

Research report

Behavioural and physiological impairments of sustained attention after traumatic brain injury

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Abstract

Sustaining attention under conditions of low external demand taxes our ability to stay on task and to avoid more appealing trains of thought or environmental distractions. By contrast, a stimulating, novel environment engages attention far more freely without the subjective feeling of having to override monotony. Our ability to maintain a goal-directed focus without support from the environment requires the endogenous control of behaviour. This control can be modulated by fronto-parietal circuits and this ability is compromised following traumatic brain injury (TBI) leading to increased lapses of attention. In this paper, we further explore a laboratory paradigm that we argue is particularly sensitive to sustained attention as opposed to other aspects of attentional control involving the selection and management of goals in working memory. The paradigm (fixed sequence Sustained Attention to Response Task—SART_{fixed}) involves withholding a key press to an infrequent no-go target embedded within a predictable sequence of numbers. We demonstrate that TBI patients in this study make disproportionately more errors than controls on this task. An analysis of response times (RTs) and EEG alpha power across the task demonstrates group differences preceding the critical no-go trial. Controls demonstrate a lengthening of RTs accompanied by desynchronization of power within the alpha band (~10 Hz) preceding the no-go trial. Conversely, the TBI group showed a shortening of RTs during this period with no evidence of alpha desynchronization. These findings suggest that TBI patients may have dysfunctional alpha generators as a consequence of their injury that impairs endogenous control during the task.

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1. Introduction

Traumatic brain injury (TBI) is the cause of a large number of hospital admissions annually, and the majority of these admissions are young adults with a normal life expectancy who have to adapt to a lifetime of disability. The immediate impact of brain injury results in focal contusions and haemorrhages together with more diffuse stretching and lacerating of nerve fibres described as diffuse axonal injury [22,30]. Attention deficits are among the most commonly observed deficits following brain injury [1,16,35] and

damage to the frontal lobes of the brain particularly the white matter connecting frontal, parietal and striatal regions are, in part, responsible for these deficits [9,10]. It has been found that frontal lobe damage in TBI patients results in a tendency to drift from intended goals and increases the frequency of action-slips that were unintended [33]. Moreover, self-reports from traumatically brain-injured patients reveal that problems with attention and concentration rate among the highest complaints for this patient group [23,36].

Robertson et al. [32] developed a task designed to be more sensitive to transient lapses of attention. In the original Sustained Attention to Response Task (SART, referred to henceforth as SART_{random}), subjects were presented with a stream of random digits appearing sequentially at a rate of approximately one per second. They were instructed to press

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a response key to each digit but to withhold their response to a designated no-go trial occurring only 11% of the time. On this task, TBI patients made significantly more errors (recorded as false presses) than controls suggesting that they found it more difficult to combat the tendency to 'drift' into a more automatic mode of responding. Moreover, performance on the SART correlated with relatives' reports of everyday absentmindedness of the patients in the Cognitive Failures Questionnaire (CFQ) [3] demonstrating that the SART may be sensitive to absentmindedness in everyday life. It was also found that the timing of accurate responses during the SART was informative of error rates. False presses could be predicted by a shortening of response times (RTs) to the trials immediately preceding the critical no-go trials, supporting the argument that errors are a result of a transient drift in controlled processing coinciding with the period prior to a critical no-go trial.

The possibility that the above findings are more a consequence of faulty inhibitory processes than an impairment of sustained attention is reduced when the SART is modified by changing the random digit presentation to a predictable ascending sequence (1,2,3,4,5,6,7,8,9,1,2...etc.). Under these conditions, where a strong anticipatory cue precedes the no-go trial, TBI patients also perform significantly worse than controls. In fact, the effect size discriminating patients and controls is greatly enhanced for the SART_{fixed} compared to the SART_{random} [21], with the controls performing near perfectly, and the TBIs continuing to make a large number of errors. One interpretation of these findings was that TBI patients were again more susceptible to transient lapses of attention because of an automated or 'task-driven' mode of responding.

In a second experiment, Manly et al. examined patterns of activation [using Positron Emission Tomography (PET)] during SART_{random} and SART_{fixed} in healthy subjects. Subtractions were carried out to evaluate whether the seemingly low demand of the SART_{fixed} would be associated with increased activation in areas associated with sustained attention compared to the SART_{random}. It was found that activation was greater in both the right dorsolateral prefrontal cortex and the right superior/posterior parietal cortex for the SART_{fixed}. One prominent interpretation of these activation patterns suggest that the prefrontal cortex may act as an endogenous controller of alertness that may be more greatly needed during the ostensibly less challenging SART_{fixed}.

Posner and Peterson [29] have proposed the existence of three main functionally and anatomically distinct attentional control systems: an orienting system that relies upon the posterior brain areas including the superior parietal lobe and temporo-parietal junction, with additional involvement from the frontal-eye fields and is involved in the selection of sensory information; an executive system, involving the anterior cingulate, lateral prefrontal cortex and the basal ganglia (particularly caudate nucleus) responsible for exercising control over lower-level cognitive functions and resolving conflicts [28]; and an alerting or sustained attention system

[29] centred on fronto-parietal regions responsible for achieving and maintaining sensitivity to incoming stimuli. It is the latter dimension that we focus on in the current article.

We argue that the SART_{fixed} may be particularly sensitive to sustained attention deficits because it holds an important advantage over traditional measures of vigilance. Early vigilance paradigms [20] have only revealed marginal decrements in performance over time when subjects are required to monitor streams of information over long periods and detect infrequent targets. Furthermore, TBI patients make a similar proportion of errors in these tasks as controls [4,34] except under conditions where the visual targets are perceptually degraded [24]. It is likely that the novelty of rare targets will engage orienting attention systems and increase the likelihood of their successful detection—this may occur even under conditions of waxing and waning endogenous control. However, if the response contingencies are reversed so that subjects must withhold a frequent response in the context of a rare target, as in the SART, the dynamics of endogenous control become far more amenable to investigation.

Importantly, the SART_{fixed} represents a clinically useful extension of the SART_{random} owing to its larger discriminative power between TBI patients and controls. The sensitivity of the SART_{fixed} to TBI patients appears to be enhanced by reducing the prepotency or behavioural relevance associated with each trial making endogenous control over the task more challenging. In the present study, we attempt to elucidate the transient nature of endogenous control of attention during the SART_{fixed} by pursuing a trial-by-trial analysis of the response time data and by examining changes in EEG alpha activity in the anticipatory period preceding the no-go trial.

Changes in EEG alpha activity that are associated with a specific experimental manipulation can be detected by frequency analysis and are characterized as increases or decreases in band power [2,12]. In contrast to ERPs that represent neuronal discharges in response to specific events, alpha band power changes have been observed in the absence of external stimulation and reflect internally induced oscillations [2,26]. An increase in alpha power, as detected by scalp electrodes, is referred to as synchronization—a state in which large populations of neurons are firing together with the same phase and frequency, producing oscillations. Alpha synchronization has been associated with a resting state of the brain where mental activity is minimal [25] and has also been linked to a state of cortical inhibition where the suppression of stimulation in visual space is required so attention can be deployed to a auditory modality [8]. Conversely, a decrease in alpha power is described as desynchronization, and this occurs when the alpha rhythm start to shift its frequency because local neural generators of alpha start to oscillate independently at a microscale. States of alpha desynchronization are linked to increased cognitive demand [12], and hence the deployment of attention at the appropriate moment.

Klimesch's work on alpha desynchronization has established that subbands are distinguishable within the range of the extended alpha band. Using a subject's individual alpha frequency, three frequency bands each with a 2 Hz width can be defined. The bands are described as lower-1 alpha, lower-2 alpha and upper alpha, upon which a typical peak alpha frequency of 10 Hz corresponds to bands 6–8, 8–10 and 10–12 Hz, respectively. Klimesch et al. [13] investigated alpha power changes within these 2 Hz bands during a visual oddball task in which subjects were instructed to count targets but ignore nontargets. The structure of this task ensured that subjects were able to detect regularities within the sequence of stimuli, and consequently, they were able to make approximate predictions as to when targets would appear. Results showed a decrease in band power in lower-1 alpha followed warning tones that preceded targets suggesting that the tone had an alerting effect only when it preceded relevant targets and not the nontargets. Interestingly, and in contrast to lower-1 alpha, there was early decrease in band power in lower-2 alpha that began prior to the warning tones suggesting endogenous preparation for the imperative stimulus. Finally, desynchronization within upper alpha was found in the poststimulus interval when subjects correctly identified the target and updated their running count. This suggests that upper alpha is most sensitive to semantic or task-specific effects.

The locations of alpha generators within the brain are widely distributed across intracortical networks and are also influenced by thalamo–cortical circuits [19]. A recent combined PET and EEG study demonstrated that an inverse relationship exists between thalamic metabolism and alpha power. As thalamic activity increases, alpha power decreases, suggesting that a desynchronized alpha state is associated with increased thalamic metabolic activity [18]. Moreover, increases in attentional performance are associated with increased thalamic activity under conditions of low arousal [27], suggesting that enhanced attentional effort is required under these conditions.

In the present study, we further investigate the disproportional errors that arise for TBI patients compared to controls during the SART_{fixed}. Previous evidence [21,32] suggests that shortening of response time reflects a 'drift' of controlled processing during the task and a lengthening of RT reflects the endogenous deployment of attention to a routine task. We investigate patterns of RT across trial items, before and after the critical no-go trial. If the timely deployment of sustained attention is important for successful performance, we predict that controls will be more able than the TBI group to modify sustained attention at key trial positions prior to the upcoming no-go trial. This will be reflected in longer response times for controls compared to patients. We also investigate whether activity within the extended alpha band (~ 10 Hz) is associated with changes in sustained attention prior to the upcoming no-go trial predicting that controls may be more able than TBI patients to desynchronize alpha as the no-go trial approaches.

Furthermore, we explore the extent to which subbands of alpha (lower-1 alpha, lower-2 alpha and upper alpha) are associated with task performance as a function of group, hypothesizing that lower-alpha power may be selectively modulated by the attentional demands of the SART_{fixed} for control subjects more so than TBI patients.

2. Method

2.1. Participants

A total of 10 traumatically brain-injured participants and 10 non-brain-injured control participants were recruited for this study. One person reported with 'moderate' posttraumatic amnesia, five were classed as 'very severe' and four were classed as 'extremely severe'. The groups were matched according to gender, age and IQ. Characteristics of both the TBI group and the control group are outlined in Table 1. The TBI patients had not experienced the following: a major psychiatric disorder, a drug or alcohol dependency, a pretrauma history of epilepsy or any other neurological disorder. The control participants also fulfilled the latter requirements and additionally, had never suffered loss of consciousness from a head injury. Participation in the study was approved by the Department of Psychology, Trinity College Dublin ethics committee and by Headway, Ireland. All participants signed an informed consent form before the beginning of the experiment.

3. Materials

3.1. Self-report measures

In order to obtain a profile of cognitive and emotional functioning that pertain to everyday events, the Hospital

Table 1
Participant characteristics for TBI and control subjects

Characteristics	TBIs	Controls
<i>Gender</i>		
M	8	8
F	2	2
<i>Age (years)</i>		
Mean	35.50	38.70
S.D.	8.73	11.10
<i>Premorbid IQ (NART)</i>		
Mean	112.23	116.68
S.D.	6.83	4.14
<i>Time since injury^a</i>		
Mean	6.5	
S.D.	6.6	

Note: M: male; F: female.

^a Years from date of injury to the time of testing.

Anxiety Depression (HAD) scale and the Cognitive Failures Questionnaire (CFQ) were administered to all participants. The HAD [37] scale is comprised of 14 items, seven of which reflect anxiety levels and seven correspond to depression levels, while the CFQ [3] measures reported slips of action and memory in everyday life.

3.2. Neuropsychological tests

All participants undertook neuropsychological tests that indexed attention, memory and planning/strategy performance. These included two subtests from the Test of Everyday Attention (TEA) [31], namely, the Telephone Search and the Telephone Search While Counting. In addition, to assess memory performance, the subtests Logical Memory I and II were administered from the Wechsler Memory Scale (WMS-III^{uk}) (1998). Finally, to measure planning/strategy, the revised Strategy Application Test (R-SAT) [17] was administered. This task presents an unstructured environment in a laboratory setting whereby the most efficient strategy is challenged by salient external cues and internal habits. The best strategy involves completing the briefest items in three separate activities: figure tracing, sentence copying and object numbering. The primary score reflecting strategy application is the proportion of items that are classified as brief.

3.3. The fixed sequence Sustained Attention to Response Task (SART_{fixed})

In this modified version of the SART_{fixed}, digits were presented sequentially from '1' through '9'. A total of 945 digits were presented sequentially (105 of each of the nine digits) over an 18.1-min period. Each trial began with the

presentation of a digit for 250 ms followed by a 100 ms mask consisting of a ring with a diagonal cross in the centre. Next, a response cue was presented for 50 ms composed of a ring with an emboldened diagonal cross followed by a second mask—identical to the first—for 300 ms. Finally, a fixation cross was presented on screen for 450 ms preceding the next trial. Fig. 1 depicts the trial sequence. The participant was instructed to respond with a left mouse press as close to the response cue as possible following each digit (go-trials) with the exception of the 105 occasions when the digit 3 (target) appeared. The inclusion of a response cue was to prevent a speed–accuracy trade-off by attempting to pace the subject's responses by regular intervals.

In keeping with Robertson et al. [32], five randomly allocated digit sizes were presented to increase the demands for processing the numerical value and to minimize the possibility that subjects would set a search template for some perceptual feature of the target trial ('3'). Digit sizes were 8, 11, 14, 17 and 20 mm, respectively. The diameter of the mask, response cue and mask-2 were 23 mm each. The digits, masks and response cue were presented centrally in white on a computer monitor against a black background. The screen (320 × 240 mm; Dell PC) was positioned approximately 40 cm from the participant. The task specifications were programmed using E-prime. A practice block including 27 trials and three critical no-response targets preceded the SART_{fixed}.

3.4. EEG acquisition

Electrophysiological data were recorded in AC mode with a gain of 500 and a band pass of 0.15–30 Hz and the A/D conversion rate was 1000 Hz. Scalp potentials were

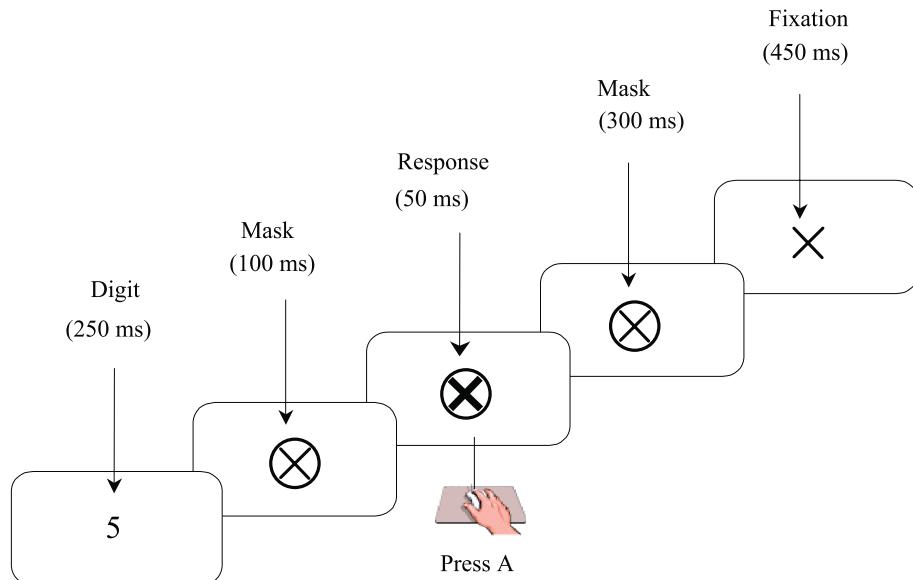


Fig. 1. A single trial sequence during the SART_{fixed}.

recorded using a 32-channel Quikcap using linked ear electrodes as ground and an anterior scalp reference site (AFz). The electrode array conformed to the International 10–20 System. Vertical eye movements were recorded with two VEOG electrodes placed above and below the left eye, while HEOG electrodes at the outer canthus of each eye recorded horizontal movements. Silver/silver-chloride (Ag/AgCl) electrodes were used at all sites. Participants were tested while seated in an armchair, with a pillow behind the head to reduce contamination of the recording by head or neck movements or muscle spindles. Recording began when electrical impedance had been reduced to less than 10 k Ω by light abrasion of the scalp. Data was recorded for a 5-min period in which subjects relaxed with their eyes open and subsequently for an 18.1-min period during the SART_{fixed}.

3.5. Signal processing

The data for each subject of both groups were epoched from –500 to +1750 relative to each presentation of the pretarget stimuli “9”, “1” and “2”. Only epochs during which no commission errors or omission errors were made were retained. Furthermore, epochs containing artifacts were rejected.

Each epoch was segmented into 11 overlapping 1-s segments, the first segment being centered on the stimulus presentation and the following segments taken in steps of 125 ms (Fig. 2). The Fourier Transform of each of the 11 segments was calculated, resulting in a standard Short Time Fourier Transform (STFT), the square of which is taken as a measure of spectral power. The ensemble-average squared STFTs for “9”, “1” and “2” were then calculated for each subject and electrode location.

Each subject's peak alpha frequency was selected by visual inspection of the average frequency spectrum over the entire task. The range of alpha and alpha-subbands were then defined relative to this Individual Alpha Frequency (IAF) as described above. The 11-point time course of power in each frequency range was then calculated by averaging the STFT across that range.

In order to compare power measures across individuals, some normalization is usually necessary to account for intersubject variability of EEG signal strength and also variability resulting from different recording conditions. To this end, a within-task reference power spectrum was calculated. Specifically, the mean squared STFTs for the “6”, “7” and “8” trials were calculated in the same way as the pretarget stimuli. The reference spectrum was calculated by averaging across time points contained within the fixation cross period (Fig. 1) of each of these trials, and subsequently across the three trials. The alpha and alpha-subband time courses were each normalized by dividing by the power in the corresponding range in the reference spectrum.

For the purposes of statistical analysis, task-related power in the extended alpha and each of the three alpha subbands was calculated by averaging across mean-squared STFT time points contained within the fixation cross period following each of the “9”, “1” and “2” stimuli. This period comes after normal event-related changes in alpha.

3.6. Procedure

Participants were assessed in two 1-h sessions. In the first session, the self-report measures and neuropsychological tests were administered, and in the second session, the EEG acquisition and SART was undertaken.

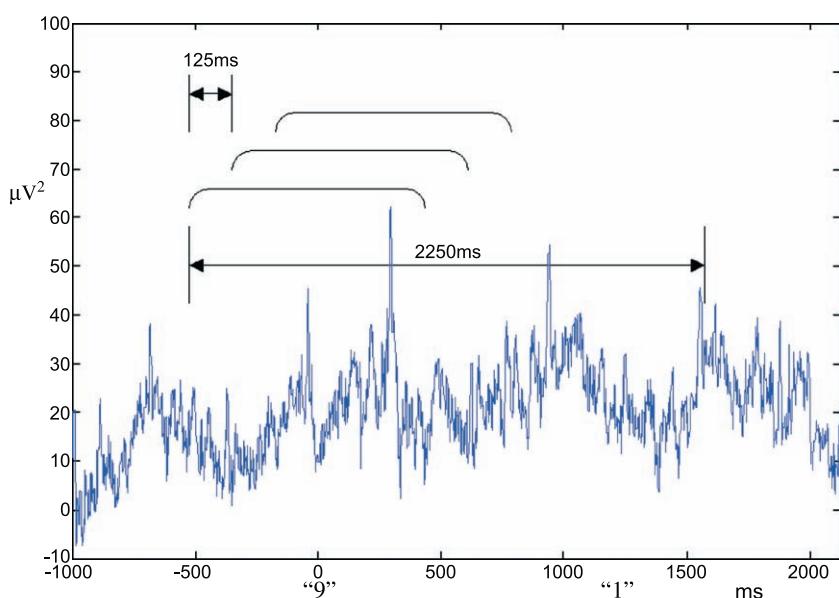


Fig. 2. Plot demonstrating stimulus epoch boundaries, and the first three of the sequence of nine STFT windows.

4. Results

4.1. Behavioural results

Table 2 details the mean scores for the self-report measures, the neuropsychological tests and the SART for each group, respectively.

No significant differences were found between the TBI and control groups for age ($t < 1$), premorbid IQ (NART) [$t(18) = 1.73, p = 0.11$] and HADS anxiety/depression scores [HAD-anxiety: $t(18) = 1.54, p = 0.41$; HAD-depression: $t(18) = 1.04, p = 0.31$].

Statistically reliable differences were found for CFQ scores between groups. Brain-injured groups reported a greater frequency of everyday cognitive failures compared to controls [$t(18) = 4.0, p = 0.006$]. The neuropsy-

chological tests showed differences in logical memory performance with brain-injured patients remembering significantly less story information both upon immediate recall [$t(18) = 2.53, p = 0.022$] and after a half-hour delay [$t(18) = 3.34, p = 0.004$]. Recognition performance between the two groups did not differ [$t(18) = 1.36, p = 19$]. Differences were also apparent between TBI and control groups on the revised strategy application test (R-SAT). TBIs completed significantly fewer brief items as a proportion of total items completed in contrast to controls [$t(18) = 5.09, p = 0.0001$]. Performance on the Telephone Search and the Telephone Search While Counting subtests of the Test of Everyday Attention (as measured by the dual task decrement scores) showed no differences between TBIs and controls [$t(18) = 1.74, p = 0.10$].

Table 2 shows that the SART_{fixed} significantly discriminated between the two groups with TBIs making reliably more commission errors compared to the control group [$t(18) = 2.43, p = 0.026$]. In order to investigate whether a commission error could be predicted by reduced attentional control prior to a target, we investigated response times (RTs) to trials '1' and '2' prior to targets for each group, respectively. A two-way mixed factorial ANOVA with group (TBIs, controls) as the between-subjects factor and trial (precommission error, precorrect withhold) as the within-subjects factor revealed no significant main effect of group [$F(1,18) = 1.06, p = 0.32$] and no group \times trial interaction ($F < 1$). A significant main effect of trial was apparent [$F(1,18) = 7.29, p = 0.015$] indicating that mean RTs to trials prior to a commission error were reliably shorter (457.00, S.D. 126.72) than mean RTs prior to a successful withhold (526.00, S.D. 133.60) irrespective of group.

There was also an RT pattern indicating differing response styles between TBIs and controls. To analyse this more closely, the task was split into pre- and post-target phases. The pretarget phase included trials 9, 1 and 2 and the posttarget phase encompassed trials 5, 6 and 7 (trial 4 was excluded from this analysis as it is associated with postprocessing of a correct withhold or error following the 3). Owing to the low trial count for commission errors, and therefore the corresponding pre- and postcommission error trials, the following analysis was restricted to go-trials only. A three-way mixed factorial ANOVA was conducted with Group (TBIs, controls) as the between-subjects factor and Phase (pretarget, posttarget) and Trial (9–1–2, 5–6–7) as the within-subjects factors. A Group \times Phase \times Trial interaction was observed [$F(2,36) = 6.70, p = 0.004$].

Lower-order interactions were then analysed separately. Firstly, a Group \times Trial ANOVA was undertaken for the pretarget phase. A significant Trial \times Group interaction was found [$F(2,36) = 8.20, p = 0.001$]. Simple main effects were conducted for TBIs and controls. The TBIs showed no RT differences for trial 9 and trial 1 items ($p = 0.837$) but they

Table 2
Self-report scores and neuropsychological test results for TBI and control subjects

	TBIs	Controls	<i>p</i>
<i>SART_{fixed}</i>			
Mean	11.40	5.10	0.026*
S.D.	5.10	3.96	
<i>Self-report measures</i>			
Hospital anxiety depression scale			
HAD—anxiety			
Mean	9.50	6.30	0.141
S.D.	3.98	5.23	
HAD—depression			
Mean	6.30	4.30	0.313
S.D.	4.50	4.11	
Cognitive failures questionnaire			
Mean	54.70	36.70	0.006**
S.D.	14.07	11.84	
<i>Neuropsychological tests</i>			
Test of everyday attention (dual task decrement score)			
Mean	2.83	0.79	0.100
S.D.	3.69	0.44	
Logical memory I (immediate recall)			
Mean	38.56	49.30	0.022*
S.D.	9.74	8.81	
Logical memory II (delayed recall)			
Mean	21.78	31.90	0.004**
S.D.	6.74	6.47	
Logical memory II (recognition)			
Mean	24.89	26.50	0.192
S.D.	2.57	2.59	
Revised strategy application task			
Mean	0.65	0.93	0.0001**
S.D.	0.15	0.07	

Note: * 0.05; ** 0.01 (significance levels).

exhibited significantly shorter RTs to trial 2 items compared to trial 1 items ($p=0.003$). Conversely, the controls showed reliably longer RTs to trial 1 compared to trial 9 items ($p=0.0004$) and maintained longer response times for trial 2, not significantly differing from trial 1 items ($p=0.927$). Group differences were only marginally significant at trial 2 with controls showing a trend towards longer RTs compared to TBIs ($p=0.077$).

Secondly, a Group \times Trial ANOVA was conducted for the posttarget phase. No main effect of Group ($F<1$) or Group \times Trial interactions [$F(2,36)=1.38$, $p=0.265$] was found but a significant main effect of Trial was apparent

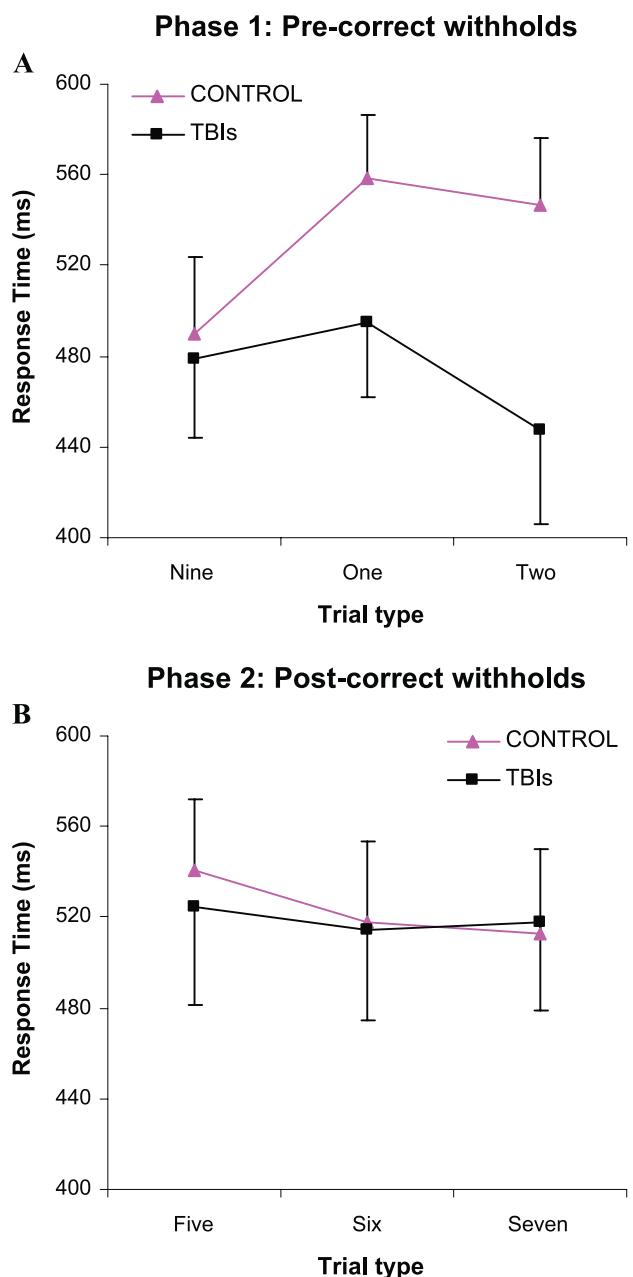


Fig. 3. Response times as a function of group (TBI, control) and phase (Phase 1: precorrect withholds; Phase 2: postcorrect withholds).

Table 3

The 2×3 mixed ANOVAs for each electrode location, with Group (TBIs, controls) as the between-subjects factor and Trial (9, 1, 2) as the within-subjects factor

Electrode	Main factors F		Interaction F Group \times Trial ($df=2,34$)
	Group ($df=1,17$)	Trial ($df=2,34$)	
CP3	$F<1$	3.696, $p=0.035^*$	3.681, $p=0.036^*$
FC3	$F<1$	2.956, $p=0.066$	3.811, $p=0.033^*$
FCZ	$F<1$	$F<1$	3.256, $p=0.051$
FT7	$F<1$	3.834, $p=0.032^*$	1.120, $p=0.338$
P3	$F<1$	$F<1$	3.614, $p=0.038^*$
P7	$F<1$	1.322, $p=0.281$	3.341, $p=0.048^*$
T7	$F<1$	6.017, $p=0.006^{**}$	3.555, $p=0.041^*$
TP7	$F<1$	2.966, $p=0.068$	3.134, $p=0.059$

Note: * 0.05 ; ** 0.01 (significance levels).

[$F(2,36)=4.38$, $p=0.020$]. Simple main effect comparisons revealed a significant decrease in response time from trials 5 to 6 ($p=0.033$) but no significant difference between trials 6 and 7 ($p=0.99$).¹ Fig. 3A and B displays the response times for each trial according to group and phase.

5. Electrophysiological results

5.1. Extended alpha band (~ 10 Hz) analysis

Alpha power at rest with eyes open was initially compared between groups. There was no significant difference in alpha power between TBI patients (mean = 0.148, S.D. = 0.152) and controls (mean = 0.045, S.D., 0.098) [$t(17)=1.724$, $p=0.108$]. Associated EEG changes within the critical period of trial 9 through to the onset of trial 3 were examined. Analysis of power within the extended alpha range (~ 10 Hz) was conducted for all electrode sites. For each site, a two-way mixed 2×3 ANOVA was conducted with Group (TBIs,² controls) as the between-subjects factor and Trial (9, 1, 2) as the within-subjects factor.

Table 3 shows the ANOVA results, confined to the electrode sites where significant/marginal main effects or interactions were found. The effects are apparent on the midline and generalized in the left hemisphere in frontal, central, temporal and parietal sites. The reported interactions were driven by a within-subjects difference in the control group demonstrated by a reduction in alpha power from trials 9 to 1 (FCZ, $p=0.026$; FC3, $p=0.001$; P7, $p=0.084$; T7, $p=0.066$) or a reduction in alpha from trials 9 to 2 (CP3, $p=0.001$; P7, $p=0.074$; T7, $p=0.002$; TP7, $p=0.033$). TBI patients failed to show evidence of alpha

¹ Bonferroni adjustments for multiple comparisons have been made for these and all subsequent tests.

² One TBI participant's data was excluded from the analysis due to excessive artifacts.

desynchronization at any electrode location during trials 9, 1 and 2 (all $p>1$) with the exception of electrode FT7. Main effect contrasts demonstrated that alpha power desynchronized from trials 9 to 2, irrespective of group, at FT7 ($p=0.016$). Fig. 4A and B depicts alpha power as a function of Group and Trial.

5.2. Alpha subbands analysis

Firstly, eyes open power was compared across groups for each subband (lower-1 alpha, lower-2 alpha and upper alpha). A 2×3 mixed ANOVA was conducted and no main effect of group [$F(1,17)=2.627$, $p=0.123$] was found indicating that power within each of the subbands did not differ as a function of group (means are displayed in Table 4). Furthermore, no main effect of Subband [$F(2,34)=1.006$, $p=0.376$] or Group \times Subband interaction ($F<1$) were observed.

Table 4

Means and standard deviations for eyes-open alpha power as a function of alpha subband and group

Alpha subband	Controls ($n=10$)	TBIs ($n=9$)
<i>Lower-1 alpha</i>		
Mean	0.031	0.152
S.D.	0.049	0.149
<i>Lower-2 alpha</i>		
Mean	0.071	0.160
S.D.	0.176	0.169
<i>Upper alpha</i>		
Mean	0.047	0.138
S.D.	0.113	0.165

Subsequent analyses explored whether alpha desynchronization varied as a function of alpha subband. ANOVAs were restricted to the electrode sites that yielded significant

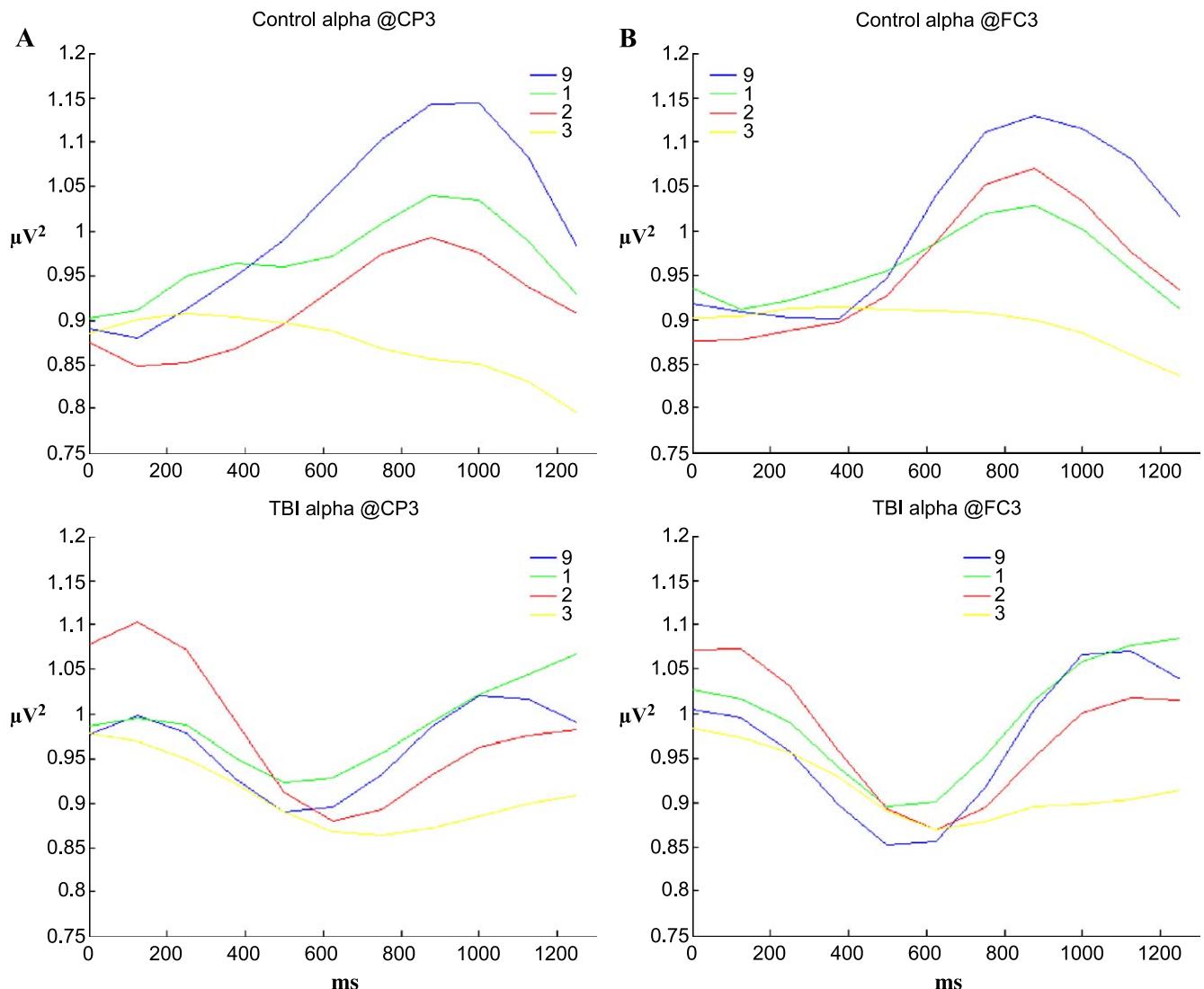


Fig. 4. Alpha power (~10 Hz) as a function of group (TBI, control) and trial (9, 1, 2) at electrode locations CP3 (A) and FCZ (B).

Table 5

The $2 \times 3 \times 3$ mixed ANOVAs for each electrode location, with Group (TBIs, controls) as the between-subjects factor and Trial (9, 1, 2) and Subband (lower-1 alpha, lower-2 alpha, upper alpha) as the within-subjects factors

Electrode	Main factors Fs Group (df=1,17)	Trial (df=2,34)	Subband (df=2,34)	Interaction Fs Subband \times Group (df=2,34)	Trial \times Group (df=2,34)	Subband \times Trial (df=2,34)	Subband \times Trial \times Group (df=4,68)
CP3	$F < 1$	$5.562, p = 0.008^{**}$	$9.972, p = 0.001^{**}$	$F < 1$	$3.120, p = 0.057$	$F < 1$	3.063, p = 0.042*
FC3	$F < 1$	$2.732, p = 0.084$	$2.664, p = 0.093$	$1.667, p = 0.208$	4.710, p = 0.018*	$F < 1$	$2.530, p = 0.058$
FCZ	$F < 1$	$F < 1$	$3.154, p = 0.076$	$F < 1$	$4.088, p = 0.026^*$	$1.700, p = 0.177$	3.525, p = 0.020*
P3	$F < 1$	$F < 1$	$2.785, p = 0.092$	$F < 1$	$3.819, p = 0.033^*$	$F < 1$	2.905, p = 0.042*
P7	$F < 1$	$1.166, p = 0.322$	$1.577, p = 0.225$	$F < 1$	3.518, p = 0.042*	$F < 1$	$F < 1$
T7	$F < 1$	$7.795, p = 0.002^{**}$	$F < 1$	$F < 1$	3.723, p = 0.032*	$2.842, p = 0.053$	$F < 1$

** $p < 0.01$, * $p < 0.05$; **BOLD** type indicates highest order interaction for each electrode site.

interactions for the extended alpha band (CP3, FC3, FCZ, P3, P7, T7). three-way $2 \times 3 \times 3$ mixed ANOVAs³ were conducted with Group as the between-subjects factor and Trial and Subband as the within-subjects factors for each electrode location. Table 5 shows that significant three-way interactions were observed at CP3, FCZ and P3 only.

Lower-order analyses examined two-way repeated measures Trial \times Subband ANOVAs for each group separately. At CP3, for the control group, a main effect of trial was found [$F(2,18) = 7.130, p = 0.019$]. No significant main effects of Subband [$F(2,18) = 3.939, p = 0.067$] or Subband \times Trial [$F(4,36) = 3.050, p = 0.065$] were observed. A simple contrast confirmed that desynchronization across all subbands was apparent from trials 9 to 2 ($p = 0.0001$). Conversely, the TBI patients showed no main effect of Trial [$F(2,16) = 1.782, p = 0.208$] and no Subband \times Trial interaction [$F(4,32) = 1.131, p = 0.359$]. Only a main effect of Subband was apparent [$F(2,16) = 6.199, p = 0.010$]. Simple contrasts demonstrated that lower-1 alpha power was enhanced relative to upper alpha power irrespective of trial ($p = 0.014$) for TBI patients.

At FCZ, for the control group, no main effect of Subband [$F(2,18) = 1.262, p = 0.307$] or Trial [$F(2,18) = 2.596, p = 0.102$] was observed. However, a marginally significant Subband \times Trial interaction was found [$F(4,36) = 3.008, p = 0.059$]. This interaction was driven by desynchronization from trials 9 to 2 in the lower-1 alpha band ($p = 0.085$) and a decline in lower-2 alpha power from trials 9 to 1 ($p = 0.071$) but no evidence of desynchronization in the upper alpha band ($p = 0.189$). By contrast, the TBI patients did not show a significant main effect of Subband [$F(2,16) = 2.236, p = 0.154$] or Trial [$F(2,16) = 2.261, p = 0.137$] and no Subband \times Trial interaction [$F(4,32) = 2.250, p = 0.108$].

At P3, the control group showed a significant Subband \times Trial interaction [$F(4,36) = 2.991, p = 0.035$]. Pairwise comparisons revealed no differences (all $p > 1$) suggesting that there was no significant change in power between trials for each level of subband. Additionally, no main effects of Subband ($F < 1$) or Trial [$F(2,18) = 3.015, p = 0.053$]

$p = 0.086$] were observed. The overall trend at P3 was consistent with a decline in all alpha subbands across trials from 9 to 2. For the TBI patients, no significant main effects of Subband [$F(2,16) = 1.177, p = 0.337$] or Trial [$F(2,16) = 1.701, p = 0.218$] was found and no significant Subband \times Trial interaction ($F < 1$).

The highest order interactions observed for sites FC3, P7 and T7 were Group \times Trial interactions (see Table 5). Trial was therefore examined separately for each group. At FC3, the interaction was driven by a decline in power from trials 9 to 1 ($p = 0.001$) for the controls but not for the TBI patients ($p = 0.513$). At T7, there was a decline in power from trials 9 to 1 ($p = 0.026$) and from trials 9 to 2 ($p = 0.001$) in the control group but no similar decline in power for the TBI patients (all $p > 1$). At P7, the controls exhibited a similar trend of decreasing power between trials 9 and 1 ($p = 0.080$) and between trials 9 and 2 ($p = 0.078$). Conversely, TBIs failed to desynchronize power across trials (all $p > 1$).

6. Discussion

The TBI participants in this study made disproportionately more errors than controls on the SART_{fixed}. This replicates Manly et al.'s findings, confirming that the paradigm has good sensitivity at discriminating error rates of TBI patients and controls. An analysis of response times across the task demonstrates group differences in response style preceding the critical no-go trial. Specifically, the controls show a slowing in latency at the beginning of the ascending sequence in anticipation for the no-go trial. The TBI group fail to show this increase in response time and, conversely, exhibited shorter RTs from immediately prior to the no-go trial. Electrophysiological differences were also found dissociating TBI patients and controls. The control group demonstrated a gradual decline in alpha power before the upcoming no-go trial. This pattern was not apparent in the TBI group (except at FT7) who showed significantly more variable alpha during this preparatory period but no overall decline. A further analysis of the alpha subbands revealed three electrode sites (FCZ, CP3 and P3) where subband interacted with group and trial. Only at FCZ was

³ While Huynh–Feldt corrections were applied to all repeated measures comparisons, unadjusted degrees of freedom (df) are reported to obviate sample size.

there tentative evidence of a modulation of the lower alpha bands that was dissociable from the upper alpha band. Here, lower-1 alpha power declined from trials 9 to 2 and lower-2 alpha power reduced from trials 9 to 1 for the control subjects. These effects were absent in TBI patients. Moreover, TBI patients showed significantly higher power in the lower-1 alpha relative to upper alpha power at CP3.

The behavioural results show that the TBI group were significantly impaired on the SART_{fixed} and our analysis of the RT data, and the lower-2 alpha power suggests that patients may be less adept at enhancing sustained attention as the upcoming no-go trial approached, supporting the argument that errors are a result of a transient drift in controlled processing [21,32]. Controls were more able than the TBI group at modifying sustained attention at key trial positions prior to the upcoming no-go trial. The areas responsible for this kind of top-down control in TBI patients may be compromised by injury either as a consequence of cortical damage at the site of impact or as a result of diffuse axonal injury. The current findings suggest that damage to either intracortical or thalamo-cortical networks after brain injury may disrupt alpha generators that are involved in the deployment of endogenous processes and impair sustained attention performance. In addition, there was some evidence of selective changes within the lower alpha bands. The desynchronization of lower alpha oscillations may be an important aspect of heightened endogenous preparation in tasks with predictable target occurrences (see Ref. [13]).

The electrode sites that yielded significant effects in this study were on the midline and lateralized to the left hemisphere. Although the spatial resolution of EEG is poor, this finding appears inconsistent with Manly et al.'s [21] PET study demonstrating that performance on the SART involves a right lateralized fronto-parietal network. Firstly, it should be noted that Manly et al. examined tonic activation during the SART across all trials ($n=90$), whereas our comparisons specifically reflect dissociations between clinical and normal groups for selected trials that predict the no-go stimulus. Consequently, the findings are not directly comparable. Secondly, the possibility that alpha power can be modulated in bilateral fronto-parietal circuits has been demonstrated recently in simultaneous fMRI and EEG studies [14,15], showing that alpha power was negatively correlated with the hemodynamic activations in bilateral fronto-parietal areas. Thirdly, it has been proposed that activation of the left dorsolateral prefrontal cortex during an event-related fMRI study [7] is involved in the representation of task goals (task set) during the SART_{fixed}. This raises the possibility that the left hemisphere effects in the current study may reflect the activation of a task-set that serves to deploy attentional control as the upcoming no-go trial approaches. Indeed, Fassbender et al. have argued that the left dorsolateral prefrontal cortex may be responsible for reactivation of task set prior to the occurrence of the predictable number 3 during the SART_{fixed}.

The assertion that TBI patients have a particular difficulty with the timely deployment of sustained attention is supported by a two-phase distinction that underpins the normal pattern of responses during the task. In Phase 1 (trials 9–2), the controls appear to enhance their sensitivity to incoming stimuli as reflected by a lengthening of RTs and desynchronization of alpha power in anticipation of the no-go trial. In Phase 2 (trials 5–8), subjects adopt a more automated mode of responding until the end of the ascending sequence indicated by a shortening of RTs during this period. The argument that control subjects simply maintain a higher state of alertness during the entirety of the task seems less convincing in light of this two-phase distinction. Moreover, the possibility that the poorer performance of the TBI group is a consequence of tonic underarousal is less tenable, in view of the fact that patients are still able to correctly withhold their responses to the no-go trial on the majority of trials (on average, 93.6 out of a possible 105), suggesting that they maintain a sufficient level of arousal throughout the task but are more sensitive to transient lapses of controlled processing. There was also no evidence of task-related increase in errors over the 18.1-min period, demonstrating that the transient lapses that occurred during the SART were not related to time-on-task, and therefore were unlikely to be directly related to levels of arousal.

If the ability to maintain sustained attention or alertness is compromised in TBI patients, then this decrement may impact upon other attentional control systems, in particular, the executive control system [29]. The predictive sequence inherent in the SART_{fixed} provides a framework for planning ahead and preparing for a correct withhold on the designated no-go trial. Although the demands of selection and goal management are minimal during the SART_{fixed} (selecting the '3' as the no-go trial and using the ascending sequence as a countdown cue), it is possible that TBI patients may be less adept at making use of these cues if their sustained attention system is compromised.

It is conceivable that individuals, when undertaking the SART_{fixed}, switch between an externally attentive mode that is responsive to stimulus processing and an internal prospective mode where current goal-states are monitored. This approach may reflect the adoption of an attention–intention cycle (see Ref. [6]). In light of the aforementioned two-phase approach to the task, the beginning of the ascending sequence may serve as a cue to reflect upon the upcoming goal and to subsequently enhance stimulus processing prior to the no-go trial. The adoption of this relatively simple strategy during the task would, at all times, be dependent on the successful maintenance of sustained attention. Therefore, slipping into a more automated mode of responding, induced by the monotony of the task or engages more appealing trains of thought would lead to a failure to implement a simple, but effective, intention–attention cycle. A recent PET study [5] has demonstrated that areas of the lateral prefrontal cortex are associated with maintaining internally

generated goals while medial prefrontal structures are recruited for the suppression of conflicting representations.

Examining the dynamics of the proposed attention–intention cycle could be achieved by investigating focal desynchronization and surround synchronization which may reflect goal monitoring in the case of the former and perceptual gating or cortical inhibition in the case of the latter [11].

In summary, the results of this study confirm that the SART_{fixed} is an effective clinical measure for discriminating sustained attention performance of TBI patients and controls. A trial-by-trial analysis demonstrated that patterns of RTs were dissociable in the TBI and control groups. The control data suggested that they adopted a two-phase approach to the task in which slowing of RTs occurred prior to the no-go trial and afterwards RTs shortened, suggesting a more automatic mode of responding. By contrast, the TBI group showed no evidence of slowing prior to the no-go trial. Secondly, electrophysiological data demonstrated that controls were able to desynchronize alpha power in anticipation of the no-go stimulus. TBI patients, by contrast, showed more variable alpha power and did not demonstrate a pattern of desynchronization prior to the upcoming no-go trial. These findings suggest that TBI patients may have dysfunction alpha generators as a consequence of their injury, which compromises endogenous control during the task. We argue that a simple attention–intention cycle may underlie successful task performance whereby the key task goal is refreshed and sustained attention is enhanced at regular and systematic points during the SART_{fixed}. Importantly, the reason for the failure of this cycle is *not* its breakdown but a failure of the sustained attention system to support the implementation of this simple but effective attention–intention cycle.

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References

- [1] S.H. Auerbach, Neuroanatomical correlates of attention and memory disturbances in traumatic brain injury: an application of neurobehavioural subtypes, *Journal of Head Trauma Rehabilitation* 1 (1986) 1–12.
- [2] E. Basar, M. Schurmann, C. Basar-Eroglu, S. Karakas, Alpha oscillations in brain functioning: an integrative theory, *International Journal of Psychophysiology* 26 (1997) 5–29.
- [3] D.E. Broadbent, P.F. Cooper, P. Fitzgerald, K.R. Parkes, The Cognitive Failures Questionnaire (CFQ) and its correlates, *British Journal of Clinical Psychology* 21 (1982) 1–16.
- [4] W.H. Brouwer, P.C. van Wolffelaar, Sustained attention and sustained effort after closed head injury: detection and 0.10 Hz heart rate variability in a low event rate vigilance task, *Cortex* 21 (1985) 111–119.
- [5] P.W. Burgess, S.K. Scott, C.D. Frith, The role of the rostral frontal cortex (area 10) in prospective memory: a lateral versus medial dissociation, *Neuropsychologia* 41 (8) (2003) 906–918.
- [6] J. Crosley Shaw, Intention as a component of the alpha-rhythm response to mental activity, *International Journal of Psychophysiology* 24 (24) (1996) 7–23.
- [7] C. Fassbender, K. Murphy, J.J. Foxe, G.R. Wylie, D.C. Javitt, I.H. Robertson, H. Garavan, A topography of executive functions and their interactions revealed by functional magnetic resonance imaging, *Cognitive Brain Research* (in press).
- [8] J.J. Foxe, G.V. Simpson, S.P. Ahlfors, Parieto–occipital 10 Hz activity reflects anticipatory state of visual mechanisms, *NeuroReport* 9 (1998) 3929–3933.
- [9] S.D. Friedman, W.M. Brooks, R.E. Jung, B.L. Hart, R.A. Yeo, Proton MR spectroscopic findings correspond to neuropsychological function in traumatic brain injury, *American Journal of Neuroradiology* 19 (10) (1998) 1879–1885.
- [10] M.R. Garnett, A.M. Blamire, R.G. Corkhill, T.A.D. Cadoux-Hudson, B. Rajagopalan, P. Styles, Early proton magnetic resonance spectroscopy in normal-appearing brain correlates with outcome in patients following traumatic brain injury, *Brain* 123 (2000) 2046–2054.
- [11] M.A. Goodale, A.D. Milner, Separate visual pathways for perception and action, *Trends in Neurosciences* 15 (1) (1992) 20–25.
- [12] W. Klimesch, EEG alpha and theta oscillations reflect cognitive and memory performance: a review and analysis, *Brain Research Reviews* 29 (1999) 169–195.
- [13] W. Klimesch, M. Russegger, H. Doppelmayr, J. Pachinger, Induced and evoked band power changes in an oddball task, *Electroencephalography and Clinical Neurophysiology* 108 (1998) 123–130.
- [14] H. Laufs, A. Kleinschmidt, A. Beyerle, E. Eger, A. Salek-Haddadi, C. Preibisch, K. Krakow, EEG-correlated fMRI of human alpha activity, *NeuroImage* 19 (4) (2003) 1463–1476.
- [15] H. Laufs, K. Krakow, P. Sterzer, E. Eger, A. Beyerle, A. Salek-Haddadi, A. Kleinschmidt, Electroencephalographic signatures of attentional and cognitive default modes in spontaneous brain activity fluctuations at rest, *Proceedings of the National Academy of Sciences of the United States of America* 100 (19) (2003) 11053–11058.
- [16] H.S. Levin, F.C. Goldstein, Neurobehavioural aspects of traumatic brain injury, in: P. Bach-y-Rita (Ed.), *Comprehensive Neurologic Rehabilitation*, vol. 2, Demos, New York, NY, 1989, pp. 53–72.
- [17] B. Levine, D. Dawson, I. Boutet, M.L. Schwartz, D.T. Stuss, Assessment of strategic self-regulation in traumatic brain injury: its relationship to injury severity and psychosocial outcome, *Neuropsychology* 14 (4) (2000) 491–500.
- [18] K.A. Lindgren, C.L. Larson, S.M. Schaefer, H.C. Abercrombie, R.T. Ward, T.R. Oakes, J.E. Holden, S.B. Perlman, R.M. Benca, R.J. Davidson, Thalamic metabolic rate predicts EEG alpha power in healthy control subjects but not in depressed patients, *Biological Psychiatry* 45 (8) (1999) 943–952.
- [19] F.H. Lopes da Silva, J.E. Vos, J. Mooibroek, A. van Rotterdam, Relative contribution of intracortical and thalamo–cortical processes in the generation of alpha rhythms, revealed by partial coherence analysis, *Electroencephalography and Clinical Neurophysiology* 50 (1980) 449–456.
- [20] F.J. Mackworth, Vigilance, arousal and habituation, *Psychological Review* 75 (1968) 308–322.
- [21] T. Manly, A.M. Owen, L. McAvinue, A. Datta, G.H. Lewis, S.K. Scott, C. Rorden, J. Pickard, I.H. Robertson, Enhancing the sensitivity of a sustained attention task to frontal damage. Convergent clinical and functional imaging evidence, *Neurocase* 9 (4) (2003) 340–349.
- [22] A.J. Mattson, H.S. Levin, Frontal lobe dysfunction following closed head injury: a review of the literature, *Journal of Nervous and Mental Diseases* 178 (1990) 282–291.
- [23] W.M. McKinlay, The short-term outcome of severe blunt head injury as reported by relatives of the injured persons, *Journal of Neurology, Neurosurgery, and Psychiatry* 44 (1981) 527–533.
- [24] R. Parasuraman, S.A. Mutter, R. Molloy, Sustained attention follow-

ing mild closed-head injury, *Journal of Clinical and Experimental Neuropsychology* 13 (1991) 789–811.

[25] G. Pfurtscheller, Event-related synchronization (ERS): an electrophysiological correlate of cortical areas at rest, *Electroencephalography and Clinical Neurophysiology* 83 (1) (1992) 62–69.

[26] G. Pfurtscheller, F.H. Lopes da Silva, Event-related EEG/MEG synchronization and desynchronization: basic principles, *Clinical Neurophysiology* 110 (1999) 1842–1857.

[27] C.M. Portas, G. Rees, A.M. Howseman, O. Josephs, R. Turner, C.D. Frith, A specific role for the thalamus in mediating the interaction of attention and arousal in humans, *Journal of Neuroscience* 18 (1998) 8979–8989.

[28] M.I. Posner, G.J. DiGirolamo, Cognitive neuroscience: origins and promise, *Psychological Bulletin* 126 (6) (2000) 873–889.

[29] M.I. Posner, S.E. Peterson, The attention system of the human brain, *Annual Review of Neuroscience* 13 (1990) 25–42.

[30] J.T.E. Richardson, *Clinical and Neuropsychological Aspects of Closed Head Injury*, Psychology Press, Hove, East Sussex, 2000.

[31] I.H. Robertson, T. Ward, V. Ridgeway, I. Nimmo-Smith, The structure of normal human attention: the test of everyday attention, *Journal of the International Neuropsychological Society* 2 (6) (1996) 525–534.

[32] I.H. Robertson, T. Manly, J. Andrade, B.T. Baddeley, J. Yiend, Oops!: performance correlates of everyday attentional failures in traumatic brain injury and normal subjects, *Neuropsychologia* 35 (6) (1997) 747–758.

[33] T. Shallice, P. Burgess, Deficits in strategy application following frontal lobe damage in man, *Brain* 114 (1991) 727–741.

[34] J.M. Spikman, A.H. van Zomeren, B.G. Deelman, Deficits of attention after closed-head injury: slowness only? *Journal of Clinical and Experimental Neuropsychology* 18 (5) (1996) 755–767.

[35] A.H. VanZomeren, W.H. Brouwer, Head injury and concepts of attention, in: H.S. Levin, J. Grafman, H.M. Eisenberg (Eds.), *Neurobehavioral Recovery from Head Injury*, Oxford Univ. Press, New York, 1987.

[36] A.H. VanZomeren, W.V.D. Burg, Residual complaints of patients two years after severe closed head injury, *Journal of Neurology, Neurosurgery, and Psychiatry* 48 (1985) 21–28.

[37] A.S. Zigmond, R.P. Snaith, The hospital anxiety and depression scale, *Acta Psychiatrica Scandinavica* 67 (1983) 361–370.