Sensing and seeing associated with overlapping occipitoparietal activation in simultaneous EEG-fMRI

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Abstract

The presence of a change in a visual scene can influence brain activity and behavior, even in the absence of full conscious report. It may be possible for us to *sense* that such a change has occurred, even if we cannot specify exactly where or what it was. Despite existing evidence from electroencephalogram (EEG) and eye-tracking data, it is still unclear how this partial level of awareness relates to functional magnetic resonance imaging (fMRI) blood oxygen level dependent (BOLD) activation. Using EEG, fMRI, and a change blindness paradigm, we found multi-modal evidence to suggest that *sensing* a change is distinguishable from being *blind* to it. Specifically, trials during which participants could detect the presence of a colour change but not identify the location of the change (*sense* trials), were compared to those where participants could both detect and localise the change (*localise* or *see* trials), as well as change blind trials. In EEG, late parietal positivity and N2 amplitudes were larger for *localised* changes only, when compared to change blindness. However, ERP-informed fMRI analysis found no voxels with activation that significantly co-varied with fluctuations in single-trial late positivity amplitudes. In fMRI, a range of visual (BA17,18), parietal (BA7,40), and mid-brain (anterior cingulate, BA24) areas showed increased fMRI BOLD activation when a change was *sensed*, compared to change blindness. These visual and parietal areas are commonly implicated as the storage sites of visual working memory, and we therefore argue that sensing may not be explained by a lack of stored representation of the visual display. Both *seeing* and *sensing* a change were associated with an overlapping occipitoparietal network of activation when compared to *blind* trials, suggesting that the quality of the visual representation, rather than the lack of one, may result in partial awareness during the change blindness paradigm.

Keywords: EEG-fMRI; change blindness; sensing; conscious awareness

Introduction

It is common for us to overestimate the amount of information that we can process and store about the world around us. Although we may assume that we would notice a cyclist entering the path of our car, or if a building on our street changed in colour, we very often miss these occurrences (Simons and Levin, 1997; Simons, 2000). The failure to detect changes between visual scenes is known as change blindness, and is used as evidence to

suggest that our internal representation of the outside world is not as complete as once thought (Noe *et al.*, 2000; Rensink, 2004). When changes to an image are disrupted in some way, for example by a distractor image or a visual saccade, we cannot use visual transients (or motion) to detect them, and are often blind to the difference (Rensink *et al.*, 1997; Kanai and Verstraten, 2004).

It was previously assumed that if we are blind to a change then we cannot provide any information about it, and that the

change should not influence our behavior in any way. Blindness to changes is thought to result from a lack of detailed representation about the pre- and post-change scenes, or an inability to successfully compare the two (Simons, 2000). If this is the case, then our knowledge when we are blind to changes should be equivalent to that when there is no change at all. Anecdotally, this does not align with the experience of observers in a change blindness experiment; it is common for them to remark that they suspected something had changed, but that they were not sure about its nature or location. This experience appears to be phenomenologically different from complete change blindness, but how this difference is reflected in behavioral and neuroimaging data is unclear.

In the domain of visual consciousness, there is a recurring debate on the nature of visual awareness; whether it is graded, or dichotomous, or a combination determined by the context. In the Global Neuronal Workspace Theory (Dehaene and Naccache, 2001; Dehaene et al., 2006), it is posited that awareness arises when inputs cross a threshold for "ignition," resulting in the distribution and maintenance of information within a "global workspace." Based on this proposal, conscious awareness is a dichotomous state, as only inputs selected by attention can spark the activation of the global workspace. This consists of a large network of connected regions, including prefrontal and parietal regions as well as the thalamic nuclei and basal ganglia (Dehaene and Changeux, 2011). Therefore, conscious awareness requires directed attention and activation of a distributed frontal-parietal network, in an "all-or-nothing" fashion.

In accordance with this, functional magnetic resonance imaging (fMRI) studies specifically investigating change blindness report that detected changes are associated with greater activation in the parietal lobe, dorsolateral prefrontal cortex, and fusiform gyrus, when compared to changes that are missed (Beck et al., 2001). Few regions were specifically activated when participants exhibited change blindness compared to no-change trials (right lingual gyrus, right fusiform gyrus, and right inferior frontal gyrus). Further, detected changes compared with correctly categorized no-change trials revealed activation in a wider network including the inferior, superior, and medial temporal gyrus, anterior interparietal sulcus, precuneus, central sulcus, inferior frontal gyrus, anterior cingulate cortex (ACC), putamen, pulvinar, and cerebellum (Pessoa and Ungerleider, 2004). A similar pattern was identified for false alarm trials, where participants reported a change when no change occurred, suggesting that activity was related to the participants' perception of the change rather than properties of the visual stimulus.

However, this "all-or-nothing" explanation of visual awareness does not align with our subjective experience of the world. Based on participants' report of a sense for something changing, we might conclude that awareness is graded. This allows for a level, or levels, of awareness lying somewhere on a continuum between full and absent awareness. In an early experiment, Rensink (2004) suggested the presence of a *sense* condition, in which observers could detect a change without fully identifying it. Observers were asked to indicate when they "thought" that something had changed, and then again when they were certain of it. He argued that this *sense* condition is both phenomenologically and perceptually distinct from the traditionally reported *see* condition in which participants are fully aware of what change occurred.

This definition has been extended and explored using electroencephalogram (EEG) and eye-tracking, with a range of results suggesting a richer visual experience than either "yes I saw a change" or "no I didn't see anything" (Thornton and Fernandez-Duque, 2001; Fernandez-Duque and Thornton, 2003; Galpin et al., 2008; Kimura et al., 2008; Busch et al., 2009; Lyyra et al., 2012; Howe and Webb, 2014; Reynolds and Withers, 2015; Chetverikov et al., 2018). The distinction could be described by the "partial awareness hypothesis" (Kouider and Dehaene, 2007; Kouider et al., 2010). While the mechanism of awareness can still be considered dichotomous and dependent on an ignition threshold, the level of detail contained within the workspace is variable. Stimuli can be represented with varying detail, based on factors such as stimulus strength, therefore giving rise to graded knowledge of its contents.

In a previous EEG experiment (Scrivener et al., 2019), we distinguished between trials in which participants could detect the presence of a colour change but not identify the location of the change (*sense* trials), versus those where participants could both detect, and localise the change (*localise* trials). We chose to measure several ERPs that are commonly linked to visual attention and awareness, including the visual P1 and N1, visual awareness negativity (VAN), N2pc, and late positivity (LP) (Koivisto and Revonsuo, 2010; Förster et al., 2020). Although suggested as one of the earliest reflection of conscious visual awareness (around 200 ms after stimulus onset), we found no statistically significant differences in the VAN ERP across conditions, contrary to previous findings (Wilenius and Revonsuo, 2007; Koivisto et al., 2008; Busch et al., 2009; Förster et al., 2020).

In a similar time window, the N2pc is characterized by an increased negativity at visual electrodes contralateral to the change location, and is increased for aware versus unaware trials (Luck and Hillyard, 1994; Schankin and Wascher, 2007). In our previous results, both awareness conditions (*localise* and *sense*) were significantly different to trials with no change detection (*blind* trials), suggesting that the N2pc is not dependent on explicit awareness. It is possible that *sense* trials elicited a shift in attention to the correct hemifield of change (and therefore an N2pc was detected), but that this was not specific enough to determine the exact location of the change.

Within the LP range (400–600 ms after change onset), all conditions were significantly different from one another. The LP overlaps with the P3 component at central parietal electrode sites, and is often associated with conscious aspects of task processing (Busch et al., 2009; Koivisto et al., 2009; Railo et al., 2011). Overall, it appears that simply "detecting" a change can be distinguished from "describing" a change, in both subjective and neuroimaging results, and that participants can *sense* a change without complete knowledge of what occurred.

The main aim of this experiment was to examine the existence and nature of the *sense* condition in the change blindness paradigm, using combined EEG-fMRI and behavioral measures. While a range of evidence posits a distinction between *sense* and *blind* conditions in EEG data, no such distinction has been made for the *sense* condition in change blindness using fMRI. One criticism of the sensing hypothesis is that participants who *sense* a change are simply applying a more liberal response criterion when completing the task, and in fact are not aware of the change at all (Simons et al., 2005). Similarly, implicit awareness of changes could also be explained by explicit mechanisms such as guessing or a process of elimination (Mitroff et al., 2002). If this is the case, then we would expect to find no significant differences between *sense* and *blind* trials in fMRI BOLD activation. This result could also support the hypothesis of visual consciousness as dichotomous. However, if sensing lies somewhere on a continuum between aware and unaware, perhaps explained by varying precision of the stimuli representation

within the global workspace, then BOLD activation for *sense* trials may be separable to both fully aware and change *blind* trials.

Further, we aimed to improve the respective temporal and spatial resolution of EEG and fMRI by measuring them simultaneously. In an extension to our previous EEG results (Scrivener *et al.*, 2019), we investigated how EEG correlates of visual awareness relate to changes in fMRI BOLD. We therefore aimed to identify brain regions with BOLD activity that co-varied with activity in the EEG data, to detect possible sources or networks associated with awareness of changes.

Materials and Methods

All materials and analysis methods were pre-registered in an open document on the Open Science Framework, where the data and analysis for this project can also be found (https://doi.org/10.17605/OSF.IO/W6BH3). Structural images were defaced using Brainstorm3 (Tadel *et al.*, 2011) in MATLAB (MathWorks, Inc., version 2014a) with SPM8.

Participants

Twenty one right-handed subjects (mean ± SD, age = 21 ± 3.6, 6 male) with no history of psychiatric or neurological disorders participated in this EEG-fMRI study. All had corrected-to-normal vision and were not colour blind (based on self-report). The experiment was approved by the University of Reading ethics committee (UREC: 16/120), and was conducted in accordance with the Declaration of Helsinki (as of 2008). All participants gave informed consent to take part, including consent to share their anonymized data. For EEG and behavioral analysis, one participant was removed due to failure to remove MRI related artifacts from the EEG, leaving $N = 20$. Four additional participants were removed from the fMRI and EEG-fMRI analysis for having motion greater than one voxel size in the fMRI data, leaving $N = 16$.

Stimuli and procedure

A change blindness task was presented using Psychtoolbox (Kleiner *et al.*, 2007), on a 1920 x 1080 LCD monitor with a 60 Hz refresh rate. The paradigm was displayed on a screen approximately 47 cm away from the center of the scanner bore. This was viewed by the participant through a mirror mounted onto the coil, at approximately 12 cm from the participant's eyes. In their left hand, the participant held an alarm ball, and in their right they held a four key button box. They had to use all of the four keys to respond to the task. Participants were asked to fixate on a central fixation cross and identify changes between consecutive displays of coloured squares. These were interrupted by a short fixation display to facilitate the change blindness phenomenon (see Fig. 1 for details on display duration). On change trials, one of the squares changed colour from the first to the second display. On no-change trials, the displays were identical. This was followed by two or three questions, depending on the participant's response to the first question.

Question 1 asked "Did you see a change?" to which participants could respond "yes" or "no." Question 2 asked participants to localise the change, based on a 2 x 2 grid from top left to bottom right. Question 3 asked how certain participants were of their responses, ranging from "1: Very Uncertain" to "4: Very Certain." If participants responded "no" change to question 1, they were asked to press any button to ensure the same number of button presses were made during each trial. We did not ask participants who did not see the change to guess a location, as our hypotheses did not relate to "implicit" change detection, as reported in Fernandez-Duque and Thornton (2000). Participants were asked to respond within a limit of 2 s for each question, and trials with any response missing were not included in further analysis.

This study had a within-subjects repeated measures design, and each participant completed 5 blocks of 50 trials, meaning a total of 250 trials. Of these 250 trials, 165 contained a change in coloured square, and the remaining trials contain no change. These trial numbers were selected after consideration of the maximum amount of time participants would be willing and able to attend to the task, especially with concurrent neuroimaging recording. The ratio was not kept at 50/50, as the trials containing the change were of most interest for analysis (Fernandez-Duque and Thornton, 2003; Howe and Webb, 2014). However, after the experiment participants were asked to report the percentage of trials that they believed contained a change (none of which were correct). After each block of 50 trials, the participants were presented with a break screen, advising them to take a break. The participant was able to continue the experiment at their discretion by pressing any button on the button box. Before beginning the main task, participants were given a short block of 10 trials in which to practice responding to the paradigm with the button box. The data from this practice block was not analysed.

Difficulty was modulated in real time by adding and removing two squares from the display, based on the assumption that more distractors increases task difficulty (Vogel *et al.*, 2005). This was to prevent floor and ceiling performance during the task as a result of individual differences (Luck and Vogel, 2013), and optimize for performance rather than to establish specific individual thresholds. Performance over the previous two trials was used to update the current trial; two consecutive correct answers added two squares, two incorrect deducted two squares, and one correct and one incorrect resulted in no change. The decision to increase or decrease the number of squares was made using responses to the localization question (Q2), as we were specifically interested in controlling the number of *sense* and *localise* trials. The number of squares always changed by two, to balance the number on the left and right hemifields of the screen. The location of the change on each trial was random, but the change occurred an equal number of times on the left and right side. The display was divided into 36 even sections, with 6 in each quadrant, and within which the squares could appear. As the colour of the squares was not related to our main hypotheses, we used seven default MATLAB colours; blue, cyan, yellow, green, white, red, and magenta (MathWorks, Inc., version 2016b).

Behavioral analysis

The trials in which a change occurred were divided into three conditions: *blind* (no change detection), *localise* (change detection and localization), and *sense* (change detection without localization). Trials in which no change occurred were divided into *correct rejection* (no change reported) and *false alarm* (change incorrectly reported). The number of false alarm trials was low, with a mean of 10 trials (range = 1 – 28, SD = 7.34), and therefore EEG analysis comparing *false alarm* to *sense* trials was not performed due to a lack of power. The percentage of *false alarm* trials was calculated in relation to the total number of no-change trials, whereas the percentage of *sense* trials was calculated in relation to the total number of change trials.
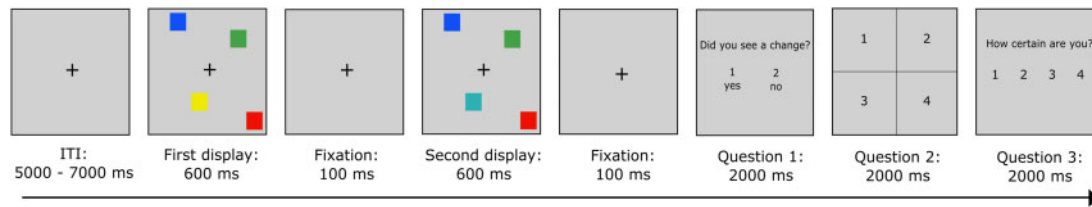
Figure 1. Illustration of the experimental paradigm. The number of squares presented varied from 2 to a maximum of 36. Question 1 asked "Did you see a change?" to which participants could respond "Yes" or "No." Question 2 asked participants to localise the change, based on a grid from top left to bottom right. Question 3 asked how certain participants were of their responses, ranging from "1: Very Uncertain" to "4: Very Certain." If participants responded "no change" to question 1, they were asked to press any button instead of the localization response

Detection accuracy for each participant was calculated based on the percentage of change trials in which they correctly detected a change. Localization accuracy was calculated as the percentage of correctly detected changes where the localization was also correct. We also recorded each participant's mean and maximum difficulty scores, with the maximum referring to the highest number of squares that were displayed to them during the experiment. Behavioral analysis was completed in JASP 2018 (version 0.8.2.0).

D'prime was calculated as a measure of participant response bias. This was calculated using the equation $d = z$(hit rate-false alarm rate) (Stanislaw and Todorov, 1999). Response bias, or criterion, was also calculated, where $c = -0.5 * (z$(hit rate-false alarm rate)) . $c = 0$ indicates no response bias to either "yes" or "no" responses. $c > 0$ indicates a bias toward "no" responses, with fewer hits, and fewer false alarms. $c < 0$ indicates bias toward "yes," with more hits but also more false alarms.

One problem faced in identifying a *sense* condition is that it is difficult to distinguish these trials from those where participants did not really see a change (similar to a false alarm during no change trials), or those where participants press the wrong response key (Mitroff *et al.*, 2002; Simons and Ambinder, 2005). Rensink (2004) found that reaction times when participants thought that they had seen a change were shorter for change trials than no-change trials, meaning that participants were slower when they were simply making a false alarm. Galpin *et al.* (2008) also found greater certainty associated with *sensing* during change trials, compared to *false alarms*. We therefore compared reaction times across awareness conditions, as well as between levels of confidence. As trial numbers were low, "very uncertain" and "uncertain" responses were combined, and "certain" and "very certain" were combined. Each awareness condition therefore had two levels of certainty; for example, *localise certain* and *localise uncertain*.

To establish if the location of the change influenced the likelihood that it was detected, we conducted two chi-square analyses. The first analysis divided the 6 x 6 grid of possible change locations into two conditions, outside and central. Changes occurring in any of the 20 outermost locations were considered to be outside changes, and the 16 central locations were considered to be central. We ran a 2 x 3 chi-square with the independent variables of location (outside/central) and awareness (*blind/localise/sense*), and the dependent variable as the frequency of trials within each condition, across participants. In the second analysis, we instead compared the side of the display in which the change occurred, resulting in a 2 x 3 chi-square for hemisphere (left/right) and awareness (*blind/localise/sense*).

A chi-square test was used to compare the frequency of each colour change occurring across awareness conditions (*see/blind*). Due to the large number of possible random colour changes, *localise*, and *sense* trials were combined into *see* trials. Each possible colour change was coded with a number between 1 and 21, such that a change in either direction was coded with the same number. colour changes with an adjusted standardized residual >1.96 were considered to have a significant difference frequency across conditions (Agresti, 2003).

EEG data acquisition

EEG data was recorded with an MRI-compatible cap equipped with carbon-wired Ag/AgCL electrodes (Braincap MR) from 64 scalp positions according to the international 10-10 system. The reference electrode was placed at FCz and the ground at AFz. An additional ECG electrode was positioned on the back to measure heart rate. An MRI-compatible EEG amplifier was used (BrainAmp MR, Brain Products) with a sampling rate of 5000 Hz. This was positioned at the back of the scanner bore and connected using ribbon cables that were secured with sandbags. Impedance was kept below 10 kΩ for EEG channels and 5 kΩ for the ECG. EEG recordings were performed with Brain Vision Recorder Software (Brain Products) and timings kept constant using a Brain Products SyncBox to synchronize EEG with the MRI system clock.

EEG pre-processing

Raw EEG data was pre-processed using Brain Vision Analyzer version 2.1 (Brain Products). Correction for the MR gradient artifact was performed using a baseline corrected sliding average of MR volumes (Allen *et al.*, 2000). Removal of cardioballistic artifacts involved the subtraction of heartbeat artifacts on a second by second basis, using a sliding average of 21 (Allen *et al.*, 1998). The delay was detected using the CBC detection solution, individually for each subject. Peaks were detected semi-automatically, with a manual check of the algorithm's estimations. Independent component analysis (ICA) (Infomax; Bell and Sejnowski, 1995) was then used to remove further BCG residual artifacts (range: 1–4 additional independent components (ICs) removed per participant). As outlined in Debener (2005), the presence of visual P1 and N1 peaks in the averaged data after pre-processing was used as an indication of the successful removal of artifacts.

The data was downsampled to 500 Hz to reduce computation time and then filtered with a high-pass filter of 0.1 Hz to remove low frequency drift (Butterworth, second order). Contrary to the pre-registration, a low-pass filter of 40 Hz (rather than 50 Hz) and a notch filter of 50 Hz were chosen to remove line noise. We used a lower cutoff frequency given the extent of high-frequency noise in the EEG data, some of which was related to the helium

pump artifact. An ideal low-pass filter should remove all 50 Hz line noise, however, an additional notch filter was included to remove any residual noise at this frequency. ICA was used to remove eye-movement artifacts (Infomax; Bell and Sejnowski, 1995). Two components were removed for each participant; one corresponding to eye-blinks and the other to lateralized eye-movements.

Further analysis was completed using EEGLab (Delorme and Makeig, 2004). Trials were marked as outliers if any ERP value was >3 SDs from the mean value of that ERP across all trials (using the MATLAB function "isoutlier"). Note that we only searched for outliers in the electrodes used for analysis (P07, P08, Cz, Pz, and CPz). Trials marked as containing outliers were excluded from further analysis (M = 7 trials, SD = 12.98), as well as those where a response to any question was not made within the response time (M = 2 trials, SD = 2.79). This step also differed slightly from the pre-registration, where we originally planned to use the data inspection feature in Brain Vision Analyzer to remove unwanted trials. However, we decided to use a more specific outlier detection method, focusing on the electrodes and time windows that were used in the analysis. We therefore chose not to remove trials where the artifacts were contained within one or two electrodes that were not used in the analysis, as this would have reduced the number of available trials.

Segments were then taken from –200 ms to 7000 ms to include the whole trial, and baseline corrected using a mean of the data within –200 ms to 0 ms, where 0 ms was the start of the first display of coloured squares (Fig. 1). We chose the baseline period to be before the first display onset, rather than the second, as we were interested in visual ERPs that occurred in response to the both displays. It has also been suggested that ERPs in response to the first presentation of stimuli are related to the subsequent perception of change (Pourtois *et al.*, 2006).

EEG analysis

To identify the peaks of the visually evoked potentials (P1 and N1), a grand average ERP was calculated across all conditions and participants, as advised in Luck and Gaspelin (2017), from electrodes P07 and P08. From here, the peaks of interest were determined by identifying the local maxima/minima of the expected peaks, using the peak detection function in BrainVision Analyzer. The mean value within a window around the peak was used instead of the peak value, as the mean is more robust against noise (Luck, 2014). A window of 40 ms around the mean was chosen as the appropriate window for visual ERPs P1 and N1. In relation to the first display onset, the first P1 was identified at 124 ms, and the first N1 at 142 ms. In relation to the second display onset, the second P1 was identified at 108 ms, and the second N1 at 168 ms.

Based on previous literature (Fernandez-Duque *et al.*, 2003; Busch *et al.*, 2010; Tseng *et al.*, 2012), the N2pc was defined as the mean within 200–400 ms after the second display at occipital electrodes PO7 and PO8. Over central parietal electrodes Cz, CPz, and Pz, the VAN was defined within a window of 130–330 ms after the second display, and the LP within a window of 400–600 ms. We used window sizes of 200 ms, defined a-priori, in an attempt to be conservative given the large variation within the literature.

To assess how differences between early visual components across detection conditions were reflected at each stimulus presentation, P1, and N1 amplitudes were compared in two separate 2 x 3 repeated measures ANOVAs, with display (first/second), and awareness (*blind/localise/sense*) as the independent variables. Differences across hemispheres in the N2pc were analysed with another 2 x 3 repeated measures ANOVA, with the independent variables of hemisphere (contralateral/ipsilateral), and awareness (*blind/localise/sense*). Amplitudes of the VAN and the LP were compared in two separate repeated measures ANOVAs with awareness (*blind/localise/sense*) as the independent variable. Where Mauchly's Test of Sphericity indicated that the assumption had been violated, Greenhouse-Geisser correction was used. All post-hoc comparisons were two-tailed, and corrected for multiple comparisons using false discovery rate where $q = 0.05$ (Benjamini and Hochberg, 1995). Effect sizes are reported as partial eta squared for ANOVA, and repeated measures Hedge's *g* for t-tests (Lakens, 2013).

Single-trial EEG analysis

As listed a-priori in our pre-registration document on the OSF, we used two methods to extract the single-trial ERP values. The first method used the raw EEG time series, while the second used EEG values extracted from the ICA component with maximum correlation with our ERP of interest. Our reason for using both methods was to increase our sensitivity for extracting meaningful single-trial values, given the reduced signal to noise ratio in EEG data recorded inside the MR environment.

Raw values: for each ERP time window, single-trial values were calculated as the mean amplitude within the predefined window for that peak. These values were then baseline corrected by subtracting the mean amplitude within the 200 ms window before the onset of the first display. We also ran a version of this analysis where single-trial values were baseline corrected using the mean of the trial from which they were taken, in an attempt to prevent any remaining linear trends in the data influencing the general linear model (GLM) model. However, this did not affect the results. Outliers were identified as trials where the amplitude was more than 3 SDs away from the mean amplitude for that ERP. As large artifacts can raise the mean amplitude, we added the additional classification of outliers at values $+/- 30 \mu V$. These outlier values were replaced by the mean value across all other trials, as outlined in Bénar *et al.* (2007).

ICA derived values: this method was similar to that mentioned above, with the exception that the single-trial values were taken from a single ICA component, identified separately for each participant. First, ICA was computed on the pre-processed data for a single subject (FastICA in EEGLab; Hyvärinen and Oja, 1997). For each IC extracted, a mean IC-ERP was calculated by averaging the time course across all trials. The average IC-ERP time courses were then correlated with the average ERP time course from the electrodes of interest in the pre-processed EEG data; for the LP this was the average ERP from the central electrodes (Cz, Pz, and CPz). The IC component with the highest correlation with the ERP of interest was inspected to ensure that the topology was as expected; for the LP this was positivity over the central electrodes. Once selected, the single-trial values were extracted from the time series of this component, as described above. For some participants, the IC with the highest correlation was an artifact component, identified by visually inspecting the component's time series, topography, and frequency spectrum in EEGLab. We also utilized the EEGLab function "ICLabel" to aid classification of artifact components. When this was the case (three participants), the IC with the next highest correlation was selected for that participant.

fMRI recording

MRI data was acquired using a 3.0-T whole-body MRI scanner (Prisma, Siemens) and a 64 channel coil for functional imaging. Interleaved slices were recorded using a 2D echo planar imaging sequence [repetition time (TR) 1630 ms; echo time (TE) 30 ms; flip

angle 90°; voxel size 3 x 3 mm; gap 3 mm; encoding direction A to P; distance factor 20%; FOV read 192 mm; number of slices 30; transversal orientation]. Three dummy scans were acquired at the beginning of each block. As well as the functional scans, an anatomical scan of the entire brain was acquired [3D MPRAGE; saggital; TE 2.37 ms; TR 1800 ms; flip angle 8°; voxel size 0.98 x 0.98 mm; FOV read 250 mm; slice thickness 0.85 mm; slices per slab 208; ascending acquisition; phase encoding direction A to P].

fMRI pre-processing

MRI images were pre-processed using the procedure recommended in SPM12 (Wellcome Department of Imaging Neuroscience, Institute of Neurology, London, UK). Functional images were first re-aligned per experimental block. These were registered to the mean image with a sixth degree spline interpolation. Following this was co-registration of the structural image to aligned functional images, segmentation of white and gray matter, normalization of functional images using the deformation field created during segmentation, and normalization of the functional to structural. The resulting data was smoothed with a 4-mm full-width-half-maximum Gaussian Kernel, and a high-pass filter with a cut off period of 128 s was applied. The registration of images was checked visually at each stage. Parameters not specified here can be assumed as the default SPM parameters.

fMRI analysis

During first-level analysis, GLMs with event-related designs were conducted in SPM12, to identify voxels activated in response to trial type (*blind/localise/sense/false alarm/correct rejection*). Regressors were created for each trial type by convolving the stimulus onset times with the canonical hemodynamic response function across all blocks (Friston *et al.*, 1994). Each regressor had a duration matched to the length of visual display, and serial correlations were corrected using the AR(1) method. All fMRI analysis was conducted in relation to the onset of the second display, where the change could occur. However, given the fast presentation of the two displays, it is possible that activation from the first display contributed to the activation recorded during the second. Each block was modeled with a separate set of regressors including time derivatives, as we did not perform slice time correction. Six motion regressors were added as nuisance variables.

For each participant we ran the following contrasts during first-level analysis; *sense > blind*, *localise > blind*, *localise > sense*, *blind > no-change*, *sense > false alarm*, and *false alarm > sense*. We then compared awareness conditions at the second-level using one-sample *t*-tests. These contrasts were not run in the other direction, for example *blind > localise*, given previous results that suggest very little activation present for *blind* trials (Beck *et al.*, 2001; Pessoa and Ungerleider, 2004). An additional paired-samples *t*-test was used to identify voxels with activation that was significantly different between the pair of contrasts *localise > blind* and *sense > blind*.

The contrasts *localise > blind* and *sense > blind* should reveal voxels with activation specific to full or partial awareness of the change, respectively, compared to no awareness. As these three conditions all contain a change in coloured squares, differences between them should reflect the participant's level of awareness. Previous fMRI contrasts for change detection (detected versus undetected) have identified activation specific to awareness of the change in the bilateral parietal lobe, right dorsolateral prefrontal cortex, and right fusiform gyrus (Beck *et al.*,

2001). The corresponding contrast here is *localise > blind*, where we may expect to find similar frontal and parietal activations.

As previously discussed, it is unclear whether the contrast *sense > blind* will result in similar activations, or whether the *sense* condition is simply another form of change blindness that can be explained by overconfidence or participant guessing. The contrast between *sense* and *false alarm* trials is useful to determine if *sensing* is similar to false alarms, meaning that participants did not detect anything changing during the change trials and were overconfident in their awareness.

In the contrast *localise > sense*, we should identify voxels specifically activated when participants can both detect and localise the change, compared to only change detection. These areas would therefore be indicated in the facilitation of complete visual awareness, compared to *sensing* alone. Contrasting *blind* and no-change trials should reveal activation specific to the presence of the change, despite the participant being unable to detect it. Previous results for this contrast suggest activation in the right lingual gyrus, right fusiform gyrus, and right inferior frontal gyrus (Beck *et al.*, 2001).

To identify voxels with activation that correlated with the change in task difficulty over time, a separate GLM model was constructed with one regressor for the onsets of all trials, and a parametric regressor using the difficulty (or number of squares presented) at each trial. To identify voxels with activation that correlated with the change in participant certainty over time, a separate GLM model was constructed with one regressor for the onsets of all trials, and a parametric regressor using the certainty value reported by the participant at each trial.

Across all fMRI analyses, we report clusters with a minimum size of 20 voxels and a cluster-level family-wise error (FWE) corrected $p < 0.001$. Extended local maxima were labeled using two methods that provided overlapping results; the automated anatomical labeling toolbox in SPM (12), with a local maximum radius of 5 mm, and the SPM Anatomy toolbox, which for compatibility reasons used an older version of MATLAB (2014a) and SPM (SPM8). MNI co-ordinates were used to label voxels according to Brodmann areas. The SPM render function was used to plot our results on the cortex of an MNI brain, and MRICron was used to create multi-slice views of the *t*-score maps for each contrast of interest.

ERP-informed fMRI analysis

For ERP-informed fMRI analysis, a first-level model with one regressor was constructed for the onset of all change trials (*blind/localise/sense*), with single-trial ERP values included as a parametric regressor. The LP ERP in response to the change display was chosen a-priori for this analysis, as significant differences have previously been identified between awareness conditions within this late parietal potential (Fernandez-Duque and Thornton, 2003; Busch *et al.*, 2010; Scrivener *et al.*, 2019). A second regressor was added for the onset of all no change trials. Motion parameters were also included as nuisance variables.

An additional (not pre-registered) ERP-informed-fMRI analysis was performed, using the visual P1 and N1 amplitudes in relation to the change display. While some results suggest that ERPs as early as the P1 and N1 reflect conscious awareness, it is debated whether this is case (Koivisto and Revonsuo, 2010; Förster *et al.*, 2020). We therefore considered it valuable to check whether any regions of the brain exhibited BOLD responses that correlated with fluctuations in these early ERP responses across time and awareness conditions.

Behavioral Results

Accuracy for question 1, in which participants had to identify a change, had a mean of 54% (range $= 39 - 69\%, \mathrm{SD} = 9\%$). Accuracy for question 2, in which participants had to localise the change, had a mean of 72% (range $= 61 - 86\%, \mathrm{SD} = 8\%$). The mean difficulty level given to each participant ranged from 6 to 23 squares ($M = 16, \mathrm{SD} = 4$), with the maximum difficulty experienced by each participant ranging from 18 to 36 ($M = 27, \mathrm{SD} = 5$). D'prime scores ranged from 0.940 to 2.30 ($M = 1.38, \mathrm{SD} = 0.38$). One person had a negative criterion, meaning that they had a response bias toward false alarms. All other participants had positive criterion, indicating a conservative response strategy ($M = 0.61, \mathrm{SD} = 0.33$). D'prime scores were significantly different from 0 in a one-sampled t-test, indicating that participants could discriminate between change and no change trials, $t(19) = 16.263, \ P < 0.001$.

Mean difficulty correlated with mean location accuracy ($r = 0.543, P = 0.013$) and d'prime ($r = -0.601, P = 0.005$), but not with mean detection accuracy ($r = -0.371, P = 0.107$). Maximum difficulty also correlated with mean location accuracy ($r = 0.537, P = 0.015$) and d'prime ($r = -0.482, P = 0.031$), but not with mean detection accuracy ($r = -0.349, P = 0.131$).

The percentage of *false alarm* trials ($12.23\%\pm8.64$) was lower than the percentage of *sense* trials ($28.07\%\pm7.73$) $t(19) = -6.815$, $P < 0.001, g_{\mathrm{rm}} = 1.85$, suggesting that *sense* trials occurred more often than participants made false alarms. Additionally, the percentage of false alarms was not significantly correlated with the percentage of *sense* trials ($r = 0.198, P = 0.403$).

Out of the 20 participants included in the analysis, 15 were slower to respond when they were *blind* to the change, and compared to no-change trials. Reaction times for *blind* trials were also significantly slower than no-change trials (0.617 ± 0.176 s), $t(19) = -3.613, \ P = 0.002, g_{\mathrm{rm}} = 0.25$. Therefore, despite being *blind* to the change, the presence of a change in the display increased reaction times, and particularly for trials where the participant was uncertain.

We found a significant effect of location of the changed item (outside/central) on awareness (*blind/localise/sense*), $\chi^2(2) = 26.68$, $p < 0.001$, as participants were more likely to be *blind* to the change when it occurred on the outside of the display (*blind* outside: 911 trials, central: 619). There were also a greater number of *sense* trials for outside changes, suggesting that these changes may be harder to localise than central changes (*sense* outside: 290 trials, central: 220). The location had the least influence on *localise* trials (*localise* outside: 627, central: 619).

The hemisphere of the display in which the change occurred (left/right) had no significant effect on participant awareness (*blind/localise/sense*), $\chi^2(2) = 4.941$, P $= 0.085$ (*blind* left: 781 trials, right: 749; *localise* left: 651, right: 607; *sense* left: 236, right: 276). Additional behavioral analysis and results can be found in the Supplementary Material.

There was a significant effect of colour change on awareness, $\chi^2(20) = 120.342, P < 0.001$. Colour change pairs with a significantly higher number of *blind* trials were: yellow/green, yellow/cyan, green/cyan, blue/cyan, and white/cyan. Colour change pairs with a significantly higher number of *see* trials were: magenta/red, cyan/red, blue/red, and white/red. Overall, colour changes including red were more likely to be detected, given the prevalence of colour pairings that included red in the *see* condition. In the other direction, colour changes including cyan were more likely to be missed, as there were more *blind* trials with cyan in the colour pairing.

EEG Results

P1 and N1

For P1 amplitudes, the main effect of awareness was not significant, $F(2, 38) = 0.568, P = 0.572., \eta^2 = 0.029$. Display was also not significant, $F(1, 19) = 0.143, P = 0.709, \eta^2 = 0.007$. The interaction between awareness and display was not significant, $F(2, 38) = 3.250, P = 0.050, \eta^2 = 0.146$ (Fig. 2). (*Blind* first display $M = 1.933$, SD $= 4.106$, second $M = 1.401, \mathrm{SD} = 5.052$; *localise* first $M = 0.606$, SD $= 2.706$, second $M = 1.108, \mathrm{SD} = 5.858$; *sense* first $M = 0.509$, SD $= 2.738$, and second $M = 2.020, \mathrm{SD} = 5.900$).

For the N1, the main effect of awareness was not significant, $F(2, 38) = 2.008, P = 0.148, \eta^2 = 0.096$. Display was also not significant, $F(1, 19) = 0.68., P = 0.797, \eta^2 = 0.004$, nor was the interaction between awareness and display, $F(2, 38) = 2.046, P = 0.143, \eta^2 = 0.097$ (Fig. 2). (*Blind* first display $M = -1.526, \mathrm{SD} = 4.096$, second $M = -2.178, \mathrm{SD} = 4.469$; *localise* first $M = -3.609, \mathrm{SD} = 4.246$, second $M = -2.783, \mathrm{SD} = 5.658$; *sense* first $M = -3.500$, SD $= 3.662$, and second $M = -2.881, \mathrm{SD} = 5.279$).

N2pc

The main effect of hemisphere on N2pc amplitudes was not significant, $F(1, 19) = 0.338, P = 0.568, \eta^2 = 0.018$, nor was the main effect of awareness, $F(2, 38) = 0.878, P = 0.424, \eta^2 = 0.044$. The interaction was not significant, $F(2, 38) = 0.572, P = 0.569$, $\eta^2 = 0.029$.

As we had strong hypotheses about the presence of an N2pc for *localise* trials, we also ran corrected post-hoc pairwise comparisons across awareness levels. A significantly increased negativity across both hemispheres was found for *localise* trials ($M = -1.573, \mathrm{SD} = 4.378$) compared to *blind* ($M = -0.810$, SD $= 4.856$) P $= 0.038$. *Blind* and *sense* ($M = -1.720, \mathrm{SD} = 5.444$) were not significantly different, $P = 0.259$, nor were *sense* and *localise*, $P = 0.862$.

Visual awareness negativity

The main effect of awareness on the VAN was not significant $F(2, 38) = 0.029, P = 0.971, \eta^2 = 0.002$. (*Blind* $M = 0.059, \mathrm{SD} = 3.427$, *localise* $M = 0.184, \mathrm{SD} = 3.093$, *sense* $M = 0.104, \mathrm{SD} = 3.295$).

Late positivity

There was a main effect of awareness on LP amplitudes $F(2, 38) = 3.776, P = 0.032, \eta^2 = 0.166$. In corrected post-hoc comparisons, *localise* trials ($M = 2.270, \mathrm{SD} = 4.130$) had a significantly greater LP amplitude than *blind* ($M = 0.032, \mathrm{SD} = 2.158$), P $= 0.024$, $P_{\mathrm{bonfRM}} = 0.028$. However, *sense* ($M = 1.069, \mathrm{SD} = 3.801$) was not significantly different to *blind*, $P = 0.130, P_{\mathrm{bonfRM}} = 0.634$ or *localise* trials, $P = 0.174, P_{\mathrm{bonfRM}} = 0.446$ (Fig. 3).

fMRI Results

Awareness

For the contrast *localise* >*blind*, increased BOLD activation was found in the bilateral occipital cortex (BA17/V1, BA18/V2, and hOC4v/V4), bilateral parietal cortex (BA40/PFt, BA3b, and BA2), left putamen (BA49), left fusiform gyrus (BA37), right insula (BA13), right pre-motor cortex (BA6, spanning middle frontal, superior frontal, and pre-central gyri), and (see Fig. 4 for significant clusters and Table 1 for additional values).

For the contrast *sense* >*blind*, increased activation was found in bilateral occipital cortex (BA17/V1, BA18/V2), left pre-motor
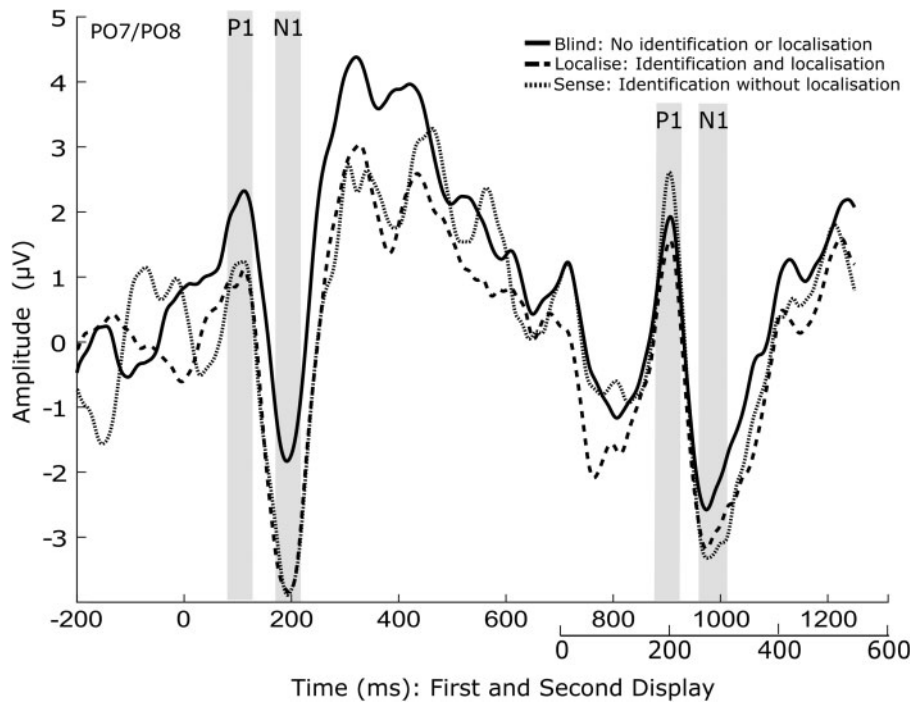
Figure 2. ERP plot showing the mean of electrodes PO7 and PO8, for each awareness condition. Condition means for the values within the shaded time windows were used for ERP analysis for the P1 and N1
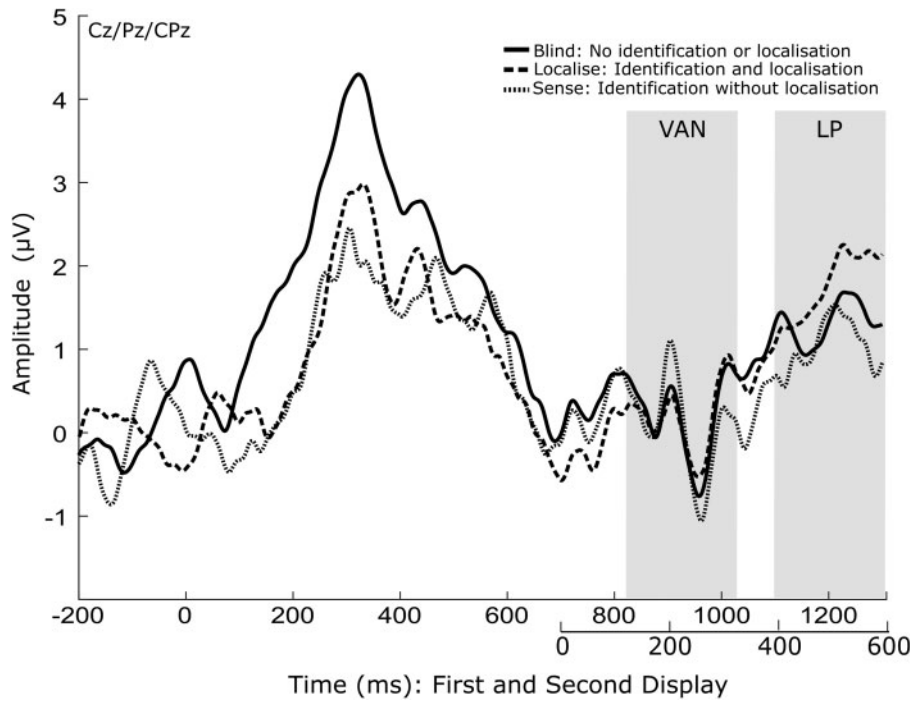

Figure 3. ERP plot showing a mean of electrodes Cz, CPz, and Pz, for each awareness condition. Condition means for the values within the shaded time window were used for ERP analysis of the VAN and LP

cortex (BA6, spanning middle frontal, superior frontal, and pre-central gyri), superior medial gyrus (BA8), parietal cortex (BA40, BA7/hIP3), and left ACC (BA32), and (see Fig. 5 for significant clusters and Table 2 for additional values).

We also looked for any activation that was significantly greater in one contrast than the other (*localise* >*blind* versus. *sense* >*blind*). However, no significant activations remained after correction for multiple comparisons. No voxels survived for the
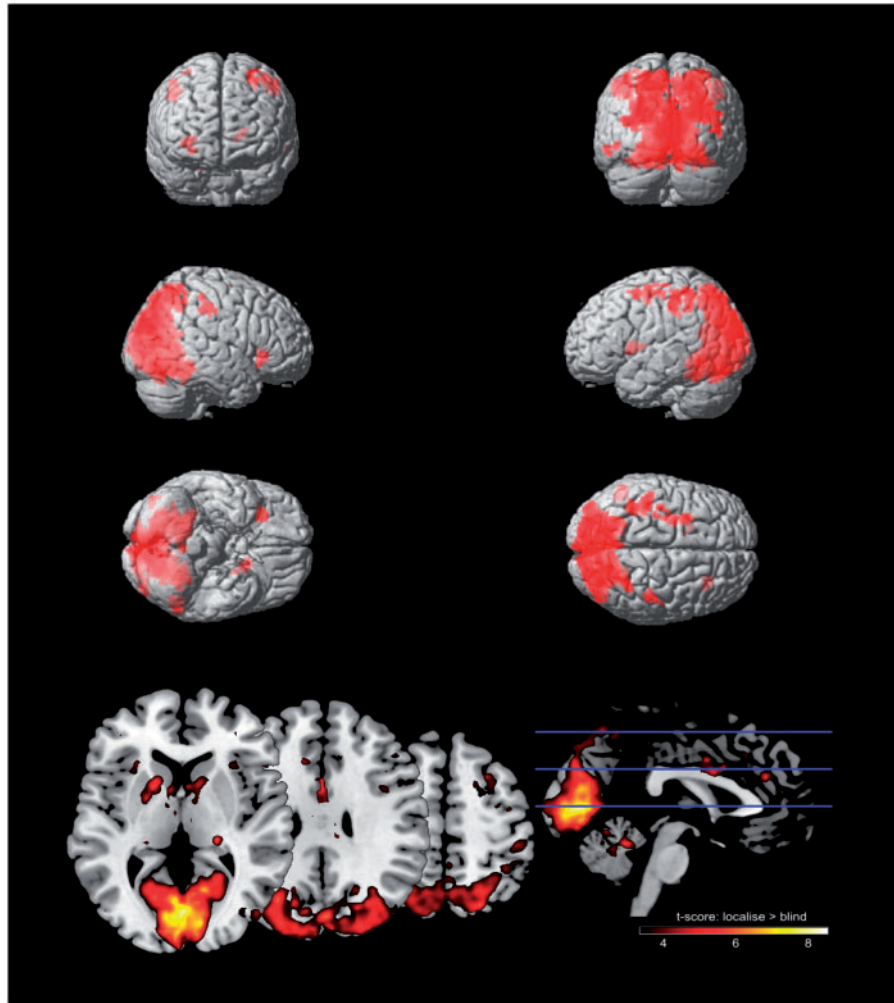
Figure 4. Top: voxels activated for the contrast *localise > blind* trials. Multiple comparisons were controlled using a cluster-level family-wise error correction where p < 0.001, as well as a minimum cluster size of 20 voxels. Bottom: *T*-scores thresholded at a minimum $t = 3$

following contrasts; *localise >sense*, *sense >localise*, *blind >no change*, *sense >false alarm*, or *false alarm >sense*.

Post-hoc conjunction analysis

Given that the contrasts *localise* versus *blind* and *sense* versus *blind* revealed similar networks of activation, we ran a conjunction analysis to determine which voxels were significantly activated in both contrasts (note that this analysis was not included in our pre-registration). To do this, we entered the two first-level contrasts for each participant into an ANOVA at the second-level (independence not assumed). We then ran a conjunction analysis across both contrasts to identify common voxels, using the conjunction null hypothesis as suggested in Nichols *et al.* (2005) . Significant activation was identified in the visual cortex (BA18, BA19) and inferior parietal cortex (BA7, BA39) (Table 5 and Fig. 6).

Difficulty and certainty

The parametric regressor of participant certainty revealed significant activation in the right visual cortex (BA17/V1, BA18/V2) and right parietal cortex (BA40) (Table 3). The parametric regressor of task difficulty (the number of squares presented per trial)

revealed significant activation in the left visual cortex (BA18, V2) (Table 4).

ERP-informed fMRI

No significant voxels were identified for the LP-informed fMRI analysis using either method for extracting the single-trial values. In addition, no significant voxels were identified using the visual P1 or N1 in relation to the change display.

Discussion

The main aim of this change blindness experiment was to distinguish between trials in which participants could both detect and localise a change in coloured square (*localise*), versus those in which they could only detect it (*sense*), or not detect it at all (*blind*), using combined EEG-fMRI. In the EEG data, the late parietal positivity ERP, *localise* trials were significantly higher in amplitude than *blind* trials as previously found (Scrivener *et al.*, 2019), but *sense* trials were not distinguishable from those where participants were *blind* to the change. Similarly, no differences were found between *sense* and *blind* trials in the N2pc or VAN. It is not clear whether this is due to false positive findings in the

Table 1. Voxels significantly activated for the contrast *localise > blind*, cluster FWE *p* < 0.001, minimum 20 voxels

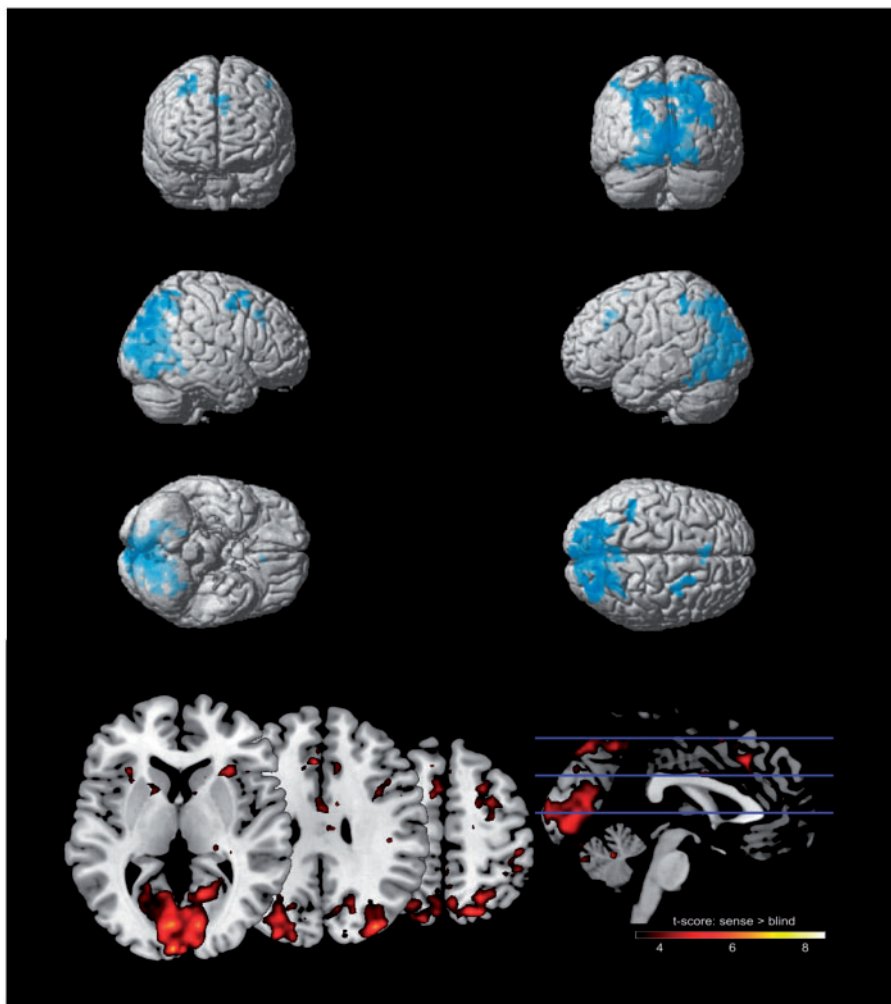| Brain region	BA	Hemisphere	Cluster size	MNI co-ordinates			Z score	T (peak level)	Cluster-level FWE (P)
				x	y	z			
Lingual gyrus	17	Right	12408	4	−82	−2	5.36	9.64	<0.001
	18 (hOC4v)	Left		−22	−70	−6			
	18	Left		−6	−76	0			
Inferior parietal cortex	40 (PFt)	Left	421	−38	−30	42	4.71	7.33	<0.001
Post-central gyrus	3 b	Left		−44	−30	52			
	2	Left		−40	−42	58			
Putamen	49	Left	90	−18	8	8	4.25	6.08	<0.001
				−22	0	6			
Middle frontal gyrus	6	Left	182	−28	4	56	4.18	5.89	<0.001
Superior frontal gyrus	6	Left		−26	−6	60			
Pre-central gyrus	6	Left		−30	−22	64			
Insula	13	Right	96	28	22	−10	4.08	5.66	<0.001
				32	28	−2			
				26	24	2			
Inferior temporal gyrus	39	Left	84	−56	−56	12	3.71	4.87	<0.001
Middle temporal gyrus	37	Left		−48	−58	−4			
				−60	−52	−6			
Inferior parietal cortex	40 (PFt)	Right	110	44	−24	38	3.62	4.69	<0.001
Post-central gyrus	3 b	Right		50	−18	40			
				40	−28	46			

Figure 5. Top: voxels activated for the contrast sense > blind trials. Multiple comparisons were controlled using a cluster-level family-wise error correction where p < 0.001, as well as a minimum cluster size of 20 voxels. Bottom: *T*-scores thresholded at a minimum *t* = 3

Table 2. Voxels significantly activated for the contrast *sense > blind*, cluster FWE *p* < 0.001, minimum 20 voxels

Brain region	BA	Hemisphere	Cluster size	MNI co-ordinates			Z score	T (peak level)	Cluster-level FWE (P)
				x	y	z			
Pre-central gyrus	6	Right	145	32	−4	48	4.94	8.07	<0.001
Middle frontal gyrus	6	Right		24	16	50			
Superior frontal gyrus	6	Right		24	8	60			
Calcarine gyrus	17	Left	5526	−4	−96	4	4.92	8	<0.001
	18	Right		12	−72	10			
Lingual gyrus	17	Left		−6	−82	2			
Superior medial gyrus	8	Left	88	−2	26	0	3.96	5.38	<0.001
Anterior cingulate gyrus	24	Left		−4	28	30			
Superior medial gyrus	8	Right		8	24	42			
Inferior parietal sulcus	40	Left	87	−48	−42	56	3.67	4.78	<0.001
	7 (hIP3)	Left		−32	−48	49			
	40	Left		−40	−44	50			

Table 3. Parametric regressor: participant certainty, cluster FWE *p* < 0.001, minimum 20 voxels

Brain region	BA	Hemisphere	Cluster size	MNI co-ordinates			Z Score	T (peak level)	Cluster-level FWE (P)
				x	y	z			
Lingual gyrus	18	Right	160	14	−74	0	4.11	5.74	<0.001
Calcarine gyrus	17	Right		6	−82	8			
				10	−88	12			
Inferior parietal cortex	40	Right	104	50	−34	48	3.84	5.12	<0.001
				48	−42	48			
				60	−34	34			

Table 4. Parametric regressor: task difficulty, cluster FWE P < 0.001, minimum 20 voxels

Brain region	BA	Hemisphere	Cluster size	MNI co-ordinates			Z Score	T (*peak* level)	Cluster-level FWE (P)
				x	y	z			
Lingual gyrus	18	Left	–	−16	−92	−6	4.23	6.02	0.003
				−10	−86	−12			

Table 5. Conjunction analysis: voxels activated for both *localise* versus *blind* and *sense* versus *blind* contrasts

Brain region	BA	Hemisphere	Cluster size	MNI co-ordinates			Z Score	T (peak level)	Cluster-level FWE (P)
				x	y	z			
Lingual gyrus	18	Left	6116	−4	−70	6	5.51	7.35	<0.001
				−4	−80	−8			
				−18	−72	−10			
Inferior parietal cortex	7	Right	445	28	−76	44	4.91	6.16	<0.001
Occipital cortex	19	Right		22	−90	22			
Inferior parietal cortex	39	Right		34	−76	38			
Inferior parietal cortex	7	Right	421	18	−70	42	4.73	5.84	<0.001
				22	−72	54			
				8	−64	52			

Cluster FWE P < 0.001, minimum 20 voxels.

previous study, the smaller signal to noise ratio in the combined EEG-fMRI data, or the relatively small sample size.

The fMRI results revealed significant differences in BOLD activation for both *localise* and *sense* trials when compared to *blind*, suggesting that they are separable to trials where participants were completely unaware of the change. These results suggest that the *sense* condition may be distinguishable from the traditional *blind* condition, meaning that subjects may have access to more information when they are able to *sense* a change. However, the contrast between *localise* and *sense* conditions
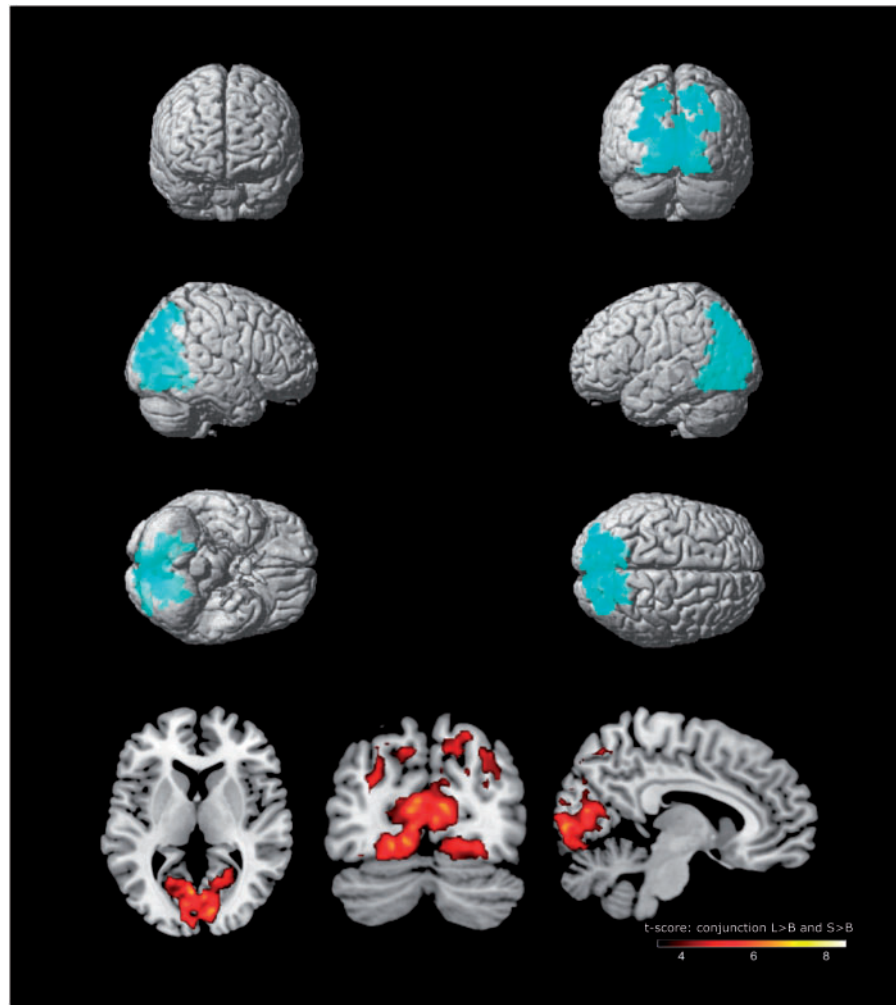
Figure 6. Conjunction analysis: voxels significantly activated for both *localise >blind* and *sense >blind* contrasts. Multiple comparisons were controlled using a cluster-level family-wise error correction where p < 0.001, as well as a minimum cluster size of 20 voxels

revealed no significant activations, and a conjunction analysis revealed overlapping activation in visual and parietal regions. These two levels of awareness may therefore be associated with activation within a similar network, and the link between brain activity and behavioral differences remains unclear.

Behavioral

One explanation for the presence of a *sense* condition in change blindness is that it reflects a liberal response criteria, such that participants report seeing a change even though they were not certain that it occurred (Simons and Ambinder, 2005). In other words, they make a "*false alarm*" during change trials. If this is the case, then these trials may be similar in number to *false alarm* trials, where participants incorrectly report a change for identical displays where they could not have seen a change. We found that participants had fewer *false alarms* than *sense* trials, and the percentage of these trials across participants was not correlated. This suggests that *sense* trials may not be attributed to a liberal response criterion of the participants, as the tendency of participants to make a *false alarm* did not influence the number of times they could *sense* a change. However, this differs from previous results, where a significant correlation was found in the

percentage of the two trial types (Scrivener *et al.*, 2019). Further behavioral data may therefore be needed to confirm this relationship.

Previous studies have also reported that participants responded "no change" more quickly for no-change trials, compared to change trials (Williams and Simons, 2000; Mitroff *et al.*, 2004). The participant's response is the same in both trial types, but the presence of a change is different. This suggests that even when they fail to detect the change in a change trial, they take longer to respond. We therefore compared reaction times for no-change trials and *blind* trials. Out of the 20 participants, 15 were slower to respond when they were blind to the change, and compared to no-change trials. Reaction times for *blind* trials were also significantly slower than no-change trials, meaning that even when participants did not notice the change, its presence increased their reaction times. It is possible that in *blind* trials, some information may be available to the participant, leading to slower reaction times, but not enough for them to be confident to report the change.

The location of the square that changed in colour during the experiment had a significant influence on the likelihood that the change was detected; changes closer to the central fixation were detected at a higher frequency across participants than those further away. One explanation is that the participants

were asked to fixate at the center of the screen, and therefore their overt attention was directed here during the trial. As attention has been found to correlate with change detection, this finding is not surprising (Rensink *et al.*, 1997).

We also found a significant effect of colour on awareness, such that changes involving the colour red were more likely to be detected, and changes involving cyan were less likely to be detected. We hypothesize that the increased salience of red squares caused a pop-out effect during these trials, leading to increase detection of the change. In the opposite direction, the colour cyan was perhaps more similar to other colours, such as white, making it more difficult to detect changes. Due to the large number of colour changes we had to collapse over the change direction (including both from and to a colour), and also over *localise* and *sense* conditions. It would be interesting to establish whether the advantage for change detection provided by the presence of red would also provide an advantage for change localization. If the colour red does initiate a pop-out effect, then we would hypothesize an advantage for both identification and localization of the change (Ball and Busch, 2015). Future studies aiming to establish more control over change detection across trials may wish to consider this effect of colour during experiment design.

Throughout the behavioral analysis presented here, the mean across trials for each participant was used as a summary statistic for further analysis. This is a relatively simple method for analysis of reaction time and accuracy data, and may not always be the most appropriate. For example, Rousselet and Wilcox (2020) outline the potential problems arising from using summary statistics, such as the mean and median, when comparing skewed distributions. Other possible approaches include hierarchical shift functions (Rouder *et al.*, 2005; Wilcox and Erceg-Hurn, 2012), designed to overcome problems caused by asymmetric distributions, and linear mixed models (Lo and Andrews, 2015; Frömer *et al.*, 2018). Using the mean to summarize complex behavior, such as reaction times, has its limitations and other statistics or methodologies may sometimes be more appropriate.

EEG

For the late parietal positivity ERP, *localise* trials were significantly higher in amplitude than *blind* trials. Other studies have also reported increased LP amplitudes for detected versus undetected changes (Fernandez-Duque and Thornton, 2003; Busch *et al.*, 2010), which has been suggested to reflect conscious awareness of changes (Railo *et al.*, 2011), and participant confidence (Eimer and Mazza, 2005). However, *sense* trials were not distinguishable from trials where participants were *blind* to the change. This contradicts our own results from a previous study where all three awareness conditions were distinguishable within the LP (Scrivener *et al.*, 2019). There is therefore increasing evidence that the LP varies reliably between detected versus undetected changes, but whether it can be detected during *sense* trials is unclear. Note that the presence of a significant LP for *localise*, but not for *sense*, should not be used as evidence that the two are different as the post-hoc comparison was not statistically significant.

For the N2pc results, it should be emphasized that the main effect of hemisphere was not significant. Therefore, the post-hoc comparison in amplitude between *localise* and *blind* trials does not reflect the traditional asymmetry of the N2pc component, with a greater negativity in the contralateral hemisphere. It can only be concluded that there was an increased negativity

for *localise* trials across both hemispheres, and may be better interpreted as an N2 component. This is a common finding, and in a review of the ERP correlates of visual awareness (Koivisto and Revonsuo, 2010) the majority of change blindness paper reported enhanced negativity in the N1–N2 range for detected changes (with the exception of Fernandez-Duque *et al.*, 2003 and Niedeggen *et al.*, 2001).

In a previous EEG study, we did find a significant N2pc for both *localise* and *sense* conditions, including a significant main effect of hemisphere (Scrivener *et al.*, 2019). We concluded that the presence of an N2pc for both awareness conditions indicated a shift in attention toward the hemisphere of the change (Luck and Ford, 1998), but that this shift in attention was not sufficient to facilitate correct localization in *sense* trials. In this experiment, we failed to find any evidence for this shift in either awareness condition, as characterized by the N2pc.

We found no statistically significant effects of awareness in the P1, N1, or VAN ERP analysis, similar to our previous results (Scrivener *et al.*, 2019). In a recent review of the ERPs associated with visual awareness (Förster *et al.*, 2020), the authors concluded that early P1 and N1 peaks are unlikely to be the earliest signature of visual awareness, and no longer discuss these peaks as possible candidates. This is due to increasing evidence against their association with conscious detection, which our EEG and ERP-informed fMRI findings cannot dispute. However, they argue that the VAN is the most likely candidate for a marker of conscious detection, and our results are contrary to several previous findings. One possible explanation is the difference in experimental paradigm. In many cases, awareness is modulated by the perceptual difficulty of the stimuli, for example by the contrast. However, the stimuli in a change blindness paradigm remain at the same contrast across all trials, and difficulty is instead modulated by the number of distractors. Another suggestion from our previous work (Scrivener *et al.*, 2019) is that the VAN requires both the location and identity of an object to be stored, such that it is available for conscious report. As our participants were not able to identify the location of the change in the *sense* condition, this may explain the lack of significant VAN ERP.

fMRI

Awareness

One aim of this experiment was to improve our knowledge of the neurological basis of the *sense* condition with the addition of fMRI results. We found largely overlapping activation for both *localise* and *sense* conditions when contrasted with trials where participants were *blind* to the change in coloured square. Both awareness conditions had significantly greater activation in the early visual cortex (B18, V2), the left supramarginal gyrus in the inferior parietal lobe (BA40), and the left pre-motor cortex (BA6).

The posterior parietal cortex and early visual cortex are commonly implicated as storage sites for the contents of visual working memory (Todd and Marois, 2005; Edin *et al.*, 2009; D'Esposito and Postle, 2015), and previous fMRI studies of change detection also found activations in these areas (Beck *et al.*, 2001; Huettel *et al.*, 2001; Pessoa and Ungerleider, 2004). Using MVPA, Christophel *et al.* (2012) identified stimuli-specific information contained in both early visual and posterior parietal areas (around the intraparietal sulcus), further implicating these regions as storage sites for visual representations. The activation of these visual and parietal regions in both *localise* and *sense* conditions suggests the presence of visual representations of the stimuli for both levels of awareness. This supports the

hypothesis that change blindness may arise from a failure to compare two displays or images, rather than a failure to encode the visual information (Hollingworth *et al.*, 2001; Simons *et al.*, 2005). Therefore, the inability of participants to localise the change during *sense* trials may not be explained by a lack of parietal representation, as activity in the dorsal stream (BA18 and BA40) was greater than during *blind* trials.

Activations found only in the *localise* contrast (but not for *sense*) were located in the primary sensory cortex (BA2, BA3b), putamen (BA49), and insula (BA13). This forms a wider network of activation than the *sense* versus *blind* contrast, including mid-brain structures. The insula and putamen are both hypothesized to act as hubs in key brain networks relating to cognitive control, and their activation specific to *localise* trials may indicate their role in facilitating full awareness of the change. More specifically, the insula forms an integrative hub between attention and salience networks (Eckert *et al.*, 2009; Menon and Uddin, 2010), balancing external attentional cues with internal performance monitoring (Uddin *et al.*, 2017). In contrast, the putamen is suggested to be a central component of a frontal-sub-cortical network (including the superior parietal and premotor cortex) related to cognitive control (van Belle *et al.*, 2014), and has anatomical connections with rostral parietal areas (Jarbo and Verstynen, 2015). Further, patients with putamen lesions show symptoms of left-sided neglect (Karnath and Rorden, 2012), which is often thought of as a disorder of attention. Overall, the pattern of findings indicates both anatomical and functional links between the putamen/insula and parietal cortex, which may explain their increased activation during *localise* trials. However, it should be noted that our fMRI sequence parameters were not specifically designed for accurate recording of mid-brain structures, which may influence the reliability of these results (Eapen *et al.*, 2011).

Activation in the ACC was found in the *sense* versus *blind* contrast. The ACC is commonly linked to functional networks underlying attention (Ungerleider and Leslie, 2000), and more specifically in boosting attention toward task-relevant stimuli (Orr and Weissman, 2009; Kim *et al.*, 2016). Further, Mitchell and Cusack (2008) found ACC activation that correlated with estimates of the number of items stored by each participant during a working memory task. If this activation reflects increased attention toward the changed stimuli, then it would be expected to occur in both awareness conditions, as attention facilitates change detection (Rensink *et al.*, 1997). However, ACC activation was not found in the *localise* condition, and therefore may not be necessary for full awareness of the change.

A more fitting explanation of the ACC activation specific to the *sense* condition is that it reflects error processing during the task. This is because *sense* trials contained a response error, where participants incorrectly localised the change. Using combined EEG-fMRI, ACC activation has been linked to error processing and is correlated with the error-related negativity in EEG (Debener, 2005; Iannaccone *et al.*, 2015). Activity in this area could therefore relate to the incorrect responses of the participants during *sense* trials. However, it should be noted that activation in the ACC is found for a wide range of tasks and the specificity of this activation is debated (Dehaene, 2018). It could be argued that *blind* trials also contain a response error, as the participant failed to report a change that did occur. This should therefore also activate the ACC, if ACC activation reflects error monitoring (and that this error monitoring need not be conscious). Compared to *blind* trials, *sense* trials contained activation in visual (BA18) and parietal (BA40) areas, and the participant correctly reported the change. However, it is also

possible that the ACC activation relates to the participant's awareness of their own failure to localise the change, which is not relevant to *blind* trials where the participant can be very confident that no change occurred.

In relation to theories of visual consciousness, our results could be interpreted in support for the "partial awareness hypothesis" given the distinction in fMRI between *blind* and *sense* trials. Although participants were aware of the change during *sense* trials, their inability to provide further information suggests a less detailed representation of the visual display. Further, *localise* trials were associated with similar activity to *sense* in visual and parietal areas, perhaps reflecting activity relating to the "all-or-nothing" ignition of change detection. However, the additional activation related to *localise* trials may characterize an improved representation that facilitated correct localization. This hypothesis is speculative, and clarity is needed on the distinction between *localise* and *sense* conditions. For example, with future work using MVPA it would be possible to determine if the pattern of information stored within the brain is similar between these two levels of awareness. This would provide more information regarding the nature of stored representations during the task, and identify regions where these representations differ. Given the behavioral and phenomenological differences between *localise* and *sense* trials, it is reasonable to expect that somewhere in the brain should contain differing representations for these two levels of awareness, and therefore be driving the variation in participant response.

Difficulty and certainty

Using participant certainty at each trial as a parametric regressor, we found significant activations in the right visual cortex (BA18, V2), and bilateral supramarginal gyrus (BA40). These regions were also found to increase with awareness of the change (*localise* and *sense* trials), possibly due to the relationship between awareness and certainty. Specifically, when participants were aware of the change and could localise it correctly, they were likely to report higher certainty in their responses.

The parametric regressor of task difficulty (the number of squares presented per trial) revealed significant activation in the visual cortex (BA18, V2). This finding likely reflects the greater visual stimulation associated with a more complex visual array. In previous literature, parietal activity has also been correlated with set size and the number of objects stored in visual working memory (Mitchell and Cusack, 2008). Activity also predicts individual differences in working memory capacity (Vogel and Machizawa, 2004). We failed to find this effect, which may be explained by the variation in set sizes that were presented across participants. Instead of presenting a number of blocks with a number of difficulty levels, the difficulty was modulated in real time depending on participant performance. Also, the change in response may not be linear in our case; during easy trials, the response may scale linearly with the number of trials, until the maximum capacity of the participant is reached. Past this point, the number of items may exceed the capacity, and therefore fail to be represented or modulate the brain activation in these regions.

ERP-informed fMRI

Our pre-registered analysis method of LP-informed fMRI revealed no significant results. The additional exploratory P1 and N1-informed fMRI was also not significant. We therefore failed to identify voxels with activation that significantly covaried with fluctuations in the EEG. It is acknowledged that EEG-BOLD couplings are weak, as they measure the effects

remaining after the mean evoked BOLD responses are explained (Liu *et al.*, 2016). However, previous combined EEG-fMRI experiments have managed to identify correlates of EEG using ERP-informed fMRI (Debener, 2005; Eimer and Mazza, 2005), even if at liberal correction thresholds.

One possible reason for the failure to find significant ERP-informed BOLD effects is the reduced signal to noise in EEG signals recorded inside the MRI environment. A second possibility is the method that we used to quantify single-trial ERPs. There is no single method for ERP-informed fMRI analysis, and we therefore chose to run two separate analysis pipelines in case of disparaging results. In the first, we used raw values from the EEG time series. This method is susceptible to noise artifacts, and any trials in which the noise signal is greater than the neurological signal of interest will reduce the chance of observing an effect across conditions. Given the increased number of artifacts in EEG-fMRI data, and the absence of perfect artifact removal routines, it is possible that the signal to noise ratio was too small in the raw single-trial ERP values.

In the second method, we used ICA to identify components matching our ERP of interest, with the hypothesis that noise signals would have a reduced contribution to the single-trial values extracted from this component (Debener, 2005; Wirsich *et al.*, 2014). However, this method also produced null results in ERP-informed fMRI analysis. A downside to this method is that its success is dependent on (i) the algorithm accurately separating ICs, and (ii) the correct selection of the components containing the ERP of interest. Other possible processing steps used in ERP-informed fMRI include linear classifiers (Goldman *et al.*, 2009; Walz *et al.*, 2015), autoregressive models (Nguyen *et al.*, 2014), and spatial laplacian filters (Liu *et al.*, 2016), to name only a few. However, it is not within the scope of our pre-registered analysis to adjust the pre-processing or analysis steps any further.

Conclusion

Overall, one of the main aims of this experiment was to establish if the *sense* condition is separable from other awareness conditions in neural signals, as measured using EEG and fMRI. While the phenomenological experience of *sensing* differs from full awareness, it remains unclear whether this arises from a distinct state of neural activation, or whether these trials can be explained by explicit behavioral mechanisms such as participant response errors or lack of confidence. The strongest evidence presented here is the difference in fMRI activation for *blind* trials compared to *sense* trials. Across our sample, there was a greater spread of activation within areas such as the early visual cortex and inferior parietal sulcus when participants suspected a change, compared to when they missed it completely. This suggests that *sense* trials were measurably different to *blind* trials, and that participants did have access to more information regarding the change.

However, the contrast between *sense* and *localise* trials, where participants had full awareness, revealed no significant differences in activation. Additionally, the conjunction analysis revealed an overlapping occipitoparietal network of activation for these two levels of awareness. This suggests common activity related to the awareness of the change itself. In line with the "partial awareness hypothesis," it may be that a degraded representation of the visual display within these regions contributed to failed localization during *sense* trials.

While we attempted to distinguish between true *sense* trials and *localise* trials with an error using participant certainty, the number of *sense certain* responses was low. This meant that dividing the awareness conditions into certain/uncertain for EEG or fMRI analysis was not feasible. Future experiments could focus on obtaining higher trial numbers, which would hopefully facilitate this analysis. However, the very nature of the *sense* condition means that participants are unlikely to be "certain" during many of the trials. One way around this would be to include a response option for participants to indicate if they think that they made a response error, although this would only identify trials where the participants were aware of their mistake.

Several previous studies investigating change detection and partial awareness have used eye-tracking data to establish that fixations can occur close to the location of a change, even in the absence of full awareness (O'Regan *et al.*, 2000; Caplovitz *et al.*, 2008; Galpin *et al.*, 2008). Given that we found an effect of change location on the likelihood of detection, it would also have been beneficial to measure participants' eye movements during the experiment, especially to investigate fixations during *sense* trials. Due to the complexity of combined EEG-fMRI and the already time-consuming set up, we did not add eye-tracking to this experiment. However, it would be an interesting avenue for future research.

In summary, our data suggests that the phenomenological experience of *sensing* a change is associated with increased activity in visual, parietal, and anterior circulate cortices, when compared to change blind trials. Given this increased activation including areas that are commonly implicated as the storage sites of visual working memory, we argue that *sensing* may not be caused by a lack of representation of the visual display. Instead, *sensing* may reflect unsuccessful comparison of the two displays (Hollingworth *et al.*, 2001; Simons *et al.*, 2005), or a degraded representation that prevents accurate localization of the change in space.

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Data Availability

The raw data, pre-processed data, and analysis scripts can be found on the Open Science Framework:https://doi.org/10.17605/OSF.IO/W6BH3.

Supplementary data

Supplementary data is available at *NCONSC Journal* online.

References

Agresti A. *Categorical Data Analysis (Vol. 482)*. John Wiley & Sons., 2003.

Allen PJ, Polizzi G, Krakow K, *et al.* Identification of EEG events in the MR scanner: the problem of pulse artifact and a method for its subtraction. *Neuroimage* 1998;**8**:229–39.

Allen PJ, Josephs O, Turner R. A method for removing imaging artifact from continuous EEG recorded during functional MRI. *NeuroImage* 2000;**12**:230–9.

Ball F, Busch NA. Change detection on a hunch: pre-attentive vision allows "sensing" of unique feature changes. *Atten Percept Psychophys* 2015;**77**:2570–88.

Beck DM, Rees G, Frith CD, *et al*. Neural correlates of change detection and change blindness. *Nat Neurosci* 2001;**4**:645–50.

Bell A. J, Sejnowski T. J. *An information-maximization approach to blind separation and blind deconvolution. Neural computation,* **7**(6), 1129–1159..

Bénar C-G, Schön D, Grimault S, *et al*. Single-trial analysis of oddball event-related potentials in simultaneous EEG-fMRI. *Hum Brain Map* 2007;**28**:602–13.

Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. *J R Stat Soci Ser B (Methodological)* 1995;**57**:289–300.

Busch NA, Fründ I, Herrmann CS. Electrophysiological evidence for different types of change detection and change blindness. *J Cogn Neurosci* 2009;**22**:1852–69.

Busch NA, Dürschmid S, Herrmann CS. ERP effects of change localization, change identification, and change blindness. *NeuroReport* 2010;**21**:371–5.

Caplovitz GP, Fendrich R, Hughes HC. Failures to see: attentive blank stares revealed by change blindness. *Conscious Cogn* 2008;**17**:877–86.

Chetverikov A, Kuvaldina M, MacInnes WJ, *et al*. Implicit processing during change blindness revealed with mouse-contingent and gaze-contingent displays. *Atten Percept Psychophys* 2018;**80**: 844–59.

Christophel TB, Hebart MN, Haynes J-D. Decoding the contents of visual short-term memory from human visual and parietal cortex. *J Neurosci* 2012;**32**:12983–9.

D'Esposito M, Postle BR. The cognitive neuroscience of working memory. *Annu Rev Psychol* 2015;**66**:115–42.

Debener S. Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. *J Neurosci* 2005;**25**: 11730–7.

Dehaene S, Naccache L. Toward a cognitive neuroscience of consciousness: basic evidence and a workspace framework. *Cognition* 2001;**79**:1–37.

Dehaene S, Changeux J-P, Naccache L, *et al*. Conscious, preconscious, and subliminal processing: a testable taxonomy. *Trends Cogn Sci* 2006;**10**:204–11.

Dehaene S, Changeux J-P. Experimental and theoretical approaches to conscious processing. *Neuron* 2011;**70**:200–27.

Dehaene S. The error-related negativity, self-monitoring, and consciousness. *Perspect Psychol Sci* 2018;**13**:161–5.

Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J Neurosci Methods* 2004;**134**:9–21.

Eapen M, Zald D, Gatenby J, *et al*. Using high-resolution MR imaging at 7T to evaluate the anatomy of the Midbrain dopaminergic system. *Am J Neuroradiol* 2011;**32**:688–94.

Eckert MA, Menon V, Walczak A, *et al*. At the heart of the ventral attention system: the right anterior insula. *Hum Brain Map* 2009;**30**:2530–41.

Edin F, Klingberg T, Johansson P, *et al*. Mechanism for top-down control of working memory capacity. *Proc Natl Acad Sci* 2009; **106**:6802–7.

Eimer M, Mazza V. Electrophysiological correlates of change detection. *Psychophysiology* 2005;**42**:328–42.

Fernandez-Duque D, Thornton IM. Change detection without awareness: do explicit reports underestimate the representation of change in the visual system? *Vis Cogn* 2000;**7**:323–44.

Fernandez-Duque D, Grossi G, Thornton IM, *et al*. Representation of change: separate electrophysiological markers of attention, awareness, and implicit processing. *J Cogn Neurosci* 2003;**15**: 491–507.

Fernandez-Duque D, Thornton IM. Explicit mechanisms do not account for implicit localization and identification of change: an empirical reply to Mitroff et al. (2002). *J Exper Psychol Hum Percept Perform* 2003;**29**:846–58.

Förster J, Koivisto M, Revonsuo A. ERP and MEG correlates of visual consciousness: the second decade. *Conscious Cogn* 2020;**80**: 102917.

Friston KJ, Holmes AP, Worsley KJ, *et al*. Statistical parametric maps in functional imaging: a general linear approach. *Hum Brain Map* 1994;**2**:189–210.

Frömer R, Maier M, Abdel Rahman R. Group-level EEG-processing pipeline for flexible single-trial-based analyses including linear mixed models. *Front Neurosci* 2018;**12**:48.

Galpin A, Underwood G, Chapman P. Sensing without seeing in comparative visual search. *Conscious Cogn* 2008;**17**:672–87.

Goldman RI, Wei C-Y, Philiastides MG, *et al*. Single-trial discrimination for integrating simultaneous EEG and fMRI: identifying cortical areas contributing to trial-to-trial variability in the auditory oddball task. *NeuroImage* 2009;**47**:136–47.

Hollingworth A, Williams CC, Henderson JM. To see and remember: visually specific information is retained in memory from previously attended objects in natural scenes. *Psychon Bull Rev* 2001;**8**:761–8.

Howe PDL, Webb ME. Detecting unidentified changes. *PLoS One* 2014;**9**:e84490.

Huettel SA, Güzeldere G, McCarthy G. Dissociating the neural mechanisms of visual attention in change detection using functional MRI. *J Cogn Neurosci* 2001;**13**:1006–18.

Hyvärinen A, Oja E. A fast fixed-point algorithm for independent component analysis. *Neural Comput* 1997;**9**:1483–92.

Iannaccone R, Hauser TU, Staempfli P, *et al*. Conflict monitoring and error processing: new insights from simultaneous EEG–fMRI. *NeuroImage* 2015;**105**:395–407.

Jarbo K, Verstynen TD. Converging structural and functional connectivity of orbitofrontal, dorsolateral prefrontal, and posterior parietal cortex in the human striatum. *J Neurosci* 2015;**35**: 3865–78.

Kanai R, Verstraten FA. Visual transients without feature changes are sufficient for the percept of a change. *Vis Res* 2004; **44**:2233–40.

Karnath H-O, Rorden C. The anatomy of spatial neglect. *Neuropsychologia* 2012;**50**:1010–7.

Kim J, Wasserman EA, Castro L, *et al*. Anterior cingulate cortex inactivation impairs rodent visual selective attention and prospective memory. *Behav Neurosci* 2016;**130**:75–90.

Kimura M, Katayama J, Ohira H. Event-related brain potential evidence for implicit change detection: a replication of Fernandez-Duque et al. (2003). *Neurosci Lett* 2008;**448**:236–9.

Kleiner M, Brainard D, Pelli D, *et al*. What's new in Psychtoolbox-3. *Perception* 2007;**36**:1.

Koivisto M, Lähteenmäki M, Sørensen TA, *et al*. The earliest electrophysiological correlate of visual awareness? *Brain Cogn* 2008;**66**:91–103.

Koivisto M, Kainulainen P, Revonsuo A. The relationship between awareness and attention: evidence from ERP responses. *Neuropsychologia* 2009;**47**:2891–9.

Koivisto M, Revonsuo A. Event-related brain potential correlates of visual awareness. *Neurosci Biobehav Rev* 2010;**34**:922–34.

Kouider S, Dehaene S. Levels of processing during non-conscious perception: a critical review of visual masking. *Philos Trans R Soc B: Biol Sci* 2007;**362**:857–75.

Kouider S, de Gardelle V, Sackur J, et al. How rich is consciousness? The partial awareness hypothesis. *Trends Cogn Sci* 2010;**14**:301–7.

Lakens D. Calculating and reporting effect sizes to facilitate cumulative science: a practical primer for t-tests and ANOVAs. *Front Psychol* 2013;**4**: 863.

Liu Y, Bengson J, Huang H, et al. Top-down modulation of neural activity in anticipatory visual attention: control mechanisms revealed by simultaneous EEG-fMRI. *Cereb Cortex* 2016;**26**: 517–29.

Lo S, Andrews S. To transform or not to transform: using generalized linear mixed models to analyse reaction time data. *Front Psychol* 2015;**6**:1171.

Luck SJ, Hillyard SA. Electrophysiological correlates of feature analysis during visual search. *Psychophysiology* 1994;**31**: 291–308.

Luck SJ, Ford MA. On the role of selective attention in visual perception. *Proc Natl Acad Sci* 1998;**95**:825–30.

Luck SJ, Vogel EK. Visual working memory capacity: from psychophysics and neurobiology to individual differences. *Trends Cogn Sci* 2013;**17**:391–400.

Luck SJ. *An Introduction to the Event-Related Potential Technique*. MIT Press, 2014.

Luck SJ, Gaspelin N. How to get statistically significant effects in any ERP experiment (and why you shouldn't): how to get significant effects. *Psychophysiology* 2017;**54**:146–57.

Lyyra P, Wikgren J, Ruusuvirta T, et al. Explicit behavioral detection of visual changes develops without their implicit neurophysiological detectability. *Front Hum Neurosci* 2012;**6**.

Menon V, Uddin LQ. Saliency, switching, attention and control: a network model of insula function. *Brain Struct Funct* 2010;**214**: 655–67.

Mitchell DJ, Cusack R. Flexible, capacity-limited activity of posterior parietal cortex in perceptual as well as visual short-term memory tasks. *Cereb Cortex* 2008;**18**:1788–98.

Mitroff SR, Simons DJ, Franconeri SL. The siren song of implicit change detection. *J Exp Psychol Hum Percept Perform* 2002;**28**: 798–815.

Mitroff SR, Simons DJ, Levin DT. Nothing compares 2 views: change blindness can occur despite preserved access to the changed information. *Percept Psychophys* 2004;**66**:1268–81.

Nguyen VT, Breakspear M, Cunnington R. Fusing concurrent EEG–fMRI with dynamic causal modeling: application to effective connectivity during face perception. *NeuroImage* 2014;**102**: 60–70.

Nichols T, Brett M, Andersson J, et al.. Valid conjunction inference with the minimum statistic. Neuroimage 2005;**25**:653–60.

Niedeggen M, Wichmann P, Stoerig P. Change blindness and time to consciousness: change blindness and time to consciousness. *Eur J Neurosci* 2001;**14**:1719–26.

Noe A, Pessoa L, Thompson E. Beyond the grand illusion: what change blindness really teaches us about vision. *Vis Cogn* 2000; **7**:93–106.

O'Regan JK, Deubel H, Clark JJ, et al. Picture changes during blinks: looking without seeing and seeing without looking. *Vis Cogn* 2000;**7**:191–211.

Orr JM, Weissman DH. Anterior cingulate cortex makes 2 contributions to minimizing distraction. *Cereb Cortex* 2009;**19**:703–11.

Pessoa L, Ungerleider LG. Neural correlates of change detection and change blindness in a working memory task. *Cereb Cortex* 2004;**14**:511–20.

Pourtois G, De Pretto M, Hauert C-A, et al. Time course of brain activity during change blindness and change awareness: performance is predicted by neural events before change onset. *J Cogn Neurosci* 2006;**18**:2108–29.

Railo H, Koivisto M, Revonsuo A. Tracking the processes behind conscious perception: a review of event-related potential correlates of visual consciousness. *Conscious Cogn* 2011;**20**:972–83.

Rensink RA, O'Regan JK, Clark JJ. To see or not to see: the need for attention to perceive changes in scenes. *Psychol Sci* 1997;**8**: 368–73.

Rensink RA. Visual sensing without seeing. *Psychol Sci* 2004;**15**: 27–32.

Reynolds MG, Withers K. Understanding the relationship between implicit and explicit change detection: evidence from scan path data. *J Exp Psychol Hum Percept Perform* 2015;**41**: 1184–9.

Rouder JN, Lu J, Speckman P, et al. A hierarchical model for estimating response time distributions. *Psychon Bull Rev* 2005;**12**: 195–223.

Rousselet GA, Wilcox RR. Reaction times and other Skewed distributions: problems with the mean and the median. *Meta-Psychology* 2020;**4**.

Schankin A, Wascher E. Electrophysiological correlates of stimulus processing in change blindness. *Exp Brain Res* 2007;**183**:95–105.

Scrivener CL, Malik A, Marsh J, et al. An EEG study of detection without localisation in change blindness. *Exp Brain Res* 2019; **237**:2535–47.

Simons D, Levin DT. Change blindness. *Trends Cogn Sci* 1997;**1**: 261–7.

Simons DJ. Current approaches to change blindness. *Vis Cogn* 2000;**7**:1–15.

Simons DJ, Ambinder MS. Change blindness: theory and consequences. *Curr Dir Psychol Sci* 2005;**14**:44–8.

Simons DJ, Nevarez G, Boot WR. Visual sensing is seeing: why "Mindsight," in hindsight, is blind. *Psychol Sci* 2005;**16**:520–4.

Stanislaw H, Todorov N. Calculation of signal detection theory measures. *Behav Res Methods Instrum Comput* 1999;**31**:137–49.

Tadel F, Baillet S, Mosher JC, et al. *Brainstorm: A User-Friendly Application for MEG/EEG Analysis*, Vol. 2011. Hindawi, 2011, e879716.

Thornton I, Fernandez-Duque D. An implicit measure of undetected change. *Spatial Vis* 2001;**14**:21–44.

Todd JJ, Marois R. Posterior parietal cortex activity predicts individual differences in visual short-term memory capacity. *Cogn Affect Behav Neurosci* 2005;**5**:144–55.

Tseng P, Hsu T-Y, Chang C-F, et al. Unleashing potential: transcranial direct current stimulation over the right posterior parietal cortex improves change detection in low-performing individuals. *J Neurosci* 2012;**32**:10554–61.

Uddin LQ, Nomi JS, Hébert-Seropian B, et al. Structure and function of the human Insula. *J Clin Neurophysiol* 2017;**34**:300–6.

Ungerleider SK, Leslie G. Mechanisms of visual attention in the human cortex. *Annu Rev Neurosci* 2000;**23**:315–41.

van Belle J, Vink M, Durston S, et al. Common and unique neural networks for proactive and reactive response inhibition revealed by independent component analysis of functional MRI data. *NeuroImage* 2014;**103**:65–74.

Vogel EK, Machizawa MG. Neural activity predicts individual differences in visual working memory capacity. *Nature* 2004;**428**: 748–51.

Vogel EK, McCollough AW, Machizawa MG. Neural measures reveal individual differences in controlling access to working memory. *Nature* 2005;**438**:500–3.

Walz JM, Goldman RI, Carapezza M, *et al*. Prestimulus EEG alpha oscillations modulate task-related fMRI BOLD responses to auditory stimuli. *NeuroImage* 2015;**113**:153–63.

Wilcox RR, Erceg-Hurn DM. Comparing two dependent groups via quantiles. *J Appl Stat* 2012;**39**:2655–64.

Wilenius ME, Revonsuo AT. Timing of the earliest ERP correlate of visual awareness. *Psychophysiology* 2007;**44**:703–10.

Williams P, Simons DJ. Detecting changes in novel, complex three-dimensional objects. *Vis Cogn* 2000;**7**:297–322.

Wirsich J, Bénar C, Ranjeva J-P, *et al*. Single-trial EEG-informed fMRI reveals spatial dependency of BOLD signal on early and late IC-ERP amplitudes during face recognition. *NeuroImage* 2014;**100**:325–36.