Linking time-on-task, spatial bias and hemispheric activation asymmetry: A neural correlate of rightward attention drift

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ABSTRACT

Biases of spatial attention may be moderated by non-spatial factors such as attentional load and time-on-task. Although these effects are thought to arise from depletion of right hemisphere processing resources, their neurophysiological bases have yet to be confirmed. We recorded posterior α-band EEG – a marker of cortical excitability linked to spatial attention orienting – from 66 non-clinical participants who detected transient, unilateral visual targets while also monitoring stimuli at fixation. Asymmetry indices were derived for both lateral target reaction times and hemispheric differences in α-activity before and after lateral target onsets. Pre-target α became more prominent over the right, relative to left, hemisphere as the task progressed over 48-min, and this change was correlated with a significant rightward shift in spatial bias. Contrary to past studies of posterior α-asymmetry and orienting, here participants did not receive pre-target cues. Thus we show that asymmetries in the hemispheric distribution of anticipatory α are not only apparent during externally-cued attention orienting, but are also sensitive to decreasing alertness over time. These data are the first to link rightward attention drift over time with change in hemispheric activation asymmetry, providing important implications for our understanding of interacting spatial attention and non-spatial alertness networks.

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1. Introduction

Healthy subjects tend to exhibit a subtle bias of visual attention favouring left space, termed 'pseudoneglect', which occurs for a variety of stimuli (Nicholls, Bradshaw, & Mattingley, 1999; Voyer, Voyer, & Tramonte, 2012) and is thought to reflect the right hemisphere's dominance of the networks governing spatial attention (Loftus & Nicholls, 2012; Mesulam, 1981). Recent research with healthy volunteers and patient groups exhibiting pathological visuo-spatial asymmetries suggest that spatial biases are regulated by non-spatial factors, such as attentional load and time-on-task (Dodds et al., 2008; Matthias et al., 2009; Peers, Cusack, & Duncan, 2006). Despite our increasing knowledge of the cognitive factors that might modulate spatial biases, our knowledge of the physiological bases of these effects remains unclear. Here we employed electrophysiology to understand the influence of attentional load and time-on-task on neural biases of spatial attention in healthy volunteers.

A number of lines of evidence suggest that non-spatial factors modulate biases of spatial attention. First, the modulatory influence of non-spatial processes on spatial bias has been documented in unilateral spatial neglect (hereafter 'neglect'), a common outcome of right hemisphere damage that is characterised by pronounced deficits in attending to contralesional stimuli despite adequate sensory processing (Corbetta & Shulman, 2011; Husain & Nachev, 2007; Husain & Rorden, 2003). Robertson et al. (1998) found that loud tones designed to increase alertness could temporarily reduce symptoms of leftward inattention in neglect patients (see also George et al., 2008). Peers et al. (2006) also demonstrated that imposing a non-spatial dual-task during a spatial attention task caused the same general rightward shift in patients with left neglect, patients with right neglect and control participants. Furthermore, it has been shown that neglect can be temporarily ameliorated by psychostimulants but exacerbated by sedatives, suggesting a critical modulatory influence of arousal (Fleet, Valenstein, Watson, & Heilman, 1987; Geminiani, Bottini, & Sterzi, 1998; Grujic et al. 1998; Lazar et al., 2002; Malhotra, Parton, Greenwood, & Husain, 2006; Mukand et al., 2001).

Second, a number of studies have shown that even in healthy populations, spatial bias is significantly modulated by sleep deprivation (Manly, Dobler, Dodds, & George, 2005), non-spatial attentional load (Peers et al., 2006; Pérez et al., 2009), diminishing alertness with time-on-task (Dodds et al., 2008; Dufour, Touzalin, & Candas, 2007) and psychostimulants (Dodds, Müller, & Manly, 2009). The links between non-spatial attention processes and spatial bias in both clinical and non-clinical populations highlight a need to understand how these mechanisms are integrated in the human brain.

At a neural level, it has been proposed (Corbetta & Shulman, 2011) that the effects of alertness and attentional load on spatial

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bias result from increased demand on a *right lateralised ventral attention network* (Coull, Frackowiak, & Frith, 1998; Pardo, Fox, & Raichle, 1991; Sturm et al., 1999, 2004) that regulates interhemispheric rivalry in the *bilateral dorsal orienting network* (Corbetta, Patel, & Shulman, 2008; Corbetta & Shulman, 2011; Husain & Nachev, 2007). The bilateral orienting network is activated by selectively attending to stimuli across space and linking them to appropriate responses, whilst the right lateralised ventral attention network has been linked to non-spatial attention capacity (Culham, Cavanagh, & Kanwisher, 2001; Schwartz et al., 2005; Vuilleumier et al., 2008) and vigilance/alertness (Paus et al., 1997; Sturm & Willmes, 2001). Decreased activation within the right lateralised ventral network may cause a more global decrease in right hemisphere activation, giving the left dorsal orienting network a competitive activation advantage over the right dorsal network, thus driving attention rightwards (Corbetta & Shulman, 2011).

Support for the above neuroanatomical model comes from an fMRI study of neglect patients with damage restricted to the right ventral attention network whose rightward spatial bias was associated with a functional imbalance in the structurally intact dorsal orienting network (Corbetta, Kincade, Lewis, Snyder, & Sapir, 2005). A recent diffusion imaging study (De Schotten et al., 2011) provides a neuronatomical basis for the pseudoneglect of healthy individuals by demonstrating a clear right lateralisation in tracts connecting the dorsal and ventral networks which was strongly related to the degree of pseudoneglect displayed by participants. To date however, a neurophysiological marker that is sensitive to interactions between spatial and non-spatial attention systems has yet to be identified.

In the present study we tested the hypothesis that a rightward attentional shift with time-on-task and attentional load is linked to changing hemispheric activation asymmetry. We recorded continuous EEG from healthy participants during a fixation-controlled spatial attention task that allowed us to separately manipulate attentional load and time-on-task. Participants detected sudden onset targets that occurred at uncued peripheral locations while performing a concurrent task at fixation. Demand on non-spatial attention was manipulated across three levels (no, low and high central load) by changing the difficulty of the task at fixation, and changes in behavioural and neurophysiological markers of spatial attention were analysed as a function of time-on-task. We capitalised on hemispheric asymmetry in α-band (8–14 Hz) as a marker of cortical activation asymmetry before and after the onset of a peripheral event. Decreased α-band activity reflects increased cortical activation or excitability, whereas increased α activity reflects cortical deactivation (Pfurtscheller, 2001; Romei et al., 2008; Romei, Rihs, Brodbeck, & Thut, 2008; Sadaghiani et al., 2010). Several recent studies employing simultaneous EEG and fMRI have demonstrated that α-band activity is negatively correlated with activity of the dorsal attention network (Laufs et al., 2003, 2006; Mantini, Perrucci, Del Gratta, Romani, & Corbetta, 2007; Sadaghiani et al., 2010; Scheeringa et al., 2009).

Thut, Nietzel, Brandt, and Pascual-Leone (2006) measured α activity over the parieto-occipital cortex during a variant of the Posner spatial cueing task (Posner, Walker, Friedrich, & Rafal, 1984) and found that preparatory hemispheric α asymmetry (expressed as a lateralisation index) during the epoch between spatial cue and target onset predicted reaction-time asymmetries for imminent peripheral targets. Spatial cueing promotes desynchronization of α (decreased α activity) at contralateral parieto-occipital sites, reflecting facilitated processing at the locus of attention (Kelly, Gomez-Ramirez, & Foxe, 2009; Rihs, Michel, & Thut, 2009; Sauseng et al., 2005; Thut et al., 2006; Yamagishi, Goda, Callan, Anderson, & Kawato, 2005) whereas synchronization (increased α activity) over ipsilateral sites, may index suppression of

unattended space (Kelly, Lalor, Reilly, & Foxe, 2006; Rihs, Michel, & Thut, 2007, 2009; Worden, Foxe, Wang, & Simpson, 2000). These cueing studies explicitly directed the attention of participants in the pre-target interval to one or the other hemifield. In the current study, by contrast, we presented targets at uncued lateral locations, eliminating any strategic top-down biasing of attention. This allowed us to investigate the impact of depleting non-spatial attention resources – either via central task load or time-on-task – on both the balance of α power between the two hemispheres and corresponding visuospatial bias. We predicted that depleting non-spatial attention resources via time-on-task and central task load would lead to a rightward shift in posterior α-asymmetry and spatial bias.

2. Method

2.1. Participants

Data were collected from 91 right-handed volunteers of Caucasian descent, reporting normal or corrected to normal vision, no history of neurological or psychiatric disorder and no head injury resulting in loss of consciousness. Event-related potential (O'Connell, Schneider, Hester, Mattingley, & Bellgrove, 2011) and molecular genetics (Newman, O'Connell, Nathan, & Bellgrove, 2012) data from a subset of these participants were previously published, however no analyses of α activity were conducted. All participants gave written informed consent, and all procedures were in accordance with the Declaration of Helsinki. Four participants responded to fewer than 75% of peripheral targets, suggesting they were insufficiently engaged in the task. These participants were excluded from further analysis. Two were excluded due to a technical error relating to response acquisition. Three participants lacked full time-on-task data within each load condition, so could not be included, and 16 participants displayed small but systematic eye movements during trials (see procedure below) and were thus excluded (Lins, Picton, Berg, & Scherg, 1993). This exclusion of participants due to systematic eye movements was necessary to ensure that peripheral stimuli were transmitted to contralateral visual cortex and that the key changes in alpha and behavioural bias could not be accounted for by systematic biases in eye movements. This left a final sample of 66 participants (40 females) aged 18–47 ($M = 24$).

2.2. Visual attention task

Full details of the current task are presented in O'Connell et al. (2011). Briefly, the task comprised short (3600 ms) rapid serial visual presentation (RSVP) streams of eight central alphanumeric characters on which participants fixated and monitored for the appearance of a target. At the same time, participants covertly monitored left and right lateralised target locations to detect a sudden-onset unilateral peripheral stimulus (Fig. 1A). The peripheral stimulus appeared in either left or right target locations or not at all (catch trials). These three trial types occurred in a randomised order and with equal probability. Participants indicated their detection of the peripheral target with a speeded button press with their right hand, with a valid response window of up to 1000 ms. Using a similar paradigm Peers et al. (2006) found response hand had no effect on spatial bias. Peripheral stimuli appeared randomly at one of two time points in the RSVP stream: at 800 ms (simultaneous with the onset of the third character) or 2000 ms (simultaneous with the onset of the sixth character). After each trial, participants were questioned whether the central RSVP stream contained a target or not and answered by making a non-speeded two choice button press for 'yes' or 'no', before the next trial began.
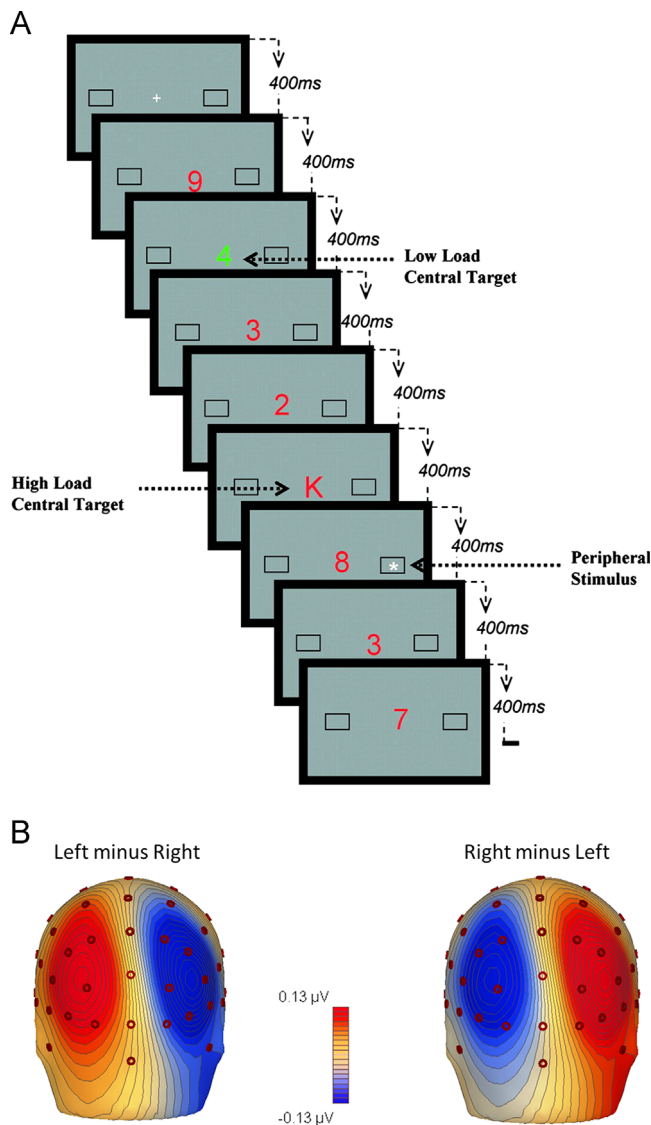
Fig. 1. (A) Schematic of a single trial from the visual attention task. Participants fixated on the central stream searching for an assigned target, while simultaneously monitoring the periphery for a stimulus that could appear to the right or left or not at all (catch trials). Attentional load was manipulated across three central load conditions: high central load (red letter as central target), low central load (any green item as central target), and no central load (no central target). Participants indicated detection of the peripheral target with a speeded button press. Detection of the central target was assessed at the end of each trial. (B) Parieto-occipital ROIs showed the largest α desynchronisation in response to contralateral target stimuli.

Attentional load at fixation was manipulated across three conditions (no, low, and high-load), completed in separate blocks in a counterbalanced order, by changing the task instructions to specify a different central target at the beginning of the condition. For the no-load condition, participants were simply instructed to fixate on the central stream while also monitoring for a peripheral target and no central target was specified. For the low-load condition, the specified central target was any green character— effectively a feature search (Treisman & Gelade, 1980). For the high-load condition, the specified target was any red *letter* within the stream of red digits—effectively a conjunction search (Treisman & Gelade, 1980). Relatively greater attention was required in the high than low central-load condition because, in the former, the central target was defined by a conjunction of features that were shared by the non-target stimuli. The central

target appeared unpredictably in 50% of the trials and its order of appearance within the RSVP stream was randomised. The onset of the central target never coincided with the onset of the peripheral target.

2.3. Procedure

Participants were comfortably seated with their head supported by a chin rest 50 cm from the screen. They were instructed to maintain central fixation and avoid blinking or moving during each trial, but were encouraged to blink and move in the short breaks between each trial, if desired. When participants had mastered a practice session of their first load condition, they were left alone in a darkened room to begin the task (described above). Participants performed the three load conditions over one session. Each load condition comprised 300 trials with participants receiving short rest periods after every 100 trials and at the end of each condition (note Thut et al. (2011) but omitted from the description of methods as time-on-task was not investigated in that study). Before beginning a new load condition, participants read on-screen instructions and the experimenter explained the task verbally then ensured the participants' comprehension of the new task. Extended intervals between conditions for task instructions meant that it was not appropriate to analyse time-on-task across the whole testing session. The duration of each load condition (approximately 48 min) ensured sufficient data for time-on-task analyses within each separate condition (see Dodds et al. (2008) for a similar approach).

2.4. Data acquisition

Continuous EEG was acquired using an Active Two Biosemi system with 64 scalp electrodes digitised at 512 Hz. Electro-oculogram (EOG) electrodes were placed above and below the left eye to measure vertical eye movements, and at the outer canthus of each eye for horizontal eye movements. Processing was performed using the EEGLAB toolbox (Delorme & Makeig, 2004) in MATLAB. Behavioural data (reaction-time and accuracy) were acquired and processed using E-Prime 2.0 (Psychology Software Tools, Pittsburgh, PA).

Following similar procedures to those of Thut et al. (2006), analyses were conducted on two symmetric occipito-parietal regions of interest (ROI) defined based on grand-average waveforms (electrode sites showing largest alpha desynchronisation in response to contralateral target stimuli, see Fig. 1B). Each ROI comprised five electrodes (left ROI: P7, P9, PO7, PO3, O1; right ROI: P8, P10, PO8, PO4, O2) which were pooled for all analyses. To ensure effects were not confounded by activation changes related to manual response preparation, a control analysis was achieved using lateralised motor-selective electrodes over the primary sensorimotor cortex (left hemisphere: C3, C5; right hemisphere: C4, C6) (Kaiser, Ulrich, & Lutzenberger, 2003; McFarland, Miner, Vaughan, & Wolpaw, 2000).

2.5. Electrophysiological artefact correction

Offline electrophysiological data were average referenced and segmented into epochs of −2000 to +1000 ms relative to peripheral target onset. Only epochs for correctly detected targets were retained for analysis. To eliminate EOG and other artifactual transients, epochs were baseline corrected relative to the 100 ms interval preceding target onset and any single trials with an amplitude deflection greater than 100 μV were rejected. Two additional steps were taken to rule out the possible influence of

horizontal eye movements. First, single trials were rejected if the bipolar horizontal EOG signal (left minus right) exceeded an absolute value of 60 μV at any time within the epoch. Second, any participants who exhibited residual systematic eye movements in their grand-average bipolar horizontal EOG signal (> 3 μV amplitude deflection corresponding to .2° change in eye position) (Lins et al., 1993) were rejected from further analysis. This led to the rejection of 16 participants.

2.6. Calculating α-band power

This study followed the criteria set out by Thut et al. (2006) for calculating the time course of α-band power during the pre and post-target intervals using a short time Fourier transform (STFT). For each of three time-blocks (first 100 trials, second 100 trials and last 100 trials) within each load condition, the epoched EEG data were band-pass filtered to isolate the α-frequency range, 8–14 Hz. The data were then rectified (negative values made positive). Initial −2000 to 1000 ms epochs were cropped to −1800 to 900 ms around the onset of peripheral targets to eliminate artefacts caused by band-pass filtering. Data were then smoothed by averaging using a moving 100 ms average window which stepped forward in 50 ms increments throughout each epoch. The ROI data were then isolated into pre and post-target epochs and averaged across all single trials. Average post-target α was measured from 0 to 900 ms, while average pre-target α was measured from either −1450 to 0 ms (for peripheral targets appearing simultaneous with the onset of the sixth character), or from −550 to 0 ms (for peripheral targets appearing simultaneous with the onset of the third character). The shorter pre-target epoch (−550 to 0 ms) was necessary for targets appearing earlier in the trial to avoid recording changes in α related to trial onset. The resulting pre and post-target α measurements were entered into condition specific matrices for export into SPSS for final analysis.

2.7. Analysis

Time-on-task was operationalised by dividing each level of load into 3 time blocks: first 100 trials, second 100 trials and final 100 trials. Behavioural data were then filtered to accept trials where participants correctly detected the peripheral stimulus and correctly identified whether the central target was present or absent (for low and high-load conditions). A measure of reaction time (RT) asymmetry was derived from peripheral RT (ms) using the following formula:

$$\text{RT asymmetry index} = \frac{(\text{left target RT}) - (\text{right target RT})}{(\text{mean left and right target RT})}$$

This index gives positive values when reaction-times are faster for right relative to the left targets (rightward spatial bias) and negative values when the opposite is true (leftward bias). A similar formula was used to calculate hemispheric asymmetry for both pre and post-target parieto-occipital α activity:

$$\alpha \text{ asymmetry index} = \frac{(\text{left ROI } \alpha) - (\text{right ROI } \alpha)}{(\text{mean left and right ROI } \alpha)}$$

This index gives positive values when α activity is greater over the right hemisphere ROI relative to the left hemisphere ROI and negative values when α activity is greater over the left hemisphere ROI relative to the right. If no asymmetry exists then the index gives a zero value (see Thut et al. (2006) for comparable use of asymmetry indices). The average post-target α asymmetry data were subjected to a time-on-task (first block vs. second block vs. final block) × load (no vs. low vs. high) × target side (left vs. right) repeated measures ANOVA (Greenhouse–Geisser corrections were

employed where the assumption of sphericity was violated). Average RT and pre-target α asymmetry data were subjected to time-on-task (first block vs. second block vs. final block) × load (no vs. low vs. high) repeated measures ANOVAs. Because we expected linear trends in RT and α asymmetry as a function of time-on-task, planned orthogonal polynomial contrasts were used where appropriate. The average asymmetry measures were also collapsed across load and time-on-task and subjected to one-sample t-tests from zero to gauge any overall asymmetry.

The effect of pre-target α asymmetry on detection of forthcoming peripheral targets was also investigated at a trial-by-trial level. Each participant's single trials were sorted in ascending order from the trial containing the most negative pre-target alpha asymmetry through to the trial containing the most positive pre-target α asymmetry. The ordered single trials were then grouped into three equal sized bins; bin 1 comprising the third of trials in which pre-target α asymmetry was most negative, through to bin 3 comprising the third of trials in which pre-target α asymmetry was most positive. The corresponding absolute RT data (as opposed to the RT asymmetry index) for these trials were then analysed using an α asymmetry (bin 1 vs. bin 2 vs. bin 3) × target side (left vs. right) repeated measures ANOVA.

3. Results

3.1. Behavioural performance

Participants detected both peripheral and central targets with near perfect accuracy (accuracy defined as correct hits; mean peripheral target accuracy 95%, mean central target accuracy 96%). Peripheral target accuracy decreased as load increased ($\chi^2 = 98.60$, $p < .001$). Wilcoxon signed-rank tests confirmed that mean accuracy was greater under no-load (99%) than low-load (95%; $Z = -6.4$, $p < .001$), which was in turn greater than high-load accuracy (92%; $Z = -5.7$, $p < .001$). There was no change in peripheral target accuracy as a function of time-on-task ($\chi^2 = 1.21$, $p = .547$). In line with peripheral target accuracy, central target accuracy was higher under low-load (97%) than high-load (94%; $Z = -6.6$, $p < .001$), and did not change with time-on-task ($\chi^2 = .92$, $p = .630$).

Peripheral target RT and α asymmetry indices were calculated to probe systematic differences in behavioural and hemispheric asymmetry as a function of central load and time-on-task. For clarity however, we also present absolute peripheral target RT (Table 1) and absolute pre and post-target α power (Fig. 2A) as a function of central load and time-on-task. Absolute RTs slowed significantly with increased central load [$F(2, 130) = 202.0$, $p < .001$] but not with time-on-task [$F(2, 130) = .724$, $p > .05$].

3.2. Linking time-on-task, pre-target α asymmetry, and RT asymmetry

As can be seen from Fig. 2A and C, absolute α activity during the pre-target epoch increased as a function of time-on-task [$F(1.67, 108.68) = 29.25$, $p < .001$, $\eta_p^2 = .31$]. However, the hemispheric α asymmetry index, rather than the absolute α magnitude, was the focus of the electrophysiological aspect of this study. Pre-target

Table 1
Absolute mean RTs (ms; SE) as a function of central task load and of time-on-task.

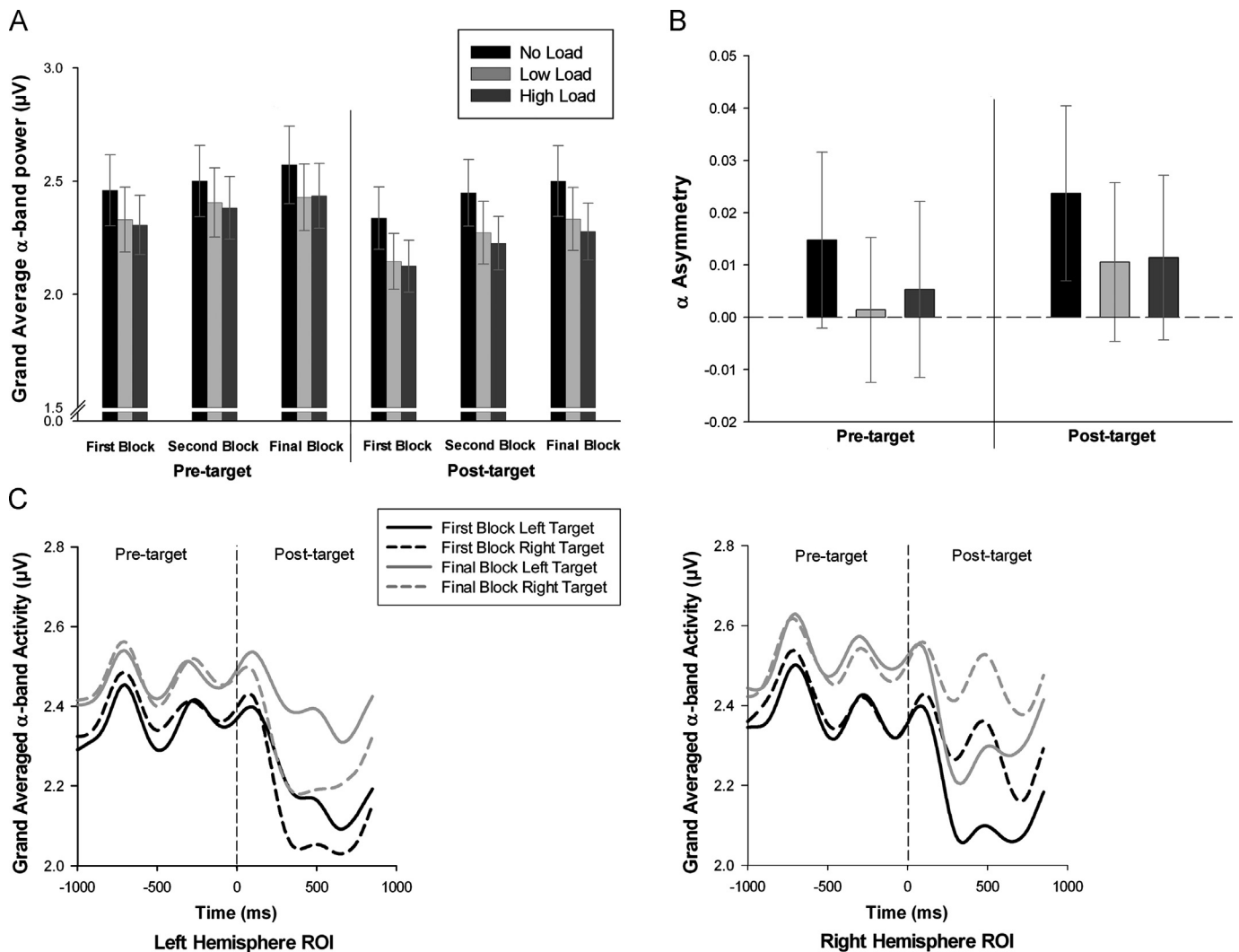|	First block	Second block	Final block
No load	413 (7.2)	410 (7.7)	412 (7.4)
Low load	502 (9.7)	505 (9.7)	499 (8.6)
High load	519 (9.8)	515 (9.6)	513 (9.3)

Fig. 2. (A) Pre and post-target α-band power as a function of time-on-task and central task load. (B) Pre and post-target α asymmetry as a function of central task load. (C) The time course of grand-average α-band oscillatory activity as a function of ROI, time-on-task (first block vs. final block) and peripheral target side.

α asymmetry and RT asymmetry data were first collapsed across central task load and time-on-task and subjected to one-sample *t*-tests to assess any asymmetry independent of load and time-on-task. Although overall pre-target α asymmetry was not significantly different from zero [$t(65)=.59$, $p>.05$], the analysis of overall RT asymmetry revealed a significant leftward bias that is characteristic of the phenomenon of pseudoneglect [$M=-.011$, $SE=.004$; $t(65)=-2.74$, $p=.008$].

Pre-target α asymmetry and RT asymmetry data were then subjected to separate time-on-task × central task load repeated measures ANOVAs. Planned orthogonal polynomial contrasts revealed a significant linear trend in pre-target α asymmetry with time-on-task [$F(1, 65)=9.41$, $p=.003$, $\eta_p^2=.13$] showing that α activity became more prominent over the right hemisphere relative to the left hemisphere ROI with time (see Fig. 3A). Crucially, a similar effect of time-on-task was observed for RT asymmetry. Planned orthogonal polynomial contrasts revealed a significant linear trend in RT asymmetry with time-on-task [$F(1, 65)=4.93$, $p=.030$, $\eta_p^2=.07$], indicating a rightward shift in spatial bias over the course of the experiment (see Fig. 3B).

To test for a link between the rightward shifts in spatial bias and hemispheric α asymmetry with time-on-task, data for RT asymmetry and pre-target α asymmetry were collapsed across load conditions and change measures were derived by subtracting

the average asymmetry during the final 100 trials (the final block) from the average asymmetry during the first 100 trials (the first block). The resulting indices of asymmetry change with time-on-task were examined with Pearson's correlations. A significant positive relationship between pre-target α asymmetry change and RT asymmetry change [$r=.32$, $p=.010$] revealed that participants who had a larger change in RT asymmetry with time-on-task also tended to have a larger change in pre-target α asymmetry, indicative of increasing α activity over of the right hemisphere relative to left hemisphere ROI with time-on-task. No significant relationship was observed between overall pre-target α asymmetry and RT asymmetry when time-on-task was disregarded.

To further explore the significant relationship between change in pre-target α asymmetry with time-on-task and change in RT asymmetry with time-on-task [$r=.32$, $p=.010$], we rank ordered participants according to their change in pre-target α asymmetry, and then divided them into three equally sized groups such that group 1 comprised the third of participants who had the least change in pre-target α asymmetry over time, and group 3 comprised those participants who had the greatest α asymmetry change over time (see Fig. 3C). A one-way between-groups ANOVA (pre-target α asymmetry groups: 1 vs. 2 vs. 3) was applied with RT asymmetry change with time-on-task as the dependent variable. This revealed a significant effect of α asymmetry change on RT
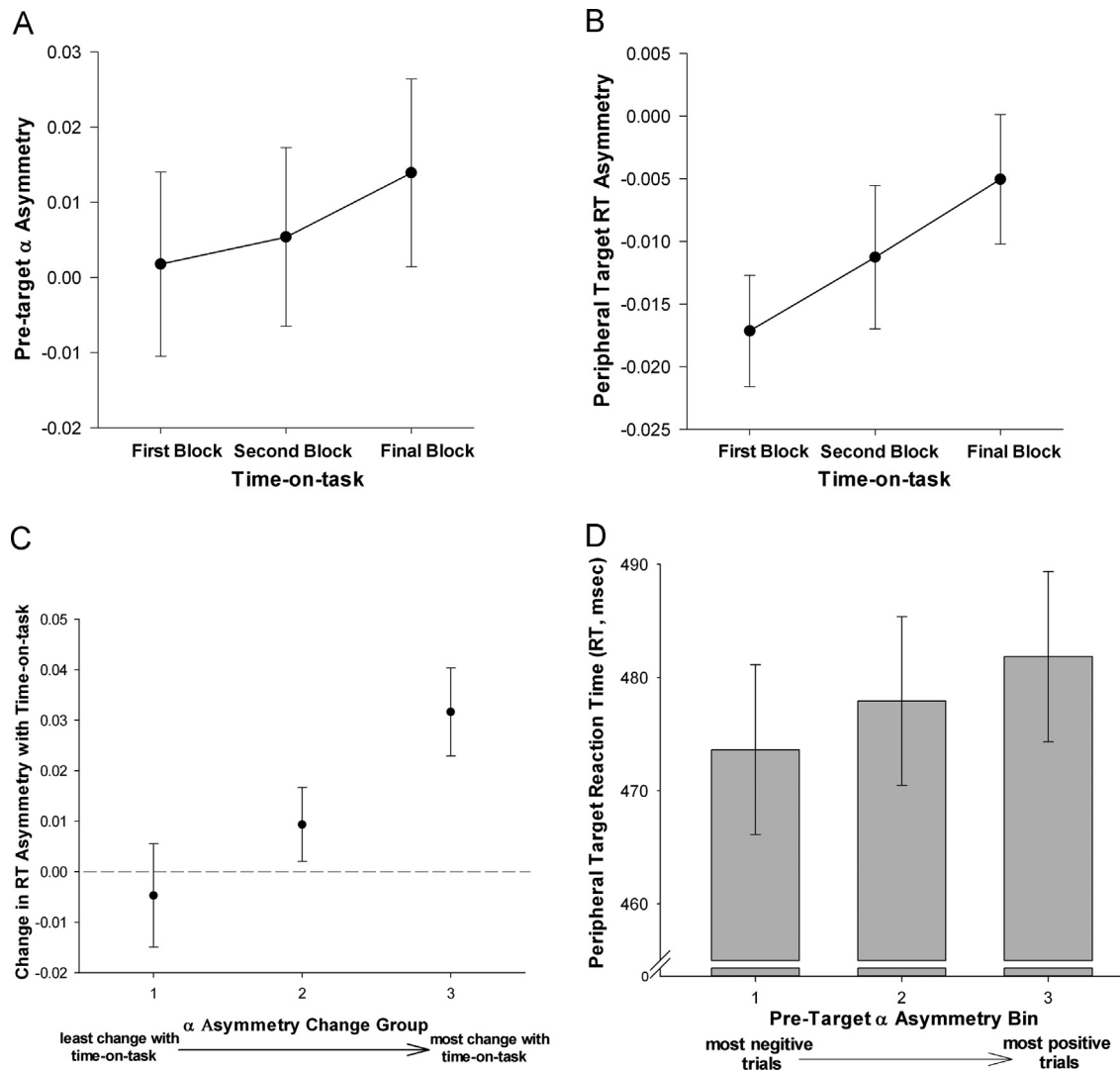
Fig. 3. (A) Increasing pre-target α asymmetry with time-on-task indicates that α activity became more prominent over the right parieto-occipital region, relative to the left, over time. (B) A leftward bias for RT that is characteristic of pseudoneglect is evident in the first block and is attenuated with time-on-task as spatial bias drifts rightward towards zero. (C) RT asymmetry change with time-on-task as a function of change in pre-target α asymmetry with time-on-task. Participants with the greatest time-on-task shift in pre-target α asymmetry had a significantly greater shift in RT asymmetry. (D) The effect of pre-target α asymmetry on peripheral target RT (ms). RT's were significantly slower during the trials in which pre-target α asymmetry was most positive (i.e. relatively decreased right hemisphere activation) compared to the trials in which pre-target α asymmetry was most negative. Error bars show standard error.

asymmetry change over time [$F(1, 65) = 4.13$, $p = .021$]. Bonferroni corrected pairwise comparisons revealed that Group 3, which had the greatest change in pre-target α asymmetry over time, had significantly greater change in RT asymmetry than Group 1 [$p = .018$]. There was no significant difference between Group 2 and Group 1 or between Group 3 and Group 2 however.

The effect depicted in Fig. 3C confirms that the subset of participants with the greatest rightward shift in pre-target α asymmetry (i.e. the greatest increase in α activity over the right hemisphere ROI relative to the left hemisphere ROI) with time-on-task also had a significantly greater shift in RT asymmetry over time.

Finally, to explore the relationship between pre-target α asymmetry and absolute target detection speed we analysed absolute peripheral target RT data (as opposed to the RT asymmetry index) as a function of pre-target α asymmetry on a trial-by-trial basis. A rank-ordered pre-target α asymmetry (bin 1 vs. bin 2 vs. bin 3) $\times$ target-side (left vs. right) ANOVA revealed a significant main effect of target-side on the absolute RT data [$F(1, 65) = 11.71$, $p = .001$, $\eta_p^2 = .15$], reflecting the overall leftward RT asymmetry/pseudoneglect discussed previously. A significant main effect of α asymmetry

[$F(2, 130) = 7.58$, $p = .001$, $\eta_p^2 = .10$] revealed that absolute RTs were slower in those trials with more positive pre-target α asymmetry (see Fig. 3D). Bonferroni adjusted comparisons confirmed that RT's were significantly slower during the third of trials in which pre-target α asymmetry was most positive, compared to the third of trials in which pre-target α asymmetry was most negative [$p = .003$] (Fig. 3D). Given that more positive α asymmetry indicates greater α activity over the right relative to the left hemisphere ROI, this finding supports the notion that hemispheric α asymmetry is a marker of alertness levels which are known to depend on activation of the right lateralized ventral attention network (Paus et al., 1997; Sturm & Willmes, 2001).

3.3. The effect of central task load on pre-target α and RT asymmetry

As can be seen from Fig. 2A, absolute α activity during the pre-target epoch decreased with increasing central load [$F(2, 130) = 3.60$, $p = .030$, $\eta_p^2 = .05$] likely reflecting greater engagement due to task difficulty and increased visual processing of stimuli at fixation. Contrary to predictions however, we did not observe a

rightward shift in hemispheric α asymmetry (Fig. 2B) or RT asymmetry with increased central task load. Furthermore, the absence of any significant load by time-on-task interactions indicated the observed shifts in α and RT asymmetry with time-on-task were not exacerbated by increasing central task load. A main effect of central load on RT asymmetry was evident [$F(2, 130) = 5.01$, $p = .008$, $\eta_p^2 = .07$], which was driven by a leftward shift in RT asymmetry from no-load to low-load [$p = .003$] and no significant difference in RT asymmetry between low-load and high-load or no-load and high-load.

3.4. Post-target α asymmetry

Absolute α activity post target increased as a function of time-on-task [$F(1.36, 88.38) = 56.06$, $p < .001$, $\eta_p^2 = .46$] and decreased with greater central task load [$F(2, 130) = 8.50$, $p < .001$, $\eta_p^2 = .12$, see Fig. 2A]. Analysis of the post target α asymmetry index revealed a robust main effect of target-side [$F(1, 65) = 84.55$, $p < .001$, $\eta_p^2 = .57$] which reflected greater α-desynchronisation over contralateral electrodes, consistent with previous research (Kelly et al., 2009; Rihs et al., 2009; Sauseng et al., 2005; Thut et al., 2006; Yamagishi et al., 2005). Planned orthogonal polynomial contrasts on the post-target α asymmetry index indicated no significant effects of increasing load or time-on-task on post-target α asymmetry and no-load by time-on-task interaction. The same methods used to observe the link between changes in pre-target α asymmetry and RT asymmetry with time-on-task (described above), were applied to post-target α asymmetry data. Pearson's correlation between post-target α asymmetry change and RT asymmetry change with time-on-task revealed a non-significant trend in the same direction as was observed in the pre-target analysis [$r = .24$, $p = .053$]. The subsequent one-way between-groups ANOVA (post-target α asymmetry change groups: 1 vs. 2 vs. 3), with RT asymmetry change over time as the dependent variable, also did not reach significance [$F(1, 65) = 2.54$, $p = .087$]. These data therefore suggest that the link between shifting asymmetry in RT bias and anticipatory pre-target α with time-on-task is more robust than that for stimulus-driven post-target α.

3.5. Additional control analysis

To ensure the pre-target α results were not confounded by activation related to manual response preparation, the same pre-target α asymmetry analyses conducted on occipito-parietal ROIs was also conducted for lateralised electrodes over the motor cortex (left hemisphere: C3 C5; right hemisphere: C4 C6). Planned orthogonal polynomial contrasts revealed no change in motor cortex α asymmetry with either time-on-task [$F(1, 65) = 1.99$, $p = .163$] or central task load [$F(1, 65) = 3.31$, $p = .073$] and no interaction between these variables [$F(1, 65) = 2.45$, $p = .122$]. The methods used to observe the link between changes in pre-target occipito-parietal α asymmetry and RT asymmetry were applied to pre-target α from the lateralised motor electrodes. Pearson's correlation between α asymmetry change and RT asymmetry change with time-on-task was not significant [$r = .05$, $p = .675$] and the one-way between-groups ANOVA (α asymmetry change groups: 1 vs. 2 vs. 3) revealed no significant effect of α asymmetry change over motor areas on RT asymmetry change with time-on-task [$F(1, 65) = 1.77$, $p = .178$]. Thus the key pre-target occipito-parietal α effects reported above were not confounded by activation related to manual response preparation.

4. Discussion

This study is the first to link changes in hemispheric activation asymmetry to a rightward shift in spatial bias with time-on-task.

Pre-target occipito-parietal α-band activity, a marker of spatial attention previously linked to the dorsal attention network (Laufs et al., 2003, 2006; Mantini et al., 2007; Sadaghiani et al., 2010; Scheeringa et al., 2009), became more prominent over the right hemisphere relative to the left hemisphere as the task progressed, and this effect was greater in participants who had a more prominent behavioural change in spatial bias over time. While previous studies of preparatory α-activity explicitly cued pre-target attention to one hemifield (e.g. Thut et al., 2006), here participants were asked to distribute their attention across both hemifields in anticipation of peripheral targets. This allowed investigation of the natural fluctuations in preparatory α asymmetry, in the absence of any strategic top-down biasing of attention. Our results support the hypothesis that the hemispheric rivalry that characterises spatial attention networks is biased by depleting attention resources and arousal (Corbetta & Shulman, 2011). These findings accord with Corbetta and Shulman's (2011) neuroanatomical model whereby an increased demand on the right-lateralised ventral 'alertness' network should disproportionately affect recruitment of the right dorsal orienting network thus driving attention rightward. Pre-target α asymmetry was also significantly associated with absolute response speed to peripheral targets, independent of time-on-task and target-side, adding further support for the notion that hemispheric α asymmetry provides a general marker of arousal—a state that is known to be regulated by the right lateralized attention network (Corbetta & Shulman, 2011; Paus, et al., 1997; Sturm & Willmes, 2001).

The present findings not only provide clues about the interactions between spatial and non-spatial attention mechanisms in the healthy brain, but may also help to elucidate current uncertainties regarding the neural underpinnings of spatial neglect. For example, Mesulam's (1981) widely accepted model of neglect proposes that the right hemisphere directs attention to both hemifields, whereas the left hemisphere only directs attention to the right hemifield. According to this view, left hemisphere damage could be compensated for by the right hemisphere, but right hemisphere damage may not be compensated for thus leading to neglect of the left hemifield. Neuroimaging studies however suggest that both the left and right dorsal fronto-parietal networks direct attention predominantly to the contralateral visual field and are symmetrically, rather than asymmetrically, organised (Corbetta, Kincade, Ollinger, McAvoy, & Shulman, 2000; De Schotten et al., 2011; Hopfinger, Buonocore, & Mangun, 2000; Kastner, Pinsk, De Weerd, Desimone, & Ungerleider, 1999; Shulman et al., 2010). Furthermore, neglect is most commonly associated with lesions to the ventral attention network (specifically the right temporoparietal junction and inferior frontal gyrus) leading to a secondary disruption of structurally undamaged dorsal regions (Corbetta et al., 2005). Accordingly, Corbetta and Shulman (2011) argue that in the case of neglect, damage to the right lateralised ventral alertness network results in abnormal ventral-to-dorsal network interaction in the right hemisphere, producing an inter-hemispheric activation imbalance of the dorsal networks. Although the current findings are consistent with Corbetta and Shulman's (2011) model, they do not accord with Mesulam's (1981) model which does not account for a rightward shift in spatial bias with time-on-task that is accompanied by a time-dependent shift in pre-target hemispheric α asymmetry.

Recent EEG and TMS work (Romei, Gross, & Thut, 2010; Thut et al., 2011) suggests that pre-target posterior α may play a causal role in the manifestation of spatial bias. Romei et al. found that lateralised TMS stimulation at alpha, but not theta or beta frequencies, selectively impaired detection of unilateral visual targets in the contralateral hemifield, and enhanced detection in the ipsilateral hemifield. The authors argued that the beneficial effect of α stimulation for ipsilateral processing suggests a trans-callosal network effect, supporting the use of an α asymmetry

index that takes activation across both hemispheres into account to best index changes in the locus of spatial attention.

Previous studies have documented rightward attentional shifts under conditions of increased cognitive load in patients with acquired and developmental disorders of attention (Bellgrove, Eramudugolla, Newman, Vance, & Mattingley, in press; Bonato, Priftis, Marenzi, Umilta, & Zorzi, 2010; Peers et al., 2006; Russell, Malhotra, & Husain, 2004). Attempts to replicate the effect in healthy participants have however yielded inconsistent results (Dodds et al., 2008; Peers et al., 2006; Pérez et al., 2009). Pérez et al. (2009) found high cognitive load induced a rightward shift in spatial bias and in post-target α asymmetry using a temporal order judgment task. However, the authors did not observe any effect of load on pre-target α asymmetry suggesting that load influenced biases of stimulus-driven processing mechanisms rather than pre-stimulus preparatory processes. This is in line with O'Connell et al. (2011) where increased load asymmetrically disrupted the N1 response to peripheral stimuli for the right, but not left, hemisphere. Here we found no effect of attentional load on pre- or post-target α asymmetry. There was a subtle leftward shift in spatial bias from the no central task condition to the low-load condition, but no significant difference between the no central task and high-load conditions or between the low and high-load conditions. These results are not wholly surprising considering the discrepant findings of past research on the relationship between attentional load and spatial bias in healthy participants (Dodds et al., 2008; Pérez et al., 2009). Bellgrove et al. (in press) asked children with attention deficit hyperactivity disorder (ADHD) and right hemisphere patients with neglect to complete a paradigm similar to that used in the current study. Both groups displayed significant rightward shifts in spatial bias with increased non-spatial attention load at fixation. Although the significant capacity limitations associated with right hemisphere damage and ADHD make these participants more susceptible to the effects of attentional load (Bellgrove et al., in press; Bonato et al., 2010; Peers et al., 2006; Russell et al., 2004), it is possible that our manipulation of non-spatial load was not sufficiently strong to induce a rightward shift in attention in healthy participants.

A possible limitation of the current methodology is that participants took rest breaks during the task. The short rest periods may have partially restored alertness levels, diminishing the effect of time-on-task. Despite this, the observation that time-on-task had a significant effect on behavioural and electrophysiological asymmetry, even with the short rest periods, may encourage greater confidence in the effect. Future research is needed to explore whether psychostimulants that abolish rightward attention drift over time (Dodds et al., 2009) also abolish the rightward shift in hemispheric α asymmetry over time. Such a finding would bolster the arguments that α-band activity is an index of cortical activation/deactivation and that spatial bias is balanced by competition between the left and right hemisphere which is sensitive to decreasing arousal.

In conclusion, this is the first study to document a link between the rightward shift in spatial bias that occurs with time on a repetitive task and asymmetry in hemispheric α-activity that changes over time. These results lend support to the hypothesis that spatial bias is balanced by rivalry between the left and right hemisphere, and suggests that this balance is sensitive to decreasing arousal with time-on-task. These results support recent findings in both healthy and clinical samples emphasising the modulation of spatial bias by non-spatial attention processes. Such results in healthy participants accord with findings in neglect suggesting that damage to the right ventral 'alertness' network may lead to an inter-hemispheric activation imbalance in structurally intact bilateral dorsal orienting networks.

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