An 'automatic pilot' for the hand in human posterior parietal cortex: toward reinterpreting optic ataxia

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We designed a protocol distinguishing between automatic and intentional motor reactions to changes in target location triggered at movement onset. In response to target jumps, but not to a similar change cued by a color switch, normal subjects often could not avoid automatically correcting fast aiming movements. This suggests that an 'automatic pilot' relying on spatial vision drives fast corrective arm movements that can escape intentional control. In a patient with a bilateral posterior parietal cortex (PPC) lesion, motor corrections could only be slow and deliberate. We propose that 'on-line' control is the most specific function of the PPC and that optic ataxia could result from a disruption of automatic hand guidance.

Suppose that in the middle of a given action, the goal is unexpectedly altered. In reaction to this change, the reorganization of ongoing action entails a modification of the predefined motor program[1]. The complex computations required to modify the motor output would be expected to delay the reaction to the goal perturbation. However, in studies of the control of simple goal-directed actions, even short-duration movements can be corrected on-line. For example, fast modifications of motor components are observed during grasping movements in response to changes in object orientation[2,3] or location[4]. Similarly, after a fast pointing movement has been programmed and initiated toward a visual target, it can be corrected without a significant increase in movement time[5,6], that is, without reprogramming a new motor output. Such on-line corrections can be observed whether or not[7] the target displacement is consciously perceived[8,9]. If the corrective system can bypass the conscious decision level, to what extent can such automatic system resist intentional control? Could corrective movements be considered as an equivalent, in normal subjects, of the neuropsychological observations of unwilled movements toward objects (for example, anarchic hand syndrome[10,11])?

Here we investigated the power of this automatic process to resist voluntary control during a pointing action. Movement interruption seems to be one of the fastest motor responses, because it is a nonspecific reaction that can be completely preprogrammed, and it can be rapidly achieved by a peripherally operating inhibition mechanism[12,13]. We therefore tested whether the 'automatic pilot' guiding on-line motor corrections is so autonomous that it can counteract a conflicting instruction to interrupt the movement in-flight. Subjects pointed at visual targets that could unexpectedly change location by a target jump (experiment 1) or a color change (experiment 2). These two types of perturbations were designed to test whether automatic corrective processes can be triggered in response to a chromatic change as well as to a target jump. They were also used as stop signals to create the conflict between the automatic correction system and voluntary motor control.

The finding that behavioral automatisms are released especially in patients with frontal lobe lesions[14,15] suggests that they depend on more-posterior brain structures. The PPC is implicated in both planning[16] and execution[17–19] of visually guided movements. In an attempt to identify the neurological basis for a specific process responsible for automatically guiding the hand to a visual target, we investigated the performance of a patient with a bilateral lesion of the PPC. In particular, we tested whether this lesion would differentially affect automatic and voluntary motor control.

Subjects were instructed to point at a green target, which remained unperturbed in 80% of the trials. In contrast to classical stop-signal protocols[20–22], we explored whether the ongoing hand movement could be corrected or interrupted. Accordingly, our stop signals consisted of a target perturbation triggered by the motor response itself (movement onset) in 20% of the trials. Thus, all movements were programmed on the basis of an identical set of information. Consequently, differences between perturbed and unperturbed trials could be attributed to the influence of the perturbation on movement execution.

Results

Inhibiting versus correcting in response to target jump

In the first experiment, one green target was initially presented, which could unexpectedly jump to the right or to the left. Subjects in the location-stop group were instructed to interrupt their ongoing movement in response to target jump; its direction was thus irrelevant. Other subjects in the location-go group were presented with the same set of stimuli, but were instructed to correct the movement in response to target jump (that is, to redirect the finger to the new target location). The experimental procedure allowed us to sample a large range of movement durations (Methods). Targets could appear at two possible locations. Final

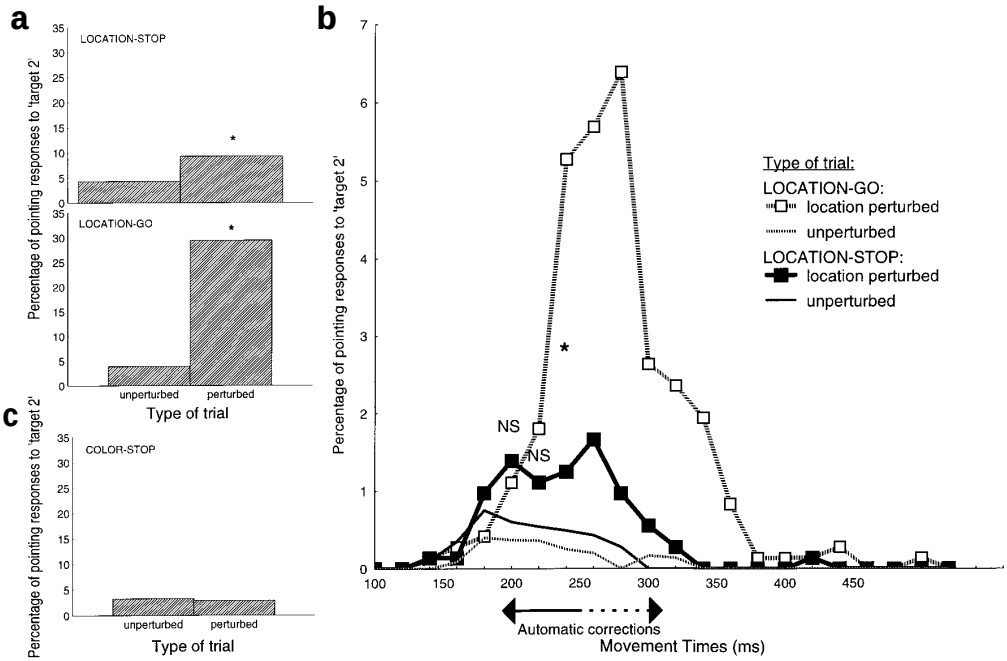

Fig. 1. Corrections made as a function of instruction and stimulus. The percentage of pointing responses to target 2 was calculated for each type of trial with respect to the total number of trials of this type completed by all subjects. (**a**) Unwilled correction movements occurred even when countermanded (experiment 1). Corrections to target 2 were made to a significant extent (*) not only by the location-go group (ANOVA repeated measures, $F_{1,5} = 136.8$; $p < 0.05$, compared with unperturbed trials) but also, despite opposite instruction, by the location-stop group ($F_{1,5} = 12.8$; $p < 0.05$). In location-stop, a total of 9% of all the perturbed trials reached target 2. (**b**) A significant percentage of correction movements appeared in response to target jump at the same movement time for the two groups (shown by the divergence between perturbed and unperturbed trials for the movement duration of 200 ms; binomial $p < 0.05$). Responses to the perturbation began to differ between the two groups only for a movement duration of 240 ms (*binomial $p < 0.05$, earliest significant divergence). This value corresponded to the shortest movement time compatible with non-automatic corrections for the location-go group, and with inhibitory control for the location-stop group. Nevertheless, automatic corrections were still produced until movement times of about 300 ms, which allowed voluntary control to fully prevail over automatic visual guidance. (**c**) A color switch between two targets was not able to elicit automatic corrections (experiment 2). Although the change in target location was physically the same as in the location-stop condition, no correction movement toward the new green target was produced by the color-stop group (ANOVA repeated measures, $F_{1,5} = 3.18$; $p > 0.1$). In this condition, responses to the perturbation always complied with the stop instruction.

hand positions were labeled as reaches to target 1 (initial target location) or target 2 (second target location) based on accuracy confidence intervals calculated for unperturbed trials. For the range of movement durations examined, pointing responses around target 2 were considered as real reactions to perturbation only when their proportion was significantly higher than the baseline proportion of unperturbed trials landing in the same area.

Strict compliance with the 'stop' instruction would cause location-stop subjects to either succeed in stopping their movement or fail to interrupt their action and therefore reach the primary target position. In contrast to this prediction, a significant percentage of corrective movements occurred despite the 'stop' instruction in the location-stop group as well as in accordance with the 'correction' instruction in the location-go group (both $p < 0.05$; Fig. 1a). After touching the displaced target, subjects of the location-stop group were aware of their mistakes and spontaneously expressed strong frustration. Irrepressible motor corrections were thus driven toward the new target location. This demonstrated that visuomotor processing of location for reach correction was systematically activated during movement execution, even when the perturbation should have led to another instructed response. This automatic visuomotor transformation was able to attract the hand away from its initially programmed trajectory. Furthermore, the occurrence of non-instructed corrections indicated that stop signal location was processed, even when it was irrelevant to the task.

Next, all recorded movements were analyzed as a function of movement time to determine the temporal distribution of pointing movements to target 2 (Fig. 1b). Movement times ranged from about 100 ms to 450 ms with a Gaussian distribution (Fig. 2). We predicted that if erroneous responses observed despite instruction resulted from a failure to inhibit automatic corrections, they would be expected to occur in a limited temporal window. Subjects in the location-go group were able to produce the instructed correction movements for movement times longer than 200 ms. In response to the same target perturbation in location, the location-stop group produced disallowed corrections over a narrower range of movement times (between about 200 ms and 300 ms). Therefore the erroneous corrections could not be explained on the basis of careless mistakes, which would be randomly distributed over the whole range of movement time. They instead seemed to reflect a fast 'hand capture' by the target, escaping the slower processes of voluntary interruption. In addition, in response to the perturbation, the number of responses classed as corrections to target 2 significantly increased within the same movement duration of 200 ms (binomial $p < 0.05$) in both location-go and location-stop groups. This similar timing suggested that the earliest corrections observed in both groups resulted from an identical visuomotor guidance, which was independent of instruction and thus automatic. Moreover, the effect of instruction appeared only for minimal movement durations of 240 ms and manifested itself as a significant divergence between responses to the perturbation by the two groups (binomial $p < 0.05$). Correction movements made for movement times ranging from 200 ms to 240 ms occurred irrespective of the instruction. They could therefore be considered

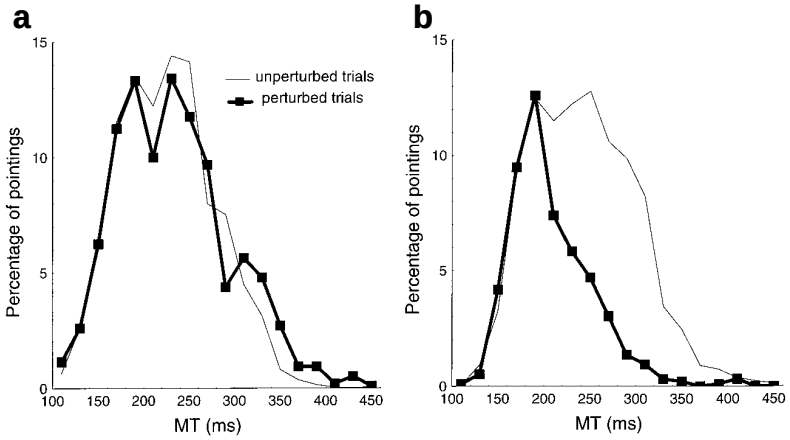

Fig. 2. Distribution of all movement durations in experiment 1 (to be compared to the occurrence of corrections to target 2; **Fig. 1b**). The number of movements falling in each duration class was randomly distributed based on the subjects' behavior. As the number of unperturbed trials was four times that of perturbed trials (80%/20%), we provide the percentage of movements occurring in each class of movement time (MT) with respect to the total number of perturbed and unperturbed trials. For unperturbed trials, normal distributions were observed. (**a**) In the location-go condition, only a very limited number of perturbed movements lasted longer than unperturbed trials. The small accessory peak at about 320 ms may have corresponded to movements involving a slow correction. (**b**) In the location-stop condition, the MT distributions for perturbed trials showed progressive suppression of touches (that is, compliance with instruction).

as fully automatic reactions to the target jump for the two groups of subjects. Finally, for movement times beyond this temporal window, a second pool of corrective movements appeared in the location-go group. As they did not appear in the location-stop group, these additional corrections observed in the location-go group must have been intentionally produced.

Is the 'automatic pilot' specific for target jumps?

In a second experiment, we tested whether this automatic visuomotor guidance occurred when the change in target location was encoded through a chromatic perturbation. Green and red targets were presented simultaneously in the two positions used in experiment 1. The subjects were instructed to point at the green one, and the color of the two targets could be unexpectedly interchanged at movement onset. Two groups of subjects were instructed either to interrupt (color-stop group) or to correct (color-go group) their ongoing movement in response to the perturbation. In contrast to the location-stop group (experiment 1), no automatic corrective movements were observed in the color-stop group ($p > 0.1$; Figs. 1c and 3d). Corrections by the color-go group involved a significant increase in movement time compared with the mean duration of unperturbed trials (ANOVA Scheffé's *post hoc*, $p < 0.05$). In the location-go group (experiment 1), corrections were made without a significant increase in movement time (ANOVA Scheffé's *post hoc*, $p > 0.40$). This was also demonstrated in experiment 3 (compare Fig. 4a and b). Thus, only intentional corrections involving additional time-consuming processes could be produced based on a color cue (Fig. 3b), and the visuomotor transformations of the hand's 'automatic pilot' may be specific to location processing.

Behavior following bilateral posterior parietal lesion

To test the neurological correlates of this automatic hand guidance, we studied a patient with a bilateral PPC lesion. The patient (I.G.), a right-handed 29-year-old woman, suffered an ischemic stroke involving Brodmann's areas 19, 18, 7 and 39 as well as the intraparietal sulcus of both hemispheres (Fig. 5). An examination two months after the stroke demonstrated a pure bilateral optic ataxia. She could move both arms normally, and showed neither somatosensory deficit nor visual deficit for the stimuli set used in the present experiments. She completed the tasks described in experiments 1 and 2, combining a location or color perturbation with a correction or interruption instruction. Several minor modifications were made to the previous protocols to compensate for the patient's pointing variability and fatigability (Methods). Three new control subjects accomplished the tasks under identical conditions and exhibited a behavioral pattern similar to that obtained in experiments 1 and 2.

I.G. was always able to verbally describe the target perturbations. When allowed to make free eye and head movements, she pointed accurately toward stationary targets. Like other patients with bilateral optic ataxia tested in central vision[23–26], she did not produce systematic pointing errors, although she exhibited a larger pointing variability. Her movement duration tended to be overall slightly longer than that of the control subjects for the unperturbed and perturbed trials (Figs. 3 and 4). Nevertheless, she consistently produced movements in the range of 200–300 ms, which allowed us to test her ability to make automatic corrections.

Whereas I.G. processed the location change normally to interrupt her movements (Fig. 3c), she exhibited an abnormal pattern of corrections across the two location conditions. In contrast to control subjects (here and in experiment 1), I.G. did not produce any disallowed corrective movements in the location-stop condition (Fig. 3c). In the location-go condition, I.G. produced mostly slow corrective movements and showed a major reduction in the pool of fast corrections with respect to controls (Fig. 3a). By contrast, she had almost-normal timing for slow corrections in response to target color changes (Fig. 3b). Together, these results suggested that only automatic corrections specific to target jumps were severely impaired, whereas slower intentional corrections could still be made. In addition, I.G. succeeded in interrupting movements in response to both types of perturbations as fast as normal subjects (about 260 ms in location-stop, Fig. 3c, and 290 ms in color-stop, Fig. 3d). This normal performance confirmed that her impairment could not be explained by a general slowing of visual and/or motor processing. We also compared the spontaneous comments made by the patient and the control subjects at the end of the experiment. Control subjects generally reported that "to redirect your movements, you don't need to concentrate as hard as when you have to interrupt them," whereas I.G. commented that "stopping is easier, because to change direction, you first need to work out the direction of the target jump."

Discussion

These experiments were aimed at creating a conflict between the hand's 'automatic pilot' and voluntary motor control. In per-

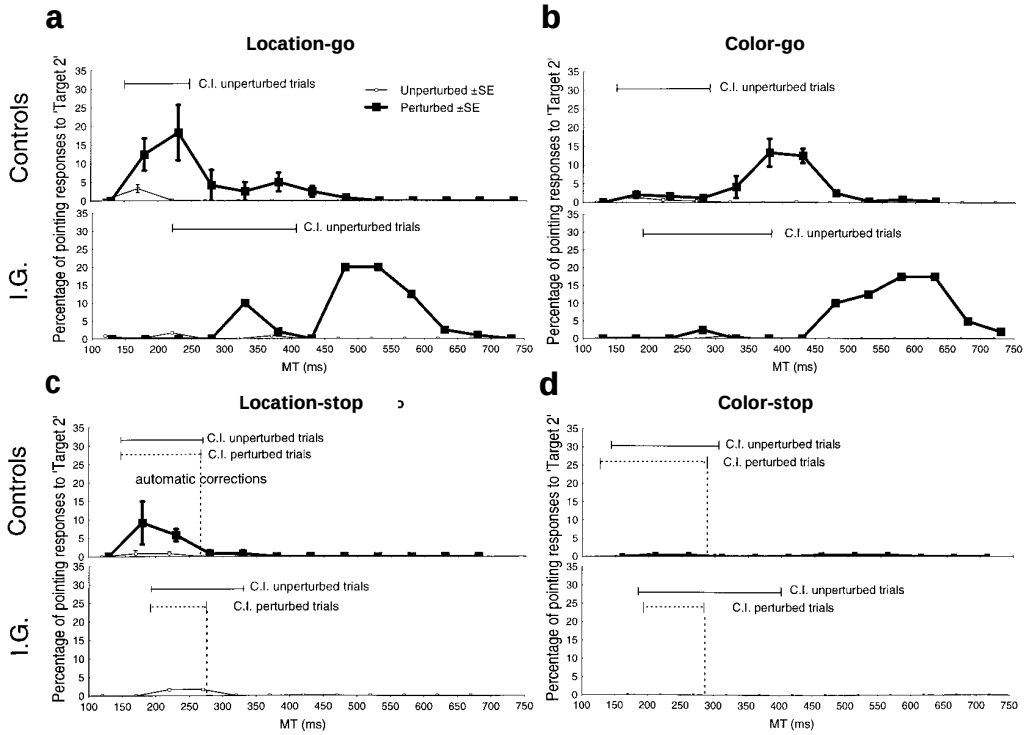

Fig. 3. Specific disruption of automatic corrections following a bilateral parietal lesion. The pointing performance of three control subjects was compared with that of patient I.G. in each of the four tested conditions. Horizontal bars, 95% confidence intervals of movement time computed for all unperturbed trials. Vertical dotted lines (**c**, **d**), upper edge of the 95% confidence interval of movement time computed for all non-interrupted perturbed trials (dotted horizontal bars). (**a**) Location-go. Controls (top) had two pools of corrections: main pool, 'fast' corrections made with movement times comparable to those in unperturbed trials (as in experiment 1); secondary pool, some 'slow' corrections made with a substantial increase of movement duration compared with that of unperturbed trials (horizontal bar). In contrast, most corrections produced by patient I.G. (bottom) caused a large increase in movement time, and very few of them could be considered as 'fast' corrections. (**b**) Color-go. The distribution of corrections was similar in the controls (top) and in the patient (I.G.; bottom): only slow corrections were observed in response to the color change. (**c**) Location-stop. The patient (I.G.; bottom) produced no corrective responses in this stop session, whereas about 10% of the perturbed trials elicited disallowed corrective responses in the controls (top; as in experiment 1). For the interruption response, the patient performed similarly to normal subjects (vertical dotted lines). (**d**) Color-stop. Patient I.G. (bottom) was again able to stop her movement for the same movement duration (280 ms) as normal subjects (top, vertical dotted lines). No automatic corrective movements were observed in response to color, either in controls or in the patient.

turbed trials, automatic corrections and voluntary inhibition were synchronously activated by a target location change at the movement onset. Unexpectedly, for movements of a given duration (Fig. 1b), disallowed corrections were produced toward the new target just as frequently as when subjects were instructed to redirect their movements. The correction system, therefore, was able to take priority over the voluntary motor control system.

The remarkable speed of hand-movement corrections[5,9] has been contrasted with the slowness of visual awareness[27]. For example, vocal reaction to target jumps occurs much later than motor reaction[28]. We provide here a related but more-direct comparison within the field of action, confirming that correction is the fastest response to a target jump. This is counterintuitive if one considers that the computation required to make a corrective movement toward an unexpected direction is complex[13]. It demonstrates that a given sensorimotor response could be automatized to such an extent that it became faster than a simpler preprogrammed stopping response.

A previous experiment[5] indicated that 'on-line' corrective processes are dissociated from conscious visual processing. In our experiment, the subjects of the location-stop group (experiment 1) had never been instructed to correct their movement in response to the target location change. Yet instead of simply complying with the stop instruction, they spontaneously made motor corrections. Our results therefore confirmed that a stimulus-driven 'automatic pilot' was activated by default and was inherent to the execution of a goal-directed action. Moreover, by requiring subjects to interrupt their movement in response to detectable target jumps, we strengthened the idea of automatic motor control proposed before[5], as the corrective processes were activated even when the subjects had another motor intention. Whereas the PPC participates in both eye and arm movements[16,29], the effects observed in the patient here could not be fully explained by an alteration of eye movements, as she had normal oculomotor behavior, and the targets were all presented within central vision.

Our results showed that neurologically normal subjects produced irrepressible corrections of simple aiming movements. This automatic behavior may be related to the environment dependency syndrome following frontal lobe lesion[14,15]. Specifically, the subservience to the external stimulus exhibited here by normal subjects could be considered as an attenuated equivalent to frontal utilization behavior[14,30]. Disallowed corrections could be attributed to a weakness of intentional control, as described in frontal patients in whom "the ongoing activity exerts a more powerful influence than the encoded intention-action"[31]. In support of this hypothesis, a preliminary experiment showed that a patient with a lesion of the dorsolateral convexity of the frontal lobe made 100% corrections when the target jump was associated with a stop instruction (location-stop condition), although she was able to verbally repeat the instruction throughout the experiment. However, the frustration expressed by our normal

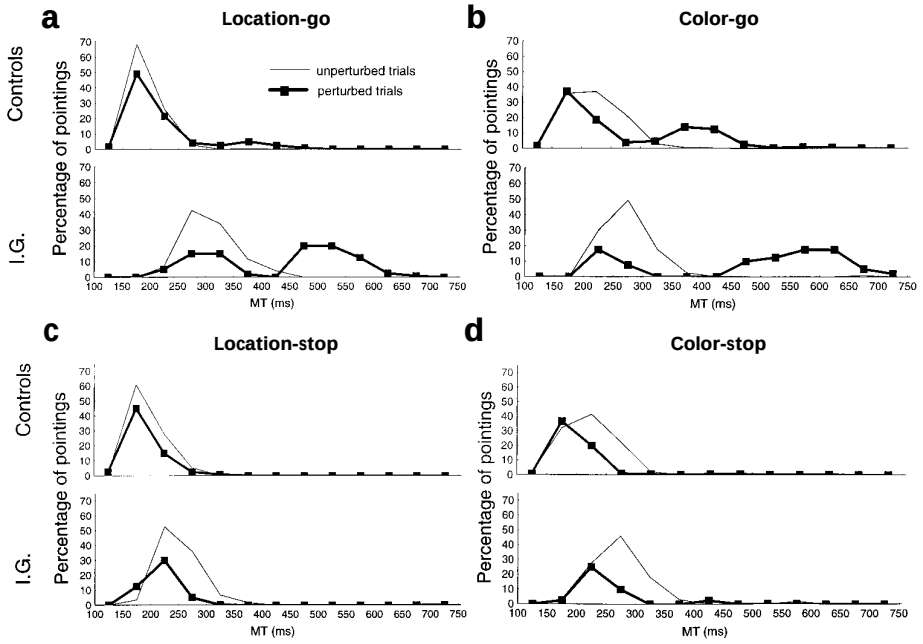

Fig. 4. Distribution of all movement durations in experiment 3 (to be compared to the occurrence of corrections to target 2; **Fig. 3**). Patient I.G. consistently performed slightly slower than control subjects. Nevertheless, she produced movements in a range compatible with automatic corrections (that is, 200–300 ms) in all conditions. Unperturbed trials had a normal distribution. In the go conditions, the MT distributions obtained for perturbed trials could become bimodal. With respect to unperturbed trials, an additional pool of slower movements resulted from slow corrections. In location-go, control subjects made mostly fast corrections: the MT distribution of perturbed trials remains very similar to that of unperturbed trials. The patient showed the same behavior in the location-go and in the color-go conditions. In the stop conditions, the MT distributions for perturbed trials showed the progressive suppression of touches.

subjects because of the corrections that escaped them stood in contrast to the usual lack of concern displayed by frontal patients. This main difference may be linked to a time factor. Impairment in frontal patients occurs for the execution of serial tasks lasting several seconds[14,31]. In contrast, normal subjects' automatic guidance was released only for simple aiming movements falling within a narrow time window. Their automatic corrections resulted from only a temporary lack of voluntary control. It may be hypothesized that normal subjects behaved in the same way as frontal patients because the speed constraints of our protocol did not leave the frontal lobe enough time to inhibit automatic corrective processes.

Interestingly, no unwanted corrections were observed when normal subjects were instructed to interrupt their movement in response to a color-cued target jump. In addition, when correction was instructed to a color cue, only slow corrective movements were produced. These findings indicated that automatic corrections could be made in response to a simple target jump, but not when the same target change was encoded through a color switch. This specificity for spatial attribute and the high-speed constraints suggested that the dorsal stream of the visual brain may be the substrate for the hand's automatic pilot. In addition to its function in location processing[16,32], the PPC exhibits particularly short neuronal latencies to visual stimulation compared with other prestriate visual areas[33]. Furthermore, parietal areas have direct anatomical projections onto the dorsal premotor cortex[27,34,35]. These two features are consistent with the requirement of a fast-operating corrective system.

In the patient with a bilateral PPC lesion, performance seemed nearly normal in response to color, which is more specific to the ventral stream of visual processing. The patient could use a target location change to trigger an interruption response or to make slow corrections, but not to make fast automatic corrections. This pattern of results fit well with the idea of a specific posterior parietal module responsible for processing on-line (automatic) motor adjustments in response to a location perturbation. Conversely, this module cannot be critically involved in the perception of location change itself or in the general ability to react to it. The patient's data thus support the electrophysiological demonstration that the PPC is a specialized visuomotor inter-

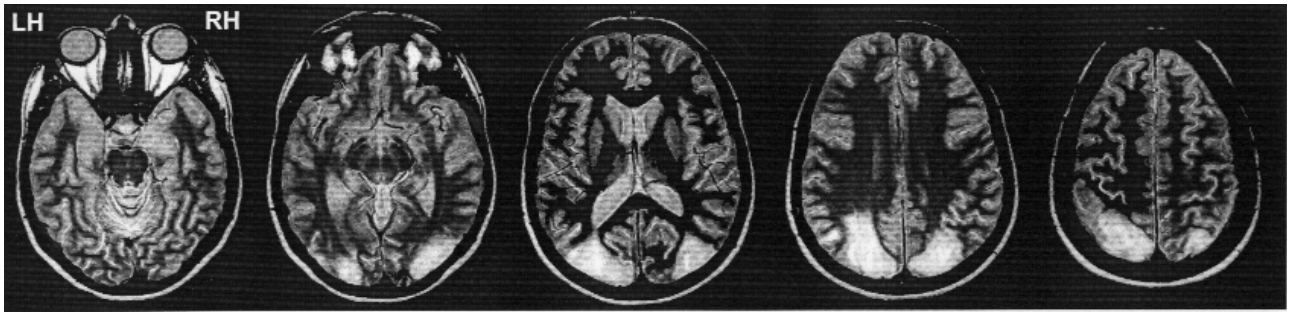

Fig. 5. The lesion of patient I.G. T2-weighted horizontal magnetic resonance imaging sections demonstrating a fairly symmetrical ischemic lesion of the posterior parietal and upper and lateral occipital cortico–subcortical regions. LH, left hemisphere; RH, right hemisphere.

face that lies between elementary stimulus analysis and motor output generation[17,36,37]. The normal stop responses produced by I.G. also confirmed that "neither part of the parietal lobe seems to be important for the non-spatial transformations of arbitrarily response selection associated with nonspatial cues in the conditional motor task"[37].

Tested in central vision, I.G. remained able to point normally to stationary targets; her automatic visuomotor guidance was specifically altered. The limited deficit shown by optic ataxia patients (including I.G.) when they are allowed to fixate on the stationary goal to reach or grasp contrasts sharply with the severe deficit observed here when I.G. had to respond to perturbations. By revealing a profound inability to make on-line corrections, we may have provided the first demonstration of a drastic visuomotor deficit in central vision in optic ataxia. If such a pattern of results is replicated in other cases, it would be tempting to reinterpret the classical deficits in optic ataxic patients. One may hypothesize that optic ataxia results from a specific deficit in on-line visuomotor control rather than a general deficit in visuomotor functions. Three arguments support this hypothesis. First, if the PPC is usually conceived as a key structure of the visuomotor system[16,27,29,35], then why do patients with optic ataxia exhibit visuomotor deficits that are mainly restricted to peripheral vision[23–26]? This incongruity suggests that the basic impairment responsible for optic ataxia needs to be identified more precisely[38]. An impairment of on-line motor control may explain the pattern of deficits in peripheral and central vision observed in I.G. and other optic ataxia patients. Indeed, when motor programming is based on foveal visual information, on-line visuomotor guidance participates in goal-directed actions only for minor final adjustments. Conversely, when an action is programmed based on imprecise peripheral visual information, visuomotor on-line control seems essential to adjust the end of the action to succeed in reaching or grasping the goal[5,39]. Target jumps similarly engage important corrective control. Such on-line processes have even been described for pointing to stationary targets in visual open loop conditions[5,8,40]. Second, the abnormally long deceleration period and large grip aperture described in optic ataxia patients[24,41] have been interpreted as a programming deficit. However, they could be reinterpreted as a deficit of on-line control processes, which are applied toward the end of the movement. This interpretation also fits with the finding[39] that transcranial magnetic stimulation of the posterior parietal lobe disrupts movement corrections made in response to a target jump but affects pointing to stationary targets only slightly. That work demonstrates that PPC is necessary for on-line motor control; our study further suggested that it is crucial only for automatic corrections but neither for movement programming nor for intentional motor control. Third, the increase in hand movement latency observed after a lesion of area 7 in monkeys[42] or in man[23,24,41] may also be explained by the need to refine the programming of movements to compensate for the on-line control deficit. This is consistent with evidence that a patient with a bilateral PPC lesion is more accurate in delayed pointing than in immediate pointing to peripheral targets[26].

Our protocol allowed us to distinguish between fast and slow corrections. As the posterior parietal lesion of I.G. differentially affected these two types of corrections, they could be considered as two distinct corrective processes. On one hand, the PPC seemed to be the key structure responsible for fast automatic corrections. On the other hand, PPC integrity was not crucial for making slow corrections or for programming movements toward stationary targets in central vision. As these last two processes remained intact in I.G., they may share common mechanisms. Slow corrective movements may result from reprogramming processes. Accordingly, two possible anatomical substrates could be proposed to account for the slower corrections among the distributed visuomotor network[27,43]. First, the human inferoparietal cortex is involved in movement preparation[44,45]. Second, the ventral stream (the inferotemporal cortex) has been proposed as an alternative structure involved in motor control when the PPC is damaged or when the action is delayed[26,29,35,46].

In the intact brain, there is a gradient among responses, ranging from perceptual responses (attributed to the ventral stream) to purely visuomotor responses (attributed to the dorsal stream)[27,38]. Although the PPC may participate in many visuospatial and visuomotor functions, its integrity may be crucial only for fast on-line motor control. Motor programming may involve other brain structures[44] and can be influenced by the intrinsic object properties processed in the ventral stream[35,46–48]. Unlike the programming of reaches, automatic corrections use only metric stimulus properties and do not require target selection. Accordingly, the color switch in our color-go condition could be used to program slow corrections but was unable to trigger the hand's 'automatic pilot'. The PPC alone is implicated in fast corrective processes because the high time constraints inherent to on-line control do not allow other visual streams to participate in motor output[27,38]. Our results therefore suggest that automatic corrections may be the most specific feature of the dorsal pathway.

Methods

Experimental setting. Stimuli were generated by software developed for a Visual Stimulus Generator board (VSG®, Cambridge Research System™; Rochester, UK) and presented on a monitor (EIZO E120™ T662; 160 Hz, 20 inches). The video monitor was mounted with a 20-inch touch frame using acoustic wave technology (Intellitouch®; Elotouch Systems™, Fremont, California). The screen was placed 30 cm from subjects' eyes; the head was fixed by a front- and chin-rest in experiments 1 and 2. A blue spot at the center of the bottom of the screen represented the starting point for vertical pointing movements. The targets were located on a radius of 22.5 cm above it, at the center of the top of the screen. Targets were small dots (2.5 mm in diameter and of 40% higher luminance than the black screen) that could appear in two positions equidistant from the starting point[49]. Targets were either red or green (isoluminant colors). The visual angle between the two target positions used in experiments 1 and 2 was 3.5 degrees. For the patient and the three control subjects of the same age (experiment 3), the distance between targets had to be slightly increased (to 6.7 degrees of visual angle), because of the patient's higher pointing variability.

Experimental procedure[49]. In experiments 1 and 2, four separate groups of six naive subjects performed the location-go, location-stop, color-go or color-stop tasks, combining two types of stimulus (target jump or color switch) and two types of instruction (correction or interruption). Each participant completed four experimental sessions (of 200 trials each) with 200, 250, 300 and 350 ms constraint of maximal movement time. A warning sound was provided if the movement time exceeded this temporal constraint, so that the subject spontaneously paced the pointing movements within the requested temporal window. Measurement began when the right fingertip left the touch screen and lasted until it hit the screen again or until the time-acquisition limit for each session was expired. Movement time and final position (x and y) were recorded for all pointing responses made within an additional extension (200 ms) of the session time limit, and were sampled to estimate the movement time subjects needed to react to stimulus perturbations according to different instructions (correction or stop). Session order was counterbalanced across participants. The different speed constraints elicited movements with a wide range of durations. A total of 4,800 pointing trials was com-

pleted by each group in experiment 1 and 2, with movement times ranging from about 100 ms to 500 ms. I.G. made slower movements, so the additional recording time extension was increased to 500 ms in experiment 3. In this experiment, three control subjects and the patient completed the same four combinations of tasks. In addition, only short sessions (100 trials) with 300 ms movement-time constraint were used and repeated twice, to include the automaticity window without tiring the patient unduly (a total of 800 pointing trials was completed by each control subject and the patient).

Data analysis. The target 1 class corresponded to pointing responses made inside the accuracy confidence interval of the initial target, calculated for the unperturbed trials at the same location and in the slowest session. For perturbed trials, pointing responses would be expected to occur in this class when no motor response was given to perturbation (that is, neither correction nor interruption of the movement). For unperturbed trials, pointing responses should occur mainly in this class, assuming they were performed with normal precision.

With the correction instruction, subjects were required to point directly to the second location of the target and to ignore its first location. Reaches made outside the accuracy confidence interval obtained for unperturbed targets were considered as target 2 responses. When pointing responses lay on both sides of this confidence interval, the number of reaches corrected toward target 2 was calculated as the number of outliers obtained between the two targets minus the number of outliers external to the two targets. For perturbed trials, reaches occurring near target 2 could result from a trajectory correction of the initial programmed movement toward the new target location. For unperturbed trials, they reflected the motor variability inherent to fast movements (that is, speed–accuracy trade-off).

With the stop instruction, subjects were required to immediately interrupt their ongoing movement and not to touch the screen. A third class of responses consisted of 'untouched trials', either complying with the stop instruction or touches occurring after the recording time defined for each session.

Statistical analysis. The percentage of touches to target 2 observed in unperturbed trials was used as a baseline to test for a significant occurrence of corrections made in response to the perturbations.

Medical case report (I.G.). This 29-year-old woman presented with severe headache, followed by dysarthria and bilateral blindness that lasted for three days. After this episode, she initially complained of being unable to see more than one item at the same time, to evaluate distances when she attempted to grasp an object or to walk on uneven ground. A diagnosis of ischemic stroke related to acute vasospastic angiopathy in the posterior cerebral arteries was established with an angiogram. Magnetic resonance imaging revealed a hyperintense signal on T2 sequencing that was fairly symmetrically located in the posterior parietal and upper and lateral occipital cortico–subcortical regions (Fig. 5). Reconstruction of the lesion[50] indicated that it involved mainly Brodmann's areas 19, 18 and 7, a limited part of area 39 and the intraparietal sulcus.

She was attentive and fully cooperative on examination two months after the onset. Her behavior in everyday life was suggestive of simultagnosia, but she showed no extinction when tested verbally with the stimulus set of all the present experiments, distinguishing the two targets in the color experiments, seeing all the jumps and being able to verbally report their direction in the location conditions. Her visual acuity was 7/10 for the right eye and 8/10 for the left eye. These results probably underestimated her genuine visual resolution, because of her low reading efficiency. Indeed, a contrast-sensitivity function showed only mild depression for intermediate spatial frequencies (ranging from 1.5 to 8 cycles per degree of visual angle) both for static and dynamic luminance gratings. Ocular fundi were normal. Visual fields showed a partial right inferior homonymous quadrantanopia with temporal crescent sparing. Pattern visual-evoked potentials generated for each eye and for each visual hemifield were normal. Recordings of saccades and smooth pursuit eye movements elicited by a light-emitting diode in the dark showed normal gain, direction and velocities. However, when she was asked to search for an object presented in the real world, she often had wandering exploratory eye movements for a few seconds before fixating on the target. She had no hemineglect syndrome during conventional testing but demonstrated bilateral optic ataxia. Reaching and grasping inaccuracy predominated for her right hand in her right peripheral hemifield. During reaching, hand posture was often inappropriate in terms of aperture and orientation, and she usually corrected her grip only through tactile feedback after she had contacted the object. However, visually elicited hand movements were generally accurate when performed in foveal vision.

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