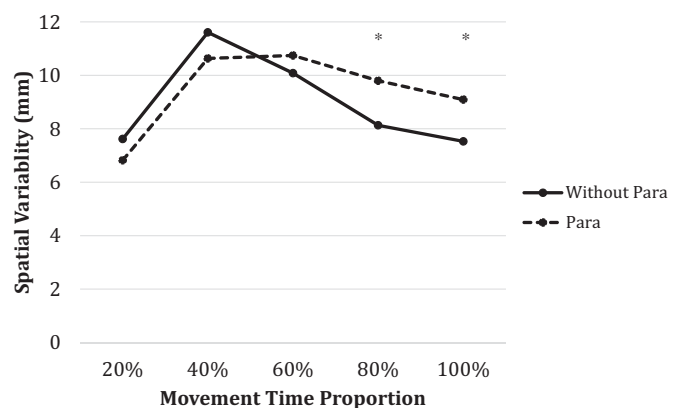


Figure 4. Mean trial-trial spatial variability (mm) of grip aperture as a function of Condition and Proportion of MT. Participants had significantly more variability at 80% and 100% of MT when reaching with the presence of paresthaesia compared to without induced paresthaesia.

showed paresthaesia impacted both the reach and grasp aspects of the action. The longer time to peak velocity is consistent with paresthaesia affecting early online control (Elliott et al. 2010). The lower peak deceleration and longer time to peak deceleration are consistent with participants needing more time to process the current limb position as they approach the shape (Note: there were no significant effects for relative times to PV or PD). Consistent with our prediction, the presence of paresthaesia led to more variability in where the index finger contacted the shape, which is evidence that there was more variability in the initial motor commands and/or ability to make online corrections. The finding that trial-trial variability of grip aperture was equivalent during the initial phase of the action, but significantly greater beginning at 80% of the movement duration, supports the suggestion that paresthaesia impacted participants' ability to engage in limb-target control processes. The presence of paresthaesia also led to systematically smaller maximum grip apertures. The smaller MGAs were reached later in the movement, regardless if measured as an absolute (time from reach onset to MGA) or in relative terms (percent MT at MGA). Thus, the temporal coordination between the reach and grasp varied with and without paresthaesia. Specifically, participants completed the reach and grasp task in a similar duration of time with and without paresthaesia, however the accuracy and temporal coordination between the parameters varied.

MGA has been shown to be typically larger during when grasping without visual feedback compared to with vision, presumably to allow for a greater margin of error in grip aperture scaling (Bradshaw and Watt 2002; Hesse and Franz 2009; Prime and Marotta 2013). We predicted that induced paresthaesia would lead to larger MGA, compared to the no paresthaesia condition, to compensate for both the lack of visual feedback and noisy proprioceptive feedback. In contrast, our results showed that MGA was smaller with paresthaesia than without, indicating a failure to take into account margins of error. It is unclear why this was the case. It is possible that paresthaesia interfered with feedback of the hand such that participants misperceived the position of their hand. As the induced paresthaesia was done through stimulation of the median nerve, the stimulation of the receptors may have created the perception of the grip aperture being wider than it was in reality. The latter explanation is consistent with a recent report of smaller movement amplitudes as a result of tendon vibration and associated illusory movement (Lavrysen et al. 2018). Capaday and Cooke (1981) also reported that participants tended to undershoot the target more when aiming to targets without vision and in the presence of tendon vibration applied to the antagonist muscle. It is not clear, however, why the illusion was towards a wider as opposed to a smaller grip. Future studies could include a perceptual task to provide further insight into the relationship between changes in perception and action (e.g., Heath et al. 2011).

The above results inform current models of limb control by providing insight into how disruption in one sensory modality influences specific limb control processes. Overall

the presence of paresthaesia appears to have interrupted early online control by disrupting the scaling of force to movement output. In the context of the multiple processes model of limb control neither movement initiation, nor the initial formation of the expected sensory consequences, were temporally impacted by the presence of paresthaesia (i.e., no differences in initiation time) when participants could prepare the movement in advance. However, both time to peak velocity and deceleration were longer when paresthaesia was present. In addition, peak deceleration was lower when paresthaesia was present. Based on the latter findings we suggest that the presence of paresthaesia affected the ability to compare the current limb position with the expected limb position. The smaller MGA that took longer to be reached is also consistent with the interpretation that participant's perception of their limbs was inaccurate and reflected a perception of more output for a given force than actually occurred. It is likely that the expected efference is based on prior experience, therefore the impact of paresthaesia was more apparent during the latter portion of the movement, that is during limb deceleration and limb-target control. Consistent with the above interpretation, participants had greater variability in both the pointing and grip aperture aspects of their reach to grasp movements. This larger variability with paresthaesia indicates that despite not having vision during the movement, participants did engage in limb-target control using available proprioception and that the presence of paresthaesia resulted in either fewer or less effective corrections during the limb-target control phase.

Findings from previous studies suggest proprioceptive information plays a larger role in encoding movement distance compared to movement direction (Lateiner and Sainburg 2003; Sainburg et al. 2003). Our results suggest that perturbing proprioceptive feedback by inducing paresthaesia did not influence distance encoding as evidenced from a lack of significant condition by distance interactions across all dependent variables. The difference in the present results may reflect differences in task and experimental details. The task in the current experiment was from a midline to lateral position as opposed to a reach in a forward direction. Differences in contributions of shoulder and elbow movement could explain the lack of effect for distance. In addition, the perturbation used in the present experiment focussed on cutaneous innervations of the median nerve, primarily at the hand and wrist. In contrast to the tendon vibration literature, in the current experiment participants had intact proprioceptive information available to them from muscles and tendons at the elbow and shoulder which likely contributed to their success at reproducing target distance. It should also be noted that spatial variability of the reaching movement as measured at the wrist was not impacted by the presence of paresthaesia, whereas the grasp site as measured at the finger was more variable in the presence of paresthaesia. We suggest that such differences in the results reflect the greater impact paresthaesia has on precision tasks, when accuracy requirements are greater. With respect to the present experiment, the more gross limb movement measured at the wrist requires the hand move to the general

area of the shape whereas finding a stable grasp site on the shape requires more precision.

Participants were aware for all conditions that they would not have vision of their environment once they began their movements. It is well established that the knowledge that vision will be removed on movement initiation leads to participants preparing their upcoming goal-directed action to account for the loss of visual feedback (Elliott and Allard 1985; Khan et al. 2006; Hansen et al. 2006; Zelaznik et al. 1983). With the knowledge that vision would not be available participants would have used a strategy in which they planned their movements to rely primarily on the movement plan. The present results provide further support that humans can flexibly adapt to the available sensory input. If visual feedback was available throughout the action participants may have shown more evidence of engaging online control. We chose not to provide online visual feedback because we predicted that if visual feedback was available then participants would substitute any use of proprioceptive feedback with visual feedback. Future work investigating how control changes with varying degrees of response to the induced paresthesia, as well as varying the amount of available visual feedback, will help to elucidate specific processes that participants may select based on the availability of sensory feedback. The reliance or use of sensory input may be based on the perceived accuracy of current or remembered limb position. If participants used an open-loop strategy then the presence of paresthesia would have minimal effect on movement planning and the accuracy of execution. That said, a number of task and individual variables may lead to an increased impact of paresthesia. Tasks with higher accuracy demands likely require accurate input from all senses to be successful as deficits are seen when peripheral sensation is compromised (Ray et al. 2019). In addition, sustained paresthesia may erode stored limb representation(s), leading to ongoing decreases in movement accuracy. Specifically, ongoing paresthesia may alter the temporal coordination between the reach and grasp components of the overall action. Overall the present study provides evidence that paresthesia leads to more conservative reach-to-grasp actions and may interfere with online control based on proprioceptive feedback. On the other hand, movement planning (i.e., RT) was not significantly longer with paresthesia with the current task conditions. Thus, for movement planning the removal of vision was more salient.

In the absence of vision, comparing the current limb position to a predictive internal model of the current estimated limb position becomes more important for completing the task successfully. The results overall support the notion that the presence of paresthesia interferes with this process. Participants performed the coordination of the reach and grasp more slowly, in particular for shapes that required added movement accuracy to successfully grasp the shape. There was also a delay in decelerating the moving limb, which may reflect a delay in updating the current limb position. However, participants were able to make corrections to their gross movement trajectories and overall scaled their grasps both when paresthesia was present and when it was

not present. Taken together, participants compensated for the added noise in the limb feedback by adjusting the temporal parameters of their reaching movement. In this way, changes in the temporal parameters of the reaching and grasp action were greater than changes in the spatial parameters. However, the increase in the endpoint variability of the index finger when paresthesia was present indicates that participants were not able to engage in online corrections as efficiently or effectively.

Participants experienced paresthesia throughout the block of trials, therefore they were able to develop a movement strategy to manage the presence of the paresthesia. This is likely similar to processes related to conditions where participants know or are unsure if they will have visual feedback (Hansen et al. 2006). In the latter cases participants prepare the upcoming action to accommodate for the fact that could have to produce the movement without vision. When comparing vision and no vision trials, humans will choose to modify movement planning time (RT) based on whether the trial has visual feedback or not. It seems that the added presence of paresthesia did not require any additional planning time (given the current task conditions), however, it did directly impact the time needed to integrate online limb feedback during movement execution. The present results provide evidence that although goal-directed movements made without vision after movement onset are thought to be under greater open-loop control, online limb regulation based on feedback from the moving limb, as well as current and past internal models of expected and predicted sensory consequences, are engaged actively. The reaching results also support the contention that paresthesia introduced noise into the feedback systems that led to participants adjusting their overall movement strategy. Together the results point to a movement strategy where participants are unsure of the precise location of their limb and are adapting an energy conservation strategy by slowing down and approaching the shape in such a way as to minimise the number and size of corrective movements.

Conclusion

The presence of temporally induced paresthesia had a direct impact on the signal-to-noise ratio within the perceptual motor system. There was an increase in within participant trial-trial variability for the precision aspect of the task (i.e., SD of index finger endpoint and SD of grip aperture). We suggest paresthesia created uncertainty in the perception of the current limb position that interfered with the integration of the online feedback with existing internal models of expected sensory consequences. In other words, when humans experience paresthesia it alters sensory feedback that in turn impacts the spatial and temporal coordination of well-learned actions, even within a few minutes. The implications of the observed changes in accuracy and coordination after a few minutes of temporarily induced paresthesia is that peripheral nerve impingements (e.g., carpal tunnel) will lead to altered movement patterns that may negatively impact performing activities of daily living by requiring

individuals to visually monitor movements normally performed without direct visual feedback.

Acknowledgements

The authors thank Jessica Sutton for her assistance with data analysis and manuscript formatting.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This work was supported by Research Manitoba under grant F313672 and the Natural Sciences and Engineering Research Council of Canada under grants 418482-2012 and 04964-18.

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