This PET H215O study uses a reaching task to determine the neural basis of the unconscious motor speed up observed in the context of urgency in healthy subjects. Three conditions were considered: self-initiated (produce the fastest possible movement toward a large plate, when ready), externally-cued (same as self-initiated but in response to an acoustic cue) and temporally-pressing (same as externally-cued with the plate controlling an electromagnet that prevented a rolling ball from falling at the bottom of a tilted ramp). Results show that: (1) Urgent responses (Temporally-pressing versus Externally-cued) engage the left parasagittal and lateral cerebellar hemisphere and the sensorimotor cortex (SMC) bilaterally; (2) Externally-driven responses (Externally-cued versus Self-initiated) recruit executive areas within the contralateral SMC; (3) Volitional responses (Self-initiated versus Externally-cued) involve prefrontal cortical areas.

These observations are discussed with respect to the idea that neuromuscular energy is set to a submaximal threshold in self-determined situations. In more challenging tasks, this threshold is raised and the first answer of the nervous system is to optimize the response of the lateral (i.e. crossed) corticospinal tract (contralateral SMC) and ipsilateral cerebellum. In a second step, the anterior (i.e. uncrossed) corticospinal tract (ipsilateral SMC) and the contralateral cerebellum are recruited. This recruitment is akin to the strategy observed during recovery in patients with brain lesions.

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Keywords: Cerebellum; Lateral corticospinal tract; PET; Sensorimotor cortex; Urgency

Introduction

In 1921 Souques described Parkinson’s disease (PD) patients who could not stand up nor walk without help, but suddenly became able to run or climb a set of stairs when facing an urgent situation. This surprising improvement of motor performance was named “paradoxical kinesia”. To account for this phenomenon it was suggested that the basal ganglia (BG) could play a much more important role in internally than in externally regulated movements.

Recently, a behavioral study was conducted by our group that led us to question the notion of “paradoxical kinesia” in PD. We used a ball-catch paradigm, to investigate the effect of motor urgency in PD patients and normal subjects (Ballanger et al., 2006). We compared reaching movements requiring to press a large plate under three conditions: self-initiated: produce the fastest possible movement; externally-cued: same as self-initiated but in response to an acoustic cue; temporally-pressing: same as externally-cued with the plate controlling an electromagnet that prevented a ball falling at the bottom of a tilted ramp. Results showed that external cues and urgent conditions increased movement speed by a similar amount in PD patients and healthy subjects (temporally-pressing > externally-cued > self-initiated). This observation suggested (1) that the so-called “paradoxical kinesia” were not so paradoxical after all, and (2) that contextual variations of movement velocity were independent of BG dysfunctions.

The main aim of the present study is to investigate the neural basis of the motor speed up observed in urgent situations. As shown in previous experiments involving healthy subjects, the pattern of cerebral activation is clearly different between self-initiated and externally guided movement (Deiber et al., 1991, 1999; Playford et al., 1992; Jahanshahi et al., 1995; Jenkins et al., 2000). The dorsolateral prefrontal cortex (DLPFC), rostral supplementary motor area (SMA), anterior cingulate cortex (ACC) and lateral premotor cortex have been consistently associated with self-generated motor task, and the caudal SMA,
primary motor cortex and basal ganglia (BG) with externally-cued movement (Deiber et al., 1991, 1999; Playford et al., 1992; Jahanshahi et al., 1995; Jenkins et al., 2000; Cunnington et al., 2002). Concerning the BG, it is worth mentioning that a significant involvement of this structure in externally-cued actions was only observed when different movements types were compared to a rest condition. No higher activation in the BG network was observed when self-initiated and externally-cued movements were compared with each other (Jahanshahi et al., 1995; Jenkins et al., 2000; Cunnington et al., 2002). An absence of systematic involvement of the BG for internally-regulated movements was also reported in electrophysiological and inactivation studies in monkeys (Kimura et al., 1992; Inase et al., 1996; Turner and Anderson, 2005).

To our knowledge, no functional imaging studies have been conducted in order to investigate the neurophysiological mechanisms that underlie the improvement of motor performance in urgent situations. The present work aims to fill this gap.

Materials and methods

Subjects

Eight healthy subjects (4 males, 4 females; mean age±S.D.: 54±8.1) participated in the study. All subjects were right-handed and did not present any neurological disease.

The study was approved by the local research ethics committee. All subjects participated after the aims of the study and the nature of the procedures had been fully explained. They signed an informed consent form according to the Declaration of Helsinki.

Task

The behavioral apparatus is shown in Fig. 1. It is similar to the one used in a previous behavioral experiment (Ballanger et al., 2006). The subjects lay supine on the scanner bed with the right hand in contact with a press button (1×0.5 cm) positioned in the middle of the abdomen in a relaxed posture. A large vertical contact plate (28×18 cm) was suspended over the scanner bed, 30 cm in front of the subjects’ hand. Contact with this plate controlled an electromagnetic catch located at the bottom of a tilted ramp. A bright green dot was positioned in front of the electromagnetic catch, to allow gaze fixation. During the experiment, horizontal EOG was recorded continuously at a frequency of 1000 Hz and monitored on an oscilloscope.

Three conditions were considered. (1) Internally-driven (or Self-Initiated). The subjects were required to “wait until they felt ready and then hit the contact plate with the palm of the hand and the fastest possible movement”. Right at the end of the reach, an auditory cue (computer beep, 200 ms) was provided to ensure the subjects to return to the starting position. The delay was computed with respect to the end of the movement to ensure a constant 6.6 s period between the start (fixation light on) and the end (fixation light blinking) of the trial. Keeping the total trial time constant was necessary to balance the number of movements across conditions. A preliminary study allowed us to determine that a 6.6 s delay was more than sufficient to allow the subjects to feel ready and complete their movement. (2) Externally-cued: the subjects were required to “react and move as fast as possible in response to an auditory cue” (computer beep, 200 ms). To prevent anticipations, this cue was presented at random times (1.5 to 2.5 s) with respect to the positioning of the hand at the starting location. After completion of the movement the green fixation light blinked for 1 s, instructing the subjects to bring their hand back to the starting position. The blink onset time was selected in order to keep the trial duration equal to 6.6 s. (3) Temporally-pressing: this condition was identical to the Externally-cued condition except that the auditory cue sounded as a ball was released at the top of the ramp (release mechanism was not visible) at random time with respect to the positioning of the hand at the starting location (1.5 to 2.5 s). The subject was asked to stop this ball with the electromagnetic catch. Ramp tilt was adjusted for each subject before the experiment during a short training session so as to ensure a failure rate roughly equal to 50%. After completion of the movement the green fixation light blinked for 1 s, instructing the subjects to bring their hand back to the starting position. The blink onset time was selected in order to keep the trial duration equal to 6.6 s. Note that vision of the rolling ball was prevented by an opaque barrier (to ensure effectiveness, subjects had been trained outside the scanner with full vision of the ball). In addition, foam was placed at the bottom and the sides of the ramp to remove any contact noise of the ball on the ramp. When the ball was missed it fell in a bucket full of foam. Consequently, the subjects had no visual or auditory cue about the ball or their performance at any time. The noise of the contact plate and magnet was audible, but present in all conditions. The presence of the ball was simply indicated to the subjects at the beginning of the scan. They were told the number of successfully caught balls at the end of the scan.

Each condition was performed during 100 s leading to a total of 15 movements and was repeated four times in a randomized order leading to a total number of 12 scans per subject.

Behavioral analysis

Movement duration (MD, interval from start position release (release of the button press) to contact with the vertical plate) was computed for all conditions. Reaction time (RT, interval from beep
to start position release) was also computed for the externally triggered conditions. An ANOVA design was used to identify significant differences between the experimental conditions. Data from all responses were incorporated in this ANOVA. The Duncan’s multiple range test was used for post-hoc comparisons of the means (Winer, 1971). Threshold for statistical significance was set at 0.05.

**PET data acquisition**

PET scanning was performed at the CERMEP (Lyon, France) on a CTI HR+ Siemens tomograph (CTI/Siemens, Knoxville, Tennessee, USA). The head was maintained in a fixed position using a molded helmet. Head position was checked before and after each scan using a laser alignment together with reference points on the Reid’s line.

A 10 min transmission scan was acquired using rotating rod sources filled with $^{68}\text{Ge}/^{68}\text{Ga}$. For each emission scan, an intravenous injection of $343\pm77$ MBq of H$_2^{15}$O through a forearm catheter placed into the brachial vein was realized. The integrated counts were collected for 90 s, starting 30 s after the injection and 10 s after the start of the behavioral task. The interval between successive H$_2^{15}$O administrations was 9 min to allow for adequate radioactivity decay. Images were reconstructed by 3D back-filtered projection (Hanning filter; cut-off frequency, 0.5 cycles/pixel), giving a transaxial resolution of 6.5 mm full width at half maximum, and displayed in a $128\times128$ pixel format with 63 planes creating 2 mm cubic voxels.

**PET data analysis**

Using CAPP software, the original emission scans in ECAT7 file format were converted to ANALYZE file format that were then processed in MATLAB 5.3 (MathWorks, Natick, MA) using the Statistical Parametric Mapping software (SPM 99, Wellcome Department of Cognitive Neurology, MRC Cyclotron Unit, London, UK). In the first stage of analysis, the 12 images from each subject were realigned to the first scan with an automated algorithm for head movement correction and then normalized into the standard stereotactic space provided in SPM. The normalized images were smoothed with an isotropic Gaussian filter of 12 mm to account for variation in gyral anatomy and individual variability in structure–function relationships, and to improve the signal-to-noise ratio. Variations in global flow across subjects were removed by proportionally scaling each image to have an arbitrary level of 50 ml/100 ml/min. rCBF changes were statistically analyzed for all voxels exceeding 80% of the mean value of the scan.

The analysis targeted the following variables:

- The increase of rCBF specific to internally-driven actions, observed contrasting the conditions *Self-initiated with Externally-cued*.
- The increase of rCBF specific to externally cued actions, observed contrasting the conditions *Externally-cued with Self-initiated*.
- The increase of rCBF specific to urgency, observed contrasting the conditions *Temporally-pressing with Externally-cued*.

The covariation of rCBF with movement duration and reaction time was also analyzed. In a first step, the mean movement duration and reaction time measured for each scan were used as covariates in a covariate only model. In a second step, the negative and positive covARIATIONS of rCBF with these covariates were determined.

Because of a priori hypothesis the analysis was restricted to voxels located into a mask that included the frontal lobes, postcentral regions, ACC, cerebellum, parietal cortex, and basal ganglia, i.e. all regions known to be involved in motor function (Deiber et al., 1991, 1999; Playford et al., 1992; Jahanshahi et al., 1995; Jenkins et al., 2000; Desmurget et al., 2001). This mask was created using VoiTool (http://www.ihb.spb.ru/~pet_lab/ VTO/VTOMain.html).

Global differences in cerebral blood flow were covaried out for all voxels within the mask and comparisons across conditions were made using *t* statistics with appropriate linear contrasts, and then converted to Z-scores. For regions located into the mask small volume corrections were applied to compensate for multiple comparisons. Only voxels which exceeded a threshold of an uncorrected *p*-value ≤ 0.001 were considered as significant. The minimal cluster size comprised at least 10 voxels. Cerebellar activations were attributed to an anatomical site according to the three-dimensional (3D)-atlas of the cerebellum of Schmahmann et al. (2000) and to the MRI atlas of the human cerebellar nuclei of Dimitrova et al. (2006), non-cerebellar activations were attributed according to the atlas of Talairach and Tournoux (1988) after images were transformed from procedures developed by M. Brett (http://www.mrc-cbu.cam.ac.uk/Imaging).

**Results**

**Behavioral results**

For movement duration (MD), a significant effect of the condition factor ($F_{2,14}=19.50, p<0.0001$) was found. More specifically: (1) MD was longer in the *Self-initiated* condition (252 ms) than in the *Externally-cued* condition (199 ms; post-hoc, $p<0.05$); (2) MD was longer in the *Externally-cued* (199 ms) than in the *Temporally-pressing* condition (164 ms; post-hoc, $p<0.05$).

For the *Externally-cued* and *Temporally-pressing* experimental conditions, reaction time (RT) showed the same pattern of variation as MD. Indeed, a significant effect of the condition factor was observed ($F_{1,7}=6.75, p<0.05$), with a RT being longer in the *Externally-cued* condition (206 ms) than in the *Temporally-pressing* condition (196 ms). These results are presented in Fig. 2.

Although RT has no real meaning in the *Self-initiated* condition, it is worth noting that the time between the positioning of the hand at the starting point and the start of the motor response was highly variable from trial to trial, in all subjects. On average, the duration between the completion of the return movement and the start of the next movement was equal to 928 ms. The mean intra-individual S.D. reached 403 ms. The mean maximal delay was equal to 2348 ms. The mean minimal delay was equal to 321 ms. These results show that movements were not performed in a rhythmical manner in the *Self-initiated* condition.

Analyses of the horizontal EOG indicated that fixation was rarely broken during the PET sessions. Saccades were sometimes detected during the return phase, when the subject had troubles finding the starting point. These episodes occurred with the same frequency in all conditions, indicating that systematic differences in oculomotor activity cannot account for systematic difference in brain activity during the three experimental conditions.
Theoretically, a change in hand velocity is not only expected to influence the movement duration, but also the motor reaction time. Indeed, the delay between EMG activity and the overt arm movement decreases slightly with movement acceleration and velocity (Desmurget et al., 2004). In addition, the time required to close the starting switch (2 mm displacement) should be shorter when MD decreases. It is thus possible that the decrease in RT in Temporally-Pressing with respect to Externally-Cued was caused by a decrease in MD. To address this possibility, we conducted a simulation of the arm movement made to the response button. We computed the effect of changing MD on the time elapsed during contact switch closure (travel: 2 mm). The two-link arm biomechanical parameters were taken from Winter (1979), and an optimal control algorithm was used to derive the arm trajectory (Guigon et al., 2007). We then computed the time elapsed during the travel of the microswitch (2 mm) at movement onset. To evaluate the robustness of this estimate, values of body mass, link lengths, and muscle time constants were varied ±25% around their chosen value (respectively 60 kg, 30 and 40 cm, 40 ms activation and excitation time constants). A Hill-type muscle model was also tested. Results indicated that changes in RT were likely to be too large to be only a byproduct of MD variations. Indeed, we found that the variations of RT due to variations MD were smaller than 5 ms (4.7 ms ±0.4 ms).

PET results

**Activation profile during self-initiated versus externally-cued movements**

Areas showing a significant rCBF increase during Self-initiated movements compared with Externally-cued movements were the right inferior frontal cortex (Brodmann areas (BA) 47 and 44) and the superior frontal gyrus (BA 8). The location, coordinates and peak Z-scores of activated areas are detailed in Table 1.

**Activation profile during externally-cued versus self-initiated movements**

Only the left (contralateral) primary motor cortex (BA 4) showed a significant rCBF increase when the Externally-cued condition compared with Self-initiated condition. The location, coordinates and peak Z-scores of activated areas are detailed in Table 1.

**Activation profile during temporally-pressing versus externally-cued condition**

Areas showing a significant rCBF increase when the Temporally-pressing condition was compared with Externally-cued condition included (1) the parasagittal cerebellar hemisphere and vermis on the left side (left lobule VI and Crus I), (2) the lateral cerebellar hemisphere, also on the left side, and (3) the sensorimotor cortex bilaterally (Figs. 3A and B). The location, coordinates and peak Z-scores of activated areas are detailed in Table 1.

**Covariation of rCBF with behavioral measures**

**Movement duration**

A negative covariation was observed between MD and the activation of the primary motor cortex (BA 4), the basal ganglia (lateral pallidum) and the inferior frontal cortex (BA 47) all in the contralateral (left) hemisphere. A negative covariation was also found in the right anterior cingulate (BA 32 and 24). These results indicate that faster movements were accompanied by greater rCBF in these areas. The location, coordinates and peak Z-scores of the activated areas are detailed in Table 2.

**Reaction time**

Reaction time showed a significant negative covariation with rCBF in the right medial frontal gyrus (BA 8), ACC (BA 32); in the left DFLPC (BA9) and SMA, as well as in the primary motor cortex bilaterally. This means that shorter RT were associated with greater rCBF in these areas. The location, coordinates and peak Z-scores of the activated areas are detailed in Table 2.

**Discussion**

Behavioral data of this study shows that healthy subjects can exhibit faster motor responses when facing urgency. PET data indicate that this modulation engage the left parasagittal cerebellum, the left lateral cerebellum and the SMC bilaterally. These key findings are discussed below.

**Behavioral data**

This study shows that healthy subjects can: (1) exceed their self-determined maximal movement speed in the context of
externally-driven conditions; (2) exceed their externally-driven maximal response speed in the context of temporally-pressing (or urgent) situations. These results confirm the observations of previous experiments showing that the amount of energy that the motor system is willing to spend is set to a submaximal threshold in the absence of explicit constraints. For instance, in sport activities, it is known that naive subjects do not jump as far or as high as they can when they try to jump as far or as high as possible. These subjects substantially improve their 'best' performance when a mark is put on the ground or in the air (for a review see Desmurget, 2006). Similarly, healthy subjects do not move as fast as they can when required to do so. They can substantially exceed their self-determined maximal movement speed to catch a falling ball (Ballanger et al., 2006). It is plausible that a submaximal threshold of muscular energy is set unconsciously to decrease the risk of tiredness or skeleto-motor injuries. This submaximal threshold would be optimal in the absence of an external challenge. However, in the context of highly challenging tasks a submaximal threshold becomes insufficient. When this happens, the subjects face a simple choice: raise the self-determined threshold or miss.

Influence of movement context on rCBF changes

Self-initiated responses and the prefrontal cortex

To explore the specific activation associated with the production of internally generated movements, we contrasted the pattern of brain activity during the Self-initiated and Externally-cued conditions. The main response was found in the prefrontal cortex (PC), including Brodmann areas 8, 44 and 47. This result is in line with previous observations suggesting that PC plays a crucial role in the preparation, planning and selection of voluntary movement (Deiber et al., 1991; Jahanshahi et al., 1995; Jenkins et al., 2000).

Table 1
Significant rCBF increases in controls

<table>
<thead>
<tr>
<th>Areas</th>
<th>Left/Right</th>
<th>x</th>
<th>y</th>
<th>z</th>
<th>Z score</th>
<th>Cluster (k)</th>
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</thead>
<tbody>
<tr>
<td>SI&gt;EC</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Superior frontal cortex (BA 8)</td>
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<tr>
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<tr>
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<td>53</td>
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<tr>
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<td>Cerebellum (Vermis)</td>
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<tr>
<td>R</td>
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<td>−36</td>
<td>58</td>
<td>3.33</td>
<td>47</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: SI: self-initiated; EC: externally cued; TP: temporally pressing; L: left; R: right.

$p_{uncorr}<0.001.$

Fig. 3. (A) Increased rCBF in the cerebellum in the Temporally-pressing condition compared to the Externally-cued conditions. (B) Increased rCBF in the ipsilateral primary motor cortex in the Temporally-pressing condition compared to the Externally-cued conditions. Data are superimposed on transverse sections of a single subject brain MRI from SPM99. $p<0.001$ uncorrected.
reaction time and movement duration

We found a greater activation in the contralateral sensorimotor cortex (SMC) than in the ipsilateral SMC. This finding agrees with previous studies where pallidal activity was found to be positively correlated with movement speed (Turner et al., 1998, 2003). On the other hand, Taniwaki et al. (2003) demonstrated that BG shows positive correlation with movement rate only in the self-initiated movements, but not in the externally-triggered movements, which is in agreement with our findings.

Finally, covariation analysis also confirmed the role of the ACC in the control of movement speed and reaction time, which is in line with other functional imaging studies (Naito et al., 2000; Kudo et al., 2004).

Temporally-pressing responses and recruitment of the anterior (i.e. uncrossed) corticospinal tract

To explore the specific activation related to urgency, we compared the Temporally-pressing to the Externally-cued conditions and showed that movements executed in the Temporally-pressing condition were associated with a dual activation in two structures that are known to be part of the neural networks that control movement velocity: the SMC (bilaterally) and the parasagittal cerebellum (on the left side). Evidence supporting the claim that the SMC is involved in the modulation of movement velocity has been summarized above. Arguments suggesting a critical involvement of the ipsilateral parasagittal cerebellum are manifold. In non-human primates, recording studies in the Purkinje cells of the cerebellum have shown that activity in the cerebellum covaries with the speed of movement (VanMeter et al., 1995; Blinkenberg et al., 1996; Rao et al., 1996; Schlaug et al., 1996; Sadato et al., 1997; Wexler et al., 1997; Turner et al., 1998).

The role of the SMC in the control of movement speed is also supported by the covariation analysis. As shown by this analysis, the activity of the contralateral motor cortex was negatively correlated with movement duration across all scans (i.e. it was positively correlated with movement speed). A similar association was found in the basal ganglia (BG) at the level of the lateral (motor) pallidum. This result is consistent with previous studies showing that activity in the BG is correlated with the speed/extent of arm movement (Turner et al., 1998, 2003), finger movement rate (Taniwaki et al., 2003) or speech volume (Liotti et al., 2003). In non-human primates, the movement-related discharge of single pallidal neurons is often correlated with the extent or velocity of movement (Georgopoulos et al., 1983; Turner and Anderson, 1997). Also, disruption of normal BG outflow by electrical stimulation, reversible inactivation, or permanent lesion affects the speed and/or the metrics of trained arm movements while preserving the directional accuracy of the movements (Hore and Villis, 1980; Horak and Anderson, 1984a,b; Mink and Thach, 1991; Kato and Kimura, 1992; Alamy et al., 1995; Inase et al., 1996; Turner et al., 2006). The same specificity is observed in BG pathologies such as PD (Berardelli et al., 2001; Desmurget et al., 2003). In light of the remarks above, one may wonder about the absence of activation in the BG for the Externally-cued versus Self-initiated contrast. In first instance one may hypothesize that this negative result reflects a false negative inference associated with the fact that velocity differences between the self-generated and externally-cued movements were not strong enough to allow reliable statistical detection at the selected threshold. In support to this view, the enhanced response in the contralateral SMC would reflect the decreased movement duration in Externally-cued movements compared to this view, the enhanced response in the contralateral SMC would reflect the decreased movement duration in Externally-cued conditions. We found a greater activation in the contralateral sensorimotor cortex (SMC) than in the ipsilateral SMC. This finding agrees with previous studies where pallidal activity was found to be positively correlated with movement speed (Turner et al., 1998, 2003). On the other hand, Taniwaki et al. (2003) demonstrated that BG shows positive correlation with movement rate only in the self-initiated movements, but not in the externally-triggered movements, which is in agreement with our findings.

Finally, covariation analysis also confirmed the role of the ACC in the control of movement speed and reaction time, which is in line with other functional imaging studies (Naito et al., 2000; Kudo et al., 2004).

### Table 2

Negative correlation of rCBF with movement parameters

<table>
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<tr>
<th>Areas</th>
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<td>39</td>
</tr>
<tr>
<td>SMA (BA 6)</td>
<td>R</td>
<td>−15</td>
<td>1</td>
<td>49</td>
<td>3.27</td>
<td>12</td>
</tr>
</tbody>
</table>

p uncorr<0.001.

Because this result has been widely discussed in the aforementioned studies, it will not be considered any further here.

### Externally-cued responses and the control of movement speed

To explore the specific activation associated with the production of externally triggered movements, we contrasted the pattern of brain activation during the Externally-cued and Self-initiated conditions. We found a greater activation in the contralateral sensorimotor cortex (SMC). This finding agrees with previous reports showing that externally-driven movements involve the same executive network as self-initiated movements (Jahanshahi et al., 1995; Jenkins et al., 2000; Cummington et al., 2002). According to this view, the enhanced response in the contralateral SMC would reflect the decreased movement duration in Externally-cued conditions, with respect to Self-initiated movements. In support to this hypothesis, studies have demonstrated that the SMC was a key element of the neural network that controls movement velocity. It is known, in particular, that neurons in the SMC encode information about movement speed/extent (Fu et al., 1993, 1995; Messier and Kalaska, 2000) or muscle force (Evarts, 1968; Cheney and Fetz, 1980; Kalaska et al., 1989) and that SMC activity, measured by functional imaging, is strongly influenced by the rate or speed of movement (VanMeter et al., 1995; Blinkenberg et al., 1996; Rao et al., 1996; Schlaug et al., 1996; Sadato et al., 1997; Wexler et al., 1997; Turner et al., 1998).

The role of the SMC in the control of movement speed is also supported by the covariation analysis. As shown by this analysis, the activity of the contralateral motor cortex was negatively correlated with movement duration across all scans (i.e. it was positively correlated with movement speed). A similar association was found in the basal ganglia (BG) at the level of the lateral (motor) pallidum. This result is consistent with previous studies showing that activity in the BG is correlated with the speed/extent of arm movement (Turner et al., 1998, 2003), finger movement rate (Taniwaki et al., 2003) or speech volume (Liotti et al., 2003). In non-human primates, the movement-related discharge of single pallidal neurons is often correlated with the extent or velocity of movement (Georgopoulos et al., 1983; Turner and Anderson, 1997). Also, disruption of normal BG outflow by electrical
cells of the lobules V and VI have demonstrated relations of single unit discharge to motor parameters such as velocity (Coltz et al., 1999) and extent (Fu et al., 1997). In humans, imaging studies have shown velocity related activity in the vermis and the lobule VI of the cerebellum ipsilateral to the moving arm (Turner et al., 1998, 2003). All these results fit well with the demonstration that patients with cerebellar lesions or dysfunctions have often longer movement duration than healthy subjects (Meyer-Lohmann et al., 1977; Trouche and Beaubaton, 1980; Bonnefoi-Kyriacou et al., 1995; Straube et al., 1997; Badescu and Lalonde, 2001).

If cerebellar activations associated with changes in movement velocity are mainly ipsilateral and if SMC responses are contralateral, two questions arise: (1) why did we fail to identify any activation in the ipsilateral parasagittal cerebellum in the covariation analyses and in the Externally-cued versus Temporally-pressing and the Externally-cued versus Self-initiated contrasts; (2) What is the meaning of the concomitant activation of the contralateral parasagittal cerebellum and ipsilateral SMC?

Regarding the first question, it is very plausible that our failure reflects a false negative inference related to the fact that velocity differences between the self-generated and externally-cued movements were not strong enough to allow statistical detection at the selected threshold. Changes in movement velocity were much more substantial in previous imaging studies where cerebellar activity was found to covary with movement velocity (Turner et al., 1998, 2003). Of course, one could argue that velocity differences were not big either for the left (contralateral) cerebellum, which showed a clear response. In this case however, metabolic activity was not subtracted across condition. Indeed, the left cerebellar activation was specific to urgency. This specificity explains also the absence of velocity-related activations in the contralateral cerebellum and ipsilateral SMC in the covariation analysis.

The remarks above lead us to our second question and to the existence of a left cerebellum — right SMC network in urgent situations. As previously emphasized, the motor network that controls movement velocity involves the ipsilateral parasagittal cerebellum, contralateral BG and contralateral SMC (Turner et al., 1998, 2003). When this network is lesioned, which happens in various neurological pathologies, such as stroke or PD, recruitment of the contralesional motor network is often observed. In PD, this activation has clearly been associated with bradykinesia. In the early-stage of the pathology, for instance, the patients with predominant unilateral motor signs, exhibit a clear activation in the contralesional primary motor cortex for the “akinetic” hand, but not for the “non akinetic” hand (Thobois et al., 2000). Stroke patients, often exhibit the same type of compensations (Desmurget et al., 2004) for a review). However, in these patients, the contralesional recruitment tends to vanish over time, as the motor system regains function (Marshall et al., 2000; Calautti et al., 2001). Contralesional persistence is, in fact, a predictor of poor recovery, which suggests that the contribution of contralesional network becomes important, as a last resort strategy, when the size of the infarct prevents ipsilesional recovery (Kolb, 1995; Hallett, 2001; Johansen-Berg et al., 2002; Ward, 2004; Desmurget et al., 2006). From these data, it is tempting to speculate that the anterior corticospinal tract (i.e. uncrossed) (ipsilateral BG and SMC) can be successfully solicited to compensate for a shortfall of the lateral (i.e. crossed) corticospinal tract (contralateral BG and SMC). In the context of the present study, this means that the anterior corticospinal tract is activated when the lateral corticospinal tract is no longer able to cope with the temporal requirements of the task. The activation of the left (contralateral to the movement) cerebellar hemisphere can also be viewed in this perspective, as the left cerebellum sends projections to the SMC via the crossed dentatothalamic pathway. In other words, in urgent situations, the motor system “boosts” its ability to respond quickly by soliciting the normally silent direct pathway. This recruitment allows the system to exceed the maximal velocity that can be reached with the sole indirect pathway.

Although cerebellar lobule VI is clearly linked to hand movement velocity, it has also been associated with saccadic activity (Noda and Fujikado, 1987; Ohtsuka and Noda, 1995; Desmurget et al., 1998, 2000). It is thus possible that the parasagittal cerebellar activation observed in the Temporally-pressing versus Externally-cued contrast is associated with difference in oculomotor activity across these conditions. This hypothesis is nevertheless very unlikely for two reasons. First, fixation was rarely broken and behavioral observations have failed to reveal any systematic differences in the number or characteristics of horizontal oculomotor responses across conditions. Second, oculomotor-related activity in the cerebellum has been shown to be bilateral (Goffart et al., 2004) and more extended along the caudal axis (lobule VII) than observed in our study (Noda and Fujikado, 1987; Ohtsuka and Noda, 1995; Desmurget et al., 1998, 2000). Note that the oculomotor hypothesis would be all the more likely to predict a bilateral cerebellar response that saccades were not constrained along one direction, when they did occur.

**Temporally-pressing responses and awareness**

Although we have no explicit measure of attentional modulation, this factor is expected to affect the movement reaction time. In the Results section, we have established that changes in movement velocity and acceleration could not fully account for the reduction in reaction time observed in the Temporally-pressing condition. This suggests that part of the change observed for RT between the Temporally-pressing and Externally-cued conditions could be associated with attentional factors.

When the Temporally-pressing and Externally-cued conditions were compared, a response was found in the left lateral cerebellum. Imaging studies in human have suggested that this region is involved in motor awareness. For instance, Allen et al. (1997) reported that the left cerebellar hemisphere was specifically involved in attentional processes. In a more recent study, Critchley et al. (2000) provided evidence that neural activity in the left lateral cerebellum covaried with the skin conductance response, a variable that is known to correlate with the level of alertness and emotional arousal. A compatible but more direct observation was also provided by Mazoyer et al. (2002) who showed that metabolic activity tended to increase in the same left cerebellar region as a function of the attentional load.

Covert orienting has also been reported to activate similar regions of the cerebellum bilaterally when the task involved attentional displacements to either side (Rosen et al., 1999; Nobre et al., 2000). Although it is unclear whether these processes are lateralized, activity in the left cerebellum in the Temporally-pressing condition could reflect covert orienting towards the ball-stopping device. This hypothesis is however weakened by the absence of activation in the parietal cortex, in contrast with the aforementioned studies. Nevertheless, in light of this set of findings, we suggest that part of the left cerebellar activation could be related to attentional modulation.
In addition, as mentioned before the RT is negatively correlated with the ACC activation. This could be interpreted in light of the role of this region in attentional processes (Carter et al., 1999; Turkens and Swick, 1999; Mazoyer et al., 2002). Indeed, several lines of evidence suggest that the ACC is involved in attentional control and conflict monitoring (Carter et al., 1999). However, in the present study no difference of ACC perfusion was noted between EC and TP conditions, which suggests that this function of the ACC is not specifically involved in motor response in TP situation but rather participates to motor control whatever the condition.

Conclusive remarks

The main message that emerges from the present study can be summarized as follows. The amount of energy that the motor system is willing to spend is set to a submaximal threshold in the absence of explicit constraints. When the task becomes more challenging, this threshold becomes inadequate. In order to respond more quickly, the nervous system starts optimizing the response of the lateral corticospinal tract (contralateral BG and SMC) and ipsilateral cerebellum. When this pathway is no longer able to cope with the demands, the anterior corticospinal tract (ipsilateral BG and SMC) and the contralateral cerebellum are solicited. This strategy is akin to the strategy observed in brain lesioned patients who tend to activate the contralesional motor networks following a degenerative (PD) or acute injury (stroke). It is also compatible with the hypothesis that our ability to modulate movement speed in urgent situations is not a hallmark of PD and thus not a basal ganglia-related deficit (Ballanger et al., 2006).

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