

## Direct Evidence for Cortical Suppression of Somatosensory Afferents during Visuomotor Adaptation

Pierre-Michel Bernier, Borís Burle, Franck Vidal, Thierry Hasbroucq and Jean Blouin

Laboratoire de Neurobiologie de la Cognition, Aix-Marseille Université, CNRS, 13331 Marseille, France

**Upon exposure to novel visuomotor relationships, the information carried by visual and proprioceptive signals becomes discrepant, often disrupting motor execution. It has been shown that degradation of the proprioceptive sense (arising either from disease or experimental manipulation) enhances performance when drawing with mirror-reversed vision. Given that the central nervous system can exert a dynamic control over the transmission of afferent signals, reducing proprioceptive inflow to cortical areas could be part of the normal adaptive mechanisms deployed in healthy humans upon exposure to novel visuomotor environments. Here we address this issue by probing the transmission of somatosensory afferents throughout the course of adaptation to a visuomotor conflict, by recording median nerve somatosensory evoked potentials. We show that early exposure to tracing with mirror-reversed vision is accompanied by substantial proprioceptive suppression occurring in the primary somatosensory cortex (S1). This proprioceptive gating is gradually alleviated as performance increases with adaptation, returning to baseline levels. Peripheral and spinal evoked potentials were not modulated throughout, suggesting that the gating acted to reduce cortico-cortico excitability directly within S1. These modulations provide neurophysiological evidence for flexibility in sensory integration during visuomotor adaptation, which may functionally serve to reduce the sensory conflict until the visuo-proprioceptive mapping is updated.**

**Keywords:** electroencephalography, proprioception, sensory conflict, vision, visuomotor adaptation

### Introduction

Under normal visual conditions, the visual estimate of limb position is congruent with the proprioceptive estimate, allowing for smooth and efficient interactions with the surroundings. Nevertheless, instances arise in which the mapping between these sensory modalities is altered, such as when using a microscope or magnifying lenses. Under such visuomotor environments, the information carried by visual and proprioceptive signals becomes incongruent, often disrupting motor execution (Harris 1963; Balslev et al. 2004; Redding et al. 2005; Bernier et al. 2007). It has been shown that humans can adapt to these discrepancies and rapidly regain near baseline levels of performance through the gradual updating of the mapping between the motor commands and their sensory consequences (Miall and Wolpert 1996; Krakauer et al. 2000; Tong and Flanagan 2003; Tseng et al. 2007). Nevertheless, despite abundant research on visuomotor adaptation, the processes by which the brain integrates unaligned visual and proprioceptive signals during visuomotor adaptation remain poorly understood.

Behavioral experiments have provided a paradoxical framework for multisensory integration upon exposure to novel visuomotor environments: degrading proprioceptive sensitivity would facilitate motor performance. For instance, being deprived of proprioception, deafferented patients are less impaired than healthy individuals when tracing with mirror-reversed visual feedback (Lajoie et al. 1992). Similarly, reducing proprioceptive acuity through repetitive transcranial magnetic stimulation over the primary somatosensory cortex enhances movement execution under conflicting visuomotor environments (Balslev et al. 2004). These facilitative effects on performance have been attributed to a reduction in interference between incongruent visual and proprioceptive signals, which in turn would optimize visual guidance of the movement.

In light of these findings, the goal of the present study was to determine whether the normal adaptive behavior in healthy humans is accompanied by a reduction in the transmission of somatosensory signals along the ascending sensory pathways. Transmission of afferent input has classically been examined by recording somatosensory evoked potentials (SEPs) arising from peripheral nerve stimulation. It was shown that the transmission of afferent signals is reduced both before and during active movement (Prochazka 1989; Brooke et al. 1997). This movement-induced gating would arise both from increased peripheral receptor discharge as well as from efferent signals associated with the motor commands (Chéron et al. 2000). Interestingly, in addition to this movement-related attenuation, the central nervous system (CNS) could also selectively suppress or facilitate the transmission of somatosensory signals as a function of task demands (Legon and Staines 2006). For instance, in contexts requiring high proprioceptive vigilance, the gating is partially alleviated to optimize performance (Staines et al. 1997). Such task-specific modulations raise the possibility that upon exposure to a novel visuomotor relationship, the CNS may seek to reduce the visuo-proprioceptive conflict by further suppressing somatosensory transmission to the cerebral cortex. Preliminary findings by Jones et al. (2001) have suggested that proprioceptive attenuation may intervene at the peripheral level. The authors found that the gradual increase in performance witnessed upon prolonged exposure to a conflicting visuomotor environment is associated with a depression in muscle spindle activity, attributing these changes to a lower fusimotor drive.

Here we test the hypothesis that along with the attenuation at the peripheral level, visuomotor adaptation is accompanied by a suppression of somatosensory input at the cortical level. This was done by recording peripheral, spinal and cortical median nerve SEPs throughout a period of adaptation to tracing with mirror-reversed vision. We hypothesized that if reducing

somatosensory input to cortical areas is part of the normal adaptive process, then we should observe an attenuation of the early SEPs at S1 during the initial stages of exposure to mirror-reversed vision, that is when the visual and proprioceptive cues can be seen as most discrepant.

## Main Experiment

### Materials and Methods

#### Subjects

Eleven healthy right-handed volunteers (6 males, 5 females, age 21–35 years, mean 27) with normal vision took part in the experiment. Experiments were conducted with the understanding and written consent of each subject, in accordance with the ethical standards of Aix-Marseille Université as well as those set out in the 1964 Declaration of Helsinki.

#### Apparatus

Subjects were seated comfortably on a reclining chair, such that their back was supported. A digitizing tablet (SummaGrid IV, 120 × 80 cm, 125 Hz, resolution < 1 mm) was fixed on a table installed in front of them. Subjects held a digitizing pen with their right hand and adopted a natural drawing position, with the forearm and elbow resting on the tablet. They had to trace the contour of an asymmetrical polygon made up of 8 segments of different lengths (dimensions 25 mm × 17.5 mm; total perimeter 84.5 mm; width of segments 0.5 mm), which was positioned approximately 30 cm ahead of subjects and 15° to their right. The tracings involved motion at the proximal and intermediate phalanges, as well as slight motion at the wrist. In the mirror condition, a round mirror (diameter 25 cm) was positioned at eye-level, approximately 15 cm in front of the subjects and 15° to their left. The mirror was tilted in all 3 dimensions, such that the tracings could not simply be performed by inhibiting the natural response and initiating a response in the opposite direction, as would have been the case if the mirror was tilted in a single dimension. In the mirror trials, a shield prevented subjects from seeing their drawing limb directly.

#### Experimental Protocol

Subjects performed the mirror-reversed task by looking at the shape and their drawing hand through the tilted mirror (mirror trials). They also performed the task with direct vision (normal trials) as well as trials in which they remained motionless (static trials). The normal and static trials provided baseline measures of SEP amplitude under motion and static conditions, respectively. The normal trials were performed at 3 instructed velocities to control for the effect of movement velocity on SEP amplitude (5 s/segment [~2.4 mm/s], 10 s/segment [~1.2 mm/s], and 20 s/segment [~0.6 mm/s]). Throughout these trials, an auditory signal was delivered at every 5-, 10-, or 20-s interval (depending on the velocity condition) and subjects were told to smoothly adjust their pace so that the pen was at a segment junction when the signal occurred. In the mirror trials, there was no specific velocity requirement per se. However, subjects were instructed not to trace faster than in the fastest normal trials (5 s/segment). An experimenter verified that they complied with these velocity requirements. Subjects were also asked not to contract the hand and forearm muscles as a means

to “freeze” the degrees of freedom, but to stay as relaxed as in the normal trials. In the static trials, subjects held the pen as if about to trace, but without actually moving. This condition thus required a minimal level of background muscular activity to hold the pen in position. Subjects visually fixated their static hand during these trials.

The experiment comprised 20 trials, each 160 s in duration, with a rest period of 30 s between each trial. Subjects initially completed a preadaptation phase, which consisted of 1 static trial and 3 normal trials (1 at each of the 3 instructed velocities). The static trial was always done first, whereas the ordering of the 3 normal trials was pseudorandomized across subjects. Subjects then completed an adaptation phase, which consisted of 12 consecutive mirror trials. Finally, to control for possible effects of fatigue or other time-related effects, subjects completed a postadaptation phase. This phase was identical to the preadaptation phase and thus consisted of 1 static trial and 3 normal trials (1 at each of the 3 instructed velocities), presented in the same order as in the preadaptation phase. Subjects were allowed to move their arm with normal vision for 5 min between the adaptation phase and the postadaptation phase to recalibrate the visuomotor relationship to its pre-adaptive level and avoid after-effects. The pre- and postadaptation phases lasted approximately 12 min each, whereas the adaptation phase lasted approximately 40 minutes.

#### Stimulation and Recording Procedures

Stimulations were delivered throughout the trials at a rate of 2.5 Hz (i.e., 400 stimulations per trial) to the median nerve at the wrist of the drawing hand (cathode proximal) with surface Ag–AgCl electrodes (2 cm center-to-center interelectrode spacing). The stimuli consisted of square-wave pulses of 0.1-ms duration. The intensity was adjusted for each subject to 90% of resting motor threshold, which was determined as the lowest intensity which evoked a twitch in the flexor pollicis brevis (FPB) muscle. The resting motor threshold was determined with subjects holding the pen as if they were to draw but without actually moving (as in the static trials). This posture incurred slight electromyographic (EMG) activity, ensuring that the established resting motor threshold would be similar to the active motor threshold. Prior to the experiment, subjects traced the shape a few times (with direct vision) with the stimulation set at 90% resting motor threshold to get used to the electrical pulses. This gave us the chance to determine if the motor threshold would be reached in fully active conditions, and to adjust the intensity of the stimulation if necessary.

Electroencephalographic (EEG) activity was recorded continuously from 64 preamplified Ag–AgCl electrodes (ActiveTwo, BioSemi) embedded on an elastic cap in accordance with the extended 10/20 system. Recording electrodes were also placed on both Erb points (brachial plexus), on the fifth cervical (Cv5) and on the mastoid process contralateral to the drawing hand. Electrooculographic (EOG) activity was recorded bipolarly with surface electrodes placed near both outer canthi and under and above the left orbit. The EEG and EOG signals were digitized online (sampling rate 1024 Hz; DC 268 Hz; 3 dB/octave) and bandpass-filtered offline (1–100 Hz; 12 dB/octave).

To determine whether forearm and hand muscle activity changed across conditions, which could have affected SEP amplitude (Jones et al. 1989), we recorded EMG activity of the right FPB, first dorsal interosseous (FDI), flexor carpi radialis

(FCR), and extensor carpi radialis (ECR) muscles. Recordings were made with a Bortec AMT-8 system (Bortec Biomedical, Calgary, Canada) with bipolar surface preamplified electrodes (2 cm center-to-center interelectrode spacing). The EMG signals were bandpass-filtered (5–350 Hz, 6 dB/octave) and digitized online (sampling rate 1 kHz) using a Keithley A/D converter device (AD-win pro, Keithley Instruments, Cleveland, OH). The data were full-wave rectified and low-pass filtered with a second-order Butterworth filter with a 10-Hz cut-off frequency to generate an envelope. Such envelopes have been shown to provide a good assessment of the total energy in the signal (Brindle et al. 2006).

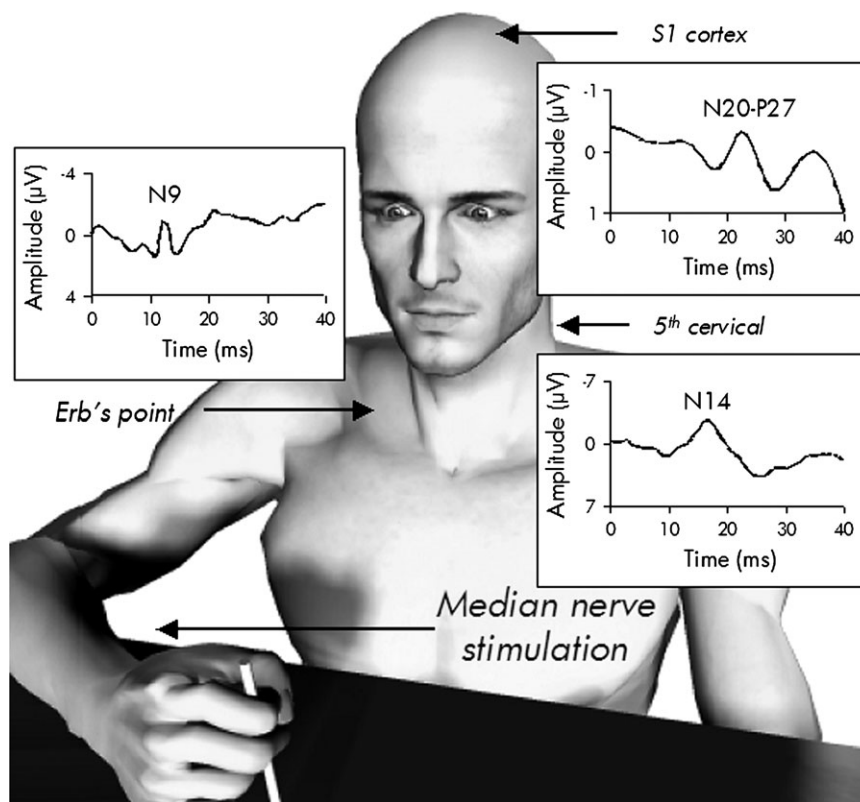
### Data Reduction

**Electrophysiological data.** Given the erratic nature of the tracings in the mirror trials and the need for sufficient data to maximize the signal-to-noise ratio of the SEP averages, the SEP traces were not averaged with respect to specific behavioral events. Rather, for each subject and each trial, SEPs were obtained by averaging all epochs time-locked to the stimulation (–100 to 250 ms), with the average amplitude of the 100-ms prestimulus epoch serving as baseline. Ocular artifacts were subtracted using the statistical method of Gratton et al. (1983) as implemented in the Brain Vision Analyzer software (Brain Products GmbH; Gilching, Germany). The monopolar recordings were visually inspected and epochs still presenting artifacts were rejected (the averages thus comprised 350–400 artifact-free traces).

The peripheral and spinal evoked potentials were recorded at Erb's point ipsilateral to the drawing hand (referenced to

contralateral Erb's point) and at Cv5 (referenced to Fpz), respectively, whereas the cortical SEPs were recorded at C3 and FCz (both referenced to Fpz) (Fig. 1). Subjects showed consistent SEP traces across all conditions, with peak latencies not differing significantly as a function of condition ( $P$  values > 0.3). The peaks of interest were the peripheral N9 (recorded at Erb's point; mean latency: 12 ms), the spinal N14 (recorded at Cv5; mean latency: 14 ms), the far-field potential P14 (recorded at C3; mean latency: 15 ms), the parietal N20 and P27 (recorded at C3; mean latencies: 19 and 25 ms, respectively), and the frontal P22 and N30 (recorded at FCz; mean latencies: 19 and 30 ms, respectively). The amplitudes of the N9, the N14, the P14 and the P22 were taken relative to prestimulus baseline, whereas the others were taken from the preceding peak of opposite deflection (peak to peak). Two subjects did not present a clear P14 across all conditions; the N20 amplitude was thus calculated from baseline for these subjects.

We pooled the mirror trials into 4 bins each containing 3 consecutive trials (m1–3, m4–6, m7–9, and m10–12). This improved the reliability of the SEP averages while keeping a fair estimate of the time course of adaptation. Separate repeated-measures ANOVAs conducted on each SEP component did not reveal significant differences between the pre- and postadaptation phases, nor between the 3 velocities at which the normal trials were performed ( $P$  values > 0.5). Therefore all trials from the static and normal conditions were collapsed and further analyses were conducted with a single mean value for the static condition and a single mean value for the normal condition. One possibility for the lack of difference in SEP amplitude as a function of velocity is that the 3 velocities used for the normal



**Figure 1.** Following median nerve stimulation at the wrist of the drawing right hand, the peripheral evoked potentials were recorded at the right Erb's point (N9 component), whereas the spinal evoked potentials were recorded at the fifth cervical (N14 component). The cortical event-related potentials were recorded over contralateral primary somatosensory cortex (N20–P27 complex). Traces from 1 representative subject in the static condition are shown in the insets.

trials were all extremely slow. Thus the difference in efferent drive and afferent discharge may have been insufficient to have a measurable influence on the magnitude of the movement-induced gating. For comparison purposes, Staines et al. (1997) found a significant effect of movement velocity on tibial nerve SEP amplitude when the rate of a pedaling motion changed from 20 to 40, 60, or 80 rpm—several orders of magnitude greater than in the present study.

We first sought to determine the influence of movement on the amplitude of the peaks of interest. To do so the SEP components were submitted to separate 6 modalities (static, normal, m1-3, m4-6, m7-9, m10-12) repeated-measures ANOVAs. In the eventuality of a significant effect, we used the Dunnett method of multiple comparisons to contrast the static condition to every other condition ( $P < 0.05$ ). The critical  $t$ -ratio used thus corrected for 5 comparisons (static vs. normal; static vs. m1-3; static vs. m4-6; static vs. m7-9; static vs. m10-12). We then specifically assessed the effect of the visuomotor conflict on SEP amplitude by contrasting the normal condition to every mirror bin using separate 5 modalities (normal, m1-3, m4-6, m7-9, m10-12) repeated-measures ANOVAs. This analysis was carried out on the SEP data expressed as a percentage of each subjects' SEP amplitude in the static condition. Because we had hypothesized that the amplitude of the SEPs would be reduced in the mirror trials compared with the normal trials, we predesignated the normal condition as the reference against which the mirror bins would be compared using the Dunnett test ( $P < 0.05$ ). Here the critical  $t$ -ratio used thus corrected for 4 comparisons (normal vs. m1-3; normal vs. m4-6; normal vs. m7-9; normal vs. m10-12).

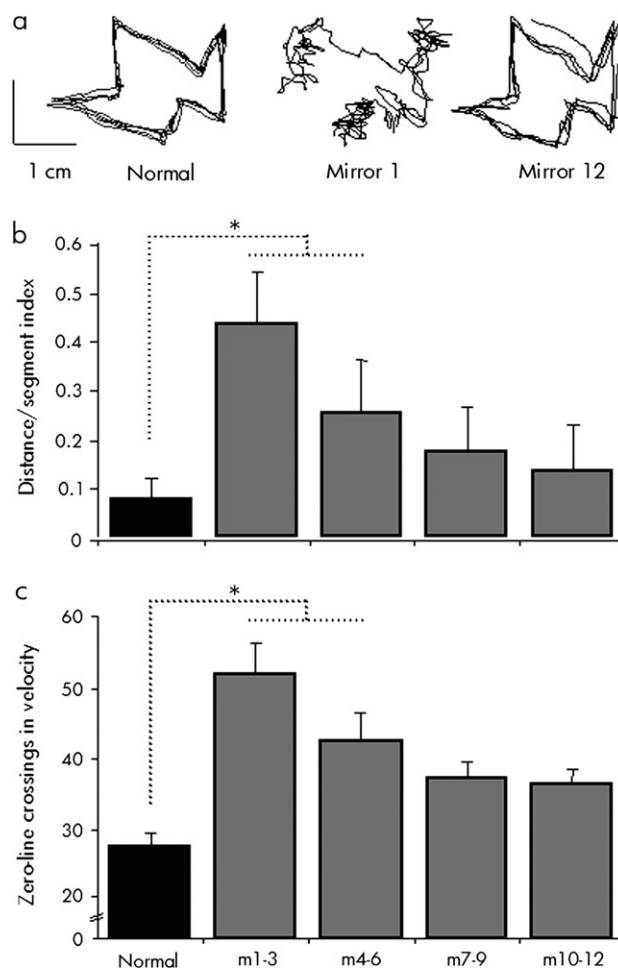
**Kinematics and EMG data.** The displacement data in the  $x$  and  $y$  directions were low-pass filtered using a second-order Butterworth filter with an 8-Hz cut-off frequency. As a reflection of subjects' efficiency in tracing the shape, we computed a distance/segment index by calculating the log of the ratio between the total distance covered by the pen and the total number of segments completed per trial [ $\log(\text{total distance}/\text{completed segments})$ ]. We also assessed tracing smoothness by measuring the number of reversals in direction per trial. This was done by calculating the total number of zero-line crossings in the velocity profiles per trial. This calculation was performed separately for the  $x$  and  $y$  velocity profiles and the 2 values were averaged to obtain a single measure per trial. The EMG recordings from the 4 muscles showed essentially tonic activity and no clear EMG bursts. To compare the EMG activity at the FPB, the FDI, the FCR and the ECR across conditions, we integrated the EMG data over the entire duration of each trial (this measure is valid because all trials were of equal duration). These data were normalized to the integrated EMG activity in the static trials.

Separate repeated-measures ANOVAs conducted on the aforementioned kinematics and EMG data did not reveal significant differences between the normal trials performed in the pre- and postadaptation phases ( $P$  values  $> 0.5$ ). Both trials at each instructed velocity were thus collapsed to obtain a single mean value for each velocity. The kinematics and EMG data were thus submitted to separate 7 modality (5 s/segment, 10 s/segment, 20 s/segment, m1-3, m4-6, m7-9, m10-12) repeated-measures ANOVAs. Tukey's test was used for post hoc comparisons ( $P < 0.05$ ).

## Results

Early exposure to mirror-reversed vision substantially perturbed subjects' tracing accuracy (Fig. 2*a*). This was reflected by a larger distance/segment index in the initial mirror trials (m1-3 and m4-6) than in the normal trials ( $P$  values  $< 0.001$ ; Fig. 2*b*). Performance increased throughout exposure, such that the distance/segment index did not differ significantly between the late mirror trials (m7-9 and m10-12) and the normal trials ( $P = 0.36$  and  $P = 0.54$ , respectively). The initial mirror trials (m1-3 and m4-6) also contained significantly more reversals in direction than the normal trials ( $P$  values  $< 0.01$ ; Fig. 2*c*). Movements became smoother with adaptation, such that the number of reversals in direction did not differ significantly between the late mirror trials (m7-9 and m10-12) and the normal trials ( $P = 0.06$  and  $P = 0.1$ , respectively). Together, these behavioral measurements reflect the gradual increase in performance typically observed during prolonged exposure to novel visuomotor environments (e.g., Harris 1963; Bernier et al. 2007; Redding et al. 2005; Sarlegna et al. 2007).

The average movement velocity in the mirror trials did not exceed that of the normal trials (Fig. S1 and videos in

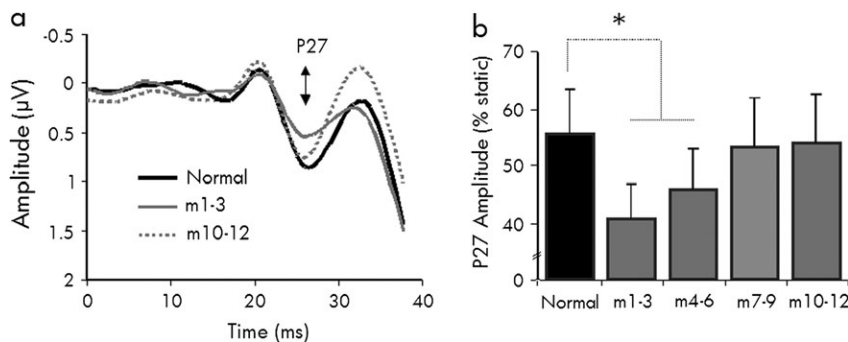


**Figure 2.** Main experiment, kinematics results. (*a*) Tracings of a representative subject in a normal trial, as well as in the first and last mirror trials. (*b*) Tracing performance as expressed by the total distance covered by the pen per segment completed in every trial (distance/segment index). A small index indicates efficient tracing as a small distance was necessary to achieve a given segment. (*c*) Number of reversals in direction as expressed by the average number of zero-line crossings on the velocity profiles per trial. Error bars: standard error of the mean.

Supplementary Material). Similarly, integrated EMG activity of the forearm and hand muscles was not significantly greater in the initial mirror trials (m1-3) than the normal trials ( $P = 0.39$  for FPB;  $P = 0.31$  for FDI;  $P = 0.29$  for FCR;  $P = 0.47$  for ECR). This confirms that subjects were not using a strategy of cocontraction to freeze the degrees of freedom in the face of the visuomotor conflict.

We found that the amplitude of the first volley of somatosensory afferents to area 1 of S1 (i.e., the parietal P27 component; Allison et al. 1991; Chéron et al. 2000; Inui et al. 2004) was significantly reduced by motion (reduction of 45% in the normal trials compared with the static trials;  $P < 0.01$ ). More importantly, in addition to this movement-induced gating, the parietal P27 showed further suppression in the initial mirror trials, being reduced by 59% for m1-3 and 54% for m4-6 compared with the static trials ( $P$  values  $< 0.001$ ). This represents a reduction of 27% for m1-3 ( $P < 0.01$ ) and 18% for m4-6 ( $P < 0.05$ ) with respect to the P27 amplitude in the normal trials (Fig. 3). Interestingly, we found that the extent of P27 suppression observed in the initial mirror trials was correlated with subjects' performance in these trials ( $r = 0.64$ ;  $P < 0.05$ ). This is represented in Figure 4, where we plotted the amplitude of the P27 component for each subject in the m1-3 bin as a function of their distance/segment index in these trials. As can be seen, subjects who performed the best upon exposure to the mirror (i.e., lowest distance/segment index) were those whose P27 gating was most important. In contrast, subjects whose performance was most deteriorated in these trials (i.e., largest distance/segment index) tended to be those for whom the least gating had occurred. As seen in Figure 3, the gating was gradually alleviated as a function of the behavioral level of adaptation, returning to baseline levels by m7-9 ( $P$  values  $> 0.5$  for m7-9 and m10-12 compared with the normal trials). No significant correlations were observed between the P27 amplitude and subjects' performance in the last 3 mirror bins ( $P$  values  $> 0.5$ ). This suggests that the P27 gating was related to performance essentially in the first mirror trials when the conflict was most important.

Similar to the P27, the amplitude of the frontal N30 presented a 40% reduction in the normal trials compared with the static trials ( $P < 0.01$ ; Table 1). However this component was not further reduced upon exposure to mirror-reversed vision ( $P = 0.15$ ). On the other hand, the peripheral N9, spinal N14, far-field potential P14, parietal N20 and frontal P22 did not change significantly across conditions ( $P$  values  $> 0.5$ ).



**Figure 3.** Main experiment, SEP results. (a) Grand average SEP traces at electrode C3 in the normal trials, as well as in m1-3 and m10-12. The arrow shows the P27 component, whose amplitude was calculated from the preceding peak of opposite deflection (N20). (b) Amplitude of the parietal P27 component, represented as a percentage of its amplitude in the static trials. A clear P27 attenuation can be seen in the initial mirror trials (m1-3 and m4-6) compared to the normal trials. Error bars: standard error of the mean.

## Control Experiment

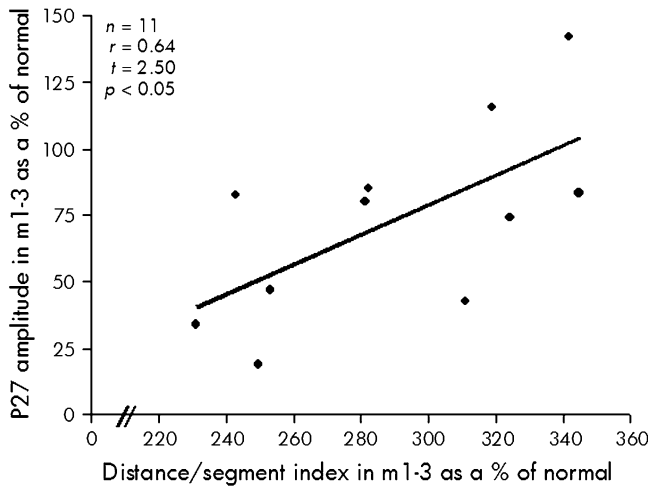
Results of the main experiment revealed that the trajectories were more erratic and contained more reversals in direction upon exposure to the visuomotor conflict than in the normal trials (Fig. 2c). Because SEP amplitude is contingent upon the kinematics of the task (Brooke et al. 1997; Staines et al. 1997), we conducted a control experiment to verify that the gating was specific to the visuomotor conflict and did not merely result from the differences in kinematics observed between conditions. To do so, we designed a condition which induced similar kinematic features as those recorded in the initial mirror trials of the main experiment, but without the presence of a visuomotor conflict.

## Materials and Methods

Eight right-handed volunteers (5 males, 3 females, age 26-39 years, mean 29) took part in the control experiment. The apparatus as well as the stimulation and recording procedures were identical to the main experiment. Subjects replicated the 2 static trials and the 6 normal trials (2 at each of the 3 instructed velocities) that were performed in the main experiment. In addition, they completed 3 control trials in which they followed the trajectory of a representative mirror 1 trial from the main experiment (Fig. 5 inset). However, in these control trials, subjects performed the tracings with direct vision rather than through a mirror. The trajectory was chosen to incur similar kinematic features as in the mirror 1 condition of the main experiment. The trajectory comprised 11 segments and had a total distance of 240 mm, which was within 1 standard deviation of the mean of the mirror 1 trials (i.e.,  $10.9 \pm 4.9$  segments and  $248 \pm 64$  mm). Throughout these control trials, an auditory signal constrained movement velocity to the mean of the mirror 1 trials (15 s/segment;  $\sim 1.5$  mm/s). Offline analyses revealed that the number of zero-line crossings in velocity did not differ significantly ( $P > 0.5$ ) between the control trials (mean = 61) and the mirror 1 trials (mean = 59).

## Results

As expected, kinematic analyses revealed that the number of zero-line crossings in velocity was significantly greater in the control trials than in the normal trials (means of 61 and 25 for the control and normal trials respectively;  $P < 0.01$ ). The distance/segment index was also significantly larger in the control condition than in the normal condition ( $P < 0.001$ ). Consistent with the results of the main experiment, we found



**Figure 4.** Main experiment, SEP results. Relationship between the amplitude of the P27 component in the m1-3 bin and the distance/segment index in these trials, expressed as a percentage of their respective values in the normal trials. Each data point represents 1 subject. Subjects who performed the best upon exposure to the mirror (i.e., lowest distance/segment index) were those whose P27 gating was most important. In contrast, subjects whose performance was most deteriorated in these trials (i.e., largest distance/segment index) tended to be those for whom the least gating had occurred.

**Table 1**

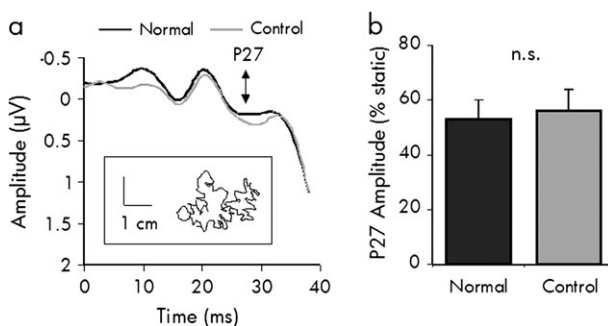
Peripheral and cortical SEP amplitudes in the static, normal, and mirror conditions<sup>a</sup>

	Static	Normal	m1-3	m4-6	m7-9	m10-12
N9	3.56 (0.52)	3.56 (0.46)	3.39 (0.43)	3.55 (0.38)	3.82 (0.36)	3.56 (0.44)
N14	2.12 (0.39)	1.97 (0.34)	2.08 (0.35)	1.84 (0.37)	1.99 (0.27)	1.96 (0.36)
N20	0.28 (0.85)	0.30 (0.57)	0.24 (0.54)	0.23 (0.56)	0.37 (0.51)	0.23 (0.67)
P27	2.38 (0.30)	1.32 (0.19)*	0.97 (0.15)*†	1.09 (0.17)*†	1.27 (0.21)*	1.29 (0.20)*
N30	1.21 (0.18)	0.73 (0.10)*	0.78 (0.13)*	0.81 (0.08)*	0.91 (0.15)*	0.86 (0.15)*

Note: \*Represents significant differences ( $P < 0.05$ ) with respect to the static trials.

†Represents significant differences ( $P < 0.05$ ) with respect to the normal trials.

<sup>a</sup>Values are shown as means ( $\pm$ SE) ( $n = 11$ ) in  $\mu$ V.



**Figure 5.** Control experiment, SEP results. (a) Grand average SEP traces at electrode C3 in the normal trials and the control trials, which were all performed with direct vision of the hand and shape. The shape used for the normal trials was the same as in the main experiment and the shape used for the control trials was the trajectory of a representative mirror 1 trial from the main experiment (inset). (b) Amplitude of the parietal P27 component, represented as a percentage of its amplitude in the static trials. Error bars: standard error of the mean.

that the parietal P27 was significantly reduced by motion (reduction of 47% in the normal trials compared with the static trials;  $P < 0.01$ ). Most importantly, however, despite large differences in kinematics, the amplitude of the P27 component

was not further reduced in the control trials compared with the normal trials ( $P > 0.5$ ) (Fig. 5b).

## Discussion

Here we show that the early arrival of somatosensory afferents to S1 (i.e., the parietal P27 component; Allison et al. 1991; Chéron et al. 2000; Inui et al. 2004) is attenuated by 27% when tracing with mirror-reversed vision compared with tracing with direct vision. Importantly, this additional gating did not take place when the tracings were performed under similar kinematic conditions as in the initial mirror trials, but with direct vision (control experiment). These results confirm that the additional P27 attenuation observed in the initial mirror trials did not result from increased peripheral receptor discharge or from a possible increase in efferent activity associated with the different kinematics. It is also unlikely that the gating resulted from a reallocation of attentional resources during exposure, as the N20-P27 response has been shown to be resistant to changes in attentional state (Arthurs et al. 2004). Interestingly, we found that the P27 amplitude returned to baseline levels as a function of the behavioral level of adaptation, indicating that the gating was functionally specific to resolving the visuomotor conflict.

The attenuation observed for the P27 was not accompanied by modulations to the peripheral N9 component. This component's robustness across conditions which presumably involved different levels of background muscle spindle activity (e.g., static vs. fastest normal condition), indicates that the gating observed at the cortical level cannot be merely attributable to a change in muscle spindle output as observed by Jones et al. (2001). The stability of the N9 also confirms that the intensity of the stimulation was constant across the experiment. In addition, the amplitude of the spinal N14 component did not change across conditions, suggesting that the gating did not take place within the first-order relay neurons in the spinal cord. Importantly, it is unlikely that the inhibition acted at the thalamic level because the parietal N20, which is generated in area 3b of S1 (Allison et al. 1991), was not modulated across conditions. Indeed, given that excitatory activity is serially transmitted from area 3b (N20) to area 1 (P27), the N20 would have been attenuated concomitantly with the P27 had the gating been subcortical (Wolters et al. 2005). These data therefore indicate that the gating acted directly within S1 to reduce cortico-cortical excitability between area 3b and area 1. These areas are known to receive proprioceptive inputs (Abbruzzese et al. 1981; Rushton et al. 1981) but also cutaneous afferents that subserve the sense of touch (Grünwald et al. 1984; Knecht et al. 1993). Because the P27 activity represents the compound activity stemming from all these receptors, it is possible that the suppression targeted both the senses of proprioception and touch. Nevertheless, in the present task the sensory conflict was probably more important between vision and proprioception than between vision and touch, and therefore the functional role of gating may have been mostly related to reducing proprioceptive input.

According to recent work, the gating signals would originate from cortical structures involved in the preparation of the motor commands (Seki et al. 2003; Voss et al. 2006; Christensen et al. 2007). One likely structure is the prefrontal cortex, which is known to regulate the transmission of proprioceptive afferents to primary sensory areas according to their task relevance (Knight et al. 1999; Staines et al. 2002). Interestingly,

using positron emission tomography, Della-Maggiore and McIntosh (2005) found increased prefrontal cortex activity in the early phase of exposure to novel visuomotor relationships compared with normal visuomotor environments. These authors proposed that this additional prefrontal activation may serve to inhibit the sensorimotor processes associated with the unadapted visuomotor mapping. The SEP methodology does not allow to identify neural activity specifically linked to generating these inhibitory signals, as the evoked potentials only represent the activity arising from the afferent volley. Nonetheless, the prefrontal areas are known to project toward S1 (Jones 1986), and it is therefore reasonable to speculate that the P27 attenuation reported here may represent the output of this inhibitory fronto-parietal network. Furthermore, our finding of a gradual release of gating through practice supports recent brain imaging work showing that the improvement in performance observed during adaptation is associated with increased activity in contralateral sensorimotor areas (Della-Maggiore and McIntosh 2005). Overall, the present findings provide empirical support to the suggestion that visuomotor adaptation is paralleled by a fronto-parietal shift in cortical activity (Staines et al. 2002; Krakauer et al. 2004).

We found that the extent to which the P27 component was suppressed in the initial mirror trials covaried with subjects' performance in these trials. This suggests a link between sensory suppression and performance, as subjects who presented the largest gating in the initial mirror trials were those who tended to perform the best. Hence, when a visuo-proprioceptive conflict occurs, the transient gating may serve to relieve some of the sensory interference, and as a result indirectly facilitate visual guidance of the movement. This is consistent with the finding that deafferented patients, who can be considered as extreme cases of somatosensory suppression, perform better than healthy individuals when exposed to novel visuomotor relationships (Lajoie et al. 1992; Guédon et al. 1998). Together with Jones et al.'s (2001) finding of a reduction in muscle spindle discharge upon exposure to a visuomotor conflict, the present evidence for cortical suppression of somatosensory afferents suggests that the CNS employs multiple mechanisms to reduce the sensory interference in the face of visuo-proprioceptive conflicts.

Adaptation is thought to result from gradually updating the mapping between the predicted sensory feedback from a motor command and the actual sensory feedback (Miall and Wolpert 1996; Krakauer et al. 2000; Tong and Flanagan 2003; Tseng et al. 2007). It is also accompanied by visual and/or proprioceptive recalibration, depending on task conditions (Harris 1963; Simani et al. 2007). In this light it may appear counterintuitive to suppress somatosensory input, as the CNS would require full access to these signals in order to adapt to the sensorimotor conflict. Therefore, despite the observed suppression at S1, the somatosensory signals may still have been faithfully conveyed to structures implicated in the adaptive process per se. In accordance with this proposal, we found that the frontal N30 component, despite being attenuated by motion, was not further reduced upon exposure to mirror-reversed vision. Although the origin of the N30 is still under debate, recent intracerebral recording studies suggest that it originates from the premotor cortex and is generated by direct thalamo-cortical inputs (Kanovský et al. 2003). Unlike the parietal P27, the N30 would be unaffected by intracortical inhibitory mechanisms (Chéron et al. 2000), and accordingly it has been

found to be modulated independently from the P27 under a variety of behavioral conditions (Allison et al. 1991; Kanovský et al. 2003). Given that the premotor cortex is implicated in updating the sensorimotor relationship (Kurata and Hoshi 1999; Lee and van Donkelaar 2006; Seidler et al. 2006), the differential gating pattern observed for the P27 and the N30 may indicate that although the CNS suppressed somatosensory signals within S1 to facilitate performance, it may have maintained a normal flow of afferent signals to structures subserving adaptation.

In conclusion, the present findings reveal that the CNS exercises a dynamic control over the flow of somatosensory signals at the cortical level during the course of visuomotor adaptation. These modulations may constitute the neural underpinning of recent computational accounts suggesting that adaptation partly results from a change in the weights attributed to visual and somatosensory cues (Smeets et al. 2006). Further research should focus on whether the transmission of visual signals is concurrently facilitated under such circumstances.

### Supplementary Material

Supplementary material can be found at: <http://www.cercor.oxfordjournals.org/>

### Funding

Délégation Générale pour l'Armement and the Centre National d'Études Spatiales; and National Sciences and Engineering Research Council of Canada scholarship to P.M.B.

### Notes

We wish to thank Frank Buloup and Jean-Luc Velay for technical help as well as Dr Jacques Grapperon for helping to collect preliminary data. We also thank Fabrice Sarlegna and Erin K. Cressman for helpful comments on the manuscript. *Conflict of Interest:* None declared.

Address correspondence to Jean Blouin, PhD, Laboratoire de Neurobiologie de la Cognition, Aix-Marseille Université, CNRS, 3, Place Victor Hugo, 13331 Marseille Cedex 3, France. Email: [jean.blouin@univ-provence.fr](mailto:jean.blouin@univ-provence.fr).

### References

- Abbruzzese G, Ratto S, Favale E, Abbruzzese M. 1981. Proprioceptive modulation of somatosensory evoked potentials during active or passive finger movements in man. *J Neurol Neurosurg Psychiatry.* 44:942-949.
- Allison T, McCarthy G, Wood CC, Jones SJ. 1991. Potentials evoked in human and monkey cerebral cortex by stimulation of the median nerve. A review of scalp and intracranial recordings. *Brain.* 114:2465-2503.
- Arthurs OJ, Johansen-Berg H, Matthews PM, Boniface SJ. 2004. Attention differentially modulates the coupling of fMRI BOLD and evoked potential signal amplitudes in the human somatosensory cortex. *Exp Brain Res.* 157:269-274.
- Balslev D, Christensen LO, Lee JH, Law I, Paulson OB, Miall RC. 2004. Enhanced accuracy in novel mirror drawing after repetitive transcranial magnetic stimulation-induced proprioceptive deafferentation. *J Neurosci.* 24:9698-9702.
- Bernier P-M, Gauthier GM, Blouin J. 2007. Evidence for distinct, differentially adaptable sensorimotor transformations for reaches to visual and proprioceptive targets. *J Neurophysiol.* 98:1815-1819.
- Brindle TJ, Nitz AJ, Uhl TL, Kifer E, Shapiro R. 2006. Kinematic and EMG characteristics of simple shoulder movements with proprioception and visual feedback. *J Electromyogr Kinesiol.* 16:236-249.

- Brooke JD, Cheng J, Collins DF, McIlroy WE, Misiasek JE, Staines WR. 1997. Sensori-sensory afferent conditioning with leg movement: gain control in spinal reflex and ascending paths. *Prog Neurobiol.* 51:393-421.
- Chéron G, Dan B, Borenstein S. 2000. Sensory and motor interfering influences upon somatosensory evoked potentials. *J Clin Neurophysiol.* 17:280-294.
- Christensen MS, Lundbye-Jensen J, Geertsen SS, Petersen TH, Paulson OB, Nielsen JB. 2007. Premotor cortex modulates somatosensory cortex during voluntary movements without proprioceptive feedback. *Nat Neurosci.* 10:417-419.
- Della-Maggiore V, McIntosh AR. 2005. Time course of changes in brain activity and functional connectivity associated with long-term adaptation to a rotational transformation. *J Neurophysiol.* 93:2254-2262.
- Gratton G, Coles MG, Donchin E. 1983. A new method for off-line removal of ocular artifact *Clin Neurophysiol.* 55:468-484.
- Grünewald G, Grünewald-Zuberbier E, Schuhmacher H, Mewald J, Noth J. 1984. Somatosensory evoked potentials to mechanical disturbances of positioning movements in man: gating of middle-range components. *Electroencephalogr Clin Neurophysiol.* 58:525-536.
- Guédon O, Gauthier G, Cole J, Vercher J-L, Blouin J. 1998. Adaptation in visuomanual tracking depends on intact proprioception. *J Mot Behav.* 30:234-248.
- Harris CS. 1963. Adaptation to displaced vision: visual, motor or proprioceptive change? *Science.* 140:812-813.
- Inui K, Wang X, Tamura Y, Kaneoke Y, Kakigi R. 2004. Serial processing in the human somatosensory system. *Cereb Cortex.* 14:851-857.
- Jones EG. 1986. Connectivity of the primate sensory-motor cortex. In: Jones EG, Peters A, editors. *Cerebral cortex.* Vol. 5. New York: Plenum Press. p. 113-183.
- Jones SJ, Halonen JP, Shawkat F. 1989. Centrifugal and centripetal mechanisms involved in the "gating" of cortical SEPs during movement. *Electroencephalogr Clin Neurophysiol.* 74:36-45.
- Jones KE, Wessberg J, Vallbo A. 2001. Proprioceptive feedback is reduced during adaptation to a visuomotor transformation: preliminary findings. *Neuroreport.* 12:4029-4033.
- Kanovský P, Bare M, Rektor I. 2003. The selective gating of the N30 cortical component of the somatosensory evoked potentials of median nerve is different in the mesial and dorsolateral frontal cortex: evidence from intracerebral recordings. *Clin Neurophysiol.* 114:981-991.
- Knecht S, Kunesch E, Buchner H, Freund HJ. 1993. Facilitation of somatosensory evoked potentials by exploratory finger movements. *Exp Brain Res.* 95:330-338.
- Knight RT, Staines WR, Swick D, Chao LL. 1999. Prefrontal cortex regulates inhibition and excitation in distributed neural networks. *Acta Psychol.* 101:159-178.
- Krakauer JW, Ghilardi MF, Mentis M, Barnes A, Veytsman M, Eidelberg D, Ghez C. 2004. Differential cortical and subcortical activations in learning rotations and gains for reaching: a PET study. *J Neurophysiol.* 91:924-933.
- Krakauer JW, Pine ZM, Ghilardi M-F, Ghez C. 2000. Learning of visuomotor transformations for vectorial planning of reaching trajectories. *J Neurosci.* 20:8916-8924.
- Kurata K, Hoshi E. 1999. Reacquisition deficits in prism adaptation after muscimol microinjection into the ventral premotor cortex of monkeys. *J Neurophysiol.* 81:1927-1938.
- Lajoie Y, Paillard J, Teasdale N, Bard C, Fleury M, Forget R, Lamarre Y. 1992. Mirror drawing in a deafferented patient and normal subjects: visuoproprioceptive conflict. *Neurology.* 42:1104-1106.
- Lee JH, van Donkelaar P. 2006. The human dorsal premotor cortex generates on-line error corrections during sensorimotor adaptation. *J Neurosci.* 26:3330-3334.
- Legon W, Staines WR. 2006. Predictability of the target stimulus for sensory-guided movement modulates early somatosensory cortical potentials. *Clin Neurophysiol.* 117:1345-1353.
- Miall RC, Wolpert DM. 1996. Forward models for physiological motor control. *Neural Netw.* 9:1265-1279.
- Prochazka A. 1989. Sensorimotor gain control: a basic strategy of motor control systems? *Prog Neurobiol.* 33:281-307.
- Redding GM, Rossetti Y, Wallace B. 2005. Applications of prism adaptation: a tutorial in theory and method. *Neurosci Biobehav Rev.* 29:431-444.
- Rushton DN, Rothwell JC, Craggs MD. 1981. Gating of somatosensory evoked potentials during different kinds of movement in man. *Brain.* 104:465-491.
- Sarlegna FR, Gauthier GM, Blouin J. 2007. Influence of feedback modality on sensorimotor adaptation: contribution of visual, kinesthetic, and verbal cues. *J Mot Behav.* 39:247-258.
- Seidler RD, Noll DC, Chintalapati P. 2006. Bilateral basal ganglia activation associated with sensorimotor adaptation. *Exp Brain Res.* 175:544-555.
- Seki K, Perlmutter SI, Fetz EE. 2003. Sensory input to primate spinal cord is presynaptically inhibited during voluntary movement. *Nat Neurosci.* 6:1309-1316.
- Simani MC, McGuire LM, Sabes PN. 2007. Visual-shift adaptation is composed of separable sensory and task-dependent effects. *J Neurophysiol.* 98:2827-2841.
- Smeets JBJ, van den Dobbelen JJ, de Grave DDJ, van Beers RJ, Brenner E. 2006. Sensory integration does not lead to sensory calibration. *Proc Natl Acad Sci USA.* 103:18781-18786.
- Staines WR, Brooke JD, Cheng J, Misiasek JE, MacKay WE. 1997. Movement-induced gain modulation of somatosensory potentials and soleus H-reflexes evoked from the leg I. Kinaesthetic task demands. *Exp Brain Res.* 115:147-155.
- Staines WR, Graham SJ, Black SE, McIlroy WE. 2002. Task-relevant modulation of contralateral and ipsilateral primary somatosensory cortex and the role of a prefrontal-cortical sensory gating system. *Neuroimage.* 15:190-199.
- Tong C, Flanagan JR. 2003. Task-specific internal models for kinematic transformations. *J Neurophysiol.* 90:578-585.
- Tseng Y-w, Diedrichsen J, Krakauer JW, Shadmehr R, Bastian AJ. 2007. Sensory prediction errors drive cerebellum-dependent adaptation of reaching. *J Neurophysiol.* 98:54-62.
- Voss M, Ingram JN, Haggard P, Wolpert DM. 2006. Sensorimotor attenuation by central motor command signals in the absence of movement. *Nat Neurosci.* 9:26-27.
- Wolters A, Schmidt A, Schramm A, Zeller D, Naumann M, Kunesch E, Benecke R, Reiners K, Classen J. 2005. Timing-dependent plasticity in human primary somatosensory cortex. *J Physiol.* 565:1039-1052.