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RESEARCH NOTES

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Initiation of rapid reach-and-grasp balance reactions: is a pre-formed visuospatial map used in controlling the initial arm trajectory?

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Abstract In order to recover balance by grasping an object for support, the CNS must rapidly move the hand toward a specific target (handhold) in the environment. The early latency (80–140 ms) of these grasping reactions would seem to preclude a role for online visual feedback in the control of the initial limb movement; however, some studies have shown that vision can influence initiation of lower-limb postural reactions at similar latency. This study explored the role of vision in controlling the initial trajectory of grasping reactions triggered by sudden unpredictable medio-lateral platform translation. Healthy young adults were instructed to recover balance by grasping a marked section of a handrail, located to their right. To reinforce a dependence on arm reactions, movement of the feet was prevented by barriers. Liquid-crystal goggles were used to occlude vision during response initiation (200 ms interval starting at perturbation onset, PO). Results showed that the initial grasping trajectory (first 100 ms) and associated muscle activation were heavily modulated to take into account the direction and speed of the perturbation-induced body motion in relation to the handrail. This modulation was unaffected by occlusion of vision at PO, indicating that information about the rail location obtained *prior* to PO was incorporated into the control. These findings are consistent with the view that the CNS tunes the initial arm trajectory by combining an egocentric spatial map, formed prior to PO, with online feedback about the body motion from non-visual inputs (somatosensory and/or vestibular). This prevents potential delays associated with visual processing and ensures very rapid onset of arm movement that is directed appropriately even though the position of the body is perturbed unpredictably with respect to the target.

Keywords Arm movement · Grasp · Postural balance · Reach · Triggered reaction · Vision

Introduction

The neural control of volitional reaching and grasping movements has received an enormous amount of attention in the motor control literature (Desmurget et al. 1998). In contrast, very little is known about the mechanisms underlying the control of rapid arm reactions that are triggered by perturbation of whole-body stability. Rapid movements to reach and grasp an external object for support can play a critical role in stabilizing the body (McIlroy and Maki 1994, 1995; Maki and McIlroy 1997; Maki et al. 1998) and are a common response to loss of balance in many situations (Maki and McIlroy 1997; Bateni et al. 2004).

The visual representation of the target is fundamental to the control of volitional pointing, reaching and grasping movements. Prior to voluntary movement execution, visuomotor computation of the object distance, orientation and size must be carried out in an egocentric frame of reference (Jeannerod 1988). However, for compensatory grasping reactions that are triggered by sudden unexpected

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or unpredictable loss of balance, the urgent need to react rapidly places severe temporal constraints on visuomotor processing. These compensatory grasping reactions are typically initiated at a latency of 80–140 ms (McIlroy and Maki 1995), over 100 ms faster than initiation of volitional reaction-time grasping movements (Maki and McIlroy 1997), yet previous findings suggest that even the earliest part of the arm trajectory incorporates information about the surrounding environment (Maki and McIlroy 1997). It is not clear whether the rapid latency of the response initiation permits sufficient time, *after* the onset of the perturbation, for the central nervous system (CNS) to acquire, process and integrate the necessary visual target information into the control of the initial arm movement.

Studies showing slow responses (>2 s) to optokinetic stimulation (Lestienne et al. 1977) and no effect of vision deprivation on the initiation of rapid (100 ms latency) triggered ankle reactions (Diener et al. 1986; Maki and Ostrovski 1993) suggest that the delays associated with visuomotor processing could preclude on-line visual control of the earliest part of triggered grasping reactions, yet there is evidence that visual information can modulate lower-limb postural reactions at latencies as early as 100 ms (Nashner and Berthoz 1978). Alternatively, it is possible that the CNS maintains and automatically updates an egocentric visual-spatial map of potential handholds, obstacles to limb movement and other salient features of the surroundings. In the event of a sudden unexpected perturbation, the CNS could then use this pre-formed map, in combination with online vestibular and/or somatosensory information about the body motion, to rapidly initiate an arm movement that is directed appropriately.

The objective of this study was to determine the role of vision in controlling the initial transport (reaching) component of compensatory grasping reactions evoked by unpredictable postural perturbation. The perturbations comprised a wide range of horizontal platform movements that were unpredictable in timing, magnitude, direction and waveform, and subjects were instructed to recover balance by grasping a nearby handrail. Vision was either occluded or left unobstructed during response initiation. We proposed that the CNS would control the initial arm trajectory by using a visual-spatial map of the environment formed *prior* to perturbation onset, in combination with online somatosensory and/or vestibular information pertaining to the whole-body movement induced by the perturbation. It was therefore hypothesized that the initial arm-muscle activation and direction of arm movement toward the handrail would be modulated according to the direction and magnitude of perturbation but would not be affected by occlusion of vision at time of perturbation onset.

Materials and methods

Twelve healthy subjects (six male, six female; aged 20–29 years) participated after signing informed consent to comply with ethics approval granted by the institutional ethics review board. All were right handed and had a visual acuity of 20/20. None had any neurological, musculoskeletal, sensorial or motor deficit, nor used a medication that may affect balance or movement control. Subjects wore a safety harness and stood upright, with arms at sides, on a computer-controlled motor-driven platform (Maki et al. 1996). A handrail was mounted on the platform to the right of the subject within reaching range (distance from rail to mid-sagittal plane was 25% of body height, rail height was 53% of body height).

Compensatory grasping reactions were evoked by sudden transient medio-lateral translation of the platform (primary waveform: 300 ms acceleration pulse followed immediately by a 300 ms deceleration pulse). Perturbation magnitude (small [0.3 m/s,1.0 m/s^2] or large [0.9 m/s, 3.0 m/s^2]) and direction (left or right) were varied unpredictably. Subjects were instructed to direct their vision at a marked 10 cm section of the handrail (~20–30 cm anterior to the shoulder, adjusted for each subject to allow comfortable grasping of the rail) prior to the start of each trial, and to recover balance by grasping this target as quickly as possible after the platform motion started. To enforce a dependence on grasping reactions, subjects were instructed not to step and barriers (0.5 m high) were placed around the feet. To deter anticipatory reactions, the platform remained motionless in ~15% of trials (“catch” trials). In ~30% of trials, an alternate perturbation waveform was used (200 ms acceleration pulse, 400 ms at constant velocity, 200 ms deceleration pulse). This waveform induced a different time course of body motion (and hence required a different arm trajectory to grasp the rail), but the start of the waveform was identical to the primary waveform. Thus, inclusion of these trials served to prevent the CNS from using the initial features of the perturbation to identify the waveform and thereby trigger a “memorized” arm movement needed to respond to that specific perturbation.

To determine the role of online visual feedback during response initiation, vision was occluded (bilaterally) for a 200 ms interval starting at perturbation onset (~50% of the time required to contact the rail), using translucent liquid-crystal goggles (Translucent Technologies Inc., Toronto, ON). In control trials, vision was unoccluded. (A third visual condition, not relevant to the current study, involved occlusion during target acquisition.) Order of testing was balanced both between and within subjects. For each visual condition, each subject performed two blocks of 14 trials (two trials at each of the four primary perturbation conditions, plus two catch trials and four trials involving the alternate perturbation waveform). To avoid the possibility that uncertainty about the availability of vision could lead subjects to disregard visual feedback in control trials, subjects were informed of the visual condition before each trial block started. Immediately prior to starting this study, subjects participated in a separate study (20 large-perturbation trials) in which behavior was unconstrained (allowed to step or grasp).

The frontal-plane trajectory of the right wrist with respect to the right shoulder was determined, at a sampling rate of 60 Hz, using a three-dimensional video-based motion-analysis system (Peak Performance; Englewood, CO). To characterize the predominant direction of the initial active arm movement (occurring prior, in vision-occluded trials, to the reinstatement of vision), we computed the angle (relative to the medio-lateral axis) of a least squares line fitted to the first 100 ms segment of the wrist trajectory (movement onset defined by velocity >5% of peak). Surface electrodes were used to record the electromyographic (EMG) activity of muscles involved in upper-arm elevation and elbow flexion (anterior deltoid and biceps, sampling rate 1000 Hz). The early EMG activation was characterized by computing the average amplitude of the rectified signal for the first 100 ms after onset of activation (i.e. time when pre-perturbation activity level exceeded by >3 SDs for >25 ms). All timing measures were defined relative to onset of platform acceleration (>0.1 m/s^2).

Three-way repeated measures analysis of variance (ANOVA) was performed to test the hypotheses (factors: visual condition, perturbation magnitude, perturbation direction) using the “primary-waveform” trials. Trials where biceps or deltoid were activated prematurely (within <60 ms of perturbation onset) were excluded from all analyses (7 of 384 trials), and trajectory-angle data from one subject were excluded due to technical problems (32 of 384 trials).

Results

The arm reactions occurred at rapid latency regardless of the task condition. The muscle activation occurred at an average latency of 98 ms (SD 16 ms) in biceps and 101 ms (SD 17 ms) in anterior deltoid, mean onset of movement (wrist velocity>5% of peak) was 126 ms (SD 24 ms), peak wrist velocity (mean 2.11 m/s, SD 0.53 m/s) was reached at 244 ms (SD 21 ms) and end of movement (wrist velocity<5% of peak) occurred at 380 ms (SD 44 ms). On average, the determined movement onset time lagged the EMG onset by ~30 ms, consistent with an active response (rather than a passive effect of the perturbation). Visual occlusion did not affect the peak wrist velocity (p=0.34) and had little effect on response latency (mean vision-related differences, within each perturbation task condition, were typically less than 5 ms).

Despite the very rapid speed of initiation, the initial arm movement triggered by the unpredictable platform motion was not a generic or stereotyped response: the direction of the initial wrist movement (first 100 ms), relative to the shoulder, was highly dependent on the perturbation direction ($F_{(1,10)}$=142.2, p<0.0001) and magnitude ($F_{(1,10)}$=60.7, p<0.0001); see Fig. 2a. Moreover, it appeared that information about the rail location was incorporated into the control of this early movement. The hand was moved horizontally toward the rail when the body fell away from the rail (Fig. 1a, b). In contrast, when the body fell toward the rail, the hand was initially moved in the opposite horizontal direction, with a greater degree of vertical motion (Fig. 1c, d). This latter pattern of motion allowed the hand to “clear” the rail, in preparation for grasping, before inadvertent contact between forearm and rail could occur.

Within each perturbation condition, occluding vision during response initiation appeared to have little influence on the direction of the initial wrist trajectory, in comparison to no-occlusion trials (Figs. 1, 2a). ANOVA confirmed that there was no significant main effect (p=0.91) or interaction due to visual condition (p‘s>0.23). Analysis of the amplitude of muscle activation (first 100 ms) in biceps and anterior deltoid yielded results that were consistent with the arm-trajectory findings, i.e. no main effects (p‘s>0.11) or interaction (p‘s>0.23) due to occlusion of vision but a high degree of modulation according to perturbation magnitude and/or direction (deltoid showed a highly significant effect due to both magnitude and direction, p‘s<0.0001; biceps was unaffected by direction, p=0.18, but was strongly influenced by magnitude, p<0.0001). Subjects were successful in grasping the rail in all trials regardless of whether vision was occluded during response initiation.

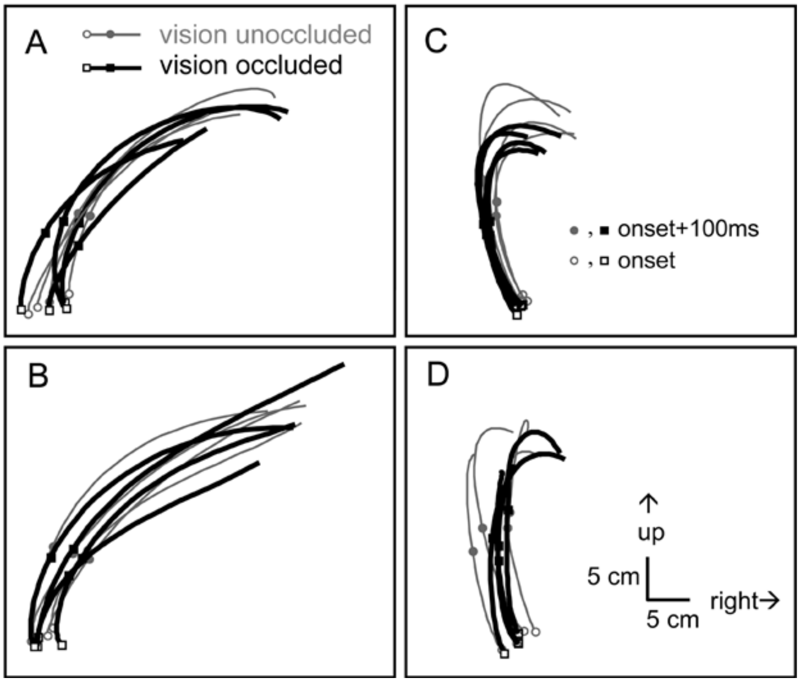

Fig. 1A–D Ensemble of wrist trajectory data from a single subject. The trajectory of the wrist, relative to the shoulder, is shown for individual trials, for falling motion away from the rail [small **(A)** and large **(B)** rightward platform translation] and for falling motion toward the rail [small **(C)** and large **(D)** leftward platform translation]. The *thicker black lines* and *thin gray lines* indicate trials where vision was or was not occluded at perturbation onset, respectively. *Symbols* indicate points on the trajectory at onset of movement and 100 ms later; each trajectory ends at time of rail contact. Note how the initial wrist trajectory (first 100 ms) was modulated to allow the rail to be grasped despite unpredictable perturbation-induced body motion (e.g. the more vertical trajectory when falling toward the rail, and the more horizontal trajectory when falling away from the rail). Although there was some trial-to-trial variability, occlusion of vision did not appear to have any systematic influence on the initial trajectory direction

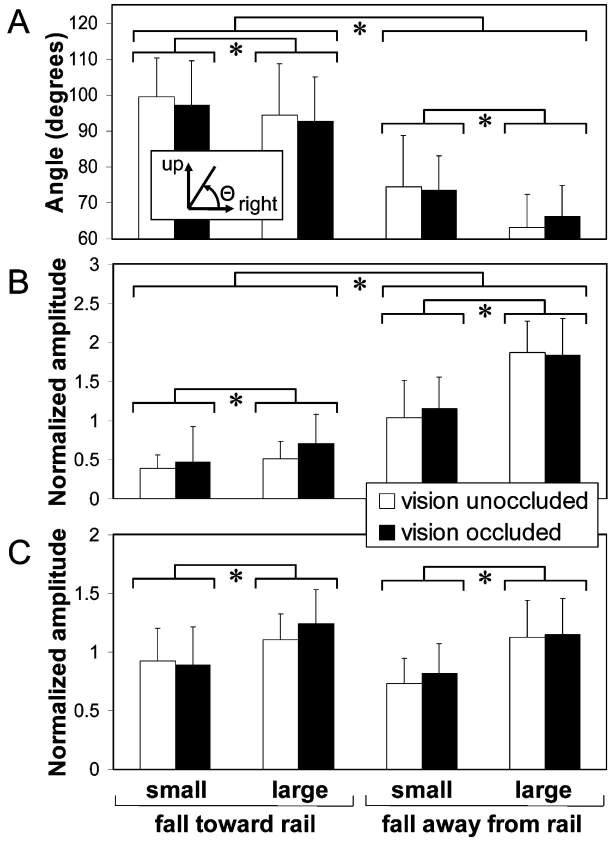

Fig. 2A–C Effect of visual condition and perturbation magnitude and direction on: **A** angle of the initial active wrist trajectory (frontal plane, relative to shoulder); **B** initial anterior deltoid activation; **C** initial biceps activation. Grand means and standard deviations are shown. Note the significant modulation of trajectory angle according to perturbation magnitude and direction, but absence of an effect due to occlusion of vision (**A**; see text for analysis details). Analysis of the amplitude of muscle activation (first 100 ms) yielded similar results. For anterior deltoid, there was a highly significant difference due to perturbation magnitude ($F_{(1,11)}$=128.9, p<0.0001) and direction ($F_{(1,11)}$=148.6, p<0.0001), but no main effect (p=0.11) or interaction (p's>0.32) due to visual condition (**B**). Biceps activation was equally large for both perturbation directions (p=0.18) but was heavily modulated according to perturbation magnitude ($F_{(1,11)}$=98.0, p<0.0001), yet there was no main effect (p=0.18) or interaction (p's>0.23) due to visual condition (**C**). (Note: *asterisk* indicates a significant main effect due to perturbation magnitude or direction, p<0.0001)

Discussion

In contrast to suggestions that the initial arm movement evoked by whole-body postural perturbation may constitute a stereotyped startle or protective response (Hsiao and Robinovitch 1998), the initial arm movement observed in the present study was not a generic response. Even the first 100 ms of muscle activation and associated arm movement were highly modulated according to the position and motion of the body with respect to the handrail, as dictated by the perturbation direction and magnitude. This allowed grasping of the handrail to occur despite large and unpredictable perturbation-induced variation in the position of the body relative to the grasping target. A previous study of perturbation-triggered grasping also showed modulation of initial deltoid activation according to perturbation conditions (McIlroy and Maki 1995).

The pronounced perturbation-dependent modulation of initial active wrist trajectory was not affected by the absence of vision during response initiation. Although it could be argued that a larger sample size might have detected a statistically significant influence due to vision, it is clear from the current data that any possible contribution of online visual feedback to the control of response initiation was very small in comparison to the perturbation-dependent modulation. The fact that the capacity to tune the initial arm movement to permit target acquisition was unaffected by the visual occlusion indicates that information about the target location acquired *prior* to perturbation onset was incorporated into the control. By instructing subjects to look directly at the target prior to perturbation onset, we promoted the ability to "remember" the target location. However, it is important to recognize that the unpredictable variation in perturbation magnitude, direction and waveform precluded the possibility of "memorizing" the required limb trajectories. In order to direct the initial arm movement in the appropriate direction, it was necessary for the CNS to combine non-visual sensory information (somatosensory and/or vestibular) about the body motion with the "remembered" visual target information.

The target location is presumably "remembered" within the framework of an egocentric visual map, formulated by combining retinotopic information with extraretinal cues pertaining to the orientation of the eyes with respect to the head (Sparks 1989). Although the present protocol may have promoted formulation of such a map, we propose that the CNS *automatically* maintains an egocentric map of potential handholds during natural behavior. This process may occur in parallel with the allocentric mapping that guides navigational behavior (Colby 1998). An automatic egocentric mapping process is supported by observations that the initial arm trajectory was directed, at rapid latency, toward the nearest handhold when a totally unexpected perturbation was delivered to a naïve subject (Maki and McIlroy 1997). Ongoing work, using time-varying obstacles, is examining factors that affect how frequently the postulated egocentric map is updated, and how long the map can be "remembered" without updating.

Initiation of rapid volitional arm movements is generally thought to involve an initial open-loop (feedforward) "ballistic" phase where visual feedback is not involved, subsequent to the initial visuomotor planning phase (Day and Marsden 1982; Georgopoulos 1988; Meyer et al. 1988; Abrams 1992). The present findings are not inconsistent with the triggering of an open-loop feedforward response; however, it is clear that this feedforward response would have to be scaled, in a complex way, according to the initial non-visual information about the body motion as well as the prior information about the target location.

A key feature of the current study was that the position of the body, relative to the target, was caused to change unpredictably both prior to and during the execution of the arm movement. While several previous studies have investigated the influence of volitional motion of the body on the control of volitional pointing movements to a remembered target (Wang and Stelmach 1998; Flanders et al. 1999; Medendorp et al. 1999; Pigeon et al. 2003), few have addressed the effect of unpredictable perturbation of the body. One such study did demonstrate that the hand trajectory, with respect to a stationary remembered target, remained invariant when volitional trunk motion was unpredictably blocked during volitional pointing movements (Ghafouri et al. 2002). This observation may indicate that similar compensatory mechanisms, involving somatosensory and/or vestibular feedback, are used to adjust volitional, as well as perturbation-triggered, reaching movements in order to offset the effects of unexpected changes in body position. Although studies of rapid lower-limb reactions (~100 ms latency) might suggest a predominant role for somatosensory drive (Runge et al. 1998), a potential contribution of vestibular afferents to the control of upper-limb reactions is supported by effects of galvanic vestibular stimulation on control of trunk-hand coordination during trunk-assisted reaching movements (Mars et al. 2003) and by findings that vestibular stimulation can influence arm-muscle activation at very rapid latency (~40 ms) (Britton et al. 1993). Platform perturbation could induce the required vestibular drive within 20–30 ms of perturbation onset (Runge et al. 1998).

Conclusion

The findings are consistent with the proposition that the CNS tunes the initial arm trajectory by combining a visual-spatial map, formed prior to the perturbation, with online feedback from non-visual inputs pertaining to the perturbation-induced body movement. Such a control strategy is compatible with the need to achieve very rapid response initiation so as to preserve postural stability. In contrast, volitional arm movement can be delayed until target information is acquired. By relying on a preformed visual spatial map, rather than online visual control, potential delays associated with visuomotor processing are avoided. Ongoing work is addressing the dynamics of the mapping process (e.g. how long a map can be "remembered" without updating), as well as the possible role of online visual control during later phases of the movement (e.g. correct for errors in the trajectory and/or facilitate grasping of the target).

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