

# Investigation of visual dream reports after transcranial direct current stimulation (tDCS) during REM sleep

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**Summary.** Neuroimaging studies have revealed regional patterns of brain activation/deactivation during REM sleep, with the posterior parietal and frontal cortex implicated as key brain regions involved in dreaming. Using a novel brain stimulation technique (tDCS), this study addressed the ongoing debate over the neuroanatomical origins of dreaming. This study examined the effect of cathodal and anodal tDCS applied simultaneously to the right posterior parietal and frontal cortex (respectively) during REM sleep on dream recall reported on awakening. It was hypothesized that such stimulation would have an inhibitory effect on local posterior parietal cortical circuitry and an excitatory effect on local frontal cortical circuitry, and that such a combination would impair dream recall on awakening from REM sleep. Three conditions (tDCS, low tDCS, blank control) were administered in a counterbalanced order in two separate studies, one employing an interval design whereby tDCS was delivered for a fixed duration, the other adopting a threshold approach with tDCS delivered in an increasing fashion until reaching the participants' arousal threshold. TDCS applied during REM sleep did not result in a significant decrease in the percentage of imagery reports, number of visualisable nouns, or imagery ratings. In sum, it appears that tDCS had no effect on reported dream imagery. However, further research would need to be carried out to determine whether such results reflect the unique properties of this stage of sleep or methodological limitations.

**Keywords:** Dreaming, transcranial direct current stimulation (tDCS), REM sleep

## 1. Introduction

In recent years neuroimaging technology has revealed regional activation during REM sleep that has been attributed to processes underlying subsequent dream reporting (Hobson, Pace-Schott, & Stickgold, 2000). Specifically, studies indicate activation of limbic and paralimbic regions of the forebrain (amygdalae, anterior cingulate cortex) and deactivation of much of the dorsolateral prefrontal and orbitofrontal cortex during REM sleep (Hobson et al., 2000). Furthermore, the results of a meta-analysis of neuroimaging studies conducted during REM and NREM sleep (Jakobson, Laird, Maller, Conduit, & Fitzgerald, 2012, Accepted for Publication) revealed that frontal deactivation featured prominently during REM sleep compared to wakefulness. Such frontal deactivation during REM sleep thus explains the loss of directed thought and deficits in other executive processes evident in dream reports (Hobson et al., 2000). In addition, the posterior parietal cortex is another area which exhibits selective activation patterns during

sleep. For instance, studies have revealed significant activation of various regions of the posterior parietal cortex during REM sleep, such as the precuneus (Hong, Gillin, Dow, Wu, & Buchsbaum, 1995), posterior part of the right parietal operculum (Maquet et al., 1996), supramarginal gyrus (Hong et al., 1995; Maquet et al., 1996), angular gyrus (Nofzinger, Mintun, Wiseman, Kupfer, & Moore, 1997), and bilateral superior and right inferior parietal lobules (Hong et al., 1995). Lesion studies reporting damage to various areas of the brain also suggest that the posterior parietal cortex is involved in dreaming. For instance, an extensive review of reported cases consistently found that lesions to posterior neocortical areas including the posterior parietal cortex (parietal operculum, PTO junction), resulted in significantly more reports of global cessation of dreaming compared to other cortical areas (Doricchi & Violani, 1992). Solms (1997) also found that of 112 reported cases of global cessation of dreaming, 47 were localized to the parietal cortex, particularly the posterior parietal cortex (supramarginal gyrus, PTO junction). Thus, a growing body of evidence from neuroimaging and lesion studies indicates that the posterior parietal cortex could be a key area involved in dream reporting.

However, the neuroanatomical origins underlying dream recall is a controversial topic with two schools of thought regarding dream generation. For instance, based primarily on findings from animal studies, the AIM model asserts that the pontine brainstem generates not only REM sleep but also dreaming by triggering other subcortical structures and cortical regions involved in dreaming (Hobson et al.,

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2000). However, although the AIM model acknowledges the important role of the cortex in dreaming, its emphasis remains on the brainstem as the generator or master switch. In contrast, Solms (1997, 2000) citing evidence from lesion and neuroimaging studies in humans, argues that forebrain cortical circuits (particularly frontal and parietal circuits) are the principal neural circuitry in the dream process. Overall, human neuroimaging and lesion data support the notion that forebrain processes are critical for dream recall, but this does not dismiss the extensive animal research suggesting a role of the brainstem in REM sleep processes and dreaming (Hobson & McCarley, 1974a; Hobson, McCarley, Pivik, & Freedman, 1974b; Hobson, McCarley, Freedman, & Pivik, 1974c; McCarley & Hobson, 1975; Pivik, McCarley, & Hobson, 1977). Although neuroimaging and lesion data in humans presents evidence challenging notions of brainstem generation of dreaming, it is unlikely that such an issue can be resolved as the data from the two types of studies (animal versus human) are not directly comparable. For instance, although dream reports can be obtained in humans, current brainstem lesion findings are debatable and recording brainstem function during sleep using neuroimaging is not possible as the technology in its current form does not possess the spatial and temporal specificity to effectively assess rapid brainstem activity conveyed to the cortex (Conduit, 1999).

Therefore, it is unlikely the relative explanatory value of such models will be resolved in the near future. These issues highlight the need to exploit new methods for testing theories of REM sleep and dream recall, and to achieve a more complete understanding of the dream recall process.

One new direction for dream research could be to use recently developed brain stimulation technologies. For example, transcranial direct current stimulation (tDCS), a technique used to non-invasively stimulate the brain through the application of a weak constant electric current, has been used increasingly as a means to alter cortical excitability (Been, Ngo, Miller, & Fitzgerald, 2007). The technique is relatively simple to administer and due to its ability to induce focal changes in cortical excitability, tDCS has been proposed to be able to demonstrate a causal link between the area targeted and the behavior under investigation (Fregni et al., 2005). The duration and direction of the effects depend not only on stimulation duration and intensity, but also on polarity, with studies demonstrating that anodal stimulation increases local cortical excitability (excitatory effect), whereas cathodal stimulation decreases local cortical excitability (inhibitory effect) (Lang, Nitsche, Paulus, Rothwell, & Lemon, 2004; Lang et al., 2005). Previous studies have shown tDCS to be an effective means of altering cortical excitability in studies targeting the motor (Boggio et al., 2006; Lang et al., 2004), somatosensory (Dieckhöfer et al., 2006), prefrontal (Kincses, Antal, Nitsche, Bártfai, & Paulus, 2003), and visual cortex (Antal, Kincses, Nitsche, & Paulus, 2003; Antal, Kincses, Nitsche, Bártfai, & Paulus, 2004). Moreover, tDCS appears to produce changes in a range of cognitive functions such as implicit probabilistic classification learning (Kincses et al., 2003), working memory (Fregni et al., 2005; Marshall, Mölle, Siebner, & Born, 2005), and tactile perception (Rogalewski, Breitenstein, Nitsche, Paulus, & Knecht, 2004).

To date, only three tDCS studies have been conducted investigating cognitive processes during sleep (Marshall, Mölle, Hallschmid, & Born, 2004; Marshall, Kirov, Brade,

Mölle, & Born, 2011; Nitsche et al., 2010). The study by Marshall et al. (2004) utilized the technique to investigate the effects of tDCS during sleep on waking memory performance. In this study, anodal tDCS was applied bilaterally at the fronto-cortical area intermittently for 30 minutes during SWS following both a declarative and procedural memory learning task. Subjects were woken and their memory recall tested. It was found that compared to a control condition, tDCS improved declarative memory performance.

In a recent study investigating the effects of tDCS on dream recall (Jakobson, Fitzgerald, & Conduit, 2012), dream reports were collected following 1.5–2 mA of simultaneous anodal and cathodal tDCS applied to the right posterior parietal and frontal cortex (respectively) during stage 2 sleep. It was found that compared to the control condition, tDCS improved recall of sleep mentation by way of an increase in two of the three measures of visual imagery. This study provided evidence for the ability of tDCS to modulate cortical excitability and reported visual imagery.

Thus, if tDCS can affect cognitive functioning in sleeping participants during stage 2 sleep, then it is not unreasonable to propose that cognitive effects may be observed when such stimulation is directed at brain regions implicated in dreaming during REM sleep. The most suitable cortical targets for tDCS would therefore be the frontal and right posterior parietal cortices due to: a) their accessibility with tDCS (compared to the anterior cingulate gyrus), b) the right hemispheres role in predominantly visuo-spatial processes (Banich & Heller, 1998), c) their implication as key cortical regions in dream reporting as evidenced by neuroimaging and lesion studies (Doricchi & Violani, 1992; Hobson et al., 2000; Jakobson, Laird, Maller, Conduit, & Fitzgerald, 2012, Accepted for Publication; Nofzinger et al., 1997), and d) our previous success manipulating dream recall when applying tDCS to these regions during stage 2 sleep (Jakobson et al., 2012).

The purpose of this study was to investigate the effect of tDCS applied to the frontal and right posterior parietal cortex during REM sleep on dream recall. This paper presents two studies in REM sleep using two different methodologies. The first study employed a method we previously used to deliver tDCS during stage 2 sleep (Jakobson et al., 2012), where tDCS was delivered during REM sleep for a fixed duration and fixed level of stimulation across participants and conditions. The second study adopted a threshold approach to delivering tDCS, whereby each tDCS presentation was delivered in an increasing fashion until reaching the participants individual arousal threshold. For both studies it was hypothesized that concurrent cathodal (negative current) and anodal (positive current) tDCS of the right posterior parietal and frontal cortex (respectively) would have an inhibitory effect on local posterior parietal cortical circuitry and an excitatory effect on local frontal cortical circuitry, and that such a combination would impair dream recall and/or alter dream quality (i.e. vividness) reported on awakening from REM sleep.

## 2. Method

### 2.1. Participants

16 participants (7 males, 9 females) aged from 18 to 38 years ( $M = 26.31$ ,  $SD = 5.90$ ) participated in study 1 and 28 participants (12 males, 16 females) aged from 19 to 38 years

( $M = 25.71$ ,  $SD = 5.46$ ) in study 2. All were recruited through campus posters and advertisements in local newspapers. Individuals with a history of abnormal sleeping patterns (i.e. shift workers), drug or alcohol abuse, learning disabilities or medical conditions were excluded from the study. Each participant provided written informed consent and was compensated for their time. This study was approved by the University Human Ethics Committee.

## 2.2. Materials

Both studies were conducted in a two-bedroom sleep laboratory located at Monash University. Polysomnography (PSG) recordings were collected using an S-Series 16 channel Polygraph with W-Series Sleep/Replay display and analysis software (Compumedics Pty, Ltd. Melbourne, Australia). Gold-plated cup electrodes (Model F- E5GH; Grass Instruments Co., CA), conductive electrode paste (Ten20, Grass Instruments Co.), surgical tape (Micropore, 3M Pharmaceuticals, MN, USA), gauze swabs (7.5 x 7.5 cm, Smith & Nephew Pty Ltd), abrasive tape (Red Dot Preparation Tape, 3M Pharmaceuticals, MN, USA), skin cleansing alcohol swabs (Briemar Nominees Pty Ltd, Australia), and 0.9% sodium chloride saline solution (Baxter, Australia) were used for electrode preparation and placement. A crepe head bandage and tubular-net were used to help keep the electrodes in place.

TDCS was delivered through a pair of conductive rubber electrodes inside saline soaked sponges (4.5cm x 7cm) via a battery-driven constant current DC stimulator (Model: Eldith #0008, Neuroconn GmbH, Germany). The saline soaked sponges were covered with an 8 x 10 cm piece of thin rubber to help restrict sponge leakage and bridging to the recording electrodes.

## 2.3. Procedure

A standard PSG recording montage was adopted (Rechtschaffen & Kales, 1968). EEG placements were to C3/A2 and C4/A1 locations according to the international ten-twenty system (Jasper, 1958). EMG placements were to the left and right mentalis musculature. EOG placements were to the left and right outer canthi and referenced to A1. EEG traces were calibrated at  $50 \mu V = 1 \text{ cm}$  with impedances below  $5 \text{ k}\Omega$ . Sleep stage scoring was manually scored according to the criteria of the AASM (Iber, Ancoli-Israel, Chesson, & Quan, 2007).

TDCS electrodes were attached to the participants scalp with the anode polarity placed at the supraorbital area of the forehead (Fpz), and the cathode polarity placed at the right side of the head (P4). There were three conditions in these studies: tDCS, low intensity tDCS (low tDCS) and a no stimulation (blank) control.

### 2.3.1 Study 1

*tDCS.* – TDCS involved a 181 second period of stimulation at an intensity of 2mA (ramping from 0–2mA over the first 30 seconds). The parameters for this condition were chosen to provide the maximum stimulation possible, without waking the participant (Iber et al., 2007). If a participant was unable to tolerate 2mA and awakened after three consecutive attempts, they were given stimulation at an intensity of 1.5mA.

*Low tDCS.* – This condition involved a 41 second period

of stimulation at an intensity of 1 mA, followed by a 140 second period in which no stimulation was given. Stimulation was applied for a short period to provide the physical sensations of mild 'tingling' or 'itching' of the scalp, similar to the tDCS condition, but with minimal effect on cortical excitability (Boggio et al., 2006; Lang et al., 2005; Rogalewski et al., 2004). In order to avoid the possibility of waking participants, a 30 second fade in period (ramping to 1mA) was administered, consistent with previous tDCS studies (Vines, Schnider, & Schlaug, 2006).

*Blank control.* – In order to account for the possibility that tDCS applied to any region of the cortex might produce measurable changes in dream reporting (i.e. a non-specific effect), a blank control condition involving no tDCS was adopted. In this condition, no tDCS was given, but the experimenter waited for a period of 181 seconds before waking the participant for the collection of dream reports. Thus, this condition served as a baseline measure with which to compare the other two conditions.

### 2.3.2 Study 2

The procedures employed were the same as for the previous study. However, here a different method of delivering the tDCS condition was employed. Specifically, this study adopted a threshold design whereby tDCS was delivered in an increasing fashion until reaching the participants' individual arousal threshold. In addition, the duration and/or intensity of the three conditions differed somewhat from the previous study as outlined below.

*tDCS.* – TDCS involved a 151 second period of stimulation at an intensity of 2 mA (ramping from 0–2 mA over the first 120 seconds).

*Low tDCS.* – This condition involved a 61 second period of stimulation at an intensity of 0.5 mA. A 30 second fade in period was administered consistent with previous studies (Vines et al., 2006).

*Blank control.* – No stimulation was given but the experimenter waited for a period of 61 seconds (consistent with the duration of the low tDCS condition).

Participants were assigned to one of the counterbalanced orders of the conditions. Prior to lights out each participant was given a test stimulation so that they were familiarized with the cutaneous sensations produced. Once participants had entered the first 2 epochs (60 seconds) of continuous REM sleep after 3am (3 epochs [90 seconds] after 2am in study 2), tDCS, low tDCS, or the blank control condition was administered, followed by a 60 second delay period (30 seconds in study 2) to allow for the cutaneous sensations to subside and to confirm REM sleep before waking the participant for dream report collection. In addition, the two stimulation conditions often interfered with the PSG recordings, making one or more channels uninterpretable. Therefore, trials were terminated by the experimenter as soon as the participant showed signs of arousal (EEG arousal or EMG activation). If the participant showed awakening or a stage shift (Iber et al., 2007) the data from that trial was not used.

## 2.4. Dream Report Collection

To wake and collect dream reports from participants, the experimenter used similar procedures to those of previous studies (Fedyszyn & Conduit, 2007; Jakobson et al., 2012; Stuart & Conduit, 2009). The experimenter called the participants name through an intercom and asked if they were



**Table 1.** The average proportion (percentage) of dream reports containing visual imagery, average participant 'vividness' ratings (scored out of 10), average 'visualisable' noun count (VNC), average total word count (TWC) and the average proportion of visualisable nouns to total words (VNC/TWC ratio) for dream reports from the tDCS, low tDCS, and blank control (no tDCS) conditions in Experiment 1.

Dream report variable	Conditions		
	tDCS	Low tDCS	Blank control (no tDCS)
Number of imagery reports	8/9 [89%]	9/9 [100%]	8/9 [89%]
Average 'Vividness' ratings (out of 10)	6 (4.27)	5.61 (3.92)	5.94 (4.07)
Average 'Visualisable' noun count (VNC)	5.5 (3.78)	4.9 (2.64)	5.3 (3.43)
Average total word count (TWC)	44.9 (30.61)	56.3 (38.96)	49.1 (29.08)
Average VNC/TWC ratio	.14 (.11)	.10 (.02)	.11 (.06)

Note. Standard deviation of each mean is listed in round brackets.

awake. Once the participant responded, the experimenter played the following pre-recorded questions: "Could you describe any thoughts or images that were going through your mind just before I called to you?", then "Is there anything else you can remember?" Participants were then asked to rate the vividness of any imagery on a scale from 1 "least vivid (hardly memorable)" to 10 "most vivid (like real)". Finally, the participant was asked "Do you think you received stimulation or no stimulation?" after which they were instructed to return to sleep. The participants' responses were recorded onto audio cassette.

## 2.5. Analysis

Participant PSG records were scored in 30 second epochs (Iber et al., 2007) by an independent sleep technologist, with no knowledge of the experimental aims and blind to conditions. The procedures adopted for quantifying the dream report data were similar to those used previously (Fedyszyn & Conduit, 2007; Jakobson et al., 2012; Stuart & Conduit, 2009). Dream report scoring was also done by an independent rater, blind to the aims and conditions of the studies. Each report was scored for (1) the number of visualisable nouns, and (2) the presence of visual imagery.

Both studies employed a repeated measures design as participants were exposed to the conditions during a single night. The order of conditions was counterbalanced across participants to accommodate for time of night effects. Of the 114 reports sampled across the two experiments, the two independent raters had 100% agreement with reports judged to contain imagery. Only seven reports (6%) contained a VNC where the raters were inconsistent. For these reports, the decision of a third independent rater was used. The distribution of all dependent variables within conditions was significantly different from a normal distribution using the Shapiro-Wilks W statistic ( $p < .05$ ). Hence, the non-parametric equivalent of a repeated measures ANOVA (Friedman chi-square) was adopted to analyze overall differences between conditions in the dream report data using SPSS 16.0 (SPSS, Inc., Chicago, USA). The non-parametric equivalent of repeated measures t-tests (Wilcoxon signed-rank tests) were used for pairwise comparisons between conditions ( $\alpha = 0.05$ , two-tailed).

## 3. Results

### 3.1. Study 1

#### 3.1.1 Sleep data

From a total of 16 participants who took part in this study, one participant was unable to sleep in the laboratory environment, two displayed insufficient REM sleep, three awoke each time stimulation was applied, and one was excluded based on the sleep stage scoring of the independent rater. Hence, the sample size was reduced to 9 right-handed participants (4 males, 5 females). Of the 9 participants administered 2 mA, only 4 were able to maintain REM sleep without signs of arousal during stimulation. Thus, the remaining participants were given stimulation at 1.5 mA and maintained REM sleep without signs of arousal. The average total sleep time was 437.72 mins (SD = 95.21) with an average of 358.47 mins in NREM (SD = 78.56) and 79.25 mins in REM sleep (SD = 29.65).

The procedure of counterbalancing the order of conditions resulted in an equivalent average time of night for each condition (tDCS = 5:28 am [SD = 1.94], low tDCS = 6:01 am [SD = 2.20], blank control = 6:45 am [SD = 2.44]). A repeated measures ANOVA showed no significant differences between the average awakening times across conditions,  $F(2, 20) = 1.30$ ,  $p = .29$ . The standard awakening procedures resulted in equivalent awakening times into REM sleep for each condition (tDCS = 291 seconds [SD = 141], low tDCS = 239 seconds [SD = 179], blank control = 342 seconds [SD = 204]). A repeated measures ANOVA showed no significant differences between the average time into REM across conditions,  $F(2, 20) = 2.74$ ,  $p = .09$ .

#### 3.1.2 Participant knowledge of the study conditions

Participants successfully guessed the tDCS condition on 33.33% of awakenings following this condition. Hence, the participants correctly identified the condition at a rate no greater than chance.

#### 3.1.3 Dream report data

The frequency of imagery reports, average visualisable noun count (VNC), average total word count (TWC), average proportion of visualisable nouns to TWC (VNC/TWC ratio),

**Table 2.** The average proportion (percentage) of dream reports containing visual imagery, average participant 'vividness' ratings (scored out of 10), average 'visualisable' noun count (VNC), average total word count (TWC) and the average proportion of visualisable nouns to total words (VNC/TWC ratio) for dream reports from the tDCS, low tDCS, and blank control (no tDCS) conditions in Experiment 2.

Dream report variable	Conditions		
	tDCS	Low tDCS	Blank control (no tDCS)
Number of imagery reports	15/24 [63%]	19/25 [76%]	18/23 [78%]
Average 'Vividness' ratings (out of 10)	4.88 (3.15)	5.97 (3.89)	5.6 (4.21)
Average 'Visualisable' noun count (VNC)	2.14 (2.35)	2.04 (2.22)	2.39 (2.48)
Average total word count (TWC)	32.54 (51.81)	35.04 (46.79)	33.93 (37.1)
Average VNC/TWC ratio	.09 (.08)	.10 (.09)	.11 (.10)

Note. Standard deviation of each mean is listed in round brackets.

and average imagery ratings for each condition are summarized in Table 1. Friedman Chi-square analyses examining differences between the tDCS, low tDCS, and blank control conditions revealed no significant difference in percentage of imagery reports [ $\chi^2(2) = 1.00, p = .61$ ], word count measures [VNC:  $\chi^2(2) = .72, p = .70$ ; TWC:  $\chi^2(2) = 1.80, p = .41$ ; VNC/TWC ratio:  $\chi^2(2) = .68, p = .71$ ], or imagery ratings [ $\chi^2(2) = .06, p = .97$ ].

## 3.2. Study 2

### 3.2.1 Sleep data

From a total of 28 participants who took part in this study, one participant was unable to sleep in the laboratory environment, one displayed insufficient REM sleep, one awoke each time stimulation was applied, and two were excluded based on the sleep stage scoring of the independent rater. Hence, the sample size was reduced to 23 right-handed participants (9 males, 14 females). The average stimulation duration for the 'tDCS' condition was 95.35 seconds (1.60 mA approximately). The average total sleep time was 411.32 mins (SD = 92.55) with an average of 350.67 mins in NREM (SD = 77.83) and 60.65 mins in REM sleep (SD = 25.44).

The procedure of counterbalancing the order of conditions resulted in an equivalent average time of night for each condition (tDCS = 6:37 am [SD = 2.13], low tDCS = 6:45 am [SD = 2.09], blank control = 6:22 am [SD = 2.20]). A Friedman chi-square showed no significant differences between the average awakening times across conditions, ( $\chi^2(2) = 1.65, p = .44$ ). The awakening procedure resulted in equivalent awakening times into REM sleep for each condition (tDCS = 318 seconds [SD = 103], low tDCS = 297 seconds [SD = 157], blank control = 366 seconds [SD = 215]). A Friedman chi-square showed no significant differences between the average time into REM across conditions, ( $\chi^2(2) = .81, p = .67$ ).

### 3.2.2 Participant knowledge of the study conditions

Participants successfully guessed the tDCS condition on 47.83% of awakenings following this condition. Hence, the participants correctly identified the condition at a rate no greater than chance.

### 3.2.3 Dream report data

The frequency of imagery reports, average visualisable noun count (VNC), average total word count (TWC), average proportion of visualisable nouns to TWC (VNC/TWC ratio), and average imagery ratings for each condition are summarized in Table 2. Friedman Chi-square analyses examining differences between the tDCS, low tDCS, and blank control conditions revealed that although there were on average fewer dream reports with visual imagery in the tDCS conditions compared to the blank control condition, this difference was not significant,  $\chi^2(2) = 2.12, p = .35$ . This was also the case for visualisable nouns [ $\chi^2(2) = .66, p = .72$ ] and to some extent the imagery ratings [ $\chi^2(2) = 5.71, p = .06$ ] where reports contained on average fewer nouns in the tDCS and low tDCS conditions and lower vividness ratings in the tDCS condition compared to the control condition, respectively. Furthermore, there was no significant overall difference in TWC and VNC/TWC ratio across conditions ( $\chi^2(2) = .24, p = .89$ ;  $\chi^2(2) = 1.79, p = .41$ , respectively).

## 4. Discussion

### 4.1. tDCS presentation during REM sleep

For both studies, statistical analyses confirmed that the time of night of awakenings and time into REM did not significantly differ between the conditions. In addition, participants correctly guessed the tDCS condition at a rate no greater than chance indicating that attempts to blind the participants to the respective condition given were successful.

### 4.2. Dream report data

For both studies, no significant difference was observed between the conditions for any of the dependent variables. This finding suggests that simultaneous cathodal and anodal tDCS of the posterior parietal and frontal cortex (respectively) had no effect on reported dream cognition using the two different delivery methodologies adopted. These findings are inconsistent with our previous research applying tDCS during stage 2 sleep (Jakobson et al., 2012). In that study the same cortical regions were targeted, applying anodal stimulation to the right posterior parietal and cathodal stimulation to the frontal cortex, but it was shown that tDCS could facilitate dream reporting during stage 2 sleep. It was

thought that this was achieved through depolarization of local posterior parietal cortical neurons and hyperpolarization of local frontal cortical neurons.

However, in this study, reversed polarity stimulation of the same regions and at the same stimulation intensity during REM sleep has not been sufficient to inhibit dream reports and presumably, inhibit local parietal and excite frontal cortical circuitry during REM sleep. The difference in results between the REM and stage 2 studies may be due to inherent physiological differences between these two sleep stages. Specifically, REM sleep is a state of heightened cortical activity (Llinas & Pare, 1991; Llinas & Steriade, 2006) when compared to other sleep stages including stage 2 sleep. Additionally, in contrast to wakefulness, REM sleep has been shown to be accompanied by a reduction of inhibitory activity in cortical neurons (Steriade, 1976). As such, REM sleep may be a state when cortical activity is not easily manipulated using tDCS when compared to stage 2 sleep.

#### 4.3. Methodological considerations

The fact that tDCS did not have an effect on the measures of reported dream imagery suggests that dreams during REM sleep are difficult to suppress, as dreams reported from REM sleep are by their very nature vivid – particularly late night REM (Fosse, Stickgold, & Hobson, 2004).

In addition, this study presented two experiments in REM sleep, the first study employing a method we previously used to deliver tDCS during stage 2 sleep (Jakobson et al., 2012) and the second a threshold design. The method used in the first study was consistent with the methodology of our previous stage 2 study, which allowed stimulation to be delivered during sleep for a fixed time interval and level of stimulation across participants and conditions. However, one disadvantage of such an approach is that the maximum levels of tDCS for each participant are not necessarily achieved. In contrast, the second study adopted a threshold design whereby tDCS was delivered in an increasing fashion until reaching the participants' individual arousal threshold. However, there was considerable variation in the stimulation intensity delivered across participants for the 'tDCS' condition due to their individual arousal thresholds, where the majority of participants across the two studies (56.25%) received up to 1.6 mA tDCS. Specifically, the mean stimulation intensities for study 1 and study 2 were 1.72 mA (SD = .26) and 1.60 mA (SD = 36.18), respectively, indicating that there was very little difference between the two studies in the overall stimulation intensity delivered. So while the aim of the threshold study was to maximize the level of stimulation delivered, only 21.74% received the full 2 mA stimulation intensity (for a period of 151 seconds) compared to 44.44% receiving the full 2 mA stimulation (for a period of 181 seconds) in the first study. Alternatively, 100% of the participants in study 1 received stimulation at an intensity equal to or greater than 1.5 mA, whereas only 43.48% in the threshold study received stimulation equal to or greater than 1.6 mA. These points indicate that some modifications to the stimulation protocol are needed in order to maximize the number of participants receiving higher intensity stimulation. This could potentially be achieved with the use of topical cutaneous anesthetic creams such as EMLA (AstraZeneca International, Pty Ltd). This would provide a method of masking the local cutaneous sensations of the tDCS and thus allow participants to sleep through the stimulation. Thus, had the current study included more participants

who were able to tolerate a greater level of the stimulation, observable effects on visual imagery and dream reporting may have been obtained. However, intensities of 1 - 2 mA is the standard protocol employed in tDCS studies which have yielded significant results. Similarly, tDCS studies during sleep have employed longer stimulation durations (up to 30 minutes) (Marshall et al., 2004; Nitsche et al., 2010) compared to that used in the current studies (up to 3 minutes). However, based on the results of our stage 2 studies, which also employed up to 3 minutes of stimulation, it was anticipated that it would also be sufficient to elicit an effect on dream recall from REM sleep. Finally, the different durations employed across the various conditions in study 2 could also have contributed to the current results, with on average more time spent in REM sleep in the 'tDCS' condition when compared to the other conditions (61 seconds). Thus, the discrepancy in the results between our previous stage 2 studies and the current REM studies highlights the need to take stimulation duration into consideration.

#### 4.4. Conclusions

In summary, using two different methodologies it appears that tDCS had no effect on the presence of dream reports with visual imagery or measures of dream quality. However, this may be due to methodological limitations of these studies, as the delivery methods employed allowed only low levels of tDCS to be delivered without waking participants. Improvements allowing higher levels of stimulation during sleep and stimulation of other cortical regions could potentially provide more definitive conclusions regarding the effectiveness of tDCS on dream imagery reported from REM sleep.

#### References

- Antal, A., Kincses, T. Z., Nitsche, M. A., & Paulus, W. (2003). Modulation of moving phosphene thresholds by transcranial direct current stimulation of V1 in human. *Neuropsychologia*, 41, 1802-1807.
- Antal, A., Kincses, T. Z., Nitsche, M. A., Bartfai, O., & Paulus, W. (2004). Excitability changes induced in the human primary visual cortex by transcranial direct current stimulation: Direct electrophysiological evidence. *Investigative Ophthalmology and Visual Science*, 45(2), 702-707.
- Antrobus, J. S., Fein, G., Jordan, L., Ellman, S. J., & Arkin, A. M. (1991). Measurement and design in research on sleep reports. In: S. J. Ellman, & J. S. Antrobus (Eds.), *The mind in sleep: Psychology and psychophysiology* (2nd ed.). New York, NY: John Wiley & Sons.
- Banich, M. T., & Heller, W. (1998). Evolving perspectives on lateralization of function. *Current Directions in Psychological Science*, 7, 1-2.
- Been, G., Ngo, T. T., Miller, S. M., & Fitzgerald, P. B. (2007). The use of tDCs and CVS as methods of non-invasive brain stimulation. *Brain Research Reviews*, 56, 346-361.
- Boggio, P. S., Castro, L. O., Savagim E. A., Braitte, R., Cruz, V. C., Rocha, R. R.,...Fregni, F. (2006). Enhancement of non-dominant hand motor function by anodal transcranial direct current stimulation. *Neuroscience Letters*, 404, 232-236.
- Conduit, R. (1999). A phenomenological investigation of the relationship between Ponto-Geniculo-Occipital (PGO) activity and dream reporting in humans: Toward an attention-based model of dreaming (Unpublished Ph.D. dissertation). La Trobe University, Melbourne, Australia.
- Dieckhöfer, A., Waberski, T. D., Nitsche, M., Paulus, W., Buch-



- ner, H., & Gobbelé, R. (2006). Transcranial direct current stimulation applied over the somatosensory cortex - Differential effect on low and high frequency SEPs. *Clinical Neurophysiology*, 117, 2221-2227.
- Doricchi, F., & Violani, C. (1992). Dream recall in brain-damaged patients: A contribution to the neuropsychology of dreaming through a review of the literature. In: J. S. Antrobus., & M. Bertini (Eds.), *The neuropsychology of sleep and dreaming*. New Jersey: Lawrence Erlbaum Associates.
- Fedyszyn, I. E., & Conduit, R. (2007). Tone induction of ocular activity and dream imagery from stage 2 sleep. *Dreaming*, 17(1), 35-47.
- Fosse, R., Stickgold, R., & Hobson, J. A. (2004). Thinking and hallucinating: Reciprocal changes in sleep. *Psychophysiology*, 41, 298-305.
- Fregni, F., Boggio, P. S., Nitsche, M. A., Berman, F., Antal, A., Feredoes, E.,... Pascual-Leone, A. (2005). Anodal transcranial direct current stimulation of prefrontal cortex enhances working memory. *Experimental Brain Research*, 166(1), 23-30.
- Hobson, J. A., & McCarley, R. W. (1974a). Multiple firings by cat cerebellar Purkinje cells in sleep and waking. *Experimental Neurology*, 44, 41-48.
- Hobson, J.A., McCarley, R. W., Pivik, R. T., & Freedman, R. (1974b). Selective firing by cat pontine brain stem neurons in desynchronized sleep. *Journal of Neurophysiology*, 37, 497-511.
- Hobson, J. A., McCarley, R. W., Freedman, R., & Pivik, R. T. (1974c). Time course of discharge rate changes by cat pontine brain stem neurons during sleep cycle. *Journal of Neurophysiology*, 37, 1297-1309.
- Hobson, J. A., Pace-Schott, E. F., & Stickgold, R. (2000). Dreaming and the brain: Toward a cognitive neuroscience of conscious states. *Behavioral and Brain Sciences*, 23(6), 793-1121.
- Hong, C. C., Gillin, J. C., Dow, B. M., Wu, J., & Buchsbaum, M. S. (1995). Localized and lateralized cerebral glucose metabolism associated with eye movements during REM sleep and wakefulness: A positron emission tomography (PET) study. *Sleep*, 18, 570-580.
- Iber, C., Ancoli-Israel, S., Chesson, A., & Quan, S. (2007). *The AASM manual for the scoring of sleep and associated events – rules, terminology and technical specifications*. Westchester, IL: American Academy of Sleep Medicine.
- Jakobson, A. J., Fitzgerald, P. B., & Conduit, R. (2012). Induction of visual dream reports after transcranial direct current stimulation (tDCs) during Stage 2 sleep. *Journal of Sleep Research*. doi: 10.1111/j.1365-2869.2011.00994.x.
- Jasper, H. H. (1958). The ten-twenty electrode placement system of the International Federation. *Electroencephalography and Clinical Neurophysiology*, 10, 495-501.
- Kincses, T. Z., Antal, A., Nitsche, M. A., Bartfai, O., & Paulus, W. (2003). Facilitation of probabilistic classification learning by transcranial direct current stimulation of the prefrontal cortex in the human. *Neuropsychologia*, 42(1), 113-117.
- Lang, N., Nitsche, M. A., Paulus, W., Rothwell, J. C., & Lemon, R. N. (2004). Effects of transcranial direct current stimulation over the human motor cortex on corticospinal and transcallosal excitability. *Experimental Brain Research*, 156(4), 439-443.
- Lang, N., Siebner, H. R., Ward, N. S., Lee, L., Nitsche, M. A., Paulus, W.,... Frackowiak, R. S. (2005). How does transcranial DC stimulation of the primary motor cortex alter regional neuronal activity in the human brain? *European Journal of Neuroscience*, 22(2), 495-504.
- Llinas, R. R., & Pare, D. (1991). Of dreaming and wakefulness. *Neuroscience*, 44, 521-535.
- Llinas, R. R., & Steriade, M. (2006). Bursting of thalamic neurons and states of vigilance. *Journal of Neurophysiology*, 95, 3297-3308.
- Maquet, P., Peters, J. M., Aerts, J., Delfiore, G., Degueldre, C., Luxen, A., & Franck, G. (1996). Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. *Nature*, 383(6596), 163-166.
- Marshall, L., Molle, M., Hallschmid, M., & Born, J. (2004). Transcranial direct current stimulation during sleep improves declarative memory. *The Journal of Neuroscience*, 24(44), 9985-9992.
- Marshall, L., Molle, M., Siebner, H. R., & Born, J. (2005). Bifrontal transcranial direct current stimulation slows reaction time in a working memory task. *BMC Neuroscience*, 6(1), 23-29.
- Marshall, L., Kirov, R., Brade, J., Mölle, M., & Born, J. (2011). Transcranial electrical currents to probe EEG brain rhythms and memory consolidation during sleep in humans. *PLoS One*, 6(2), e16905.
- McCarley, R. W., & Hobson, J. A. (1975). Discharge patterns of cat pontine brain stem neurons during desynchronized sleep. *Journal of Neurophysiology*, 38, 751-766.
- Nielsen, T. A. (2000). A review of mentation in REM and NREM sleep: 'Covert' REM sleep as a possible reconciliation of two opposing models. *Behavioral and Brain Sciences*, 23(6), 851-866.
- Nitsche, M. A., Jakoubkova, M., Thirugnanasambandam, N., Schmalfluss, L., Hulleman, S., Sonka, K.,... Happe, S. (2010). Contribution of the premotor cortex to consolidation of motor sequence learning in humans during sleep. *Journal of Neurophysiology*, 104, 2603-2614.
- Nofzinger, E. A., Mintun, M. A., Wiseman, M., Kupfer, D. J., & Moore, R. Y. (1997). Forebrain activation in REM sleep: An FDG PET study. *Brain Research*, 770, 192-201.
- Pivik, R. T., McCarley, R. W., & Hobson, J. A. (1977). Eye movement-associated discharge in brain stem neurons during desynchronized sleep. *Brain Research*, 121, 59-76.
- Rechtschaffen, A., & Kales, A. (1968). *A manual for the standardized terminology, techniques and scoring system for sleep stages of human subjects*. Washington, DC: Public Health Services, U.S. Government Printing Office.
- Rogalewski, A., Breitenstein, C., Nitsche, M. A., Paulus, W., & Knecht, S. (2004). Transcranial direct current stimulation disrupts tactile perception. *European Journal of Neuroscience*, 20(1), 313-316.
- Solms, M. (1997). *The neuropsychology of dreams: A clinico-anatomical study*. New Jersey: Lawrence Erlbaum Associates.
- Solms, M. (2000). Dreaming and REM sleep are controlled by different brain mechanisms. *Behavioral and Brain Sciences*, 23(6), 793-1121.
- Steriade, M. (1976). Cortical inhibition during sleep and waking. In: T. Desiraju (Eds.), *Mechanisms in transmission of signal for conscious behavior*. Amsterdam: Elsevier.
- Stuart, K., & Conduit, R. (2009). Auditory inhibition of rapid eye movements and dream recall from REM sleep. *Sleep*, 32(3), 399-408.
- Vines, B. W., Schnider, N. M., & Schlaug, G. (2006). Testing for causality with transcranial direct current stimulation: Pitch memory and the left supramarginal gyrus. *Neuroreport*, 17, 1047-1050.