Cortical Dynamics of Anticipatory Mechanisms in Interception: A Neuromagnetic Study

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Abstract

■ Humans demonstrate an amazing ability for intercepting and catching moving targets, most noticeably in fast-speed ball games. However, the few studies exploring the neural bases of interception in humans and the classical studies on visual motion processing and visuomotor interactions have reported rather long latencies of cortical activations that cannot explain the performances observed in most natural interceptive actions. The aim of our experiment was twofold: (1) describe the spatio-temporal unfolding of cortical activations involved in catching a moving target and (2) provide evidence that fast cortical responses can be elicited by a visuomotor task with high temporal constraints and decide if these responses are task or stimulus dependent. Neuromagnetic brain activity was recorded with whole-head coverage while subjects were asked to catch a free-falling ball or simply pay attention to the ball trajectory. A fast, likely stimulus-dependent, propagation of neural activity was observed along the dorsal visual pathway in both tasks. Evaluation of latencies of activations in the main cortical regions involved in the tasks revealed that this entire network of regions was activated within 40 msec. Moreover, comparison of experimental conditions revealed similar patterns of activation except in contralateral sensorimotor regions where common and *Catch*-specific activations were differentiated. ■

INTRODUCTION

Interception is a rapid motor action that is largely dependent on visual inputs. Because moving toward a target is time-consuming, interception requires an estimation of the expected future target position in space and time. In that respect, it has been proposed that humans use anticipatory mechanisms based on on-line visual information (Lee, 1976) that can be combined with a priori implicit knowledge of the rules of physics for the target motion (Zago & Lacquaniti, 2005; McIntyre, Zago, Berthoz, & Lacquaniti, 2001; Lacquaniti, Carrozzo, & Borghese, 1993) for estimating time to contact (TTC). Severe timing constraints are inherent to this task. Accordingly, success in interception requires a fast processing of information (target motion processing, TTC estimate) and a fast transmission of the outcomes to motor centers so that the appropriate anticipatory motor action can be triggered at the right time.

Humans, indeed, demonstrate noticeably good performances in being fast and accurate in high-speed interception behavior. Expert cricketers show reaction times of about 200 msec when adjusting their batting (McLeod, 1987) with temporal accuracy about the instant of impact of less than 2.5 msec (Regan, 1997). In laboratory conditions, correction of initiated or ongoing movements in response to an unconsciously perceived change in target location (Prablanc & Martin, 1992; Paulignan, Jeannerod, MacKenzie, & Marteniuk, 1991; Pelisson, Prablanc, Goodale, & Jeannerod, 1986; Georgopoulos, Kalaska, & Massey, 1981) can occur within only 140 and 110 msec, respectively (Day & Lyon, 2000; Brenner & Smeets, 1997). The timing of brain activations involved in visual and motor processes for interception should therefore be compatible with these behavioral data.

Electroencephalography (EEG) and magnetoencephalography (MEG) studies have already reported on fast processing of visual information in a variety of cognitive tasks, but preferentially involving the ventral visual pathway [e.g., object categorization (Thorpe, Fize, & Marlot, 1996), face recognition (Seeck et al., 1997), response to emotional words (Ortigue et al., 2004)]. Fast visual processing and propagation of information from visual to visuomotor coordination centers should also be expected in a task-like interception involving the dorsal visuomotor pathway.

Surprisingly, few functional studies have addressed the issue of information processing and its time course during interception until now. In monkeys, Merchant, Battaglia-Mayer, and Georgopoulos (2004b) have reported relationships between activations in parietal and frontal areas and different stimulation parameters during interception using a joystick of a target following a circular trajectory. In humans, some studies involving visuomotor tasks (such as tracking) have described activation patterns with functional magnetic resonance

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imaging (MRI) that involved the dorsal visual pathway (Oreja-Guevara et al., 2004; Turner, Grafton, Votaw, Delong, & Hoffman, 1998; Kertzman, Schwarz, Zeffiro, & Hallett, 1997; Grafton, Mazziotta, Woods, & Phelps, 1992). These studies, however, do not provide any temporal information on brain activation, which is critical in a fast interception task. Nishitani, Uutela, Shibasaki, and Hari (1999) have recorded magnetic fields evoked by the direction change of a moving target in an eye–hand pursuit task. This kind of task, just like interception, necessitates continuous processing of target motion and eye–hand movement coordination. Relatively long latencies of activations (about 230 msec) in the occipital cortex in response to target direction changes were reported. Further, activation latencies in parietal and frontal areas (253–275 and 258–269 msec, respectively) were clearly not able to account for the short reaction times observed in behavioral data. Finally, although the medio-temporal area V5 is known to be dedicated to motion processing (Watson et al., 1993; Zeki et al., 1991), Nishitani et al. (1999) did not report any significant activation in this region.

The primary goal of the present study is then to provide a synopsis of the spatio-temporal unfolding of cortical activations elicited by an interception task that engages visuomotor coordination with severe timing constraints. In this context, we specifically assume that fast responses should be detectable by noninvasive neuroimaging techniques with high temporal resolution. The experimental paradigm therefore consisted of MEG recordings during the ecological situation of catching a falling ball with the hand. This situation was balanced with a control condition in which subject had only to watch the ball falling. We provide evidence of fast brain responses elicited by the high-speed stimulus present in both tasks, that extended along the dorsal pathway, from striate and extrastriate visual areas to distant central motor regions. We also show catch and non-catch-specific activations in sensorimotor areas.

METHODS

Subjects

Seven healthy right-handed subjects [5 women (S1 to S5), 2 men (S6 to S7)] with normal vision, ages between 22 and 35 years, and with no particular training in high-speed ball games, gave informed consent to participate to our experiment, which was carried out in accordance with the rules of the La Salpêtrière Hospital Ethical Committee.

Experimental Setup

Subjects were seated in the Paris 151-channel MEG system [synthetic third-order gradiometers (Vrba et al., 1999), 2 cm coil diameter, 5 cm baseline; VSM MedTech, Coquitlam, BC, Canada] (Figure 1). A 10-cm-long vertical tube was fixed to the MEG gantry, pointing right above their legs. Subjects were asked to visually fixate a red light-emitting diode (LED) located at the lower extremity of the tube, from which a white tennis ball was released (125 g and 6 cm diameter, i.e., 6.32° of visual angle on average). Thus, the ball trajectory was projected on the inferior visual field of the subject. All subjects were able to see the complete fall of the ball (70 cm distance, visual angle of 70°) while still fixating the LED. Subjects wore plastic earphones diffusing white noise to cover any mechanical sounds that could be caused by the falling ball. Because of the variability in head shapes and positions in the sensor helmet, the distance between the hand and the initial release position of the ball was adapted individually to each subject. These adjustments guaranteed that subjects were able, first, to see the fixation point and, second, to keep their head as close as possible to the MEG sensors. Consequently, the resulting times of flight of the ball could not be kept strictly constant across subjects (see Data Analysis).

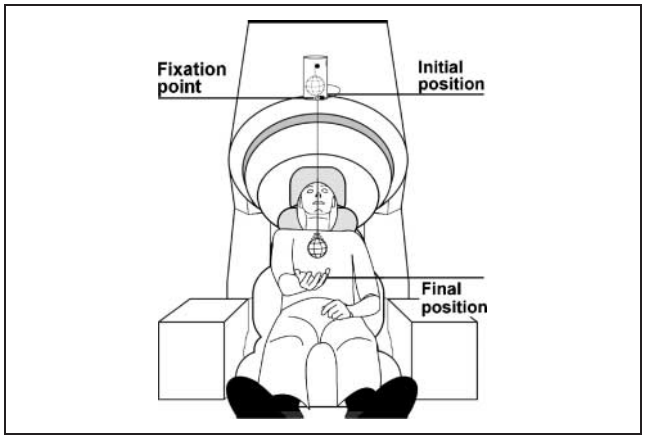

Figure 1. Experimental setup: The ball was initially hidden in the tube just above the fixation point (LED). The ball then started to fall toward the subject's open hand. Subjects had to grasp the ball at the time of impact while maintaining their eyes on the fixation point.

During the main experiment (''*Catch*''), subjects had to maintain their right arm semi-extended, with their hand supinated in the sagittal plane under the vertical tube, while their left hand had to remain still along their body. Ball release was triggered manually by the experimenter for each trial. The ball had no initial velocity and started to fall down toward the subject's hand at about 50 cm in front of the subject's eyes. Subjects were therefore not required to reach out for the ball to catch it, but rather had to grasp the ball when arriving in their hand while still watching the fixation point.

One hundred ball releases (trials) were performed within a single recording run for subsequent computation of average individual evoked magnetic fields. After the subject had caught and then released the ball, it was pulled back up into the tube by the experimenter before the next trial could begin within the next 1 to 10 sec.

Although this release was triggered manually and pseudorandomly by the experimenter, we cannot exclude that subjects were anticipating the next ball release during the 100 trials, just like a tennis player does while facing a tennis ball machine during training and getting prepared to the next launch.

A control condition (''*No-catch*'') was performed by all subjects in a subsequent run starting after few minutes of rest. In this control, both subject's hands were lying along their legs. The ball was dropped by the experimenter as in the *Catch* condition and fell on a thick and soft blanket covering subject's legs and absorbing the impact. The subjects' task consisted in looking at the fixation point and direct their attention on the falling ball but without catching it.

Data Acquisition

MEG, electrooculographic (EOG) and electromyographic (EMG) (bipolar surface electrodes; right *flexor carpi* muscles or *wrist flexors*) data were simultaneously recorded (1250 Hz sampling rate; [0.3, 600] Hz band-pass filter).

Head localization was achieved by placing coils on three anatomical landmarks (nasion and periauricular points) at the beginning of each run. A sound signal recorded through a microphone placed near the hand of the subject provided a time reference for impact of the ball in the hand. Data acquisition was triggered by the ball cutting the beam of an optical detector located at the lower extremity of the release tube. This event also corresponded to the time of release of the ball (t = 0 sec) as it simultaneously entered the subject's field of view. Data were collected between 0.5 sec before and 1.5 sec after the ball release. All subsequent analysis will focus on the [−0.5 sec, +0.5 sec] time window about ball release, which includes time to impact for all subjects.

T1-weighted (IR gradient-echo) MRI of individual subject anatomy was completed after MEG recording. To facilitate MEG/MRI registration, vitamin E pills were placed on the same anatomical landmarks as the ones used as fiducials during the MEG session.

Data Analysis

The time of impact was obtained from the latency of the first peak from the microphone signal. The ball time of flight ranged from 349 to 372 msec (370 ± 17 msec, mean ± standard deviation) across subjects for the *Catch* condition and slightly longer for thee control condition (between 364 and 422 msec; 392 ± 27 msec). Time to impact across trials was fairly stable within subjects (individual standard deviations ranging from 3 to 12 msec and 3 to 14 msec for the *Catch* and *No-catch* conditions, respectively).

EMG data were rectified and averaged relative to t = 0 sec for each subject. Initiation time of EMG was defined as the time when the EMG signal averaged on a 10-msec sliding window reached one standard deviation above the average EMG signal computed over the baseline (t = −0.5 to t = 0 sec).

MEG data were digitally band-pass filtered ([1.26, 40] Hz) off-line using a zero-lag filter and compensated for sensor DC offset. Trials contaminated by eye blinks and/or eye movements were discarded. Remaining trials were then averaged relative to the time of ball release (t = 0 sec). For group analysis purposes, a cross-subjects grand average of MEG was computed after individual evoked MEG time series were coregistered to a single average sensor array with a method adapted from Uutela, Taulu, and Hamalainen (2001).

Source Estimation

The individual anatomy obtained from MRI was prepared for source localization: automatic extraction and tessellation of scalp and gray matter were processed with the BrainVISA software solution (http://brainvisa.info). Elementary current dipoles were placed normally at each node of the cortical tessellation, forming a 6000-source distribution over the cortex. For each subject, cortically constrained imaging of the neural generators of MEG surface magnetic fields was achieved by the linear minimum-norm estimator (MNE) with subject-specific spherical head model (Hamalainen & Ilmoniemi, 1994) available in the BrainStorm software (Baillet, Mosher, & Leahy, 2001; http://neuroimage.usc.edu/brainstorm). Time series of each elementary dipole from the distributed source model were normalized with regard to their respective baseline, thereby yielding a Z-score map of deviations of individual currents. Hence, for each time step t and each dipole i:

$$Z_i(t) = \frac{(x_i(t) - \mu_i)}{\sigma_i},$$

with $x_i(t)$ the original source amplitude as estimated from the MNE model; μ_i the empirical mean and σ_i the standard deviation of source amplitude of dipole i over the baseline (taken as [−0.4 sec, −0.1 sec] before ball release). Absolute Z-score maps were then thresholded above $Z = 10$ for subsequent analysis and interpretation. Cortical activations above this threshold were therefore considered as significantly deviating from baseline with $p < \frac{1}{10^2} = .01$, uncorrected, as specified by the Tchebychev upper bound (Papoulis, 1991).

Data Reduction

The grand-average of Z-normalized dipole intensity maps over subjects was obtained following affine coregistration

of brain surfaces into the MNI referential (BrainStorm software). The resulting average of normalized current densities was finally interpolated over the cortical surface of the MNI Colin27 brain template (Holmes et al., 1998) and thresholded to $Z > 15$ for display.

Visual inspection of the timeline of the resulting current map revealed strong activation within three main brain areas that were tagged as regions of interest (ROIs) (see Results section for detailed anatomical descriptions). Two regions were found being bilaterally activated. Hence, for cross-checking purposes, the third ROI was defined symmetrically over both hemispheres. Consequently, each ROI was manually identified on every individual anatomy, while respecting the consistency of its anatomical location with a spatial surface area of 5 cm^2 (see Figure 2). The instantaneous maximum source amplitude at each time instant was finally extracted as a measure of the regional activation within each ROI.

To account for the intersubject variability of activation latencies (defined as time-to-peak measures), amplitudes of regional activation in each ROI were integrated over an adaptive time window of ±20% about peak latencies of interest.

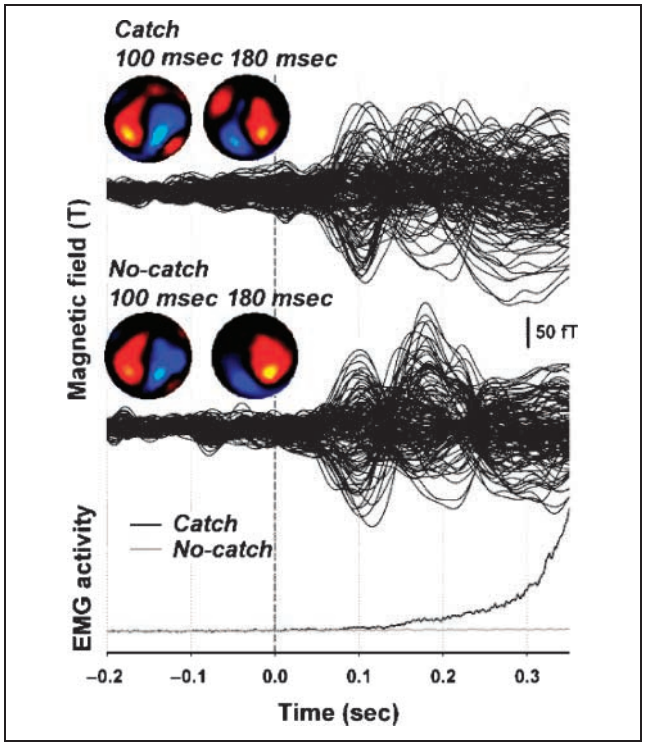

Figure 3. (Top) Grand-averaged (n = 7) evoked field for the *Catch* and *No-catch*. conditions. Topographical maps for the two first main MEG peaks are inserted at the corresponding latencies. (Bottom) Averaged (n = 7) EMG signal from right wrist flexor muscles for the *Catch* (black) and *No-catch* (gray) conditions.

RESULTS

The *Catch* condition was correctly performed by subjects as confirmed by the early build up of anticipatory muscular activity in the right wrist muscles (Figure 3, bottom). This activity was detected in all subjects with, however, quite variable onset ([70, 190] msec range; 111 ± 40 msec after ball release, mean ± standard deviation). As expected, no EMG activity was found during the control *No-catch* condition except for Subject S4, who demonstrated some impact specific activation in some trials. These trials were removed from the analysis.

Waveform Analysis

The first component (labeled ''M100'') of the grand-averaged evoked field recorded on scalp surface (Figure 3, top) started at about 50 msec and peaked around 100 msec after ball release in both experimental conditions, with no significant difference [two-way analysis of variance (ANOVA) with repeated measures, $p > .05$]

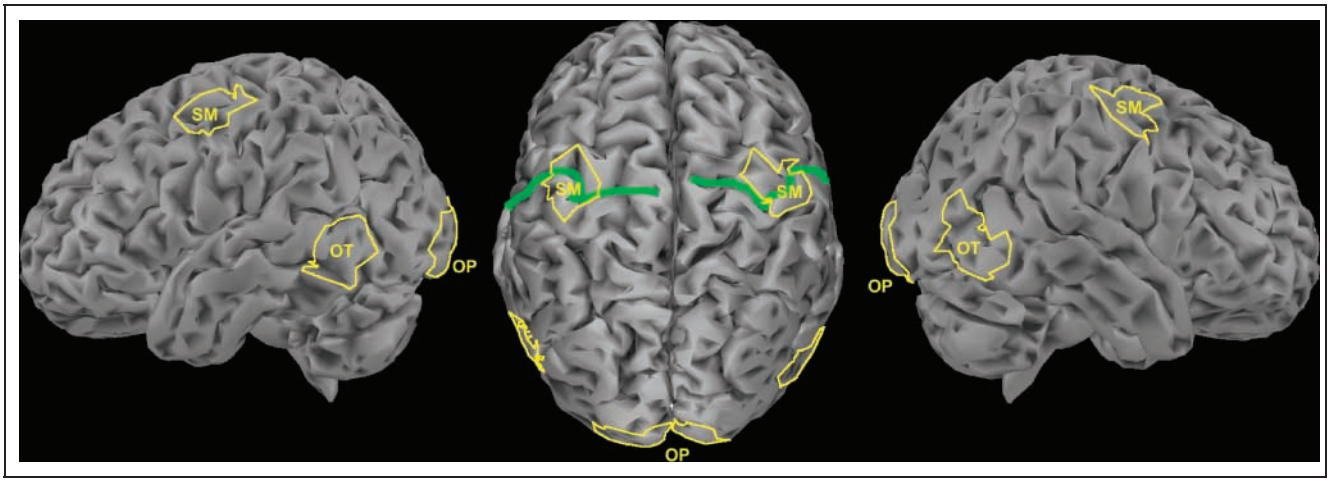

Figure 2. Localization and extent over individual anatomy of the ROIs OP, OT, and SM (mean area ≈ 5 cm^2). See Results for definition of the ROIs.

in peak latency between the *Catch* (108 ± 13 msec) and the *No-catch* conditions (103 ± 11 msec). For a given subject, the latencies of responses in both experimental conditions were similar (±9 msec). Topographical maps revealed that this component was related to a bilateral occipito-temporal distribution of magnetic field (see inserts above traces in Figure 3, top) in both conditions.

A second component (labeled ''M200''), rising significantly later than the M100 [$F(1, 6) = 106.5$, $p < .05$], was observed on the grand-averaged evoked field at about 180 msec after ball release in both experimental conditions (182 ± 22 msec for the *Catch* condition and 179 ± 25 msec for the *No-catch* condition, two-way ANOVA, $p > .05$). However, topographical maps at this latency revealed a differential distribution of magnetic field on the left hemisphere between both conditions: a symmetric parieto-occipital pattern of magnetic fields was present in both conditions (see Figure 3, top), whereas a more anterior central contralateral activity was observed in the *Catch* condition only.

Latency values reported here must be considered carefully as a slow anticipatory activity was shown to build up from about 50 msec before ball release (see Figures 3 and 5). We cannot completely reject the hypothesis of a trial-specific anticipation that could have risen despite the pseudorandomization of release times. As this activity could bias the observed latencies relative to ball release, we will only consider relative latencies (i.e., durations between the earliest and subsequent peak latencies of activation).

Source Distribution

The minimum norm solution for evaluation of source activation (Figure 4) revealed remarkably fast propagation

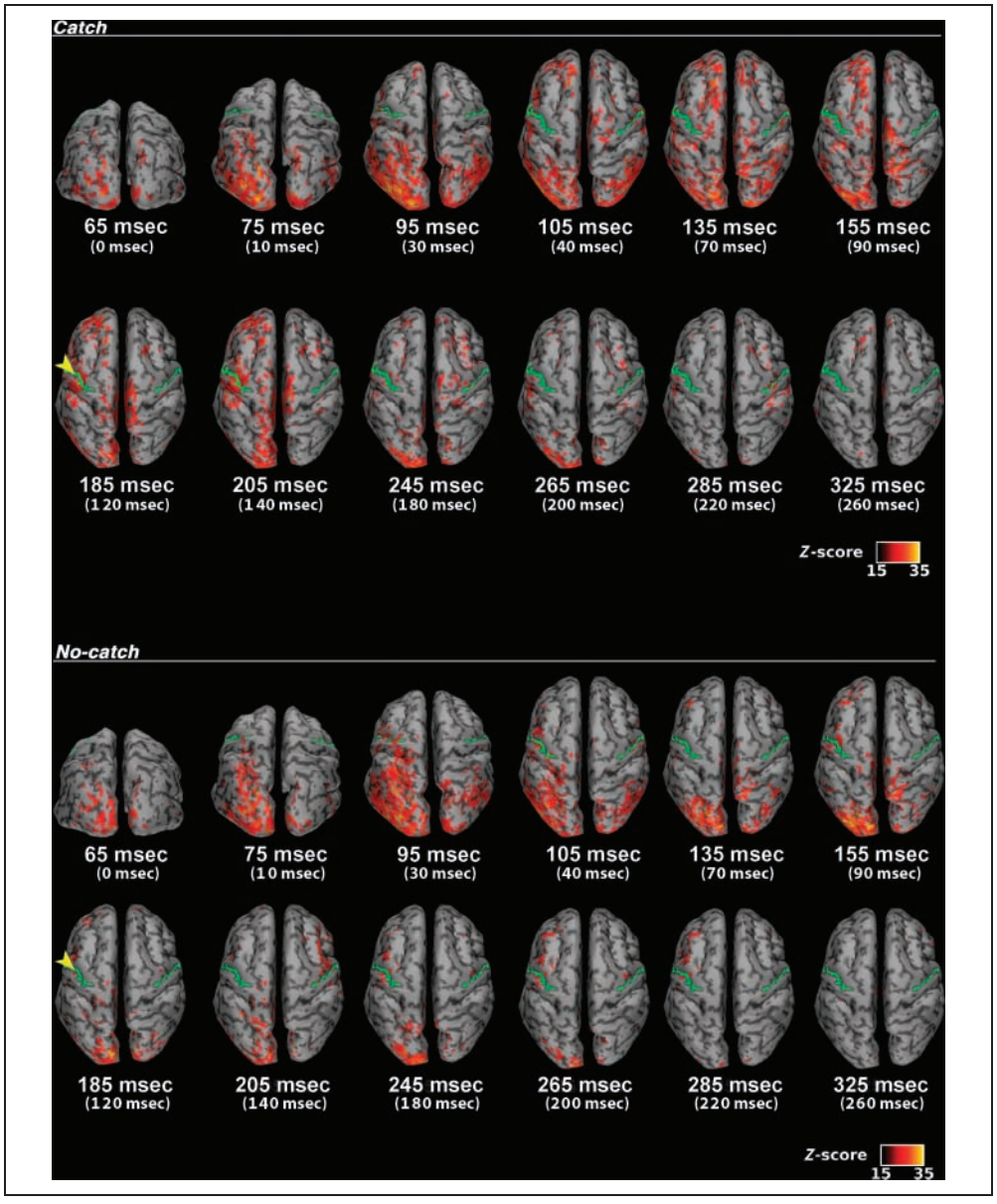

Figure 4. *Z*-normalized current dipole intensity maps averaged over subjects after normalization into the MNI space. Group data are represented over the inflated surface of the Colin27 template and maps thresholded above $Z = 15$ ($p < .005$, uncorrected) for clarity. Left and right central sulci are underlined in green. For each map, relative latencies are provided into brackets. Note the *Catch*-specific activation of the sensorimotor region 120 msec after activation in the occipital pole area (yellow arrow). Initial occipital view of the brain is gradually rotated to a top view to present the parietal and frontal activations after 30 msec.

of brain activations over the contralateral visuomotor dorsal pathway for all subjects in the *Catch* condition. Major sustained activations were initiated in the occipital pole and rapidly unfolded to the occipito-temporo-parietal junction and mainly over the left posterior parietal cortex within about 30 msec, and reached the left superior frontal lobe within 70 msec. Left contralateral sensorimotor regions then became activated 90–120 msec after occipital activations. The activity over the left superior parietal and sensorimotor region then persisted for about 50 msec. The two components, M100 and M200, identified on the averaged evoked field, corresponded respectively to the large activation of the occipito-temporo-parietal junction and to the superior parietal and sensorimotor regions. The remarkable point here is that the activity in the sensorimotor cortex appeared only 90 msec after activation in the striate and extrastriate cortex. During control condition (*No-catch*), about the same pattern of activation was found in the occipital, occipito-temporo-parietal, and superior parietal cortex, but differed in the frontal and sensorimotor cortex. Especially, the large sensorimotor activation seen 90 msec after activity started in the occipital pole was absent, confirming what had been observed on topographical sensor maps (see Waveform Analysis).

ROI Analysis

We identified three ROIs from the grand average of source activations during the first 200 msec (see Figure 2): the occipital pole (OP), the occipito-temporal region (OT), and the primary sensorimotor hand area (SM). Only these regions were considered for further analysis. Activations in the left superior parietal and superior frontal lobe sustained too large a latency jitter to be retained as ROIs. Specific anatomical locations of the three retained ROIs were: the occipital pole in OP; the intersection point of the inferior temporal sulcus (ITS) and its ascending limb (ALITS) in OT; and the central sulcus at the location of maximal evoked activation at the ball impact in the SM. Figure 5A displays the time course of the grand average of normalized neural currents in all three ROIs in both hemispheres.

Four ANOVAs with repeated measures were conducted on peak amplitude measures in each ROI to test

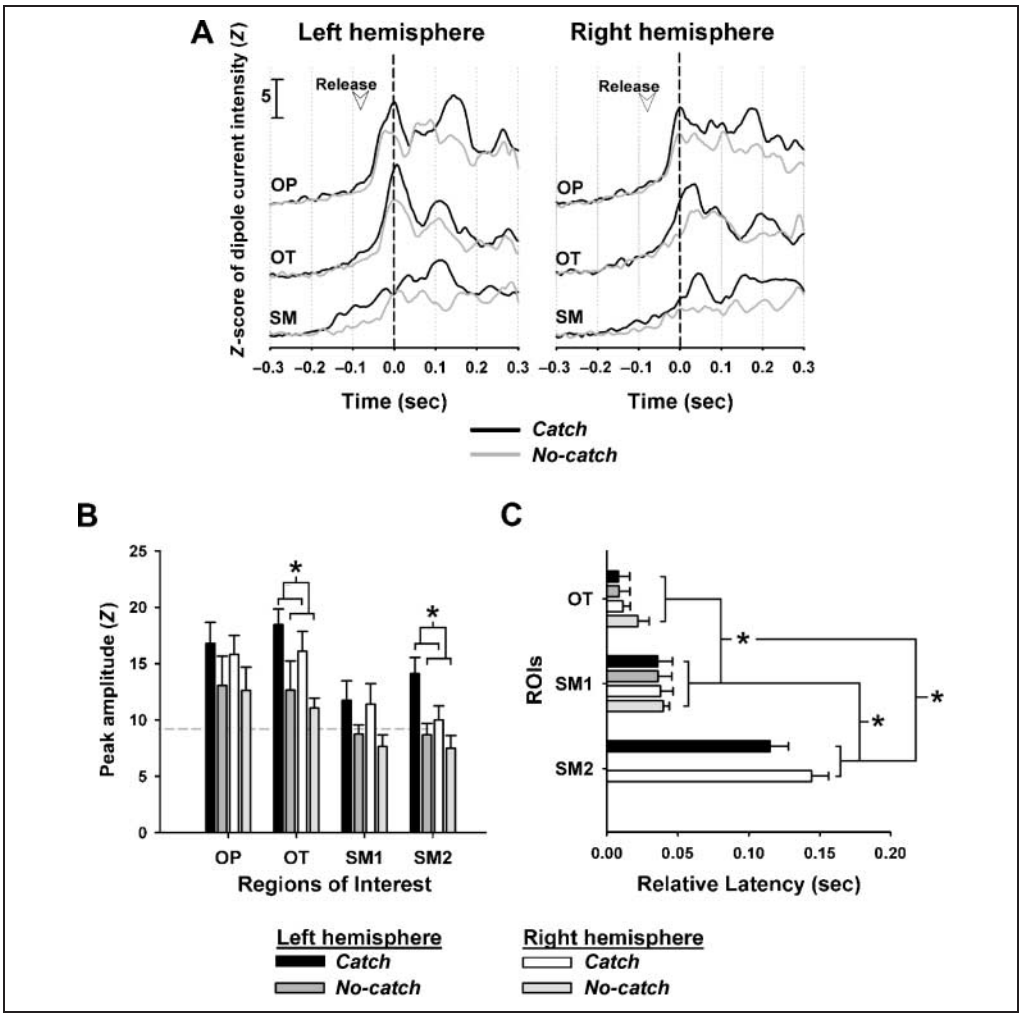

Figure 5. (A) Time course of mean neural current magnitudes within all ROIs relative to the first peak of activation in the OP region ($t = 0$). (B) Normalized amplitudes (*Z*-scores) of neural current dipole intensity. (C) Latencies of main peak of neural current intensity, relatively to ball release time, for each ROI in both hemispheres.

for differences with respect to the factors ''hemisphere'' and ''experimental condition.'' In OP and OT, peaks of activity above threshold ($Z = 10$) were present in both hemisphere and conditions. Amplitude during the *Catch* condition was systematically higher than during the *No-catch* condition in both hemispheres, but this difference was only significant in the OT area [$F(1, 6) = 11.55$, $p < .05$] (Figure 5B). Two peaks of activity were identified in the SM area and differed with respect to their specificity to the experimental conditions. The earliest peak reached above threshold in both hemispheres in the *Catch* condition but was only present (although slightly under-threshold) on the left side in the *No-catch* condition. However, no significant difference in amplitudes between conditions and hemispheres was found. On the contrary, the latest and predominant peak was specifically found in the *Catch* condition on the left side that is contralateral to the acting hand. At the same latency, a decrease of activity was clearly observed (see Figure 5A) on the right (res. left) side in the *Catch* (res. *No-catch*) condition. The ANOVA revealed a significant effect of the experimental condition [$F(1, 6) = 10.81$, $p < .05$] but failed to demonstrate a significant effect of the hemisphere.

Detailed analysis of the peak latencies evidenced remarkably fast-propagating brain processing from perception to action along the dorsal pathway. Activations in all three bilateral ROIs peaked within 40 msec in a postero-anterior sequence.

Figure 5C displays the relative latencies of the main peaks of activity with respect to the peak latency in the OP region. A three-way (Hemisphere × Condition × ROI) ANOVA with repeated measures revealed a significant effect of the ROI [$F(3, 18) = 135.71$, $p < .01$] on peak latencies. However, a post hoc analysis (Newman–Keuls) revealed that the activity in the OT region peaked at about the same time than in the OP. No significant differences in latencies were found between hemispheres or conditions (see Table 1).

Table 1. Relative Peak Latencies of Activity (msec) in Each ROI with Respect to Corresponding Peak Latencies in the OP Region (Absolute Peak Latencies in OP are Shown between Brackets)

	Left Hemisphere		*Right Hemisphere*	
	Catch	*No-catch*	*Catch*	*No-catch*
OP	0 (89 ± 10)	0 (90 ± 16)	0 (88 ± 14)	0 (87 ± 16)
OT	8 ± 21	9 ± 20	11 ± 14	22 ± 21
SM1	36 ± 28	36 ± 26	38 ± 24	40 ± 11
SM2	115 ± 34	–	144 ± 32	–

Values corresponding to peaks whose amplitudes fall below the threshold ($Z = 10$) and are significantly different ($p < .05$) from those in the other condition and/or hemisphere are left blank.

More distinctively, activity reached the SM cortex through superior parietal cortex significantly later than in OP and OT regions (Newman–Keuls, $p < .05$). The first peak of activity that was present in the *Catch* and *No-catch* conditions arose about 40 msec after the peak activity in the OP. The second component, specific to the *Catch* condition, peaked at about 115 msec in the hemisphere contralateral to the catching hand (see Table 1 for the respective latencies).

In summary, a fast propagation pattern of activations was evidenced in both experimental conditions from occipital and occipito-temporal areas to the central cortex within 40 msec. This pattern is not related to the catching action as revealed by the absence of EMG activity during the *No-catch* condition. Moreover, a later *Catch*-specific peak of activation reached the SM region within 115 msec.

DISCUSSION

We used a simple but ecological catching paradigm to help unveil the sequence of main cortical activations engaged in interceptive actions. Subjects were asked to catch a free-falling ball with the hand while fixating its point of appearance in their field of view. Our study reveals a fast propagation of activations—reaching from occipital to SM regions—over the left dorsal visual pathway within about 40 msec. This finding indicates that the brain may perform all the necessary information processing from perception to appropriate action in such a remarkably short time interval.

Methodological Considerations

We found an anticipatory activity beginning about 50 msec before ball release that was particularly clear on the time course of SM sources. This activation cannot be explained by the phase-preserving filters that were applied to the data. One hypothesis is that this early deflection in the signal reflects a trial-specific anticipation of ball release that could shorten the absolute latencies related to ball appearance. However, the absolute latencies of the first peak of activation we found in the OP (about 80 msec) in response to motion could not be considered as specifically early (e.g., 40–70 msec; see Moradi et al., 2003; Buchner et al., 1997; Uusitalo, Virsu, Salenius, Nasanen, & Hari, 1997; Ffytche, Guy, & Zeki, 1995) but rather correspond to the classical P1 component to motion onset [65–120 msec; see (Bonmassar et al., 2001; Probst, Plendl, Paulus, Wist, & Scherg, 1993; Ahlfors, Ilmoniemi, & Hamalainen, 1992)]. On the contrary, the fast propagation of activation in OP and SM regions is in accordance with the timing constraints of the task. However, as we cannot firmly exclude that some implicit pace emerged in the trial series, we decided to present relative latencies of

activations with respect to the earliest activations found in the occipital cortex.

Main Activations for Catching

We found a propagating wave of cortical activations that unfolded principally over the dorsal visual stream that is known to support visuomotor coordination processes (Milner & Goodale, 1993; Goodale & Milner, 1992). Our approach consisted of the detailed analysis of the spatio-temporal cascade of activations restricted to representative, functionally task-relevant ROIs located along this pathway and that were clearly identifiable from the functional images of neural currents. These regions included the bilateral posterior occipital (OP), OT, and SM cortices. Our analysis clearly leave out several cortical and subcortical regions that are likely involved in the task. However, given the spatial resolution of the MEG, our objective was not to provide an exhaustive list of the areas involved in the task, but rather, a synopsis of the spatio-temporal unfolding of cortical activations. In the following, we briefly discuss the role of the selected areas in the task.

The OP ROI may well match the lower areas of the visual cortex V1, V2, and V3A. These areas are known to respond to static but also to moving visual stimuli (Culham, He, Dukelow, & Verstraten, 2001; Sunaert, Van Hecke, Marchal, & Orban, 1999).

The OT ROI was found to support rather widespread activations. However, OT probe was located at the intersection point of the ITS and its ascending limb (ALITS) defined by Dumoulin et al. (2000) as the best landmark to localize area V5, that is believed to match monkey areas MT and MST (see Culham et al., 2001). However, given the spatial resolution of our source imaging method, we cannot exclude a contribution of surrounding areas responsive to different components of visual motion [e.g., V3/V3a (Ahlfors et al., 1999; Tootell et al., 1997) or KO (Orban et al., 1995)]. The role of V5 in the processing of visual motion is extensively documented in literature (e.g., Tootell et al., 1997; Watson et al., 1993; Zeki et al., 1991), and its activation is obvious in our task. It is interesting to note that activation of V5 has also been reported in visuomotor (Oreja-Guevara et al., 2004) and catching tasks (Schenk, Ellison, Rice, & Milner, 2005; Schenk, Mai, Ditterich, & Zihl, 2000).

The SM ROI is assumed to be the cortical output toward spinal motor neurons. Its activation during the *Catch* condition was then highly expected. However, we also found a nonspecific activation of M1 during the *No-catch* condition that could suggest a role in the timing of movement initiation. This hypothesis is discussed below.

Significant activations in the left superior parietal cortex and in the left superior frontal lobe were found in all subjects, but with ample variability in localization, spatial extension, and latencies, which prevented us from considering this region as an additional ROI.

Catch vs. *No-catch*

On the methodological point of view, the very similar current density maps we found from two independent sets of data confirm the reliability of the source reconstruction algorithm.

Indeed, we found remarkably similar spatio-temporal pattern of activation in the *Catch* and *No-catch* conditions not only in visual regions as expected but also in parietal and even SM areas, although with lower intensity (non-catch-specific activation in the SM region 40 msec after activations of the OP region). This was true on average data but also for single subjects. This suggests that similar mechanisms are involved in both tasks until a significant difference is observed in the primary motor cortex 115 msec after onset of activation in occipital areas (catch-specific activation).

How can we explain the parietal activations and especially this activity in the primary motor cortex in the *No-catch* condition? We propose that information processing in this kind of tasks is stimulus rather than task dependent. The underlying idea is that success of interceptive (or avoidance) tasks is conditioned by a fast reaction time. When we intercept or avoid a fast approaching object, we did not always intend to (and then did not prepare for) do(ing) it because we are simply not always able to anticipate this event as in a laboratory environment. In the more critical situations, we rather need to react to the approaching stimulus as fast as possible. Then, it is likely that fast stimuli are processed rapidly whether we intend to act upon them or not. If we have to act, at least we are in time to. This implies that any region involved in the processing of stimulus parameters should be involved even when no action is required. Neuronal activity in the primary motor cortex has been found to be related to the motion parameters of the stimulus during interceptive ''go'' and ''no-go ''tasks with a joystick (Port, Kruse, Lee, & Georgopoulos, 2001). Similar results have been found during presentation of a different kind of purely visual stimulation (optic flow, circular real and, in a less extent, apparent motion) (Merchant, Battaglia-Mayer, & Georgopoulos, 2004a; Merchant, Battaglia, & Georgopoulos, 2001). These authors also trained monkeys to intercept a target following a circular motion in a go/no-go task (Merchant et al., 2004b) and showed that parietal area 7a and the primary motor cortex were activated during both tasks, suggesting that processing of target parameters and action preparation took place in these two areas. More recently, Field and Wann (2005) used functional MRI to study the areas activated by a TTC judgment task using looming visual stimuli. Despite the absence of any interception movement, the authors report activations that were

specific of TTC estimate in several areas of the left dorsal stream and especially in the superior parietal and motor cortex. Although we cannot provide any direct evidence of an activity in M1 related to the stimulus parameters or a TTC estimate (the ball always felt with the same kinematics), the temporal resolution of the MEG allowed us to dissociate a common early SM component presumably involved in the processing of visual information and its integration with motor parameters and a later component more likely involved in the triggering of action.

An alternative explanation could arise from the fact that the *No-catch* session was always performed after the *Catch* session (as a control condition). One could argue that simply watching the ball falling after having caught it one hundred times evokes a nonvoluntary simulation of the action. However, this hypothesis seems unlikely. First, we did not ask subjects to simulate grasping during the *No-catch* session but only to focus their attention on the falling ball. Second, kinesthetic imagery is a conscious process (Jeannerod, 1994) that requires strong attention from the subject and a training session in most cases. However, we cannot reject the hypothesis of a habituation phenomenon that could have biased the results of the *No-catch* condition and could be easily tested by intermixing the experimental conditions.

Timing along the Dorsal Visuomotor Pathway

We found fast propagation of activations within the extended network of brain areas involved in the anticipatory motor activity required to catch the ball. Our findings indicate that only about 40 msec (from peak to peak) were necessary for brain activations to travel from visual to motor cortices over the dorsal pathway. This chronometry is well in line with behavioral parameters of interceptive actions. Although anticipatory priming of the response cannot be excluded, the addition of classical latencies (65–120 msec) of activations in the primary visual cortex with the 40-msec propagation time to SM areas is very much compatible with reaction times typically reported in the literature (about 140 msec; Day & Lyon, 2000; Brenner & Smeets, 1997). In an intermodal selective attention paradigm, Foxe and Simpson (2002) have shown that the processing of visual information through the dorsal pathway could, however, be achieved within 80 msec after stimulus onset. We show in this study that similar time ranges of activation can be found in humans along the dorsal stream, in the context of a highly anticipatory visuomotor task triggered in response to a high-speed visual stimulus.

OT Region

The first signal component we describe peaks at about 100 msec (M100) in both experimental situations. We could explain it by at least two sources of activation, one in the OP region, the other, more lateral on the OT region. These regions are likely to match V1/V2 and V5, respectively, and sustain very similar temporal patterns of activation. Peak latencies in these two regions were separated by only 10 msec, on average, considering both hemispheres and both experimental conditions. Moreover, they were clearly temporally overlapping for some subjects. Some previous MEG studies reported delayed early component in response to motion onset (150–210 msec; see Amano, Kuriki, & Takeda, 2005; Anderson, Holliday, Singh, & Harding, 1996). In these studies, the ''M150'' peak was also explained by two source components localized in V1/V2 and V5+ that were successively activated within 15–50 msec, only with a slightly longer duration than the one we report here. Because of the generalized anticipatory activation that could have shortened the latencies we are reporting, we cannot exclude that the M100 component in our study matches the ''M150'' peak of activation. However, some other studies (Schellart, Trindade, Reits, Verbunt, & Spekreijse, 2004; Moradi et al., 2003; Uusitalo et al., 1997) reported on M100-compatible earlier latencies but that were modeled by sources located only in the striate cortex, likely in V1/V2. A second component was, however, described as including activation in V5+, concomitant or slightly later (0–20 msec) than activation in V1/V2 (see Moradi et al., 2003; Uusitalo et al., 1997). Note that Schellart et al. (2004) reported a 35-msec delay between striate and extrastriate sources, the latter being localized in V3/V3a that could have contributed to our OT activation as well. Our results are also well in line with VEP studies the classical P1 (likely originating in V1/V2) and N2 (possibly originating in V5+) components peaking at 110–135 msec and 150–170 msec, respectively, but often overlapping (Delon-Martin et al., 2006; Henning, Merboldt, & Frahm, 2005; Kremlacek, Kuba, Chlubnova, & Kubova, 2004). Whatever the absolute latency of these activations, we were able to show that a fast (if not simultaneous) propagation of signal over striate and extrastriate signals can be observed in response to a fast stimulus that subjects had to act on it or not.

SM Regions

The second peak of activation (M200) was explained by at least one source localized in the SM cortex. This source showed a first peak of activation 40 msec after activation in the OP in both experimental condition and then a second at 115 msec but only in the *Catch* condition. The few studies that report relative latencies of activation in M1 with respect to visual areas are in accordance with our findings. Foxe and Simpson (2002) reported that propagation of activations from occipital to frontal regions could be completed within 30 msec. Moreover, the absolute latencies of magnetic responses in this area [from 145 msec (early peak) to 212 msec (late peak)] are in agreement with previous studies on

visually triggered actions in monkeys (Riehle, 1991) and humans (Thut et al., 2000; Schluter, Rushworth, Mills, & Passingham, 1999). Specifically, Schluter et al. (1999) used transcranial magnetic stimulation (TMS) to disrupt a cued movement task. Movement was slowed down by TMS when applied at 140 msec over the anterior premotor cortex, and 220 msec when applied over the SM cortex. These latencies are very close to those reported in our study.

In conclusion, we used MEG to study a simple but ecological catching task and report a fast (about 50 msec) propagation of activation related to visual motion processing for action over the visuomotor dorsal pathway. This wave of activation involved at least three regions that we localized in V1/V2, V5+, and M1 using a distributed source solution constrained on individual anatomy. The first two regions were activated during catching and simple observation of the falling ball in a similar manner. As expected, the M1 area evidenced a *Catch*-specific activation but also some earlier activations common to the *Catch* and *No-catch* conditions that suggest a common stimulus-dependent processing of a fast-moving object that could be caught. The very close, sometimes overlapping, latencies found in V1/V2 and V5+ is promising for subsequent studies. Early latencies in visual motion areas relative to V1/V2 have indeed been related to a possible subcortical route that would bypass V1/V2 and could be triggered by fast stimuli (Ffytche et al., 1995). The superior colliculus could then be connected to V5 through the pulvinar and still play a role in some condition. This hypothesis is of particular interest in the framework of interception because the superior colliculus is largely involved in interception and avoidance behavior in submammalian species. It is then very tempting to propose that this phyllogenetically old cerebral structures could have remained functional in humans and play a particular role in fast interception. However, more precise study of absolute latencies, if not subcortical data, would be necessary to investigate this hypothesis.

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REFERENCES

Ahlfors, S., Ilmoniemi, R., & Hamalainen, M. (1992). Estimates of visually evoked cortical currents. *Electroencephalography and Clinical Neurophysiology, 82,* 225–236.

Ahlfors, S., Simpson, G., Dale, A., Belliveau, J., Liu, A., Korvenoja, A., et al. (1999). Spatiotemporal activity of a cortical network for processing visual motion revealed by MEG and fMRI. *Journal of Neurophysiology, 82,* 2545–2555.

Amano, K., Kuriki, I., & Takeda, T. (2005). Direction-specific adaptation of magnetic responses to motion onset. *Vision Research, 45,* 2533–2548.

Anderson, S. J., Holliday, I. E., Singh, K. D., & Harding, G. F. (1996). Localization and functional analysis of human cortical area V5 using magneto-encephalography. *Proceedings of the Royal Society of London, Series B, Biological Sciences, 263,* 423–431.

Baillet, S., Mosher, J., & Leahy, R. (2001). Electromagnetic brain mapping. *IEEE Signal Processing Magazine, 18,* 14–30.

Bonmassar, G., Schwartz, D. P., Liu, A. K., Kwong, K. K., Dale, A. M., & Belliveau, J. W. (2001). Spatiotemporal brain imaging of visual-evoked activity using interleaved EEG and fMRI recordings. *Neuroimage, 13,* 1035–1043.

Brenner, E., & Smeets, J. (1997). Fast responses of the human hand to changes in target position. *Journal of Motor Behavior, 29,* 297–310.

Buchner, H., Gobbele, R., Wagner, M., Fuchs, M., Waberski, T., & Beckmann, R. (1997). Fast visual evoked potential input into human area V5. *NeuroReport, 8,* 2419–2422.

Culham, J., He, S., Dukelow, S., & Verstraten, F. (2001). Visual motion and the human brain: What has neuroimaging told us? *Acta Psychologica, 107,* 69–94.

Day, B., & Lyon, I. (2000). Voluntary modification of automatic arm movements evoked by motion of a visual target. *Experimental Brain Research, 130,* 159–168.

Delon-Martin, C., Gobbele, R., Buchner, H., Haug, B. A., Antal, A., Darvas, F., et al. (2006). Temporal pattern of source activities evoked by different types of motion onset stimuli. *Neuroimage, 31,* 1567–1579.

Dumoulin, S., Bittar, R., Kabani, N., Baker, C., Le Goualher, G., Bruce, P., et al. (2000). A new anatomical landmark for reliable identification of human area V5/MT: A quantitative analysis of sulcal patterning. *Cerebral Cortex, 10,* 454–463.

Ffytche, D., Guy, C., & Zeki, S. (1995). The parallel visual motion inputs into areas V1 and V5 of human cerebral cortex. *Brain, 118,* 1375–1394.

Field, D. T., & Wann, J. P. (2005). Perceiving time to collision activates the sensorimotor cortex. *Current Biology, 15,* 453–458.

Foxe, J., & Simpson, G. (2002). Flow of activation from V1 to frontal cortex in humans. A framework for defining ''early'' visual processing. *Experimental Brain Research, 142,* 139–150.

Georgopoulos, A. P., Kalaska, J. F., & Massey, J. T. (1981). Spatial trajectories and reaction times of aimed movements: Effects of practice, uncertainty, and change in target location. *Journal of Neurophysiology, 46,* 725–743.

Goodale, M. A., & Milner, A. D. (1992). Separate visual pathways for perception and action. *Trends in Neurosciences, 15,* 20–25.

Grafton, S., Mazziotta, J., Woods, R., & Phelps, M. (1992). Human functional anatomy of visually guided finger movements. *Brain, 115,* 565–587.

Hamalainen, M. S., & Ilmoniemi, R. J. (1994). Interpreting magnetic fields of the brain: Minimum norm estimates.

Medical & Biological Engineering & Computing, 32, 35–42.
Henning, S., Merboldt, K., & Frahm, J. (2005). Simultaneous recordings of visual evoked potentials and BOLD MRI activations in response to visual motion processing. *NMR in Biomedicine, 18,* 543–552.
Holmes, C. J., Hoge, R., Collins, L., Woods, R., Toga, A. W., & Evans, A. C. (1998). Enhancement of MR images using registration for signal averaging. *Journal of Computer Assisted Tomography, 22,* 324–333.
Jeannerod, M. (1994). The representing brain: Neural correlates of motor intention and imagery. *Behavioral and Brain Sciences, 17,* 187–245.
Kertzman, C., Schwarz, U., Zeffiro, T., & Hallett, M. (1997). The role of posterior parietal cortex in visually guided reaching movements in humans. *Experimental Brain Research, 114,* 170–183.
Kremlacek, J., Kuba, M., Chlubnova, J., & Kubova, Z. (2004). Effect of stimulus localisation on motion-onset VEP. *Vision Research, 44,* 2989–3000.
Lacquaniti, F., Carrozzo, M., & Borghese, N. (1993). The role of vision in tuning anticipatory motor responses of the limbs. In A. Berthoz (Ed.), *Multisensory control of movement* (pp. 379–393). Oxford: Oxford University Press.
Lee, D. (1976). A theory of visual control of bracking based on information about time-to-collision. *Perception, 5,* 437–459.
McIntyre, J., Zago, M., Berthoz, A., & Lacquaniti, F. (2001). Does the brain model Newton's laws? *Nature Neuroscience, 4,* 693–694.
McLeod, P. (1987). Visual reaction time and high-speed ball games. *Perception, 16,* 49–59.
Merchant, H., Battaglia, M., & Georgopoulos, A. (2001). Effects of optic flow in motor cortex and area 7a. *Journal of Neurophysiology, 86,* 1937–1954.
Merchant, H., Battaglia-Mayer, A., & Georgopoulos, A. (2004a). Neural responses in motor cortex and area 7a to real and apparent motion. *Experimental Brain Research, 154,* 291–307.
Merchant, H., Battaglia-Mayer, A., & Georgopoulos, A. (2004b). Neural responses during interception of real and apparent circularly moving stimuli in motor cortex and area 7a. *Cerebral Cortex, 14,* 314–331.
Milner, A., & Goodale, M. (1993). Visual pathways to perception and action. *Progress in Brain Research, 95,* 317–337.
Moradi, F., Liu, L., Cheng, K., Waggoner, R., Tanaka, K., & Ioannides, A. (2003). Consistent and precise localization of brain activity in human primary visual cortex by MEG and fMRI. *Neuroimage, 18,* 595–609.
Nishitani, N., Uutela, K., Shibasaki, H., & Hari, R. (1999). Cortical visuomotor integration during eye pursuit and eye–finger pursuit. *Journal of Neuroscience, 19,* 2647–2657.
Orban, G., Dupont, P., De Bruyn, B., Vogels, R., Vandenberghe, R., & Mortelmans, L. (1995). A motion area in human visual cortex. *Proceedings of the National Academy of Sciences, U.S.A., 92,* 993–997.
Oreja-Guevara, C., Kleiser, R., Paulus, W., Kruse, W., Seitz, R., & Hoffmann, K. (2004). The role of V5 (hMT+) in visually guided hand movements: An fMRI study. *European Journal of Neuroscience, 19,* 3113–3120.
Ortigue, S., Michel, C., Murray, M., Mohr, C., Carbonnel, S., & Landis, T. (2004). Electrical neuroimaging reveals early generator modulation to emotional words. *Neuroimage, 21,* 1242–1251.
Papoulis, A. (1991). *Probability, random variables, and stochastic processes*. New York: McGraw-Hill.
Paulignan, Y., Jeannerod, M., MacKenzie, C., & Marteniuk, R. (1991). Selective perturbation of visual input during prehension movements: 2. The effects of changing object size. *Experimental Brain Research, 87,* 407–420.
Pelisson, D., Prablanc, C., Goodale, M. A., & Jeannerod, M. (1986). Visual control of reaching movements without vision of the limb: II. Evidence of fast unconscious processes correcting the trajectory of the hand to the final position of a double-step stimulus. *Experimental Brain Research, 62,* 303–311.
Port, N., Kruse, W., Lee, D., & Georgopoulos, A. (2001). Motor cortical activity during interception of moving targets. *Journal of Cognitive Neuroscience, 13,* 306–318.
Prablanc, C., & Martin, O. (1992). Automatic control during hand reaching at undetected two-dimensional target displacements. *Journal of Neurophysiology, 67,* 455–469.
Probst, T., Plendl, H., Paulus, W., Wist, E., & Scherg, M. (1993). Identification of the visual motion area (area V5) in the human brain by dipole source analysis. *Experimental Brain Research, 93,* 345–351.
Regan, D. (1997). Visual factors in hitting and catching. *Journal of Sports Sciences, 15,* 533–558.
Riehle, A. (1991). Visually induced signal-locked neuronal activity changes in precentral motor areas of the monkey: Hierarchical progression of signal processing. *Brain Research, 540,* 131–137.
Schellart, N., Trindade, M., Reits, D., Verbunt, J., & Spekreijse, H. (2004). Temporal and spatial congruence of components of motion-onset evoked responses investigated by whole-head magneto-electroencephalography. *Vision Research, 44,* 119–134.
Schenk, T., Ellison, A., Rice, N., & Milner, A. (2005). The role of V5/MT+ in the control of catching movements: An rTMS study. *Neuropsychologia, 43,* 189–198.
Schenk, T., Mai, N., Ditterich, J., & Zihl, J. (2000). Can a motion-blind patient reach for moving objects? *European Journal of Neuroscience, 12,* 3351–3360.
Schluter, N., Rushworth, M., Mills, K., & Passingham, R. (1999). Signal-, set-, and movement-related activity in the human premotor cortex. *Neuropsychologia, 37,* 233–243.
Seeck, M., Michel, C., Mainwaring, N., Cosgrove, R., Blume, H., Ives, J., et al. (1997). Evidence for rapid face recognition from human scalp and intracranial electrodes. *NeuroReport, 8,* 2749–2754.
Sunaert, S., Van Hecke, P., Marchal, G., & Orban, G. (1999). Motion-responsive regions of the human brain. *Experimental Brain Research, 127,* 355–370.
Thorpe, S., Fize, D., & Marlot, C. (1996). Speed of processing in the human visual system. *Nature, 381,* 520–522.
Thut, G., Hauert, C., Blanke, O., Morand, S., Seeck, M., Gonzalez, S., et al. (2000). Visually induced activity in human frontal motor areas during simple visuomotor performance. *NeuroReport, 11,* 2843–2848.
Tootell, R., Mendola, J., Hadjikhani, N., Ledden, P., Liu, A., Reppas, J., et al. (1997). Functional analysis of V3A and related areas in human visual cortex. *Journal of Neuroscience, 17,* 7060–7078.
Turner, R., Grafton, S., Votaw, J., Delong, M., & Hoffman, J. (1998). Motor subcircuits mediating the control of movement velocity: A PET study. *Journal of Neurophysiology, 80,* 2162–2176.
Uusitalo, M., Virsu, V., Salenius, S., Nasanen, R., & Hari, R. (1997). Activation of human V5 complex and rolandic regions in association with moving visual stimuli. *Neuroimage, 5,* 241–250.

Uutela, K., Taulu, S., & Hamalainen, M. (2001). Detecting and correcting for head movements in neuromagnetic measurements. *Neuroimage, 14,* 1424–1431.

Vrba, J., Anderson, G., Betts, K., Burbank, M., Cheung, T., Cheyne, D., et al. (1999). 151-Channel whole-cortex MEG system for seated or supine positions. In T. Yoshimoto, M. Kotani, S. Kuriki, & H. Karibe (Eds.), *Recent advances in biomagnetism* (pp. 93–96). Sendai: Tohoku University Press.

Watson, J., Myers, R., Frackowiak, R., Hajnal, J., Woods, R., Mazziotta, J., et al. (1993). Area V5 of the human brain: Evidence from a combined study using positron emission tomography and magnetic resonance imaging. *Cerebral Cortex, 3,* 79–94.

Zago, M., & Lacquaniti, F. (2005). Visual perception and interception of falling objects: A review of evidence for an internal model of gravity. *Journal of Neural Engineering, 2,* S198–S208.

Zeki, S., Watson, J., Lueck, C., Friston, K., Kennard, C., & Frackowiak, R. (1991). A direct demonstration of functional specialization in human visual cortex. *Journal of Neuroscience, 11,* 641–649.