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Neural mechanisms involved in error processing: A comparison of errors made with and without awareness

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The ability to detect an error in one's own performance and then to improve ongoing performance based on this error processing is critical for effective behaviour. In our event-related fMRI experiment, we show that explicit awareness of a response inhibition commission error and subsequent post-error behaviour were associated with bilateral prefrontal and parietal brain activation. Activity in the anterior cingulate region, typically associated with error detection, was equivalent for both errors subjects were aware of and those they were not aware of making. While anterior cingulate activation has repeatedly been associated with error-related processing, these results suggest that, in isolation, it is not sufficient for conscious awareness of errors or post-error adaptation of response strategies. Instead, it appears, irrespective of awareness, to detect information about stimuli/responses that requires interpretation in other brain regions for strategic implementation of post-error adjustments of behaviour.

Keywords: Neural mechanisms; Error processing; fMRI

Introduction

Our ability to monitor ongoing performance is an executive function critical to behavioural control, in particular the processing of errors, which serves an adaptive function in signalling to an individual that an ongoing task has increased in difficulty and that the intervention of other attention or control processes would potentially be advantageous (Gehring et al., 1993; Ullsperger and von Cramon, 2001). The neural basis of error-processing has become a key research interest in cognitive neuroscience, not only because of its importance to these cognitive skills and to the mechanisms by which cognitive control is implemented, but also because understanding its cortical network may offer insights into the dysfunctions of self-monitoring seen in a range of clinical conditions (Carter et al., 2001; Forman et al., 2004; Gehring et al., 2000; Kaufman et al., 2003; Mathalon et al., 2003). Studies of neural responses to performance errors have suggested that the prefrontal (PFC) and anterior cingulate (ACC) cortices are critical to error processing (Garavan et al., 2003), but the precise roles these regions play remains debated (Bush et al., 2000).

To date, neuroimaging studies have focussed primarily on identifying the neural regions involved in error detection (Kiehl et al., 2000; Menon et al., 2001). An important distinction exists between error detection and error awareness. The cognitive neuroscience theories that characterise how a performance error is processed by the brain focus almost exclusively on error detection, without assuming that an individual is conscious of this process (see Yeung et al. (2004) for an interesting exception). It is therefore possible for an error to be detected by the brain and behavioural correction to occur, without the individual being aware of either phenomenological experience. For the purposes of this study, error awareness is defined as the explicit recognition of a performance error via a specific 'awareness' button press response. Nieuwenhuis and colleagues (2001) were the first to examine the neural correlates of error awareness, identifying with event-related potentials (ERPs) that the error-negativity (Ne/ERN), typically localised to the ACC region and associated with error detection (Dehaene et al., 1994; Gehring et al., 1993; Scheffers et al., 1996), following unperceived eye-movement errors did not correspond with conscious awareness of an error. Rather, another ERP component, a positivity associated with errors or Pe, directly related to error awareness. The Pe is argued to be a P3-like

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positivity that is maximal at midline parieto-central scalp sites (Falkenstein et al., 2000; Vidal et al., 2000).

The site and specificity of the error positivity response is of great interest, as it may reveal cortical regions critical to error awareness. Previous studies attempting to localise the source of the Pe response have yielded mixed results, finding a distribution of sites that included dorsolateral, cingulate, mesiotemporal and orbitofrontal cortex using intracranial recording (Brazdil et al., 2002), while ERP source localisation studies have typically suggested ACC generators (Herrmann et al., 2004; van Veen and Carter, 2002). One limitation of these three studies was the absence of the type of 'awareness' comparison performed by Nieuwenhuis et al. (2001) between errors recognised by participants and those of which they remained unaware. The specificity of this response and its likely cortical generator are of great interest, as researchers have already begun to probe the neurobiological basis of error processing deficits (including error awareness) in clinical conditions such as Alzheimer's disease and schizophrenia by measuring the Pe (Mathalon et al., 2002; Mathalon et al., 2003).

Here, we utilised the higher spatial resolution of fMRI to address the neural mechanisms that are associated with error awareness and post-error behaviour.

Methods

Subjects

Thirteen subjects (6 female, mean age 28, range: 21–41) participated in the experiment; all were right-handed and reported no history of neurological symptoms. Subjects were fully informed of the nature of the research and provided written consent for their involvement in accordance with the Institutional Review Board of the Nathan Kline Institute.

Behavioural task

To examine conscious recognition of errors, we developed the Error Awareness Task (EAT) (see Fig. 1), a motor Go/No-go response inhibition task in which subjects make errors of commission of which they are aware (Aware errors) or unaware (Unaware errors). The task presents a serial stream of single colour words in congruent fonts, with the word presented for 900 ms followed by a 600 ms inter-stimulus interval. Subjects were trained to respond to each of the words with a single 'Go trial' button press and withhold this response when either of two different circumstances arose. The first was if the same word was presented on two consecutive trials (Repeat No-go), and the second was if the word and font of the word did not match (Stroop No-go). By having competing types of response inhibition rules, we aimed to vary the strength of stimulus–response relationships, whereby representations of rules competitively suppress one another such that the more prepotent rule would suppress the weaker rule and so produce a significant number of errors, a small proportion of which may go unnoticed due to focussing primarily on the prepotent rule. In particular, we aimed to capitalise on the overlearned human behaviour of reading the word rather than the colour of the letters (the Stroop effect) and so predispose subjects to monitor for the Repeat rather than the Stroop No-gos. Subjects were trained to press a different 'error awareness' button on the trial following any commission errors and were not required to make the standard Go response.

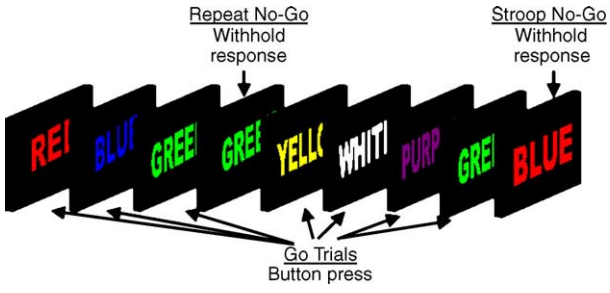

Fig. 1. The Error Awareness Task required subjects to respond with a button press to a stream of colour words and withhold their response when either a word was repeated on consecutive trials or the font and word were incongruous. Subjects were trained to press a different button following any commission errors.

An 'Oddball condition' was also administered to identify activations associated with the changed response demands of the Aware errors. This condition replicated the stimuli and timing from the EAT task except that No-go trials were replaced with the word 'STOP'. Subjects were instructed to respond to each trial with the 'Go trial' button and press the 'error awareness' button on the trial following 'STOP' stimuli, though they were not required to inhibit their response to the 'STOP' trials. Oddball events therefore represented similar response and decision requirements to Aware errors, without the subject making an error.

Six blocks (5 EAT and 1 Oddball) of 225 trials were administered to subjects during fMRI data acquisition. An event-related design was employed, distributing 128 No-go events pseudo-randomly throughout the serial presentation of 1125 Go trials, having the dual advantage of mixing frequent responses and infrequent response inhibitions to maintain response prepotency and separating the events of interest sufficiently so that correct and failed response inhibition events could be analysed separately without signal cross-contamination. Subjects were informed prior to the final block of trials that the Oddball condition was to begin, which contained 25 Oddball trials distributed within 225 Go trials.

Scanning parameters

All scanning was conducted on a 1.5 T Siemens VISION scanner in which foam padding was used to restrict head movements. Contiguous 5 mm sagittal slices covering the entire brain were collected using a single-shot, T2*-weighted echo planar imaging sequence (TE = 50 ms; TR = 2000 ms; FOV = 256 mm; 64 × 64 mm matrix size in-plane resolution). High-resolution T1-weighted structural MPRAGE images (FOV = 256 mm, isotropic 1 mm voxels) were acquired following functional imaging to allow subsequent activation localisation and spatial normalisation. Stimuli were delivered using an IFIS-SA stimulus-delivery system (MRI Devices Corp., Waukesha, Wisconsin), which was equipped with a 640 × 480 LCD panel. This shielded LCD screen is mounted on the head-coil, directly in the subjects' line of vision.

All analyses were conducted using AFNI software (Cox, 1996). Following image reconstruction, the time-series data were time-shifted using Fourier interpolation to remove differences in slice acquisition times and motion-corrected using 3D volume registration (least-squares alignment of three translational and three rotational parameters). Activation outside the brain was also removed using edge detection techniques. No subjects showed