

**ADAPTATIONS IN VISUAL AND PROPRIOCEPTIVE PROCESSING
FOLLOWING SHORT- AND LONG-TERM VISUAL DEPRIVATION**

by

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ABSTRACT

While significant cortical plasticity is known to take place during prolonged visual deprivation, little is known about adaptation to acute onset vision loss. Here, we use a two-experiment study to investigate adaptations in processing visual and proprioceptive information in acute onset vision loss. Experiment I: Visual input was removed for 2 hours to simulate acute onset short-term vision loss. Participants performed 80 trials of a reaching and grasping task pre- and post-deprivation (160 trials). Proprioceptive control (No-Vision) was used for the first 40 trials, followed by 40 trials using visual control (Vision). In all trials participants grasped a circular target in response to an auditory tone. Prior to the initiation of each trial, the subject's arm was passively moved to the target location and returned to the start position by an experimenter. Kinematic measurements (e.g. limb position, grip aperture) were obtained using 3 infrared markers (thumb, forefinger, and wrist) and an OptoTrak Certus (Northern Digital, Inc.). Experiment II: On the Experimental day, Visual input was removed for 8 hours to simulate acute onset long-term vision loss. During the 8-hr deprivation, participants performed normal daily activities with the assistance of an experimenter. On the control day, participants performed these same activities with full vision. Pre- and post each 8 hour period participants performed the same grasping task as Experiment I. In addition to the grasping task, participants completed an oddball detection task following the grasping task. Somatosensory and visual evoked potentials were recorded in response to tactile and visual stimuli. Both tasks involved detecting stimulus onset to either the right or left index finger. Results of both experiments suggest a dual-phase adaptation: improving on movement speed rather than corrective capacity in early adaptation, and favouring planning more proprioceptively accurate movements later in adaptation.

PREFACE

This study was approved by the University of British Columbia Clinical Research Ethics Board (Experiment I: H07-01734; Experiment II: H11-01271). Data were collected at the University of British Columbia (Kelowna, BC), by Karen Bourns, Darian Cheng, Francisco Colino, Robert Hermosillo, and Brendan Cameron. Experiment II sought to build off results of Experiment I. Dr. Gordon Binsted helped with design, equipment acquisition, and funding for both studies. Karen Bourns was responsible for implementation and execution, including subject recruitment, data collection, data analysis, and writing of the manuscript. Francisco Colino provided code for behavioural protocols, while Darian Cheng and Krista Fjeld contributed significantly to analysis of the EEG data.

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CHAPTER 1: INTRODUCTION

1.1 Background

Programming and execution of movement is heavily dependent on visual and proprioceptive information (see Glover, 2004 for review). Together, vision and proprioception construct features of the environment and the objects within it. For example, when we perceive a glass of water sitting on a table, we see the size and shape of the glass, and its location relative to the rest of the environment. Integration of proprioceptive feedback tells us where that glass of water is located relative to our own body, specifically to the hand that wishes to reach for it. To reach out and take a drink from the glass, dimensions of the glass projected to the visual system ensure we grasp it appropriately for the size and shape, while the distance of the glass from our body is used to approximate the length of our reach. Many simple every day tasks require the close interaction of these two systems.

One can imagine, then, how these movements become disrupted when input from either sensory modality is restricted. If we can no longer see the glass, we have no information about size or shape, how full it may be, or where it is in the environment. Without proprioceptive input we have no reference of where we are in the environment or where our hand is relative to the glass, how far we need to move the arm to contact it, or what amount of force is being applied to hold it. Without vision, we lack information about what we are interacting with and where it lies in our environment; without proprioception, we lose our knowledge of where we exist within the environment, relative to the objects that comprise it. When one modality is compromised, we must rely on the other, in combination with the remaining senses, to construct a movement.

One notable exception is when we are reaching for something in a known location. If you keep the glass of water on your nightstand, in generally the same place every night, chances are you can successfully reach out and grasp it in the dark (i.e. without visual feedback) to take a

drink. The consistent, repetitive location generates a visual memory that can be used in place of active visual feedback. In conjunction with proprioceptive memory of the glass location relative to where you are in the bed, you are able to garner enough information about the environment to successfully complete the desired movement. Visual and proprioceptive memory can be used in a way as an alternate sense, if others become compromised.

1.2 The Importance of Vision

In 1899, Woodworth purported that any sense could justifiably be used to construct a movement, but the most accurate source of information related to that movement would be used preferentially. In his experiments comparing ocular versus muscle sense to perform a line drawing task, Woodworth showed a complete dependence on the eyes and disregard for muscle sense. He noted the importance of vision in planning but also for controlling a movement in his two-component limb control model. He describes a rapid initial aiming movement that is relatively consistent, termed the *ballistic phase*, and a *current control phase* in which visual feedback is facilitated to make corrections to the initial movement. These phases highlight the planning and control aspects of executing a movement. The ballistic phase is intended to bring the limb to the target in a general sense (based on a planned movement), while the control phase consists of smaller movements attempting to correct prior errors (based on visual feedback). Perhaps the simplest example of the importance of vision comes from reaching experiments performed in light versus dark, where we see greater accuracy of movements performed with the lights on than off (Zelaznik, Hawkins, & Kisselburgh, 1983). Notably, performance was equivocal between light and dark when movements were rapid, thus suggesting a substantial processing delay associated with using visual feedback. We have since come to accept that when visual information is available it is used maximally and predominantly to structure our perception of that environment and our movements within it (Heath, 2005).

1.3 Visual Feedback

Woodworth's work sparked much discussion about the relative contributions of the 'ballistic' (i.e. planning) and 'current control' (i.e. feedback control) phases to the accurate production of goal directed action. The last century has yielded a large and varied debate regarding the relative contributions of these processes and significant delineation of limb-control models (see Elliott, Hansen, Grierson, Lyons, Bennett, & Hayes 2010 for a review). Due to the delay in neural processing of visual information, early phases of movement are typically thought to operate in an open-loop manner (Sainburg, 2010). In an open-loop model, feedback is not used to control a movement directly, but rather sensory information obtained prior to the movement (Plamondon, 1995; Plamondon & Alimi, 1997) determines its parameterization. The use of visual feedback to adjust movement trajectory is known as a closed-loop system (see: Elliott, Helsen, & Chua, 2001). While feedback control is generally agreed to subservise accurate performance, it is significantly influenced by the transmission and processing delay (Elliott & Allard, 1985; Keele & Posner, 1968; Zelaznik et al., 1983; see Elliott and Khan 2010 for a comprehensive review). As a result, most current positions posit the temporal overlap of open and closed-loop phases, although the relative contributions and timings remain controversial (see: Plamondon, 1997).

Although visual feedback is necessary for accurate trajectory corrections, when we know visual feedback may not be available, we prepare for a 'worst-case scenario' (Elliott, Hansen, Mendoza & Tremblay, 2004; Hansen, Glazebrook, Anson, Weeks, & Elliott, 2006). Typically, more time is spent planning the movement and therefore reaction times are slower. When vision is known to be available, planning and control becomes much more efficient. Hansen et al. (2006) suggest that pre-cuing the target location is more important than knowing whether or not you will have visual feedback. That is, the visual information given prior to the movement can

be used to execute the movement in the absence of vision. However, in order to most effectively plan and control a movement, we must have vision of the target and the limb (Proteau & Carnahan, 2001).

1.4 Proprioceptive Feedback

In addition to vision, proprioceptive feedback also assists in movement planning and control. Proprioceptive information is relayed to the brain through mechanoreceptors in the muscle and joints, and stretch receptors in the skin (Kandel, Schwartz, & Jessel, 2000). These receptors provide a sense of where the body is within the environment and relative to itself without having to use vision. The mechanoreceptors give a general sense of where the limb is in space, both when stationary (limb-position sense), and during movement (kinesthesia). This helps control the speed and accuracy of aiming movements. Specifically, sensory information is received from three types of mechanoreceptors: muscle spindles, joint capsule receptors, and Golgi tendon organs. Muscle spindles are activated by nerves innervating muscles and serve to relay information about changes in muscle length. This input is interpreted by the brain as information about stretch and tension occurring in the muscle. Located in the muscles themselves, muscle spindles provide information about relative joint angles and body segment position. Joint capsule receptors also provide information about joint angles, through sensing movements such as flexion and extension. Golgi tendon organs provide information about contractile force and therefore help control grasp. These mechanoreceptors lie in the muscle tendons at the attachment of the muscle belly and are a continual source of information on tension of contracting muscles. Tactile receptors in the skin also provide input about stretch, as well as postural information.

1.5 Neuroanatomy

As detailed in Kandel et al. (2000), our visual perception of an object or environment is

comprised from overlapping visual fields projecting onto the retinas. While central vision projects to both retinas, images from the right visual field project onto the temporal half of the left retina (left temporal hemiretina) and the nasal half of the right retina (right nasal hemiretina), and vice versa. Images from the periphery project solely to the nasal hemiretina of the ipsilateral eye. Exiting the retina via the ganglion cells, visual information then travels along the optic nerve until it reaches the optic chiasm. Here, fibers from the temporal hemiretinae continue ipsilaterally, while fibers from the nasal hemiretinae cross over and continue contralaterally, generating left and right branches of the optic tract. This crossing over of optic nerve fibers means that the right optic tract carries input from the left visual field, and the left optic tract carries input from the right visual field.

Both optic tracts project this visual information to the brain, specifically the pretectum and superior colliculus in the midbrain, and the lateral geniculate nucleus in the thalamus. The pretectum controls papillary reflexes responsible for dilating and constricting the pupil in response to different light conditions; the superior colliculus controls the rapid eye movements to multiple fixations within the visual field known as saccades. The majority (90%) of visual input is sent to the lateral geniculate nucleus, which is then relayed to the primary visual cortex (V1) in the occipital lobe.

1.6 Visual Processing: Dorsal and Ventral Streams

Our understanding of what happens to this visual input on reaching V1 has developed over several decades and, to a large extent, is still up for discussion. At the core, however, lies a general understanding that there exists an anatomical separation between information processed about an object's identifying features and its location. Schnieder (1968) was the first to suggest this division, postulating the retinotectal pathway (from the retina to the superior colliculus) to be responsible for processing object location, and the geniculostriate pathway (from the lateral

geniculate nucleus to V1) for processing object identification. Though no longer an accepted hypothetical system, Schnieder's 1968 model paved the way for that of Ungerleider and Mishkin (1982) that assigned these roles to the posterior parietal cortex, and the inferior temporal cortex, respectively. Each region was thought to receive separate information streams from V1, which lead to the development of the ventral and dorsal streams: ventral carries identification information and terminates in the inferotemporal cortex; dorsal carries spatial information and terminates in the posterior parietal region (Ungerleider & Mishkin, 1982).

Milner and Goodale (1992) elaborated on this dual-stream model to consider perception of an object, and the output processing for actions related to that object. Visual information at V1 is further processed through the two cortical pathways: dorsal and ventral (see Goodale & Milner, 1992; Milner & Goodale, 2006 for review). The dorsal cortical pathway carries visual information pertaining to movement and object location from V1 through the secondary visual cortex (V2) to the middle temporal area. From here, the information is sent to the posterior parietal cortex and used to plan movement and control the limb and eye movements involved in executing it. The ventral cortical pathway also moves from V1 through V2, on its way to visual area V4 in the extrastriate cortex and finally the inferior temporal cortex. Ventral stream processing typically deals with information related to object form (i.e. colour, shape, orientation) and recognition. Together, these pathways allow us to recognize objects in the environment and create the desired actions to interact with them.

Glover (2004) has since refined function of the dorsal stream, suggesting a sub-division where separate systems exist to process information for planning and for control. Planning is undertaken prior to movement initiation by the inferior parietal lobe using a visual representation, and overlaps with the onset of the control phase, which gradually exudes influence on the movement as it progresses via the superior parietal lobe using visual feedback

for comparison to the original movement plan (Glover, 2004).

1.7 Neuroplasticity

Pascual-Leone describes cortical plasticity as a structural and functional reorganization of the nervous system, typically occurring in response to development, experiences, environmental pressures, or damage to the brain (Merabet & Pascual-Leone, 2010). Vision loss can promote plasticity by causing areas of the brain normally associated with vision to be recruited by the remaining senses (Merabet & Pascual-Leone, 2010). Vision loss is thought to increase demand on other senses in order to functionally adapt to blindness. Blind participants must use other modalities, such as touch and hearing, for spatial information when vision is unavailable (Pascual-Leone & Hamilton, 2001).

Consistent with this functional reallocation, plasticity in blind participants has been purported to enhance their tactile discrimination capacity over that of sighted participants (Merabet & Pascual-Leone, 2010). Sighted participants blindfolded for five days have shown increased tactile performance on a Braille character discrimination task over non-blindfolded, sighted participants (Kauffman, Théoret, & Pascual-Leone, 2002). This increased tactile performance of blindfolded, sighted participants was attributed to plasticity caused by a lack of visual input; previously masked existing neural connections in the visual cortex were recruited and used instead for tactile discrimination (Kauffman et al., 2002).

Hamilton & Pascual-Leone (1998) suggest that in order to read Braille, significant cortical changes must take place such that tactile acuity is enhanced enough to detect finite differences in stimuli. This plasticity has been evidenced by observed enlargement of cortical sensorimotor areas linked to the Braille reading finger, relative to the same areas in normal sighted participants (Pascual-Leone & Torres, 1993). Additionally, it seems that in the blind the occipital cortex can be recruited for tactile processing. In the early blind, V1 becomes active

during tactile discrimination tasks, such as Braille reading (Sadato et al., 1996). Furthermore, repetitive transcranial magnetic stimulation (rTMS) to this region during Braille reading distorts the tactile representation of the Braille letters and increases error rates in reading success (Cohen et al., 1997).

While these studies provide evidence of cortical plasticity in response to vision loss, they do so only with respect to the tactile sensory modality. Little is known about what plastic changes occur related to movement when we encounter sudden or acute vision loss. This information is vital to how we approach rehabilitation techniques for varying degrees and onsets of blindness, from ophthalmic surgery patient care to traumatic vision loss. By simulating acute vision loss in normal-sighted individuals, we may observe changes in how people interact with their environment. These behavioural changes could provide insight into learning strategies that best facilitate adaptation to vision loss. Additionally, this information could be relevant to rehabilitation of congenitally blind individuals with treatable conditions who have recently regained vision.

1.8 Research Question

As previously stated, without vision, we rely on proprioception to provide us with the necessary feedback to plan and coordinate movements. Proprioception may be the key to how we navigate our environment when vision is unavailable. Therefore, the present research aims to determine the role of proprioception in motor tasks when vision is absent, and what changes in the brain may result from vision loss with respect to proprioception. Experiment I served as a pilot study and sought to determine how visual and proprioceptive information is used in the planning and control of movement following acute onset short-term vision loss. Experiment II was designed to elaborate on these findings following long-term vision loss. An electroencephalography (EEG) task was introduced to assess how any resulting plasticity is

reflected in behaviour and neural activity. I hope my findings will provide insight as to what is experienced by the brain in the event of acute vision loss. Any observed adaptation may provide information that can be integrated into how we approach rehabilitation of varying degrees and onsets of blindness.

1.9 Hypotheses

This study is comprised of two experiments. Experiment I simulates short-term (2 hours) vision loss, while Experiment II simulates long-term (8 hours) vision loss to approximate a ‘first day’ experience. In Experiment I, it was my aim to simulate an ‘early-onset’ vision loss scenario similar to what someone experiences in the first hours of vision loss. I hypothesized that performance of tactile discrimination tasks while blindfolded for 2 hours would promote onset of proprioceptive plasticity. I further expected a larger contribution of proprioceptive information in response to the period of visual deprivation, and that this would have a synergistic effect on the use of vision when it became available in post-tests.

In Experiment II, the aim was to extend our findings into a long-term vision loss scenario. I hypothesized that a prolonged period of simulated vision loss (8 hours) would promote further plasticity in the brain and thereby enhance the observed results of Experiment I. I expected behavioural changes in the control of reaching and grasping reflective of this plasticity and any concurrent use of vision and proprioception. Additionally, I predicted sensitivity to tactile stimuli to increase, but decrease to visual stimuli in response to prolonged lack of visual input to the brain. Significant behavioural and cortical changes would facilitate a better understanding of the underlying structures contributing to visual and proprioceptive control of reaching and grasping movements.

CHAPTER 2: EXPERIMENTS

Previous studies have investigated neural changes in response to long-term vision loss over a matter of days (Kauffman et al., 2002; Pascual-Leone & Hamilton, 2001), or specific to auditory or tactile processing (Weisser, Stilla, Peltier, Hu, & Sathian, 2005). The effect of early phases of vision loss specifically related to movement, however, is not known. The goal here was to look at the first-day experience of acute vision loss and how this alters processing of visual and proprioceptive information. Two experiments were employed to separate what changes take place in the early stages of vision loss from those that happen in the long-term.

In other studies examining proprioception for movement, it is common that some visual pre-cue to the target is presented during or prior to the task, likely generating a visual memory representation of that target location (Elliott, 1988). This visual memory can be used as a proprioceptive reference on a subsequent trial where vision is unavailable, and therefore as a source of information contributing to the movement. These studies, then, lack a true assessment of proprioception as it is confounded by the generated visual memory. In order to limit the formation of such visual memories, an assisted passive movement was used to orient participants to the target position without giving them a visual or spatial cue (Paillard and Brouchon, 1974). This method was tested in Experiment I and retained for Experiment II.

In both experiments, post-deprivation behavioural changes during the movement were taken as indices of proprioceptive or visual influence on movement planning and control. The same reaching and grasping task was used in both experiments to investigate any differences in behaviour between post- 2-hour and 8-hour data, which may provide insight to the progression of any observed adaptation.

2.1 Experiment I

2.1.1 Background

In Experiment I a 2-hour visual deprivation was used to investigate the role of proprioception in planning and controlling reaching and grasping movements when vision is unavailable. The central aim was to assess if a 2-hour period of visual deprivation was enough time to promote plastic changes, and how that would spur adaptation in movement behaviours. Kinematics of reaching and grasping were assessed during trials where vision was available, and trials where vision was unavailable, both pre- and post-deprivation. During the deprivation period, a series of tasks were performed in an attempt to stimulate plasticity related to tactile discrimination and serve to enhance proprioceptive environmental cues. As mentioned previously, Braille reading tasks are often associated with encouraging tactile plasticity. Other forms of tactile discrimination, such as piano playing, also stimulate plasticity. Spatial differentiation thresholds in musicians are observed to be lower than those of non-musicians, and correlated to the amount of time practicing the tactile skill (Ragert, Schmidt, Altenmüller, & Dinse, 2004).

The prediction was that, just as the proprioceptive system is relied on when vision is absent during a trial, proprioception would be favoured following the 2-hour visual deprivation. In post-deprivation trials where vision is unavailable, an up-regulation of proprioceptive processing could improve movement planning efficacy. This up-regulation may also enhance or even supersede the processing of visual information for planning and controlling the movement in post-deprivation trials where vision was available for the duration of the movement.

2.1.2 Methods

2.1.2.1 Participants

Ten participants (5 male, 5 female, mean age: 24 ± 3.0) from the University of British Columbia Okanagan campus participated in this study. All participants were self-reported right-hand dominant with normal or corrected to normal, 20/20 visual acuity. This study was approved by the University of British Columbia Research Ethics Board and all participants provided written informed consent prior to participating in the study.

2.1.2.2. Apparatus

Participants were seated at a table with three marked positions: Home (edge), Short (20cm), and Long (35cm). Infrared emitting diodes (IREDS) were fitted to the subject's right index finger and thumb, with a third IRED affixed to the knuckle of the second metacarpal bone. The table was oriented such that the subject faced an OptoTrak 3020 (Northern Digital, Inc.). Liquid crystal goggles (Translucent Technologies, Inc.) were used to remove visual input when necessary.

2.1.2.3 Stimuli

Four possible target stimuli were presented to the subject, comprised of two target sizes and two target distances. For each trial, participants reached to either a Small (5cm diameter) or Large (7cm diameter) section of black PVC pipe, 4cm in height. The target was placed at either the Short (20cm) or Long (35cm) location on the table by an experimenter. Target position and size were randomized using a custom MatLab code such that 10 trials of each possible combination were completed, for a total of 40 trials per block.

2.1.2.4 Design

Two blocks of reaching and grasping trials were performed pre and post 2-hour deprivation. The first block was always the no vision (NV) condition, and the second always the

full-vision (FV) condition. Both blocks were performed pre- and post-deprivation, in the same order (NV then FV) in order to assess behavioural changes immediately on regaining vision when the post-test was performed. Participants completed 160 Good trials in total; bad trials (i.e.: missed IREDs, failure to contact target, $150 < RT > 500\text{ms}$) were marked in the data output and re-randomized into the trial conditions list.

2.1.2.5 Procedure

Prior to the initiation of each trial, participants rested their hand at the home position and were instructed to completely relax the arm while the experimenter moved their arm and hand to the placed target and back to the home position. On returning to the home position, the trial was initiated, and following a 2000ms delay, an auditory tone sounded (2500Hz). Participants were instructed to move as quickly and accurately as possible to the target in response to this tone, then grasp the target between the thumb and forefinger, lift it slightly from the table surface, then return their hand to the home position. In the first block (NV), vision was removed for the duration of the block. In the second block (FV), vision was available from 2000ms prior to the auditory tone to movement end, with vision removed between trials while the target was placed and during the passive arm movement.

During the deprivation period, participants completed tactile discrimination tasks to help promote proprioceptive plasticity. These tasks included a block sorting task, and a block-building task. The sorting task was comprised of three timed trials performed twice during the deprivation, once at the beginning and again just prior to the end. Participants were instructed to match, by feel, 6 different block shapes to their corresponding shaped holes on a bucket lid and drop them through. The building task involved mirroring pre-built Lego shapes on a Lego building surface. Participants had to use the circular projections on the Lego board to determine

positions and on the blocks to determine shapes. There were two tasks with shapes of increasing difficulty, and participants were timed per shape.

2.1.2.6 Recording and Analysis

Kinematic data were recorded from the IREDs using the OptoTrak 3020 (Northern Digital, Inc.) at a sampling frequency of 200Hz. These data provided the dependent variables for each condition for further analysis: reaction time, movement time, peak grip aperture, grip aperture at percent of movement deciles, time after peak grip aperture, and time after peak velocity. A 2(Pre, Post) X 2(No Vision, Vision) X 2(Small, Large) X 2(Long, Short) repeated measures analysis of variance (ANOVA) was conducted on all dependent variables with the exception of those including deciles. For these analyses, a 2(Pre, Post) X 2(No Vision, Vision) X 2(Small, Large) X 2(Long, Short) X 11(Deciles 0% to 100%) repeated measures ANOVA was run. Using a simple main effects approach, interactions involving two or more means were deconstructed. Post-hoc pairwise comparisons were then conducted using one-way ANOVA with Bonferroni correction for multiple comparisons.

Variability of limb position at each decile percentage of movement amplitude in the y-axis was correlated to movement endpoint using correlation coefficients (R). Proportion of explained variance (R^2) was calculated between movement endpoint and percent of movement trajectory (i.e. decile percentage of movement amplitude in the y-axis) to provide insight to the planning and control of the movement (see: Elliott, Binsted, & Heath, 1999; Heath, 2005; Heath, Westwood, & Binsted, 2004; Messier & Kalaska, 1999). This method assesses how predictive limb position at one decile is of the movement endpoint; it measures the proportion of variability in the movement endpoint explained by a prior position within the movement. Heath (2005) suggests that movements executed purely based on planning should carry significant R^2 values throughout the movement. This would mean that online error corrections are not being made,

and the movement is proceeding based on how it was originally planned. However, if online control were being used to correct movement errors, one would expect to see little or no relationship between limb position during movement and eventual endpoint.

2.1.3 Results

Reaction time (RT) analysis yielded two significant main effects: Vision $F(1,9) = 10.89$, $p < .05$, with RT significantly faster when vision was available (means: FV = 307.38ms; NV = 326.97ms); and Target Distance $F(1,9) = 6.21$, $p < .05$, where RT to nearer targets was significantly faster than to far (means: Short = 310.45ms; Long = 323.90ms). There was a 3-way interaction involving Pre-Post, Vision, and Target Distance $F(1,9) = 6.03$, $p < .05$. Breaking down the interaction showed a main effect of Target Distance $F(1,9) = 5.70$, $p < .05$ present in Pre but not in Post; and a main effect of Vision $F(1,9) = 21.83$, $p < .05$ present in Post but not in Pre (Figure 1). The interaction then was driven by Target Distance in the Pre-test, where RT was faster to nearer targets than far (means: Pre, Short = 320.42ms; Pre, Long = 334.98ms), and Vision in the Post-test, where RT was faster when vision was available than when not (means: Post, V = 291.66ms; Post, NV = 321.65ms).

For movement time (MT), main effects were also observed in Vision $F(1,9) = 13.47$, $p < .05$, and Target Distance $F(1,9) = 114.83$, $p < .05$. MT was faster when vision was available (Figure 2; means: V = 750.79ms; NV = 886.28ms) and to nearer targets (means: Short = 735.81ms; Long = 901.26ms). Time after peak velocity (TAPV) data showed main effects of Vision $F(1,9) = 13.98$, $p < .05$, and Target Distance $F(1,9) = 75.16$, $p < .05$. More time was spent from peak velocity to movement end when vision was unavailable (means: NV = 531.43ms; V = 425.24ms) and when targets were further away (means: Long = 543.32ms; Short = 413.35ms). Both of these results were related to the main effects of Vision and Target Distance observed

with respect to MT, as greater TAPV in NV and to further targets contribute to an overall increase in MT.

Analysis of peak grip aperture (PGA) showed main effects of Vision $F(1,9) = 61.99, p < .05$, and Target Size $F(1,9) = 16.31, p < .05$. PGA was significantly narrower when vision was available, more closely approximating actual target size (Figure 3; means: V = 98.39mm; NV = 106.70mm). Target Size also influenced PGA, with a significantly smaller PGA adopted when reaching to Small targets, and significantly larger when reaching to Large targets (means: Small = 98.22mm; Large = 106.88mm). A two-way interaction of Vision and Target Size $F(1,9) = 13.21, p < .05$ revealed significantly larger PGA when grasping Large targets than Small in both visual conditions, and significantly larger grip PGA adopted when vision was unavailable, regardless of Target Size (means: NV, Large = 109.97mm; NV, Small = 103.43mm; V, Large = 103.78mm; V, Small = 93.00mm).

Time after peak grip aperture (TAPGA) analysis revealed main effects of Vision $F(1,9) = 15.21, p < .05$, and Target Distance $F(1,9) = 53.06, p < .05$. Less time was spent after PGA when vision was available (means: V = 305.64ms; NV = 409.67ms), and to nearer targets (means: Short = 317.39ms; Long = 397.91ms). A two-way interaction between Vision and Pre-Post $F(1,9) = 5.57, p < .05$ revealed main effects of Vision in Pre, $F(1,9) = 21.11, p < .05$, and Post $F(1,9) = 7.97, p < .05$, but no main effect of Pre-Post (Figure 4).

Decile analysis (i.e. percent of movement execution) of grip aperture (GA) in all trials showed main effects of Vision $F(1,9) = 29.95, p < .05$, Target Size $F(1,9) = 19.14, p < .05$, and Decile $F(10,90) = 99.45, p < .05$. Similar to PGA, larger GA was used when vision was unavailable (means: NV = 74.60mm; FV = 67.57mm) and when reaching to Large targets (means: Large = 74.36mm; Small = 67.81mm). The main effect of Decile reflects the natural

progression of grip aperture throughout movement, therefore GA is significantly different at each decile from the preceding decile, with the exception of the 70% to 80% decile (Table 1).

Two-way interactions were found between Vision and Decile $F(10,90) = 10.01, p < .05$, and Target Size and Decile $F(10,90) = 14.99, p < .05$. Decile-by-decile analysis of these interactions showed a significant effect of Vision where GA was significantly larger when vision was unavailable from 10% to movement end, and a significant effect of Target Size where GA was significantly smaller to Small targets from 20% to movement end (Table 1). A three-way interaction was observed between Vision, Target Size, and Decile $F(10,90) = 2.45, p < .05$, with further analysis showing a significantly larger grip apertures when reaching to Large targets from 60% to movement end when vision was unavailable, and from 20% to movement end when vision was available (Table 2).

Due to the repeated trial nature of the experiment and a necessity to retain block order (i.e. blocks could not be counterbalanced), there exists a possibility of learning effects related to the task that could wash out any post-deprivation effects. To combat this, further analysis of GA over movement deciles was conducted solely on the last 5 trials of each Pre-test block, and the first 5 trials of each Post-test block. Main effects of Vision $F(1,9) = 7.11, p < .05$, and Decile $F(10,90) = 118.81, p < .05$ were observed again, as in analysis of all GA deciles. A significant two-way interaction was observed between Pre-Post and Decile $F(10,90) = 2.26, p < .05$, however further analysis showed no within-decile significance of Pre-Post. A two-way interaction between Vision and Decile $F(10,90) = 6.15, p < .05$ similar to that of the complete GA decile analysis was found, with significance in the 30% to 70% movement deciles. As this analysis method of GA revealed no novel result beyond that of the original analysis of all GA deciles, further discussion and analysis considered all GA deciles.

Analysis of the proportion of explained variance (R^2) calculated between movement endpoint and each movement decile yielded main effects of Decile $F(10,90) = 195.68, p < .05$, and Vision $F(1,9) = 17.68, p < .05$. Decile effects reflect on the natural change in correlation with movement endpoint through each 10% of movement progression, while the effect of Vision reflects greater correlation of limb position to movement endpoint when vision is unavailable (Figure 5). Two-way interactions were observed between Decile and Vision $F(10,90) = 5.87, p < .05$, and Decile and Target Distance $F(10,90) = 1.98, p < .05$. Further analysis of the two-way interactions showed a significant effect of Vision where limb position was more strongly correlated with endpoint from the 20% decile until movement end when vision was unavailable (Figure 5), and a significant effect of Target Distance where at 60% and 70% movement, limb position was more strongly correlated to movement endpoint when reaching for nearer targets than to far (R^2 means: 60%, Short = 0.51; 60%, Long = 0.39; 70%, Short = 0.69; 70%, Long = 0.59) as a result of being nearer to movement end.

Standard deviations of limb position were used to calculate variability within each decile. Analysis of these data showed main effects of Decile $F(10,90) = 25.86, p < .05$, Vision $F(1,9) = 10.93, p < .05$, and Target Distance $F(1,9) = 12.15, p < .05$. The Decile main effect reflects on the natural change in variability of limb position through each 10% of movement progression (Figure 6). The main effect of vision was expected, as variability of limb position was more significant when visual feedback was unavailable (Figure 6). Main effects of Target Distance show significantly less variability in limb position overall when reaching to nearer targets than far (means: Short = 13.82mm; Long = 17.58mm).

A two-way interaction between Decile and Vision $F(10,90) = 2.81, p < .05$ showed a significant effect of Vision where variability was significantly greater when vision was unavailable from the 40% movement decile to movement end (Figure 6). A two-way interaction

between Decile and Target Distance $F(10,90) = 12.82, p < .05$ again showed significantly less variability in limb position when reaching to nearer targets than far, specifically from 10% to 60% movement (Table 3).

2.1.4 Discussion

Main effects of vision in RT, MT, and PGA fit our expectations of how the visual system alters movements when vision is unavailable. We see increased reaction times, movement times, and peak grip aperture sizes consistent with generating a more conservative movement in response to a lack of visual input, thereby increasing likelihood of target contact (Figures 1-3). Significant effects of Target Distance in RT and MT are also predictable; RT and MT are both faster to nearer targets, and slower to those that are further away. Main effects in TAPV mirror those found in MT; more time spent after peak velocity in NV and to further targets explains why we see longer MT in these conditions as well.

MT, TAPV, and PGA main effects are consistent Pre and Post 2-hour visual deprivation, which suggests that despite this acute restriction of visual input, these aspects of general reaching and grasping behaviour are unaffected. However, the interaction observed between Pre-Post, Vision, and Target Distance in RT provides some evidence of the task learning we would expect to see given the repeated trial nature of the experiment. In the Pre-test RT is still faster to nearer targets than to far, but is generally slow regardless of visual condition, suggesting overall poorer performance of the task initially. Post-deprivation, only visual condition affects RT; RT is significantly faster in FV than in NV, with no effect of Target Distance. Following the deprivation, participants may have a previously internalized a proprioceptive representation of target distance to use for planning their movements; therefore they are only affected by a lack of visual input for controlling movement in Post-test NV trials (Figure 1).

Overall, a larger grip aperture is used to approach larger targets, and a smaller aperture is adopted when reaching to smaller ones (Figure 3). The interaction of Vision and Target Size showed that while this strategy is also used in the NV condition, grip aperture is greater in NV to both target sizes relative to those in FV. TAPGA also follows this pattern; more time is spent after PGA in NV and provides further evidence of a conservative grasping strategy (Figure 4). This strategy is maintained Pre and Post-deprivation, however the difference in TAPGA between NV and FV is significantly smaller Post-deprivation. As PGA tends to occur later in movement, changes in time spent after PGA is likely indicative of changes in control (Glover, 2004).

Although not a significant result on its own, it seems that Post-deprivation, more time is spent post-PGA in FV relative to Pre-deprivation, even though the visual information available is unchanged Pre-to-Post. This may suggest a shift in feedback use favouring proprioceptive information in planning the movement, resulting in more time spent controlling the movement in its final phases.

Decile analysis of grip aperture was performed to highlight any differences Pre-Post in NV and FV conditions, specifically post-PGA at approximately 70% movement (Jakobson & Goodale, 1991). Unfortunately, this analysis did not reveal any novel differences pre- and post-deprivation, but did further confirm retention of expected reaching and grasping behaviours in NV and FV. Further grip aperture decile analysis was performed on the last five trials of each Pre-test condition and first five trials of each Post-test condition to overcome any wash-out effects of multiple trials incurred post-deprivation. Despite an interaction of Pre-Post and Decile, further analysis showed this was likely driven by a main effect of Decile and not by Pre-Post differences. Neither analysis provided insight into where and what changes might be taking place post-PGA.

Correlation (R^2) and variability analyses also did not yield Pre-Post differences in reaching and grasping behaviour. Correlation analysis showed higher R^2 values in NV (Figure 5), reflecting a dependence on planning to execute the movement in the absence of visual feedback to make online corrections (Heath, 2005). Lack of Pre-Post differences in R^2 suggests that planning and control phases of the movement are active at relatively the same decile of movement, pre- and post-deprivation. While planning and control parameters may be changing relative to RT and TAPGA, they are still operating at the expected phases of movement. Variability analysis using standard deviations of limb position within each decile showed an increased variability of limb position in NV from 40% movement onwards (Figure 6). This specific decile shows a shift from dependence on the planned movement to controlling the movement using visual feedback in FV.

Overall, our data confirms considerable differences in goal directed movements executed with and without vision. Pre-Post consistencies of these data suggest that the 2-hour visual deprivation did not affect reaching and grasping behaviour on the whole, and that participants are still performing the action in the same general fashion. However, subtle differences in TAPGA may be the first indication of greater change occurring in the way movements are planned and controlled. Correlation analysis data suggests that the timing of planning and control phases remain the same Pre-Post. Given this, it is not necessarily a case of planning or control operating for differing periods of time. It is more likely that post-deprivation differences in TAPGA are reflecting a change in how the information available is being used.

Typically, available visual information is used predominantly over proprioceptive information (Heath, 2005); here we are seeing a trade-off in the use of visual information for proprioceptive information to plan a movement. This may reflect a shared mechanism operating to pick and choose which information will be used dominantly, and suggests that this mechanism

may be susceptible to sensory perturbations such as vision loss. In order to further investigate this mechanism and its operation, we move to an 8-hour visual deprivation to further delineate visual and proprioceptive input to movement planning and control.

2.2 Experiment II

2.2.1 Background

The pilot work of Experiment I showed limited evidence of a potential trading-off of visual and proprioceptive information processing in movement control following two hours of visual deprivation. The aim of Experiment II was to further delineate this trade-off by extending the bout of deprivation to 8-hours. I hypothesized that the longer, 8-hour bout of vision loss would further facilitate any plastic changes occurring. Behavioural changes in the control of reaching and grasping then would reflect this plasticity and any concurrent use of vision and proprioception. It was predicted that this plasticity would also be seen as neurological changes evidenced by an electroencephalography (EEG) task, specifically in decreased sensitivity to visual stimuli but increased sensitivity to tactile stimuli. Simply using tactile navigation of daily activities has been suggested to influence behavioural changes following long-term visual deprivation (Kauffman et. al, 2002). As such, instead of completing the tactile discrimination tasks performed in Experiment I, participants went about their normal daily activities with the help of an experimenter when necessary.

Based on the known occurrence of plastic changes in response to prolonged sensory deprivation (See: Merabet & Pascual-Leone 2010; Pascual-Leone, Amedi, Fregni, & Merabet, 2005; Pascual-Leone & Hamilton, 2001), an EEG protocol was employed to measure any changes in neural activation post-deprivation. An oddball detection task (adapted from Eimer, Cockburn, Smedley, & Driver, 2001) was employed to elicit event related potentials (ERPs) in the brain in response to the detection of infrequent tactile and visual stimuli. Differences in these ERPs following the deprivation would highlight changes to oddball stimuli detection ability (i.e.: changes to tactile and visual processing), and could be used to determine more specific regions of where these changes occur through cortical topography. It was hoped that changes in ERP

latencies and amplitudes following 8-hours of visual deprivation would help to determine when and where these plastic changes actually begin to take place.

2.2.2 Methods

2.2.2.1 Participants

Twelve participants (7 male, 5 female, mean age: 25 ± 4.1) from the University of British Columbia Okanagan campus participated in this study. All participants were self-reported right-hand dominant with normal or corrected to normal, 20/20 visual acuity. This study was approved by the University of British Columbia Research Ethics Board and all participants provided written informed consent prior to participating in the study.

2.2.2.2 Apparatus

Behavioural:

Participants were seated at a table with three marked positions: Home (5cm from edge), Short (20cm from Home), and Long (35cm from Home). The table and subject faced an OptoTrak Certus (Northern Digital, Inc.) motion capture camera. IREDS were attached to the subject's right index finger, thumb, and knuckle of the second metacarpal bone. Liquid crystal goggles (Translucent Technologies, Inc.) were used to remove visual input when necessary.

EEG:

Participants were fitted with an EEG cap (Brain Products, Inc.) and seated with their arms resting on a table at either side of a central fixation cross located 25cm from the table edge. For the tactile ERP task, vibration motors encased in plastic tubing were taped to both index fingertips. To mask any mechanical noise from the motors, white noise was presented through a speaker directly in front of the subject. For the visual ERP task, green light emitting diodes (LEDs) were taped to the dorsal side of both index fingertips.

2.2.2.3 Stimuli

Behavioural:

Four possible target stimuli were presented to the subject, comprised of two target sizes and two target distances. For each trial, participants reached to either a Small (5cm diameter) or Large (7cm diameter) section of black PVC pipe, 4cm in height. The target was placed at either the Short (20cm) or Long (35cm) location on the table by an experimenter. Target position and size were randomized using a custom MatLab code such that 10 trials of each possible combination were completed, for a total of 40 trials per block.

EEG:

In the tactile condition, we sought to elicit somatosensory evoked potentials (SSEPs) in response to vibration of the index fingertips. Each block consisted of 100 vibration trials, where vibration stimuli were presented for 50ms. In each block, the probability of non-target (non-oddball) stimuli was 0.8, and the probability of target (oddball) stimuli was 0.2. Prior to the initiation of each block, participants were told which hand was more likely to receive the stimulus; these trials comprised the non-target stimuli. A stimulus delivered to the hand opposite these instructions was denoted a target stimulus.

In the visual condition, we looked at visual evoked potentials (VEPs) in response to a 10ms illumination of a green LED on the dorsal surface of the index fingertips. The number of trials per block and stimulus probabilities within each block was the same as the tactile condition. Participants were again instructed as to which hand the stimulus would more frequently appear.

2.2.2.4 Design

All participants completed a Control and an Experimental day. To protect against order effects, day condition was counterbalanced among participants. On both days, participants

completed a pre- and post-test surrounding an 8-hour time period. On the Control day, this 8-hour period was spent doing normal daily activities with full vision; on the Experimental day, visual input was removed using translucent orthoptic eye patches (Nexcare, 3M Inc.) for the duration of the 8-hour period. The pre- and post-test were comprised of the same tasks, performed in the same order (i.e. No Vision then Full Vision). Each subject participated in a behavioural reaching and grasping task, and an oddball detection task with EEG recording pre and post.

Behavioural:

Two blocks of reaching and grasping trials were performed pre and post 8-hour deprivation. The first block was always the NV condition, and the second always the FV condition. Block order was consistent pre and post-deprivation. Participants completed 160 Good trials in total; Bad trials (i.e.: missed IREDs, failure to contact target, $150 > RT > 500\text{ms}$) were marked in the data output and re-randomized into the trial conditions list.

EEG:

Within the tactile and visual conditions, there were two blocks: one where the stimulus was more often presented to the right hand, and the other more often to the left hand. Condition order was counterbalanced across all 12 individuals. Participants performed the same condition and block order on all four testing occasions. Any Bad trials (i.e.: missed stimuli, blinking, yawning, sneezing) were marked in the EEG output and re-randomized into the trial list using custom MatLab code. 400 total Good EEG trials were completed.

2.2.2.5 Procedure

Behavioural:

Prior to the initiation of each trial, participants rested their hand at the home position and were instructed to completely relax the arm while the experimenter moved their arm and hand to

the placed target and back to the home position. On returning to the home position, the trial was initiated, and following a 2000ms delay, a DC-6V Piezoelectric buzzer sounded for 100ms. Participants were instructed to move as quickly and accurately as possible to the target in response to the buzzer, then grasp the target between the thumb and forefinger, lift it slightly from the table surface, then return their hand to the home position. In the first block (NV), vision was removed for the duration of the block. In the second block (FV), vision was available from 2000ms prior to the auditory tone to movement end, with vision removed between trials while the target was placed and during the passive arm movement.

EEG:

Participants started with either vibration motors or LEDs taped to both index fingers, and the arms resting at the edge of the table, about 20cm on each side of the fixation cross. For the vibration blocks, the hands were supinated to prevent motor contact with the table surface. The hands were pronated during the visual blocks such that the LEDs on the dorsal fingertips were easily visible. Prior to each block, the subject was notified which hand the stimulus would be more frequently presented to. For each trial, the stimulus was presented, followed by a 1000ms pause and a brief auditory tone (6000Hz). Participants were instructed to respond on this tone with “left” or “right”, indicating which hand they felt the stimulus on. Trials were presented in rapid succession with the subject blinking between trials (while responding).

2.2.2.6 Recording and Analysis

Behavioural:

Kinematic data were tracked and recorded from the IREDs using an OptoTrak Certus (Northern Digital, Inc.) at a sampling frequency of 200Hz. Dependent variables used for analysis included: reaction time, movement time, peak grip aperture, grip aperture at percent of movement deciles, time after peak grip aperture, and time after peak velocity. A 2(Control,

Experimental) X 2(Pre, Post) X 2(No Vision, Vision) X 2(Small, Large) X 2(Long, Short) repeated measures analysis of variance (ANOVA) was conducted on all dependent variables, with the exception of those including deciles. For decile analyses, a 2(Control, Experimental) X 2(Pre, Post) X 11(Deciles 0% to 100%) X 2(No Vision, Vision) X 2(Small, Large) X 2(Long, Short) repeated measures analysis of variance (ANOVA) was run. Variability of limb position and proportion of explained endpoint variance were again correlated to decile percentages of movement amplitude in the y-axis. Pairwise post-hoc comparisons with Bonferroni correction were conducted on effects involving more than 2 means using one-way ANOVA. A simple main effects approach was used to decompose interactions prior to pairwise comparisons. Higher order effects with explained variance less than 1% following omega squared (ω^2) analysis will not be discussed, but are presented in Table 4.

EEG:

EEG was recorded from a custom electrode workspace of 15 Ag/AgCl scalp electrodes using a BrainVision amplifier (BrainProducts, Germany) and EasyCap 10-20 system (EasyCap, Germany). Scalp electrodes included were: Fpz, Fz, AFz, Fcz, IO, Fp2, Cz, C3, C4, Pz, P3, P4, Oz, O1, O2. Fp2 and IO electrodes were used to record the electro-oculogram (EOG). All electrode impedances were $\leq 5k\Omega$, and the amplifier bandpass was 0.1-40Hz with a 60Hz notch filter. Both EOG and EEG measures were sampled at 500Hz and recorded using BrainVision Recorder software (BrainProducts, Germany).

Analyses of EEG data were conducted using BrainVision Analyzer software (BrainProducts, Germany). All data were segmented into 1200ms epochs, starting 200ms prior to and ending 1000ms after stimulus onset. All trials were corrected to a baseline measure from 200ms prior to stimulus onset. Eyeblink trials were removed with a Gratton-Coles ocular correction algorithm. Other artifacts (i.e. voltages greater than $50\mu V$ or less than $0.5\mu V$) were

also removed from the analysis. Vibration trials were averaged within hand condition (right or left) and separate of LED trials, which were also averaged within hand condition. Additionally, within each of these averages, oddball stimuli were averaged separately of non-target stimuli. Secondary analysis provided a grand average of each of these conditions for pre- and post-deprivation bouts on both Control and Experimental days, for a total of 32 grand averages.

From the averaged ERP waveforms, four peaks were used for analysis: P1, N1, P2, N2, and P3, where P and N indicate positive (downward-going) and negative (upward-going) peaks, respectively. P1 is the initial peak and represents a sensory response to visual stimuli. N1 and P2 follow P1 and also contain visual components (Luck, 2005). N2 follows P2 and occurs in response to repeated stimuli, with larger amplitude responses to occasional interfering stimuli (Luck, 2005). P3 occurs last in the waveform and is task-dependent; in oddball tasks, P3 is typically larger in response to infrequent stimuli (Luck, 2005). Peak detection was performed over average ranges within each condition to mark P1 (vibration: 27ms-116ms; LED: 38ms-119ms), N1 (vibration: 86ms-184ms; LED: 87ms-186ms), P2 (vibration: 147ms-240ms; LED: 137ms-248ms), N2 (vibration: 213ms-325ms; LED: 201ms-314ms) and P3 (vibration: 313ms-456ms; LED: 306ms-469ms) peaks. Resulting peak amplitudes and latencies were further analyzed using a 2(Control, Experimental) X 2(Pre, Post) repeated measures ANOVA.

2.2.3 Results

2.2.3.1 Behavioural Results

Throughout the analysis of Experiment II, main effects of Vision, Target Distance, and Target Size were commonly observed for RT, MT, PGA, TAPGA, and TAPV. Where Vision was found to have a main effect, RT, MT, TAPGA, and TAPV were all longer in NV than FV; PGA was found to be larger in NV than FV; variability in limb position was greater in NV than

FV; and correlation between prior limb position and movement endpoint was greater in NV than FV. Where Target Distance was found to have a main effect, RT, MT, TAPGA, and TAPV were all longer to Long targets than to Short, and variability in limb position was greater when targets were further away. Where Target Size was a main effect, PGA was larger to Large targets and smaller to Small targets. These main effects were consistent with the same outcomes seen in Experiment I and will not be further interpreted in this section; means are presented in Table 5.

There were also several interactions involving Vision with Target Distance and/or Target Size. While further analysis of these interactions revealed significant effects, these effects were also in keeping with those observed for these parameters in Experiment I. That is, these results re-iterated those already found to show significant differences between reaching in Vision and No Vision, reaching for near targets versus far targets, and reaching to Small targets versus Large targets. In all of these interactions there was a significant effect of Vision where PGA was smaller and MT, TAPGA, and TAPV were all shorter when reaching in Vision trials than in No Vision. MT, TAPGA, and TAPV were all shorter when reaching to nearer targets regardless of visual condition. PGA was smaller when reaching to Small targets regardless of visual condition, and TAPGA and TAPV were both shorter when reaching to Large targets when vision was unavailable. In combination, these results provided no additional information of interest and were deemed not directly relevant to further discussion. These interactions are not included in this section but are presented with their means and standard deviations in Table 6.

2.2.3.1.1 Planning

Reaction time (RT) analysis yielded main effects of Vision $F(1,11) = 69.04, p < .05$ (Figure 7), and Target Distance $F(1,11) = 7.44, p < .05$ (Table 5). A two-way interaction of Vision and Target Distance $F(1,11) = 8.91, p < .05$ was also present. Further analysis showed the interaction to be driven by a significant effect of Vision to both Long $F(1,11) = 57.01, p < .05$

and Short $F(1,11) = 67.07, p < .05$ targets and significant main effect of Target Distance, but in only the Vision condition $F(1,11) = 35.18, p < .05$ (means: NV, Long = 262.77ms; NV, Short = 262.93ms; FV, Long = 225.80ms; FV, Short = 215.71ms).

Analysis of movement time (MT) showed main effects of Vision $F(1,11) = 81.64, p < .05$ (Figure 7) and Target Distance $F(1,11) = 520.38, p < .05$ in keeping with Experiment I (Table 5). Novel main effects were observed for Pre-Post $F(1,11) = 17.77, p < .05$ (Figure 8) and Target Size $F(1,11) = 6.92, p < .05$. MT was significantly shortened in the Post-test relative to the Pre-test (means: Pre = 983.62ms; Post = 938.31ms). When reaching to Small targets, MT was faster than when reaching to Large targets (means: Small = 969.55ms; Large = 952.38ms).

Two-way interactions were observed between Control-Experimental (CE) and Vision $F(1,11) = 4.98, p < .05$; and Vision and Target Size $F(1,11) = 9.05, p < .05$. The interaction of CE and Vision seems to be driven by the significant main effect of Vision where MT was slower in NV trials on both days (means: Control, NV = 1127.32ms; Control, FV = 806.42ms; Experimental, NV = 1136.04ms; Experimental NV = 774.07ms); no other CE differences were found on further analysis.

Analysis of the interaction between Vision and Target Size showed an effect of visual condition to both Small $F(1,11) = 96.61, p < .05$ and Large targets $F(1,11) = 66.26, p < .05$, where MT was faster when reaching with vision, regardless of target size. An effect of Target Size was found only in the NV reaching condition $F(1,11) = 10.31, p < .05$, where reaching to Large targets was faster than to Small (means: FV, Small = 790.39ms; FV, Large = 790.10ms; NV, Small = 1148.71ms; NV, Large = 1114.65ms).

Three-way interactions for MT were found between CE, Pre-Post, and Target Distance $F(1,11) = 5.36, p < .05$; CE, Vision, and Target Distance $F(1,11) = 5.67, p < .05$; and Pre-Post, Vision, and Target Distance $F(1,11) = 13.64, p < .05$. The CE, Pre-Post, Target Distance

interaction proved to be driven by the Target Distance main effect where reaching to Short targets is faster than to Long in all conditions, with no true CE differences (Table 7).

Analysis of the three-way CE, Vision, Target Distance interaction yielded a main effect of Target Distance in the NV $F(1,11) = 303.68, p < .05$ and FV $F(1,11) = 403.25, p < .05$ conditions, again with significantly faster MT when reaching to Short targets than to Long (Table 7). A two-way interaction was also found of CE and Target Distance in the Vision condition $F(1,11) = 11.45, p < .05$, with a main effect of CE but seen only in the Vision condition and to Short targets $F(1,11) = 5.83, p < .05$; in FV, reaching to Short targets was faster on the Experimental day than Control (Table 7). Overall, these interactions were also driven by the effect of Target Distance on MT, where further targets correspond with greater MT.

Deconstructing the three-way Pre-Post, Vision, Target Distance interaction showed main effects of Pre-Post $F(1,11) = 8.14, p < .05$ and Target Distance $F(1,11) = 303.67, p < .05$ in the NV condition; MT was faster in the Post, NV condition than Pre, NV condition, and faster to Short targets than Long within each of these conditions (Table 7). An interaction of Pre-Post and Target Distance $F(1,11) = 10.36, p < .05$ with a main effect of Pre-Post in NV to further targets $F(1,11) = 17.52, p < .05$, but not near targets; therefore, Pre to Post in the NV condition MT is faster specifically to Large targets across these conditions (Table 7). Main effects of Pre-Post $F(1,11) = 7.61, p < .05$ and Target Distance $F(1,11) = 403.31, p < .05$ were also found in the Vision condition, with main effects of Pre-Post also found to both Short $F(1,11) = 6.62, p < .05$ and Long $F(1,11) = 7.19, p < .05$ targets also in this condition. These results are interpreted in the same manner as those found in the NV condition; Pre to Post in the FV condition MT is faster, though faster MT to Short targets was observed both within and across these conditions (Table 7).

Analysis of peak grip aperture (PGA) yielded main effects of Vision $F(1,11) = 14.99, p < .05$ (Figure 1), and Target Size $F(1,11) = 117.37, p < .05$ (Table 5). A two-way interaction was found between CE and Target Size $F(1,11) = 10.18, p < .05$, but further analysis revealed this interaction to be driven by the main effect of Target Size with larger PGA to Large targets, present in both the Control, $F(1,11) = 133.23, p < .05$; and Experimental conditions $F(1,11) = 86.09, p < .05$ (means: Control, Large = 97.75mm; Control, Small = 91.28mm; Experimental, Large = 99.07mm; Experimental, Small = 93.63mm).

A three-way interaction was also observed, between CE, Vision, and Target Size $F(1,11) = 6.44, p < .05$, with further analysis also showing no effect of CE. Main effects of Vision $F(1,11) = 13.66, p < .05$ and Target Size $F(1,11) = 128.56, p < .05$ were present (Table 7). A two-way interaction of Vision and Target Size $F(1,11) = 20.78, p < .05$ yielded further main effects of Vision such that PGA was larger in NV when reaching to both Small $F(1,11) = 16.26, p < .05$, and Large targets $F(1,11) = 10.02, p < .05$ (Table 7). Additionally, there was a main effect of Target Size in both the Vision $F(1,11) = 271.66, p < .05$ and No Vision conditions $F(1,11) = 29.34, p < .05$, where reaching to Large targets in both visual conditions adopted a larger PGA (Table 7).

2.2.3.1.2 Control

Time after PGA (TAPGA) analysis found main effects of Vision $F(1,11) = 80.80, p < .05$ and Target Distance $F(1,11) = 445.97, p < .05$ (Table 5), and novel main effects of Pre-Post $F(1,11) = 12.36, p < .05$ and Target Size $F(1,11) = 13.17, p < .05$. TAPGA was significantly shorter Post 8-hour visual deprivation (Figure 8), and significantly shorter after reaching to Large targets than Small (means: Large = 786.07ms; Small = 807.99ms). A two-way interaction was observed between CE and Vision $F(1,11) = 6.13, p < .05$, but further determined to be driven by the main effect of Vision present in both the Control $F(1,11) = 77.06, p < .05$, and

Experimental conditions $F(1,11) = 76.82, p < .05$, without any accompanying significance of CE (means: Control, NV = 945.81ms; Control, FV = 658.35ms; Experimental, NV = 955.53ms; Experimental, FV = 628.42ms).

Three three-way interactions were observed. CE, Pre-Post, and Target Distance $F(1,11) = 5.61, p < .05$ was found to be driven by main effects of Pre-Post and Target Distance, with no significant effects of CE (Table 7). CE, Vision, and Target Distance $F(1,11) = 6.45, p < .05$ revealed significant CE differences only to Short targets in Vision $F(1,11) = 7.20, p < .05$; TAPGA was significantly shorter when reaching to Short targets in the FV condition on the Experimental day (Table 7).

Lastly, Pre-Post, Vision, and Target Distance $F(1,11) = 14.44, p < .05$ showed significant effects of Vision to Short $F(1,11) = 41.64, p < .05$ and Long $F(1,11) = 78.12, p < .05$ targets in the Pre condition, as well as to Short $F(1,11) = 81.58, p < .05$ and Long $F(1,11) = 116.38, p < .05$ targets in the Post condition. Target Distance was a significant factor while reaching with Vision $F(1,11) = 200.55, p < .05$ and No Vision $F(1,11) = 232.47, p < .05$ in the Pre condition, and reaching with Vision $F(1,11) = 208.64, p < .05$ and No Vision $F(1,11) = 88.65, p < .05$ in the Post condition. Overall, this interaction reflects significantly shorter TAPGA when reaching to Short targets than Long, and in FV than NV, consistently Pre- and Post-deprivation (Table 7).

Analysis of time after peak velocity (TAPV) yielded main effects of Vision $F(1,11) = 82.45, p < .05$ and Target Distance $F(1,11) = 340.78, p < .05$ (Table 5), and novel main effects of Pre-Post $F(1,11) = 10.34, p < .05$ and Target Size $F(1,11) = 7.97, p < .05$. TAPV was significantly shorter Post 8-hour visual deprivation (Figure 8), and when reaching to Large targets than Small (means: Large = 602.01ms; Small = 618.96ms). A two-way interaction was found between CE and Vision $F(1,11) = 6.24, p < .05$, but later determined to be driven by the main effect of Vision, with shorter TAPV in FV than NV significant in both the Control $F(1,11)$

= 71.12, $p < .05$ and Experimental $F(1,11) = 84.25$, $p < .05$ conditions; no CE significance was found in either visual condition (means: Control, NV = 791.19ms; Control, FV = 516.21ms; Experimental, NV = 801.22ms; Experimental, FV = 485.94ms).

Two three-way interactions were observed: CE, Pre-Post, and Target Distance, and Pre-Post, Vision, and Target Distance. CE, Pre-Post, and Target Distance $F(1,11) = 7.34$, $p < .05$ yielded a two-way interaction of CE and Target Distance on further analysis, observed in the Pre condition $F(1,11) = 7.87$, $p < .05$ with shorter TAPV when reaching to Long targets during the Control day Pre-test, and shorter TAPV when reaching to Short targets during the Experimental day Pre-test (Table 7). However, this interaction proved to be driven by the significance of Target Distance present in the Pre $F(1,11) = 345.80$, $p < .05$ and Post $F(1,11) = 162.85$, $p < .05$ conditions, with TAPV shorter when reaching to Short targets than Long (Table 7); CE was not significant in either condition, to either target distance. Breaking down Pre-Post, Vision, and Target Distance $F(1,11) = 10.59$, $p < .05$ revealed significance on all levels, with main effects of Vision and Target Size and an interaction of these two conditions observed both Pre and Post. TAPV was significantly shorter when reaching to Short targets than Long, in FV than NV, and Post-deprivation than Pre (Table 7).

2.2.3.1.3 Pre-to-Post Differences

Three-way interactions involving CE and Pre-Post found in TAPV and TAPGA analysis, in addition to those previously mentioned as found in MT, were further analyzed to investigate the role of CE differences Pre-to-Post. A Pre-to-Post change value was calculated by subtracting Post-test means from Pre-test means to yield the difference between Pre and Post within that day. The repeated measures analysis was re-run on the calculated differences in an attempt to assess what kind of changes in these variables took place throughout the course of each day. Paired, one-tailed t-tests were then performed on the resulting means; results are presented in Table 8.

Main effects involving Target Distance encountered during the analysis of pre-to-post differences reflect the main effects discussed previously with the original results of these interactions. As such, these main effects will not be re-addressed, though means are included in Table 8.

Analysis of pre-to-post differences in MT showed two significant two-way interactions, between CE and Target Distance $F(1,11) = 8.50, p < .05$ and Vision and Target Distance $F(1,11) = 13.65, p < .05$. Pre-to-post difference when reaching to Long targets was greater on the Experimental day, equating to faster MT to Long targets following the Experimental day (Figure 9, left; Table 8). The CE and Target Distance interaction was also driven by an effect of Target Distance present on the Experimental day, with a greater pre-to-post change to Long targets than Short (Figure 9, left; Table 8). The interaction of Vision and Target Distance was driven by an effect of Target Distance in NV, with greater pre-to-post differences to Long targets than Short (Table 8).

Interactions of CE and Target Distance $F(1,11) = 9.42, p < .05$ and Vision and Target Distance $F(1,11) = 14.44, p < .05$ were also found in analysis of TAPGA pre-to-post differences. These interactions were driven by the same effects seen with respect to MT: greater pre-to-post differences were seen to Long targets than Short following the Experimental day (Figure 9, middle), and reaching to Long targets than Short during the Experimental day and in NV conditions (Table 8).

Analysis of TAPV pre-to-post differences again showed significant two-way interactions of CE and Target Distance $F(1,11) = 10.84, p < .05$, and Vision and Target Distance $F(1,11) = 10.59, p < .05$. The driving effects of the CE and Target Distance interaction were the same as those discussed for MT and TAPGA (Figure 9, right). The interaction of Vision and Target Distance was driven by an effect of Vision when reaching to Long targets, with greater pre-to-

post differences seen in NV, and also by an effect of Target Distance present in NV, with greater pre-to-post differences seen to Long targets than Short (Table 8).

Overall, pre-to-post differences yielded by this analysis involving CE effects were deemed more relevant to further discussion than those related to effects solely of Vision and Target Distance; as such, the results of Vision and Target Distance interactions will not be discussed further, and discussion of pre-to-post difference analysis will be restricted to interactions of CE and Target Distance (Figure 9).

2.2.3.1.4 Decile Analyses

Decile analysis (i.e. percent of movement execution) of grip aperture (GA) in all trials showed main effects of Decile $F(10,110) = 227.61, p < .05$; Vision $F(1,11) = 16.71, p < .05$; Target Size $F(1,11) = 165.30, p < .05$; and Target Distance $F(1,11) = 10.26, p < .05$. The main effect of decile reflects the natural change in grip aperture as the movement progresses, where GA at each decile was significantly different from the prior decile, with the exception of the 60% to 70% movement decile (Table 9). GA was significantly larger in NV than FV (means: NV = 65.84mm; FV = 61.89mm), to Large targets than Small (means: Large = 66.39mm; Small = 61.33mm), and to Short targets than Long (means: Short = 64.98mm; Long = 63.26mm).

Several significant two-way interactions involving Decile were present. CE and Decile $F(10,110) = 2.52, p < .05$ identified significance of CE at the 90% stage of movement trajectory, with GA significantly larger on the Experimental day at 90% (Table 10). Decile and Vision $F(10,110) = 7.01, p < .05$ showed significance of Vision from 10% to 50% of movement, with larger GA adopted in NV in these deciles (Figure 10; Table 10). Decile and Target Size showed a significant impact of Target Size on grip aperture from 30% to movement end, with GA significantly larger to Large targets than Small (Table 10). Decile and Target Distance $F(10,110) = 13.26, p < .05$ provided less concise insight into how target distance affected grip aperture,

with significantly smaller GA to Long targets occurring at 0%, 30-50%, and 100% deciles, and significantly larger GA to Long targets from 70% to 90% deciles (Table 10).

A three-way interaction between Decile, Vision, and Target Size $F(10,110) = 2.43, p < .05$ yielded a significant influence of Target Size, with larger GA to Large targets from 30% movement to movement end in Vision, and from 50% movement to movement end in No Vision (Table 11). CE, Decile, and Target Distance $F(10,110) = 1.94, p < .05$ showed significantly larger GA used in the Experimental condition than Control, specifically to Short targets from 20% to movement end, and Long targets from 10% to movement end (Table 12). This interaction also showed varied results of Target Distance significance, with differences in the Control condition at 30-50% and 80% to movement end, and differences in the Experimental condition at 0%, 30-50%, 70%, and 80% of movement trajectory; due to the varied nature of these differences, they were deemed not relevant to further discussion.

The three-way interaction of Decile, Vision, and Target Distance $F(1,11) = 7.06, p < .05$ was broken down into the following significant effects: Target Distance in Vision from 30-60%, and 80% to movement end; Target Size in No Vision at 0%, and from 20-40% movement trajectory; Vision to near targets from 10-50% movement trajectory; and Vision to far targets at 10%, and from 40-50% movement trajectory (Table 13). These effects varied in their directions and did not further contribute to our analysis or discussion.

Decile, Target Size, and Target Distance $F(10,110) = 2.69, p < .05$ yielded effects of Target Size to Short targets from 30% to movement end, and Long targets at 0% and from 20% to movement end. Target Distance affected reaching to Large targets at 0%, 30-50%, and 100% of movement; to Small targets, Target Distance was significant from 30-50% and 70-90% movement trajectory (Table 14). Target Size and Target Distance effects drove this interaction;

this reiterated previous results involving these parameters and did not contribute novel information to the analysis or discussion.

Three-way interaction of CE, Vision, and Target Distance $F(1,11) = 5.30, p < .05$ was further analyzed to reveal no significant CE effects. In the Control condition, GA was significantly larger to Long targets both in Vision $F(1,11) = 8.44, p < .05$ and in No Vision $F(1,11) = 11.05, p < .05$ (Table 15). In the Experimental condition, GA was significantly larger to Long targets, but only in No Vision $F(1,11) = 9.95, p < .05$ (Table 15). Significant effects of Vision were present in the Control condition, with larger GA in No Vision, and when reaching to both Short $F(1,11) = 10.39, p < .05$ and Long targets $F(1,11) = 5.32, p < .05$; in the Experimental condition, the same effect was found, again to both Short $F(1,11) = 22.37, p < .05$ and Long targets $F(1,11) = 10.02, p < .05$ (Table 15).

Further analysis of grip aperture over movement deciles was conducted on the last 5 trials of each Pre-test block and the first 5 trials of each Post-test block to address any potential learning or order effects. Main effects were present for Decile $F(10,110) = 199.32, p < .05$ and for Vision $F(1,11) = 11.46, p < .05$. The main effect of decile reflects the natural change in grip aperture as the movement progresses, where GA at each decile was significantly different from the prior decile, with the exception of the 60% to 70% movement decile (Table 16). GA was significantly larger in NV than FV (means: NV = 64.10mm; FV = 59.83mm).

A two-way interaction was found for CE and Decile $F(10,110) = 2.52, p < .05$, but further analysis showed no significant within decile differences between Control and Experimental mean grip apertures (Table 17). Two-way interaction of Decile and Vision $F(10,110) = 6.75, p < .05$, however, revealed significant differences between grip apertures in Vision and No Vision from 10-50% of movement trajectory. During this early phase of movement, grip apertures are significantly larger in No Vision than in Vision (Table 17).

A two-way interaction of Pre-Post and Vision $F(1,11) = 6.30, p < .05$ was broken down to show significantly larger GA in NV than FV in Post $F(1,11) = 38.95, p < .05$ (means: Post, NV = 65.48mm; Post, FV = 59.56mm) and significantly larger GA Post 8-hour visual deprivation than Pre-deprivation specifically in the No Vision condition $F(1,11) = 5.11, p < .05$ (means: Pre, NV = 62.72mm; Post, NV = 65.48mm).

A three-way interaction between Decile, Pre-Post, and Vision $F(10,110) = 3.30, p < .05$ revealed no significant effect of Vision in Pre, but significance of Vision in the Post condition from 10-50% movement (Table 18). This component of the interaction was likely driven by the interaction of Decile and Vision showing significance at the same percentages of movement (10-50%), where grip apertures are significantly larger in No Vision than in Vision during this phase of movement. There was a significant difference Pre-Post in the No Vision condition only at 20% and 30% movement, with larger grip apertures adopted in these two phases in the Post-test than in Pre (Table 18).

2.2.3.1.5 Variability and Correlation Analyses

Analysis of the proportion of explained variance (R^2) calculated between movement endpoint and each movement decile yielded main effects of Decile $F(10,110) = 282.74, p < .05$; Vision $F(1,11) = 17.61, p < .05$; and Target Distance $F(1,11) = 5.80, p < .05$. As expected, as movement progressed hand position at each decile became more predictive of movement endpoint with significance from 20% until movement end (Table 19). Mean hand position during No Vision trials was more predictive of movement endpoint than during Vision trials (means: NV = 0.45; FV = 0.35), and in reaching to Short targets than to Long (means: Short = 0.43; Long = 0.37).

A two-way interaction of Decile and Vision $F(10,110) = 8.42, p < .05$ showed that mean hand position from 10-60% of movement was significantly more predictive of movement

endpoint in the No Vision condition than in Vision (Table 20). In a two-way interaction of Pre-Post and Vision $F(1,11) = 5.67, p < .05$, a significant effect of Vision $F(1,11) = 20.71, p < .05$ in the Post test showed that mean hand position in No Vision trials significantly more predictive of movement endpoint than in Vision trials (means: Post, NV = 0.47; Post, FV = 0.32). An effect of Pre-Post $F(1,11) = 10.71, p < .05$ in showed mean hand position in Vision trials was significantly more predictive of movement endpoint in the Pre-test (means: Pre, FV = 0.39; Post, FV = 0.32).

A three-way interaction of Decile, Pre-Post, and Vision $F(10,110) = 2.00, p < .05$ showed significance of Vision in the Post-test with mean hand position more predictive of movement endpoint in No Vision trials than Vision from 10-60% movement, and significance of Pre-Post in the Vision condition with mean hand position more predictive of movement endpoint in the Pre-test from 20-60% of movement (Table 21). A second three-way interaction was found between Pre-Post, Target Size, and Target Distance $F(1,11) = 9.14, p < .05$, though seemingly driven by the previously established main effect of Target Distance as further analysis showed no other significance (Table 22).

Standard deviations of limb position were used to calculate variability within each decile. Main effects were found for Decile $F(10,110) = 182.60, p < .05$; Vision $F(1,11) = 29.54, p < .05$; and Target Distance $F(1,11) = 48.47, p < .05$. The main effect of Decile was significant for movement duration, with variability in limb position significantly different at each 10% increment of from that prior (Table 23). Variability in limb position was significantly greater in No Vision trials than in Vision trials (means: NV = 16.67; FV = 12.82), and to further targets than to near (means: Long = 15.36; Short = 12.32).

A two-way interaction of Decile and Vision $F(10,110) = 12.08, p < .05$ showed significantly greater variability of limb position during No Vision trials than in Vision at 10-40%

and 70-90% movement (Table 23). A two-way interaction of CE and Target Size $F(1,11) = 6.96$, $p < .05$ was found, but revealed no other significance on further analysis. Two-way interaction of Decile and Target Size $F(10,110) = 6.24$, $p < .05$ showed significantly greater variability in limb position to Large targets than to Small at 30-40% of movement, while interaction of Decile and Target Distance $F(10,110) = 55.38$, $p < .05$ showed significantly greater variability in limb position to Long targets than to Short from 10-60% of movement (Table 24).

A three-way interaction of Decile, Vision, and Target Distance $F(10,110) = 9.43$, $p < .05$ was present. Mean variability of limb position was significantly greater to Long than Short targets in Vision from 20-70% of movement, and to Long than Short targets in No Vision from 0-40% movement. Variability in this interaction was also significantly greater in No Vision trials to Short targets than in Vision from 20-40% movement and 60% to movement end; and finally, significantly greater in No vision to Long targets than in Vision from 10-30% and 80% movement (Table 25).

2.2.3.2 EEG Results

To identify critical features of the EEG waveform, peak detection was performed over average ranges within each condition: P1 (vibration: 27ms-116ms; LED: 38ms-119ms), N1 (vibration: 86ms-184ms; LED: 87ms-186ms), P2 (vibration: 147ms-240ms; LED: 137ms-248ms), N2 (vibration: 213ms-325ms; LED: 201ms-314ms) and P3 (vibration: 313ms-456ms; LED: 306ms-469ms) peaks. Resulting peak amplitudes were analyzed using a 2(Control, Experimental) X 2(Pre, Post) repeated measures ANOVA. In order to obtain a 2(Control, Experimental) X 2(Pre, Post) model from the original 2(Control, Experimental) X 2(Pre, Post) X 2 (Left, Right) X 2(Oddball, Non-Oddball), the Oddball-Non-Oddball difference was calculated, then Left-Right conditions were collapsed to remove laterality inherent in the oddball paradigm. Amplitude data were first analyzed to determine the electrodes with the largest effects for both

the LED and vibration conditions. Averaged visual evoked potential amplitudes and somatosensory evoked potential amplitudes are presented in Figures 11 and 12, respectively. Latency data corresponding to these electrodes were then analyzed separately; this analysis revealed no significant results.

Amplitude analysis revealed significant amplitude effects in primarily the O1 and O2 electrodes, for both conditions. There were no significant results for N1 and N2 peaks in both the LED and vibration conditions, and no P3 results for the vibration condition. P1 amplitude in the LED condition was maximal at O2 (2.01 μ V), with a main effect of Experimental condition $F(1,11) = 8.30, p < .05$. P1 amplitude in the vibration condition was maximal at O1 (1.75 μ V), with a main effect of Experimental condition $F(1,11) = 4.94, p < .05$, and a two-way interaction of Control/Experimental and Pre-Post $F(1,11) = 7.59, p < .05$. Two-tailed paired t-tests revealed this interaction to be driven by a Pre-Post effect in the Experimental condition ($p < .05$).

P2 amplitude in the LED condition was maximal at O2 (2.63 μ V), with a two-way interaction of Experimental condition and Pre-Post $F(1,11) = 13.40, p < .05$. This interaction was driven by Pre-Post effects in Control ($p = .03$) and Experimental condition effects in both Pre ($p = .05$) and Post ($p = .05$). P2 amplitude in the vibration condition was maximal at O1 (2.47 μ V), with a two-way interaction Experimental condition and Pre-Post $F(1,11) = 5.52, p < .05$, that proved to be non-significant on further analysis. P3 amplitude in the LED condition was maximal at O2 (3.15 μ V), with a two-way interaction of Experimental condition and Pre-Post $F(1,11) = 8.30, p < .05$, which also proved non-significant on further analysis.

2.2.4 Discussion

2.2.4.1 Planning and Control

Main effects of vision in RT, MT, and PGA reflect expected reaching and grasping behaviours when vision is unavailable (Figure 7). Reaction and movement times are both significantly increased in the absence of vision, and peak grip aperture is significantly larger, as a wider grasp is employed to increase probability of target contact. Time after PGA (TAPGA) is also longer in the NV condition, also suggesting use of a wide grasp. A larger grip aperture is generally used to larger targets than to small, as evidenced by a main effect of Target Size in PGA. This reflects maintenance of normal behaviour despite visual perturbation; it occurs regardless of the presence of vision, but GA is significantly larger to larger targets in NV than to larger targets in FV, as if the absence of vision somehow exacerbates conservative grasping strategies.

Main effects of Target Distance in RT and MT show faster RTs and MTs to nearer targets, and longer RTs and MTs to further targets, as expected. Interactions of these main effects with the main effect of vision in both RT and MT further reinforce this effect. Main effects of vision and Target Distance are also seen in time after peak velocity (TAPV), with more time spent after TAPV in NV and to Long targets. These reinforce expected behaviours, as further targets take longer to reach to, and a reach in NV is more exploratory, resulting in longer time spent reaching than when vision is available.

Absence of Pre-Post and Control-Experimental effects for RT and PGA may suggest they are unaffected by the prolonged deprivation, or some kind of washout or adaptation is negating any potential effect. This is not a novel suggestion, as in subjects blindfolded for 5 days (Kauffman et. al, 2002), newly observed activation of V1 in response to tactile stimuli was significantly reduced within just hours of blindfold removal (Pascual-Leone et. al, 2005). MT,

TAPV, and TAPGA, however, are longer Pre than Post (Figure 2). While RT and grasping strategy (PGA) are staying the same Pre-to-Post, it seems that the movement features change throughout the day; overall MT becomes shorter, suggesting a learning of task or target location, while the timing of PV and PGA seems to be shifted later in the movement. This late shift corresponding to less time spent after PV and PGA reflects a decrease in time spent controlling movement; PGA itself reflects more on movement control than planning (Glover, 2004), and PV represents the initiation of movement deceleration, where it has been suggested the majority of movement regulation occurs (Chua & Elliott, 1993).

2.2.4.2 Pre-to-Post Differences

Three-way interactions of Pre-Post with Control-Experimental and Target Distance for MT, TAPV, and TAPGA were further analyzed as Pre-Post differences to show pre-to-post changes in each of these measures. With respect to MT, speeding of MT was observed pre-to-post in both Control and Experimental conditions, with the decrease in MT pre-to-post significantly larger in the Experimental condition (Figure 9). Faster observed movement time seems to be related to less time spent after PV and PGA, as previously mentioned. This is further evidenced by pre-to-post differences in TAPV and TAPGA; both decrease pre-to-post, and significantly more in the Experimental condition (Figure 9).

2.2.4.3 Decile Analyses

Results of decile analysis for all trials show mainly normal behaviours, with a larger grip aperture adopted at most deciles of NV movements than in FV (Figure 10). Many higher order interactions seem to be driven by this main effect of vision. Of interest, however, is the three-way interaction of Control-Experimental with decile and Target Distance; grip aperture is larger to both target distances in the Experimental condition than Control. On the Experimental day, a larger grip aperture is used for all targets, and is adopted sooner in the movement. It is possible

that an up-regulation of proprioception in the planning phase has caused participants to become more aware of distance in their reaching movements, and use more accurate approximations of target size sooner during movement, requiring less correction later in the movement.

Further grip aperture decile analysis on the last five trials of each Pre-test condition and first five trials of each Post-test condition to overcome any wash-out effects failed to produce novel effects beyond those noted when all trials were included in the analysis. However, a three-way interaction of decile with Pre-Post and vision may suggest some changes occurring within each day, as vision only creates a significant effect on grip aperture over deciles in the post-test. This observation provides support that adaptation is occurring throughout both days. Unfortunately, lacking any Control-Experimental effect leaves us unable to determine if adaptation is occurring in the same manner on each day.

2.2.4.4 Variability and Correlation Analyses

Similarly, Correlation (R^2) analysis supported expected behaviours with no significant effects of the Control-Experimental conditions. Prior limb position was more predictive of movement endpoint in NV, reflected in higher R^2 values in NV than FV. In the absence of vision, then, planning is used to formulate later online corrections, following expected behaviour (Heath, 2005). In reaching to further targets, prior limb position is less predictive than when reaching to near targets; this decreased predictability may be introduced by innately longer movement times to targets that are further away, thus decreasing R^2 . This decrease in predictability of movement endpoint when reaching to further targets is echoed in variability analysis of standard deviations of limb position within each decile. Variability analysis also showed expectedly greater variability in NV than FV.

2.2.4.5 Adaptations in Control

While much of the data support that general behaviours are unchanged, of particular interest are the three-way interactions of Control-Experimental with Pre-Post and Target Distance. The decreased MT pre-to-post, most significantly in the Experimental day, in conjunction with the observed decreases in TAPV and TAPGA in the same condition would suggest improvements in grasping accuracy as a result of the visual deprivation. Participants are executing movements faster while spending less time after PGA and PV, and therefore less time controlling movement. Typically, when vision is available, participants tend to spend more time after PV, presumably as they use this information to drive online corrections (Elliott et al. 1991, Elliott et al., 2001). A possible optimization of proprioception in earlier planning phases may have decreased need for corrections, as evidenced by the use of less conservative grasping strategy on the Experimental day.

2.2.4.6 Cortical Evidence of Adaptation

We expected any adaptations in planning and control evidenced by behavioural changes to be reflected in changes in neural activity, demonstrating some kind of plastic change. Differences, if any, were predicted to be mostly over parietal and occipital electrodes; parietal to detect any tactile plasticity by vibration detection, and occipital to detect any visual plasticity in LED detection, both brought on by the visual deprivation of the Experimental day. Our analysis, however, revealed maximal significant changes in only occipital electrodes, specifically O1 and O2. This is not entirely unexpected, given our experimental manipulation dealt with primarily the removal of vision and thus likely created more of a change in visual processing than tactile.

Significant changes at these electrodes were only observed to P1 and P2 peaks in both LED and vibration conditions, and P3 only in the LED condition. P1 is largest at lateral occipital electrodes and is typically observed in a waveform regardless of task specificity (Luck, 2005); in

LED conditions, this occurs in response to the visual stimulus of illuminating the LED, but in the vibration condition this may have been due to some other visual distraction. P2 is typically observed in central or anterior regions of the scalp, and may be appearing in these occipital regions in overlap with N1, N2, and P3 peaks (Luck, 2005). P3 is typically maximal in parietal electrodes, here possibly in overlap with the occipital electrodes. P3 in oddball paradigms is of large amplitude, in response to the infrequency of stimuli but their expectation by the participant (Verleger, Jaskowski, & Wauschkuhn, 1994).

Observation of these effects lends some support that the oddball paradigm itself was successful, however their inconsistency prevents any real determination of significant cortical changes (Figures 11 and 12). It is possible 8 hours is simply not enough time for higher cortical changes to occur, and observed changes to planning and control of movement are related to lower-level adaptations occurring sub-cortically. Significant plastic changes may require more drastic or long-term perturbation to vision in order to see profound changes like occipital activation in response to tactile tasks (Sadato et. al, 1996), and increased tactile error rates in response to occipital stimulation (Cohen et. al, 1997).

Overall, our behavioural results suggest a decreased time using online control post-deprivation regardless of visual input, which may translate to an increased use of proprioceptive information for planning movements initially, and controlling movements even when vision is available. Despite indications of behavioural adaptation, we do not see any consistently significant electrophysiological changes that would suggest accompanying cortical plasticity. It is possible this change in behaviour stems from sub-cortical adaptations to the nervous system, such as at the level of the spinal cord relative to the muscles involved in the movement.

This is most likely due to a quick re-adaptation upon regaining vision post-deprivation (see: Pascual-Leone et. al, 2005). Our experimental protocol necessitated performing the

reaching and grasping task first and the EEG oddball task second; it seems likely that during the performance of the grasping task (where vision is regained in the last two blocks), and preparation for the EEG task (i.e. fitting the EEG cap and ensuring low impedance values prior to recording), any higher adaptation that would have been visible in the electrophysiological data may have been completely washed out. Therefore, future experimentation where the main focus is cortical adaptation and the EEG task could be performed right at vision onset following the deprivation could serve to provide a clearer picture of the cortical adaptations that occur in response to 8 hours of visual deprivation.

CHAPTER 3: GENERAL DISCUSSION AND CONCLUSIONS

3.1 Behaviour

These experiments were performed in hopes of determining the role of proprioception in movement planning and control when vision is unavailable, and how this role changes as a result of short- and long-term visual deprivation. The first experiment served as a pilot study to explore the changes in movement planning and control that arise in response to acute onset short-term vision loss with respect to use of visual and proprioceptive inputs. In the second experiment, the aim was to elaborate on behavioural changes observed in the pilot study, and correlate these changes to any cortical plasticity that may have occurred as a result. Both experiments yielded results that support a continuation of typical reaching and grasping behaviour on a general scale, including faster reaction and movement times to nearer targets and with vision available, and vice versa without. Grip aperture was consistently scaled to target size with vision and conservative, wider grasps adopted in no-vision. While perhaps not an extreme enough perturbation, these results do suggest that while some changes to actual movement structure may occur, the fundamental parameters of the movement are unyielding to visual deprivation.

In Experiment II there was a reduction of conservative grasping strategy, occurring on the Experimental day, following 8 hours of visual deprivation. This was not observed in Experiment I. It is possible that this less conservative grasp was associated with an increase in proprioceptive information in planning, leading to movements with more accurately approximated grasps. In Experiment I, we also saw elements of this up-regulation, but it seemed to be functioning more in the control phase, evidenced by longer time spent after peak grip aperture in the post-deprivation, full-vision condition. In both experiments, peak grip aperture did not change pre-to-post.

3.2 Dual-phase Adaptation

Movement time and time after peak velocity were unchanged as a result of the 2-hour deprivation in Experiment I. In the second experiment, however, we saw shorter movement times and time spent after peak velocity in the post-test. Reaction time was improved pre-to-post in Experiment I, but showed no significant difference at the end of either day in Experiment II. It seems that adaptation in movement time and time after peak velocity begins to occur somewhere within the 2 to 8 hour time frame, while early adaptations in reaction time are washed out as the deprivation is prolonged. These observations suggest an initial phase of adaptation and a later phase of adaptation within the 8 hours. Initially, the focus is on learning how to perform movements more quickly (i.e. decreasing reaction times, movement times), while accuracy becomes the goal later in the deprivation (i.e. less conservative grasping, less time spent in the control phase). Breaking adaptation up into stages may be a coping mechanism used by the brain to assess what kind of changes have occurred, and how to respond in a way that restores functionality.

Subtle differences in time after peak grip aperture observed in Experiment I might highlight the transitioning of these phases. At first, we saw a tradeoff in the use of visual information for proprioceptive information to plan and control a movement. Later, we saw less time invested in control and more use of proprioception to coordinate more accurate movements requiring less correction. Contrary to Woodworth's (1899) and consistent with Heath (2005), findings that visual input is preferred over proprioceptive input when vision is available, it is possible here that residual proprioceptive influence or a previously constructed visual memory is responsible for some of the proprioceptive input to movement planning that is decreasing the need for online correction. The presence of less correction supports an open loop mode of control, where visual feedback is not used to correct movements (Plamondon, 1995; Plamondon

& Alimi, 1997), and deviates from the expectation that vision is being used preferentially over other information when it is available (Heath, 2005).

3.3 Corrections

Time after peak velocity and time after peak grip aperture provide us with an approximate timing of the control phase. In Experiment I, time after peak grip aperture was not significantly different following the deprivation. However, an observed decrease in the difference of this parameter between no-vision and full vision post-deprivation seems to suggest a longer time spent after peak grip aperture when vision was available during the post-test. According to Elliott et al. (1991), this is typical behaviour, as the visual information available is actively used to perform corrections during this time. As the difference between reaching in full-vision and no-vision decreased post-test, however, our experiment suggests even more time is being spent in this corrective phase than pre-deprivation.

The case is much the opposite in Experiment II. Movement time, time after peak velocity, and time after peak grip aperture are all decreased pre-to-post. Therefore, less time was spent after peak velocity and in a corrective phase even with vision available, deviating from the behaviour observed by Elliott et al. (1991). Experiment II shows a shift of peak velocity and peak grip aperture to later in the movement, a change that was not observed in Experiment I. Here, then, less time was spent controlling the movement, as if the proprioceptive information gathered was contributed to planning a movement that requires less correction.

3.4 Limitations and Future Considerations

Lack of significant cortical changes that reflect this increased use of proprioceptive information in planning and less energy focused on online control suggests that while 8 hours may induce behavioural differences, it may not be a significantly long enough deprivation to cause long-term plastic changes. If any cortical adaptation did in fact occur, it may have been

washed out due to our experimental procedure and the time course between vision onset post-deprivation and actual performance of the EEG task. Ideally, the EEG task would have also been performed immediately when participants regained vision following the deprivation, but the constraints of our reaching and grasping task prevented this from being a possibility. In the future, separating the two tasks into independent experiments built around an 8-hour deprivation may provide more notable results and clues to what plastic changes are occurring.

From these experiments, we see that there are indeed early adaptations that occur in the first hours of vision loss. In order to observe more drastic changes, it is apparent that visual deprivation (or other sensory perturbations) likely needs to occur for more than 8 hours; for example, the five-day deprivation that induced improvement in tactile discrimination of Braille characters (Kauffman et al., 2002). Although this is very likely true, there remains the possibility that tactile sensitivity and the planning and control of movements vary drastically in their adaptations to vision loss. These studies aimed to investigate movements rather than just tactile discrimination, and may suggest that adaptation does indeed occur in a different manner.

3.5 Conclusions

In Experiment I, I hypothesized an increase in proprioceptive plasticity and expected this increased proprioceptive awareness to improve movement accuracy; thereby decreasing time spent controlling movements. I did observe evidence of enhanced proprioceptive influence on movement, but more so in the control phase of the movement, leading to increased time during this phase and contradicting our hypothesis. In Experiment II, I had predicted an enhancement of the observations in Experiment I with respect to the behavioural task. Further evidence of proprioceptive up-regulation was observed, and appeared to be more influential on the planning phase of movement rather than control. Therefore, my predictions of the 2-hour experiment seem to better fit the observations of the 8-hour experiment.

It was also expected that the 8-hour deprivation would increase sensitivity to tactile stimuli and decrease sensitivity to visual stimuli in the EEG task. My observations were mainly in the occipital electrodes and more related to visual stimuli than tactile; it seems tactile sensitivity is relatively unchanged by the deprivation. While differences were observed in response to visual stimuli, no consistent pattern emerged to suggest whether these differences reflected an increased or decreased sensitivity to visual stimuli. Previously mentioned issues with experimental procedure may be responsible for the lack of conclusive electrophysiological observations.

It seems that adaptation to acute vision loss occurs in several complex stages, where different aspects of behaviour are affected. Early adaptation to short-term deprivation favours improving on movement speed rather than corrective capacity, and we see more time spent controlling movements as a result. In the later phase of adaptation to long-term acute vision loss (i.e. during the 8 hours), we see a focus on planning more proprioceptively accurate movements, thus decreasing the conservative approach to reaching and grasping in both conditions.

Somewhere between our simulated 8-hour 'first day' and five consecutive days (see: Kauffman et al., 2002) lies the onset of more significant adaptation to vision loss. These experiments suggest there is an increased reliance on proprioceptive information as vision becomes less available (either directly or via memory), but where this information ends up being used to contribute to the movement remains unclear. It is possible that it continues to fluctuate until more permanent plastic changes occur. Therefore, further experimentation should focus on different intervals of visual deprivation between 8 hours and several days, as well as streamline procedures to prevent washout of any plastic changes due to re-adaptation when vision is once again available.

Further study would contribute to a narrow body of literature on how visual deprivation affects movement planning and execution. The experience of transient vision loss is not uncommon; many who go through laser eye surgery will lose vision for a number of days or weeks. Optic injuries can have traumatic impacts on vision and may result in temporary or life-long blindness, the latter of which may also be experienced due to genetics or disease. Knowledge of what is occurring in the brain in response to all of these events is vital to how we approach accommodating and rehabilitating people experiencing them. Simulating long and short-term vision loss provides us the opportunity to investigate these adaptations in a non-invasive fashion.

TABLES

Table 1: Mean grip apertures and standard deviations for decile main effect and two-way interactions of decile analysis for Experiment I.

Decile	Visual Condition		Target Size	
	No Vision	Vision	Large	Small
<i>Decile</i>	<i>Mean Grip Aperture (mm)</i>			
0%	27.12 (± 8.78)	26.50 (± 8.88)	27.40 (± 9.25)	26.84 (± 8.23)
10%	29.69 (± 9.22)*	28.40 (± 9.05) [†]	29.99 (± 9.63)	29.40 (± 8.68)
20%	40.60 (± 11.23)*	37.43 (± 10.39) [†]	41.93 (± 11.73)	39.27 (± 9.90) [‡]
30%	56.70 (± 12.56)*	51.80 (± 11.14) [†]	59.29 (± 13.76)	54.10 (± 9.97) [‡]
40%	72.11 (± 11.20)*	65.85 (± 9.97) [†]	75.33 (± 12.89)	68.89 (± 8.30) [‡]
50%	85.13 (± 9.25)*	78.60 (± 8.64) [†]	88.67 (± 10.99)	81.59 (± 7.17) [‡]
60%	94.31 (± 9.17)*	88.53 (± 9.35) [†]	98.2 (± 10.53)	90.42 (± 8.22) [‡]
70%	98.80 (± 9.53)*	94.43 (± 10.51) [†]	103.11 (± 10.49)	94.49 (± 9.20) [‡]
80%	98.84 (± 8.75)	96.05 (± 9.90) [†]	103.57 (± 9.56)	94.11 (± 8.77) [‡]
90%	93.46 (± 7.10)*	91.61 (± 7.75) [†]	98.88 (± 7.92)	88.04 (± 7.38) [‡]
100%	85.16 (± 5.95)*	84.04 (± 6.24) [†]	91.54 (± 6.97)	78.79 (± 6.01) [‡]

* Significant main effect of Decile ($p < .05$)

† Significant effect of Vision ($p < .05$)

‡ Significant effect of Target Size ($p < .05$)

Table 2: Mean grip apertures and standard deviations for three-way interaction of Vision, Target Size, and Decile for Experiment I decile analysis.

<i>Decile</i>	No Vision		Vision	
	Large	Small	Large	Small
	<i>Mean Grip Aperture (mm)</i>			
0%	27.97 (±9.34)	27.50 (±8.03)	26.82 (±9.23)	26.17 (±8.56)
10%	31.18 (±9.92)	30.81 (±8.88)	28.8 (±9.47)	27.99 (±8.65)
20%	45.02 (±13.69)	42.53 (±10.92)	38.84 (±11.20)	36.01 (±9.63)*
30%	63.98 (±16.76)	59.21 (±11.61)	54.61 (±12.90)	48.99 (±9.65)*
40%	80.92 (±15.63)	75.81 (±9.66)	69.74 (±12.18)	61.96 (±8.33)*
50%	94.06 (±12.41)	89.26 (±8.25)	83.28 (±11.12)	73.92 (±7.10)*
60%	102.74 (±10.11)	97.43 (±8.98)*	93.66 (±11.81)	83.40 (±7.94)*
70%	106.36 (±8.91)	99.98 (±9.16)*	99.86 (±12.42)	88.00 (±9.57)*
80%	105.54 (±8.06)	97.71 (±8.40)*	101.60 (±11.38)	90.51 (±9.51)*
90%	100.25 (±7.38)	90.37 (±7.31)*	97.51 (±8.86)	85.71 (±7.98)*
100%	92.16 (±6.85)	80.42 (±6.16)*	90.92 (±7.34)	77.16 (±6.32)*

* Significant effect of Target Size ($p < .05$)

Table 3: Mean variability of limb position and standard deviations for two-way interactions of decile analysis for Experiment I.

<i>Decile</i>	Target Distance	
	Long	Short
	<i>Variability of Limb Position (mm)</i>	
0%	9.19 (± 3.76)	8.05 (± 3.38)
10%	9.73 (± 3.62)	8.40 (± 3.35)*
20%	14.97 (± 5.44)	10.84 (± 3.59)*
30%	23.47 (± 9.60)	15.03 (± 4.57)*
40%	28.81 (± 9.93)	18.54 (± 5.49)*
50%	27.96 (± 7.41)	19.52 (± 5.49)*
60%	22.44 (± 4.39)	17.75 (± 4.51)*
70%	16.78 (± 3.04)	14.98 (± 3.64)
80%	13.66 (± 3.10)	13.39 (± 3.50)
90%	13.06 (± 3.51)	12.85 (± 3.67)
100%	13.26 (± 4.27)	12.65 (± 3.64)

* Significant effect of Target Distance ($p < .05$)

Table 4: Omega squared (ω^2) of 4-way and higher interactions for Experiment II.

Interaction	ω^2
Grip Aperture Deciles (all trials)	
<i>CE x Decile x Pre-Post x Vision x Target Size</i> <i>F(10,110) = 4.01, p < .05</i>	0.20
<i>CE x Decile x Vision x Target Size x Target Distance</i> <i>F(10,110) = 3.45, p < .05</i>	0.17
<i>Decile x Vision x Target Size x Target Distance</i> <i>F(10,110) = 2.03, p < .05</i>	0.08
<i>CE x Decile x Vision x Target Size x Target Distance</i> <i>F(10,110) = 3.19, p < .05</i>	0.15
<i>CE x Decile x Pre-Post x Vision x Target Size x Target Distance</i> <i>F(10,110) = 1.98, p < .05</i>	0.08
STDEV	
<i>Decile x Vision x Target Size x Target Distance</i> <i>F(10,110) = 4.41, p < .05</i>	0.22

Table 5: Means and standard deviations for main effects of Vision, Target Distance, and Target Size for Reaction Time (RT), Movement Time (MT), Peak Grip Aperture (PGA), Time After Peak Grip Aperture (TAPGA), and Time After Peak Velocity (TAPV) for Experiment II.

Main Effect	Means and Standard Deviations
RT	
<i>Vision</i>	NV = 262.85ms (± 40.83); FV = 220.75ms (± 33.85)
<i>Target Distance</i>	Long = 244.28ms (± 36.64); Short = 239.32ms (± 36.70)
MT	
<i>Vision</i>	NV = 1131.68ms (± 154.47); FV = 790.25ms (± 136.78)
<i>Target Distance</i>	Long = 1037.12ms (± 137.43); Short = 884.81ms (± 126.80)
<i>Target Size</i>	
PGA	
<i>Vision</i>	NV = 98.89mm (± 7.80); FV = 92.09mm (± 3.96)
<i>Target Size</i>	Large = 94.52mm (± 5.17); Small = 96.35mm (± 6.16)
TAPGA	
<i>Vision</i>	NV = 950.67ms (± 128.02); FV = 806.04ms (± 108.34)
<i>Target Distance</i>	Long = 857.76ms (± 111.17); Short = 736.29ms (± 106.64)
<i>Target Size</i>	Large = 786.07ms (± 105.54); Small = 807.99ms (± 112.30)
TAPV	
<i>Vision</i>	NV = 796.21ms (± 123.24); FV = 501.08ms (± 98.70)
<i>Target Distance</i>	Long = 711.12ms (± 102.74); Short = 586.16ms (± 93.89)
<i>Target Size</i>	Large = 602.01ms (± 88.34); Small = 618.96ms (± 96.59)

Table 6: Means and standard deviations for two-way interactions of Vision with Target Distance and/or Target Size for Movement Time (MT), Peak Grip Aperture (PGA), Time After Peak Grip Aperture (TAPGA), and Time After Peak Velocity (TAPV) for Experiment II.

Interaction	Means and Standard Deviations	
MT		
<i>Vision x Target Distance</i> $F(1,11) = 26.63, p < .05$		
<u>No Vision:</u>		<u>Vision:</u>
Long = 1221.46ms (± 158.06)		Long = 852.77ms (± 142.09)*
Short = 1041.90ms (± 152.72) [†]		Short = 727.72ms (± 132.08)* [†]
PGA		
<i>Vision x Target Size</i> $F(1,11) = 22.68, p < .05$		
<u>No Vision:</u>		<u>Vision:</u>
Large = 100.97mm (± 7.25)		Large = 95.85mm (± 3.93)*
Small = 96.81mm (± 8.52) [‡]		Small = 88.33mm (± 4.12)* [‡]
TAPGA		
<i>Vision x Target Size</i> $F(1,11) = 12.98, p < .05$		
<u>No Vision:</u>		<u>Vision:</u>
Large = 930.70ms (± 125.64)		Large = 641.44ms (± 115.05)*
Small = 970.64ms (± 132.18) [‡]		Small = 645.33ms (± 118.89)*
<i>Vision x Target Distance</i> $F(1,11) = 17.75, p < .05$		
<u>No Vision:</u>		<u>Vision:</u>
Long = 1022.86ms (± 127.95)		Long = 692.67ms (± 119.69)*
Short = 878.49ms (± 130.17) [†]		Short = 594.10ms (± 114.02)* [†]
TAPV		
<i>Vision x Target Size</i> $F(1,11) = 9.65, p < .05$		
<u>No Vision:</u>		<u>Vision:</u>
Large = 778.550ms (± 119.65)		Large = 500.73ms (± 98.42)*
Small = 813.86ms (± 129.18) [‡]		Small = 501.42ms (± 99.77)*
<i>Vision x Target Distance</i> $F(1,11) = 39.24, p < .05$		
<u>No Vision:</u>		<u>Vision:</u>
Long = 871.27ms (± 127.18)		Long = 550.96ms (± 102.91)*
Short = 721.14ms (± 120.52) [†]		Short = 451.19ms (± 96.17)* [†]

* Significant effect of Vision ($p < .05$)

† Significant effect of Target Distance ($p < .05$)

‡ Significant effect of Target Size ($p < .05$)

Table 7: Means and standard deviations for three-way interactions for Movement Time (MT), Peak Grip Aperture (PGA), Time After Peak Grip Aperture (TAPGA), and Time After Peak Velocity (TAPV) for Experiment II.

Interaction	Means and Standard Deviations	
MT		
<i>CE x Pre-Post x Target Distance</i> $F(1,11) = 5.36, p < .05$		
	<u>Control, Pre:</u>	<u>Control, Post:</u>
	Long = 1052.10ms (± 177.75)	Long = 1027.40ms (± 149.78)
	Short = 904.53ms (± 182.57) [‡]	Short = 883.47ms (± 137.95) [‡]
	<u>Experimental, Pre:</u>	<u>Experimental, Post:</u>
	Long = 1077.25ms (± 113.63)	Long = 991.72ms (± 144.14)**
	Short = 900.59ms (± 102.59) [‡]	Short = 850.66ms (± 127.29)** [‡]
<i>CE x Vision x Target Distance</i> $F(1,11) = 5.67, p < .05$		
	<u>Control, NV:</u>	<u>Control, FV:</u>
	Long = 1219.47ms (± 178.20)	Long = 860.02ms (± 166.24) [†]
	Short = 1035.17ms (± 178.35) [‡]	Short = 752.82ms (± 155.97) ^{†‡}
	<u>Experimental, NV:</u>	<u>Experimental, FV:</u>
	Long = 1223.45ms (± 151.58)	Long = 845.52ms (± 126.42) [†]
	Short = 1048.63ms (± 145.29) [‡]	Short = 702.63ms (± 113.74)** ^{†‡}
<i>Pre-Post x Vision x Target Distance</i> $F(1,11) = 13.64, p < .05$		
	<u>Pre, NV:</u>	<u>Pre, FV:</u>
	Long = 1258.83ms (± 169.57)	Long = 870.52ms (± 149.93) [†]
	Short = 1056.54ms (± 169.83) [‡]	Short = 748.58ms (± 144.80) ^{†‡}
	<u>Post, NV:</u>	<u>Post, FV:</u>
	Long = 1184.09ms (± 151.55)**	Long = 835.02ms (± 137.34)** [†]
	Short = 1027.26ms (± 142.32) [‡]	Short = 706.86ms (± 123.97)** ^{†‡}
PGA		
<i>CE x Vision x Target Size</i> $F(1,11) = 6.44, p < .05$		
	<u>Control, NV:</u>	<u>Control, FV:</u>
	Large = 100.42mm (± 8.14)	Large = 95.09mm (± 3.70) [†]
	Small = 95.03mm (± 8.18) ^{††}	Small = 87.53 mm (± 3.84) ^{†, ††}
	<u>Experimental, NV:</u>	<u>Experimental, FV:</u>
	Large = 101.53mm (± 7.65)	Large = 96.61mm (± 5.02) [†]
	Small = 98.58mm (± 9.98) ^{††}	Small = 89.13mm (± 5.15) ^{†, ††}
TAPGA		
<i>CE x Pre-Post x Target Distance</i> $F(1,11) = 5.61, p < .05$		
	<u>Control, Pre:</u>	<u>Control, Post:</u>
	Long = 869.97ms (± 143.21)	Long = 849.53ms (± 121.48)
	Short = 751.77ms (± 152.53) [‡]	Short = 737.05ms (± 115.90) [‡]
	<u>Experimental, Pre:</u>	<u>Experimental, Post:</u>
	Long = 890.89ms (± 92.85)	Long = 820.66ms (± 120.11)**
	Short = 746.59ms (± 88.59) [‡]	Short = 709.77ms (± 110.34) [‡]

Interaction**Means and Standard Deviations****TAPGA**

CE x Vision x Target Distance $F(1,11) = 6.45, p < .05$

Control, NV:

Long = 1020.55ms (± 139.86)

Short = 871.08ms (± 146.98)[‡]

Experimental, NV:

Long = 1025.16ms (± 128.69)

Short = 885.90ms (± 129.88)[‡]

Control, FV:

Long = 698.95ms (± 141.32)[†]

Short = 617.74ms (± 135.07)^{†‡}

Experimental, FV:

Long = 686.39ms (± 105.31)[†]

Short = 570.45ms (± 97.30)^{*†‡}

Pre-Post x Vision x Target Distance $F(1,11) = 14.44, p < .05$

Pre, NV:

Long = 1054.04ms (± 136.58)

Short = 886.97ms (± 141.61)[‡]

Post, NV:

Long = 991.68ms (± 124.96)^{**}

Short = 870.01ms (± 124.96)[‡]

Pre, FV:

Long = 706.82ms (± 126.37)[†]

Short = 611.39ms (± 125.73)^{†‡}

Post, FV:

Long = 678.51ms (± 116.13)^{**†}

Short = 576.81ms (± 106.80)^{*†‡}

TAPV

CE x Pre-Post x Target Distance $F(1,11) = 7.34, p < .05$

Control, Pre:

Long = 722.56ms (± 137.97)

Short = 605.10ms (± 141.35)[‡]

Experimental, Pre:

Long = 743.72ms (± 91.97)

Short = 594.45ms (± 84.93)[‡]

Control, Post:

Long = 703.80ms (± 104.90)

Short = 583.33ms (± 99.23)[‡]

Experimental, Post:

Long = 674.39ms (± 109.62)^{**}

Short = 561.78ms (± 102.72)[‡]

Pre-Post x Vision x Target Distance $F(1,11) = 10.59, p < .05$

Pre, NV:

Long = 902.78ms (± 140.18)

Short = 732.78ms (± 136.55)[‡]

Post, NV:

Long = 839.77ms (± 119.74)^{**}

Short = 709.50ms (± 112.40)[‡]

Pre, FV:

Long = 563.59ms (± 113.46)[†]

Short = 466.77ms (± 110.41)^{†‡}

Post, FV:

Long = 538.42ms (± 95.06)[†]

Short = 435.61ms (± 87.88)^{†‡}

* Significant effect of CE ($p < .05$)

** Significant effect of Pre-Post ($p < .05$)

† Significant effect of Vision ($p < .05$)

†† Significant effect of Target Size ($p < .05$)

‡ Significant effect of Target Distance ($p < .05$)

Table 8: Means and standard deviations for main effects and interactions of Pre-to-Post differences analysis for Movement Time (MT), Time After Peak Grip Aperture (TAPGA) and Time After Peak Velocity (TAPV) for Experiment II.

Interaction	Means and Standard Deviations	
MT		
<i>Target Distance</i> $F(1,11) = 5.22, p < .05$		
	Long = 55.12ms (± 35.60)	Short = 35.50ms (± 42.53)
<i>CE x Target Distance</i> $F(1,11) = 8.50, p < .05$		
	<u>Control:</u>	<u>Experimental:</u>
	Long = 19.76ms (± 49.63)	Long = 85.53ms (± 83.19)*
	Short = 16.85ms (± 76.77)	Short = 49.94ms (± 73.11)**
<i>Vision x Target Distance</i> $F(1,11) = 13.65, p < .05$		
	<u>NV:</u>	<u>FV:</u>
	Long = 74.73ms (± 59.22)	Long = 35.50ms (± 43.90)
	Short = 29.28ms (± 70.02)**	Short = 41.72ms (± 53.79)
TAPGA		
<i>Target Distance</i> $F(1,11) = 5.73, p < .05$		
	Long = 45.34ms (± 34.20)	Short = 25.77ms (± 39.59)
<i>CE x Target Distance</i> $F(1,11) = 9.42, p < .05$		
	<u>Control:</u>	<u>Experimental:</u>
	Long = 16.35ms (± 42.79)	Long = 70.23ms (± 73.04)*
	Short = 11.78ms (± 67.25)	Short = 36.92ms (± 66.90)**
<i>Vision x Target Distance</i> $F(1,11) = 14.44, p < .05$		
	<u>NV:</u>	<u>FV:</u>
	Long = 62.63ms (± 55.31)	Long = 28.31ms (± 40.07)
	Short = 16.96ms (± 59.73)*	Short = 34.59ms (± 49.23)
TAPV		
<i>CE x Target Distance</i> $F(1,11) = 10.84, p < .05$		
	<u>Control:</u>	<u>Experimental:</u>
	Long = 15.01ms (± 43.67)	Long = 69.33ms (± 65.95)*
	Short = 17.41ms (± 70.91)	Short = 32.67ms (± 69.41)**
<i>Vision x Target Distance</i> $F(1,11) = 10.59, p < .05$		
	<u>NV:</u>	<u>FV:</u>
	Long = 63.00ms (± 57.28)	Long = 25.09ms (± 38.15) [†]
	Short = 23.28ms (± 66.77)**	Short = 31.15ms (± 53.22)

* Significant effect of CE ($p < .05$)

** Significant effect of Target Distance ($p < .05$)

† Significant effect of Vision ($p < .05$)

Table 9: Mean grip apertures and standard deviations for decile main effect for Experiment II decile analysis.

Decile	
<i>Decile</i>	<i>Mean Grip Aperture (mm)</i>
0%	16.80 (± 4.00)
10%	20.52 (± 4.25)*
20%	37.98 (± 10.06)*
30%	58.85 (± 12.37)*
40%	75.47 (± 10.71)*
50%	85.59 (± 7.38)*
60%	89.19 (± 5.66)*
70%	87.78 (± 5.11)
80%	84.00 (± 4.14)*
90%	79.00 (± 2.69)*
100%	75.27 (± 2.22)*

* Significant main effect of Decile ($p < .05$)

Table 10: Mean grip apertures and standard deviations for two-way interactions of decile analysis for Experiment II.

<i>Decile</i>	Day		Visual Condition	
	Control	Experimental	No Vision	Vision
	<i>Mean Grip Aperture (mm)</i>			
0%	16.85 (±4.81)	16.76 (±4.00)	16.87 (±3.88)	16.74 (±4.20)
10%	20.39 (±5.62)	20.66 (±3.67)	22.11 (±4.95)	18.94 (±4.23) [†]
20%	37.44 (±11.95)	38.53 (±8.78)	41.74 (±13.01)	34.23 (±(8.63)) [†]
30%	57.29 (±14.02)	60.40 (±11.57)	64.33 (±15.50)	53.36 (±11.40) [†]
40%	73.63 (±11.56)	77.32 (±10.91)	81.30 (±12.84)	69.64 (±10.51) [†]
50%	83.91 (±7.49)	87.26 (±8.32)	89.71 (±9.00)	81.74 (±7.54) [†]
60%	87.84 (±5.35)	90.54 (±6.85)	90.52 (±8.02)	87.86 (±4.68)
70%	86.87 (±5.31)	88.69 (±5.53)	87.31 (±7.43)	88.25 (±3.44)
80%	83.60 (±4.61)	84.41 (±4.10)	84.05 (±5.42)	83.96 (±3.44)
90%	78.89 (±3.14)	84.38 (±3.16)*	79.54 (±3.24)	78.45 (±2.41)
100%	75.31 (±2.90)	75.23 (±2.48)	74.97 (±2.31)	75.58 (±2.29)

* Significant effect of CE ($p < .05$) in CE x Decile

† Significant effect of Vision ($p < .05$) in Vision x Decile

<i>Decile</i>	Target Size		Target Distance	
	Large	Small	Long	Short
	<i>Mean Grip Aperture (mm)</i>			
0%	16.77 (±4.00)	16.83 (±4.00)	16.73 (±4.01)	16.87 (±4.00) [†]
10%	20.53 (±4.37)	20.51 (±4.13)	20.60 (±4.54)	20.45 (±4.02)
20%	38.35 (±10.33)	37.62 (±9.81)	36.97 (±11.44)	39.00 (±9.05)
30%	59.88 (±12.92)	57.81 (±11.88)*	55.62 (±13.88)	62.07 (±11.36) [†]
40%	77.16 (±11.33)	73.79 (±10.21)*	71.89 (±12.48)	79.05 (±9.24) [†]
50%	88.01 (±7.71)	83.16 (±7.32)*	83.23 (±9.01)	87.94 (±6.19) [†]
60%	92.29 (±5.67)	86.09 (±5.94)*	88.62 (±6.08)	89.76 (±5.61)
70%	91.56 (±5.11)	84.01 (±5.33)*	88.29 (±4.98)	87.28 (±5.33) [†]
80%	88.57 (±4.03)	79.43 (±4.41)*	84.60 (±4.24)	83.40 (±4.07) [†]
90%	84.63 (±2.64)	73.74 (±2.81)*	79.32 (±2.68)	78.68 (±2.72) [†]
100%	81.13 (±2.30)	69.41 (±2.17)*	75.12 (±2.22)	75.42 (±2.24) [†]

* Significant effect of Target Size ($p < .05$) in Target Size x Decile

† Significant effect of Target Distance ($p < .05$) in Target Distance x Decile

Table 11: Mean grip apertures and standard deviations for three-way interaction of Decile, Vision, and Target Size for Experiment II grip aperture decile analysis.

<i>Decile</i>	No Vision		Vision	
	Large	Small	Large	Small
	<i>Mean Grip Aperture (mm)</i>			
0%	16.87 (± 3.87)	16.87 (± 3.89)	16.68 (± 4.22)	16.79 (± 4.18)
10%	22.20 (± 5.27)	22.01 (± 4.67)	18.86 (± 4.13)	19.02 (± 4.33)
20%	42.00 (± 13.38)	41.49 (± 12.69)	34.71 (± 8.56)	33.75 (± 8.81)
30%	64.69 (± 16.02)	63.96 (± 15.10)	55.07 (± 11.82)	51.66 (± 11.24)*
40%	82.10 (± 13.30)	80.51 (± 12.59)	72.22 (± 11.27)	67.07 (± 10.00)*
50%	91.54 (± 8.91)	87.87 (± 9.45)*	84.48 (± 8.20)	78.45 (± 7.11)*
60%	93.33 (± 7.54)	87.72 (± 8.82)*	91.26 (± 5.09)	84.46 (± 4.47)*
70%	90.81 (± 7.30)	83.81 (± 7.87)*	92.32 (± 3.55)	84.20 (± 3.51)*
80%	88.29 (± 5.53)	79.81 (± 5.59)*	88.86 (± 3.18)	79.06 (± 3.78)*
90%	84.54 (± 3.29)	74.50 (± 3.39)*	84.13 (± 2.30)	72.78 (± 2.55)*
100%	80.76 (± 2.42)	69.17 (± 2.25)*	81.50 (± 2.34)	69.65 (± 2.28)*

* Significant effect of Target Size ($p < .05$)

Table 12: Mean grip apertures and standard deviations for three-way interaction of Decile, Control-Experimental (CE), and Target Distance for Experiment II grip aperture decile analysis.

<i>Decile</i>	Control		Experimental	
	Long	Short	Long	Short
	<i>Mean Grip Aperture (mm)</i>			
0%	14.92 (± 4.30)	15.04 (± 4.26)	16.68 (± 3.97)	16.83 (± 4.02)
10%	18.10 (± 4.95)	18.15 (± 5.08)	20.84 (± 4.25) [†]	20.48 (± 3.22)
20%	32.32 (± 11.50)	34.32 (± 10.19)	37.58 (± 10.54) [†]	39.48 (± 7.43)*
30%	47.74 (± 13.60)	54.10 (± 11.88)	57.55 (± 13.22) [†]	63.26 (± 10.44)*
40%	61.79 (± 11.75)	69.11 (± 9.22)	74.28 (± 12.66) [†]	80.36 (± 9.49)*
50%	72.10 (± 8.17)	77.08 (± 5.69)	85.36 (± 9.68) [†]	89.17 (± 7.42)*
60%	77.26 (± 5.01)	78.89 (± 4.96)	90.31 (± 7.27) [†]	90.77 (± 6.70)*
70%	77.54 (± 4.54)	76.90 (± 4.98)	89.35 (± 5.37) [†]	88.04 (± 5.84)*
80%	74.95 (± 4.34)	73.67 (± 3.91)	84.89 (± 3.98) [†]	83.93 (± 4.33)*
90%	70.51 (± 2.82)	69.73 (± 2.80)	79.31 (± 2.94) [†]	78.91 (± 3.04)*
100%	66.77 (± 2.62)	67.12 (± 2.54)	75.12 (± 2.47) [†]	75.33 (± 2.51)*

* Significant effect of CE to Short targets ($p < .05$)

† Significant effect of CE to Long targets ($p < .05$)

Table 13: Mean grip apertures and standard deviations for three-way interaction of Decile, Vision, and Target Distance for Experiment II grip aperture decile analysis.

<i>Decile</i>	No Vision		Vision	
	Long	Short	Long	Short
	<i>Mean Grip Aperture (mm)</i>			
0%	16.76 (± 3.85)	16.98 (± 3.91)**	16.70 (± 4.22)	16.77 (± 4.18)
10%	22.00 (± 5.45)	22.22 (± 4.53)	19.21 (± 4.35) [‡]	18.68 (± 4.15) [†]
20%	39.33 (± 14.41)	44.15 (± 12.10)**	34.60 (± 9.83)	33.85 (± 7.90) [†]
30%	59.44 (± 17.59)	69.21 (± 14.21)**	51.81 (± 12.41)	54.92 (± 10.69) ^{*†}
40%	77.01 (± 15.59)	85.60 (± 10.51)**	66.78 (± 11.63) [‡]	72.50 (± 9.59) ^{*†}
50%	87.61 (± 11.48)	91.80 (± 7.29)	78.85 (± 8.54) [‡]	84.08 (± 6.75) ^{*†}
60%	90.52 (± 8.79)	90.53 (± 7.72)	86.71 (± 5.33)	89.00 (± 4.41) [*]
70%	88.02 (± 7.55)	86.61 (± 7.46)	88.56 (± 3.44)	87.94 (± 3.72)
80%	84.50 (± 5.58)	83.60 (± 5.37)	84.71 (± 3.64)	83.20 (± 3.30) [*]
90%	79.86 (± 3.26)	79.22 (± 3.34)	78.77 (± 2.50)	78.14 (± 2.36) [*]
100%	74.88 (± 2.31)	75.05 (± 2.37)	75.36 (± 2.31)	75.79 (± 2.30) [*]

* Significant effect of Target Distance in Vision ($p < .05$)

** Significant effect of Target Distance in NV ($p < .05$)

[†] Significant effect of Vision to Short targets ($p < .05$)

[‡] Significant effect of Vision to Long targets ($p < .05$)

Table 14: Mean grip apertures and standard deviations for three-way interaction of Decile, Target Size, and Target Distance for Experiment II grip aperture decile analysis.

<i>Decile</i>	Large		Small	
	Long	Short	Long	Short
	<i>Mean Grip Aperture (mm)</i>			
0%	16.67 (± 3.95)	16.88 (± 4.06) [†]	16.80 (± 4.07)**	16.87 (± 3.94)
10%	20.72 (± 4.74)	20.35 (± 4.07)	20.49 (± 4.36)	20.54 (± 3.98)
20%	37.63 (± 12.14)	39.07 (± 8.89)	36.31 (± 10.80)**	38.93 (± 9.26)
30%	56.77 (± 14.65)	62.99 (± 11.74) [†]	54.48 (± 13.16)**	61.14 (± 11.09) ^{*‡}
40%	73.55 (± 13.09)	80.77 (± 9.91) [†]	70.24 (± 11.99)**	77.34 (± 8.79) ^{*‡}
50%	85.48 (± 9.38)	90.55 (± 6.41) [†]	80.99 (± 8.86)**	85.33 (± 6.36) ^{*‡}
60%	91.50 (± 6.36)	93.09 (± 5.29)	85.73 (± 6.10)**	86.44 (± 6.19) [*]
70%	91.77 (± 5.40)	91.35 (± 4.94)	84.81 (± 4.88)**	83.21 (± 5.91) ^{*‡}
80%	88.82 (± 4.17)	88.33 (± 3.98)	80.39 (± 4.53)**	78.48 (± 4.31) ^{*‡}
90%	84.40 (± 2.61)	84.31 (± 2.73)	74.23 (± 2.87)**	73.05 (± 2.80) ^{*‡}
100%	80.86 (± 2.31)	81.39 (± 2.32) [†]	69.38 (± 2.17)**	69.45 (± 2.18) [*]

* Significant effect of Target Size to Short targets ($p < .05$)

** Significant effect of Target Size to Long targets ($p < .05$)

[†] Significant effect of Target Distance to Large targets ($p < .05$)

[‡] Significant effect of Target Distance to Small targets ($p < .05$)

Table 15: Mean grip apertures and standard deviations for three-way interaction of Control-Experimental (CE), Vision, and Target Distance for Experiment II grip aperture decile analysis.

	Control		Experimental	
	No Vision	Vision	No Vision	Vision
	<i>Mean Grip Aperture (mm)</i>			
<i>Long</i>	63.95 (± 0.96)	60.72 (± 5.09) [†]	65.97 (± 6.04)	62.40 (± 4.41) [†]
<i>Short</i>	66.19 (± 5.29) [*]	62.56 (± 4.61) ^{*†}	68.24 (± 4.81) [*]	62.94 (± 3.65) [†]

* Significant effect of Target Distance ($p < .05$)

† Significant effect of Vision ($p < .05$)

Table 16: Mean grip apertures and standard deviations for Decile main effect in last 5 Pre-test trials and first 5 Post-test trials in decile analysis for Experiment II.

Decile	
<i>Decile</i>	<i>Mean Grip Aperture (mm)</i>
0%	16.65 (± 3.84)
10%	20.96 (± 4.68) [*]
20%	38.77 (± 11.28) [*]
30%	59.12 (± 13.51) [*]
40%	75.54 (± 11.92) [*]
50%	85.46 (± 8.70) [*]
60%	89.34 (± 6.34) [*]
70%	88.02 (± 5.40)
80%	84.20 (± 4.38) [*]
90%	79.16 (± 2.92) [*]
100%	75.38 (± 2.62) [*]

* Significant main effect of Decile ($p < .05$)

Table 17: Mean grip apertures and standard deviations for two-way interaction of last 5 Pre-test trials and first 5 Post-test trials analysis for Experiment II.

<i>Decile</i>	Day		Visual Condition	
	Control	Experimental	No Vision	Vision
	<i>Mean Grip Aperture (mm)</i>			
0%	16.64 (±4.54)	16.66 (±4.12)	16.52 (±3.70)	16.78 (±4.05)
10%	21.05 (±6.22)	20.87 (±3.93)	22.60 (±5.93)	19.32 (±4.14)*
20%	38.89 (±13.77)	38.64 (±9.83)	42.71 (±15.42)	34.83 (±8.78)*
30%	57.96 (±14.83)	60.29 (±13.20)	65.19 (±17.60)	53.05 (±12.07)*
40%	73.80 (±12.01)	77.28 (±12.78)	82.12 (±14.64)	68.95 (±11.51)*
50%	83.83 (±8.36)	87.09 (±10.09)	90.15 (±11.27)	80.77 (±8.34)*
60%	87.89 (±5.87)	90.79 (±8.04)	91.15 (±9.41)	87.52 (±4.77)
70%	86.83 (±5.12)	89.21 (±6.54)	87.61 (±8.08)	88.43 (±3.40)
80%	83.40 (±4.52)	84.99 (±4.88)	83.99 (±5.65)	84.41 (±3.82)
90%	78.93 (±3.24)	79.40 (±3.51)	79.77 (±3.84)	78.56 (±2.67)
100%	75.60 (±3.49)	75.16 (±2.67)	75.39 (±2.87)	75.46 (±2.77)

* Significant effect of Vision ($p < .05$) in Vision x Decile

Table 18: Mean grip apertures and standard deviations for three-way interaction of Decile, Pre-Post, and Vision in last 5 Pre-test trials and first 5 Post-test trials analysis for Experiment II.

<i>Decile</i>	Pre		Post	
	No Vision	Vision	No Vision	Vision
	<i>Mean Grip Aperture (mm)</i>			
0%	16.60 (±4.11)	16.81 (±4.64)	16.44 (±3.75)	16.75 (±4.05)
10%	21.20 (±5.05)	19.69 (±5.41)	24.00 (±7.88)	18.95 (±4.05)*
20%	39.80 (±13.01)	37.07 (±10.17)	45.62 (±18.21) [†]	32.59 (±9.42)*
30%	61.47 (±19.27)	54.54 (±13.31)	68.92 (±17.03) [†]	51.57 (±12.01)*
40%	78.52 (±18.99)	69.11 (±12.01)	85.73 (±11.83)	68.80 (±12.11)*
50%	87.25 (±14.18)	80.23 (±8.63)	93.06 (±9.91)	81.30 (±9.08)*
60%	90.17 (±10.42)	86.85 (±5.02)	92.13 (±9.57)	88.19 (±5.62)
70%	87.54 (±8.78)	87.68 (±3.80)	87.67 (±8.60)	89.19 (±4.75)
80%	83.86 (±6.23)	84.05 (±4.77)	84.12 (±5.60)	84.77 (±4.96)
90%	79.77 (±4.78)	79.10 (±4.26)	79.77 (±4.17)	78.01 (±3.56)
100%	75.07 (±4.31)	76.15 (±4.80)	75.54 (±2.76)	74.77 (±3.19)

* Significant effect of Vision in Post ($p < .05$)

[†] Significant effect of Pre-Post in NV ($p < .05$)

Table 19: Mean proportion of explained variance (R^2) and standard deviations between sequential decile and movement endpoint for Decile main effect in correlation analysis for Experiment II.

Decile	
<i>Decile</i>	R^2
0%	0.06 (± 0.05)
10%	0.09 (± 0.04)
20%	0.10 (± 0.09)*
30%	0.13 (± 0.10)*
40%	0.18 (± 0.10)*
50%	0.26 (± 0.10)*
60%	0.38 (± 0.12)*
70%	0.56 (± 0.14)*
80%	0.77 (± 0.11)*
90%	0.93 (± 0.04)*
100%	1.00 (± 0.00)

* Significant main effect of Decile ($p < .05$)

Table 20: Mean proportion of explained variance (R^2) and standard deviations between sequential decile and movement endpoint for two-way interaction of Vision and Decile in correlation analysis for Experiment II.

<i>Decile</i>	Visual Condition	
	No Vision	Vision
	R^2	
0%	0.09 (± 0.07)	0.03 (± 0.09)
10%	0.14 (± 0.05)	0.04 (± 0.08)*
20%	0.16 (± 0.10)	0.05 (± 0.11)*
30%	0.19 (± 0.12)	0.06 (± 0.10)*
40%	0.26 (± 0.13)	0.09 (± 0.10)*
50%	0.37 (± 0.15)	0.15 (± 0.10)*
60%	0.48 (± 0.18)	0.27 (± 0.10)*
70%	0.60 (± 0.20)	0.51 (± 0.11)
80%	0.76 (± 0.15)	0.78 (± 0.08)
90%	0.92 (± 0.05)	0.94 (± 0.03)
100%	1.00 (± 0.00)	1.00 (± 0.00)

* Significant effect of Vision ($p < .05$)

Table 21: Mean proportion of explained variance (R^2) and standard deviations between sequential decile and movement endpoint for three-way interaction of Decile, Pre-Post, and Vision in correlation analysis for Experiment II.

	Pre		Post	
	No Vision	Vision	No Vision	Vision
<i>Decile</i>			R^2	
0%	0.09 (± 0.09)	0.02 (± 0.11)	0.10 (± 0.11)	0.03 (± 0.17)
10%	0.09 (± 0.14)	0.06 (± 0.10)	0.20 (± 0.13)	0.02 (± 0.11)*
20%	0.11 (± 0.17)	0.12 (± 0.16)	0.20 (± 0.19)	-0.02 (± 0.11)* [†]
30%	0.15 (± 0.18)	0.14 (± 0.15)	0.24 (± 0.19)	-0.01 (± 0.12)* [†]
40%	0.22 (± 0.19)	0.16 (± 0.14)	0.31 (± 0.19)	0.03 (± 0.13)* [†]
50%	0.32 (± 0.19)	0.21 (± 0.13)	0.41 (± 0.20)	0.09 (± 0.13)* [†]
60%	0.44 (± 0.19)	0.33 (± 0.12)	0.41 (± 0.20)	0.27 (± 0.10)* [†]
70%	0.59 (± 0.19)	0.56 (± 0.10)	0.62 (± 0.23)	0.47 (± 0.16)
80%	0.76 (± 0.15)	0.79 (± 0.06)	0.76 (± 0.17)	0.76 (± 0.11)
90%	0.93 (± 0.05)	0.95 (± 0.03)	0.91 (± 0.07)	0.94 (± 0.03)
100%	1.00 (± 0.00)			

* Significant effect of Vision ($p < .05$)

[†] Significant effect of Pre-Post ($p < .05$)

Table 22: Mean proportion of explained variance (R^2) and standard deviations between sequential decile and movement endpoint for three-way interaction of Pre-Post, Target Size, and Target Distance in correlation analysis for Experiment II.

	Pre		Post	
	Large	Small	Large	Small
			R^2	
<i>Long</i>	0.36 (± 0.11)	0.41 (± 0.10)	0.35 (± 0.12)	0.35 (± 0.11)
<i>Short</i>	0.44 (± 0.11)	0.43 (± 0.15)	0.40 (± 0.14)	0.44 (± 0.09)

Table 23: Mean variability in limb position and standard deviations for main effect of Decile and two-way interaction of Decile and Vision in variability analysis for Experiment II.

<i>Decile</i>	<i>Variability (mm)</i>	Visual Condition	
		No Vision	Vision
0%	4.12 (± 2.01)	4.08 (± 1.84)	4.16 (± 2.21)
10%	5.37 (± 1.60)*	6.07 (± 1.29)	4.67 (± 2.09) [†]
20%	15.50 (± 2.90)*	20.27 (± 5.57)	10.72 (± 1.68) [†]
30%	28.58 (± 4.74)*	35.02 (± 7.20)	22.14 (± 3.47) [†]
40%	30.92 (± 3.72)*	33.63 (± 5.12)	28.21 (± 4.46) [†]
50%	24.61 (± 3.54)*	24.74 (± 4.70)	24.47 (± 4.67)
60%	17.22 (± 2.97)*	17.82 (± 3.07)	16.62 (± 4.39)
70%	12.17 (± 2.19)*	13.94 (± 2.12)	10.40 (± 3.50) [†]
80%	0.77 (± 0.11)*	11.10 (± 2.01)	7.30 (± 2.79) [†]
90%	9.20 (± 1.89)*	8.85 (± 2.05)	6.25 (± 2.64) [†]
100%	7.55 (± 1.92)*	7.83 (± 2.09)	6.07 (± 2.64)

* Significant effect of Decile ($p < .05$)

† Significant effect of Vision ($p < .05$)

Table 24: Mean variability in limb position and standard deviations for two-way interactions of Decile and Target Size and Decile and Target Distance in variability analysis for Experiment II.

<i>Decile</i>	Target Size		Target Distance	
	Large	Small	Long	Short
0%	4.12 (± 2.04)	4.13 (± 1.99)	3.99 (± 1.95)	3.77 (± 1.85)
10%	5.35 (± 1.68)	5.39 (± 1.57)	5.51 (± 1.44)	4.60 (± 1.61) [†]
20%	15.73 (± 2.83)	15.26 (± 3.16)	17.50 (± 3.89)	11.66 (± 1.78) [†]
30%	29.49 (± 5.12)	27.66 (± 4.72)*	31.94 (± 5.79)	21.85 (± 3.43) [†]
40%	32.19 (± 4.16)	29.65 (± 3.97)*	33.21 (± 4.63)	24.99 (± 2.57) [†]
50%	25.53 (± 3.64)	23.69 (± 4.10)	25.31 (± 4.31)	21.01 (± 2.51) [†]
60%	17.50 (± 3.09)	16.94 (± 3.56)	17.11 (± 3.48)	15.31 (± 2.35) [†]
70%	12.03 (± 2.34)	12.31 (± 2.89)	11.91 (± 2.48)	11.00 (± 1.97)
80%	8.82 (± 1.77)	9.58 (± 2.83)	9.05 (± 2.12)	8.27 (± 1.84)
90%	7.04 (± 1.48)	8.06 (± 3.06)	7.36 (± 2.22)	6.85 (± 1.85)
100%	6.46 (± 1.51)	7.45 (± 3.15)	6.71 (± 2.23)	6.37 (± 1.87)

* Significant effect of Target Size ($p < .05$)

† Significant effect of Target Distance ($p < .05$)

Table 25: Mean variability in limb position and standard deviations for three-way interaction of Decile, Vision, and Target Distance in variability analysis for Experiment II.

<i>Decile</i>	No Vision		Vision	
	Long	Short	Long	Short
	<i>Variability (mm)</i>			
0%	4.27 (± 1.90)	3.89 (± 1.82)*	4.20 (± 2.29)	4.12 (± 2.19)
10%	6.90 (± 1.28)	5.25 (± 1.41)*	4.80 (± 2.09) [†]	4.53 (± 2.12)
20%	24.78 (± 7.93)	15.76 (± 3.42)*	12.41 (± 1.88) [†]	9.03 (± 1.90)* [†]
30%	41.45 (± 9.84)	28.59 (± 5.07)*	26.43 (± 3.93) [†]	17.84 (± 3.82)* [†]
40%	37.62 (± 6.93)	29.64 (± 4.22)*	32.96 (± 5.13)	23.46 (± 4.44)* [†]
50%	25.90 (± 5.72)	23.58 (± 4.30)	27.89 (± 5.75)	21.06 (± 3.88)*
60%	17.69 (± 3.66)	17.96 (± 2.82)	18.66 (± 5.34)	14.57 (± 3.77)* [†]
70%	13.75 (± 2.47)	14.12 (± 2.14)	11.56 (± 4.30)	9.25 (± 3.23)* [†]
80%	11.16 (± 2.18)	11.04 (± 2.31)	8.06 (± 3.69) [†]	6.53 (± 2.66) [†]
90%	8.76 (± 2.19)	8.94 (± 2.33)	6.89 (± 3.57)	5.61 (± 2.56) [†]
100%	7.56 (± 2.16)	8.10 (± 2.29)	6.71 (± 3.52)	5.44 (± 2.60) [†]

* Significant effect of Target Distance ($p < .05$)

[†] Significant effect of Vision ($p < .05$)

FIGURES

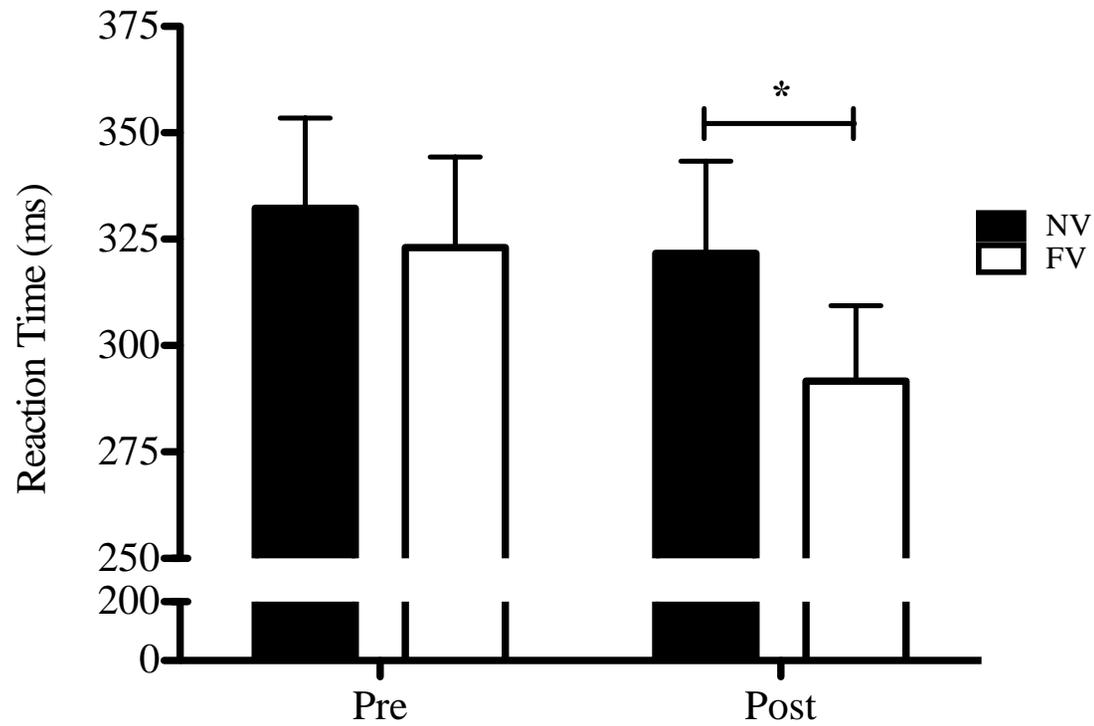


Figure 1: Reaction time (RT) Pre- and Post- 2-hour visual deprivation in Full Vision (FV) and No Vision (NV) conditions. RT was significantly faster in FV in the Post-test ($p < .05$).

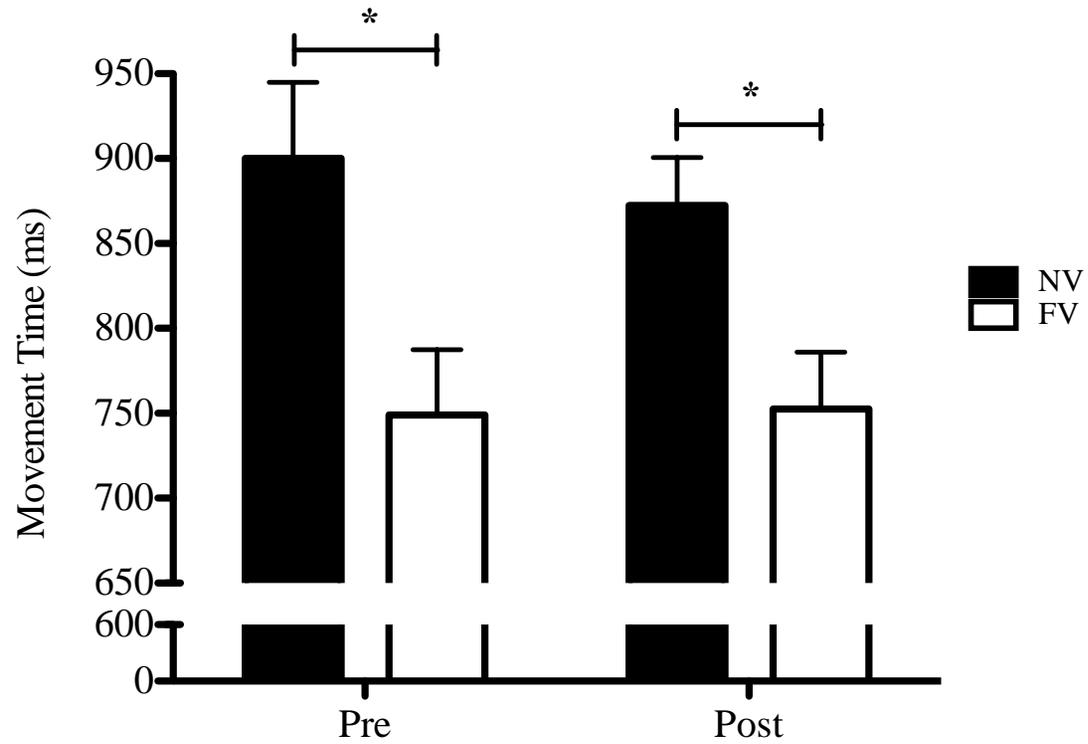


Figure 2: Movement Time (MT) Pre- and Post- 2-hour visual deprivation in Full Vision (FV) and No Vision (NV) conditions. MT was significantly longer in NV, both Pre- and Post-deprivation ($p < .05$).

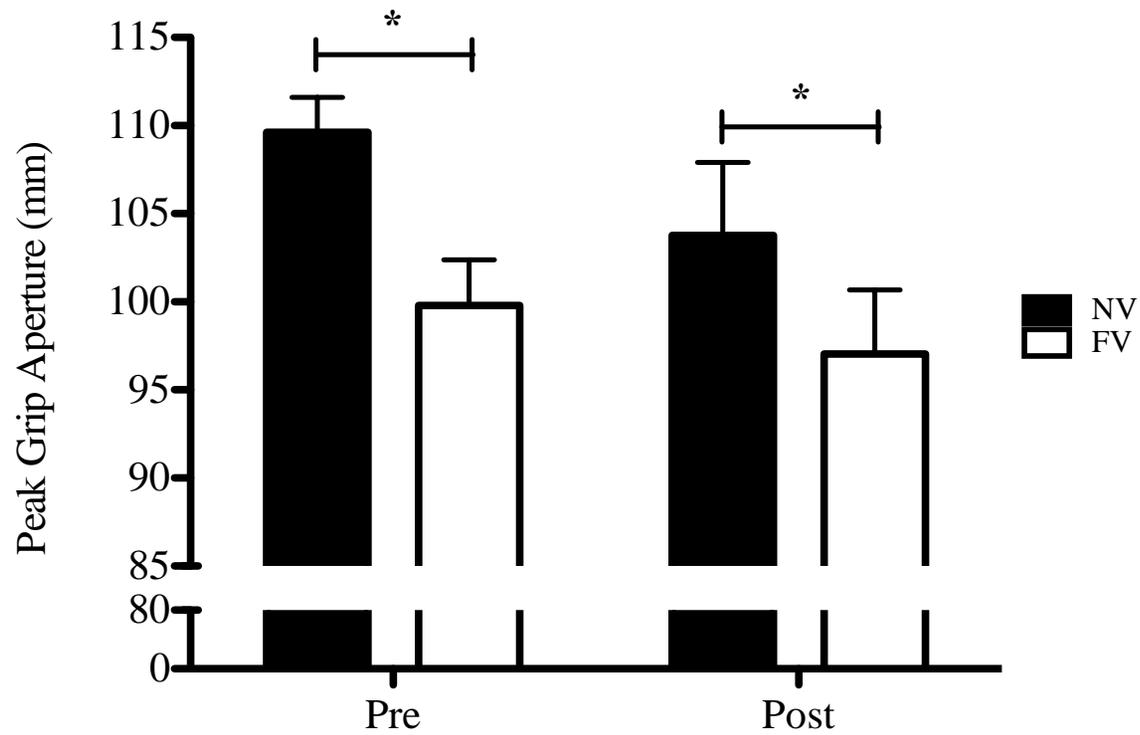


Figure 3: Peak grip aperture (PGA) Pre- and Post- 2-hour visual deprivation in Full Vision (FV) and No Vision (NV) conditions. PGA was significantly narrower in FV conditions, both Pre- and Post-deprivation ($p < .05$).

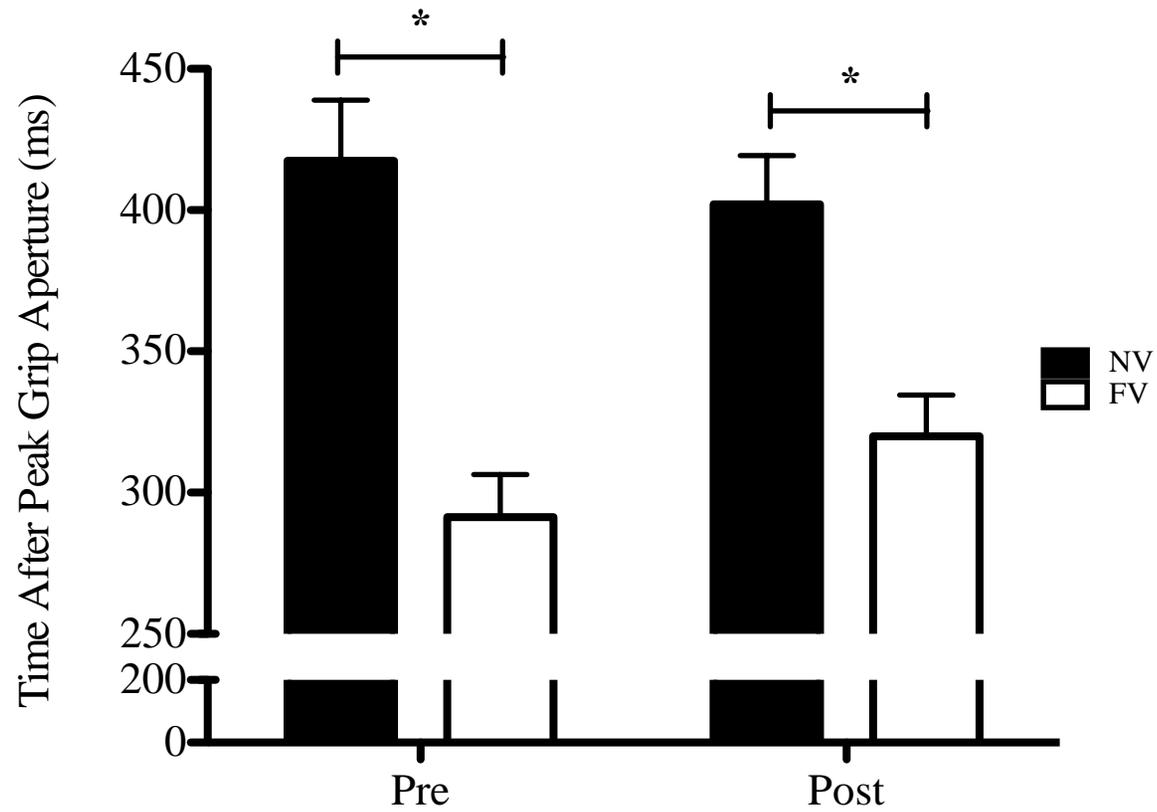


Figure 4: Time after peak grip aperture (TAPGA) Pre- and Post- 2-hour visual deprivation in Full Vision (FV) and No Vision (NV) conditions. TAPGA was significantly longer in NV conditions both Pre- and Post-deprivation ($p < .05$).

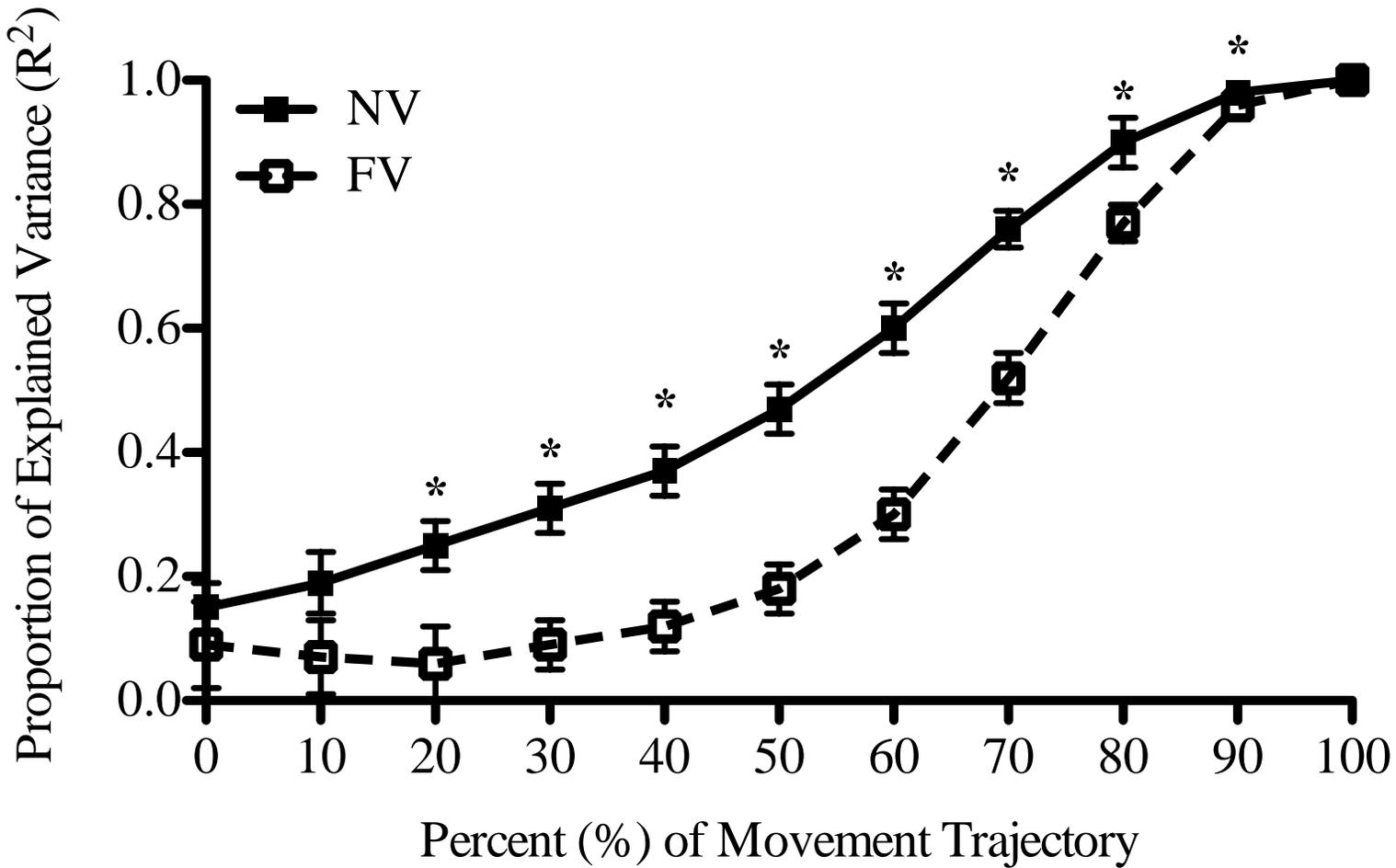


Figure 5: Proportion of explained variance (R^2) across deciles in Full Vision (FV) and No Vision (NV) conditions. Prior limb position was significantly more correlated with movement endpoint in NV, specifically from 20% movement trajectory onward ($p < .05$).

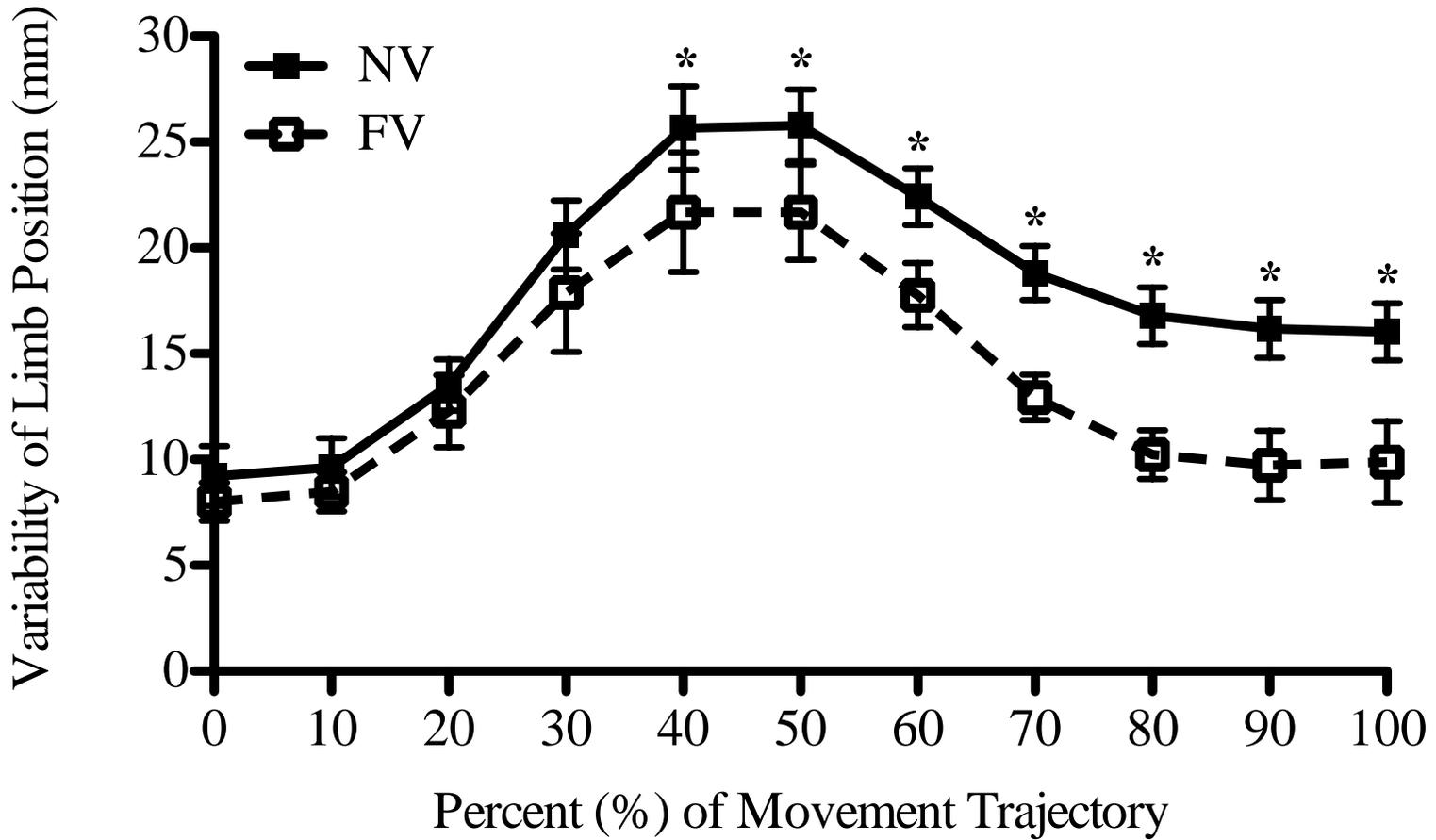


Figure 6: Variability of limb position across deciles in Full Vision (FV) and No Vision (NV) conditions. Variability of limb position within each decile was significantly greater in NV, specifically from 40% of movement trajectory onward ($p < .05$).

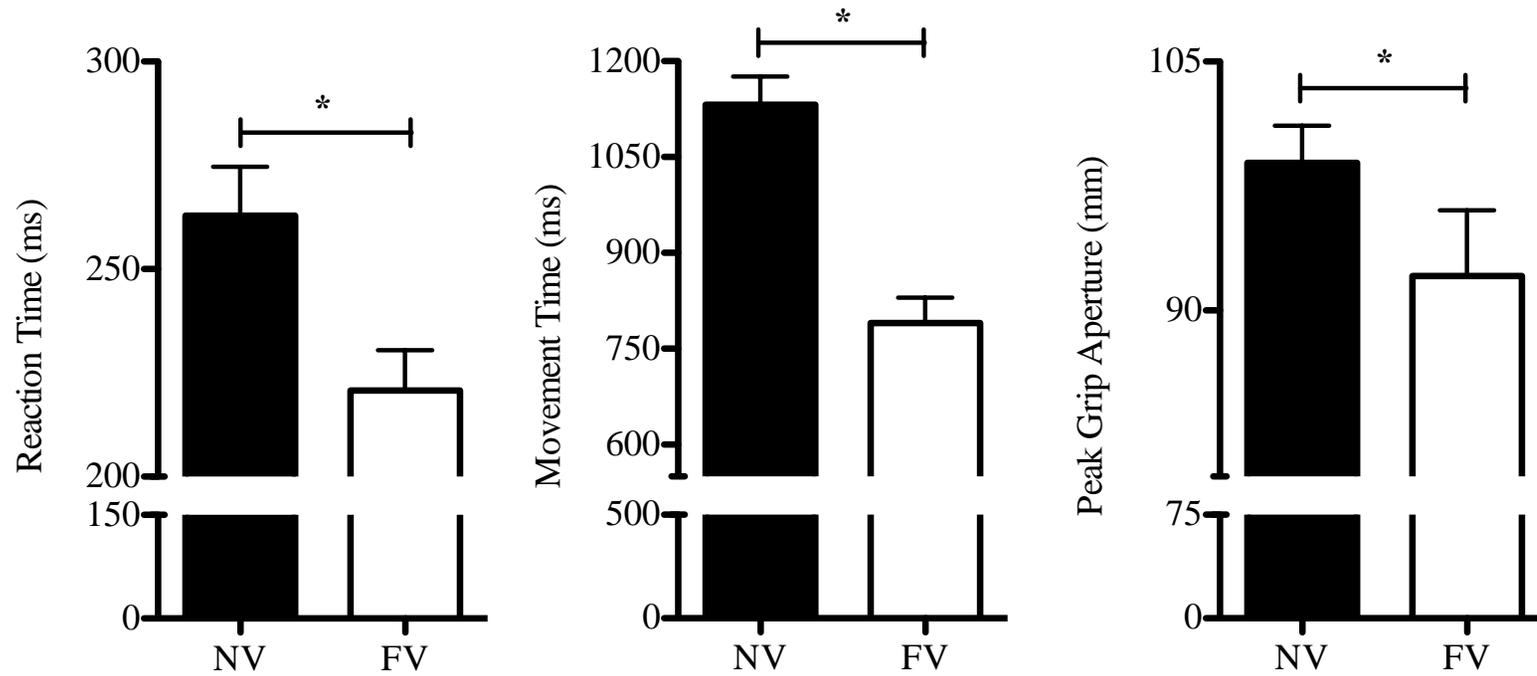


Figure 7: Main effect of Vision for Reaction Time (RT; left), Movement Time (MT; middle), and Peak Grip Aperture (PGA; right). RT and MT were significantly faster in FV than NV ($p > .05$), and PGA was significantly smaller in FV than NV ($p < .05$).

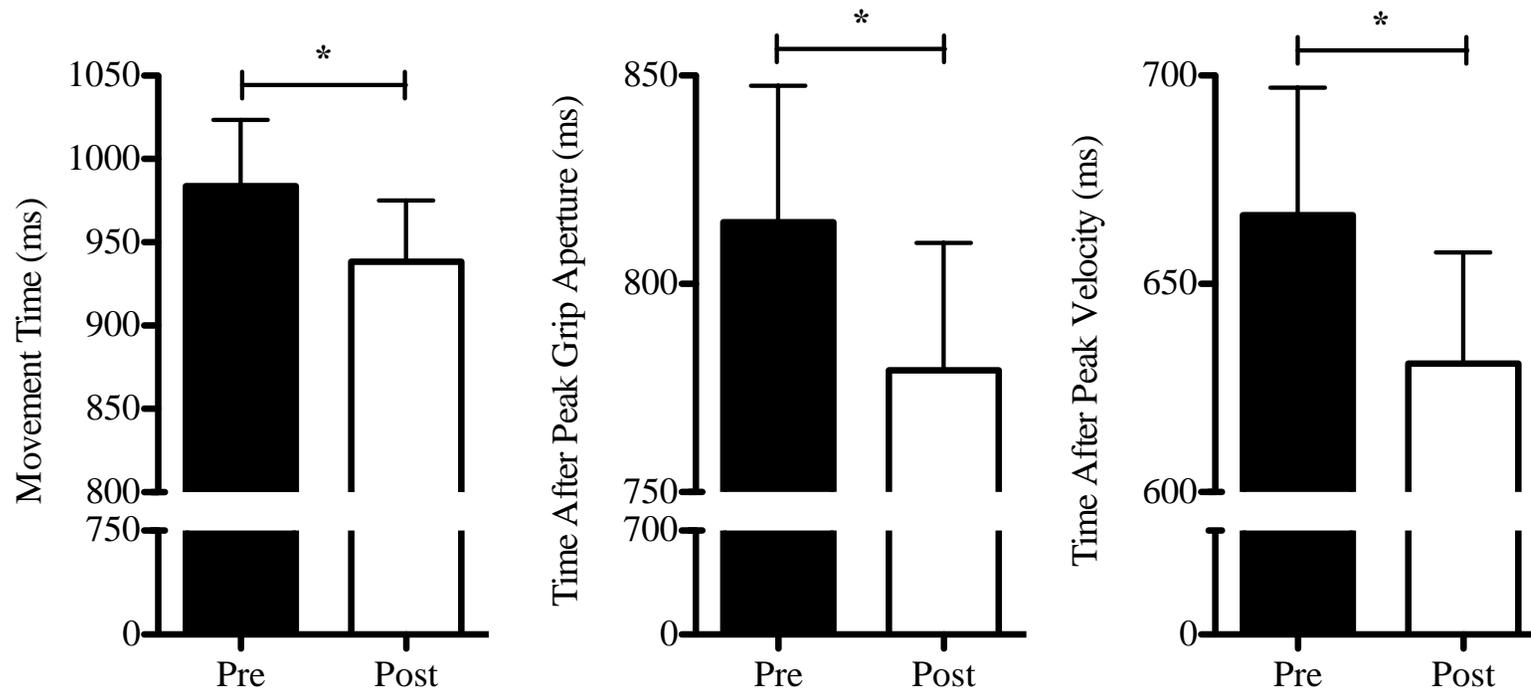


Figure 8: Main effect of Pre-Post Movement Time (MT; left), Time After Peak Grip Aperture (TAPGA; middle), and Time After Peak Velocity (TAPV; right). MT, TAPGA, and TAPV were all significantly faster Post 8-hour visual deprivation ($p < .05$).

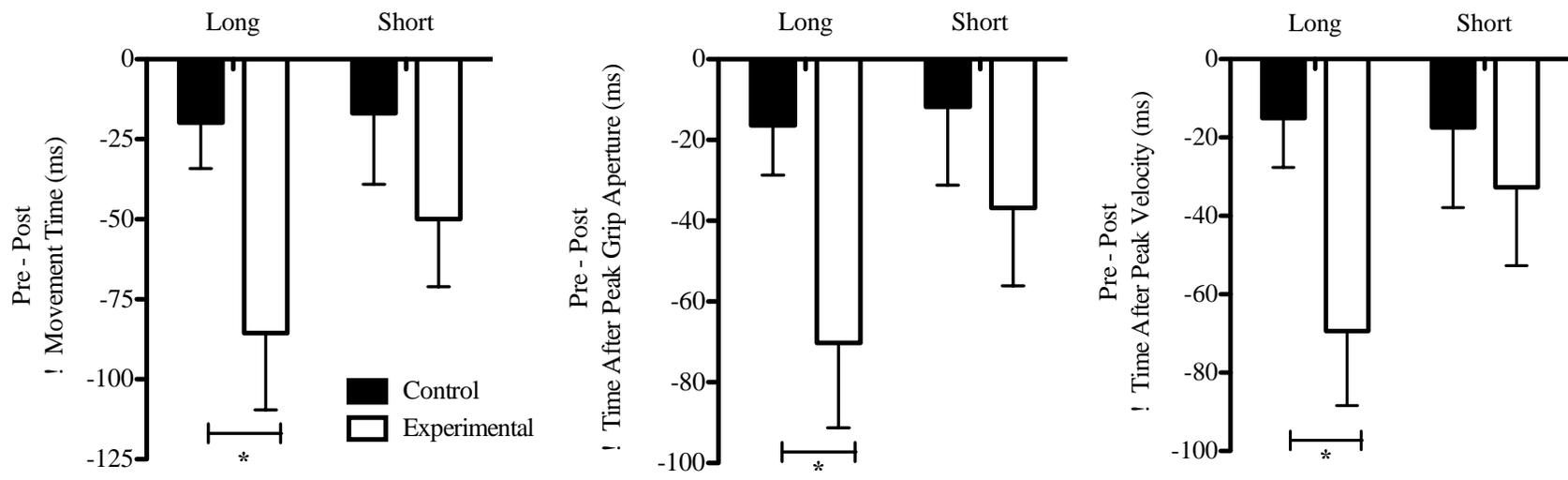


Figure 9: Pre-to-Post change in Movement Time (MT; left), Time After Peak Grip Aperture (TAPGA; middle) and Time After Peak Velocity (TAPV; right) to Long and Short targets for Control and Experimental days. Pre-to-Post differences were significantly larger on Experimental day for MT, TAPGA, and TAPV ($p < .05$).

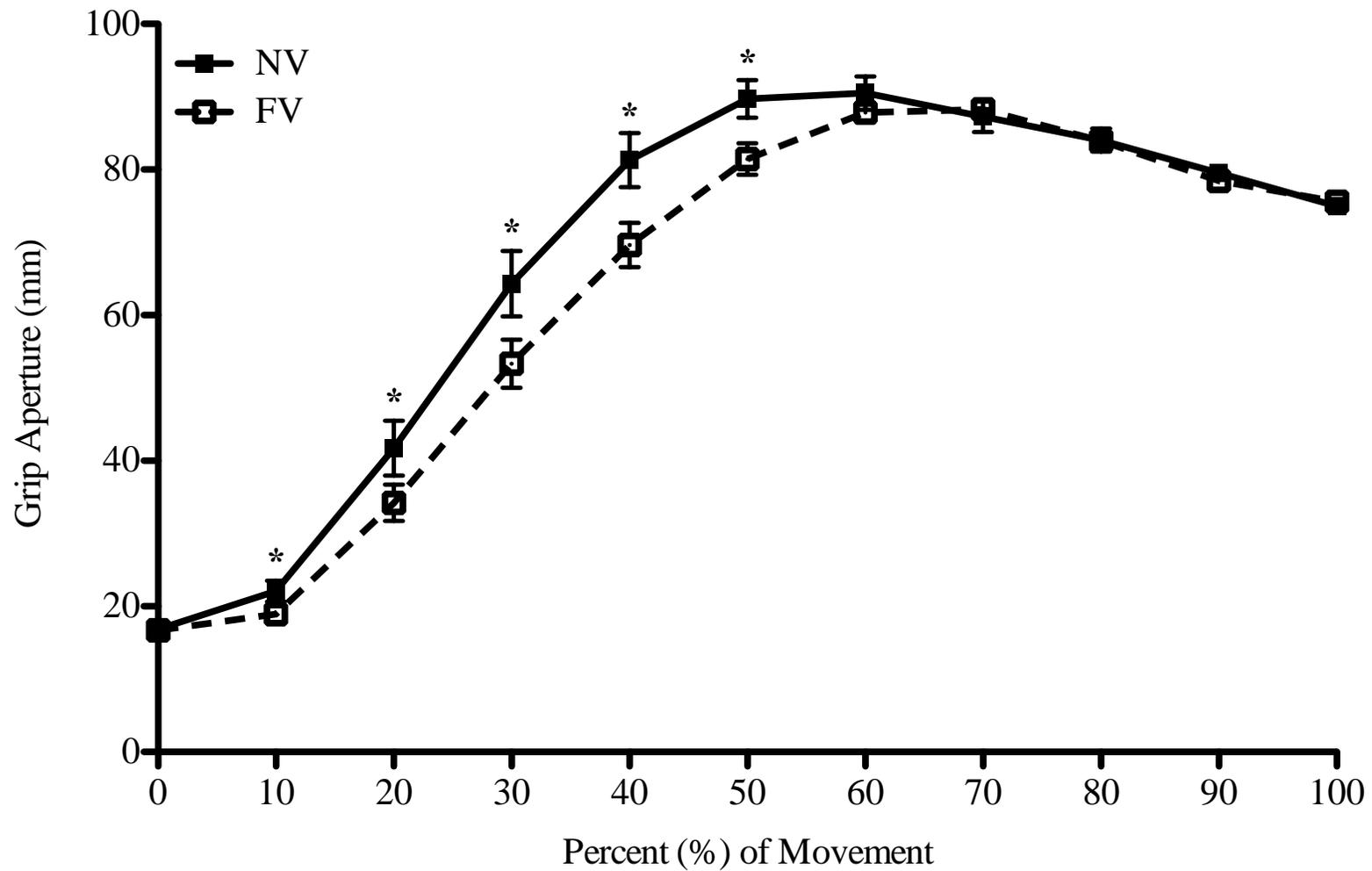


Figure 10: Grip aperture across 10% movement deciles in Full Vision (FV) and No Vision (NV) conditions. Grip aperture within each decile was significantly greater in NV, specifically from 10% to 50% of movement trajectory ($p < .05$).

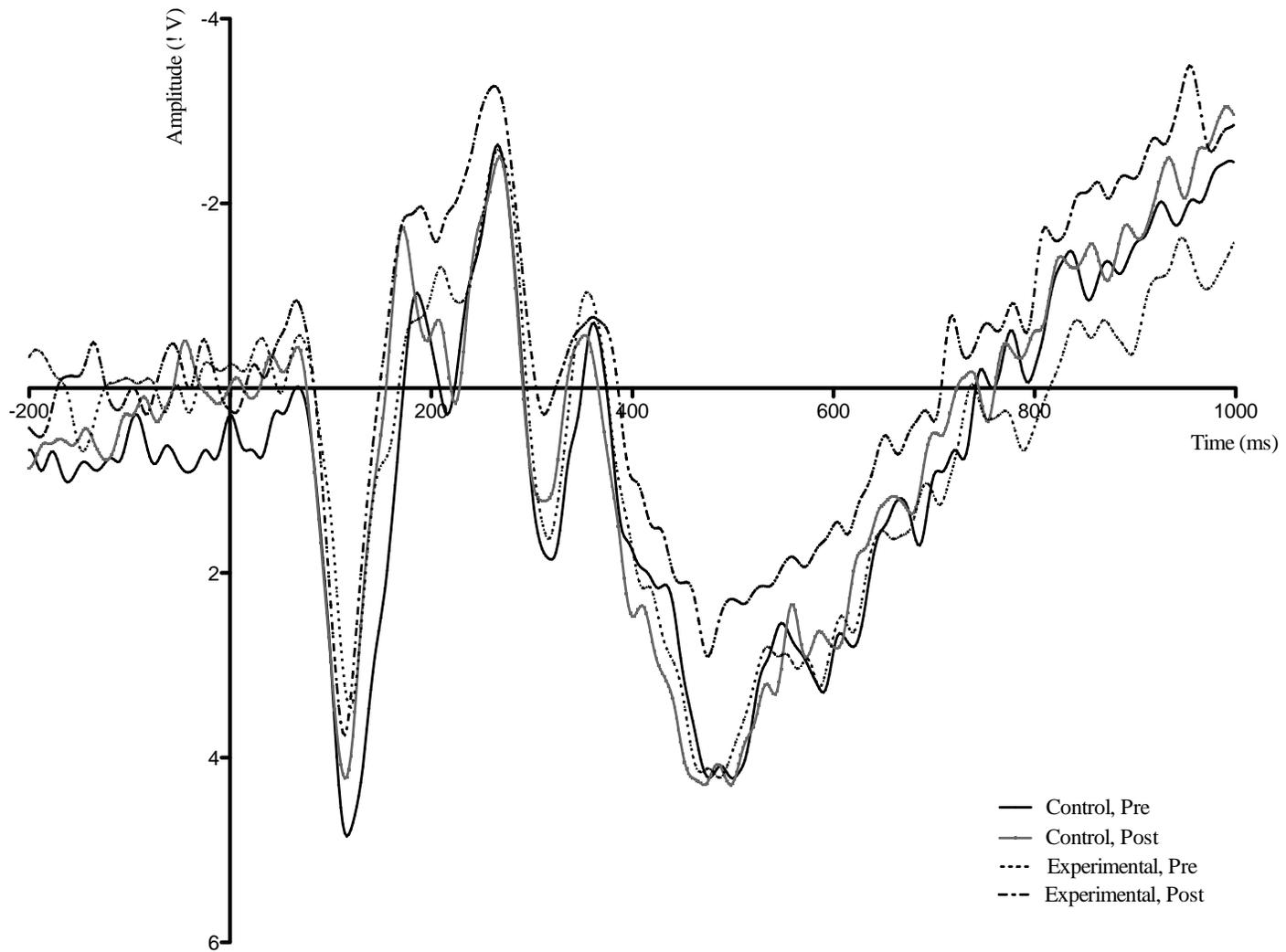


Figure 11: Averaged visual evoked potentials (LED condition), O2 electrode.

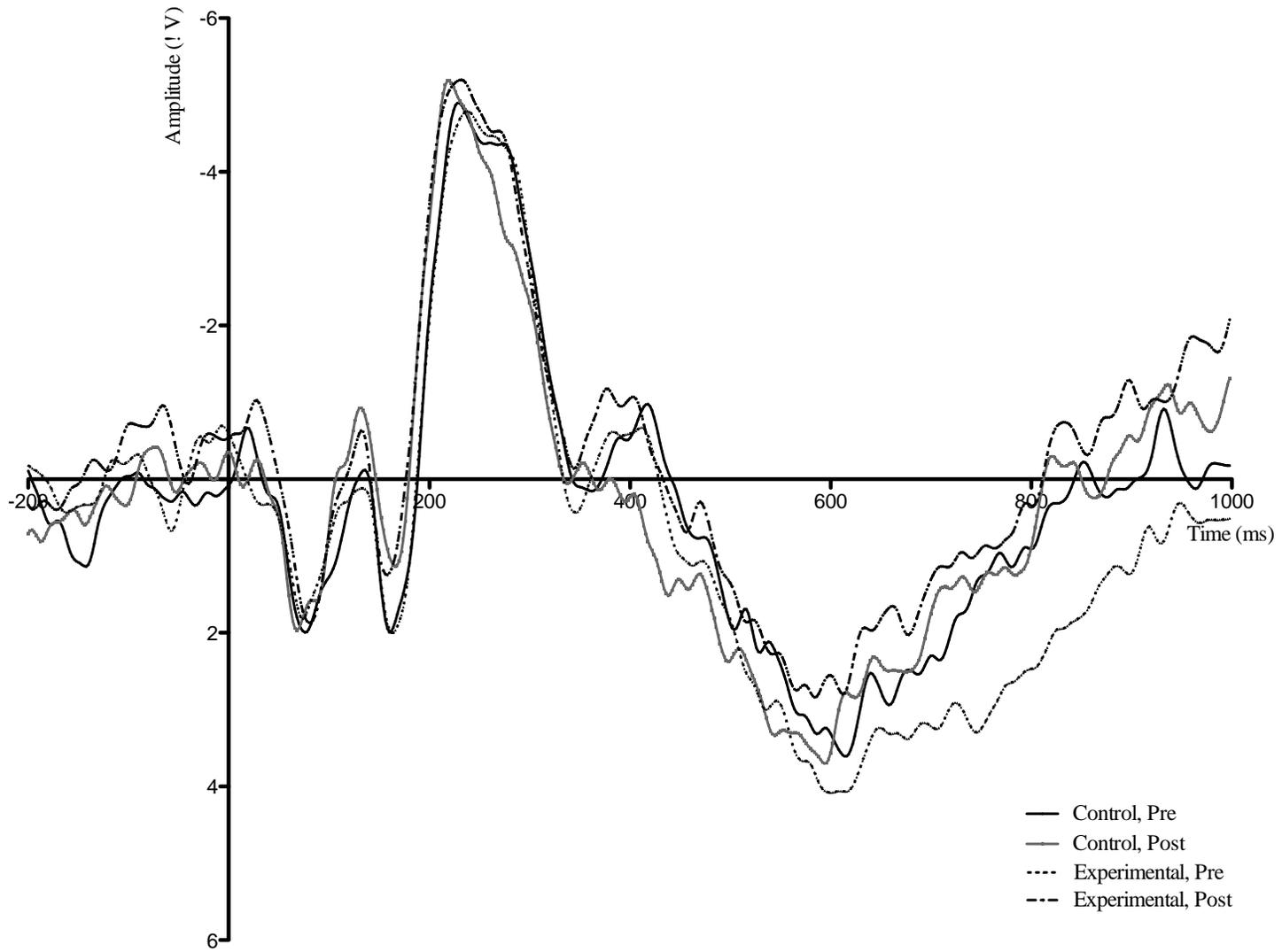


Figure 12: Averaged somatosensory (tactile) evoked potentials (Vibration condition), O2 electrode.

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APPENDIX A: MEANS TABLES

Table 26: Means and standard deviations across subjects for Experiment I behavioural data.

	Pre-test		Post-test	
	No Vision	Vision	No Vision	Vision
<i>Reaction Time (ms)</i>	332.3 (± 66.80)	323.09 (± 67.16)	321.65 (± 68.56)	291.66 (± 56.07)
<i>Movement Time (ms)</i>	900.17 (± 141.53)	749.00 (± 121.18)	872.40 (± 89.38)	752.59 (± 105.77)
<i>Peak Grip Aperture (mm)</i>	109.63 (± 6.29)	99.78 (± 8.19)	103.77 (± 13.12)	97.01 (± 11.56)
<i>Time After Peak Grip Aperture (ms)</i>	417.37 (± 72.84)	291.35 (± 47.72)	401.97 (± 54.68)	319.93 (± 46.18)
<i>Time After Peak Grip Velocity (ms)</i>	544.17 (± 93.41)	422.54 (± 83.84)	518.69 (± 61.95)	427.94 (± 66.62)

Table 27: Means and standard deviations across subjects for Experiment II behavioural data.

	<i>Control</i>			
	Pre-test		Post-test	
	No Vision	Vision	No Vision	Vision
<i>Reaction Time (ms)</i>	256.28 (±57.24)	217.33 (±47.32)	254.64 (±56.07)	215.39 (±44.90)
<i>Movement Time (ms)</i>	1131.79 (±204.55)	824.83 (±175.41)	1122.85 (±158.12)	788.01 (±152.42)
<i>Peak Grip Aperture (mm)</i>	97.84 (±7.53)	90.97 (±3.37)	97.60 (±9.77)	91.65 (±5.17)
<i>Time After Peak Grip Aperture (ms)</i>	947.46 (±163.56)	674.28 (±151.46)	944.16 (±129.28)	642.41 (±130.00)
<i>Time After Peak Velocity (ms)</i>	796.12 (±159.20)	531.54 (±140.04)	786.26 (±120.20)	500.88 (±105.83)

	<i>Experimental</i>			
	Pre-test		Post-test	
	No Vision	Vision	No Vision	Vision
<i>Reaction Time (ms)</i>	283.02 (±37.61)	234.99 (±38.99)	205.96 (±39.69)	215.31 (±34.56)
<i>Movement Time (ms)</i>	1183.57 (±156.89)	794.27 (±132.41)	1088.50 (±157.89)	753.88 (±133.09)
<i>Peak Grip Aperture (mm)</i>	99.40 (±8.91)	92.10 (±5.46)	80.21 (±7.75)	93.65 (±6.82)
<i>Time After Peak Grip Aperture (ms)</i>	993.54 (±136.57)	643.94 (±110.77)	734.02 (±110.89)	612.90 (±113.10)
<i>Time After Peak Velocity (ms)</i>	839.44 (±138.54)	498.73 (±97.15)	610.41 (±105.15)	473.16 (±98.37)

Table 28: Means and standard deviations across subjects for Experiment II EEG data.

	Control		Experimental	
	Pre-Test	Post-Test	Pre-Test	Post-Test
<i>VIB P1 (O1 electrode)</i>	2.22 (± 1.23)	1.68 (± 0.77)	1.20 (± 0.40)	1.90 (± 0.63)
<i>LED P1 (O2 electrode)</i>	2.04 (± 1.07)	2.66 (± 1.30)	1.85 (± 1.16)	1.50 (± 0.73)
<i>VIB P2 (O1 electrode)</i>	3.10 (± 1.72)	2.41 (± 1.35)	1.76 (± 1.24)	2.62 (± 0.95)
<i>LED P2 (O2 electrode)</i>	3.90 (± 2.30)	2.90 (± 2.04)	2.55 (± 1.50)	3.24 (± 1.85)
<i>LED P3 (O2 electrode)</i>	2.03 (± 1.17)	3.26 (± 1.75)	3.03 (± 1.70)	2.20 (± 1.24)