Touching on the cause of delusions

JOHN HARRIS (REPORT DIGITAL)

SARAH-JAYNE BLAKEMORE, *winner of the 2002 Award for Outstanding Doctoral Research Contributions to Psychology, investigated our 'self-monitoring' mechanism and what happens when it fails.*

My fingers pick up the pen, but I don't control them. What they do is nothing to do with me. (Mellors, 1970, p.13)

DELUSIONS of alien control are symptoms associated with schizophrenia in which people misattribute self-generated actions to an external source (Schneider, 1959). The actions in question can be mundane, such as picking up a cup or combing one's hair. Auditory hallucinations are common in schizophrenia, and normally consist of hearing speech or voices (Johnstone, 1991). Both delusions of alien control and auditory hallucinations are included as 'first rank' features in schizophrenia (Schneider, 1959). But what can such phenomena tell us about how we know that our own actions belong to us? How are we able to distinguish self-generated sensory events from those that arise externally? Here, I describe behavioural and brain-imaging experiments designed to investigate these questions.

Did I do that?

Normally, we can readily detect whether a movement is self-generated or externally caused. When I make an arm movement I know that the movement is my own and do not confuse it with a passive arm movement; similarly I can distinguish a self-generated touch from an external one, and know when a voice is my own and when it belongs to someone else. It has been proposed that an internal predictor, or 'forward model', uses information about intentions to enable this distinction between self-generated and externally generated sensory events (Wolpert *et al*., 1995; Wolpert *et al*., 2001). Forward models use an 'efference copy' (a copy of the motor command: von Holst, 1954) to make a prediction of the consequences of the motor act. This prediction is compared with the actual sensory feedback from movement and this comparison is used to cancel the sensory effect of the motor act. This predictive system is useful because it can be used to filter incoming sensory signals, picking out sensory stimulation that occurs as a necessary consequence of self-produced motion (which is associated with little or no sensory discrepancy resulting from the comparison between predicted and actual sensory feedback) and sensory information caused externally, such as touch produced by an external object or agent (where the sensory discrepancy is greater).

An impairment in such a predictive system could cause a lack of attenuation of the sensory consequences of self-produced actions, which would therefore be indistinguishable from externally generated sensations. This would result in the interpretation of one's own movements as being externally caused or one's thoughts as external voices – delusions of alien control and auditory hallucinations (Blakemore *et al*., 2002; Frith, 1992; Frith *et al*., 2000).

Why can't I tickle myself?

Evidence suggests that the sensory consequences of some self-generated movements are perceived differently from identical sensory input when it is externally generated. An example of such differential perception is the phenomenon that people cannot tickle themselves (e.g. Weiskrantz *et al*., 1971). It has been argued that efference copy produced in parallel with the motor command underlies this phenomenon. To investigate this proposal, we asked participants 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.

Delays and trajectory rotations were produced by a robotic interface – participants moved a robotic arm with their left hand and this movement caused a second foam-tipped robotic arm to move across their right palm (see Figure 1). By

WEBLINKS

Wellcome Department of Imaging Neuroscience: *www.fil.ion.ucl.ac.uk*

Sensorimotor Control Lab: *www.hera.ucl.ac.uk* using this equipment so that the tactile stimulus could be delivered under remote control by the participant, delays of 100, 200 and 300ms were introduced – without telling the participant – between the movement of the left hand and the touch on the right palm. In a further condition trajectory rotations of 30°, 60° and 90° were introduced between the direction of the left-hand movement and the direction of the resultant stimulus on the right palm.

Under all delays and trajectory rotations the left hand made the same wavelike 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 result of increasing the delay or trajectory rotation is that the sensory stimulus no longer corresponds to that which would be normally expected based on the efference copy produced in parallel with the motor command. As the delay or trajectory rotation increases, the sensory prediction becomes less accurate.

The results showed that participants rated the self-produced tactile sensation as being significantly less tickly, intense and pleasant than an identical stimulus produced by the robot (Blakemore, Frith *et al.*, 1999). Furthermore, participants reported a progressive increase in the tickly rating as the delay was increased between 0ms and 200ms and as the trajectory rotation was increased between 0° and 90°. These results support the hypothesis that the perceptual attenuation of self-produced tactile stimulation is due to precise sensory predictions. As the sensory feedback deviates from the prediction of the forward 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.

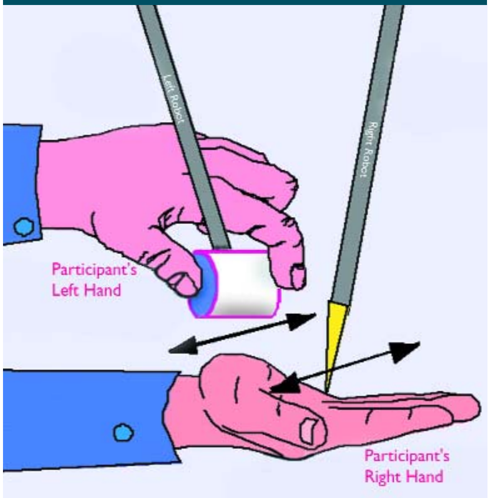
Schizophrenia and self-monitoring

People with delusions of control feel as if their intentions are being monitored and their actions made for them by some external force. This could theoretically arise if there were an impairment of either the prediction or the comparison process of the forward model (Frith *et al.*, 2000). For example, if the comparison process were impaired and always produced a high level of sensory discrepancy despite the accuracy of the sensory prediction, then self-produced sensations would be associated with high levels of sensory discrepancy despite being accurately predicted. In this way, self-produced stimulation could be interpreted as being externally produced.

To test the hypothesis that delusions of control and auditory hallucinations occur due to a defect in self-monitoring, we investigated whether individuals with auditory hallucinations or delusions of control or both are abnormally aware of the sensory consequences of their own movements. Individuals with a diagnosis of schizophrenia, bipolar affective disorder and depression were divided into two groups on the basis of the presence or absence of auditory hallucinations and delusions of control. These patient groups and a group of age-matched healthy control participants were asked to rate the perception of a tactile sensation (a piece of soft foam) on the palm of their left hand. The tactile stimulation was either self-produced by the participant's right hand or externally produced by the experimenter.

The results demonstrated that healthy control participants and participants with neither auditory hallucinations nor passivity symptoms experienced self-produced stimuli as less intense, tickly and pleasant than identical externally produced tactile stimuli. In contrast, participants with those symptoms did not show a decrease in their perceptual ratings for tactile stimuli produced by themselves compared with those produced by the experimenter (Blakemore *et al.*, 2000). 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.

FIGURE 1 Tickling equipment



The physiological basis

Neurophysiological data demonstrate that neuronal responses in somatosensory cortex are attenuated by self-generated movement. Active touch is attenuated in primary somatosensory cortex of animals (Chapman, 1994) compared with passive and external touch of an identical tactile stimulus. It is possible that this movement-induced somatosensory attenuation 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 (Miall *et al.*, 1993; Wolpert *et al.*, 1998), neurophysiological (Simpson *et al.*, 1995) and functional neuroimaging data (Imamizu *et al.*, 2000).

To investigate the hypothesis that the somatosensory cortex and the cerebellum are involved in modulating the sensation of a self-produced tactile stimulation, we used functional magnetic resonance imaging to examine the neural basis of self-produced versus externally produced tactile stimuli in humans (Blakemore *et al.*, 1998). Healthy participants were scanned while a tactile stimulation device allowed a piece of soft foam to be applied to the participant's left palm. The touch stimulus was produced either by the participant's right hand or by the experimenter.

The results showed an increase in activity of the secondary somatosensory cortex (SII) and the anterior cingulate cortex (ACC) when participants experienced an externally produced, relative to self-produced, tactile stimulus. The activity in the ACC in particular may have been related to the increased tickliness and pleasantness of externally produced compared with self-produced tactile stimuli. Previous studies have implicated this area in affective behaviour and positive reinforcement (Vogt & Gabriel, 1993). Alternatively, the activity in ACC might be related to the requirement to monitor the sensations the participants were experiencing (Frith & Frith, 1999; Lane *et al.*, 1997).

While the decrease in activity in SII and ACC 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 ACC modulation. In SII and ACC, 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 significantly activated by externally produced tactile stimulation. This pattern suggests that the cerebellum distinguishes between movements depending on their specific sensory consequences. Further analysis showed that the cerebellum may be involved in providing the signal that is used to attenuate the somatosensory response to self-produced tactile stimulation (Blakemore, Wolpert *et al.*, 1999).

A second experiment supported the proposal that the cerebellum distinguishes between movements depending on their specific sensory consequences (Blakemore *et al.*, 2001). In this experiment participants were scanned using positron emission tomography while generating a tactile stimulus on the palm of their hand, as before. This time, however, the tactile stimulation was produced under remote control via a robotic interface, as described earlier. Blood flow in the right cerebellar cortex significantly correlated with delay. This suggests that activity in this region increases as the actual feedback from movement deviates from the predicted sensory feedback. On the basis of these results we proposed that the cerebellum is involved in signalling discrepancies between the predicted and actual consequences of action.

Conclusion

The ability to distinguish between self-produced movements is an important part of a 'Who' system, which allows one to link an action with its cause (Georgieff & Jeannerod, 1998). Our results suggest that a cerebellar-parietal network is involved in the Who system, specifically in the prediction and attenuation of the sensory consequences of self-generated actions. It is possible that overactivation of this neural system contributes to the misattribution of actions to an external source in delusions of control. Indeed, there is evidence that overactivity of the parietal cortex and cerebellum occurs during self-generated movements in individuals with delusions of control, and subsides when the same individuals are in remission (Spence *et al.*, 1997). It is possible that malfunctioning in this network leading to overactivity produces the feeling of 'otherness' associated with self-produced movements in delusions of control.

■ *Dr Sarah-Jayne Blakemore is at the Institute of Cognitive Neuroscience, University College London. E-mail: s.blakemore@ucl.ac.uk. The research described in this article was carried out at the Wellcome Department of Cognitive Neurology, UCL.*

References

Blakemore, S-J., Frith, C.D. & Wolpert, D.W. (1999). Spatiotemporal prediction modulates the perception of self-produced stimuli. *Journal of Cognitive Neuroscience, 11*, 551–559.

Blakemore, S-J., Frith, C.D. & Wolpert, D.W. (2001). The cerebellum is involved in predicting the sensory consequences of action. *NeuroReport, 12*, 1879–1885.

Blakemore, S-J., Smith, J., Steel, R., Johnstone, E. & Frith, C.D. (2000). The perception of self-produced sensory stimuli in patients with auditory hallucinations and passivity experiences: Evidence for a breakdown in self-monitoring. *Psychological Medicine, 30*, 1131–1139.

Blakemore, S-J., Wolpert, D.M. & Frith, C.D. (1998). Central cancellation of self-produced tickle sensation. *Nature Neuroscience, 1*, 635–640.

Blakemore, S-J., Wolpert, D.M. & Frith, C.D. (1999). The cerebellum contributes to somatosensory cortical activity during self-produced tactile stimulation. *NeuroImage, 10*, 448–459.

Blakemore, S-J., Wolpert, D.M. & Frith, C.D. (2002). A framework for understanding abnormalities in the control and awareness of action. *Trends in Cognitive Science, 6*, 237–242.

Chapman, C.E. (1994). Active versus passive touch: Factors influencing the transmission of somatosensory signals to primary somatosensory cortex. *Canadian Journal of Physiology and Pharmacology, 72*, 558–570.

Frith, C.D. (1992). *The cognitive neuropsychology of schizophrenia.* Hove: Lawrence Erlbaum.

Frith C.D. & Frith, U. (1999). Interacting minds – A biological basis. *Science, 286*, 1692–1695.

Frith, C.D., Blakemore S-J. & Wolpert D.M. (2000). Abnormalities in the awareness and control of action. *Philosophical Transactions of the Royal Society of London: Biological Sciences, 355*(1404), 1771–1788.

Georgieff, N. & Jeannerod, M. (1998). Beyond consciousness of external reality. A 'Who' system for consciousness of action and self-consciousness. *Consciousness and Cognition, 7*, 465–477.

Imamizu, H., Miyauchi, S., Tamada, T., Sasaki, Y., Takino, R., Pütz, B. *et al.* (2000). Human cerebellar activity reflecting an acquired internal model of a new tool. *Nature, 403*, 192–195.

Johnstone, E.C. (1991). Defining characteristics of schizophrenia. *British Journal of Psychiatry, Supplement 13*, 5–6.

Lane, R.D., Fink, G.R., Chua, P.M. & Dolan, R.J. (1997). Neural activation during selective attention to subjective emotional responses. *NeuroReport, 8*, 3969–3972.

Mellors, C.S. (1970). First-rank symptoms of schizophrenia. *British Journal of Psychiatry, 117*, 15–23.

Miall, R.C., Weir, D.J., Wolpert, D.M. & Stein J.F. (1993). Is the cerebellum a Smith predictor? *Journal of Motor Behaviour, 25*, 203–216.

Schneider, K. (1959). *Clinical psychopathology*. New York: Grune & Stratton.

Simpson, J.L., Wylie, D.R. & De Zeeuw, C.I. (1995). On climbing fiber signals and their consequence(s). *Behavioural and Brain Sciences, 19*, 384.

Spence, S.A., Brooks, D.J., Hirsch, S.R., Liddle, P.F., Meehan, J. & Grasby, P.M. (1997). A PET study of voluntary movement in schizophrenic patients experiencing passivity phenomena (delusions of alien control). *Brain, 120*, 1997–2011.

Vogt, B.A. & Gabriel, M. (Eds.) (1993). *Neurobiology of cingulate cortex and limbic thalamus.* Boston, MA: Birkauser.

von Holst, E. (1954). Relations between the central nervous system and the peripheral organs. *British Journal of Animal Behaviour*, 2, 89–94.

Weiskrantz, L., Elliot, J. & Darlington, C. (1971). Preliminary observations of tickling oneself. *Nature, 230*, 598–599.

Wolpert, D.M., Ghahramani, Z. & Jordan, M.I. (1995). An internal model for sensorimotor integration. *Science, 269*, 1880–1882.

Wolpert, D.M., Miall, R.C. & Kawato, M. (1998). Internal models in the cerebellum. *Trends in Cognitive Science, 2*, 338–347.

Wolpert, D.M., Ghahramani, Z. & Flanagan, R. (2001). Perspectives and problems in motor learning. *Trends in Cognitive Science, 5*, 487–494.