Role of the posterior parietal cortex in updating reaching movements to a visual target

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The exact role of posterior parietal cortex (PPC) in visually directed reaching is unknown. We propose that, by building an internal representation of instantaneous hand location, PPC computes a dynamic motor error used by motor centers to correct the ongoing trajectory. With unseen right hands, five subjects pointed to visual targets that either remained stationary or moved during saccadic eye movements. Transcranial magnetic stimulation (TMS) was applied over the left PPC during target presentation. Stimulation disrupted path corrections that normally occur in response to target jumps, but had no effect on those directed at stationary targets. Furthermore, left-hand movement corrections were not blocked, ruling out visual or oculomotor effects of stimulation.

A 'motor plan' may be defined as a set of muscle commands determined before initiation of movement[1]. This concept has provided theoretical underpinnings for many studies on the generation of visually directed pointing movements[2,3]. Thus, most authors subordinate the importance of feedback loops, which allow updating of the trajectory throughout its execution. Here we demonstrate the existence of such loops and try to identify their functional substrates.

The minimal delay needed for a visual or proprioceptive signal to influence ongoing movement has been estimated[3] at around 100 ms. This led to the suggestion that sensory feedback loops could not be used to control trajectory during rapid hand movement[4]. Following this logic, visually directed movements must be driven by predefined motor plans, and feedback loops can act only as the arm slows, toward the end of movement[1,3,5]. This dual model was challenged by experiments showing that hand trajectory could be amended early and smoothly when the target location was slightly modified at movement onset[6,7]. To explain this, it was proposed that feedback mechanisms use motor outflow and movement-related changes in motor apparatus state, as well as sensory information. According to this view, an error signal generated by comparing an internal estimate of hand location to visual target position is used to modify an ongoing motor command. Such forward models, supported by computational neuroscience[8,9], predict immediate availability of hand position and velocity information, allowing feedback control for fast movements.

PPC has been implicated in sensorimotor integration[10,11] and visually guided movements[12,13]. Here we propose that this structure is specifically involved in both formation of internal representations of instantaneous hand location (forward model) and computation of dynamic motor errors (instantaneous differences between hand and target locations). This predicts that disruption of PPC function after movement onset should block any trajectory adjustment based on comparison of hand and target position. To test this prediction, we asked five right-handed subjects to 'look and point' with the right hand to visual targets in the peripheral visual field without viewing the moving limb. In some trials, the target was stationary; in others, it changed position (jumped) during the saccade. From a functional point of view, these two types of trials are identical; when a subject is required to point to a peripheral target, eye and arm muscles are activated nearly simultaneously[3]. Thus, the initial motor command to the arm is based on an extra-foveal visual signal that is subsequently shown to be inaccurate[14]. At the end of the saccade (roughly simultaneous with hand movement onset, because inertia is much higher for arm than for eyes[7]), the target location is recomputed on the basis of foveal information. As previously shown[15], the updated visual signal is then used to adjust the ongoing trajectory. Interestingly, slight target movement during the saccade is not perceived as a jump, because visual motion perception is suppressed during saccades[16]. As a consequence, moving the target between saccades may simply add to the initial error. Functional similarity between movements directed to displaced and stationary targets support the hypothesis that, despite larger path corrections in jump trials, stationary trials engage similar corrective processes[3,6–8].

Cortically mediated functions can be inhibited with single TMS pulses[17,18]. In the present experiment, single TMS pulses were applied over PPC just after movement onset to inhibit putative on-line control mechanisms. The characteristics of movements with or without stimulation were compared for both stationary and jump trials. Jump trials verified that TMS inhibited on-line control, and stationary trials estimated movement accuracy in the absence of feedback-related adjustments. Here the term feedback refers exclusively to the internal loop that compares estimated target position with hand location derived from a forward model to generate an error signal[6–8], and not to other

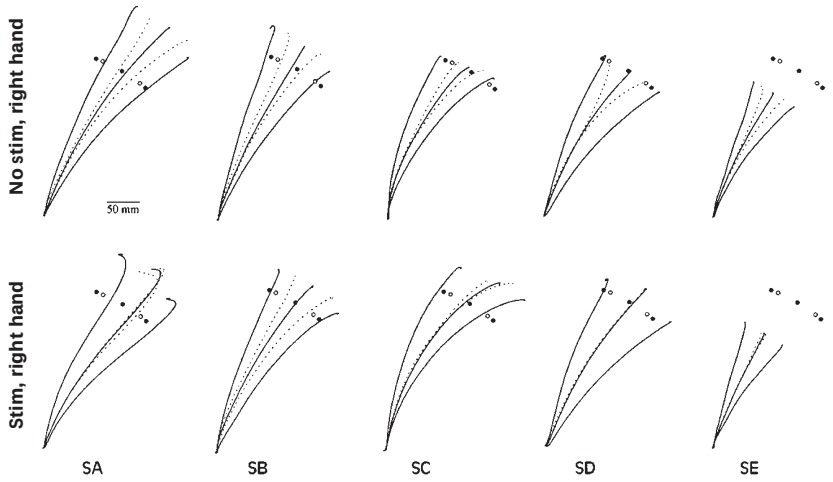

Fig. 1. Mean hand paths produced by all subjects (SA–SE) with the right, dominant hand in the non-stimulated (upper row) and stimulated (lower row) conditions. Mean trajectories were computed after temporal normalization of the individual trials ($n = 10$). The black continuous curves represent the mean paths directed at stationary targets (20, 30 and 40 degrees). The gray dashed curves represent the mean paths directed at jumping targets (30 − 22.5 and 30 − 37.5 degrees). Black circles indicate stationary target locations, whereas white circles represent jumping target locations.

loops involved in trajectory control (for instance, those comparing expected and actual proprioceptive inputs[19]).

Results

Subjects were questioned at the end of the experiment. None reported detection of the target jumps in perturbed trials nor perceived interference with movement accuracy or movement characteristics resulting from stimulation. Mean movement duration was slightly higher in the non-stimulated (490 ms) than in the stimulated condition (461 ms), as would be expected if feedback loops entailing delay are inhibited by TMS[20]. For trials with a 30-degree target, with or without TMS, mean movement duration was not significantly increased by 'target jump' (non-stimulated, 499 ms from 488 ms; stimulated, 466 ms from 458 ms; $p > 0.70$). The weakness of this effect supports the hypothesis that stationary and jump trials did not involve distinct computational mechanisms. To establish that subjects did not use special strategies in the stimulated condition, and that TMS did not affect movement initiation processes, we compared mean position, mean velocity and mean acceleration vectors between stimulated and non-stimulated conditions at the time of peak acceleration (125 ms). This timepoint was chosen based on psychophysical experiments suggesting that the initial movement pulse is independent of feedback loops[3,7]. Analyses revealed no significant influence of TMS on the initial movements ($p > 0.10$).

Smooth modifications of the hand path were observed in all subjects when the target location was changed during the saccade without stimulation (**Fig. 1**). Remarkably, path adjustments were disrupted in four of five subjects when TMS was applied (**Fig. 1**). For subjects SD and SE, hand trajectory was identical in jump trials initially directed to the 30-degree target and no-jump trials ($p > 0.05$). For subjects SA and SC, only minor adjustments were detected at the very end of the movement, following cessation of the TMS effect. TMS did not impair hand trajectory modification in SB.

For stationary targets, TMS significantly shifted movement mean endpoints in all subjects ($p < 0.001$). Remarkably, even in the four subjects in which trajectory corrections in response to target displacement were substantially disrupted, this shift was modest in amplitude (**Fig. 2**). Three of four subjects showed increased mean error magnitude in response to stimulation (SC, SD, SE; 22 mm; $p < 0.0001$). Surprisingly, the remaining subject showed a significant decrease (SA; 26 mm; $p < 0.0001$). This observation may be explained by individual bias in the dynamic estimation of the hand location resulting from large errors in the evaluation of the initial state of the motor apparatus[21]. In such subjects, feedback loops introduce systematic errors that vanish if on-line trajectory corrections are prevented. Theoretically, systematic biases in the dynamic estimation of the hand location should not affect the variable errors. If TMS disrupts feedback loops, these errors should increase. Our observations agree with this prediction (**Fig. 2**). For the four subjects showing disruption of feedback control, movement endpoint variability was 35% larger on average with stimulation.

Because definition of target location in a body-centered frame of reference is mediated by parietal cortex[2,11] and because eye movements are affected by stimulation in posterior parietal cortex[22,23], one cannot exclude *a priori* visual or oculomotor origins for deficits observed with TMS. To address this possibility, we examined two control conditions in which subjects were instructed to point with the non-dominant left hand. If TMS influences representation of target location, it should affect accuracy regardless of the hand used. Alternatively, if TMS prevents hand trajectory corrections, and if parietal control of reaching is lateralized, as suggested by several studies[24–26], no stimulation effect should be observed for left-hand pointing. Our experimental observations were consistent with the latter hypothesis (**Fig. 3**). Indeed, on-line

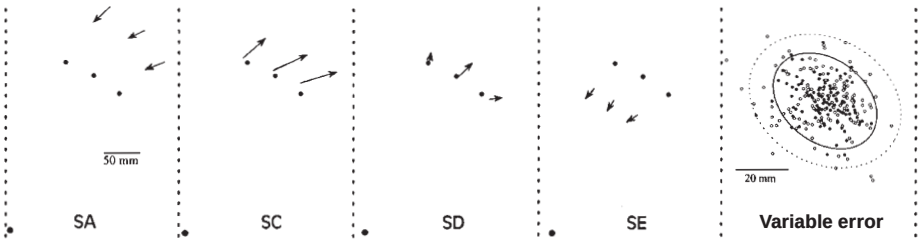

Fig. 2. Influence of the TMS pulse on movement accuracy for the four subjects who showed a total or near-total disruption of trajectory corrections in the stimulated condition. The first four columns display the systematic shift of the movement mean endpoint. (Arrows are oriented from the non-stimulated mean endpoint to the stimulated mean endpoint; large black circle represents the hand starting point; small circles represent target locations.) The last column displays the mean endpoint variability ellipses normalized for direction and position (stimulated, white circles, dotted ellipse; non-stimulated, black circles, continuous ellipse). Note that the scale for the last column is larger than for the first four columns.

movement control was not inhibited by TMS when pointing with the non-dominant hand to a jumping target. In the trials aimed at stationary target addition, only SC showed a significant alteration of the final accuracy ($p < 0.001$); mean movement endpoints were unchanged in the other subjects ($p > 0.10$). Independence of these effects from oculomotor and visual factors is not surprising. Indeed, a TMS study[22] suggests that the saccade-related area in PPC is relatively focal and more medial than the stimulation site in our experiments. Also, in agreement with previous reports[7], stimulation generally occurred at the end of the saccade, which roughly corresponded to hand-movement onset. Late stimulation of PPC by TMS does not affect saccade accuracy[23], suggesting that this cortical area may be involved more in saccade triggering than in saccade control[27].

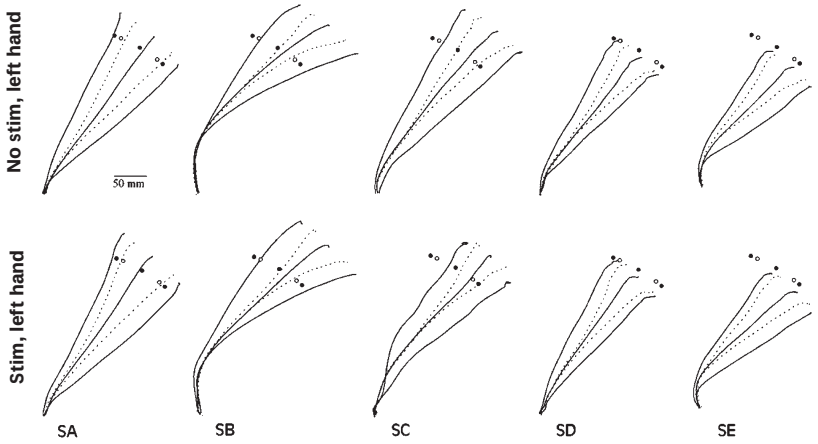

Fig. 3. Mean hand paths produced by all the subjects (SA–SE) using the left, non-dominant hand without (non-stimulated, upper row) or with (stimulated, lower row) TMS. Key, same as in Fig. 1.

To confirm regional specificity of TMS in abolishing corrections, we additionally tested two of the four subjects in whom movement-feedback loops were disrupted (SA, SC) during stimulation of a non-motor temporal area (Brodmann's areas 20, 21) at the same intensity as used for PPC. Under this condition, ability to update the hand trajectory in response to target displacement was not disrupted. Stimulation of the hand area of primary motor cortex (M1) in the same two subjects induced a transient twitch at the wrist, but it did not disrupt ability to modify the hand path when the target moved. As shown by off-line EMG recording, magnetic stimulation applied over M1 was too low to induce discernible activation of the shoulder and elbow primary muscles.

As expected, three-dimensional magnetic resonance imaging at the end of the experiment showed that the effective stimulation sites overlay PPC (**Fig. 4**). For all subjects, the stimulated area included the intraparietal sulcus and the adjacent cortex in the superior and inferior parietal lobule. Although the stimulated area varied slightly among subjects, we were unable to identify a clear correlation between stimulation site and behavioral effects. However, note that the stimulation site extended more medially in the subject with a TMS-related deficit for the non-dominant hand (SC) than in other subjects. From these results, it is not possible to identify specific PPC subareas involved in on-line trajectory updating. The reasons for the lack of feedback inhibition under stimulation in one of the subjects (SB) are unclear; this may have resulted from anatomical variability among subjects, variation in area of stimulation or disparity in neural responsiveness to TMS.

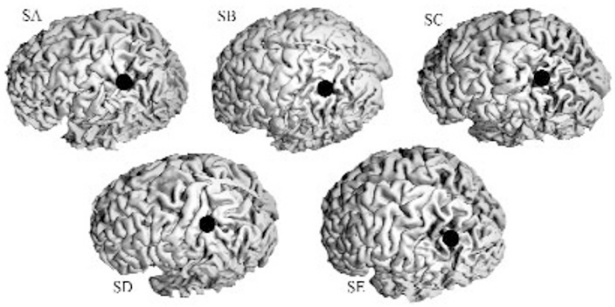

Fig. 4. TMS location sites determined by three-dimensional MRI for all subjects. Dark circles indicate the position of high-intensity signal markers placed on the subjects' skull at the stimulation site. For the sake of clarity, each individual brain is oriented along the stimulation axis (along the coronal axis, orthogonal to the line tangent to the skull at the stimulation site).

Discussion

Here we show that the right-hand path corrections that normally occur in response to the updating of target location at the end of a saccade are disrupted by perturbation of left PPC activity at movement onset. However, the accuracy of movements directed at stationary targets is unaffected by this same disruption of the internal feedback loops that adjust the current motor command by comparing the relative positions of the hand and target. Because no feedback disruption is observed for the hand ipsilateral to the stimulation site, these effects cannot be mediated by purely visual or oculomotor deficits.

Single-neuron recording[13], brain lesion[3,28,29] and imaging[12,24] studies indicate that PPC is critically involved in the execution of visually directed movements. Although PPC involvement in planning processes has been emphasized, most authors acknowledge that this area may also be involved in on-line movement correction[12,13,29]. Our data support this hypothesis by demonstrating that trajectory corrections are disrupted by preventing PPC from functioning properly after movement onset. This result corresponds to neurophysiological studies showing that neurons in area 7A of non-human primates change reach responsiveness as the unseen hand approaches a visual target[13]. It also supports the notion that the PPC functions as a 'neural comparator' to compute the current motor error and allow updating of the muscle activation pattern. PPC has access to target position in a body-centered frame of reference[2,11]. In addition, it can evaluate the current location of the hand by integrating proprioceptive signals from the somatosensory areas and efferent copy signals from the motor regions[10,30]. Finally, it can influence the current motor command through direct and indirect projections to the main motor structures[31,32]. The finding that no TMS-related deficit was observed in the present study for left-hand pointing suggests that the stimulation did not perturb target localization. Instead, TMS affected computation of the motor error, estimation of current hand position and/or transmission of the corrective signal to the motor centers. Further experiments will be required to distinguish these possibilities. It is worth mentioning that the absence of TMS-related deficit for left-hand pointing is coher-

ent with recent imaging studies[24,26] and with the observation that relatively dorsal lesions of PPC (intraparietal sulcus, superior parietal lobule, superior part of the inferior parietal lobule) impair pointing with the contralateral hand into the contralateral space[25,28,33]. An alternate explanation of our results might be that PPC prevents path corrections toward irrelevant stimuli via inhibition of specific cerebral centers. If this were the case, stimulating PPC would lead the motor system to disregard target location updates at the end of the saccade. Although possible, this hypothesis is at odds with our knowledge of both the connectivity of PPC and its role in motor control.

The present experiment bears directly on debates regarding the logic of the motor system in executing visually directed movements. Although the idea that such movements are driven by predetermined motor plans is prevalent, it has also been suggested that continuous control loops comparing locations of hand and target may be used to generate the motor command in real time[34]. This latter idea is supported by two main arguments: first, progressive definition of the motor output seems to be computationally easier than completely predetermining a motor command[34,35]; second, a control scheme that builds the motor command in real time predicts trajectories that successfully capture the kinematic characteristics of visually directed reaching[8,35]. This impressive ability to computationally model behavioral observations however, is not echoed by the results of the present experiment. Indeed, our data indicate that disruption of the feedback loops mediating comparison between target position and estimated current hand location does not produce errant or dramatically inaccurate trajectories, as would be expected if this type of loop were used to generate the motor command in real time.

The present study is compatible with the following model: when a subject points at a target in the peripheral visual field, a motor command is sent to the arm on the basis of extra-foveal visual information. After the completion of the saccade, the central nervous system refines its estimate of the target location based on combined input from retinal and extra-retinal signals[6,7,15]. Concurrently, dynamic proprioception and efferent copy signals are linked together by the PPC to estimate hand location. This structure then compares these two spatial codes and computes the dynamic motor error that is used by the motor centers to update the ongoing trajectory.

Methods

Apparatus and experimental procedure. The experimental device (similar to one described[36]) consisted of a horizontal table in front of which the subjects were seated comfortably. Table height was adjusted to the level of the lower part of the subject's sternum. An array of six light emitting diodes (LEDs) and a half-reflecting mirror were suspended over the pointing surface. Subjects saw virtual images of the LEDs through the mirror, in the plane of the pointing table. They could not cover the diodes, which prevented an indirect estimation of the movement final error. The LEDs were located on a circle centered on the hand starting point (radius, 25 cm), at –20 (left hemispace), 20, 22.5, 30, 37.5 and 40 degrees. The subject's head was fixed with a chin-rest and positioned along a line between the hand starting point and the zero-degree target. The hand starting point was located 25 cm in front of the subject's head. With the index fingertip at the starting point, the forearm rested on the table in a semi-flexed position. Hand movements were recorded with an ELITE system at a sampling frequency of 100 Hz. Eye movements were recorded binocularly using DC electro-oculography. In a typical trial, the –20 degree LED was turned on for visual fixation. Then after a random delay ranging from 1000 ms to 1500 ms, this LED was turned off, and a target was randomly presented at 20, 30 or 40 degrees. In one third of the trials involving the 30-degree LED, target location was moved to 22.5 or 37.5 degrees as the saccade reached peak velocity. The TMS pulse was initiated by an infrared switch as the subject's index finger was lifted from the starting point. Because of saccadic suppression, changes in target location were not consciously perceived by the subjects[16]. For each target (unperturbed, 20, 30 or 40 degrees; perturbed, 30 ± 22.5 or 30 ± 37.5 degrees) and each experimental condition (with or without TMS; reaching with right or left hand), 10 repetitions were done. The experimental protocol was approved by the Emory University institutional Human Investigation Committee.

Stimulation. A custom TMS stimulator generated cosine pulses of total duration 180 μs. The iron-core coil produced a magnetic field distribution comparable to that of a 5 cm × 10 cm figure-eight coil[37]. Measurements made in a model head[17] indicate that isopotential contours of the induced electric field have an oval shape, with long axis parallel to the central windings of the coil and the maximum electric field directly beneath the center. Models of the induced electric field suggest that, with a TMS pulse adjusted to 120% of motor threshold, the cortical area exposed to an electric field was approximately 3.2 cm × 1.7 cm. To reduce the likelihood of activating the motor cortex, parietal stimulation was done with the stimulator aligned at an oblique angle, placing the central windings parallel to the central sulcus. During the experiment, TMS pulse intensity was adjusted to 120% of the motor threshold. At the end of the experiment, 30-second EMG recordings were done for each subject while the entire limb was held in moderate tonic contraction and PPC was stimulated at a frequency of 0.5 Hz. EMG analyses involving the primary flexor and extensor muscle groups for the shoulder, the elbow and the wrist confirmed the absence of TMS-related muscle activation during PPC stimulation. Three steps were used to locate the parietal stimulation site. First, the hand motor area was identified using TMS. Second, Talairach coordinates from previous PET studies[38] were used to calculate the translation vector required to reach medial intraparietal sulcus of the PPC from the hand motor cortex. Third, the stimulator was moved 4.5 cm caudal and 0.5 cm medial along this vector. To locate temporal stimulation sites, we used the same procedure but moved the stimulator 7 cm lateral from the hand motor area.

Magnetic resonance imaging (MRI). Images of individual subject brain anatomy were determined with a high-resolution MRI scans. Scans were acquired on a 1.5-Tesla Philips Gyroscan NT scanner. A T-1 weighted, three-dimensional, fast-field echo pulse sequence of 160 contiguous 1.3-mm coronal sections was obtained (TR, 33 ms; TE, 12 ms; flip angle, 35 degrees). Stimulation sites were defined by marking the skull of the subjects with a small capsule that produced high-intensity signals on the MRI images.

Statistical analyses. Mean movement variation as a function of the experimental factors was tested using two-way inter-individual ANOVAs (stimulation, 2 levels, stimulated, non-stimulated; target location, 3 levels, 20, 30, 40). Vectorial quantities were compared using two-way MANOVAs. Intra-individual one-way ANOVAs were used to determine whether the movement trajectory was different for the control (stationary, 30 degree) and perturbed trials (30 ± 22.5 degree, 30 ± 37.5 degree). Path curvature, movement time, peak acceleration, peak velocity and time-to-peak for acceleration, velocity and deceleration were considered. End-point coordinates (x, y) were compared using one-way MANOVAs. Intra-individual two-way MANOVAs for endpoint coordinates (stimulation, 2 levels, stimulated, non-stimulated; target location, 3 levels, 20, 30, 40 degrees) were used to evaluate stimulation-induced differences in accuracy of the movements directed at stationary targets. Additionally, two-way ANOVAs were used to analyze magnitude of the vector joining the target to the movement endpoint. Variable error was defined for each subject, each experimental condition and each target eccentricity, as the isodensity ellipsoid within which 95% of the endpoint population fell. To allow synthetic presentation of this parameter (**Fig. 2**), we normalized endpoint populations by standardizing the individual scatters for direction and location[39].

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