Role of the Posterior Parietal Cortex in the Initiation of Saccades and Vergence: Right/Left Functional Asymmetry

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ABSTRACT: **This study explored in humans the role of the posterior parietal cortex (PPC) in saccades, vergence, and combined saccade-vergence movements by means of transcranial magnetic stimulation (TMS). TMS was applied to the right PPC at 80 ms, 90 ms, or 100 ms after target onset in experiment 1, and to the left PPC in experiment 2. Control experiments were also run in which TMS was applied over the primary motor cortex at 90 ms after target onset. Relative to no-TMS trials, TMS over the right PPC prolonged significantly the latency of almost all eye movements (saccades in either direction, convergence, divergence, and components of combined eye movements). Such latency increase was significant mostly when TMS was delivered 90 ms after target onset. In contrast, TMS of the left PPC increased the latency only for saccades to right, convergence, and convergence combined with rightward saccades; latency increase occurred for all time windows of TMS deliver (80, 90, or 100 ms after target onset). TMS over the vertex had no effect on the latency for any type of eye movement. TMS of either the left or the right PPC or of the motor cortex did not alter the accuracy of any type of eye movement. Thus, the effects of TMS on latency are time-, area-, and eye-movement–specific. We suggest that the right PPC is involved primarily in the processing of fixation disengagement, whereas the left PPC participates in the initiation of eye movements via different spatial selective mechanisms that concern exclusively targets to the right and/or to near.**

KEYWORDS: **humans; TMS; PPC; latency; saccade; vergence**

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INTRODUCTION

Saccades and vergence eye movements are used extensively to explore the three-dimensional visual environment. Saccades are the fast stereotyped movements of the eyes in the same direction, allowing fixation to change rapidly. Vergence eye movements allow the angle of visual axes to adjust according to the viewing distance, and bring the target on the fovea of each eye; they are of major importance for single binocular vision and for stereopsis. In natural conditions, saccades and vergence most frequently are combined. Among all movements, vergence is the most fragile and the first to be altered by lesions, disease, and fatigue. Yet its cortical substrate is little explored, particularly in humans, and this contrasts with increasing knowledge for saccades.

For saccade control, a large cortical network is known to be involved, including the posterior parietal cortex (PPC), the frontal eye field (FEF), and the prefrontal cortex (PFC).[1,2] Electrical stimulation of the monkey PPC triggers saccades.[3–5] Lesions of this area result in increasing latency of reflexive visually guided saccades in monkey as well as in humans.[6,7] Functional magnetic resonance imaging (fMRI) shows that this area is highly activated during preparation of visually guided saccades.[8] There is some evidence in monkeys, and more recently in humans, that the PPC is involved in the control of vergence eye movements[9,10]

The goal of the studies presented here is to investigate in healthy humans the role of the right versus left posterior parietal cortex in the initiation of the saccades, vergence, and combined eye movements. Single pulse TMS is used to create interference with the cortical processing of PPC at specific time points during the preparation period of the eye movements. The results show area-, time-, and movement-specific effects of TMS on latency. We argue for a differential role of the left and right PPC in the initiation of saccades and vergence, alone or combined. Detailed presentation of the two experiments can be found elsewhere.[11,12]

METHODS

Subjects

Five healthy adult subjects, three females and two males, participated in each experiment. Four of the subjects participated in both experiments. Subject ages ranged from 29 to 46 years (mean: 37.0 ± 6.4). All subjects had normal or corrected-to-normal vision. Binocular vision was assessed with the TNO test of stereoacuity; all individual scores were normal, 60″ of arc or better. Each subject gave informed consent to participate in the study. This investigation was approved by the local ethics committee and consistent with the Declaration of Helsinki.

TMS Localization

A single-pulse TMS was applied by a MagStim 200 magnetic stimulator with a figure-of-eight coil (each wing: 70 mm diameter). The right PPC was stimulated in experiment 1, and the left PPC in experiment 2. The coil was placed 3 cm posterior and 3 cm lateral to the vertex. These criteria were also used in prior studies.[13,14] The

posterior parietal cortex (PPC) is located in the caudal part of the parietal lobe, including the superior and inferior parietal lobules. Such placement of the coil involved stimulation of the region of the posterior part of the intraparietal sulcus, which appears to play an important role in the control of eye movements. The coil was placed down to the scalp, with its handle oriented backward and 45° leftward relative to the midline.[15] The PPC was stimulated at 60 to 80% of total stimulator output, depending on the subjects; blinks were monitored online and the stimulator output was adjusted to avoid frequent blinks. Similar capacity stimulation has been used by others.[16] The rising time of the pulse was 5 μs, the decay lasting 160 μs, and a click occurred simultaneously with the stimulation discharge. For the control experiment, TMS of the primary motor cortex was performed by placing the coil on the vertex, with the handle oriented backward. For no-TMS reference experiments, the stimulator was switched on, but the coil was placed 30 cm over the head of the subject and oriented toward the ceiling.

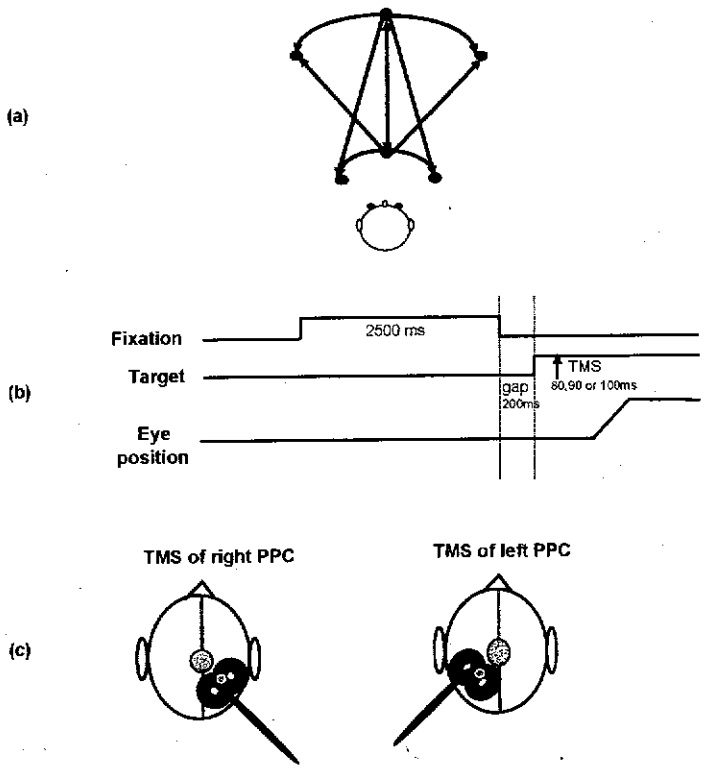

FIGURE 1. (**a**) LED targets and eye movements required saccades at far or at near, pure vergence along the median plane, and combined convergent or divergent movements. (**b**) Temporal arrangement of fixation and target LEDs in a given trial; the target appears after a gap period of 200 ms. (**c**) The TMS stimulator was placed at the right and left PPC in experiments 1 and 2, respectively. TMS was delivered at 80, 90, or 100 ms after target onset.

Visual Display

The visual display consisted of LEDs placed at two isovergence circles: one at 20 cm from the subject, and the other at 150 cm. On each circle three LEDs were used: one at the center and the others at ±20°. The required mean vergence angle for fixating any of the three LEDs was 17° at the close circle. On the far circle, it was 2.3° (see FIG. 1a).

Oculomotor Procedure

In a dark room, the subject, seated in a chair adapted with a medical collar, viewed binocularly the three-dimensional visual display of the LEDs. All LEDs were highly visible, as at each trial only one LED was lit at a time.

Main Oculomotor Task

In order to elicit short-latency reflexive eye movements, we used the gap paradigm described below. Each trial started by lighting a fixation LED at the center of one of the circles (far or near). After a 2.5-s fixation period, the central LED was turned off; following a gap of 200 ms, a target-LED was turned on for 2 s (see FIG. 1b). When the target LED was on the center of the other circle it called for a pure vergence eye movement, along the median plane. When it was at the same circle it called for a pure saccade, and when it was lateral and on the other circle the required eye movement was a combined saccade with vergence eye movement (see FIG. 1a). All target LEDs for saccades were at 20°. All targets along the median plane required a change in ocular vergence of 15°; similarly, combined movements required a saccade of 20° and a vergence of 15°. In each block, the three types of eye movements were interleaved randomly.

In experiment 1, two subjects performed six blocks of 60 trials with TMS of the right PPC and four blocks of 60 trials without TMS. Three subjects performed two blocks of 60 trials with TMS of the right PPC and two blocks of 60 trials without TMS. Four subjects performed one block of 60 trials with TMS over the vertex.

In experiment 2, all subjects performed five blocks of 60 trials with TMS over the left PPC, two blocks of 60 trials without TMS, and one block of 60 trials with TMS over the vertex. For both PPC experiments, TMS was delivered at 80, 90, or 100 ms after target onset. In no-TMS blocks, the click was also delivered at 80, 90, or 100 ms after target onset. For the control vertex experiment the TMS was delivered at 90 ms. Before and after each block, a calibration task was performed, in which subjects made saccades to ±20° LED targets at far and at near. The order of the blocks (TMS at PPC, no-TMS, or TMS at vertex) was pseudorandom to avoid fatigue effects.

Eye Movement Recording

Horizontal movements from both eyes were recorded simultaneously with the IRIS device (Skalar Medical, Delft, The Netherlands). The head was stabilized by placing the chin on a frontal rest. Eye position signals were low-pass filtered with a cut-off frequency of 200 Hz and digitized with a 12-bit analog-to-digital converter and each channel was sampled at 500 Hz.

FIGURE 2. Examples of eye movements stimulated; *arrows and markers* indicate the start (*i*) and the end (*e*) of each type of eye movement.

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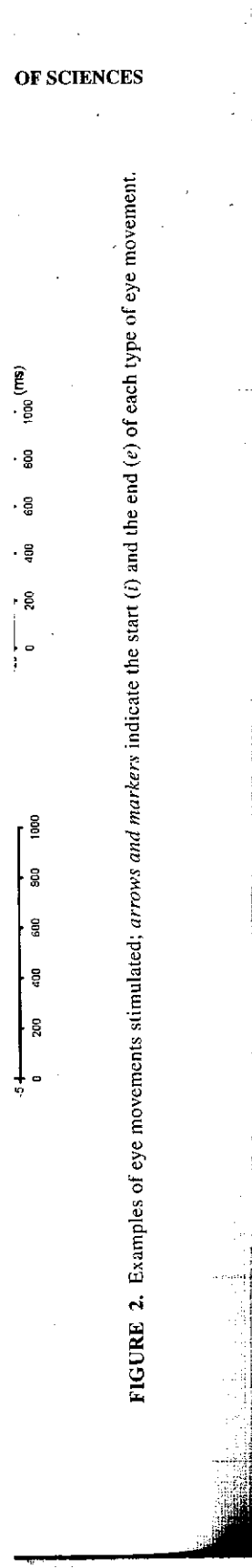

FIGURE 2. Examples of eye movements stimulated; *arrows and markers* indicate the start (*i*) and the end (*e*) of each type of eye movement.

Data Analysis

Calibration factors for each eye were extracted from the saccades recorded in the calibration task; a linear function was used to fit the calibration data. From the two individual calibrated eye position signals we derived the mean of the two eyes (the conjugate or saccade signal), and the difference between the two eyes (the disconjugate or vergence signal). The onset of saccade (pure or combined) was defined as the time when eye velocity exceeded 5% of saccadic peak velocity; the offset was taken as the time when eye velocity dropped below 10 deg/s. The onset and the offset of the vergence (pure or combined) were defined as the time point when the eye velocity exceeded or dropped below 5 deg/s, respectively. These criteria are standard.[17,18] From these markers, we measured the latency of eye movements, for example, the difference between target onset and eye movement initiation (markers *i* in FIG. 2). The eye movement amplitude is the difference between the marker *e* (end of movement) and the marker *i* (start of the movement) (see FIG. 2). Eye movements in the wrong direction, anticipatory movements (latency shorter than 80 ms), and slow movements (latencies longer than 400 ms), or movements contaminated by blinks were rejected (rate of rejection 10%, range 8% to 15%). After checking the homogeneity of variance of individual means, the one-way ANOVA test was used to examine the effect of TMS for each type of eye movement; the subject was the random factor, and the TMS condition was the fixed factor (no-TMS, TMS over PPC). The LSD *post hoc* test was used for paired comparisons between any two conditions.

RESULTS

Experiment 1: Effect of TMS of the Right PPC on the Latency of Eye Movements

Isolated Saccades and Vergence

FIGURE 3a presents the group mean latencies of saccades to right and left of convergence and divergence; data are shown for the reference condition (no-TMS), and for TMS of the right PPC delivered at 80 ms, 90 ms, and 100 ms after target presentation. The one-way ANOVA showed a significant effect of TMS for all types of eye movements: saccades in either direction ($F_{3,12} = 4.9$, $P < .02$ for saccades to right; $F_{3,12} = 5.0$, $P < .02$ for saccades to left), convergence ($F_{3,12} = 3.95$, $P < .04$), and divergence ($F_{3,12} = 5.8$, $P < .01$). Significant differences between TMS and no-TMS are shown by asterisks (LSD *post hoc* tests significant at $P < .05$). When TMS is delivered at 90 ms after target onset, latency prolongation is significant for all movements, whereas when TMS is delivered at 80 or 100 ms, significant latency prolongation is observed for divergence only.

Saccade-Vergence Combined Movements

FIGURE 3b shows mean latencies of saccade, convergence, and divergence components of combined movements for no-TMS and TMS conditions. The ANOVA showed significant TMS effects for saccade ($F_{3,12} = 4.27$, $P < .03$) and divergence components ($F_{3,12} = 3.75$, $P < .04$). Again, effects were significant only when TMS was delivered at 90 ms after target onset (see asterisks).

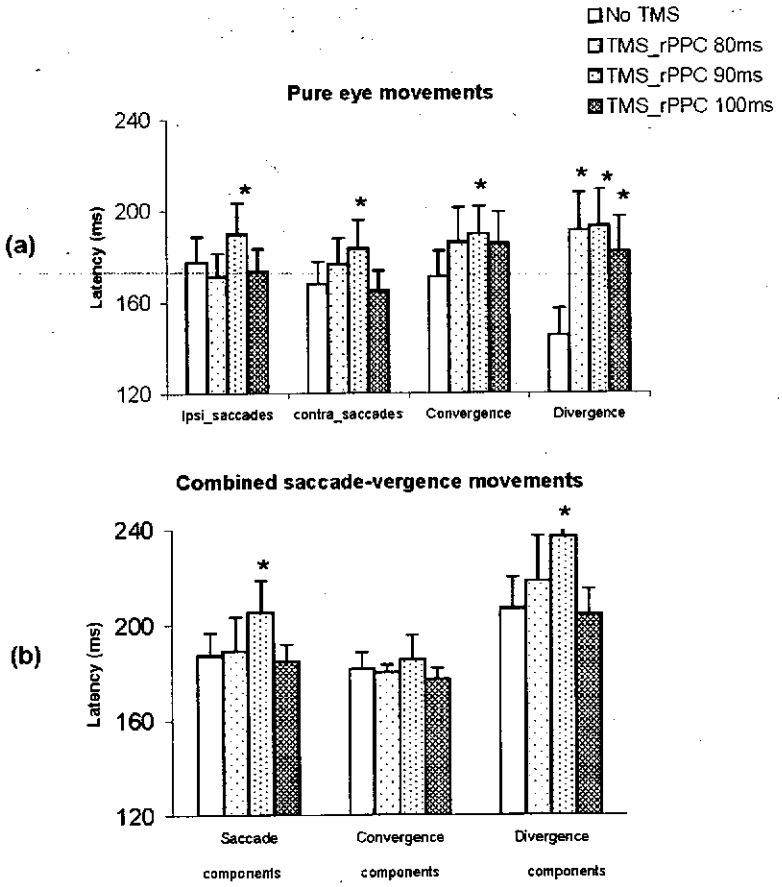

FIGURE 3. (**a**) Group mean latency for no-TMS, and for various TMS conditions for saccades ipsilateral or contralateral to the stimulated right PPC, for convergence and divergence. (**b**) Group mean latency for saccade, convergence, and divergence components of combined movements. *Vertical bars* are standard errors. *Asterisks* indicate significant differences between TMS and no-TMS conditions (LSD *post hoc* test at $P < .05$).

Correlation between Saccade and Vergence Latency

Yarbus[19] was the first to study the initiation of combined eye movements, and pointed out that vergence starts before the saccade. Yang *et al.*[18] reported a high rate of mild asynchrony (10 to 20 ms) of the latency of the two components. Thus, combined eye movements involve the initiation of a complex motor program, and the two components may not be perfectly synchronized. Nevertheless, Tagaki *et al.*[17] reported that the latencies of the two components are highly correlated, and that both components are influenced similarly by the fixation task (latency of both components decreases in a gap paradigm in which the fixation dot disappears before target onset, relative to synchrony or overlap conditions). We examined how well the two compo-

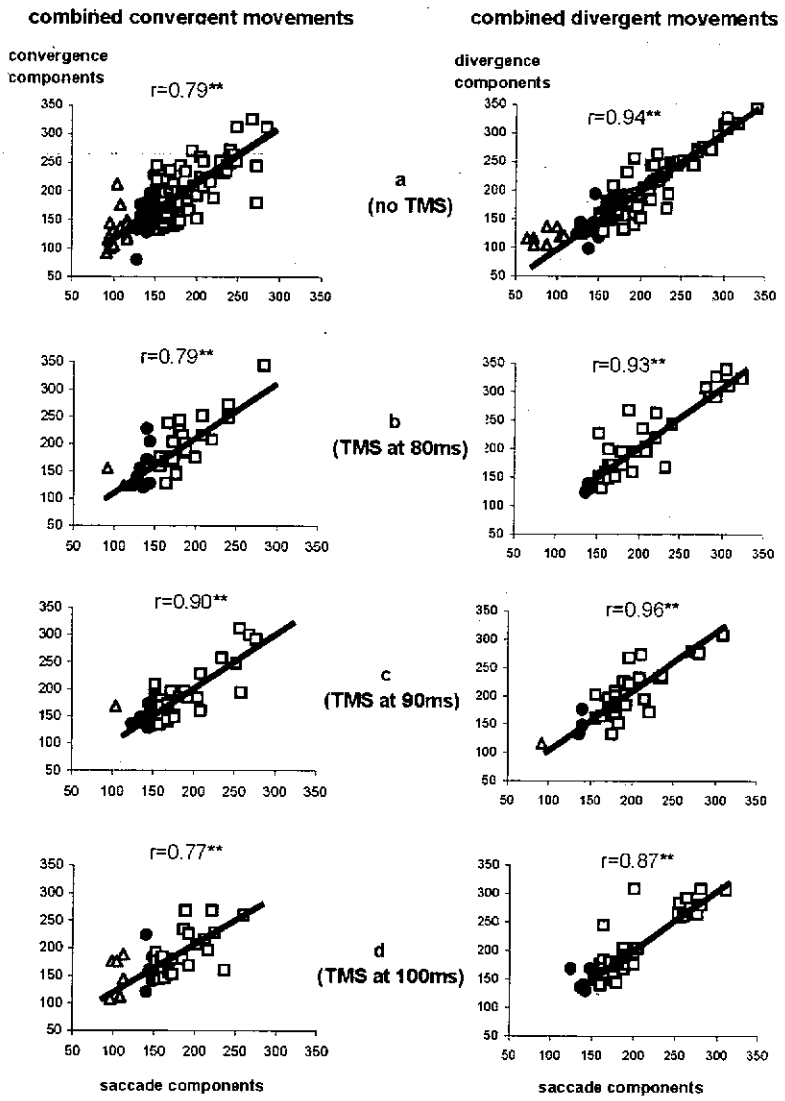

FIGURE 4. Correlation between latency of saccades and vergence components of combined movements in the various TMS and no-TMS conditions. (△) Movements with express type of latency (80 to 120 ms); (●) movements with fast regular type of latency (121 to 150 ms); (□) movements with the slow regular type of latency (151 to 400 ms). Correlation coefficients were high for all conditions.

nents were correlated. FIGURE 4 shows latency correlation between the saccade and vergence components for the no-TMS condition and the various TMS conditions. The coefficient of correlation was high for all conditions (r from 0.77 to 0.96, $P <$ .05) and TMS did not alter this correlation. Takagi *et al.*[17] suggested a common decision mechanism for both components. Consistent with this, Chaturvedi and Van Gisbergen[20] provided evidence for a common target selection and amplitude computation process of stimuli in direction and in depth. Our data are compatible with the studies just cited and models of common triggering of the two components.

Additional Observations

TMS of the motor cortex delivered at 90 ms after target onset caused no significant latency prolongation relative to no-TMS for any type of eye movement ($P >$ .05). For the reference no-TMS condition, the accuracy of eye movements was normal; the mean gain value (i.e., movement amplitude/target excursion amplitude) was 0.93 ± 0.09 for saccades, 0.96 ± 0.19 for convergence, and 0.67 ± 0.09 for divergence. TMS at the right PPC or of the motor cortex had no effect on accuracy ($P > .05$).

Experiment 2: Effects of TMS of the Left PPC on the Latency of Eye Movements

Saccades, Convergence and Divergence

FIGURE 5a shows mean latencies of saccades to the left and to the right, of convergence, and of divergence for the TMS and no-TMS conditions. The one-way ANOVA test applied for each type of movement showed a significant TMS effect only for saccades to the right, that is, contralateral to the stimulated site ($F_{3,12} = 23.8$, $P < .001$), and for convergence ($F_{3,12} = 9.27$, $P < .002$). TMS effects were significant for these types of eye movements regardless of the point at which TMS was delivered (80, 90, or 100 ms after target onset; all comparisons were significant at $P < .001$ and are indicated by asterisks in FIG. 5A).

Combined Eye Movements

Group means of latencies of left or right saccade components for convergence and divergence in TMS and no-TMS conditions are shown in FIGURE 5b and 5c. The ANOVA test showed a significant TMS effect only for saccade components to the right ($F_{3,12} = 3.7$, $P < .05$) and for convergence components combined with rightward saccades ($F_{3,12} = 18.89$, $P < .001$); asterisks in FIGURE 5b indicate significant *post hoc* comparisons (LSD test). For these two types of eye movements, latency prolongation was significant regardless of the time point at which TMS was delivered (80, 90, or 100 ms after target onset).

Additional Observations

As in experiment 1, control studies with TMS of the vertex at 90 ms after target onset produced no changes in latency for any type of eye movements (all $P > .05$). Again, TMS over the left PPC or the vertex had no effect on the accuracy of any of the eye movements.

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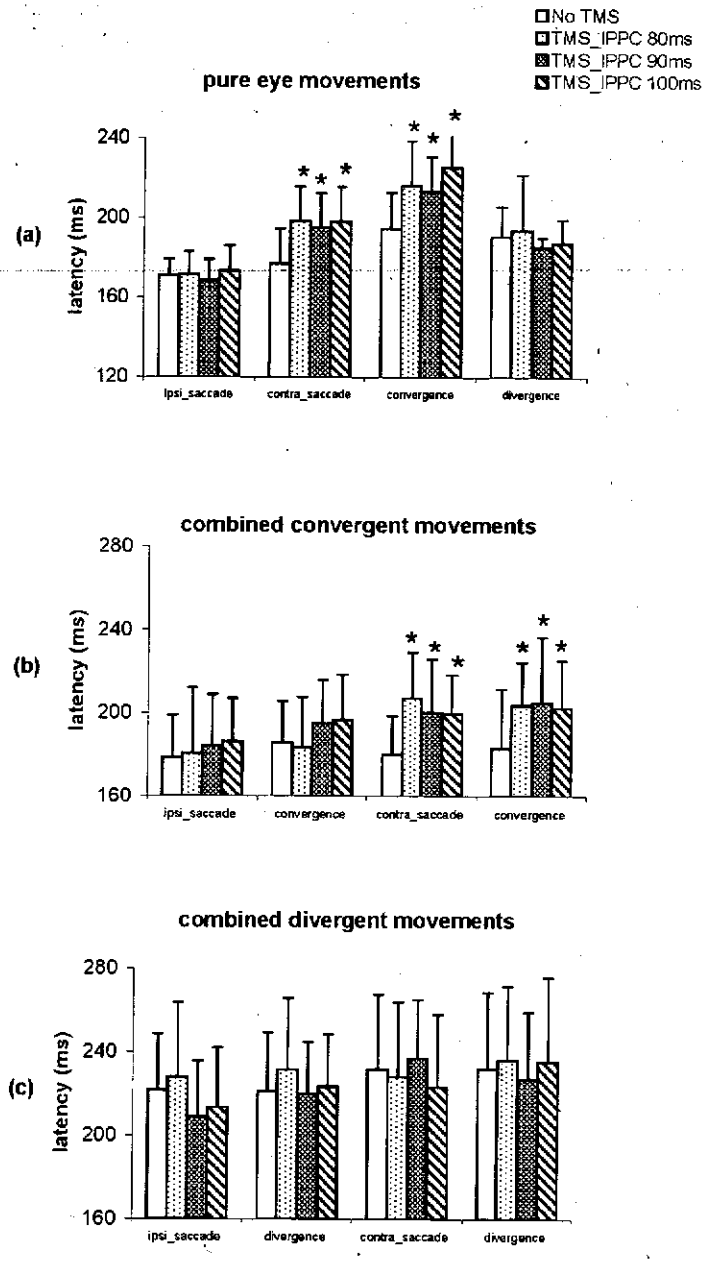

FIGURE 5. (**a**) Group mean latency for no-TMS, and for various TMS conditions for saccades ipsilateral or contralateral to stimulated left PPC, for convergence and divergence. (**b**) Group mean latency for saccade and convergence components of combined convergent movements. (**c**) Group mean latency for saccade and divergence components of combined divergent movements. Other notations as in FIGURE 3.

In summary, TMS of the left PPC caused significant latency prolongation only for saccades to the right, that is, contralateral to the stimulated site, and for convergence, pure or combined with a saccade to the right. Importantly, such prolongation occurred for all time windows tested.

DISCUSSION

The main findings are the following: TMS of the right PPC increases the latency of almost all eye movements when delivered at 90 ms after target onset; for divergence only, there is a latency increase even when TMS is delivered at 80 or 100 ms after target onset. For combined movements, TMS increases the latency of both components similarly and does not alter the tight correlation between the latencies of the two components. In contrast, TMS over the left PPC increases the latency only for certain types of eye movements: saccades to right, convergence, and convergence combined with rightward saccades. Contrary to the right PPC, TMS of the left PPC affects latencies of eye movements for all time windows studied (80, 90, or 100 ms after target onset). TMS over the vertex did not increase the latency for any types of eye movements. Thus, the effects of TMS of the right PPC on latency are time-specific and concern almost all movements, whereas the effects of the left PPC are selective for some movements but wide in time.

We attribute latency prolongation to interference with the triggering signal that the PPC should deliver to the superior colliculus (SC), thereby lengthening the latency of eye movements in three-dimensional space. Next, we will discuss the functional asymmetry between left and right PPC.

Left/Right Functional Asymmetry

Patient studies with right PPC lesions showed marked bilateral increase of saccade latency;[21] our findings for bilateral saccade latency increase after TMS of the right PPC are compatible with this. They are also compatible with prior TMS studies.[13,14] On the other hand, our observation of TMS-induced increase of vergence latency is consistent with physiological studies showing activation of the parietal cortex prior to vergence movements in humans[22] and in monkeys.[10] Based on the observation of bilateral effects on saccade latencies after right PPC lesions, Pierrot-Desseilligny *et al.*[1,21] suggested that the right PPC is involved in the initiation of saccades in either direction via a common mechanism, which is related to fixation disengagement. Our current data support this idea and extend it to vergence eye movements in depth, alone or combined. Disengagement of ocular motor fixation is a prerequisite for any eye movement to occur. Thus, the right PPC would have an omnidirectional and omnidepth function in triggering all eye movements in three-dimensional space by disengaging oculomotor fixation.

In contrast, the left PPC seems to be involved in the initiation of the eye movements via a different mechanism. Recall that TMS over left PPC caused significant latency increases for rightward saccades, for convergence, and for combined convergent and right movements. Such increases occurred for all three time windows of TMS delivery studied (80 ms, 90 ms, and 100 ms after target onset). First, the saccade data are in agreement with the study of patients with lesions of left PPC by

Pierrot-Deseilligny *et al.*,[7] who showed that latency increase was significant only for saccades made contralateral to the lesion. They are also consistent with the study of Leff *et al.*,[23] who found that repetitive TMS over the left PPC slowed the whole array of rightward reading saccades.

Findlay and Walker[24,25] proposed a model in which there exists separation of the pathways controlling the "when" and the "where" information for the triggering of saccades. The where stream is a set of interconnected activity maps, resulting in a "salience map," from which the saccadic target location is selected. In contrast, the when stream is envisaged as a single individual signal whose activity level varies. The competitive interaction of the fixate center (when system) and the move center (where system) may occur in the different brain centers and determines the initiation of the saccades. In the context of such a model, we suggest that the left PPC could be more involved in the where pathway, whereas the right PPC is involved in the when pathway.

Again, in the light of the present study, the model of Findlay and Walker could be extended to include a where stream for convergence control. It is also interesting that TMS of the left PPC prolonged latency similarly when delivered at 80, 90, or 100 ms after target onset, whereas TMS of the right PPC produced time-specific effects. The wide window for the left PPC is compatible with the idea of spatial processing that takes a certain amount of time. As suggested by Findlay and Walker, the where signal would be based on progressive building of a salience map in competition with the fixation center, whereas the when signal would be a single signal.

Left PPC and Space Segregation

The findings of the left PPC suggest segregation for shifting gaze in the near space along the median plane or to the right. Neurophysiological and neuropsychological studies have shown that the near and the far space (within and beyond arm's reach) are coded in different brain areas and by different mechanisms.[26] The frontal lobe of the monkey has been proposed to be involved in far-space representation.[27] Near space, on the other hand, seems to be presented in frontal area 6 and in the rostral part of the inferior parietal lobe, area 7b and area VIP.[28–30] Weiss *et al.*[31,32] measured regional cerebral blood flow with positron emission tomography in normal subjects who performed manual bisection or made line bisection judgment. They found that near-space presentation enhanced left occipital–parietal, parietal, and premotor cortex activity, whereas far-space presentation enhanced activations in the occipital cortex extending into the medial occipitotemporal cortex bilaterally. Taken together, evidence from different studies indicates that the PPC, especially the left PPC, is involved in the control of the eyes and hands within the near space. Convergence is the type of eye movement allowing the transition from the far to the near space and its initiation could be particularly dependent on the left PPC.

Conclusion and Clinical Implications

This study shows the importance of the PPC on the initiation of saccade and vergence eye movements used to explore three-dimensional space, and suggests different mechanisms for the left and right PPC. In young children, latencies of all eye movements are long, particularly those of convergence.[18] In children with vertigo

and balance problems that occur despite normal vestibular function, latencies of eye movements are also abnormally long.[33] Increased latencies in such cases could be due to immaturity or hypofunction of the parietal cortex. The experiments presented here also indicate that rapid initiation of saccades to the right and of convergence depend on good functioning of both the right and the left posterior parietal cortex. Efficient control of saccades, particularly from left to right, is important for reading; convergence movements are also needed in order to realign the visual axes, for example, after the transient divergence occurring during the saccades.[34] In light of the TMS studies presented here, one can better appreciate the complexity and highly cortical nature of ocular motor control during reading.

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