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# Self-Alert Training: Volitional modulation of autonomic arousal improves sustained attention

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**Abstract**

The present study examines a new alertness training strategy (Self-Alert Training, SAT) designed to explore the relationship between the top–down control processes governing arousal and sustained attention. In order to maximally target frontal control systems SAT combines a previously validated behavioural self-alerting technique [Robertson, I. H., Tegner, R., Tham, K., Lo, A., & Nimmo-Smith, I. (1995). Sustained attention training for unilateral neglect: Theoretical and rehabilitation implications. *Journal of Clinical and Experimental Neuropsychology*, *17*, 416–430] with an autonomic arousal biofeedback protocol in which participants learn to modulate their own arousal levels. The SAT protocol was first validated with a group of 23 neurologically healthy participants and then independently tested in a group of 18 adults with ADHD to determine its clinical utility. Half of the participants in each group were assigned to a placebo condition to control for non-specific effects. All participants performed the sustained attention to response task (SART) during pre- and post-training testing sessions to assess training effects on sustained attention. By the end of SAT all participants were able to modulate their own arousal levels without external prompting. Comparison of pre- and post-training baseline data indicated that, as predicted, SAT was associated with increased levels of autonomic arousal accompanied by improved accuracy on the SART. In contrast, participants in the placebo condition exhibited a gradual reduction in arousal over time and increased reaction time variability indicative of a vigilance decrement. These data demonstrate that the recruitment of top–down control processes during volitional modulation of arousal leads to improved sustained attention. These findings have important implications for the rehabilitation of attention deficits arising from frontal dysfunction.


*Keywords:* Sustained attention; Arousal; Prefrontal cortex; Neuropsychological training; Electrodermal activity

## 1. Introduction

Sustained attention is a core executive function and can be defined as the ability to maintain an alert, goal-directed focus in the absence of exogenous stimulation (Robertson & Garavan, 2004). Failures of sustained attention occur when there is a transient decrease in mindful, endogenous control of behaviour leaving one prone to goal-neglect and distraction by irrelevant stimuli. An increased susceptibility to attentional lapses in everyday life is a common consequence of frontal lobe damage (Manly & Robertson, 1997) and has also been highlighted in a number of prominent clinical conditions including attention-deficit/hyperactivity disorder (ADHD) (Barkley, 1998; Swanson et al., 2004). Difficulty maintaining goal-directed attention is one of the most frequently cited problems reported by individuals with a traumatic brain injury (Van Zomeren & Burg, 1985) and there is evidence that such deficits can interfere with rehabilitative efforts in non-cognitive domains. For example, the capacity to self-sustain attention has been shown to predict motor recovery following right hemisphere stroke over a 2-year period (Robertson, Ridgeway, Greenfield, & Parr, 1997b). Hence, there is a clear imperative for the development of interventions that can address these deficits.

The available evidence from fMRI, PET and pharmacological studies indicates that sustained attention is achieved through a primarily right lateralised, multimodal cortical network that

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includes the anterior cingulate gyrus, the right dorsolateral prefrontal cortex and the inferior parietal lobule with prominent reciprocal connections to the thalamus and noradrenergic brainstem targets (Posner & Peterson, 1990; Sturm & Willmes, 2001). The cortical sustained attention network monitors and modulates firing rates in subcortical arousal structures and hence calibrates the state of alertness according to current goals and task demands (Foucher, Otzenberger, & Gounot, 2004; Kinomura, Larsson, Gulyas, & Roland, 1996). The prefrontal cortices appear to be particularly important in exercising this top–down control as evidenced by the prevalence of sustained attention difficulties in patients with frontal dysfunction (e.g. Wilkins, Shallice, & McCarthy, 1987). Furthermore, recent imaging evidence shows that brief lapses of attention are preceded by momentary reductions of activity in the anterior cingulate and right prefrontal cortex (Weissman, Roberts, Visscher, & Woldorff, 2006).

One of the most elementary examples of cognitive rehabilitation of sustained attention deficits was reported by Manly, Hawkins, Evans, Woldt, and Robertson (2002) who found that the performance of traumatically brain injured participants on an executive control task was markedly improved with the introduction of brief auditory alerts. The alerts bore no relevance to the task other than to cue participants to be more aware of what they were doing (i.e. the cues were non-contingent). Manly and colleagues instructed their participants to use each alert as a cue that would remind them of their current task goal. A similar technique has been shown to have beneficial effects on a test of sustained attention for neurologically healthy participants (Manly et al., 2004) and children with ADHD (O'Connell, Bellgrove, Dockree, & Robertson, 2006). Lesion studies have found that the ability to increase response readiness following an external cue, known as phasic alertness, is not affected by right hemisphere damage (Sturm & Willmes, 2001). The alerts presumably have their effect by briefly activating the frontal control network via ascending thalamic-mesencephalic projections and re-orienting attention to the task at hand. Hence, bottom–up influences on the sustained attention network can be exploited to compensate for reduced top–down control. An important question that follows from these studies is whether or not it is also possible to target top–down control processes directly and hence achieve lasting effects.

Top–down influences on arousal have been frequently explored within the field of biofeedback. During biofeedback participants receive real time visual or auditory information conveying the current level of an otherwise covert biomarker and learn to exert volitional control over that particular process (Critchley, Melmed, Featherstone, Mathias, & Dolan, 2002; Critchley, Tang, Glaser, Butterworth, & Dolan, 2005; Lubar, 2003; Nagai, Critchley, Featherstone, Trimble, & Dolan, 2004). Biofeedback relaxation strategies in which participants learn to decrease sympathetic arousal have proven efficacy for clinical conditions associated with chronically high levels of arousal such as anxiety disorders and stress (Lubar, 2003). Few studies, however, have explored the potential for this approach to contribute to cognitive rehabilitation.

One arousal biomarker that can be reliably modulated during biofeedback interventions is electrodermal activity (EDA), a measure of covert modulation of autonomic system activity that is linked to emotional and cognitive states. EDA is recorded as changes in electrical conductance due to sympathetic stimulation of eccrine sweat glands in the skin and can be characterised as tonic (Skin Conductance Level, SCL) or transient (Skin Conductance Response, SCR). SCL reflects the basal sympathetic tone while SCRs have been widely used in psychological research as a measure of emotional responsiveness and attentional engagement (Dawson, Schell, & Filion, 2000; Frith & Allen, 1983; Hugdahl, 1995). The autonomic system is subject to descending cortical and subcortical influences on hypothalamic and brainstem mechanisms and there is evidence that volitional modulation of SCRs during biofeedback activates many of the same frontal control regions that have been implicated in top–down sustained attention (Critchley et al., 2002; Nagai et al., 2004). For example, Nagai et al. (2004) imaged participants while they performed two separate EDA-biofeedback relaxation and arousal sessions. The fMRI data pointed to a dissociation of the neural systems controlling tonic and transient changes in arousal. Tonic changes were negatively correlated with activity in a 'default mode' network incorporating the ventromedial PFC and orbitofrontal cortex while modulation of SCRs was associated with increased activation of a distributed network that included the dorsal ACC, lateral prefrontal cortices, the insula, the thalamus and the hypothalamus. The degree of overlap between frontal regions involved in SCR modulation and sustained attention provides a basis for hypothesising that training participants to modulate their own SCRs should lead to improvements in sustained attention. Here we examine a new endogenous cueing technique called Self-Alert Training (SAT) which seeks to capitalise on the known relationships between sustained attention and arousal. Instead of reducing arousal, the goal of SAT is to teach participants to transiently increase their arousal at regular intervals in order to offset the periodic decreases in endogenous control that are a major determinant of momentary lapses of attention.

The behavioural strategies involved in SAT arise from an earlier intervention developed by Robertson and colleagues which was designed to remediate the sustained attention deficits of a group of patients with right-hemisphere lesions arising from stroke (Robertson, Tegner, Tham, Lo, & Nimmo-Smith, 1995). That intervention occurred while patients performed a variety of routine everyday tasks (e.g. reading or sorting). Intermittently, the experimenters re-directed the patients' attention to the task by combining a loud noise (clapping) with an instruction to attend. Thus, as in Manly et al. (2002), the authors used the intact bottom–up alerting pathways to re-orient attention. Patients were then gradually taught to initiate this alerting procedure themselves using a self-generated verbal cue. By the end of training, patients had learned to 'self-alert' without needing to generate verbal cues at all. Thus, patients acquired the ability to endogenously activate the sustained attention system without requiring any external cue. After training, all participants showed clinically significant improvements on the training tasks and on a number of untrained attention demanding tasks.

SAT extends Robertson et al.'s (1995) behavioural training strategy with the addition of a biofeedback arousal protocol. Dur-

ing SAT participants do not perform any particular tasks other than observing and modulating their EDA. The objective of SAT is to gradually acquire the ability to control alertness levels in a task-independent manner that can be potentially applied to a variety of settings. In light of the evidence discussed above, it is hypothesised that participants who apply the SAT strategies should be able to exert greater conscious, top–down control over their sustained attention system. This technique was first validated in the current study with healthy adult participants and then independently tested in a group of adults diagnosed with attention-deficit/hyperactivity disorder (ADHD) to determine its clinical utility.

ADHD is one of the most prevalent developmental disorders and is characterised by core behavioural symptoms of inattention, impulsivity and hyperactivity (APA, 2000). According to the DSM-IV criteria, poor sustained attention is a key behavioural characteristic of ADHD and a number of recent meta-analyses have confirmed that adults with ADHD show reliable deficits on neuropsychological measures that require sustained attention (Epstein, Johnson, Varia, & Conners, 2001; Hervey, Epstein, & Curry, 2004; Schoechlin & Engel, 2005; Woods, Lovejoy, & Ball, 2002). Convergent lines of research have identified decreased activation of predominantly fronto-striatal brain regions and abnormalities in the transmission of neurotransmitters such as dopamine and noradrenaline as the likely neurobiological basis of this disorder and a number of studies have indicated that these frontal abnormalities are predominantly right lateralised (Casey et al., 1997; Semrud-Clikeman et al., 2000; Zametkin et al., 1990). ADHD appears, therefore, to represent a good candidate for remediation by SAT.

The training procedures reported in this paper last between 30 and 40 min and therefore do not represent an extended attempt at cognitive remediation. Our primary goal was to examine the relationship between the top–down control processes governing arousal and sustained attention. Training effects on sustained attention were evaluated using pre- and post-training baseline testing sessions in which participants performed the Sustained Attention to Response Test (SART, Robertson, Manly, Andrade, Baddeley, & Yiend, 1997a). During the SART participants are presented with a predictable series of single digits (1–9) and must withhold on the occurrence of a predictable No-Go target. The undemanding nature of this task encourages the adoption of a routine response set which increases its sensitivity to brief momentary lapses of attention. This task has been shown to activate the right hemispheric sustained attention network (Manly et al., 2003) and has proven to be a sensitive clinical measure, discriminating patients with frontal brain injury (Manly et al., 2003; O'Keeffe, Dockree, & Robertson, 2004; O'Keeffe et al., 2007) and ADHD (Johnson et al., 2007; O'Connell, Bellgrove, Dockree, & Robertson, 2004; Shallice et al., 2002) from their neurologically healthy peers. Performance deficits are typically seen in the form of increased errors of commission (failing to withhold to the No-Go target), increased errors of omission (failing to respond to a Go stimulus) and greater reaction time variability indicative of reduced endogenous control of performance. Here, transient EDA changes in the form of SCRs were measured during both baseline testing sessions as an independent measure of the extent of self-alerting. The following training effects were predicted:

(1) Participants who implement SAT strategies during SART performance would show increased arousal, as measured by SCR magnitude, relative to untrained participants.
(2) Increased volitional control of arousal would lead to a reduction in momentary failures of attention (errors of commission).
(3) Increased activation of the sustained attention system following SAT would also be reflected in on-going performance measures such as changes in reaction time variability and errors of omission.

## 2. Methods

### 2.1. Participants

#### 2.1.1. Non-ADHD group

Participants were recruited by poster advertisement at the university campus and randomly assigned to the SAT or placebo condition. The SAT group contained 11 participants (5 females, 1 left-handed) with a mean age of 22 years (S.D. = 2.7) and the placebo group contained 12 participants (5 females) with a mean age of 24 years (S.D. = 4.2). Exclusion criteria were any known neurological condition, severe head trauma, psychosis, learning disability or reading disability. SAT and placebo groups were matched for sex, handedness, age [$F_{(1,21)} = 1.6, p = 0.2$] and everyday absent mindedness, as measured by the Cognitive Failures Questionnaire (Broadbent, Cooper, Fitzgerald, & Parkes, 1982) [SAT mean = 46.9, S.D. = 14.5; Placebo mean = 36.1, S.D. = 13.3; $F_{(1,21)} = 3.6$, $p = 0.07$].

#### 2.1.2. ADHD group

Eighteen participants with ADHD volunteered for the present study following a telephone call or mail advertisement. All patients had existing diagnoses made by a trained psychiatrist attached to the Eastern Healthboard of Ireland. Nine patients were currently taking psychostimulant medication, four had taken stimulant medication in the past but had stopped and five were stimulant-naive. Before inclusion in the study all participants were screened with a telephone interview addressing personal and family history of ADHD, learning disability, psychiatric, neurological or medical disorders, use of medication and substance abuse. Also, prior to testing all participants completed the Conners' Adult ADHD Rating Scale (CAARS; (Conners, Erhardt, & Sparrow, 2003)) and the Wender Utah Rating Scale (WURS), a retrospective measure of ADHD symptoms in childhood (Ward, Wender, & Reimherr, 1993). The observer versions of both scales were also administered to a close family member or partner. Finally, the Standard Clinical Interview for DSM-IV (SCID) was administered by a trained psychiatrist in order to assess comorbid Axis I disorders (Spitzer, Williams, Gibbon, & First, 1992). Participants were excluded if they reported any previous history of psychosis (or if psychosis was indicated by SCID interview), organic brain disorder, epilepsy, serious head injury or learning disability. Comorbid Axis I disorders in the patient group included lifetime depression ($n = 1$), current depression ($n = 1$), bipolar disorder ($n = 1$), current anxiety disorder ($n = 1$) and substance abuse ($n = 4$, alcohol and cannabis use). The 18 participants were randomly assigned to separate SAT-ADHD and Placebo-ADHD groups that were matched for age, sex, handedness, estimated IQ, Wender Utah Rating Scale (WURS) Self- and other-rated childhood symptom scores and Conners' Adult ADHD Rating Scale (CAARS) Self- and other-rated symptom scores. Means, standard deviations and significance levels for each of these variables are summarised in Table 1.

All participants gave written informed consent and all procedures were approved by the ethical review board of the School of Psychology, Trinity College Dublin in accordance with the 1964 Declaration of Helsinki. All participants reported normal or corrected-to-normal vision.

Table 1
Summary of demographic data of ADHD group participants

| | ADHD group | | | |
|---|---|---|---|---|
| | SAT[a] | Placebo[a] | $F_{(1,16)}$ | $p$ |
| *N* | 9 (2 female) | 9 (1 female) | | |
| Age | 23.6 (6.5) | 23.8 (3.3) | 0.01 | 0.9 |
| eIQ | 106.4 (11.0) | 112.9 (6.9) | 1.9 | 0.2 |
| WURS self | 62 (13.1) | 66.5 (14.1) | 0.5 | 0.5 |
| WURS other | 63.4 (11.3) | 60.0 (19.7) | 0.2 | 0.7 |
| CAARS DSM-IV inattention self | 82.7 (8.8) | 81.2 (8.9) | 0.1 | 0.7 |
| CAARS DSM-IV hyperactivity self | 74.3 (11.7) | 76.3 (10.2) | 0.2 | 0.7 |
| CAARS DSM-IV total self | 82.3 (10.5) | 84.9 (5.6) | 0.4 | 0.5 |
| CAARS DSM-IV inattention other | 71.7 (7.7) | 70.3 (11.2) | 0.1 | 0.8 |
| CAARS DSM-IV hyperactivity other | 67.7 (14.4) | 64.1 (20.8) | 0.2 | 0.7 |
| CAARS DSM-IV total other | 72.1 (9.4) | 72.3 (7.9) | 0.01 | 0.9 |

*T*-scores are reported for each of the Conners Adult ADHD Rating Scale (CAARS) measures.
[a] Values are mean (S.D.).

### 2.2. Pre-training baseline

All participants completed 4 blocks of a modified version of the SART. The stimuli for the SART were presented sequentially from '1' through '9'. For each block, 225 digits were presented, representing 25 runs of the 1–9 sequence. Digit font sizes varied between 100, 120, 140, 160 and 180 in arial text. The five allocated digit sizes subtended 1.39°, 1.66°, 1.92°, 2.18° and 2.45°, respectively, in the vertical plane, at a viewing distance of 152 cm. Digits were presented 0.25° above a central white fixation cross on a grey background. The task specifications were programmed and stimuli were delivered using the Presentation® software package (Version 0.75, http://www.neurobs.com). For each trial, a digit was presented for 150 ms followed by an Inter-Stimulus-Interval (ISI) of 1000 ms. Participants were instructed to respond with a left mouse button press with their right forefinger upon presentation of each digit (Go trials) with the exception of the 25 occasions per block when the digit 3 (No-Go target) appeared, where they were required to withhold their response. Participants were instructed to time their button presses to the offset of each stimulus. This kind of 'response-locking' has been shown to reduce inter-individual variability and eliminate speed accuracy trade-offs (Manly, Davison, Heutink, Galloway, & Robertson, 2000; Stuss, Murphy, Binns, & Alexander, 2003). Participants were asked to press the left mouse button to each number except for 3. The task included 200 Go stimuli and 25 No-Go stimuli.

Ten of the Go stimuli were coloured grey, all other stimuli, including the No-Gos were coloured white. Whenever a grey Go stimulus appeared participants were instructed to press the left mouse button as for all Go stimuli, and to say the word 'grey' to indicate to the experimenter that they had noticed the colour change. To avoid confusion participants were told that there were no grey 3s and grey digits occurred only on the digits 5,6,7,8 or 9 to avoid interference with the task of withholding on the No-Go target. In the present study, Grey stimuli were introduced as a cue for participants to implement the Self-Alert Training (SAT) during the post-testing phase. Asking participants to say "grey" when a grey digit appeared provided a way of verifying awareness of these stimuli and also as a means of controlling for the effect of vocalisation on EDA in the post-test condition (i.e. it made it possible to isolate changes in arousal that were specifically due to self-alerting). To reduce the extent to which grey numbers interrupted ongoing responding participants were told that the experimenters were not interested in how quickly they could say "grey" after seeing a grey digit but rather that they were just seeking an indication that participants had noticed the change in colour. Thus, grey stimuli did not interrupt the prepotent Go response set limiting any additional dual-task demand. Timing of task stimuli and the basic response requirements are demonstrated in Fig. 1.

During SART testing participants were seated in front of a Dell Latitude laptop at a distance of approximately 60 cm from the screen. The experimenter was seated at a separate table behind the participant and recorded the number of times participants correctly identified grey stimuli.

SAT or placebo training commenced immediately on completion of baseline testing.

### 2.3. SAT protocol

During SAT participants were taught to gain volitional control of their EDA trace by following three main steps:

Step 1: Eliciting SCRs by external alerting

Participants were allowed to view the EDA reading on-line and the meaning of this measurement was briefly explained. The participant was presented with a loud alerting sound (experimenter clapping and calling "*wake up*!") in order to demonstrate the responsiveness of SCRs to changes in arousal. The participant was shown their SCR to this external alert in real time (see Fig. 2). Since there tend to be large individual differences in the magnitude of SCRs, the scale of measurement in BIOPAC was adjusted for each individual to ensure that arousal responses were clearly visible, as is common practice in other biofeedback protocols (e.g. Critchley et al., 2002). The experimenter asked the participant to try to make a link between what they felt inside and the increases they saw in the red line. This step was repeated 5 times, and each time the participant was able to view increases in the EDA waveform online. A resting period of at least 20 s was provided following each alert to allow the waveform to return to a resting baseline. Participants were also instructed to relax as much as possible in between each cue in order to reduce the number of non-specific SCRs and hence ensure that increases in arousal were more clearly observable in the EDA waveform.

Step 2: Cued internally generated SCRs

In this second stage, the loud alerting cue was removed and the aim was for the participant to begin producing internally driven increases in response to a verbal cue from the experiment (the word 'now' spoken

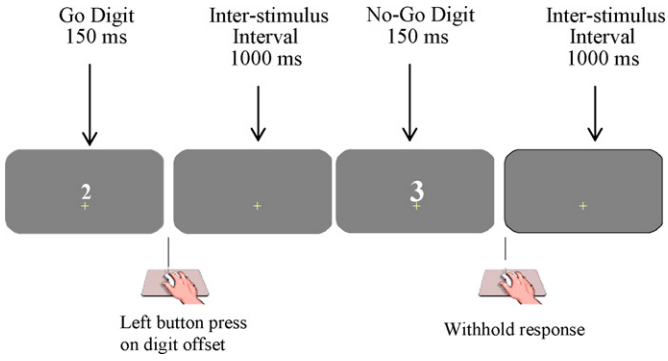


Fig. 1. SART task schematic. Demonstrates the sequence of events contained within a Go trial (the digit 2) and a No-Go trial (the digit 3).

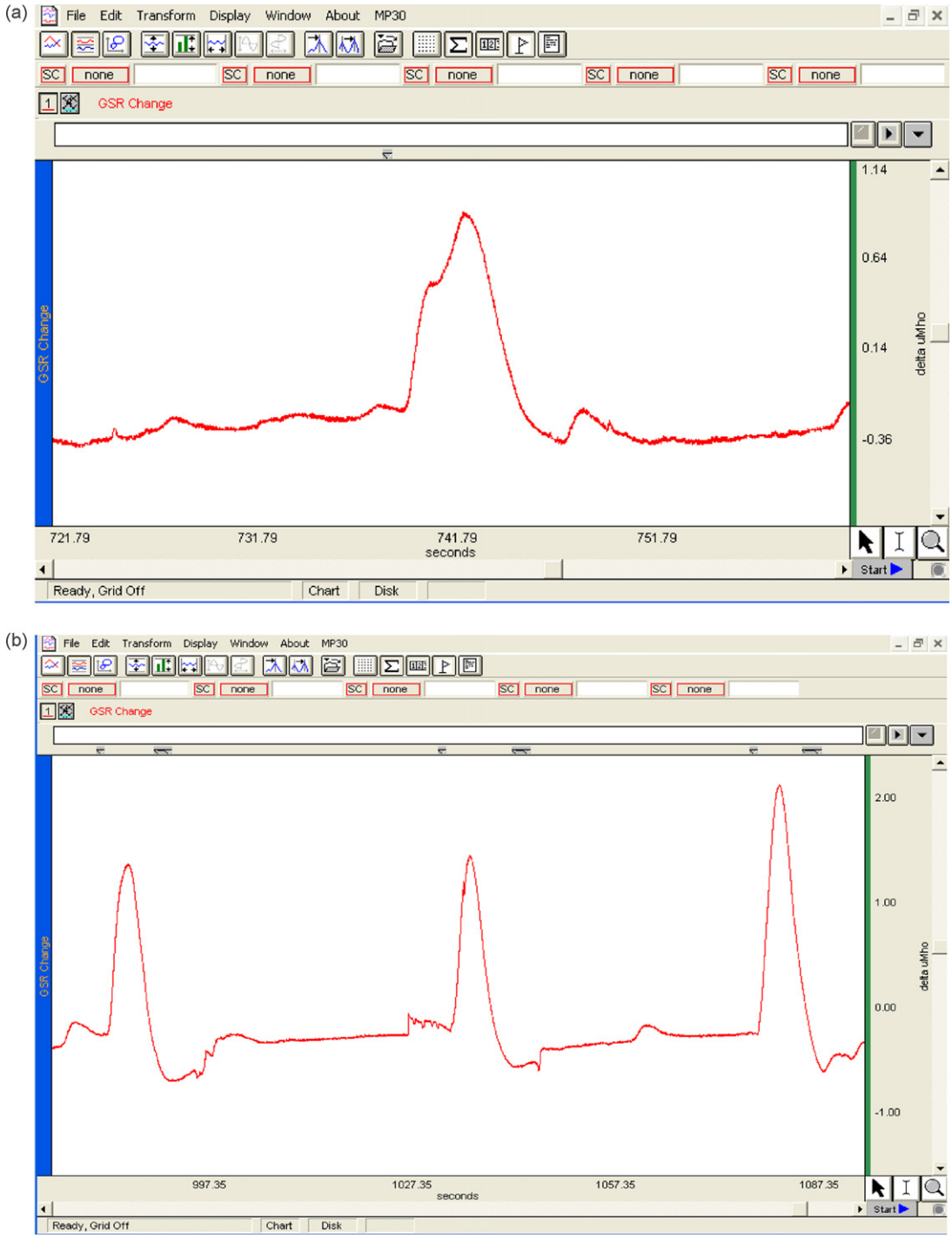


Fig. 2. (A) An example from a single subject of a Skin Conductance Response (SCR) to an external alert. The alert was provided by the experimenter during SAT and its occurrence is marked online (small triangle above the SCR reading). The participant is able to view the subsequent arousal response in real time. (B) An example from a single subject of three 'self-alert' SCRs generated by a participant during SAT. This EDA trace covers a 90 s period. The participant has learned to produce substantial increases in arousal without any prompting from the experimenter.

at a normal volume). The participant was asked to try to recreate the sudden increase in alertness they felt the first time that the experimenter clapped.

The participant was instructed to keep trying to make the red line go as high as possible for about 10–20 s after each cue. A gap of at least 20 s was allowed between cues. This step was repeated until the participant could generate at least 5 clear increases in amplitude. In between each attempt, the participant was instructed to relax in order to reduce the number of non-specific SCRs and thus ensure that an increase in arousal would be readily observable.

Step 3: Un-cued internal generation of SCR amplitude change

In the final step of SAT, the participant learned to take complete control of their EDA trace without any prompting from the experimenter. The participant was asked to say the word 'now' when they were initiating a self-alert to allow the experimenter to mark the EDA trace at the appropriate time. The experimenter marked the EDA trace

when the participant indicated that they were self-alerting. This step was repeated, with visual feedback, until the participant could generate at least 5 increases in amplitude. The participant was instructed to leave at least 20 s after each attempt to allow the EDA trace to return to baseline and ensure that increases in arousal were readily observable. An example of successful self-alerting during training is provided in Fig. 2.

This procedure was then repeated but this time visual feedback was withdrawn and the participant was not able to view the EDA trace. The participant was asked to say 'now' when they were self-alerting and the experimenter marked the trace. This final step was repeated until the participant could generate at least 5 increases in amplitude.

Typical duration of training was 30–40 min.

### 2.4. Placebo training

The aim of the placebo training procedure was to control for the key non-specific elements of SAT, including interaction with the experimenter, positive feedback and the placebo effect. Video game practice has been commonly used as a placebo condition in studies of cognitive rehabilitation. In the present study participants were trained on the video game 'Tetris'.

As in SAT, the experimenter began the placebo condition by allowing the participant to view the EDA reading on-line and explaining the basic premise of the measure. Participants were then presented with the computer game Tetris. In order to enhance the credibility of the sham training the experimenter explained to the participant that previous research has indicated that playing certain computer games can actually increase one's ability to concentrate over time. The basic premise of the computer game training was that the participant must attempt to establish a top speed on Tetris, defined as the highest speed at which the participant was able to last for 3 min without a "game over". The speed of the Tetris game remained constant until it was reset by the experimenter. All participants started at level 7 and progressed up one level each time they managed to last for 3 min. Time was recorded on a stopwatch and the experimenter called out each passing minute so that participants could keep track of their progress. After each step participants were given positive feedback by the experimenter. If a participant failed repeatedly to last 3 min at a given level the experimenter set the short-term goal of beating their previous time. Participants continued to practice Tetris for 30 min.

### 2.5. Post-training baseline

On completion of training participants took a 5–10 min break before beginning the post-training baseline testing. As at pre-testing, participants performed four blocks of the SART and the experimenter again recorded the number of times that participants correctly identified grey stimuli. The SAT and placebo groups were given slightly different instructions. SAT participants were asked to use the self-alerting technique during SART performance to enhance their levels of alertness. Participants were instructed to use the grey coloured digits as a cue to 'self-alert' and to continue to say "grey" each time. The experimenter emphasised that the participant should use each grey digit as a cue for self-alerting. Participants in the Placebo condition were instructed to use each grey digit as a cue to remind themselves what they were doing and to concentrate harder on the task at hand. Again participants were asked to say the word "grey" each time. These instructions were repeated before each SART block to ensure compliance.

### 2.6. EDA acquisition and analysis

EDA measurements were taken from all participants during pre- and post-testing with a 5 channel BIOPAC MP30B unit, calibrated to skin conductance responses (SCRs) in microsiemens (μS). Two Ag/AgCl BIOPAC electrodes, with contact areas of approximately 6 mm, were filled with SIGNA electrode gel and secured with a velcro strap to the volar surface of the distal phalanges of the index and middle fingers of the participant's non-dominant hand. EDA data was analysed using Matlab 6.1 according to previously established criteria (Dawson et al., 2000). A rise in skin conductance level (SCL) was considered to be a response (SCR) if its onset was between 0.5 and 4.5 s after a particular event (presentation of No-Go stimulus or alert). The SCR for a given accepted trial was measured by taking the maximum positive value within the interval 0.5–4.5 s and subtracting the nearest preceding local minimum within that interval. Hence the amplitude of SCRs was calculated as a peak-to-peak measure. The criterion for the smallest acceptable SCR was set at 0.02 μS. The average SCR amplitude on each SART block was calculated for all participants.

### 2.7. Statisical analyses

Variables analysed included errors of commission, errors of omission, reaction time variability and SCR amplitude. Variability of reaction time for correct Go responses (GoRT) was calculated as the average standard deviation of GoRT per block per participant. Separate averages were calculated for each participant for the four SART blocks pre-training and the four blocks post-training. Each variable was entered into a repeated-measures ANOVA with two levels of Group (SAT vs. Placebo) and two levels of Phase (pre-training vs. post-training). SAT and placebo groups within each of the non-ADHD and ADHD groups were compared directly. The ADHD and non-ADHD groups were recruited as part of independent validations of the SAT technique. Consequently, the groups were not matched for key demographic variables and direct statistical comparison of the two groups was not conducted.

## 3. Results

The two SAT and placebo group pairings were successfully matched for baseline levels of commission errors, errors of omission, GoRT variability and SCRs to the cues. Table 2 summarises the means, standard deviations and significance levels for each of these variables.

Average cue detection was very high for the non-ADHD groups pre- [treatment group mean = 98%, S.D. = 1.6, placebo group mean = 98%, S.D. = 1.3, $F_{(1,21)} = 0.05$, $p = 0.8$] and post-training [treatment group mean = 99%, S.D. = 0.8, placebo group mean = 98%, S.D. = 0.7, $F_{(1,21)} = 0.2$, $p = 0.7$]. Similarly, ADHD participants had little difficulty detecting the grey cues and detection rates were close to perfect both pre- [SAT-ADHD mean = 98%, S.D. = 0.9, Placebo-ADHD mean = 99%, S.D. = 0.8, $F_{(1,16)} = 0.3$, $p = 0.6$] and post-training [SAT-

Table 2
Comparison of pre-training baseline behavioural and EDA data for non-ADHD and ADHD groups

| | Non-ADHD group | | | | ADHD group | | | |
|---|---|---|---|---|---|---|---|---|
| | SAT[a] | Placebo[a] | $F_{(1,21)}$ | $p$ | SAT[a] | Placebo[a] | $F_{(1,16)}$ | $p$ |
| $N$ | 11 | 12 | | | 9 | 9 | | |
| Mean commission errors | 3.8 (3.3) | 3.1 (0.2) | 0.46 | 0.5 | 6.2 (1.9) | 6.3 (3.2) | 0.01 | 0.9 |
| Mean errors of omission | 0.4 (0.6) | 0.9 (0.9) | 2.3 | 0.14 | 6.1 (5.4) | 4.4 (3.5) | 0.7 | 0.4 |
| Mean RT variability | 82.9 (28.3) | 90.4 (46.8) | 0.21 | 0.65 | 170.3 (45.5) | 180.1 (64.9) | 0.1 | 0.7 |
| Grey SCRs | 0.503 (0.30) | 0.606 (0.36) | 0.4 | 0.52 | 0.169 (0.10) | 0.261 (0.24) | 1.2 | 0.3 |

[a] Values are mean (S.D.).

Table 3
Effect of SAT on the SART performance and autonomic arousal of participants in the non-ADHD group

| | SAT | | Placebo | | $F_{(1,21)}$ | $p$ |
|---|---|---|---|---|---|---|
| | Pre[a] | Post[a] | Pre[a] | Post[a] | | |
| Mean commission errors | 3.8 (3.3) | 2.5 (2.3) | 3.1 (0.2) | 2.94 (2.6) | 7.3 | 0.03* |
| Mean errors of omission | 0.4 (0.6) | 0.65 (0.97) | 0.9 (0.9) | 1.9 (2.4) | 2.4 | 0.1 |
| Mean RT variability | 82.9 (28.3) | 81.2 (47.4) | 90.4 (46.8) | 138.8 (88.3) | 4.1 | 0.05* |
| Grey SCRs | 0.503 (0.30) | 0.64 (0.38) | 0.606 (0.36) | 0.376 (0.15) | 8.4 | 0.011* |

*$p < 0.05$.
[a] Values are mean (S.D.).

Table 4
Effect of SAT on the SART performance and autonomic arousal of participants in the ADHD group

| | SAT | | Placebo | | $F_{(1,16)}$ | $p$ |
|---|---|---|---|---|---|---|
| | Pre[a] | Post[a] | Pre[a] | Post[a] | | |
| Mean commission errors | 6.2 (1.9) | 4.19 (2.2) | 6.3 (3.2) | 6.7 (4.3) | 4.85 | 0.04* |
| Mean errors of omission | 6.1 (5.4) | 4.5 (3.5) | 4.4 (3.5) | 3.8 (2.8) | 0.51 | 0.48 |
| Mean RT variability | 170.3 (45.5) | 159.3 (44.0) | 180.1 (64.9) | 202.9 (91.7) | 5.5 | 0.03* |
| Grey SCRs | 0.169 (0.10) | 0.230 (0.18) | 0.261 (0.24) | 0.142 (0.13) | 8.9 | 0.009** |

*$p < 0.05$, **$p < 0.01$.
[a] Values are mean (S.D.).

ADHD mean = 99%, S.D. = 0.7, Placebo-ADHD mean = 99%, S.D. = 0.7, $F_{(1,16)} = 0.1$, $p = 0.7$].

By the end of SAT all participants were able to generate 5 SCRs without any external prompting or visual feedback.

### 3.1. Effect of SAT on sustained attention

Behavioural data for the pre- and post-training baselines are summarised separately for the non-ADHD and ADHD groups in Tables 3 and 4.

#### 3.1.1. Errors of commission

Training effects on commission errors for non-ADHD and ADHD groups are illustrated in Fig. 3.

*3.1.1.1. Non-ADHD group.* A repeated-measures ANOVA revealed a significant main effect of Phase (pre-training vs. post-training) [$F_{(1,21)} = 7.3$, $p < 0.05$] and a phase by group interaction [$F_{(1,21)} = 5.0$, $p < 0.05$]. There was no main effect of group [$F_{(1,21)} = 0.03$, $p = 0.87$]. Post hoc tests with Bonferroni corrections revealed a significant effect of phase on the treatment group [$p < 0.01$] that was absent in the control group [$p = 0.9$]. On average, participants who received SAT made 35% fewer errors of commission at the post-training baseline versus a 4% decrease for participants who received placebo training. Ten of the SAT participants showed a reduction in errors of commission and one participant showed no change. Seven participants in the Placebo group also showed a reduction in errors of commission, one participant showed no change while four participants made more errors after training.

*3.1.1.2. ADHD group.* A repeated-measures ANOVA indicated a significant phase (pre-training vs. post-training) by group interaction [$F_{(1,16)} = 4.85$, $p < 0.05$] but there was no main effect

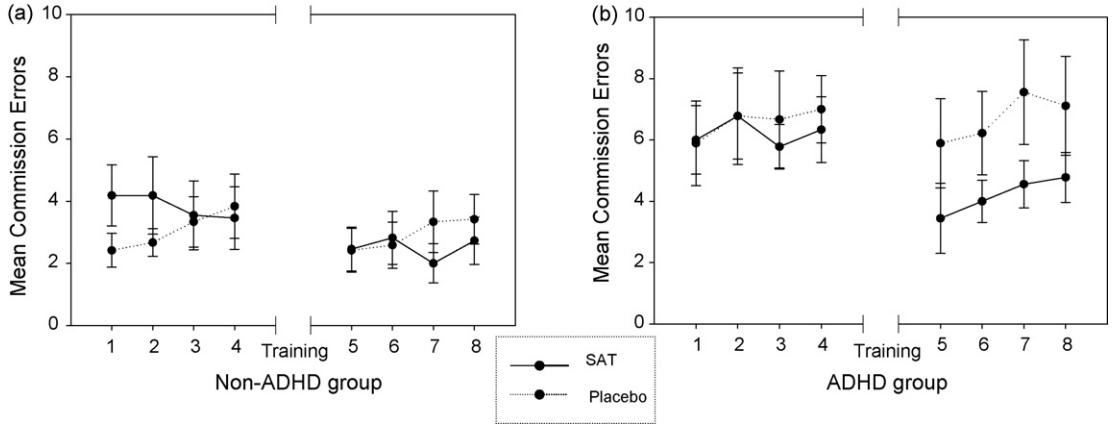


Fig. 3. Effect of SAT on mean errors of commission during SART performance. (a and b) Both SAT groups exhibited a significant decrease in errors of commission post-training. No significant change was seen in either of the placebo groups.

of phase [$F_{(1,16)} = 2.3$, $p = 0.16$] or of group [$F_{(1,16)} = 0.95$, $p = 0.3$]. Post hoc pairwise comparisons with Bonferroni corrections revealed a significant decrease in commission errors post-training in the SAT-ADHD group [$p < 0.05$] that was absent in the Placebo-ADHD group [$p = 0.6$]. SAT participants made 32% fewer errors post-training while participants in the placebo condition made an average of 6% more errors post-training. Six participants in the SAT-ADHD group showed an improvement in performance, one participant showed no change and two participants made more errors post-training. In contrast only one participant showed improved performance following Placebo training, two showed no change, and six made more errors post-training.

### 3.1.2. Errors of omission

*3.1.2.1. Non-ADHD group.* Errors of omission were rare amongst the non-ADHD participants occurring on 1.3% of Go trials. A repeated-measures ANOVA revealed a main effect of phase on errors of omission [$F_{(1,21)} = 5.9$, $p < 0.05$] but the phase by group interaction did not reach significance [$F_{(1,21)} = 2.4$, $p = 0.14$] and there was no main effect of group [$F_{(1,21)} = 2.7$, $p = 0.2$]. Thus, there was a general increase in errors of omission over time that was not significantly altered by SAT.

*3.1.2.2. ADHD group.* Although more frequent than in the non-ADHD group, errors of omission were rare amongst participants with ADHD occurring on just 2.6% of Go trials. A repeated-measures ANOVA indicated no significant main effects of phase [$F_{(1,16)} = 2.7$, $p = 0.12$] or group [$F_{(1,16)} = 0.5$, $p = 0.5$] and no phase by group interaction [$F_{(1,16)} = 0.5$, $p = 0.5$]. Thus SAT did not alter task performance as measured by errors of omission.

### 3.1.3. Reaction time variability

Training effects on variability in reaction time for non-ADHD and ADHD groups are illustrated in Fig. 4.

*3.1.3.1. Non-ADHD group.* The mean standard deviation of reaction time (GoRT variability) was calculated separately for each group pre- and post-training. A repeated-measures ANOVA revealed no main effect of phase [$F_{(1,21)} = 3.6$, $p = 0.07$] or group [$F_{(1,21)} = 1.7$, $p = 0.2$]. A significant phase by group interaction was found [$F_{(1,21)} = 4.1$, $p < 0.05$] and post hoc Bonferroni tests revealed that this effect was driven by significant increases in GoRT variability in the placebo group over time [$p < 0.01$] that were not evident in the treatment group [$p = 0.9$]. As can be seen in Fig. 4a these data suggest that while the participants in the Placebo group became more variable in their responding on the SART over time, SAT participants were able to maintain a more consistent level of performance before and after training.

*3.1.3.2. ADHD group.* For the ADHD group SAT had a strikingly similar effect on GoRT variability as that observed in the non-ADHD group. A repeated-measures ANOVA indicated that there was no main effect of phase [$F_{(1,16)} = 0.7$, $p = 0.4$] or group [$F_{(1,16)} = 0.8$, $p = 0.4$] but there was a significant phase by group interaction [$F_{(1,16)} = 5.5$, $p < 0.05$]. Post hoc Bonferroni *t*-tests revealed that this interaction was driven by a significant increase in GoRT variability post-training in the Placebo-ADHD group [$p < 0.05$] which was absent in the SAT-ADHD group [$p = 0.3$]. As in the non-ADHD, the participants who received Placebo training became more variable over time while participants who received SAT training were able to maintain a more consistent level of performance.

### 3.2. Effect of SAT on arousal levels

As a measure of self-alerting, SCRs following each cue were measured and averaged separately for the pre- and post-training baselines. SCR data are summarised in Tables 3 and 4. Training effects on autonomic arousal for non-ADHD and ADHD groups are illustrated in Fig. 5.

### 3.2.1. Non-ADHD group

A repeated-measures ANOVA revealed no main effect of phase on SCR magnitude [$F_{(1,21)} = 0.6$, $p = 0.5$] and no main effect of group [$F_{(1,21)} = 0.3$, $p = 0.6$]. A significant phase by group interaction was found [$F_{(1,21)} = 8.4$, $p < 0.01$] and post hoc Bonferroni *t*-tests indicated that this effect was driven by a drop in SCR magnitude over time in the placebo group [$p < 0.05$] and a marginally significant increase in the SAT group [$p = 0.07$].

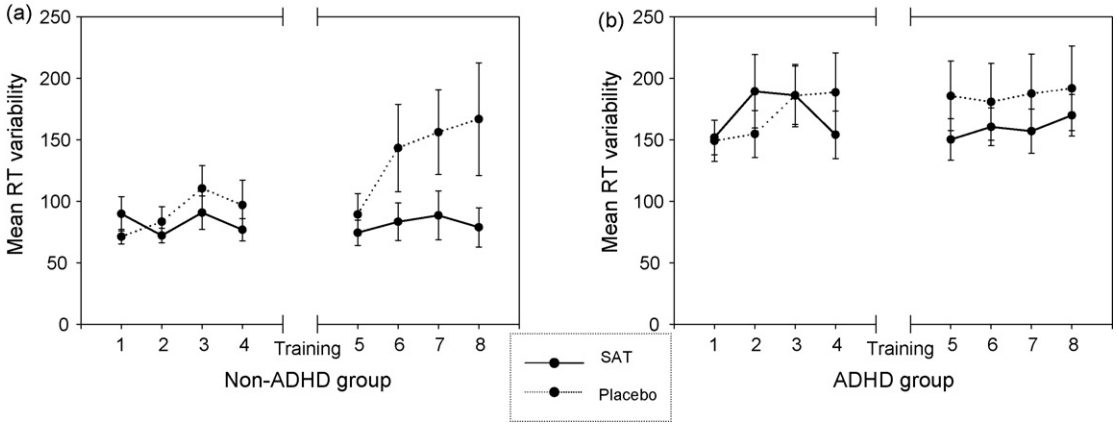


Fig. 4. Effect of SAT on mean reaction time variability for each SART block pre- and post-training. (a and b) Both placebo groups exhibited a clear increase in variability over time while the SAT groups maintained a more consistent performance after training.

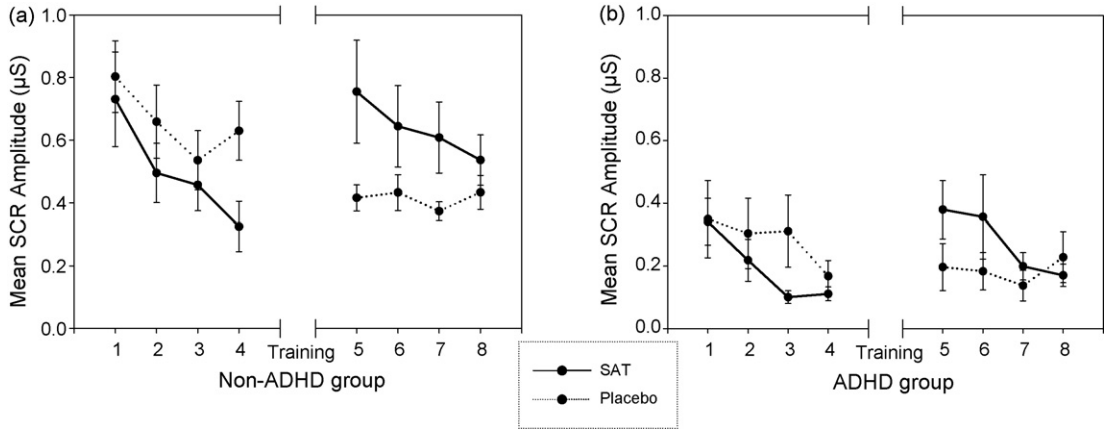


Fig. 5. Mean Skin Conductance Response amplitude (measured in μS) elicited by cues embedded in the SART pre- and post-training. (a and b) The SAT groups showed increased arousal responses after training while arousal in the placebo group gradually decreased over time.

These findings suggest that participants in the placebo condition experienced an overall drop in arousal responses to cues over time whereas participants who received SAT increased their arousal responses following cues post-training. These data therefore confirm that SAT participants did engage in self-alerting during post-training blocks of the SART.

### 3.2.2. ADHD group

No main effect of phase [$F_{(1,16)} = 0.9$, $p = 0.4$] or group [$F_{(1,16)} = 0.001$, $p = 0.9$] was found but there was a significant phase by group interaction [$F_{(1,16)} = 8.9, p < 0.01$]. Post hoc Bonferroni *t*-tests indicated that this effect was driven by a drop in SCR magnitude over time in the placebo group [$p < 0.05$] that was not found in the SAT-ADHD group [$p = 0.16$]. This result suggests that SAT had the effect of preventing a decrement in arousal over time as opposed to actually increasing arousal beyond previous levels. However, closer inspection of the changes in SCRs over time illustrated in Fig. 5b suggests that participants who had received SAT had increased arousal for the first two blocks post-training but that this effect dissipated by blocks 3 and 4. A further repeated-measures ANOVA was conducted comparing post-cue SCRs for the four blocks of baseline testing to the first two blocks post-training. Again, there was no main effect of phase [$F_{(1,16)} = 1.8, p = 0.2$] or group [$F_{(1,16)} = 0.2, p = 0.7$] but there was a significant phase by group interaction [$F_{(1,16)} = 8.4, p < 0.01$]. This time however, post hoc Bonferroni *t*-tests indicated that participants who received SAT experienced a significant increase in arousal during the first two blocks after training [$p < 0.01$], while there was no such change amongst participants in the placebo condition [$p = 0.3$].

## 4. Discussion

These data indicate that, as predicted, increased volitional modulation of autonomic arousal following Self-Alert Training (SAT) produced improvements in behavioural and physiological indices of sustained attention. The analysis of EDA data confirms that participants were able to implement the SAT alerting technique while they performed an untrained neuropsychological task. The absence of any performance gains in the placebo condition confirms that these improvements are unlikely to arise from non-specific effects. In addition to benefiting neurologically healthy adults, we have also demonstrated that SAT is effective in addressing sustained attention deficits in adults with ADHD. During training, participants with ADHD showed no difficulty in modulating their EDA responses and were able to fulfil the criteria of producing five un-cued SCRs within the 30-min training period. After training, participants in the ADHD group successfully modulated their own arousal levels during the SART and made comparable performance gains to those achieved by non-ADHD participants. The training did not involve practicing a neuropsychological task and, as a result, these behavioural effects represent a generalisation of training effects to an untrained neuropsychological task.

Both participant groups that received SAT showed clear improvements on the key behavioural indices of sustained attention. A 35% decrease in commission errors in the non-ADHD sample represents a substantial difference considering that these improvements were made from an already high baseline level of task performance. Similarly, participants in the ADHD group substantially improved their performance following SAT making 32% fewer errors of commission and maintaining a consistent level of RT variability. In contrast, participants in the sham-training placebo condition showed no significant change in commission error rates and exhibited a gradual increase in GoRT variability over time.

As illustrated in Figs. 4 and 5, for participants in the placebo condition, GoRT variability and arousal levels (as measured by SCRs) appeared to follow a similar time-on-task trend with variability gradually increasing over the 8 blocks and arousal levels gradually decreasing. Previous work using vigilance paradigms has demonstrated that when a task is monotonous and unstimulating accuracy rates tend to decline with time due to diminishing arousal (e.g. Parasuraman, Nestor, & Greenwood, 1989; Paus et al., 1997). Previous work has linked increasing reaction time variability to decreased efficiency of frontal control mechanisms (Bellgrove, Hester, & Garavan, 2004; Stuss et al., 2003) and a recent study by Johnson et al. (2007) has suggested that increased GoRT variability on the SART is attributable, at least in part, to a progressive slowing of reaction times. This work

indicates that subtle time-on-task effects for RT can become apparent over far shorter time periods than may be apparent when using accuracy measures alone. Thus, whereas errors of commission are primarily sensitive to brief reductions in the top–down control of sustained attention, the continuous response control that is indexed by trial-by-trial measures of GoRT variability may be more sensitive to any changes in arousal. In the present study, the fact that block-by-block increases in GoRT variability were accompanied by gradual decreases in arousal, as measured by SCR amplitude, strongly suggests that the placebo group experienced a vigilance decrement. In contrast, neither of the SAT groups showed a decline in arousal nor an increase in variability indicating that implementing the training strategies allowed participants to offset a vigilance decrement as well as increasing the engagement of endogenous control processes as indicated by reduced error rates. Given the link between RT variability and prefrontal functioning, these effects are consistent with our initial hypothesis that the volitional modulation of arousal following SAT may preferentially target frontal regions.

The comparison of SCR amplitudes before and after training indicates that both SAT groups did successfully implement the training strategy during SART performance. Non-ADHD participants who received SAT showed an increase in arousal post-training while participants in the placebo condition experienced a gradual drop in arousal. A similar interaction of Group and Phase was observed in the ADHD group however this effect appears to have been driven primarily by decreased arousal in the placebo group since arousal levels did not change significantly in the SAT group. A more sensitive block-by-block analysis revealed that the ADHD SAT group did show clear signs of self-alerting during the first two blocks of SART post-training but that these effects dissipated by the third and fourth blocks. This drop-off may be a reflection of a decreased capacity for volitional modulation of arousal in ADHD. The fact that the adult ADHD group successfully completed training and were able to increase their arousal during the first two blocks of post-testing, suggests that increasing the length and intensity of the training session might be necessary to achieve lasting effects. It is also worth noting that the ADHD group in this study was not medicated during testing. It may be that the focus required to facilitate cognitive remediation is better achieved when the putative right frontal dysfunction that is thought to underpin sustained attention deficits in ADHD, is controlled pharmacologically (see Loo et al., 2003).

In summary, our results are consistent with the initial hypothesis that increased volitional control of arousal would lead to improvements in sustained attention. While past rehabilitative efforts (Manly et al., 2004; O'Connell et al., 2006) have targeted the sustained attention network via its bottom–up influences, SAT targeted sustained attention via its top–down influences. SAT may be particularly beneficial for adults suffering from ADHD since this disorder produces relatively subtle neuropsychological abnormalities that do not preclude direct training within the affected domains. This experiment has demonstrated that a relatively simple cognitive intervention can lead to substantial neuropsychological improvements. The possibility that extended SAT and implementation of training strategies in everyday life would lead to lasting improvement in frontally mediated cognitive function is an interesting possibility worthy of further investigation.

We emphasise that the SAT protocol used in the present study was a brief version designed to investigate, in a proof-of-concept fashion, whether repeated volitional modulation of autonomic arousal would trigger increased top–down control of sustained attention, potentially mediated by fronto-parietal networks. The training procedure lasted between 30 and 40 min in each case and therefore did not represent an extended attempt at cognitive remediation. Nevertheless, it was found that by the end of training all participants were able to phasically modulate their arousal levels in an endogenous manner without any visual feedback and without any external prompting from the experimenter. A limitation of the design used in this study is that participants were cued to self-alert during SART performance. The inclusion of cues made it possible to measure the magnitude of arousal responses during self-alerting hence providing a valuable objective marker of training efficacy. However, a key benefit of self-alerting is that it can, in theory, be performed in the absence of any external cue and could therefore be applied in a wide range of real world settings. Future work should investigate whether similar improvements in performance can be achieved when participants are not cued to self-alert. Further work is also required to establish whether or not these gains can be transferred to real world settings. From a rehabilitation perspective, the use of alerts that are independent of task or participant characteristics provides a highly flexible means of triggering controlled behaviour that is potentially applicable to a range of real-world settings (see Fish et al., 2007; Levine et al., 2000).

These data show that the SAT protocol was successful in training healthy adult participants to gain control over their own arousal levels with a consequent improvement in performance on an untrained sustained attention task. This work has demonstrated that an improved understanding of the neural networks governing sustained attention can inform the development of new and effective remedial strategies.

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