

Why can't you tickle yourself?

Sarah-Jayne Blakemore,^{CA} Daniel Wolpert and Chris Frith

Wellcome Department of Cognitive Neurology, Institute of Neurology, University College London, 12 Queen Square, London WC1N 3BG, UK

^{CA}Corresponding Author

It is well known that you cannot tickle yourself. Here, we discuss the proposal that such attenuation of self-produced tactile stimulation is due to the sensory predictions made by an internal forward model of the motor system. A forward model predicts the sensory consequences of a movement based on the motor command. When a movement is self-produced, its sensory consequences can be accurately predicted, and this prediction can be used to attenuate the sensory effects of the movement. Studies are reviewed that demonstrate that as the discrepancy between predicted and actual sensory feedback increases during self-produced tactile stimulation there is a concomitant decrease in the level of sensory attenuation and

an increase in tickliness. Functional neuroimaging studies have demonstrated that this sensory attenuation might be mediated by somatosensory cortex and anterior cingulate cortex: these areas are activated less by a self-produced tactile stimulus than by the same stimulus when it is externally produced. Furthermore, evidence suggests that the cerebellum might be involved in generating the prediction of the sensory consequences of movement. Finally, recent evidence suggests that this predictive mechanism is abnormal in patients with auditory hallucinations and/or passivity experiences. *NeuroReport* 11:11–16 © 2000 Lippincott Williams & Wilkins.

Key words: fMRI; Forward models; Schizophrenia; Tickleing

INTRODUCTION

Detecting the consequences of our own actions: We can readily distinguish between sensations that are produced by our own movements and sensations that are caused by a change in the environment. This ability is important because it enables us to pick out stimuli that correspond to potentially biologically significant external events from stimuli that arise simply as a consequence of our own motor actions. It has been proposed that information about motor commands is used to distinguish the sensory consequences of our own actions from externally produced sensory stimuli [1–5], giving us the ability to monitor and recognise as our own, self-generated limb movements, touch, speech and thoughts [6]. This kind of mechanism can be used to maintain perceptual stability in the presence of self-produced movement. For example, during eye-movements an efference copy of the motor command is used to predict the effects of the movement [7–9]. In order to determine the location of an object relative to the head, its retinal location and the gaze direction must be known. As the eye muscles are thought to contain no sensory receptors used to determine the gaze direction, Helmholtz [7] proposed that the gaze direction is determined by predicting the eye location based on the efference copy of the motor command going to the eye muscles. Using this estimate of eye position together with the object's retinal location, the object's true position in space can be determined. When the eye is moved without using the eye muscles (for example by gently pressing on the eye lid

with the finger), the retinal location of objects changes, but the predicted eye position is not updated, leading to the perception that the world is moving. Prediction can also work in other sensory modalities to filter sensory information, attenuating the component that is due to self-movement (re-afference) from that due to changes in the outside world, and it is this use of prediction that forms the focus of this review. In order to generate sensory predictions, it is postulated that the central nervous system contains a central monitor [6] or internal 'forward model' [3,10,11].

Forward models mimic aspects of the external world and the motor system in order to capture the forward or causal relationship between actions and their outcomes [2,10,12] (Fig. 1). An efference copy of the motor command [8] is used to generate continuously predictions of the sensory consequences (or corollary discharge [9]) of the ongoing motor act. This prediction is then compared with the actual sensory feedback (re-afference) from the movement. Self-produced sensations can be correctly predicted on the basis of motor commands, and there will therefore be little or no sensory discrepancy resulting from the comparison between the predicted and actual sensory feedback. This accurate prediction can be used to attenuate the sensory effects of self-produced movement. In contrast, externally generated sensations are not associated with any efference copy and therefore cannot be predicted by the forward model. By removing or attenuating the component of sensory feedback that is due to self-produced movement it is possible to accentuate the feedback that is caused by

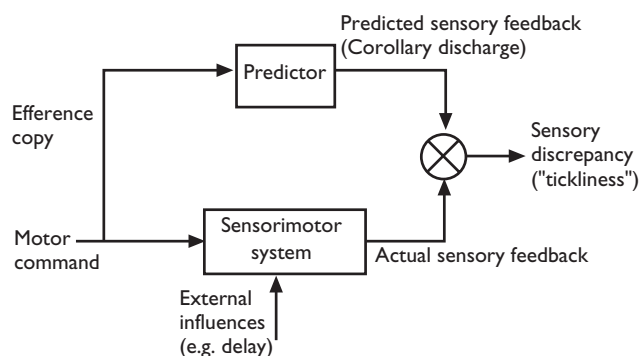


Fig. 1. A model for determining the sensory consequences of a movement. An internal forward model makes predictions of the sensory feedback based on the motor command. These predictions are then compared with the actual sensory feedback. Self-produced sensations can be correctly predicted on the basis of the motor command and are associated with little or no sensory discrepancy resulting from the comparison between predicted and actual sensory feedback. As the sensory discrepancy from this comparison increases (for example by increasing the delay or trajectory rotation between the movement and its sensory consequences) so does the likelihood that the sensation is externally produced. By using such a system it is possible to cancel out the effects on sensation induced by self-motion and thereby distinguish sensory events due to self-produced motion from the sensory feedback caused by the environment, such as contact with objects.

external effects. This process therefore filters incoming sensory information for perhaps the more relevant component of information.

Forward models in schizophrenia: Frith [6] proposed that a defect in this kind of central 'self-monitoring' mechanism might underlie auditory hallucinations and passivity phenomena, which are 'first rank' features in schizophrenia [13]. Auditory hallucinations normally consist of hearing spoken voices [14,15]. The essence of passivity experiences (or delusions of control) is that the subject experiences his or her will as replaced by that of some other force or agency [16]: 'My fingers pick up the pen, but I don't control them. What they do is nothing to do with me... The force moved my lips. I began to speak. The words were made for me' [17].

Frith has suggested that these abnormal experiences arise through a lack of awareness of intended actions. Such an impairment might cause thoughts or actions to become isolated from the sense of will normally associated with them. This would result in the interpretation of internally generated voices or thoughts as external voices (auditory hallucinations and thought insertion) and of one's own movements and speech as externally caused (passivity of experiences). We have suggested that the experience of passivity arises from a lack of awareness of the predicted limb position based on the forward model [18,19]. Thus the patient is aware of the intention to move and of the movement having occurred, but is not aware of having initiated the movement. It is as if the movement, although intended, has been initiated by some external force. In a variation on this theme, Spence [20] has suggested that the problem is to do with the timing of awareness.

The perception of the sensory consequences of actions:

Evidence suggests that the sensory consequences of some self-generated movements are perceived differently from an identical sensory input that is externally generated. An example of such differential perception is the phenomenon that people cannot tickle themselves [21,22]. In Weiskrantz *et al.*'s psychophysical study, a tactile stimulus that transversed the sole of the subject's foot was administered either by the experimenter or the by the subject. Subjects rated the self-administered tactile stimulus as less tickly than the externally administered tactile stimulus. When the experimenter moved the subject's hand to tickle their foot, the tickle strength was reduced, but not to the level of the self-administered tactile stimulus. The differences in response were attributed to the mode of delivery: self-administered tactile stimulation produces both efference copy, in accordance with the motor command, and re-efference produced by the arm movement; passive arm movement produces only re-efference, and externally administered tactile stimulation produces neither efference copy nor re-efference. The authors therefore concluded that although re-efference plays a role, the signal used for attenuation is based mainly on the efference copy signal produced in concordance with a self-generated movement.

One explanation of these results is that there is a general gating of all incoming sensory stimulation during self-generated movement. Indeed, this kind of sensory gating during movement has been documented in humans [23–26]. For example, detection thresholds for an electrically induced twitch of the arm muscle are attenuated by voluntary movements of the stimulated arm [25]. Such findings suggest that the perception of sensory stimulation might be attenuated simply if self-generated movement occurs simultaneously with the stimulus: the movement might not necessarily have to produce the sensory stimulus in order for it to be attenuated. This, however, is inconsistent with the theoretical approach of forward models we have outlined, which posits that in order for sensory attenuation to occur, the specific sensory consequences of the movement must be predicted accurately. According to our hypothesis, the sensory stimulation would have to correspond to the movement producing it in order for perceptual attenuation to occur. If this hypothesis is true, there are two further possibilities. First, the sensory stimulation might have to correspond exactly to the movement producing it for any perceptual attenuation to occur. Alternatively, the amount of perceptual attenuation might be proportional to the accuracy of the sensory prediction.

To investigate this, in an experiment that was based on Weiskrantz and colleagues' study, we asked subjects to rate the sensation of a tactile stimulus on the palm of their hand, and examined the perceptual effects of altering the correspondence between self-generated movement and its sensory (tactile) consequences. This was achieved by introducing parametrically varied degrees of delay or trajectory rotation between the subject's movement and the resultant tactile stimulation. The result of increasing the delay or trajectory rotation is that the sensory stimulus no longer corresponds to that normally expected based on the efference copy produced in parallel with the motor command. Therefore as the delay or trajectory rotation increases, the sensory prediction becomes less accurate. This

has three possible effects on the sensation. First, if sensory attenuation is due to a general movement-induced sensory gating, then since movement occurs under all delays and trajectory rotations the sensation would remain at the same level of attenuation under all conditions. Second, if sensory attenuation relies on a completely accurate prediction of the sensation, then no attenuation would occur under any delay or trajectory rotation. Third, sensory attenuation could be proportional to the accuracy of the prediction, in which case as the delay or trajectory rotation is increased the intensity of the sensation should increase.

Sixteen subjects were asked to rate the sensation of a tactile stimulus on the palm of their right hand under several conditions. In all conditions the tactile stimuli applied to the hand was the same, a sinusoidal movement (amplitude 1.5 cm; frequency 2 Hz) of a piece of soft foam across the right palm. The piece of foam was attached to a robotic arm. The only difference between conditions was the causal nature of the stimulus. In the externally produced tactile stimulation condition the stimulus was generated by the robot and therefore not related to any movement the subject made. In all the remaining conditions the tactile stimulus was self-produced as the movement of the foam was determined by the movements of the subject's left hand. In these conditions the subjects held an object attached to the end of a second robot and were required to move this over their right palm sinusoidally. The motion of the left hand determined the position of the first robot, attached to which was the piece of foam that made contact with the subject's right palm. The motion of the left hand therefore produced the tactile stimulus on the right palm.

In the self-produced tactile stimulation condition, the tactile stimulus corresponded exactly to the movement of the subject's left hand. This condition corresponds to the normal situation in which subjects use their left hand to move a physical rod across the palm of their right hand (equivalent to the self-produced tickling condition in the study by Weiskrantz and colleagues). By using this robotic interface so that the tactile stimulus could be delivered under remote control by the subject, delays of 100, 200 and

300 ms were introduced between the movement of the left hand and the tactile stimulus on the right palm. In a further condition, trajectory rotations of 30°, 60° 90° were introduced between the direction of the left hand movement and the direction of the tactile stimulus on the right palm. Under all delays and trajectory rotations the left hand made the same sinusoidal movements and the right hand experienced the tactile stimulus. Only the temporal or spatial correspondence between the movement of the left hand and the sensory effect on the right palm was altered.

The results showed that subjects rated the self-produced tactile sensation as being significantly ($p < 0.001$) less tickly, intense and pleasant than an identical stimulus produced by the robot [27]. Furthermore, subjects reported a progressive increase in the tickly rating as the delay was increased between 0 ms and 200 ms ($p < 0.0005$; Fig. 2a) and as the trajectory rotation was increased between 0 and 90° ($p < 0.01$; Fig. 2b).

These results support the hypothesis that the perceptual attenuation of self-produced tactile stimulation is due to precise sensory predictions, rather than a movement-induced non-specific attenuation of all sensory signals. When there is no delay or trajectory rotation the model correctly predicts the sensory consequences of the movement, so no sensory discrepancy ensues between the predicted and actual sensory information, and the motor command to the left hand can be used to attenuate the sensation on the right palm. As the sensory feedback deviates from the prediction of the model (by increasing the delay or trajectory rotation) the sensory discrepancy between the predicted and actual sensory feedback increases, which leads to a decrease in the amount of sensory attenuation.

The physiological basis of the perceptual modulation of self-produced sensory stimuli: Electrophysiological data demonstrate that neuronal responses in somatosensory cortex are attenuated by self-generated movement. Active touch is gated in primary somatosensory cortex of rats [28] and monkeys [29–31] compared to passive and external

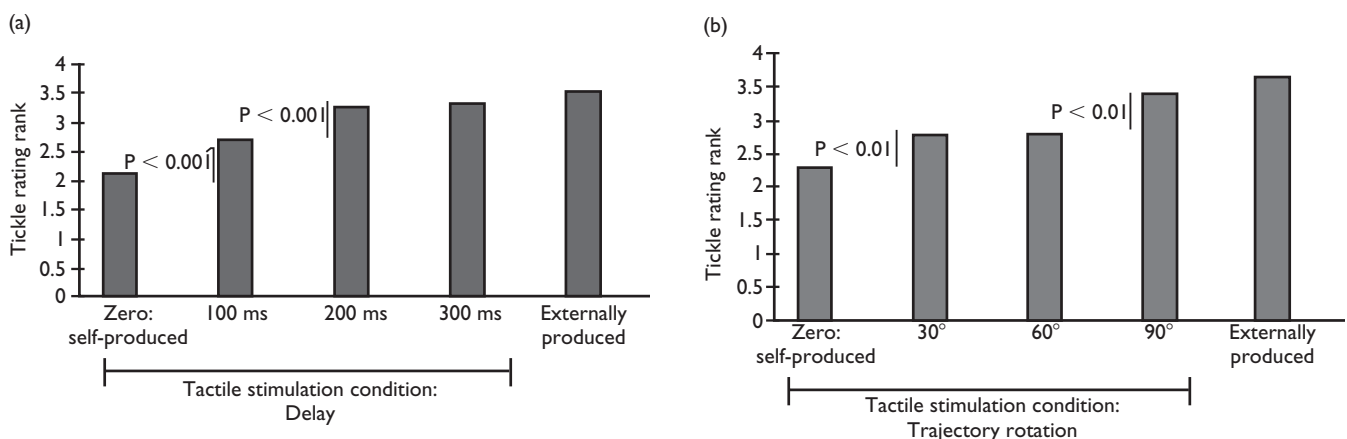


Fig. 2. Graph to show that the tickliness of a tactile stimulus increases with increasing delay (a) and trajectory rotation (b) between the movement of the left hand and the tactile stimulus on the right palm. These results suggest that the perceptual attenuation of self-produced tactile stimulation is based on specific sensory predictions made by a forward model.

touch of an identical tactile stimulus. It is possible that this movement-induced somatosensory gating is the physiological correlate of the decreased sensation associated with self-produced tactile stimuli in humans. In order for somatosensory cortex activity to be attenuated to self-produced sensory stimuli, these stimuli need to be predicted accurately. The cerebellum is a possible site for a forward model of the motor apparatus that provides predictions of the sensory consequences of motor commands. This proposal has been supported by computational [10,11,32,33], neurophysiological [34–38] and functional neuroimaging data [39].

To investigate the hypothesis that the somatosensory cortex and the cerebellum are involved in modulating the sensation of a self-produced tickle, we used fMRI to examine the neural basis of self *vs* externally produced tactile stimuli in humans [40]. Six healthy subjects were scanned while a tactile stimulation device allowed a sinusoidal tactile stimulus (a piece of soft foam moving at 2 Hz; amplitude 1.5 cm) to be applied to the subject's left palm either by their right hand or by the experimenter. To examine the neural correlates of self-produced tactile stimuli we employed a factorial design with the factors self-generated movement of the right hand *vs* rest, and tactile stimulation on the left palm *vs* no stimulation. There were four conditions: self-generated tactile stimulation; self-generated movement without tactile stimulation; externally generated tactile stimulation; and rest. Using this design we were able to assess the difference in brain activity during self-generated relative to externally generated tactile stimulation while factoring out activity associated with self-generated movement and tactile stimulation. Analysis of the imaging data resulted in the creation of statistical parametric maps [41] reflecting the two main effects, movement and tactile stimulation, and the interaction between these two factors.

The results showed an increase in activity of the secondary somatosensory cortex (SII; Fig. 3) and the anterior cingulate gyrus (ACG; Brodmann Areas 24/32; Fig. 4) when subjects experienced an externally produced tactile

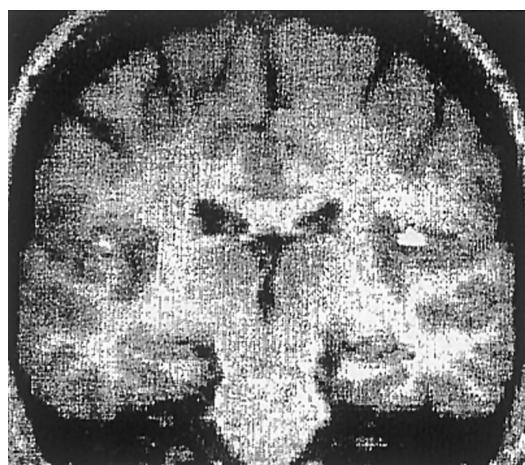


Fig. 3. The secondary somatosensory cortex showed significantly ($p < 0.05$ corrected for multiple comparisons) less BOLD signal when the tactile stimulus was self-produced relative to when it was externally produced.



Fig. 4. The anterior cingulate cortex showed significantly ($p < 0.05$ corrected for multiple comparisons) less BOLD signal when the tactile stimulus was self-produced relative to when it was externally produced. The reduction in activity in somatosensory and anterior cingulate cortex to self-produced tactile stimuli might be the physiological correlate of the reduced perception associated with this type of stimulation.

stimulus relative to a self-produced tactile stimulus. The reduction in activity in these areas to self-produced tactile stimulation might be the physiological correlate of the reduced perception associated with this type of stimulation. The activity in the ACG in particular may have been related to the increased tickliness and pleasantness of externally produced compared to self-produced tactile stimuli. Previous studies have implicated this area in affective behaviour and positive reinforcement [42–44].

While the decrease in activity in SII and ACG might underlie the reduced perception of self-produced tactile stimuli, the pattern of brain activity in the cerebellum suggests that this area is the source of the SII and ACG modulation. In SII and ACG, activity was attenuated by all movement: these areas were equally activated by movement that did and that did not result in tactile stimulation. In contrast, the right anterior cerebellar cortex was selectively deactivated by self-produced movement that resulted in a tactile stimulus, but not by movement alone, and was significantly activated by externally produced tactile stimulation (Fig. 5). This pattern suggests that the cerebellum differentiates between movements depending on their specific sensory consequences. We suggest that the cerebellum is involved in predicting the specific sensory consequences of movements and in providing the signal that is used to attenuate the somatosensory response to self-produced tactile stimulation [45].

The perception of the sensory consequences of actions in patients with auditory hallucinations and/or passivity experiences: To test the hypothesis that certain symptomatology associated with schizophrenia is due to a defect in self-monitoring, as proposed by Frith [6], we investigated whether patients with auditory hallucinations and/or passivity experiences are abnormally aware of the sensory consequences of their own movements. Age-matched patients with a diagnosis of schizophrenia, bipolar affective disorder or depression were divided into

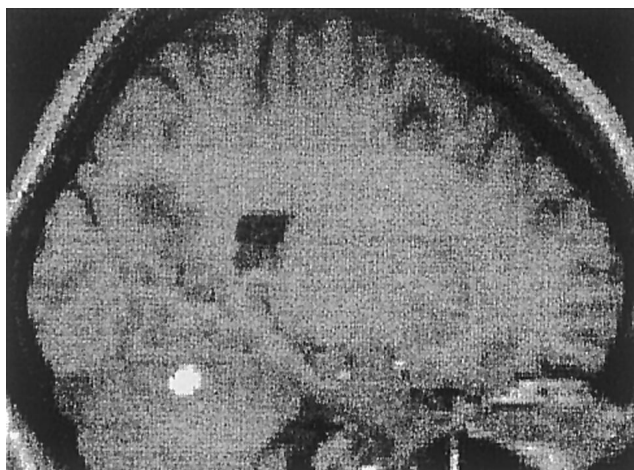


Fig. 5. The right anterior cerebellar cortex showed significantly ($p < 0.05$ corrected for multiple comparisons) less BOLD signal when the tactile stimulus was self-produced relative to when it was externally produced. There was a significant difference between the BOLD response in this area when a self-generated movement resulted in tactile stimulation compared to when it resulted in no tactile stimulation. This pattern suggests that the cerebellum differentiates between movements depending on their specific sensory consequences.

two groups on the basis of the presence ($n = 15$) or absence ($n = 23$) of auditory hallucinations and/or passivity experiences. These patient groups and 15 age-matched normal control subjects were asked to rate the perception of a tactile sensation on the palm of their left hand. The tactile stimulation was either self-produced by movement of the

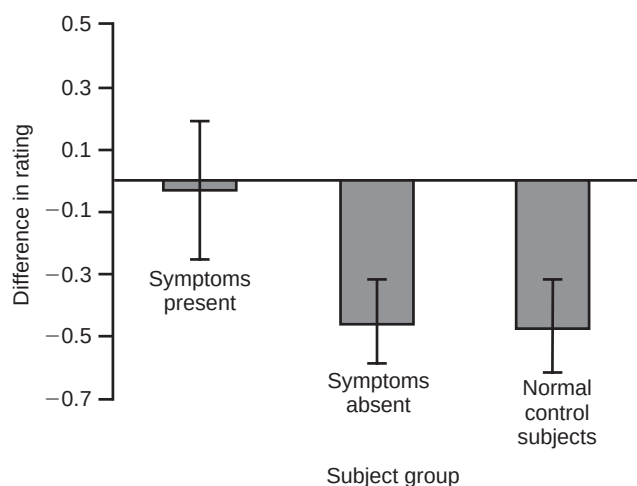


Fig. 6. Graph showing the mean (tickly, pleasant and intense combined) perceptual rating difference between self-produced and externally produced tactile stimulation conditions for the three subject groups: patients with auditory hallucinations and/or passivity, patients without these symptoms and normal control subjects. There was no significant difference between the perceptual ratings in the two conditions for patients with auditory hallucinations and/or passivity, hence the mean rating difference was close to zero. In contrast, there was a significant difference between the perceptual ratings in the two conditions for patients without these symptoms and in normal control subjects: both groups rated self-produced stimulation as less tickly, intense and pleasant than externally produced stimulation.

subject's right hand or externally produced by the experimenter. The results demonstrated that normal control subjects and patients with neither auditory hallucinations nor passivity experienced self-produced stimuli as less intense, tickly and pleasant than identical, externally produced tactile stimuli. In contrast, patients with these symptoms did not show a decrease in their perceptual ratings for tactile stimuli produced by themselves as compared to those produced by the experimenter (Fig. 6) [46]. These results support the proposal that auditory hallucinations and passivity experiences are associated with an abnormality in the forward model mechanism that normally allows us to distinguish self-produced from externally produced sensations. It is possible that the neural system associated with this mechanism, or part of it, operates abnormally in people with such symptoms.

REFERENCES

1. Jeannerod M. *The Neural and Behavioural Organisation of Goal-directed Movements*. Oxford: Oxford University Press; 1988.
2. Wolpert DM, Ghahramani Z and Jordan MI. *Science* **269**, 1880–1882 (1995).
3. Wolpert DM. *Trends Cogn Sci* **1**, 209–216 (1997).
4. Decety J. *Rev Neurosci* **7**, 285–297 (1996).
5. Jeannerod M. *The Cognitive Neuropsychology of Action*. Cambridge: Blackwell; 1997.
6. Frith CD. *The Cognitive Neuropsychology of Schizophrenia*. Hove: Lawrence Erlbaum Associates; 1992.
7. Helmholtz H. *Handbuch der Physiologischen Optik*. 12th edition. Hamburg: Voss; 1967.
8. Holst E. *Br J Anim Behav* **2**, 89–94 (1954).
9. Sperry RW. *J Comp Physiol Psychol* **43**, 482–289 (1950).
10. Ito M. *Int J Neurol* **7**, 162–176 (1970).
11. Wolpert DM, Miall RC and Kawato M. *Trends Cogn Sci* **2**, 338–347 (1998).
12. Jordan MI. *Psychonomic Bull Rev* **6**, 504–510 (1996).
13. Schneider K. *Clinical Psychopathology*. New York: Grune and Stratton; 1995.
14. Hoffman RE. *Behav Brain Sci* **9**, 503–517 (1986).
15. Johnstone EC. *Br J Psychiatry Suppl.* **13**, 5–6 (1991).
16. Wing JK, Cooper JE and Sartorius N. *Description and Classification of Psychiatric Symptoms*. London: Cambridge University Press; 1974.
17. Mellors CS. *Br J Psychiatry* **117**, 15–23 (1970).
18. Frith, CD, Blakemore S-J and Wolpert DM. *Brain Res Rev* **31**, 357–363 (2000).
19. Frith CD, Blakemore S-J and Wolpert DM. *Phil Trans R Soc Lond: Biol Sci* (in press 2000).
20. Spence SA. *Philos Psychiatry Psychol* **3**, 75–90 (1996).
21. Weiskrantz L, Elliott J and Darlington C. *Nature* **230**, 598–599 (1971).
22. Claxton G. *Percept Motor Skills* **41**, 335–338 (1975).
23. Angel RW and Malenka RC. *Exp Neurol* **77**, 266–274 (1982).
24. Chapman CE, Bushnell MC, Miron D et al. *Exp Brain Res* **68**, 516–524 (1987).
25. Collins DF, Cameron T, Gillard DM and Prochazka A. *J Physiol* **508**, 635–643 (1998).
26. Milne RJ, Aniss AM, Kay NE and Gandevia SC. *Exp Brain Res* **70**, 569–576 (1988).
27. Blakemore S-J, Frith CD and Wolpert DW. *J Cogn Neurosci* **11**, 551–559 (1999).
28. Chapin JK and Woodward DJ. *Exp Neurol* **785**, 654–669 (1982).
29. Jiang W, Chapman CE and Lamarre Y. *Exp Brain Res* **84**, 342–354 (1991).
30. Chapman CE and Ageranioti-Belanger SA. Comparison of the discharge of primary somatosensory cortical (SI) neurones during active and passive tactile discrimination. *Proc Third IBRO World Congress Neurosci* August 4–9, Montreal, Quebec, p. 317 (1991).
31. Chapman CE. *Can J Physiol Pharmacol* **72**, 558–570 (1994).
32. Paulin MG. A Kalman filter theory of the cerebellum. In: Arbib EMA and Amari ES, eds. *Dynamic Interactions in Neural Networks: Models and Data*. Berlin: Springer Verlag; 1989, pp. 241–259.

33. Miall RC, Weir DJ, Wolpert DM and Stein JF. *J Motor Behav* **25**, 203–216 (1993).
34. Oscarsson O. In: Courville J, DeMontigny C and Lamarre Y, eds. *The Inferior Olivary Nucleus: Anatomy and Physiology*. New York: Raven Press; 1980.
35. Gellman R, Gibson AR and Houk C. *J Neurophysiol* **54**, 40–60 (1995).
36. Andersson G and Armstrong DM. *Neurosci Lett Suppl.* **22**, S27 (1985).
37. Andersson G and Armstrong DM. *J Physiol (Lond)* **385**, 107–134 (1987).
38. Simpson JL, Wylie DR and De Zeeuw CI. *Behav Brain Sci* **19**, 384 (1995).
39. Imamizu M, Miyauchi S, Tamada T *et al.* *Nature* **403**, 192–195 (2000).
40. Blakemore S-J, Wolpert DM and Frith CD. *Nature Neurosci* **1**, 635–640 (1998).
41. Friston KJ, Frith CD, Liddle PF *et al.* *J Cerebr Blood Flow Metab* **10**, 458–466 (1990).
42. Vogt BA, Finch DM and Olson CR. *Cerebr Cortex* **2**, 435–445 (1992).
43. Vogt BA and Gabriel M, eds. *Neurobiology of Cingulate Cortex and Limbic Thalamus*. Boston: Birkhauser; 1993.
44. Porrino LJ. *Psychopharmacology (Berl)* **112**, 343–51 (1993).
45. Blakemore S-J, Wolpert DM and Frith CD. *NeuroImage* **10**, 448–459 (1999).
46. Blakemore S-J, Smith J, Steel R *et al.* *Psychol Med* (In press 2000).

Acknowledgements: The work discussed in this review was supported by a grant from the Wellcome Trust. S.-J.B. is supported by a Wellcome Trust 4-year PhD Programme in Neuroscience at University College London. The research involving patients was performed in the Department of Psychiatry at the Royal Edinburgh Hospital with Joanna Smith, Robby Steel, Eve Johnstone. The costs of the work in Edinburgh were met by a grant from the Stanley Foundation.