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Perception of self-generated action in schizophrenia

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Introduction. Self-generated actions involve central processes of sensorimotor integration that continuously monitor sensory inputs to ensure that motor outputs are congruent with our intentions. This mechanism works automatically in normal conditions but becomes conscious whenever a mismatch happens during the execution of action between expected and current sensorimotor reafferences. It is now admitted in the literature that sensorimotor processes as well as the ability to predict the consequences of our own actions imply the existence of a forward model of action, which is based on efference copies. Recently, it has been proposed that positive symptoms expressed by schizophrenic patients, such as delusions of control or thought insertions, arise because of a deficiency in this forward model, and more particularly, because of a lack of awareness of certain aspects of motor control derived from such an internal model. Method. To test further this hypothesis, 19 schizophrenic patients (10 with and 9 without Schneiderian symptoms) and 19 control subjects performed a visuo-motor conflict task and had verbally to report the felt position of their hand at the end of each trial. Results. Under this experimental procedure, schizophrenic patients—whatever their clinical phenotype—failed to switch to a conscious representation of their hand movements, and then consequently to maintain their level of performance for the sensorimotor adjustment in comparison with controls. Conclusion. Our findings suggest two facts. First, that a functional monitoring of action, based on a forward
model, is partially preserved in schizophrenic patients. Second, that the primary cognitive deficit associated with positive symptoms cannot entirely be reduced to an impairment in the conscious motor control representation.

Before its execution, any voluntary action implies the mental representation of the goal and of the environmental constraints, as well as the internal and external states of our body. Thus, the representation of an action would contain both the specification of the means necessary to allow its realisation and the anticipation of these expected effects on the external world, so that it can be proposed that we in fact ‘actualize’ our projects more than merely execute them.

The ability to predict the consequences of one’s own movements even before they are carried out is a fundamental aspect of motor control of which neurocognitive determinants remain largely debated. In order to give an account of this ability, the notion of ‘internal model’, originated from computational studies, is now usually employed. Accordingly, the concept of ‘internal model’ is used to set up systems that are able to simulate or mimic the effects of an action of the machine on the external environment, the effects of the external changes resulting from that action on the machine itself, and so on (Wolpert, Ghahramani, & Jordan, 1995; Wolpert & Ghahramani, 2000). Applied to the study of human behaviour, this concept implies a continuous integration, through a forward model and by a recursive process, between the reafferent sensory inflow and the efference copy of motor outflow to evaluate the consequence of the motor commands sent to the effector. Although the relative importance of the feedforward and the feedback mechanisms continues to be discussed, this integrative model of motor control in which these two processes are permanently combined in the nervous system is now supported by several authors (Blakemore, Goodbody, & Wolpert, 1998; Desmurget & Grafton, 2000; Kawato, 1999; Nelson, 1996).

Another recurrent question, which is intrinsic to the question of motor control, concerns the nature of the mental processes implied in the conscious monitoring of our acts. Indeed, recognising that our actions are congruent with our intentions is one of the fundamental aspects of self-consciousness. Nevertheless, several studies in the literature have shown that normal subjects are poorly aware of many aspects of their own behaviour, even when they act intentionally. For example, even if the body orientation and configuration are precisely represented in the central nervous system, we have a poor subjective sense of limb position and/or motion (Coello, Orliaguet, Prablanc, & Barraud, 1997; Fourneret & Jeannerod, 1998; van Beers, Sittig, & Denier van der Gon, 1998). The weakness of action consciousness raises a crucial problem: how can we identify ourselves as the authors of our own actions when at the same time we do not know exactly what we do?
Recently, Frith (1992; Frith, Blakemore, & Wolpert, 2000) has developed an interesting theory according to which the capacity to identify ourselves as the source of our own actions would be directly related to our faculty to have a conscious access to the anticipated representation of our acts. This theory is mainly constructed from his previous works on schizophrenics patients (Frith, 1987; Frith & Done, 1989) presenting experiences of alien control, i.e., including symptoms initially described by Kurt Schneider (1959) such as delusions of control, auditory hallucinations, thought insertion, and passivity phenomena. According to Frith, the disruption takes place not only at the level of the generation of a forward model (based on the production and storage of the efference copy) but also at the level of the representation (and its access to consciousness) of the predicted state of the system (Frith et al., 2000). In other words, even though the “willed” actions of these patients are being correctly carried out, because they are deprived of the awareness of this state, they do not feel in control and consequently attribute their intentions to an external agent.

Despite the inherent problems in attempting to conduct research into such hypothetical constructs as willed intentions and self-monitoring, Frith’s model has received mixed support from experimental verification. While the reality of an action attribution mechanism dysfunction is largely confirmed among patients suffering from Schneiderian symptoms (Bredion, Amador, David, Malaspina, Sharif, & Gorman, 2000; Franck et al., 2001; Keefe, Arnold, Bayen, & Harvey, 1999; Stirling, Hellewell, & Quraishi, 1998), the assumption of a direct relation between this disruption and a possible deficit in the production of efference copies—which could play a role in the labelling of self-produced actions—was recently called into question (Fourneret, Franck, Slachevsky, & Jeannerod, 2001; Kopp & Rist, 1994; Trillenberg, Heide, Junghanns, Blakenburg, Arolt, & Kömpf, 1998). Indeed, the automatic control, the feeling of being aware, and the self-monitoring of an action should be conceived as three distinct levels of representation, each depending on specific but complementary neural processes.

To test this hypothesis further we reproduced in a group of schizophrenic patients with and without Schneiderian symptoms, an experiment previously proposed by Slachevsky, Pillon, Fourneret, Pradat-Diehl, Jeannerod, and Dubois (2001) for studying the relationship between action monitoring and action consciousness. The experimental design was based on a conflict—created by an angular bias of increasing amplitude—between what subjects actually do with their hand and what they see from the displacement of their hand. More specifically we were interested in the schizophrenic patients’ ability to become aware not only of the mismatch but especially of the hand correction necessary for improving the visuo-motor adjustment with respect to the bias by an anticipation process.
METHOD

Subjects

A total of 19 right-handed (Edinburgh Inventory; Oldfield, 1971) patients with schizophrenia (age range 20–49 years; mean = 30.57) from the Vinatier Hospital in Lyon were selected for this study. All met the DSM-IV criteria for schizophrenia as assessed by the Mini International Neuropsychiatric Interview (MINI) and were clinically stable with no change in medication for more than 1 month prior to entering the experiment. A total of 19 healthy subjects were also selected through advertising addressed to students and technical staff of ISC and hospital institutions. Control subjects were free from both current DSM-IV Axis I psychiatric diseases and schizophrenia spectrum personality disorder. Furthermore, none of them had a family history of psychopathology. Exclusion criteria for all subjects included possible brain damage, neurological or medical illness that could affect the nervous system, alcohol or drugs abuse, and substance dependence. There were no significant differences between schizophrenic and control groups for age and sex, but the normal controls had received significantly more education; \( \chi^2 (df = 1) ; p < .03 \). Table 1 lists demographic and clinical characteristics for all subjects. Patients as well as control subjects

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Demographic and clinical characteristics of controls and patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Controls (n = 19) (Mean ± SD)</td>
</tr>
<tr>
<td>Demographic variables</td>
<td>[</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>34.52 ± 8.48</td>
</tr>
<tr>
<td>Sex (Male/Female)</td>
<td>12/7</td>
</tr>
<tr>
<td>Education (yrs)</td>
<td>12 ± 2.47</td>
</tr>
</tbody>
</table>

Estimated illness duration (yrs)

- 7.53 ± 5.94

Neuropsychological evaluations

| Token test         | –                  | 32.92 ± 1.60                      | –     |
| IQ (Raven PM 47)   | –                  | 80 ± 0                            | –     |

Medication

| Antipsychotic (mg/day CPZ Eq) | –                  | 496.57 ± 350.64                   | –     |
| Simpson Angus Scale (SAS)    | –                  | 5.05 ± 5.25                       | –     |

SAPS/SANS scores

| SAPS total score | –                  | 33.78 ± 21.02                     | –     |
| SANS total score | –                  | 47.73 ± 19.15                     | –     |

\( ^a \) Mann-Whitney comparisons between schizophrenics and controls.
\( ^b \) Chi-square (df = 1); p significantly below .05 level.
were naive about the purpose of the experiment. Finally, all subjects gave their informed consent for participating to this study.

**Diagnostic criteria**

Psychiatric diagnoses of patients were established in accordance with their referent therapist. Schizophrenic symptoms were assessed by the Scale for Assessment of Positive Symptoms (SAPS; Andreasen, 1984) and the Scale for Assessment for Negative Symptoms (SANS; Andreasen, 1983). The mean SAPS score was 33.78 (SD = 21.02) and the mean SANS score was 47.73 (SD = 19.15). In addition, a Schneiderian sub-score was calculated by adding seven items (item 2: voices commenting; item 3: voices conversing; item 15: delusions of being controlled; item 16: delusions of mind reading; item 17: thought broadcasting; item 18: thought insertion; item 19: thought withdrawal) from the SAPS. Each item was quoted from 0 to 5, according to the severity of clinical symptoms, for a total of 35. To assess the nature of this sub-scale score, we calculated a Spearman’s correlation score between its two parts (verbal hallucinations: items 2 and 3 / influence syndrome: items 15–19) which confirmed the homogeneity of this two group of symptoms ($R = 0.72 ; p < .0004$). This sub-scale score enabled us to classify the patients in two groups: S1 and S0, according to the presence or absence of Schneiderian symptoms, respectively. Ten patients had a sub-score superior to 3 and were classified as “Schneiderian” (mean score = 12.80; SD = ± 7.33). For the remaining nine “non-Schneiderian” patients the mean value was 0.88 (SD = ± 1.26). All patients were receiving antipsychotic and medication [mean dose = 496.57 ± 350.64 mg of chlorpromazine or equivalent]. The 10 patients reporting Schneiderian symptoms did not differ from the rest of the schizophrenic group regarding age, illness duration, and neuropsychological variables. Nevertheless, these patients were significantly more medicated than the other non-Schneiderian patients ($U = 10.5 ; Z = −2.81 ; p < .004$) but without that implying more extrapyramidal side-effects of type (mean score of the Simpson Angus Scale for the schizophrenic group = 5.05 ± 5.25) (Simpson & Angus, 1975; see Table 2).

**Apparatus**

The experimental device consisted of a 30 × 45 cm graphic tablet placed on a regular table, and connected to a computer (see Figure 1). The computer screen was placed horizontally at 62 cm above the graphic tablet. A circular mirror (35 cm in diameter) was placed horizontally halfway between the screen and the tablet. The subjects sat on a chair facing the table and held in their right hand a stylus connected to the graphic tablet computer. The right hand was placed on the tablet, below the mirror, and hidden from the subject. When tracing a line on the tablet, the subjects could see through the mirror a red line appearing on the
computer screen in exact coincidence with the displacements of the tip of the stylus on the tablet. The output of the graphic tablet was processed by the computer using a simple algorithm for adding a linear directional bias. When the bias was set to the right, e.g., at 15°, a line traced in the sagittal direction on the tablet appeared to the subject to deviate to the right at an identical angle (see Figure 2).

**Procedure**

At the beginning of each trial, the subjects placed the stylus tip on the starting point located on the tablet close to the body midline (a green point, 3 × 4 mm). They were instructed to reach a yellow target (3 × 4 mm) located on the sagittal axis at 22 cm from the starting point. They had to draw a continuous line as straight as possible, keeping the hand in contact with the graphic tablet. The duration of each trial was limited to 5.5 seconds. After the trial had been completed or after this delay, the screen was blanked and a new trial started when the subjects placed the stylus on the starting position.

### TABLE 2

Demographic and clinical characteristics of the two groups of schizophrenic patients (with and without Schneiderian symptoms)

<table>
<thead>
<tr>
<th></th>
<th>Schizophrenic group with Schneiderian symptoms (n = 10)</th>
<th>Schizophrenic group without Schneiderian symptoms (n = 9)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td></td>
</tr>
<tr>
<td><strong>Demographic variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>31 ± 10</td>
<td>30 ± 9.3</td>
<td>n.s.</td>
</tr>
<tr>
<td>Sex (Male/Female)</td>
<td>7/3</td>
<td>7/2</td>
<td>n.s.</td>
</tr>
<tr>
<td>Education (yrs)</td>
<td>11 ± 2.6</td>
<td>9.6 ± 1.6</td>
<td>n.s.</td>
</tr>
<tr>
<td><strong>Estimated illness duration (yrs)</strong></td>
<td>8.3 ± 6.4</td>
<td>6.6 ± 5.6</td>
<td>n.s.</td>
</tr>
<tr>
<td><strong>Neuropsychological evaluations</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Token test</td>
<td>32.7 ± 1.8</td>
<td>33.2 ± 1.3</td>
<td>n.s.</td>
</tr>
<tr>
<td>IQ (Raven PM 47)</td>
<td>80 ± 0</td>
<td>80 ± 0</td>
<td>n.s.</td>
</tr>
<tr>
<td><strong>Medication</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antipsychotic (mg/day CPZ Eq)</td>
<td>689.5 ± 385.6</td>
<td>282.2 ± 105.6</td>
<td>&lt;.004</td>
</tr>
<tr>
<td>Simpson Angus Scale (SAS)</td>
<td>3.7 ± 5.6</td>
<td>6.6 ± 4.7</td>
<td>n.s.</td>
</tr>
<tr>
<td><strong>SAPS/SANS scores</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAPS total score</td>
<td>47 ± 15</td>
<td>19.1 ± 16.8</td>
<td>&lt;.003</td>
</tr>
<tr>
<td>SANS total score</td>
<td>50.7 ± 19.8</td>
<td>44.4 ± 19.10</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

*a* Mann-Whitney comparisons between schizophrenic groups

bChi-square (df = 1); p significantly below .05 level.
Figure 1. Experimental set-up. Upper part: the output of the graphic tablet, displayed on the computer screen, is seen by the subject through the mirror placed above his/her hand. Lower part: subject’s view of the computer screen seen in the mirror.
Figure 2. Graphic representation of one trial (12th) for one control subject who was aware of his manual correction. The heavy line represents the visual reafference as seen by the subject during the trajectory. The light line indicates the hand trajectory—unseen by the subject—on the digitiser tablet. The dashed line represents the amplitude and the direction of the bias. The distance separating the target from the starting point was 222 mm.
Subjects first ran 10 practice trials with no bias. In the experimental session, 20 biased trials were performed. The amplitude of the bias increased from 1° to 20° to the right, increasing by 1° at each new trial. The instruction given to the subjects was to trace a line as straight as possible, to be able to reach the visual target. In order to do so, the subjects had to compensate the visuo-motor discordance and thus to trace a line with a deviation to the left roughly equal to the angular bias. At the end of each trial, the subjects were asked whether the line they had traced was the same as the line they had seen. The angle at which they became aware of the conflict (i.e., when they answered ‘‘no’’) was recorded. In order to estimate also the motor performance they thought they had carried out, an abacus with lines drawn on a card at different angles (from –30 to +30 degrees) had been placed in front of them. These lines were numbered from 1 to 31. Lines 1–15 deviated to the left with respect to the axis of the card, lines 17–31 to the right. The line numbered 16 was aligned with the card axis (see the insert on Figure 5). The question to the subjects was: ‘‘According to your own impression, which line corresponds to the actual trajectory drawn by your hand’’. The verbal response had to be given by reading the corresponding number on the card.

Data analysis

Sensori motor adjustment to the bias was evaluated by computing the square of the Root Mean Squared Error (RMSE) or $a^2$:

$$a^2 = \frac{1}{x_f - x_i} \int_{x_i}^{x_f} (f_s - f_r)^2 \, dx$$

where:

- $x_i$ is the initial position of the stylus tip,
- $x_f$ the final position of the stylus tip,
- $f_s$ the real trajectory realised by the subject, and
- $f_r$ the ideal trajectory to correct the deviation.

This ratio corresponds to the average deviation of the trajectory made by the subject from the theoretical line that would perfectly compensate for the bias (Cunningham, 1989). In other terms, the more the score of this ratio approaches 0, the more the trajectory can be considered as sagittal. In addition, this measure has the advantage of avoiding cancellation between right and left side deviations from the ideal line. The threshold of conscious perception for the bias (angle at which the subjects explicitly reported a feeling of incongruity between what they did and what they saw) and consecutively the verbal responses for each subject were recorded and analysed. Because of variance heterogeneity (Levene’s Test), analyses of significant effects between the three groups (S0, S1, and C) for the
sensori motor adjustment ($a^2$) and for the distribution of verbal responses were run with non-parametric tests (Kruskal-Wallis ANOVA by ranks).

RESULTS

Overall performance

All subjects performed the session without difficulty. Only the trials for which the subjects had covered more than 90% of the distance separating the two targets were analysed. The number of valid trials was less for the schizophrenic patients ($N = 328$) than for the control group ($N = 373$) $\chi^2 = 36.09$ (df = 1); $p < .00001$.

A Kruskal-Wallis ANOVA with the percentage of the distance covered between the two targets showed no significant difference between the controls and the schizophrenic patients, $H(1; N = 701) = 0.21$; $p < .64$. This difference did not improve over successive trials.

Visuo-motor adaptation

The three groups significantly differed for the ability to compensate for the deviation, $H(2; N = 701) = 79.26$; $p < .00001$, schizophrenic patients being less efficient than controls. Regarding the change over time of the median value of $a^2$ across trials, one observes that the ability of the patients to correct the perturbation declines strongly as the angular bias increases, compared with the control subjects (see Figure 3). This effect is clearly visible after 10 degrees of perturbation. Beyond this angle, whereas the performance of the control subjects tended to be stationary in spite of the increase in angular bias, $H(9; N = 186) = 7.58$; $p < .57$, that of the patients continued to degrade significantly, $H(9; N = 159) = 22.61$; $p < .007$, with no difference between the two schizophrenic sub-groups, $H(1; N = 159) = 0.86$; $p < .35$.

Awareness of the visuo-motor conflict

Only two subjects out of the three groups (one control and one patient belonging to the group S1) did not become aware of the visuo-motor conflict.

The threshold for conscious perception of the angular deviation was significantly different between the three groups, $H(2; N = 36) = 8.28$; $p < .01$. The median value was $6.50^\circ$ for the controls and $13^\circ$ for the schizophrenic patients (respectively $13^\circ$ for group S0 and $11^\circ$ for group S1) (see Figure 4).

Figure 5 displays the median value of the readings of the abacus for each group according to the increase in angular bias. The readings of the control subjects differ from those of the schizophrenic patients, $H(2; N =701) = 136.12$; $p < .00001$. Whereas control subjects were roughly able to perceive the effective trajectory of their hand (directed to the left), they largely underestimated the amplitude of the angle compared to the real value of the correction carried out
Figure 3. Visuomotor adaptation to the conflict between what the subject does and what the subject sees as expressed by the change of $a^2$ (the square of the Root Mean Squared Error) as a function of successive trials for the two groups of schizophrenic patients [with (S1) and without (S0) Schneiderian symptoms] and the control subjects.

Figure 4. Threshold for the conscious perception of the bias for the three sub-groups.
The purpose of the present experiment was to investigate the monitoring of action and its conscious accessibility among patients with schizophrenia and healthy controls in a visuo-motor conflict task. The major finding was twofold: the schizophrenic patients showed weaker ability than controls to explicitly detect visuo-motor mismatch, especially for biases of small amplitudes;
concerning their capacity for sensorimotor adjustment, the schizophrenic patients were overall less efficient than control subjects in compensating for angular deviation, in particular for large biases and even if they became aware of the deviation. The worse motor performances realised by the schizophrenic patients, whatever their clinical phenotype (with or without Schneiderian symptoms), seem to be the consequence of a failure to have a reliable conscious access to the relevant information concerning the effective trajectory of their movements. This results are complementary with respect to previous results obtained with the same patients, but in a different task. When exposed to a constant bias (15°), over successive trials, they were able to learn how to compensate for this bias, even if visual control of the motor output was partially blocked. However, unlike normal controls who were all able to report their compensatory strategy, only 9/19 were able to do so (Fournieret et al., 2001).

This type of impairment in our patients cannot be explained by the presence of a central motor system deficit or the side-effects of neuroleptic medication due to the weak mean score on the Simpson-Angus scale for extrapyramidal symptoms. In addition, we could find no information in the literature on possible effects of medication on tasks similar to those in the present work, or on impairment in self-monitoring (Stirling et al., 1998). Thus, the poor correction performances shown by schizophrenic subjects in our experiment are more likely to reflect a disturbance of the cognitive aspects of action processing, including the perceptual awareness of one’s own actions, and perhaps at a higher level, of the sense of agency, as supported by several recent works (Daprat et al., 1997; Franck et al., 2001; Georgieff & Jeannerod, 1998).

Our experimental paradigm involves at least two stages. First, in accordance with the instruction, the subject had to program a specific movement, a process that presumably involves setting up internal representations of the expected visual and kinaesthetic feedback that this action will subsequently generate (Desmurget & Grafton, 2000; Wolpert et al., 1995). Second, the subject had to judge whether the movement displayed in the mirror corresponded to the requested movement, that is, to the final result intended by the action (i.e., to trace a line straight ahead to reach the visual target). In order to do so, the expected visual feedback—derived from the “forward model” described by computational studies (Wolpert & Ghahramani, 2000)—had to be registered and compared with the actual visual feedback. In addition, the reafferent visual information also had to be compared with the kinaesthetic feedback (here, only the proprioceptive information, because the cutaneous information was prevented) in order to determine whether the movement displayed in the mirror matched what the subject’s hand was actually doing. Whereas this latter level clearly belongs to the category of a conscious decision, the former can largely function implicitly.

It is usually accepted that the selection of a spatial target, the intention to grasp it, and the execution of the action itself, mainly imply perceptual-motor
integration processes, which involve activation of the posterior parietal cortex (PPC) (Sakata & Taira, 1994; Stein, 1996). Besides the role of the PPC in planning processes, most authors acknowledge that this area may also be involved in “on-line” movement correction (Clower et al., 1996; Rushworth, Johansen-Berg, & Young, 1998). According to this view, the PPC could function as a “neural comparator”, combining proprioceptive signals from the somatosensory areas and efference copy signals from the motor regions to provide, in case of mismatch, a current error signal that is used by the motor centres to update the ongoing trajectory. In fact, given that the PPC also has access to the target position—encoded in a body-centred frame of reference—this structure seems to be specifically involved both in building an internal representation of instantaneous hand location and in computing the dynamic motor errors (Desmurget et al., 1999; Wolpert & Ghahramani, 2000). In most daily actions, these “on-line” movement corrections are achieved automatically, without any conscious access to the physical parameters of actions. Nevertheless in case of unfamiliar situations, the subject may have to shift to another explicit effortful operating mode in order to optimise his or her capacities for adaptation. In other words, the subject will produce overt responses bearing on predictions, estimations, and comparisons. Consequently, awareness of incongruous events is a preliminary and necessary condition for substituting implicit processes by a more effective strategy of control (Jeannerod, 1997; Shallice, 1988; Willingham, 1998). Applied to our experiment, it implies that subjects must become aware of the mismatch between the actual kinaesthetic feedback and the predicted feedback based on the intention of action (to trace a line as straight as possible). This conscious perception seems to be supported by the frontal cortex (Slachevsky et al., 2001) and more specifically by an activation of the right lateral prefrontal area (Fink et al., 1999).

However, the lack of awareness of the visuo-motor conflict, expressed by schizophrenic patients, is not sufficient by itself to explain their poor adaptation when the angular deviation increases. In fact, as Figure 5 shows, what they lacked by comparison with controls is the ability to have conscious access to the effective representation of their hand position. More exactly, they behaved as if the visual information was “singled out” to the detriment of the proprioceptive information, constituting therefore the unique information available for conscious decision. As the apparent result of their gesture was roughly in conformity with the instruction (trace a visual line straight ahead), the patients inferred that they had produced the correct movement in spite of the visuo-motor discordance. By contrast, it is clear that control subjects based their responses on the “reading” of the proprioceptive signal. The distribution of their verbal answers indicates that they were able to consciously monitor their hand trajectory in spite of contradictory evidence from other signals (the visual feedback and endogenous signals arising from the motor command to trace a
line straight ahead). In addition, this interpretation is reinforced by the negative correlation found between the pattern of the answers and the angular bias increase, which confirms that normal subjects tended to give verbal responses in the opposite direction to the perturbation and consequently in the direction in which the hand had effectively moved. Concerning the schizophrenic patients, even if they based their responses preferentially on the visual feedback, the positive correlation also stresses the fact that they did not monitor only this exteroceptive information. A tempting explanation could be that, although they were unaware of the proprioceptive information, they paid greater attention to endogenous signals related to the central level of movement generation. Thus, in our experiment where the hand had to be gradually ‘‘pulled’’ away from the increased bias, we suppose that the schizophrenic patients may have monitored the resistance exerted by this external force against the effort that they said they felt to move in the desired direction.

How can we now integrate these results with the current knowledge about bodily awareness? By this, we refer mainly to the concept of ‘‘body image’’ understood as an intentional representation of the body, as opposed to the ‘‘body schema’’ which is subpersonal and does not refer explicitly to the self (see Gallagher, 1995). It is nowadays accepted in literature that corporeal consciousness and more generally mechanisms underlying awareness imply a large neural network where somatosensory, parietal, temporal, and frontal cortices play a crucial role (Courtney, Ungerleider, Keil, & Haxby, 1997; Frith & Dolan, 1996; Lumer, Friston, & Rees, 1998). In this light, our results suggest decomposition of goal-directed action into the following steps:

1. During the first part of the test, the subject is able to correct the visuo-motor conflict by an automatic and implicit mechanism of sensorimotor integration, which preferentially implies the superior parietal lobe (Wolpert, Goodbody, & Husain, 1998).

2. It is only after a certain degree of discordance that the frontal cortex is activated to control the process of visuo-motor transformations in order to maintain the performance level. In this case, the frontal lobes may amplify the strength of the kinaesthetic memory traces to allow for the mismatch perception (Slacheksky et al., 2001; Willingham, 1998).

3. As regards the incapacity of the schizophrenic patients in our task to indicate the correct direction of their hand, and considering the notion of hypofrontality usually found in most neuroimaging studies of schizophrenia (Andreasen et al., 1992; Spence, Hirsch, Brooks, & Grasby, 1998), we suggest that the prefrontal areas play a key role in the conscious access of the internal representation of hand position stored in the superior parietal lobe (Desmurget & Grafton, 2000; Wolpert et al., 1998). In other words, we propose that in the large cortical network underlying the conscious representations of our own body, the frontal
cortex is necessary to maintain the mental representations above a certain level of “activation” to permit their access to awareness.

CONCLUSION

Despite the broad cognitive and motor behavioural disorders associated with schizophrenia, the present study suggests at least two points: (1) even if a lack of awareness of certain aspects of motor control can account for the poor effectiveness of willed actions in schizophrenia, this deficiency does not appear to be related to a failure in the central mechanism by which an internal “forward model” is generated from the intended sequence of motor commands, as suggested by Frith’s theory (Frith, 1992; Frith et al., 2000). In other words, schizophrenic patients do not seem to express a deficit in the production or the storage of efferent copy. (2) Moreover, and in this experimental paradigm, our results indicate that the impaired capacity of schizophrenic patients to have conscious access to the representation of their hand movement—whatever its cause—is not specific to those suffering from positive disorders. Thus, this fact seems to suggest that the functional relation evoked since Feinberg (1978) between deficit of the self-monitoring of action and the disorders of agency does not appear to be so direct.

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