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Frontal lesions impair the attentional control of movements during motor learning

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Abstract

This study examined the effects of frontal lobe lesions on the control of movements during motor learning. We compared the performance of patients with unilateral frontal or temporal excisions and controls in two-dimensional aiming movements during adaptation to a transformed visuomotor mapping. Subjects tried to reach a fixed target on a graphics tablet using indirect visual control from a monitor in either: (1) the standard visuomotor mapping, (2) a full inversion of motor space preserving the axis of movement, or (3) a mirror-like inversion of one axis of motor space. In the standard mapping, all groups showed precise and rapid aiming movements. In the full inversion condition, frontal lobe patients showed a stronger tendency than others to initiate movements in the natural direction (capture errors) during adaptation. In the mirror-like inversion, frontal patients showed deficits in both movement initiation and movement corrections. These control deficits disappeared with practice. These data provide evidence for a critical role of frontal cortex in the attentional control of unpracticed movements in man. © 1999 Elsevier Science Ltd. All rights reserved.

1. Introduction

The execution of simple visually guided movements is little affected by lesions to premotor or prefrontal cortex [16,17,47,55]. This contrasts with the significant effects of parietal lesions which often affect the precision of pointing, reaching and manipulation movements [3,15,26,42] This lack of effect of frontal lesions is also surprizing in light of the increased activity in these structures before and during simple visually guided movements as evidenced from single-cell recordings in monkeys [29,37] and functional imaging of regional activation in man [8,43,49].

> Frontal lesions can affect movement programming and selection in special conditions. For example, frontal lesions affect the programming and execution of sequential movements in monkeys and man [5,12,25,31,33–35,39,57]. Frontal lesions can also affect movements which require inhibiting prepotent automatic movements such as antisaccades [22] or spatially-inverted choice responses [11]. In monkeys, premotor lesions can impair indirect reaching movements around a transparent obstacle which involves inhibiting direct reaching movements [36]. Finally, frontal lesions produce deficits in the selection of movements associated with arbitrary cues, which can be interpreted as a deficit in movement selection in novel stimulus-response mappings [11,23,24,44,45].

A common characteristic of movements affected by frontal lesions is that their context or sensorimotor mapping is unfamiliar and that they thus require a more important contribution from voluntary control. Frontal cortex has long been implicated in the more voluntary or attentional aspects of actions as opposed to the more automatic or well-learned aspects [17,40,53]. However, few studies have tested

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this dissociation directly in visually-guided movements by comparing the performance of unpracticed and practiced movements. In novel unpracticed situations, performance depends a great deal on attentional control, while after acquisition attentional control is much less necessary as well-learned programs take over control of many portions of movements [28,46,51,56].

One reason that frontal lesions produce so few problems in visually guided movements may be that most of these movements are well practiced. This study examined whether frontal lesions affect unpracticed movements during motor learning in man. Some evidence points to a role of frontal cortex in unpracticed movements. For example, cerebral activation in man appears to increase in frontal areas during the acquisition of motor skills [14,19–21,32,41,50,52]. If frontal cortex activity is necessary for the attentional control of movements, frontal lesions should impair unpracticed movements in a motor learning situation.

Unpracticed movements can be observed during adaptation to new sensorimotor mappings and some studies have examined sensorimotor adaptation after frontal cortical damage. Movements during prism adaptation have shown mixed results, some showing no effects of frontal lesions [58]; others showing some adaptation deficits after frontal lesions but few initial performance problems [6]. Depending on the procedure used, prism adaptation may involve adaptation of several motor modalities including eye, head and or arm movements at different moments in the task and may therefore be too complex to directly address the question of attentional motor control problems.

Some studies have examined mirror-reversed movements. Some case studies have shown adaptation problems after frontal lesions and some have not [1,4,10]. We recently examined mirror tracing performance in patients with frontal excisions [7]. In this situation, frontal lobe patients were slower than temporal lobe patients as expected but also showed more frequent oscillatory movements, suggesting an impaired visuomotor control. However, mirror tracing provides a very coarse measure of sensorimotor control.

The present study was designed to provide a more direct test of the effect of human frontal damage on inverted movements. We compared the performance of patients with unilateral frontal lobe lesions to that of patients with temporal lobe lesions on simple visually guided aiming movements during adaptation to a transformed visuomotor mapping. If frontal lesions affect attentional motor control, patients with frontal lesions should show problems in inverted movements during adaptation but not after learning when the newly formed programs can take over control of the performance.

2. Method

2.1. Subjects

Eleven patients with unilateral frontal lobe excisions (7 right, 4 left) were compared to 11 patients with unilateral anterior temporal lobe excisions (7 right, 4 left) and to 11 control subjects with no history of neurological injury. The three groups were matched for age [$M = 41$ years] and education level [$M = 12$ years]. Six frontal resections and 10 temporal resections were performed to alleviate a drug-resistant epilepsy. Six subjects underwent frontal or temporal tumor resections, including two meningioma, one glioblastoma, and two astrocytoma in frontal lobes and one temporal meningioma. All patients were tested at least one year following surgery. Informed consent to participate in the study was obtained according to the rules of the hospital.

The frontal lesions are shown in Fig. 1 and were derived from the surgeon's drawings in eight cases and from MRI images in three cases. All frontal patients showed some damage to dorsal prefrontal cortex, three showed ventral prefrontal damage, eight patients showed lateral premotor cortex damage, nine patients also showed anterior cingulate damage; and five showed medial premotor damage. In temporal excisions, the anterior portion of the lobe (5–6 cm) was removed including the hippocampus but preserving Heschl's gyri. All epilepsy patients showed a marked reduction in seizure frequency after surgery (two frontal patients and five temporal patients were seizure-free). Ten frontal patients as well as seven temporal patients were on anticonvulsant medication. Two patients had received radiation as adjuvant treatment (5000 rads in 6 weeks).

None of the patients exhibited gross sensory or motor impairments in a standard neurological examination. Neuropsychological evaluations revealed no deficit in language comprehension or production nor in episodic memory. Also, none of the patients showed problems in rapid tapping, discrimination of line orientation, nor any gross visuospatial deficit including neglect (line bisection and letter search), ideomotor apraxia (symbolic gestures and simulation of object use), and optic ataxia (reaching objects of different widths and orientations). Frontal lobe patients were however significantly poorer than temporal lobe patients in verbal fluency (animals: $P = 0.02$; letter: $P = 0.10$), a timed attentional search task ($P = 0.01$), the Stroop task ($P < 0.05$), Luria's graphic series ($P = 0.04$) and sequential hand gestures ($P = 0.02$).

2.2. Materials and stimuli

Movements were performed on a graphics tablet

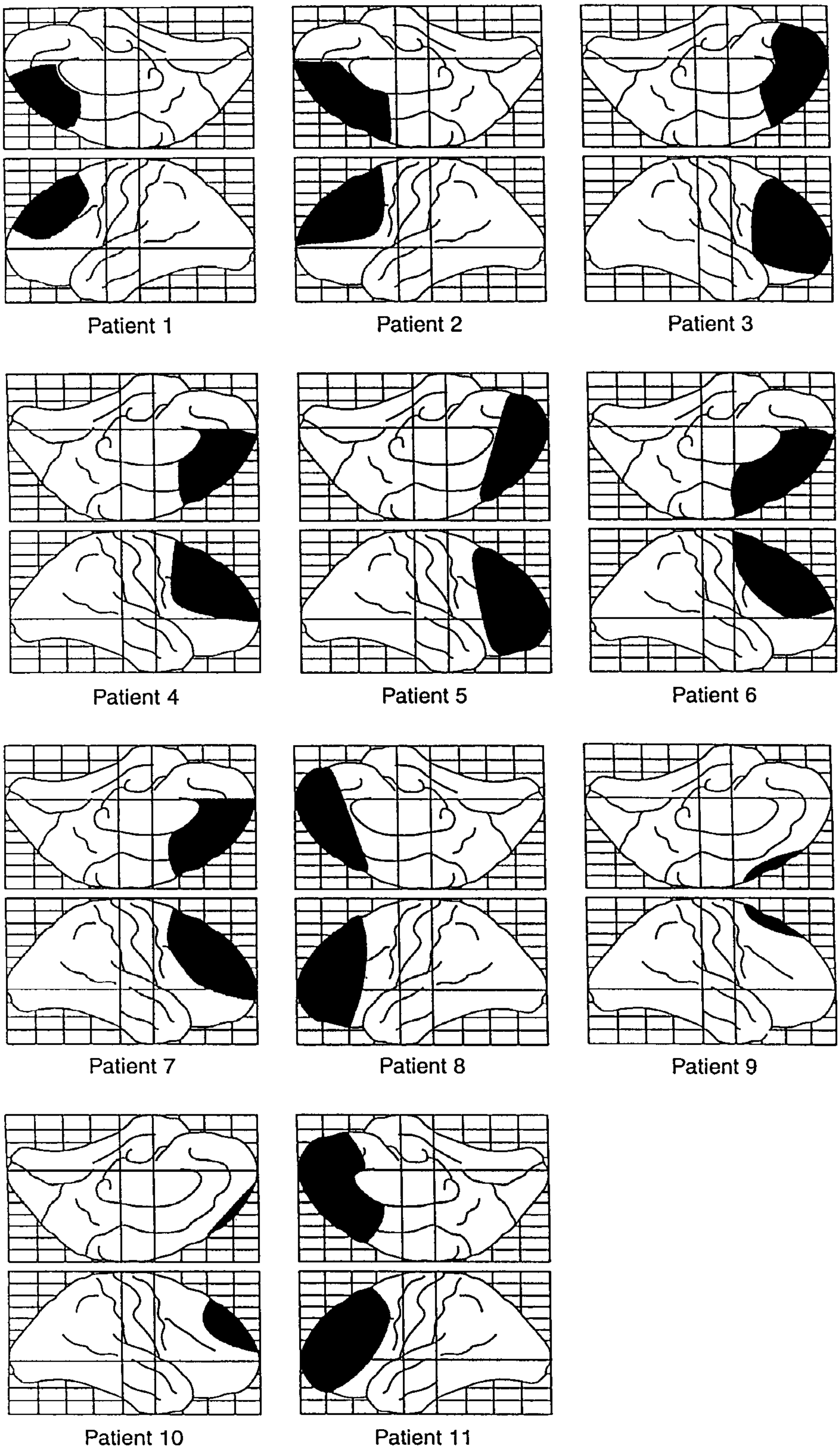

Fig. 1. Schematic drawings of the extent of the frontal resections on lateral and medial views of the Talairach grid.

(30 × 30 cm) which was placed under an occluding screen to hide the hand from view. The position of the stylus on the tablet was sampled at a rate of 12 Hz. Stimuli appeared on a computer monitor 1.0 m in front of the subject. The cursor linked to the movement of the stylus was a white spot 1 cm in diameter. The targets of movements were white circles 2 cm in diameter.

2.3. Procedure

The aiming task was adapted from Cunningham [9]. Subjects had to move the cursor on the screen from a central starting point to a fixed target appearing in the periphery. Targets appeared in one of four peripheral positions, 40° above or below the horizontal axis on the right or left of midline. Subjects were tested in three movement conditions. In the *baseline condition*, there was no transformation of visuomotor space except the usual transformation of the horizontal tablet surface to the vertical monitor surface. In this condition, aiming movements are executed rapidly and in a straight line [9]. In the *full inversion condition*, both horizontal and vertical directions of the cursor were inverted in relation to stylus movement. To reach the target on the screen, the subject had to move the stylus in the opposite direction on the tablet (e.g. lower-left instead of upper-right). This full inversion of movement direction requires only a brief adaptation from the natural mapping because it preserves the axis of movement and thus requires the same motor coordination pattern as natural movements [9,30]. However, this condition should induce capture errors in the initiation of the movement since it requires selecting an initial direction opposite to the natural one.

The critical condition was the *single-axis inversion condition*. In this condition, the motion of the cursor on the screen was inverted in the horizontal axis but not in the vertical axis. This condition is similar to mirror inversion in which only the vertical axis of movement is inverted compared to the standard mapping. Single-axis inversion produces significant movement errors during several trials before adaptation takes place [9]. The peripheral target remained present for the duration of the trial: 3.0 s in the baseline condition, 6.0 s in the full inversion condition and 11.0 s in the single-axis inversion condition. At the end of each trial, the experimenter returned the subject's hand at the center of the tablet.

Subjects were tested in a fixed sequence of conditions: eight trials were used to familiarize the subject with the apparatus followed by 24 trials in the baseline condition, 48 trials in the full inversion condition, 16 more trials in the baseline condition aimed at removing any aftereffect of the full inversion, and 64 trials in the single-axis inversion condition. The fixed sequence of conditions was used so that the full inversion condition would help familiarize subjects with adaptation to new mappings. Each consecutive sequence of four movements contained one trial for each target position in a pseudo-random order and there were no repetitions of target positions. Performance was analyzed across target positions by using the median of each group of four consecutive trials (trial quadruplets). Adjacent conditions were separated by pauses of about 3 min. Movement parameters examined included the initial direction of movements, movement precision measured by the total length of the movement, and average movement speed.

3. Results

3.1. Initial direction of movement

The initial direction of aiming movements was quantified by the direction of the third sampling point of the movement. This point was chosen because it occurred 240 ms into the movement before any feedback-based correction of the movement is possible. Initial directions were highly variable both across trials and across subjects and they were thus analyzed using three categories: (1) adequate initiations, defined as initial directions within 30° of the perfect direction; (2) capture errors, defined as initial directions within 30° of the direction of the visually presented target; and (3) other erroneous directions. The 30° criterion was used because all control subjects showed initial directions within this interval after adaptation in all conditions.

In the baseline condition, all subjects showed an adequate initiation in all 24 trials. In the full inversion condition, capture errors were observed mostly in the first trials. Capture errors were averaged for trial quadruplets 2–4 so as not to include initial directions that were not based on previous exposure to the transformed space (quadruplet 1) and because of the very low frequency of these errors after trial quadruplet 4. The frontal group produced significantly more frequent capture errors than the other two groups in the full inversion condition (frontals vs temporals: Mann–Whitney $U = 30.0$, $P < 0.02$; temporals vs controls: $U = 49.5$, n.s.). No other types of errors were observed in this condition.

In the single-axis inversion condition, both capture errors and other directional errors were observed (see Table 1). These two types of errors were averaged over quadruplets 2–8 because of their very low frequency after quadruplet 8. The analyses indicated that the frontal group made significantly more capture errors than other groups (F vs T: $U = 32.5$, $P < .03$, T vs C:

Table 1
Mean error rates (%) in the initial direction of aiming movements in the full inversion condition and in the single-axis inversion condition in patients with frontal lesions, patients with temporal lesions and controls

	Frontal	Temporal	Control
Full inversion			
Capture errors	25.0	8.3	8.3
Single-axis inversion			
Capture errors	25.0	10.7	3.6
Other errors	39.3	28.6	14.3

U = 37.5, n.s.) but not more of other types of errors (F vs T: U = 42.0, n.s.; T vs C: U = 50.0, n.s.).

3.2. Movement precision

The precision of aiming movements was measured using the total length of the movement. Fig. 2 shows group averages of movement length for consecutive trial quadruplets in the three conditions. In the baseline condition, movements were very close to a straight line in all groups. An ANOVA on the first and last trial quadruplets showed no significant group differences [F(2,30) = 1.8, n.s.], no significant effect of practice [F(1,30) < 1.0, n.s.], and no interaction [F(2,30) < 1.0, n.s.].

In the full inversion condition, an ANOVA on the first and last trial quadruplets showed a significant effect of practice [F(1,30) = 7.7, P < 0.01], but no significant group effect [F(2,30) = 1.6, n.s.], and no significant group-by-trial interaction [F(2,30) = 1.6, n.s.]. To quantify the aftereffect of the full inversion condition, an ANOVA was performed on the first and last trials of baseline performance which followed the full-inversion condition. In this analysis, there was a significant effect of practice [F(1,30) = 3.4, P = 0.02], indicating that a measurable after effect was produced by the full-inversion condition, but there was no group effect [F(2,30) < 1.0, n.s.] and no interaction [F(2,30) = 1.4, n.s.].

In the single-axis inversion condition, frontal lobe patients appear to show abnormal movement lengths in the early phase of adaptation but not after practice. An ANOVA was performed on the first, middle, and last trial quadruplets to determine whether the performance of the groups became similar early or late in adaptation. The analysis showed that there was a significant group effect [F(2,30) = 5.6, P < 0.01; F vs T: t(20) = 2.9, P < 0.01; T vs C: t(20) = 0.01, n.s.] and that all groups

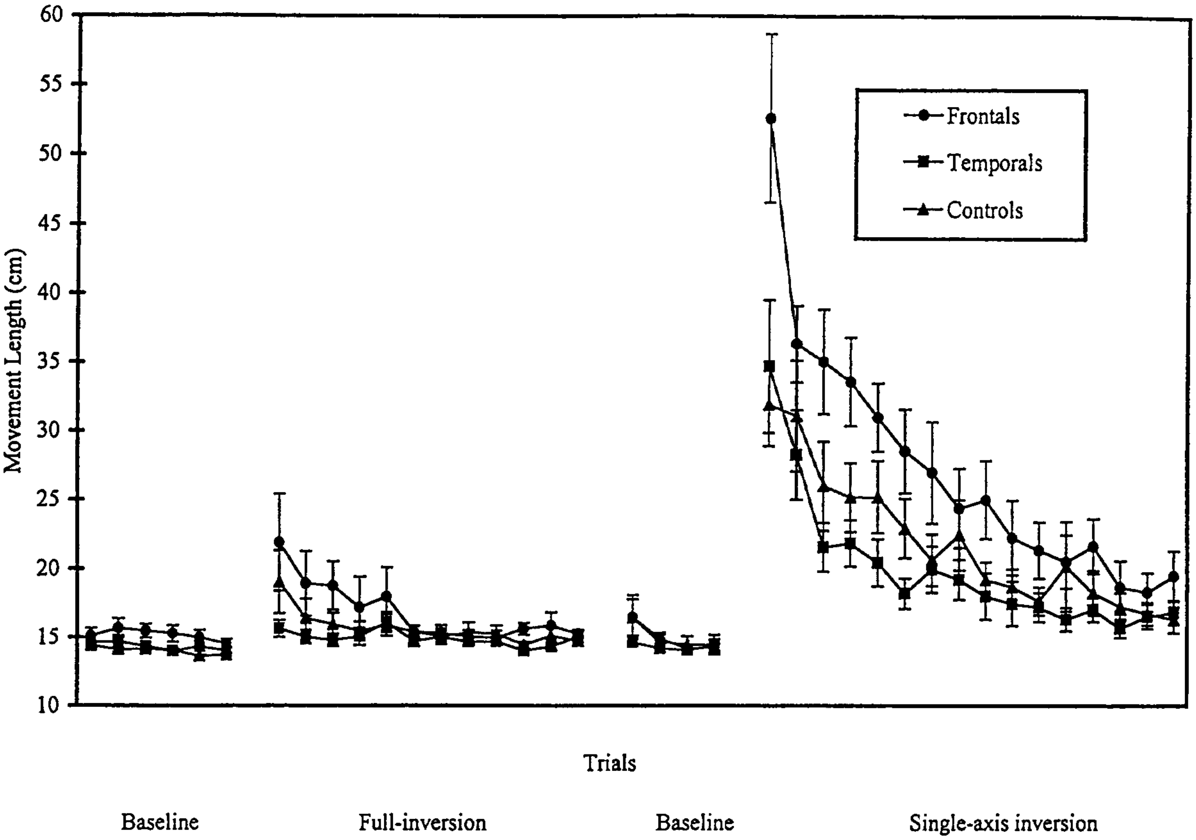

Fig. 2. Average length of aiming movements as a function of practice in the normal visuomotor space, in a full inversion of visual feedback, and in a mirror-like inversion of feedback.

improved with practice [$F(2,60) = 48.4$, $P < 0.0001$]. There was also a significant Group-by-Trial interaction [$F(4,60) = 3.6$, $P = 0.01$] and simple effects showed that the performance of the frontal lobe patients had reached a normal level after the first half of practice [F vs T: $t(20) = 1.5$, n.s.; T vs C: $t(20) = 1.0$, n.s.].

3.3. Movement speed

Fig. 3 shows the measures of average movement speed obtained in the three conditions of the task. In the baseline condition, movement speed was very similar in the three groups. An ANOVA on the first and last trial quadruplet showed that movement speed increased with practice [$F(1,30) = 9.0$, $P < 0.005$], but there was no significant group effect [$F(2,30) < 1.0$, n.s.] and no significant Group-by-Trial interaction [$F(2,30) < 1.0$, n.s.]. In the full inversion condition, the same pattern of results was obtained: a significant practice effect [$F(2,30) = 34.1$, $P < 0.0001$], but no significant group effect [$F(2,30) = 1.0$, n.s.] and no significant interaction [$F(2,30) < 1.0$, n.s.].

In the single-axis inversion condition, average movement speed showed a relatively high variability throughout acquisition in all three groups but the temporal and control groups appeared to produce faster movements with practice. An ANOVA on the first, middle, and last trial quadruplets showed a significant practice effect [$F(2,60) = 16.6$, $P < 0.0001$], no significant group effect [$F(2,30) = 1.1$, n.s.], and a significant interaction [$F(4,60) = 4.4$, $P < 0.005$]. Analyses of the simple effects suggest that both the temporal group and the control group showed a significant improvement of speed with practice [Temporal: $t(10) = 4.2$, $P < 0.0002$; Control: $t(10) = 4.6$, $P < 0.0001$] but that the frontal group did not [$t(10) = 0.02$, n.s.].

We examined the individual performances of frontal lobe patients and found no differences between patients with right-sided lesions and patients with left-sided lesions nor between patients with sparing of premotor regions or medial frontal regions and other patients on any measure.

4. Discussion

Frontal lesions did not affect baseline aiming movements, confirming that patients with frontal lobe

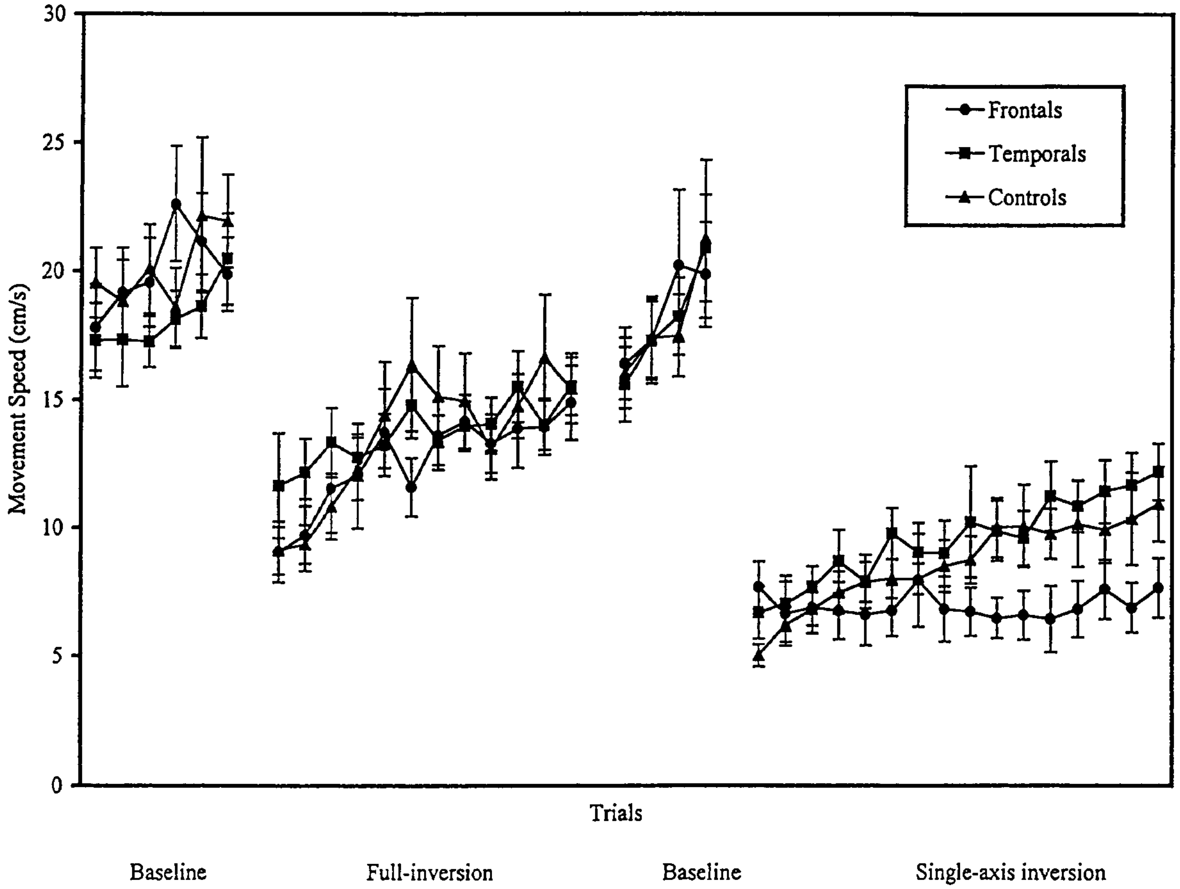

Fig. 3. Average speed of aiming movements as a function of practice in the normal visuomotor space, in a full inversion of visual feedback and in a mirror-like inversion of feedback.

lesions can produce simple visually guided two-dimensional movements toward these targets with little difficulty. Of course, results may have been different in a task requiring high movement speed, high precision (small targets) or frequent direction changes, all variables which increase the contribution of attentional control to movement performance. However, the good baseline performance of frontal lobe patients helps differentiate the effects obtained here from the aiming and reaching difficulties of optic ataxia observed after parietal lobe lesions [42].

The full inversion condition required an inversion of the general direction of movements while preserving the motor coordination pattern of natural movements. In this condition, all groups showed a rapid adaptation, as expected from a previous study [9]. Frontal patients produced more capture errors than others in this condition, indicating that they had more problems selecting the initial direction of movement. This deficit suggests that inverted arm movements can elicit movement selection errors. This result parallels the deficits previously observed in the execution of anti-saccades or inverted choice responses [11,22,35]. The initial direction errors in frontal lobe patients contributed to some degree to an increase in movement length in frontal patients since capture errors had to be corrected to complete the movements. However, movement lengths were only slightly increased in frontals compared to others. This is probably due to the fact that movements in full inversion preserve the axis or motor coordination pattern of natural movements. These movements thus require well-practiced movements once the initial movement selection problem is solved.

The single-axis inversion of motor space produced an adaptation phase lasting several trials. In this condition, frontal lobe patients showed problems in specifying both the initial direction of movement and the corrections during the movement. This deficit suggests that frontal lesions can affect the execution of simple visually guided movements when they involve a novel sensorimotor mapping. The motor control problems observed disappeared within 32 trials of practice, indicating that the deficit is restricted to the control of unpracticed movements. Unpracticed movements are generally thought to require a mode of control that is distinct from well learned movements which is often referred to as attentional control [38,46,54,56]. The attentional control mode is thought to be a relatively slow but flexible control system which is dependent on error feedback to select movements. In contrast, the control of practiced movements relies more on faster and more precise processes which use the acquired associations or program relevant to the task for the selection of movements, relying less on feedback except at certain critical moments. Our results suggest that these two modes of motor control are at least partly dissociated in the brain and that frontal cortex is critical for the attentional control of movements and not for the programmed control of movements.

Frontal lesions have often been shown to produce attention problems [48]. There is also ample evidence that frontal lesions affect novel tasks preferentially [17,55] and some of these deficits have been labelled problems of attention to action [38,41]. However, few controlled studies have directly compared similar actions performed under attentional and non-attentional control. Our data suggest that at least some of the novel performance deficits after frontal lesions, could be due to a role of frontal cortex on attentional motor control required in unpracticed situations.

In monkeys, premotor cortex lesions produce well-documented problems in movement selection in arbitrary S-R mappings [40]. Premotor lesions produce few problems in visually guided movements despite the fact that these movements produce significant activations in premotor cortex [8,40,47,49]. The present data suggest that visually guided movements show deficits after frontal lesions only when they are performed in novel visuomotor mappings.

The deficit observed here shows many similarities with that shown in monkeys with premotor lesions when trying to reach objects on the other side of a transparent obstacle [36]. In both tasks, the arm movement had to be adapted to a novel visual context and an habitual movement had to be inhibited. Both the indirect reaching deficit in monkey and the unpractised movement deficit in man suggest that frontal lesions make the attentional motor control system too inefficient to override the natural visuomotor mapping before the new mapping is learned. The deficit appears to be linked to the relative strengths of competing inhabitual and habitual movements. Moll and Kuypers [36] suggested that the key factor in the deficit was the poor inhibition of the natural movement. Both the inhibition of inappropriate movements and the facilitation of the correct movement are part of attentional control, but Moll and Kuypers suggest that a deficit should not be observable when no response inhibition is required. Future work should thus examine if frontal patients have problems with some portions of natural visually guided movements which do not require inhibition of habitual movements but do require attentional control, such as the termination of fast movements to small targets or unpredictable direction changes [28]. Our hypothesis is that frontal lesions will affect portions of movements in which attentional control is required whether strong prepotent habitual movements have to be inhibited or not.

4.1. Visuomotor learning

The effect of practice on the performance of frontal lobe patients indicates that unilateral premotor and prefrontal lesions do not prevent visuomotor adaptation to new spatial mappings. It may be that the control problem produced by unilateral lesions is not sufficient to significantly affect acquisition rate. This will have to be verified with bilateral frontal lesions. Motor learning problems have been reported after damage to striatal structures which have strong connections to frontal cortex. Patients with Huntington's disease or Parkinson's disease can show problems in visuomotor acquisition as well as visuomotor control [2,13,18,27,59,]. However, few studies have compared the effects of frontal and striatal dysfunction in the same motor learning situation. Also, it is still unclear whether there is a correlation between the control and acquisition problems.

The absence of acquisition problems in the frontal lobe group may also be linked to the type of task used. Our task produced a relatively rapid acquisition and other tasks which produce more control problems and require more practice may show different results. The present data suggest that the systems responsible for acquiring new visuomotor mappings can function despite some damage to the attentional motor control system. This is consistent with the suggestion that the system involved in acquisition is at least partly dissociable from that involved in attentional motor control. There is evidence for increases in blood flow in frontal as well as posterior cortical and subcortical areas during acquisition of motor skills [14,19–21,32,41,52,50,]. This suggests that frontal cortex may be part of a distributed system involved in sensorimotor learning without being critical for acquisition.

Frontal lobe patients were not significantly slower than others in natural aiming movements or in the first trials of adaptation. However, they did not show the normal increase in movement speed during adaptation to the transformed mapping. Dorsal frontal lesions often produce a slowing in many complex actions and this effect may be another instance of this general slowing or bradykinesia [17]. This slowing is poorly understood. In the present task, it may indicate that some level of attentional control is required even after 64 trials. This will have to be further investigated. Nevertheless, the rate of improvement in precision in the frontal group indicates that unilateral frontal lesions do not critically affect sensorimotor acquisition in this task.

Our data cannot address the question of which portions of premotor and/or prefrontal cortex are more critical for attentional motor control in man. In monkeys, indirect reaching movements are affected after premotor lesions [36]. In functional brain imaging in man, there is evidence that prefrontal and cingulate regions may be more active during early stages of motor learning than after learning [14,19,41,43,50]. Task differences may exist as well. More work combining different methodological approaches on the same task will be necessary to delineate the various regions involved in attentional motor control and in motor learning.

In conclusion, frontal lobe lesions affect visually guided movements during adaptation to a transformed visuomotor mapping and this problem disappears with practice. These data provide direct support for the suggestion that frontal structures are necessary for the attentional control of unpracticed movements but not for the programmed control of well learned movements.

Acknowledgements

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