How the brain perceives causality: an event-related fMRI study

Sarah-Jayne Blakemore,[1,CA] Pierre Fonlupt,[1] Mathilde Pachot-Clouard,[2] Céline Darmon,[1,2] Pascal Boyer,[3] Andrew N. Meltzoff,[4] Christoph Segebarth[2] and Jean Decety[1,4]

[1]Brain Activation and Mental Processes, INSERM U280, Lyon 69424, Cedex 03; [2]Unité Mixte INSERM/UJF U438, LRC CEA, Grenoble, France; [3]College of Arts and Sciences, Washington University, St Louis, MO; [4]Center for Mind, Brain and Learning, University of Washington, Seattle, WA, USA

[CA]Corresponding Author

Received 25 July 2001; accepted 24 September 2001

Detection of the causal relationships between events is fundamental for understanding the world around us. We report an event-related fMRI study designed to investigate how the human brain processes the perception of mechanical causality. Subjects were presented with mechanically causal events (in which a ball collides with and causes movement of another ball) and non-causal events (in which no contact is made between the balls). There was a significantly higher level of activation of V5/MT/MST bilaterally, the superior temporal sulcus bilaterally and the left intraparietal sulcus to causal relative to non-causal events. Directing attention to the causal nature of the stimuli had no significant effect on the neural processing of the causal events. These results support theories of causality suggesting that the perception of elementary mechanical causality events is automatically processed by the visual system. *NeuroReport* 12:3741–3746 © 2001 Lippincott Williams & Wilkins.

Key words: Causation; Cognitive neuroscience; Event-related fMRI; Human; Launching displays

INTRODUCTION

Detection of the causal relationships between events is fundamental for understanding what is happening in the world around us. The understanding of the causal nature of events has been the subject of debate for centuries. Hume argued that we have no direct way of knowing that one event or action will cause another: he claimed that causality is not something that can be directly perceived [1]. Instead, he suggested that understanding the causal relationships between events requires inference on the basis of previous, learned contingencies. A contrasting view to that of Hume was put forward by Michotte [2], whose empirical research on visual displays involving causality led him to propose that the perception of causality is direct, automatic and possibly innate. A classic form of mechanical causality studied by Michotte was the so-called launching effect, exemplified by one billiard ball hitting another. In Michotte's visual stimuli, one object (A) moves until it touches another item (B), at which point A stops and B starts moving (see Fig. 1 for example). Michotte interviewed adult subjects who viewed such displays and found that they perceive them as 'A causes the motion of B'.

Many studies since Michotte's have supported his suggestion that the perception of causality in launch displays is fast (occurring < 250 ms after the interaction between the objects in the displays), automatic and highly stimulus driven [3,4]. The perception of causality also exists for displays involving apparent motion [5] and other types of object interactions, such as one object pulling another [6] and objects causing other objects to burst [7]. Developmental studies have shown that the ability to perceive causality from simple motion displays emerges early in life [8–10].

Top-down processes, in particular the knowledge that the displays are not really causal, appear to have little influence on the perception of mechanical causality [11].

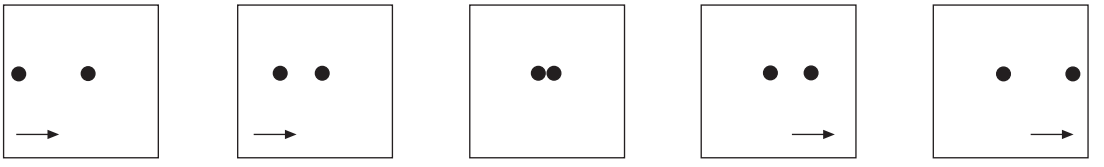

Fig. 1. Example of a launching display (causality event used in the current experiment). In this causality event a ball rolls across the screen from left to right and after 1 s collides with and is perceived to cause movement of a second ball.

Based on the evidence that causal perception is a fast, automatic, bottom-up process, which is distinct from higher-level cognitive interpretations [12], it has been speculated that such causal perception may conform to modular perceptual processing [13] and may be hardwired in the visual system [4]. The idea is that the visual system works to recover the causal structure of the world by inferring properties such as mechanical causality, in the same way that it recovers the physical structure of the world by inferring properties such as 3D shape [4].

Little is known about the neural correlates of the perception of causality in Michotte-like displays. Electrophysiological studies in the monkey have shown that cells at a posterior location in the superior temporal sulcus (STS) that are sensitive to hand–object interactions are especially responsive if the observed hand causes an object to move [14,15]. Such STS cells do not respond if the hand and object movements are not perceived as causally related due spatial and temporal gaps between them. If the response of STS neurons in these studies was due to the causal nature of the hand–object interactions, then an equivalent region of the human STS (the posterior portion of the middle temporal gyrus) may be involved in processing mechanical causation. The psychophysical evidence suggests that the perception of causality is an automatic process that is mandatory given particular visual input [4]. If this is the case, regions specifically responsive to visual movement (the V5/MT/MST complex) [16] and regions involved in extracting properties such as depth from motion (including V5/MT/MST and the intraparietal sulcus) [17] may be activated by stimuli that evoke the perception of mechanical causation. In addition, higher visual areas involved in processing spatial relationships between moving objects and conveying motion information, including the intraparietal sulcus and the parietal lobe [18–20], may be recruited to detect causality in visual displays.

The present study was designed to test the hypotheses that the perception of mechanical causality is automatically processed by the visual system and that its neural processing is not affected by higher-lever processes such as attention to causation. To investigate these hypotheses, we used event-related fMRI to evaluate the neural correlates of perceived causality in simple Michotte-like launching displays. In order to investigate whether there is a top-down influence of attention to causality on the processing of these basic causal events, we employed a factorial design that varied two factors: causality *vs* no causality, and attention to causality *vs* no attention to causality. To manipulate attention to causality, subjects were asked to make two different types of response to each visual event. They were asked to detect either the direction of motion of the stimuli (thus their attention was directed away from causality) or the presence of causal relationships between the stimuli (thus their attention was directed towards causality) in the displays. If there is no top-down influence of cognitive task on the perception of causality, then the nature of the task (detecting stimulus motion direction or the presence of causality) should have no effect on the neural processing of causal stimuli [11]. In other words, paying attention to the causal nature of the stimuli should not influence the way in which the brain processes these causal stimuli.

MATERIALS AND METHODS

Subjects: Eight healthy right-handed volunteers (four females; age range 20–25 years) took part in the study, which was performed in accordance with the local Ethics Committee. Written, informed consent was obtained from all subjects prior to participation according to the Declaration of Helsinki.

Materials and Methods: During scanning, subjects viewed four types of visual event, each of which lasted 2 s and was followed by a 2 s response period. There was one experimental event. In the *causality* event a blue ball rolls horizontally across the screen and after 1 s collides with a red ball, which is positioned in the centre of the screen in the path of the blue ball (as in Fig. 1). The red ball moves horizontally off the screen. There were two control events. In the *non-causality* event the blue ball rolls across the screen as in the causality event, but passes underneath the red ball, which is positioned above the path of the blue ball, so no contact is made between them. In a *visual transient* event, a blue ball rolls across the screen and changes colour (to red) after 1 s. This event was designed to control for the visual transient event (the collision between the blue and red balls) that occurred at 1 s in the causality event but not in the non-causality event. As a baseline stimulus, the *null* event comprised a black fixation point in the centre of a white screen.

Each stimulus image was made up of 512×512 pixels and 256 colours. Each event lasted 2 s and the screen was updated at 30 images/s. The position of the blue ball's starting point (top, middle or bottom of the screen), the colour of the balls, and the direction of motion (right to left or left to right) were varied. The variation of these factors was balanced between conditions. After each of the four event types there was a 2 s interval in which subjects saw a fixation cross and were asked to make a response based on the preceding stimulus by pressing one of two buttons.

Each subject underwent four scanning sessions. Within each session, each of the three visual events was repeated 21 times. In addition, 26 null events were included in each session, five of which occurred at the end of the session. The order of presentation of the stimuli was optimised [21,22] by generating 10000 random permutations of the integers between 1 and 84 and dividing the series into four blocks of 21 numbers, each of which represented the presentation order of the four different types of event. This ensured sufficient temporal jitter between sequential trials of the same type [21–26]. The efficiency of the sequence orders was assessed [21,22] (implemented in SPM99) and the optimal sequence orders with regards to an efficiency criterion relating to the contrasts causality *vs* non-causality events and causality *vs* visual transient events were selected.

Factorial nature of design: We employed a factorial design with two factors: causality *vs* no causality and attention to causality *vs* no attention to causality. After each visual event in sessions 1 and 2, subjects were instructed to make a response with the index or middle finger of their right hand depending on the direction of motion of the ball (the attention to motion direction task). After each visual event in sessions 3 and 4, subjects were instructed to make

a response with the index or middle finger of their right hand based on the presence or absence of a causal relationship between the balls (the attention to causality task). The ordering of the tasks was not counterbalanced between sessions in order to avoid biasing subjects' attention towards causality in the attention to motion direction task. The design was fully factorial, as shown in Table 1.

Subjects were instructed to respond with their right index finger after each null event in all sessions. All subject responses were recorded for subsequent reaction time analysis. Instructions were provided visually at the beginning of sessions 1 and 3 and subjects practised the tasks for ~2 min at the beginning of these sessions.

Data acquisition: A Philips NT MRI scanner operating at 1.5 T was used to acquire both T1-weighted structural images and gradient echo-planar T2*-weighted MRI image volumes with blood oxygentation level dependent (BOLD) contrast (TR = 2 s; TE = 45 ms; matrix = 64 × 64 mm; FOV = 256 × 256 mm^2). For each subject, data were acquired in four scanning sessions. A total of 178 volumes were acquired per session, plus 10 dummy volumes, subsequently discarded, to allow for T1 equilibrium effects. Each functional brain volume comprised 23 5 mm axial slices with in-plane resolution of 4 × 4 mm positioned to cover the whole brain. The acquisition of a T1-weighted anatomical image occurred after session 2 for each participant. The total duration of the experiment was around 40 min/subject.

Statistical analysis: Reaction times and accuracy of subject responses after each event were recorded and subsequently analysed using a two-way ANOVA.

Functional imaging analysis used the technique of statistical parametric mapping, implemented in SPM99 (http://www.fil.ion.ucl.ac.uk/spm). For each subject, a set of 712 fMRI scans was acquisition-corrected to correct for sampling bias effects caused by different slices being acquired at different times relative to the haemodynamic response. The scans were then realigned to correct for interscan movement and stereotactically normalised using sinc interpolation [27] into the standard space defined by the Montreal National Institute template. After normalisation the image volumes had a resolution of 4 × 4 × 5 mm. The scans were then smoothed with a Gaussian kernel of 8 mm full-width half maximum to account for residual inter-subject differences [27].

The analysis of the functional imaging data entailed the creation of statistical parametric maps representing a statistical assessment of hypothesised condition-specific effects [28]. Four event types were modelled: causality, non-causality, visual transient and the null events. These effects were modelled by convolving a delta function at one second after each event onset with the haemodynamic response function, and its two partial derivatives, to create regressors of interest. The events corresponding to the subject responses were modelled as a regressor of no interest, as were low frequency drifts in signal (cut-off 120 s). Areas of significant change in brain activity were specified by appropriately weighted linear contrasts of the condition-specific effects and determined using the *t*-statistic on a voxel to voxel basis.

Statistical analysis was performed to examine the simple effects of the three visual events compared with the null stimulus, the main effects of causality *vs* non-causality and causality *vs* visual transient, and the interaction between causality and experimental task (see Table 1). Examination of the interaction reflects the statistically significant differential effects of causal stimuli in the context of attention to causality or attention to stimulus motion direction. This allowed us to search for an interaction with experimental task in regions that previously demonstrated an effect of causality. The presence of a significant interaction (at a reduced statistical threshold of $p < 0.001$ uncorrected) in these regions would suggest that causality-evoked activation depends on the experimental task.

The statistical contrasts were used to create an SPM{t}, which was transformed into an SPM{Z} and thresholded at $p < 0.05$ (corrected on the basis of the theory of random Gaussian fields for multiple comparisons across the whole brain volume examined). We report regions that survive correction at $p < 0.05$ plus those regions surviving an uncorrected threshold of $p < 0.001$ for which we had an *a priori* hypothesis for their activation.

RESULTS

Behavioural responses: Behavioural responses indicated that subjects perceived the causal events as involving a causal relationship between the balls whereas they did not perceive the non-causal and visual transient events as involving a causal relationship between the balls. An ANOVA performed on the response times demonstrated that there was no significant difference between reaction times of subject responses in the causal and non-causal conditions during either of the two tasks (detection of motion direction and detection of causality; Table 2; $F = 0.066$; $p = 0.98$; $df = 1,24$).

Functional imaging results: The analysis of the simple effects of each of the three visual events compared with the null stimulus revealed significant activations in cortical regions involved in processing the various aspects of moving, coloured visual stimuli, as would be expected (Table 3).

Several areas responded significantly more strongly to causal events than to both non-causal and visual transient

Table 1. Factorial design.

	Attention to causality absent, subjects asked to detect motion direction	Attention to causality present, subjects asked to detect presence of causality
Causality present	Causality event	Causality event
Causality absent	Non-causality event	Non-causality event

Table 2. Average reaction times of responses, calculated from the onset of the 2 s response window after each event, for all subjects combined in the four conditions of interest.

Condition	Average (± s.d.) response time (ms)
Causality; attention to movement direction	869.35 ± 101.26
Non-causality; attention to movement direction	860.90 ± 81.70
Causality; attention to causality	867.69 ± 96.96
Non-causality; attention to causality	915.88 ± 109.70

events (Table 4). To isolate regions that were significantly more activated by causal events than by the two visual control events, the contrast comparing causal and non-causal events was masked (inclusively) by the contrast comparing causal events and visual transient events at $Z = 3.09$. The BOLD response in the resulting regions was significantly higher during causal events than during non-causal events and visual transient events. These areas were located in the MT/V5 bilaterally, STS bilaterally and the border of the intraparietal sulcus and angular gyrus on the left (Fig. 2).

The nature of the task (detecting stimulus motion direction or detecting the presence of causality) had no significant influence on the brain regions involved in processing causal stimuli. There was no significant modulation of the activity in bilateral MT/V5, STS or the left intraparietal sulcus when attention was directed to causality as opposed to when attention was directed to stimulus motion direction. The absence of such an interaction demonstrates that the task manipulation used in the current study had no significant top-down effect on the neural processing of the causal stimuli. However, the possibility of a type II error in this result cannot be ruled out: the particular task manipulation used in this study may simply have been ineffective at elucidating a top-down effect on causality-evoked activity, which may be elucidated by some other task manipulation.

Table 3. Regions activated by the simple effects of each of the three visual events compared with the null event.

Region	Coordinates	Z ($p < 0.05$, corrected)
Causal−null		
Right visual cortex*	28,−76,−15	Infinite
Left extrastriate cortex*	−48,−72,5	Infinite
Right frontal eyefields	28,−4,55	7.65
Left frontal eyefields	−32,−4,60	6.94
Non-causal−null		
Right extrastriate cortex*	8,−84,−5	Infinite
Left extrastriate cortex*	−20,−64,60	Infinite
Right frontal eyefields	28,−4,55	7.16
Left frontal eyefields	−32,−4,60	6.28
Visual transient−null		
Right extrastriate cortex*	8,−84,−5	Infinite
Left extrastriate cortex*	−12,−80,5	Infinite
Right frontal eyefields	28,0,55	Infinite
Left frontal eyefields	−32,−8,50	6.86

*Visual cortex activations resulting from each contrast extend from extrastriate cortex to temporal and parietal lobes; voxel of maximum intensity within each cluster is reported.

Table 4. Regions that were significantly more activated by causal events than by non-causal and visual transient events.

Region	Coordinates	Z ($p < 0.001$)
Right medial temporal area (MT/V5)	52,−60,5	4.81
Left medial temporal area (MT/V5)	−56,−68,0	4.08
Right superior temporal lobe (STS)	64,−40,0	3.75
Left superior temporal lobe (STS)	−60,−48,5	5.90
Left intraparietal sulcus/angular gyrus	−44,−52,55	3.62

This contrast was masked (inclusively) by the contrast comparing causal events and visual transient events at $Z = 3.09$. The BOLD response in the reported regions was significantly higher during causal events than during non-causal events and visual transient events.

DISCUSSION

The present study investigated the neural correlates of perceived causality in collision events of the type used by Michotte. The results support the hypothesis that the elementary, billiard-ball type of mechanical causality is automatically processed by the visual system. Our interaction analysis suggests that directing attention to the presence of causality has no effect on the neural processing of this type of causality.

Our fMRI results lend brain imaging data to support the theory originally put forward by Michotte that the perception of causality is directly processed by the visual system [2]. The Michotte-like launching displays used in the current study, which were perceived by the subjects as involving a causal relationship between the stimuli, activated MT/V5 and STS bilaterally and the left intraparietal sulcus to a significantly greater extent than similar non-launching displays. These results support the proposal that the visual system works to recover the causal structure of the world by inferring properties such as causality, just as it works to recover the physical structure of the world by inferring properties such as 3D shape [4]. In particular, the current results suggest that MT/V5, STS and the left intraparietal sulcus may play a particularly important role in detecting causality in visual events. MT/V5, which is a region specialised for processing visual motion [16,17], may be fine-tuned to process causality in moving visual stimuli such as launching displays. STS in primates has previously been shown to contain cells that are especially responsive to hand–object interactions that are perceived to be causal [14,15]. Our results suggest that human STS may be specialised to process the nature of very basic causal interactions between objects.

The intraparietal sulcus is known to contain cells that code spatial relationships [18–20]. That activity in the left intraparietal sulcus was significantly greater for causal than for non-causal stimuli in the current study may have been due to the presence of the specific spatial relationships between the balls in the causal displays that were absent in the non-causal displays. It was the spatial relationship between the red and the blue ball that determined their collision in the causal displays. Thus, the left intraparietal sulcus may be specifically involved in proces-

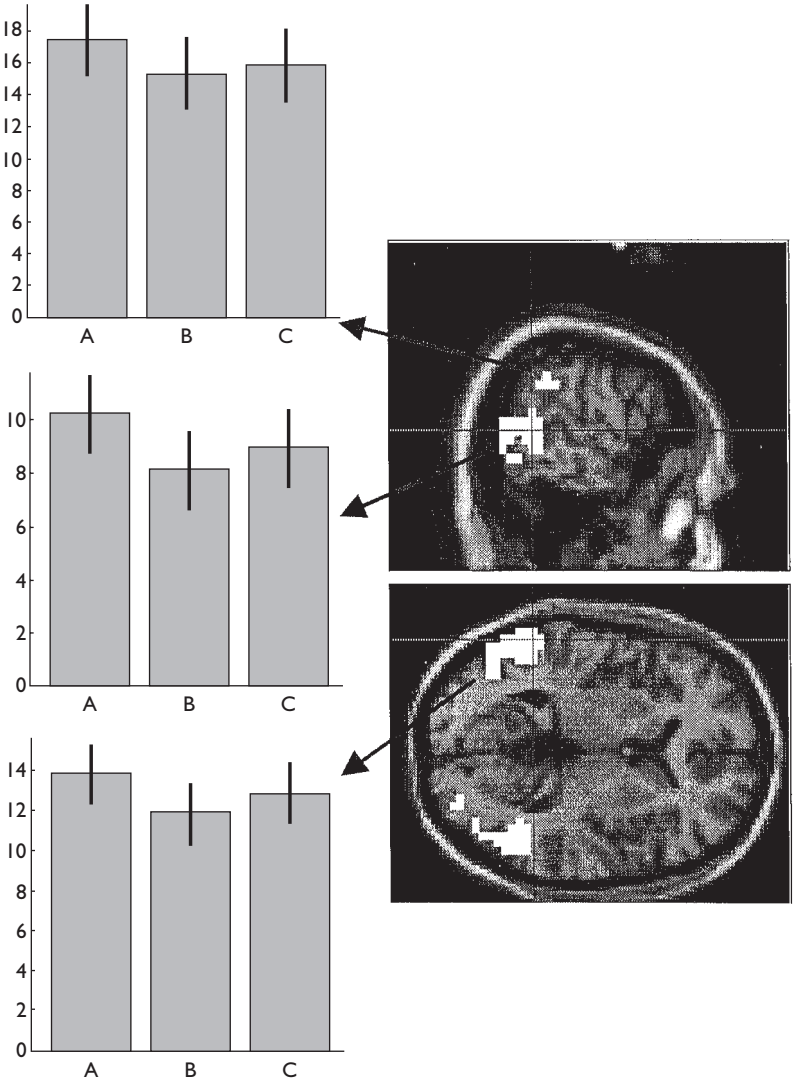

Fig. 2. Image showing activation of bilateral MT/V5, bilateral STS and the left intraparietal sulcus resulting from the comparison between causal and non-causal events and inclusively masked by the comparison between causal and visual transient events at $Z = 3.09$. The graphs show the relative haemodynamic response (in arbitrary units) to causal (A), non-causal (B) and visual transient (C) events in the voxels of maximum intensity in the left intraparietal sulcus (top), left STS (middle) and the left MT/V5 (bottom). A similar pattern of haemodynamic response was observed in right MT/V5 and right STS.

sing spatial relationships that involve some kind of causal contingency between objects.

The major difference between the conditions in the current study was the perception of a causal contingency between the balls in the causality events and the lack of such a causal contingency in the control events. Although the stimuli were designed to be as similar as possible on all factors other than causality, the possibility that the visuospatial motion in the causality event is more complex than in the other two visual events cannot be ruled out. The conclusion that the increased activity in MT/V5, the STS and the left intraparietal sulcus during the causality events was specifically related to the perceived causal nature of the events and not to some other visuospatial factor requires further experimentation.

Past experiments have demonstrated that perceiving causality in visual displays is automatic, irresistible and unaffected by higher-level processes. In this way, perceiving mechanical causality seems to be a bottom-up process, with top-down processes having little influence on the percept of causality [11]. The current fMRI experiment sought to test this hypothesis by investigating neural responses to causal displays during two different judgement contexts. Subjects were asked to make responses based on either the direction of motion of the stimuli (thus their attention was directed away from the causal nature of the stimuli) or the presence of causality in the displays (their attention was directed to the causal nature of the stimuli). Attention to causality had no significant effect on the neural processing of causality. This supports the hypothesis that attention to simple billiard-ball causality has little or no top-down influence on the way in which

the brain processes such causal stimuli. However, caution is required when interpreting this result. The possibility that the particular task manipulation used in this study was simply ineffective at elucidating a top-down effect on causality-evoked activity, which may be elucidated by some other task manipulation, cannot be ruled out. This question requires further investigation.

CONCLUSION

This study was designed to investigate the neural correlates of the perceived causality in Michotte-like launching displays. The results are consistent with the view that the perception of simple mechanical causality reflects relatively low-level perceptual processing. Activity in bilateral MT/V5 and STS and the left intraparietal sulcus was significantly greater to visual events that involved causality than to similar visual events that did not involve causality. In addition, directing attention to the causal nature of the stimuli had no significant influence on the brain regions involved in processing causality. These results support theories of causality that claim that the visual system is wired to recover the causal structure of the world.

REFERENCES

1. Hume D. *Treatise of Human Nature*. Oxford: Oxford University Press, 1739/1978.
2. Michotte A. *The Perception of Causality*. New York: Basic Books; 1946/1963.
3. White PA. *The Understanding of Causation and the Production of Action.* London: Erlbaum; 1995.
4. Scholl BJ and Tremoulet PD. *Trends Cogn Sci* **4**, 299–309 (2000).
5. Gordon IE, Day RH and Stecher EJ. *Perception* **19**, 17–20 (1990).
6. White PA and Milne A. *Am J Psychol* **110**, 573–602 (1997).
7. White PA and Milne A. *J Exp Psychol Gen* **128**, 499–516 (1999).
8. Leslie AM. *Perception* **11**, 173–186 (1982).
9. Oakes L and Cohen L. *Cogn Dev* **5**, 193–207 (1990).
10. Baillargeon R, Kotovsky L and Needham A. The acquisition of physical knowledge in infancy. In: Sperber D, Premack D and Premack AJ, eds. *Causal Cognition*. Oxford: Clarendon Press; 1995, pp. 79–116.
11. Schlottmann A and Shanks D. *Q J Exp Psychol* **44**, 321–342 (1992).
12. Pylyshyn ZW. *Behav Brain Sci* **22**, 341–423 (1999).
13. Fodor JA. *The Modularity of Mind*. Cambridge, MA: MIT Press; 1983.
14. Perrett DI, Mistlin AJ, Harries MH and Chitty AJ. Understanding the visual appearance and consequences of hand actions. In: Goodale MA, ed. *Vision and Action: The Control of Grasping*. Bristol: Intellect Books; 1990, pp. 163–180.
15. Perrett DI, Harries MH, Bevan R *et al*. *J Exp Biol* **146**, 87–114 (1989).
16. Watson JD, Myers R, Frackowiak RS *et al*. *Cerebr Cortex* **3**, 79–94 (1993).
17. Orban GA, Sunaert S, Todd JT *et al*. *Neuron* **24**, 929–940 (1999).
18. Sakata H, Shibutani H, Kawano K and Harrington TL. *Vision Res* **25**, 453–463 (1985).
19. Coull JT and Nobre AC. *J Neurosci* **18**, 7426–7435 (1998).
20. Bremmer F, Schlack A, Duhamel JR *et al*. *Neuroimage* **14**, S46–51 (2001).
21. Dale AM. *Hum Brain Mapp* **8**, 109–114 (1999).
22. Friston KJ, Zarahn E, Josephs *et al*. *Neuroimage* **10**, 607–619 (1999).
23. Dale AM and Buckner RL. *Hum Brain Mapp* **5**, 329–340 (1997).
24. Buckner RL, Goodman J, Burock M *et al*. *Neuron* **20**, 285–296 (1998).
25. Josephs O and Henson RNA. *Phil Trans R Soc Lond B Biol Sci* **354**, 1215–1228 (1999).
26. Miezin FM, Maccotta L, Ollinger JM *et al*. *Neuroimage* **11**, 735–759 (2000).
27. Friston KJ, Ashburner J, Frith CD *et al*. *Hum Brain Mapp* **3**, 165 (1995).
28. Friston KJ, Holmes AP, Worsley KJ *et al*. *Hum Brain Mapp* **2**, 189-210 (1995).

Acknowledgements: We are grateful to R. Henson for advice at the analysis stage of this study. This work was supported by the INSERM France, the Programme Cognitique from the French Ministry of Education and the Wellcome Trust, UK. S.-J.B. is supported by a Wellcome Trust International Travelling Research Fellowship.