Pilot Study: REM Quality, Dream Recall and Sleep-maintenance in Patients with PCA stokes

Caitlyn Greyling

Department of Psychology

University of Cape Town

Supervisor: Mark Solms

Co-Supervisor: Danyal Wainstein

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ABSTRACT

Based on the dopaminergic theory of dreaming it was proposed that dream loss would be related to disturbed sleep. This pilot study explored whether changes in rapid eye movement (REM) sleep quality and quantity were related to dream loss in stroke patients. As dream recall is reported most frequently from REM sleep, this portion of sleep was the focus of this study. It was predicted that dream loss would lead to reduced REM sleep quality and quantity. Polysomnographic (PSG) recordings were obtained for two consecutive nights in the sleep laboratory at the Cape Sleep Centre for patients who ceased to dream following thrombotic infarctions in the posterior cerebral artery (PCA) territory with corresponding occipital lesions. In addition, PSG recordings were completed for patients with the same neuropathology who continued to dream. All patients were selected by referral from neurological specialists at Gatesville Medical Centre, Cape Town. Accordingly, the sample size for the pilot study was dependent on the availability of patients with the correct lesions. Multiple measures of REM sleep quality and quantity, as well as general sleep efficiency, for dreaming *(n*= 4), non-dreaming (*n*=5), and recovered-dreaming patients (*n*=3) were described. Trends emerged in the pilot data that suggest that dream loss is related to reduced REM quality and quantity. The implications of these results for the dopaminergic hypothesis of dreaming are discussed. Furthermore, the benefits of conducting a main study to test the sleep protection hypothesis of dreaming are discussed as well.

Keywords: dreaming, dream loss, disturbed sleep, posterior cerebral artery (PCA) stroke, REM sleep quality, Alpha activity, pilot study.

REM Quality, Dream Recall and Sleep-maintenance

The discovery of REM sleep in 1953 (Aserinsky & Kleitman, 1953) and the subsequent positive correlation of REM sleep to dreaming (Dement & Kleitman, 1957), acted as an impetus for a surge in dream research. Countless theories emerged trying to establish why people dream (Breger, 1967; Crick & Mitchison, 1983; Hobson & McCarley, 1977). However many of these theories did not distinguish between the potential physiological functions of REM sleep and a physiological function for dreams. As REM sleep was shown to be casually generated by structures in the pons (Jones, 1979; Jouvet, 1967) early theories argued that dreaming was generated by cholinergic mechanisms in the pontine brainstem as well (Hobson & McCarley, 1975). However, theories have subsequently been revised in light of evidence that pontine brainstem lesions do not result in the global cessation of dreaming (Solms, 1997). Conversely, Solms (1997) identified the parieto-tempero-occipital (PTO) junction and the limbic ventro-mesial frontal white matter as being the primary driving force behind the process of dreaming. This is in light of the fact that damage to either of these regions has been found to produce a complete loss of dreaming (Solms, 1997). Contrary, then, to Hobson and McCarley (1977), Solms (1997) hypothesized, on the basis of these lesion studies, that dreaming is primarily controlled by the dopaminergic forebrain system and not by cholinergic brainstem mechanisms. Ample evidence demonstrates that dreaming and REM sleep are *doubly dissociable states* (Bischof & Bassetti, 2004; Foulkes & Vogel, 1965 Poza & Marti Massó, 2006; Solms, 2000), showing that dreaming is not only an epiphenomenon of REM processes, for this reason it is viable to pursue the scientific exploration of a potential physiological function for dreaming.

In the last few decades, in particular, multiple lines of research have pointed to the posterior cortical regions and the white matter tracts surrounding the frontal horns of the lateral ventricles as being fundamentally involved in the generation of dreams (Bischof & Bassetti, 2004; Doricchi & Violani, 1992; Poza & Marti Massó, 2006; Solms, 1997; Yu, 2007). In the following subsections, a review of clinical and experimental findings of patients with focal brain lesions demonstrated that these two brain areas play a vital role in the dreaming brain.

Freud's Dream Theory

Posterior Cortical Lesions. Posterior cortical lesions have been shown to be related to cessation of dreaming. However, precise localization of the lesion site has not been conclusively established. In a clinico-anatomical study conducted by Solms (1997), 361 patients suffering a range of neurological illnesses were questioned about the fluctuations in the nature or frequency of their dreams following their injury or illness. The findings from these clinical interviews were compared with the results from neurological tests, CT and MRI scans. Based on his comparison, Solms reported that damage to the parieto-tempero-occipital (PTO) junction (Figure 1) resulted in loss of visual dreaming. The loss of dreaming due to posterior cortical lesions, in particular occipital lobe lesions, is in some sense expected as the cessation of dreaming in such cases would be attributable to the inability to perpetually construct dream imagery (Dumont, Braun & Guimond, 2007; Solms, 2000). More interestingly damage to the second area, the ventro-mesial frontal white matter, is hypothesized to disrupt dreaming due to a loss of motivational capacity (Solms, 1997).

Figure 1. Dorsolateral prefrontal cortex (DLPFC)

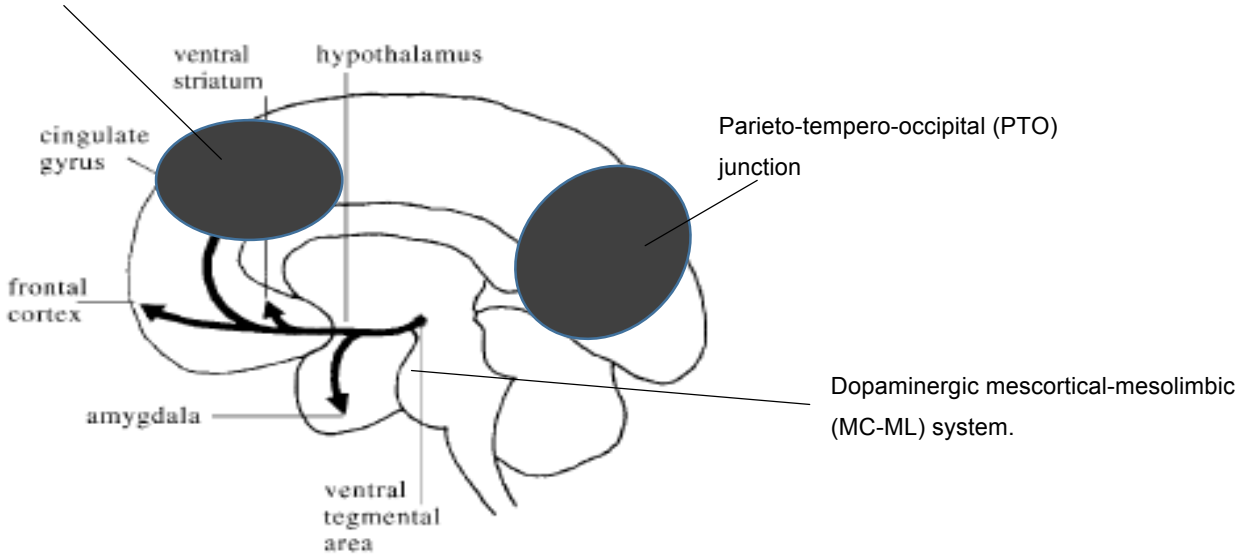

Figure 1. Dorsolateral prefrontal association cortex and posterior association cortex. The DLPFC serves as the highest order level of processing responsible for motor planning and organization, as well as the regulation, of intellectual function and action, especially with regards to impulse control (Pochon et al., 2001).The PTO junction which forms the posterior association cortex in the brain is not in control of primary sensory experiences, but takes part in sensory integration (Yu, 2007). Solms (1997, 2000) hypothesises that this is where dreaming is visuo-spatially constructed. Adapted from "Dreaming and REM sleep are controlled by different brain mechanisms," by M. Solms, 2000, *Behavioral and Brain Sciences*, 23, p. 846. Copyright 2000 by Cambridge University Press

Ventro-mesial Frontal Lesions. As with the posterior lesion site, Solms (1997) used clinico-anatomical observations to demonstrate that bifrontal lesions in the ventro-mesial region were associated with complete cessation of dreaming. This specific group of forebrain structures is known as the dopaminergic mescortical-mesolimbic (MC-ML) system (Figure 1). The role of the dopaminergic ML-MC system in dream generation has been seen with bilateral lesions in the ventro-mesial frontal white matter resulting in dream loss (Domhoff, 2001). Further evidence supporting the dopaminergic dream theory were pharmacological studies which showed that drugs (such as L-Dopa) increased levels of dopamine in the ventro-mesial quadrant of the frontal lobes intensified the vivacity and emotionality of dreaming (De Gennaro, Marzano, Cipolli & Ferrara, 2012; Hartmann et al., 1980). This added to earlier findings that chronic levodopa therapy, which increases levels of forebrain dopamine, was responsible for the generation of new dream phenomena (Sharf et al., 1978). The fact that dreaming ceases following a surgical procedure known as *modified prefrontal leucotomy* in which the dopaminergic pathway running through the MC-ML system is transacted also supports the view that the ventro-mesial frontal white matter is involved in the dream generation (Jus et al., 1973). Moreover, loss of dream mentation has been related with Parkinson's disease, which is largely regarded as the result of depleted levels of forebrain dopamine (Sandyk, 1997). Lastly, an increase in dopamine release within the MC-ML system has been reported in humans during REM sleep (Gottesmann, 2004).

SEEKING System

The dopaminergic MC-ML system is a central component of what Panksepp (1998) refers to as the SEEKING system—a model "psychobehavioural emotional and motivational system of the mammalian brain" (Perogamvros & Schwartz, 2012, p. 1936) which drives all mammals to interact with their environment. While the SEEKING system is extremely active during sleep (Dahan et al., 2007; Gottesmann, 2004), the dorsolateral prefrontal cortex (DLPFC) is deactivated during sleep (Solms, 2002; Figure 1). In light of this evidence, Solms (1997, 2000) hypothesises that since the latter brain region is disengaged during sleep, the appetitive urges (that manifest in the form of thoughts and actions) which ordinarily take shape here during waking cannot be carried out during sleep and therefore have to be redirected. Thus, Solms (2000) asserts that the appetitive urges stemming from the dopaminergic MC-ML system (Figure 2) are reverted to the PTO region, where they are experienced as dreams.

As the MC-ML system has been shown to be activated during sleep and with "SEEKING" behaviours during waking, a paradox is evident. How can one maintain sleep when a system is activated that during waking typically motivates one to actively engage in SEEKING behaviour? The deduction drawn by the dopaminergic theory based on the literature reviewed above is that dreams maintain sleep by generating a virtual SEEKING experience where motivational behaviour can be carried out without awakening (Solms, 2000). This *sleep protection hypothesis* is important as sleep disruption has been shown to have various short- and long-term consequences including high blood pressure, impaired concentration and depression (Chokroverty, 2010).

The above hypothesis closely resembles Freud's dream theory. Freud (1900) was one of the first to outline an extensive theory of the function of dreaming that would later form the foundation of psychoanalysis. In his book, *The Interpretation of Dreams* (1900), Freud argued that dreams are part of a process of unconscious wish-fulfilment[1]. Moreover, Freud asserted that is through the fulfilment of these unconscious desires, of which the desire to remain asleep is the most important, that dreams function to maintain sleep. Following Freud's death and the subsequent discovery of REM sleep and dreaming being highly correlated (Aserinksy & Kleitman, 1955; Dement & Kleitman, 1957) the search for the neural correlates of dreaming from a neuroscientific perspective commenced in earnest. As Freud's dream theory is the bedrock of psychoanalysis, there is immense significance in empirically assessing whether dreaming is a function of sleep maintenance. For the reason that, a global cessation of dreaming has been shown to be related to posterior cortical lesions (Solms, 1997), it can be theorized that patients with such lesions will be unable to redirect the surge of neural activity that is normally associated with REM sleep (Solms, 2000). Consequently, it can be argued that by testing the quality and quantity of REM sleep in patients who have ceased to dream following posterior lesions, specifically occipital lesions, compare to patients with the same neuropathology who continue to dream it will be possible to test Freud's dream theory. One such measure of REM quality that may be affected by dream loss is Alpha activity (8-13 Hertz).

[1] Freud's view is that all dreams are a form of wish-fulfilment- in other words, attempts by the unconscious to resolve a conflict. Because the content in the unconscious is often disturbing in form, a "censor" in the preconscious alters the information before transferring it to the conscious (Freud, 1900).

The Relevance of Alpha Activity to the Sleep Protection Dream Hypothesis

The discovery of an association between EEG-defined REM sleep and dream recall (Dement & Kleitman, 1957) has resulted in a surge of scientific endeavours seeking to identify the electrophysiological correlates of dreaming which has been met with mixed results (Morel, Hoffman & Moffitt, 1991; Williamson, Csima, Galin & Mamelak, 1986; Wollman & Antrobus, 1987). Alpha activity (8-13 Hertz) is one of the most prominent correlates that have been distinguished in this regard, and is universally acknowledged as an indication of a state of relaxed wakefulness (Pivik and Harman, 2009). Furthermore, reduced amounts of Alpha signify sleep onset, and the occurrence of Alpha activity throughout sleep is assumed to be a sign of arousal[2] (Pivik and Harman, 1995).

Alpha Activity Associated with Sleep-maintaining Processes. In 1973, a form of Alpha disturbance during sleep in psychiatric patients was documented by Hauri and Hawkins and termed 'alpha-delta sleep'. Alpha-delta sleep refers to sleep punctuated by Alpha activity during Stage 3 and 4 of non-rapid eye movement sleep (NREM), stages of sleep which are typically supposed to be comprised of lower frequencies (i.e., < 4 Hertz). Sleep was reported to be maintained during these EEG activities until it was further noted that this type of sleep replaced *slow-wave sleep*[3] in some patients. Consequently, such subjects complained of *nonrestorative sleep.* Subsequent reports have confirmed the association of Alpha intrusions in clinical populations that complain of nonrestorative sleep (Mahowald, Mahowald, Bundlie & Ytterberg, 1989; Moldofsky, Scarisbrick, England, & Smythe, 1975; Moldofsky, 1993; Wittig, Zorick, Blumer, Heilbronn, & Roth, 1982). Therefore, increases in Alpha activity are related to disturbed sleep.

Reduced Alpha Power Associated with Dream Recall from Stage REM. Despite the incongruities in research attempting to determine the electrophysiological correlates of dreaming, there does appear to be a relationship between Alpha activity and dreaming. Hong et al. (1996) found a negative correlation linking Alpha power (8- 12 Hertz) over central and

[2] Arousals can be briefly described in terms of transient phenomena marked by a 3 to 14 second intrusion of alpha, beta, or theta waves resulting in fragmented sleep without behavioural waking. Arousals are represented as a number per hour (Arousal Index; AI) and an AI of up to 10 is normal in middle aged adults (Chokroverty, 2009).

[3] Slow-wave sleep is composed of stage 3 and stage 4 non-rapid eye movement sleep and is usually referred to as deep sleep.

parietal EEG sites, analogous with Broca's area and Wernicke's areas, with expressive and receptive language in dream reports. Alpha power in central and occipital O2[4] derivations was similarly reported to be negatively correlated with degree of visual content in both congenitally blind and sighted subjects (Bertolo et al., 2003). Furthermore, increased Alpha activity (11.72 -13.67 Hertz) in the central area was reported to be negatively correlated with SOREMP[5] (Sleep Onset Rapid Eye Movement Periods) dreams and positively correlated with NREMP (Sleep Onset Non Rapid Eye Movement Periods) dreams (Takeuchi, Ogilvie, Murphy & Ferrelli, 2003).These studies suggest that reduced Alpha activity may herald successful dream recall.

In keeping with the notion above, dream recall was reported to be negatively correlated with Alpha power, especially middle Alpha activity (9.5 – 11.5 Hertz) in REM sleep as well as Stage 2 sleep (Esposito, Nielsen & Paquette, 2004). More recently, REM sleep was shown to be positively correlated with low frontal Alpha activity and high Alpha and Beta activity in occipital derivations (Chellappa, Frey, Knoblauch & Cajochen, 2011). Consequently, Chellappa et al. (2011) demonstrated that offline facilitation of sleep mentation is related to reduced REM Alpha activity, signifying that this particular reduction in Alpha activity is associated with dream recall. In addition, Marzano et al. (2011) found that morning REM had a higher Theta frequency (5-7 Hertz) and Stage 2 sleep had lower Alpha oscillatory activity (8- 12 Hertz) related with successful dream recall. Therefore despite the inconsistencies in the EEG correlates of dreaming, there appears to be some relation to Alpha activity. Furthermore this relationship, between Alpha activity and dreaming, may even differ as a function of sleep stage.

Alpha Activity in Dreaming and Sleep. In summary, not only has decreased Alpha activity been shown to be related to mentation during sleep, but interestingly, an increase in activity has also been shown to be related to disturbed sleep. As dreaming is related to reduced alpha activity it is reasonable to propose that *dream loss may be related to an increase in alpha activity, and that this increase may in turn signify less consolidated sleep*.

[4] Using the 10/20 System of electrode placement, 02 refers to the occipital electrode site on the right hemisphere of the head.

[5] Atypical beginning of sleep by entering into REM periods within 15 minutes of sleep onset (Spriggs, 2002). The study by Takeuchi et al. (2003) experimentally induced SOREMPs and NREMPs in healthy patients using the Sleep Interruption Technique (SIT) to investigate the quantitative and qualitative between SOREMPs and NREMPs dreams.

This is further supported by the inverse relationship between SOREMP dreams and Alpha activity reported by Takeuchi et al. (2003). Therefore, if dream loss is found to be related to an increase in the amount of Alpha activity in REM sleep, this will provide additional evidence that dreaming may be a sleep maintaining mechanism.

Quality of Sleep in Non-dreamers

Consistent with this hypothesis, Solms (1997) tested this hypothesis in a clinical investigation by asking patients with various brain injuries and illnesses, affecting both anterior frontal regions and the posterior PTO region, to subjectively rate their sleep quality, and found that non-dreamers rated their sleep quality as significantly worse.

Furthermore, Bischof and Bassetti (2004) reported a case study of a patient who experienced cessation of dreaming after bilateral occipital stroke. While normal REM amounts, REM density[6], and REM latency[7] were documented, unaware of the potential significance, the authors also reported that the patient showed signs of sleep-maintenance insomnia. More recently, Poza and Marti Massó (2006) published a case study of a patient who completely ceased dreaming following a unilateral left tempero-occipital hematoma that resulted from a cerebral arteriovenous malformation (AVM). Again, despite normal REM sleep, the authors reported that the patient experienced nonrestorative sleep following the neurological damage. These findings suggested that disturbed sleep may be associated with dream loss as a result of neurological injury or illness. However, it is imperative to note that REM sleep was not investigated thoroughly in the latter case studies (Bischof and Bassetti, 2004; Poza and Marti Massó, 2006). Thus, the effect that dream loss has on the quality and quantity of REM sleep remains to be investigated.

Conclusion

All the evidence reviewed here renders Freud's (1900) hypothesis that dreams protect sleep to be empirically testable and falsifiable. Since Freud's dream theory is the bedrock of psychoanalysis, finding empirical support for the Freudian dream theory will add greater credibility to the field of psychoanalysis. Additionally, this would contribute to knowledge on the function of dreams considering that there are many theories that propose a physiological function for dreams but none have been empirically established to date (Solms & Malcom-

[6] The frequency of eye movements per unit of time during REM sleep (Spriggs, 2002).

[7] The period of time it takes to reach the first REM episode from sleep onset (Spriggs, 2002).

Smith, 2009). Based on the dopaminergic theory of dreaming (Solms, 2000; Yu, 2007) it it proposed that dream loss would be related to disturbed sleep. More, specifically it hypothesized that dream loss would lead to reduced REM sleep quality and quantity.

Aims and Objectives

As this was a pilot study, the aim was not hypothesis significance testing. The purpose of pilot studies should be to descriptively discuss findings related to the validity and successful implementation of a planned main study (Arain, Campbell, Cooper, & Lancaster, 2010; Thabane et al., 2010). Null hypothesis significance testing requires powered sample sizes. As pilot studies do not typically have large sample sizes (and powered samples sizes in particular) it is not appropriate to carry out hypothesis significance testing (Shanyinde, Pickering & Weatherall, 2011). Accordingly, the primary objective for this external pilot study is to describe the preliminary data with regards to the hypotheses of the main study. The following hypotheses are proposed for the main study:

H_1: Patients who have cessation of dreams following posterior cerebral artery (PCA) stroke will show increased Alpha activity in REM sleep compared to patients with PCA stroke who do dream.

H_1: Patients who cease to dream following posterior cerebral artery (PCA) stroke will show reduced REM sleep quality and quantity compared to patients with PCA stroke who do dream.

Additional objectives of this pilot study intended to test the (1) process, (2) resources, and (3) scientific basis of the planned main study (Thabane et al., 2010):

Process

1. Assess the feasibility and suitability of eligibility criteria for the main study's sample.
2. Test polysomnographic recording and electroencephalographic data analysis methods.

Resources

3. Assess suitability of software and equipment available for conducting the main study.

Scientific

4. Estimate the effect sizes of the pilot data as there is a dearth in the current literature to inform estimates of sample size for future research.

Method

Participants

All participants were selected from referrals by neurological specialists at Gatesville Medical Centre[8]. In total there were 12 participants (5 woman, 7 men, M_{age}= 54.58 years) between the ages of 42 and 67 years. The control group (dreaming participants) consisted of four participants (2 women, 2 men, M_{age}= 56.25 years). The non-dreamers consisted of five participants (3 women, 2 men, M_{age}=54.20 years). The recovered-dreamers consisted of three participants (3 men, M_{age}= 53.00 years).

The inclusion criterion was thrombotic infarctions in the posterior cerebral artery (PCA) territory. Thrombotic strokes were considered preferable for this pilot study as they create more circumscribed damage (see Appendix A for the Magnetic Resonance Images of patients). Due to this method of selection, sample size was highly dependent on the availability of patients with the correct lesions for this study. It was predicted, based on the study by Solms (1997), that the occurrence of such patients is not extremely rare. A further strict inclusion criterion was that patients had grossly intact REM cycles, which was documented by the neurological specialist at Gatesville Medical Centre, Cape Town, and confirmed in the sleep laboratory.

Exclusion criteria included the presence of any other sleep or neurological disorder that might confound the results, or the use of any medications that could affect sleep architecture. Patients were subsequently divided into non-dreamers and dreamers in order to compare sleep efficiency[9]. Patients in the control group were also selected from a similar age bracket to patients in the quasi-experimental groups to control for the effects that age has been shown to have on sleep quality (Redline *et al.,* 2004). Added to this was a control for amnesia so that patients who failed to recall dreams were not doing so due to being amnestic,

[8] This research is a pilot study deriving some of its data from a previous Master's quantitative multi-case study. The Master's study (Cameron-Dow, 2012) investigated general sleep quality and quantity in patients who had a thrombotic stroke in the region of the posterior cerebral arteries and who had experienced a total cessation of dreaming compare to patients with same neuropathology who continued to dream. However, REM quality in particular was not investigated in detail.

[9] Two non-dreaming and three dreaming patients were taken from Cameron-Dow's (2012) Master's study. While an additional three patients met the criteria for the non-dreamers group; another participant was added to the dreamers group and three patients were selected for the third group (the recovered-dreamers group) for this pilot study.

but had equivalent memory scores to dreamers. This method of selection controls for the confounding effect that may be associated with the experience of neurological damage, as stroke has been reported to have an impact on sleep quality (Chokroverty & Montagna, 2009).

Non-dreaming patients (Quasi-experimental group). The non-dreaming patients consisted of patients who had met the above selection criteria, but who also ceased dreaming. Therefore a strict inclusion criterion was that patients had not dreamed since their stroke. This was confirmed subjectively via patients' dream accounts and objectively in the sleep laboratory by awakening patients during REM sleep and asking them whether or not they were dreaming.

Recovered-dreaming patients (Quasi-experimental group). The recovered dreaming patients consisted of patients who were initially non-dreamers as they reported that they could not remember dreaming since the onset of their stroke, but upon awakening during REM sleep in the sleep lab they recalled having vague dreams.

Dreaming patients (Control group). The dreaming patients consisted of patients who had the same neuropathology as the quasi-experimental group, but who still dreamed. Therefore, the strict inclusion criterion was that patients subjectively reported normal dreams. This was then confirmed objectively in the sleep laboratory by awakening patients during REM sleep and asking them whether they were dreaming or not.

Measures

Dream recall. Nocturnal REM-sleep interviews were used to confirm the subjective dream recall reports of dreaming, non-dreaming and recovered dreaming patients. This is a common method for establishing dream presence (Benson & Greenberg, 1969; Brown, 1972; Efron, 1968; Goodenough, Lewis, Shapiro, Jaret, & Sleser, 1965; Jus et al., 1973; Kerr, Foulkes & Jurkovic, 1978; Murri, Massetani, Siciliano & Arena, 1985; Solms, 2000). During the first night in the sleep laboratory patients were awakened according to EEG-defined REM sleep and asked whether they were dreaming or not. More specifically, patients were awakened 10 minutes after the onset of the second REM period and 15 minutes after the onset of the third REM period, or were interviewed after spontaneous awakenings during REM sleep.

Subjective sleep quality. Subjective sleep quality of patients was assessed using the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman & Kupfer, 1989). The PSQI evaluates sleep quality and disturbances for a 1-month timeframe using self-rated indexes (Buysse et al., 1989). A global score of sleep quality was generated from summation of seven component scores. The seven components included: 1) subjective sleep quality, 2) sleep duration, 3) sleep disturbances, 4) sleep latency, 5) habitual sleep efficiency, 6) daytime dysfunction and 7) use of sleeping medication. A global score greater than 5 distinguished good sleepers from poor sleepers and generated a diagnostic sensitivity of 89.6% and specificity of 86.5% (Buysse et al., 1989)) The PSQI was scored according to the standard scoring procedures delineated by the test manual. PSQI allowed comparison between physiologic sleep parameters and the recording of the patient's perceived sleep experience. There is evidence of the reliability and validity of the PSQI in the elderly (Buysse, Reynolds, Monk et al., 1991; Gentili et al., 1995) and in stroke patients (Backhaus, Junghanns, Broocks, Riemann & Hohagen, 2002; Carpenter & Andrykowski, 1998).

Polysomnographic measures. The polysomnographic (PSG) recordings were done on a portable Alice © 5 *Respironics* polygraphic amplifier in the sleep laboratory at Gatesville Medical Centre, Cape Town. The following recording montage was used in the study as recommended by American Association of Sleep Medicine (AASM; Iber, Ancoli-Israel, Chesson and Quan, 2007): electroencephalogram (EEG; 4 leads, 2 channels), electrooculogram (EOG; 2 channels), and the submental electromyogram (EMG; chin and leg). More specifically, the following referential montage was used: F_z - A_2, C_Z - A_2, C_3 - A_2, O_2 - A_2 (see the electrode placement below in Figure 4). Eye movement was detected on two EOG channels when the spikes of opposite polarity occur simultaneously with a minimum amplitude of 35 microvolts[10] (μV) and a maximum duration of 3 seconds (sec). Rapid eye movement was detected when the ratio of spike amplitude to time was greater than 400 μV /sec. Slow eye movement was detected when the ratio of spike amplitude to time was less than 150 μV/sec and event duration was more than 1 sec. Oscillations bursts between 7.5 Hertz to 13 Hertz greater than 18.38 μV were marked as Alpha waves.

[10] Micro-volt is defined as one millionth of a volt and is the standard unit of measurement for recording and reading polysomnographic waves (Spriggs, 2002).

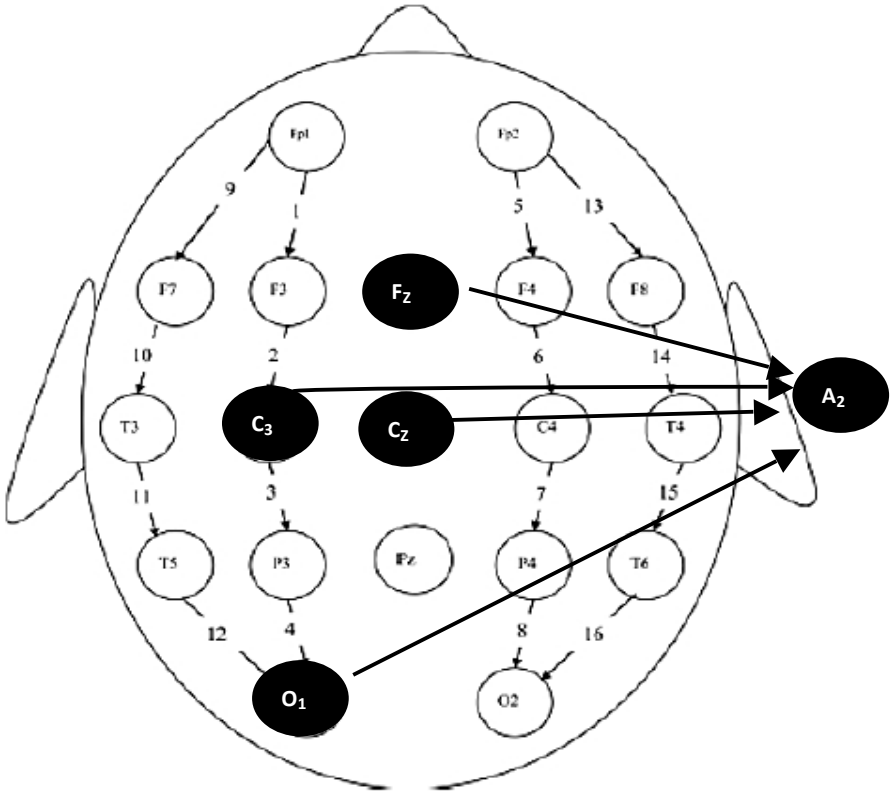

Figure 2. Electrode placement in pilot study. Referential montage with occipital (O1) and central (C3) electrodes placed over the left hemisphere of the head, using the International 10/20 System (Jasper, 1958). The conventional contra-lateral mastoid reference was employed (Pivik et al., 1993).

Design

The study used a quasi-experimental between-groups design. Differences in measures of REM quality and quantity, as well as general sleep efficiency, were descriptively compared between neurological patients who still dream and patients with the same neuropathology who do not dream. Therefore the dependent variables for the pilot study included the following measures of REM quality: micro-arousal[11] index for REM (MI), REM density[12] and Alpha activity (8-13 Hertz) in REM[13]. REM quantity was described with the following measures: percentage of REM spent in sleep period time (SPT) and longest REM period[14]. Lastly, the measures of general sleep efficiency included: PSQI, sleep efficiency[15] (SE) and sleep onset latency[16] (SL). The independent variable is a between-subjects factor with three levels, as the neurological patients are divided into three groups: *dreamers*, *non-dreamers,* and *recovered dreamers*.

The pilot study analysed data previously collected for a Master's study (Cameron-Dow, 2012) which followed the ethical guiding principles delineated by the Health Profession Council of South Africa (HPCSA) for research concerning human subjects. Guidelines specified by the University of Cape Town (UCT) Codes for Research were also adhered to. In addition, ethical approval was also acquired from the Psychology Department's Research Ethics Committee as well the Faculty of Health Sciences Research Ethics Committee at UCT respectively. As the pilot study analysed the data already collected for the Master's study, it is similarly ethically sound. (REC. REF. 163/2010).

Data Analysis

[11] In this pilot study, micro-arousals were characterized as arousal phenomena occurring for less than 15 seconds. Micro-arousal index is defined as the average number of micro-arousals per hour of sleep time.

[12] Defined in terms of the frequency of eye movements in REM sleep (Spriggs, 2002).Calculated by dividing the total minutes of rapid eye movements by total minutes of REM sleep and then multiplied by 100 (Spriggs, 2009).

[13] Alpha activity in REM was calculated for the pilot study as a percentage by dividing the number of Alpha events in REM by the total duration of REM sleep (in minutes) for the entire study.

[14] Longest REM period was the REM cycle with the longest duration (in minutes) for the entire study.

[15] The percentage of Total Recording Time that participant was asleep; calculated by dividing Total Sleep Time by Total Recording Time (Spriggs, 2009).

[16] The time it takes from lights out to sleep onset (measured in minutes).

Sleep Staging. The polysomnographic recordings were manually analysed and scored for 30-s epochs at the Cape Sleep Centre, Gatesville Medical Centre, Cape Town by a certified polysomnographic technologist according to standard sleep guidelines as set by the AASM (Iber, Ancoli-Israel, Chesson, & Quan, 2007). Thereafter, the data was compiled into a comprehensive sleep report using Alice © 5 *Respironics* software. See Appendix D for standard definitions for sleep macrostructure measurements.

FFT analysis. Traditional analyses of EEG activity have evaluated data on the time domain, but data can also be converted to the frequency domain (Zappulla, 1991). While data evaluated on the time domain examines variations in amplitude as a function of time, digital computers have made it possible to extract and quantify this information in terms of frequency, amplitude and phase (Pivik *et al.,*, 1993). Quantitative EEG (qEEG) provides a method to quantify features of the EEG that have usually been scored visually according to general sleep staging criteria (Zappulla, 1991). A unique advantage of qEEG is the ability to quantify features of the EEG that are not observable from visual inspection of the traditional time-domain record (Zappulla, 1991). Fast Fourier Transform (FFT) or spectral analysis has become a common way of analysing EEG data in addition to the more conventional sleep staging methods that are reliant on visual scoring. Specifically, FFT uses computerised technologies and software to analyse the average EEG power spectrum generated by the different wave-forms in a specified time-frame of EEG recording (Chen, & Black, 2005). In this pilot study's FFT analyses, the data trends for all three groups were computed to a 6-second window of time throughout the entire study. The EEG in each frequency band, i.e. Delta (0.5-4Hz), Theta (4-7Hz), Alpha (8-12Hz) and Sigma (13-15z), is quantified according to the root-mean square average amplitude within that band (Pivik *et al*., 1993). The FFT analysis trend then provides the relative power[17] of the constituent frequencies of the EEG channels over a 6-second window.

In this pilot study we are only interested in the relative power of Alpha activity for REM sleep. Accordingly, after the EEG was quantified the amount of Alpha activity in REM sleep for the two consecutive nights in the sleep lab was averaged for each participant and then subsequently averaged across each of the 3 groups: dreamers, non-dreamers and recovered-dreamers. Average differences in these groups during REM may indicate

[17] Relative power is a measure of the quantity of EEG activity in a frequency band divided by the amount in all bands (Pivik *et al*., 1993).

differences in sleep consolidation as well as the appearance of more Alpha in the non-dreamers, which would be in line with the hypothesis of the main study, indicating disturbed sleep.

Statistical Tests. Descriptive statistics for all three groups were conducted for measures of REM sleep quality and quantity as well as general sleep efficiency. Pilot data was described in terms of relevant theory and what could be expected in the planned main study based on this data. The pilot study will look at effect sizes by conducting univariate analyses of variance (ANOVA) for the multiple measures of REM sleep quality and quantity, as there is a dearth in current literature. However these will be interpreted with caution for sample size calculations.

Procedure

The same procedure was used for the non-dreaming, dreaming and recovered dreaming patients. Patients were informed of the main purpose of the study and the procedures involved. In addition, each patient was told that they are free to withdraw from the study at any stage, without consequence, should he or she wish to. Informed consent and permission from each participant and attending physicians was obtained respectively before any data was collected (Appendix B). The data was collected in the sleep laboratory at Gatesville Medical Centre, Cape Town. Permission for conducting this study was obtained from the institution.

Sleep study. The sleep study took place over two consecutive nights at the Gatesville Medical Centre, Cape Town. Patients were restricted in the use of caffeine-containing liquids and other stimulants. The first night served as an orientation night and a confirmation of basic sleep/dream activity. The second night functioned as the experimental night. Patients were connected to a polysomnograph for both nights and asked to sleep as they would normally at home. During both nights the patients were monitored by the principal researcher and a qualified sleep laboratory nurse.

First night. Nocturnal REM-sleep interviews were conducted on the first night to confirm the presence or absence of dreaming. Interviews were comprised of brief questions with regards to whether or not patients were dreaming and what was going on in their minds prior to awakening. Polysomnography was used to confirm that patients in both groups were

experiencing REM sleep cycles. Nocturnal interviews using the REM awakening method were kept standard in order to avoid any experimenter bias as the interviewer was not blind to the status of patients as a dreamer or non-dreamer.

Second night. Patients were not awaked by the researcher during the night. Polysomnographic recordings were used to measure the quality and quantity of sleep. In the morning, patients were debriefed and asked whether they feel as though their quality of sleep in the sleep laboratory was similar to their quality of sleep at home. Patients were thanked for their participation in the study and received compensation in accordance with the participation agreement (see Appendix B).

Results

Comparison of Non-dreaming, Dreaming and Recovered-dreaming Groups

An analysis of all the general sleep parameters was beyond the scope of this project, but most were nonetheless included in the calculations of the REM quality, REM quantity and general sleep efficiency measures, and were accordingly included in the Table 1 and Table 2 for the sake of completeness. Weighted averages of multiple measures of general sleep efficiency, as well as REM quality and quantity were compared for two consecutive nights in the sleep laboratory, as well as the average of both nights for non-dreaming, dreaming and recovered-dreaming patients. Figure 3 indicates the sampling and flow of participants through the pilot study.

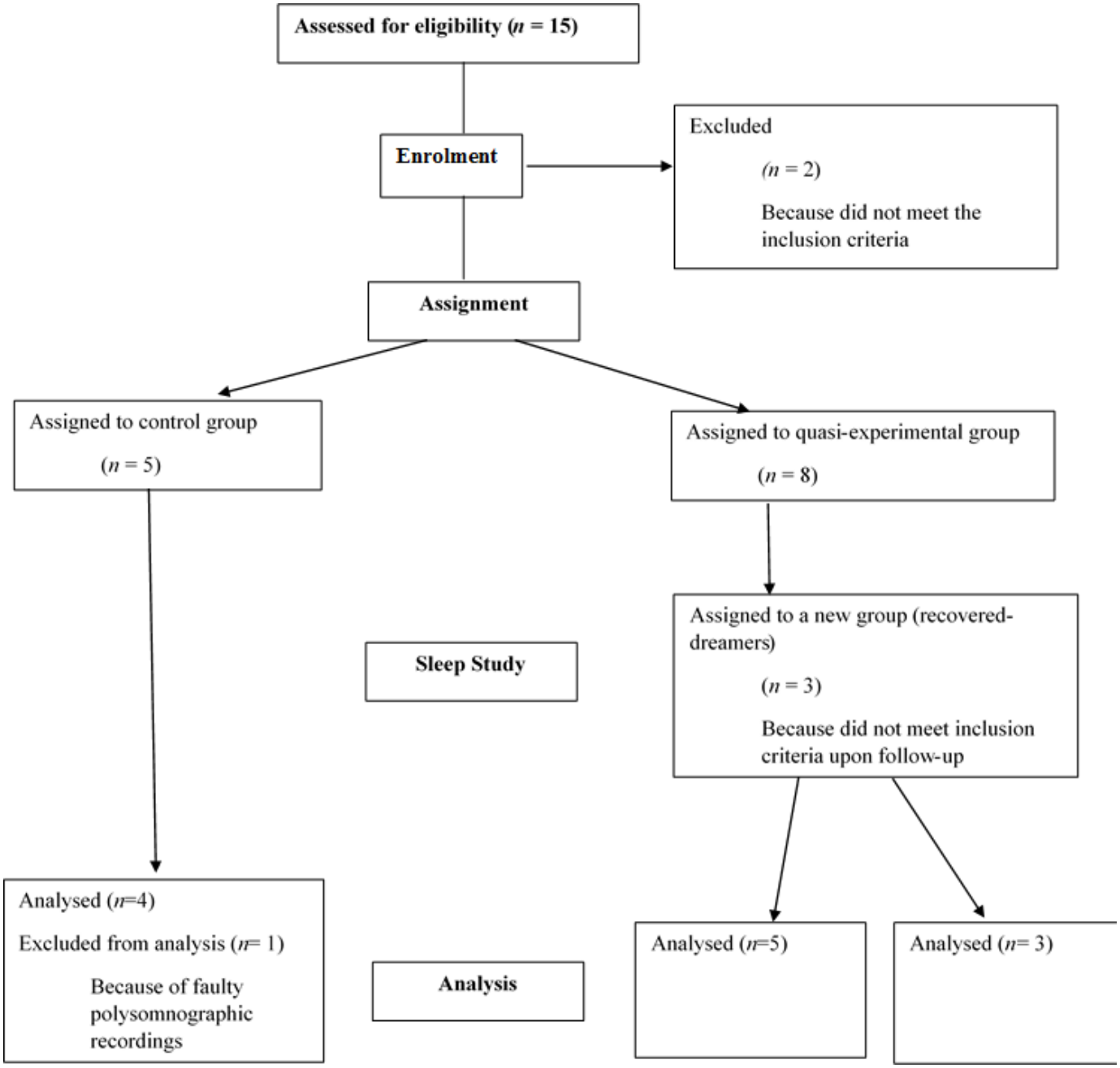

Figure 3. Participant flow chart. Fifteen participants were chosen. Two participants were later excluded due to the incorrect neuropathology for this study while an additional participant was subsequently excluded from the analyses due to faulty polysomnographic recordings.

Table 1

Sleep Quantities: Comparison of Non-dreaming, Dreaming and Recovered-dreaming Means

		Non-Dreamers	Dreamers	Recovered-Dreamers
TST	Night 1	230.80	332.12	280.33
	Night 2	380.00	381.75	325.50
	Average of Both Nights	305.40	356.94	302.92
SPT	Night 1	353.80	476.25	366.83
	Night 2	443.80	473.13	422.33
	Average of Both Nights	398.8	474.69	394.58
TIB	Night 1	443.40	504.25	427.33
	Night 2	490.60	516.25	473.33
	Average of Both Nights	467.00	510.25	450.33
REM Duration	Night 1	24.80	52.13	31.00
	Night 2	*49.00*	*74.50*	*46.67*
	Average of Both Nights	36.90	63.31	38.83

Note: Non-Dreamers (n=5); Dreamers (n=4); Recovered-Dreamers (n=3).

TST= Total Sleep Time: Total number of minutes spent in sleep. Calculated: R+ N1+ N2+N3

SPT= Sleep Period Time: Sleep Onset -> Last Sleep Page (measured in minutes)

TIB= Time in Bed: Lights off -> Lights on (measured in minutes)

REM Duration: defined as the total number of minutes spent in Rapid Eye Movement sleep.

Table 2

Sleep Events: Comparison of Non-dreaming, Dreaming and Recovered-dreaming Means

		Non-Dreamers	Dreamers	Recovered-Dreamers
Alpha	Night 1	33.20	47.00	27.33
	Night 2	138.40	103.50	47.33
	Average of Both Nights	85.80	75.25	37.33
Awakenings	Night 1	22.00	17.25	22.00
	Night 2	27.60	21.00	21.67
	Average of Both Nights	24.80	19.13	21.83
Arousals	Night 1	140.00	82.00	173.67
	Night 2	158.20	73.67	140.67
	Average of Both Nights	149.10	77.83	157.17
Micro-Arousals	Night 1	6.20	15.00	17.00
	Night 2	13.80	18.75	18.33
	Average of Both Nights	10.00	16.87	17.67
REMs	Night 1	65.60	282.75	92.00
	Night 2	304.40	316.75	206.67
	Average of Both Nights	185.00	299.75	149.33

Note: Non-Dreamers (n=5); Dreamers (n=4); Recovered-Dreamers (n=3).

REMs= Rapid Eye Movements: total number of rapid eye movement events in sleep.

General Sleep Efficiency

The efficiency of general sleep was analysed according to subjective reports of sleep quality for all three groups with the global PSQI as well as for electroencephalographic measures of sleep quantity and quality (Table 3).

Pittsburgh Sleep Quality Index. The PSQI did not reveal any striking differences between dreamers (*M* = 7.25, *SD* =1.71) and non-dreamers (*M* = 7.80, *SD* =2.59). However it was interesting to note that, on average, recovered-dreamers (*M* = 5.67, *SD* =4.62) reported slightly fewer difficulties overall than non-dreaming and dreaming patients.

Sleep Efficiency Index. On the first night in the sleep laboratory, dreamers and recovered-dreamers spent a substantial 15% and 14% longer time as a percentage of time from lights off to lights on in sleep, respectively, compared to non-dreamers (Figure 3). That said, there were no striking differences between the three groups for the second night. On average over both nights, dreamers (*M* = 70.45, *SD* =10.98) spent marginally more time as a percentage of time from lights off to lights on in sleep compared to non-dreamers (*M* = 62.63, *SD* =15.38); while there were no prominent differences between recovered-dreamers (*M* = 67.25, *SD* =11.93) and non-dreamers (Table 3).

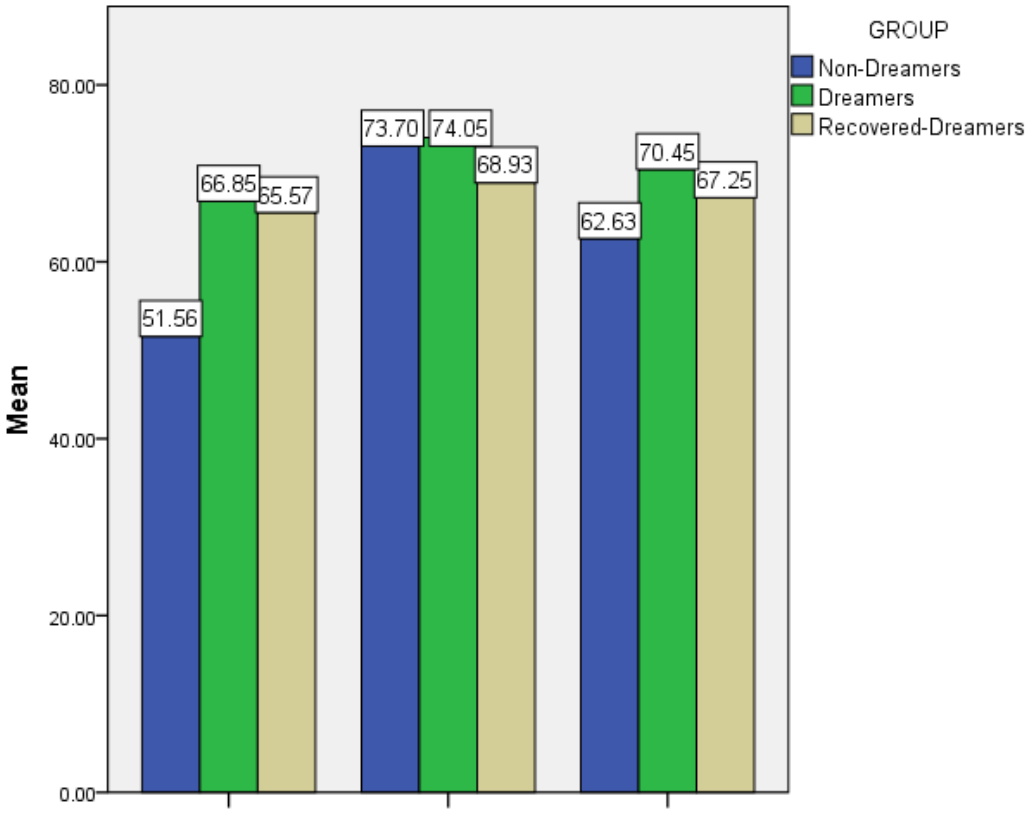

Figure 3. Sleep Efficiency Index

Sleep Onset Latency. Analysis of sleep onset latency (Table 6), indicated that on average over both nights the dreamers (M = 23.62, SD = 6.32) spent a substantially shorter amount of time trying to fall asleep in comparison to non-dreaming (M = 62.70, SD = 49.02) and recovered-dreaming (M = 55.67, SD = 23.48) patients. However when each night was looked at individually, on the first night in the sleep laboratory, the dreaming patients spent a fourth of the amount of time trying to fall asleep in comparison to non-dreamers and a third of the amount of time till sleep onset compared to recovered-dreamers (Figure 4) . While the difference in sleep onset latency between the three groups for the second night in the sleep laboratory was less distinct. In addition, the recovered-dreamers took longer than the non-dreamers to enter Stage 1 sleep for night 2.

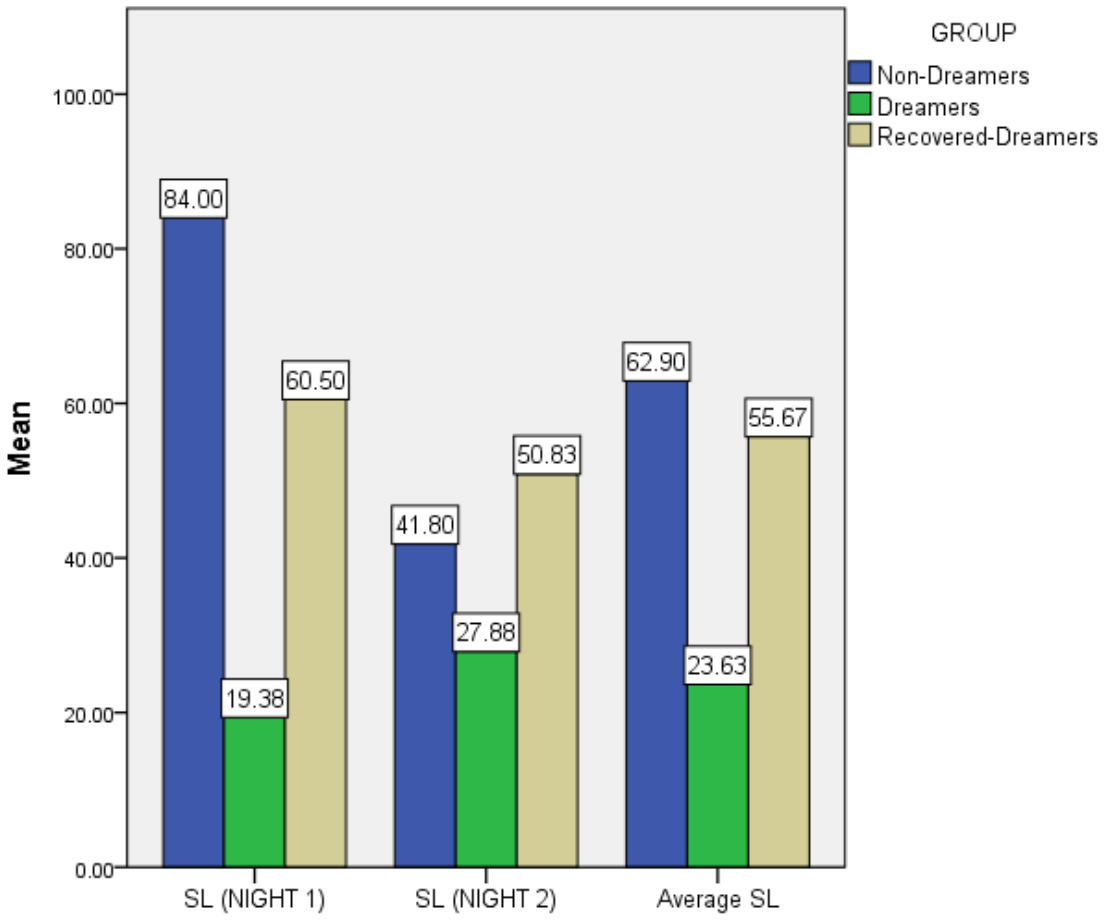

Figure 4. Sleep Onset Latency

Table 3.

Sleep Efficiency: Comparison of Non-dreaming, Dreaming and Recovered-dreaming Means

		Non-Dreamers	Dreamers	Recovered-Dreamers
PSQI		7.80	7.20	5.70
SE	Night 1	51.56	66.85	65.57
	Night 2	73.70	74.05	68.93
	Average of Both Nights	62.63	70.45	67.25
SL	Night 1	84.00	19.37	60.50
	Night 2	41.80	27.87	50.83
	Average of Both Nights	62.90	23.62	55.67

Note: Non-Dreamers (n=5); Dreamers (n=4); Recovered-Dreamers (n=3).

Note: The global PSQI score is a summation of seven component scores that each have a possible range of 0-3. That said the global PSQI score ranges from 0-21with a score of '0' indicating no difficulty and a score of '21' indicative of severe difficulties in all areas.

SE= Sleep Efficiency: Percentage of time spent in sleep from lights off to lights on. Calculated as a percentage of TST/ TIB (Time in Bed).

SL= Sleep Onset Latency = Total number of minutes it takes from lights out to sleep onset.

REM Quantity

REM as a percentage of sleep period time. Analysis of the percentage of sleep period time spent in REM sleep (Figure 5) indicated that on average dreamers spent 5% more time in REM sleep than non-dreamers for the average of both nights (M = 13.20 , SD = 3.31 and M = 8.02 , SD = 4.81 respectively) and for each night independently (Table 4). There was only a marginal difference between recovered-dreamers and non-dreamers for the percentage of sleep period time (SPT) spent in REM for both nights on average (M = 9.77, SD = 3.40 and M = 8.02, SD = 4.81 respectively) and for each night taken separately.

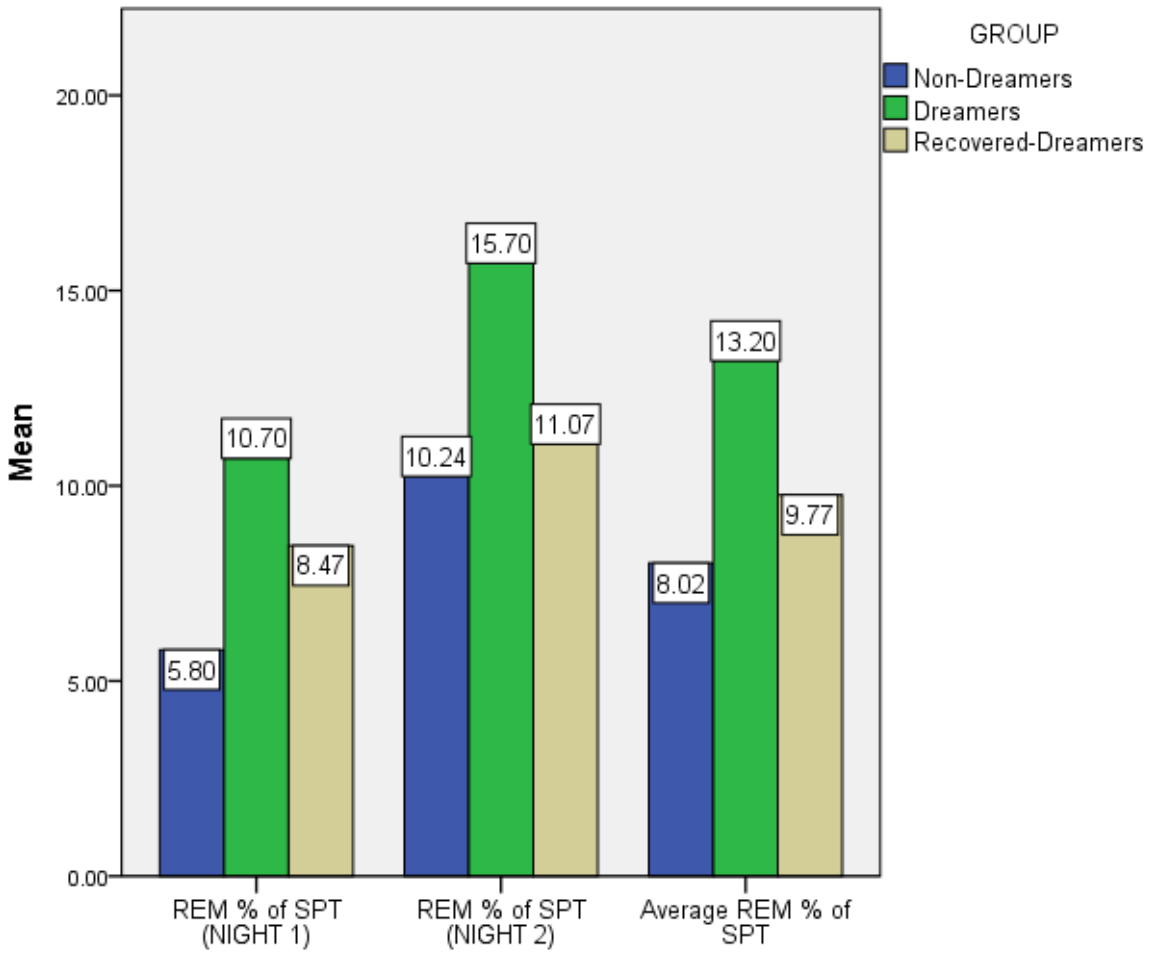

Figure 5. REM percentage of Sleep Period Time (SPT)

Longest REM period. Analysis of the longest REM period (Table 4) revealed large differences between dreamers (M = 29.50, SD = 13.28) and non-dreamers (M = 9.20, SD = 8.34) for the first night in the sleep laboratory whereas the differences were not as distinct for the second night (M = 34.62, SD = 5.71 and M = 23.90, SD = 12.86, respectively). On average over both nights, dreamers' longest REM period was twice as long as the duration of the non-dreamers' longest REM period (Figure 6). There was no striking difference between non-dreamers (M = 16.55, SD =8.51) and recovered-dreamers (M = 18.17, SD = 3.82) on average for both nights and for each night taken independently.

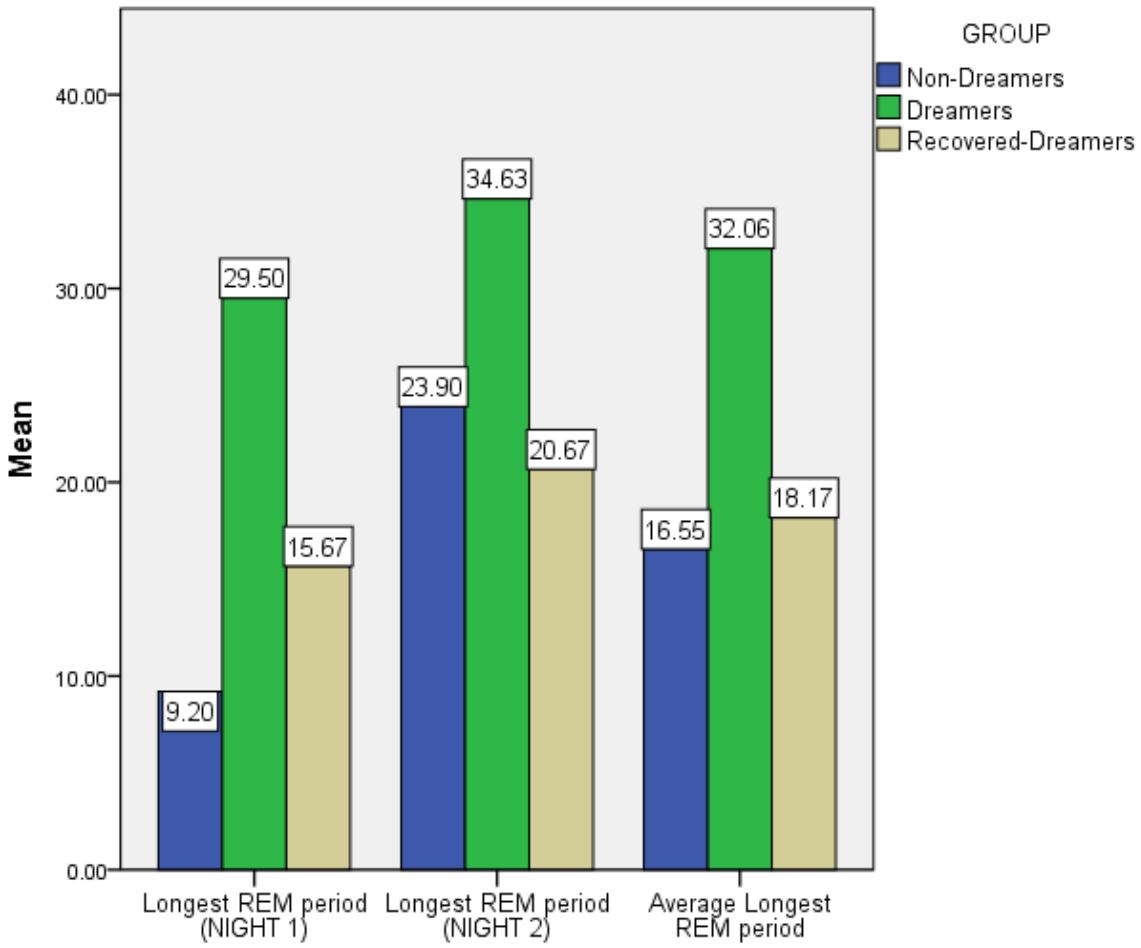

Figure 6. Longest REM period (in minutes)

Table 4.

REM Quantity: Comparison of Non-dreaming, Dreaming and Recovered-dreaming Means

		Non-Dreamers	Dreamers	Recovered-Dreamers
REM % SPT	Night 1	5.80	10.70	8.47
	Night 2	10.24	15.70	11.07
	Average of Both Nights	8.02	13.20	9.77
Longest REM period	Night 1	9.20	29.50	15.67
	Night 2	23.90	34.62	20.67
	Average of Both Nights	16.55	32.06	18.17

Note: Non-Dreamers (n=5); Dreamers (n=4); Recovered-Dreamers (n=3).

REM % SPT: defined as the total time in minutes spent in REM sleep from sleep onset.

REM % TST: defined as a percentage of the total time spent in REM sleep from the total time in minutes spent in Stages R+ N1+ N2+N3.

Longest REM period: defined as the total number of minutes of the longest REM cycle.

REM Quality

% Alpha. Analysis of the percentage of Alpha activity in REM sleep (Table 5) indicated that there were no striking differences between the dreaming (M = 1.27, SD = 1.48), recovered-dreaming (M = 1.48, SD = 2.20) and non-dreaming (M = 1.88, SD = 1.58) patients for both nights on average, as well as for the first night in the sleep laboratory. Whereas for the second night, the was a fairly larger percentage of Alpha activity in REM sleep for non-dreamers compare to dreaming and recovered-dreaming patients respectively (Figure 7).

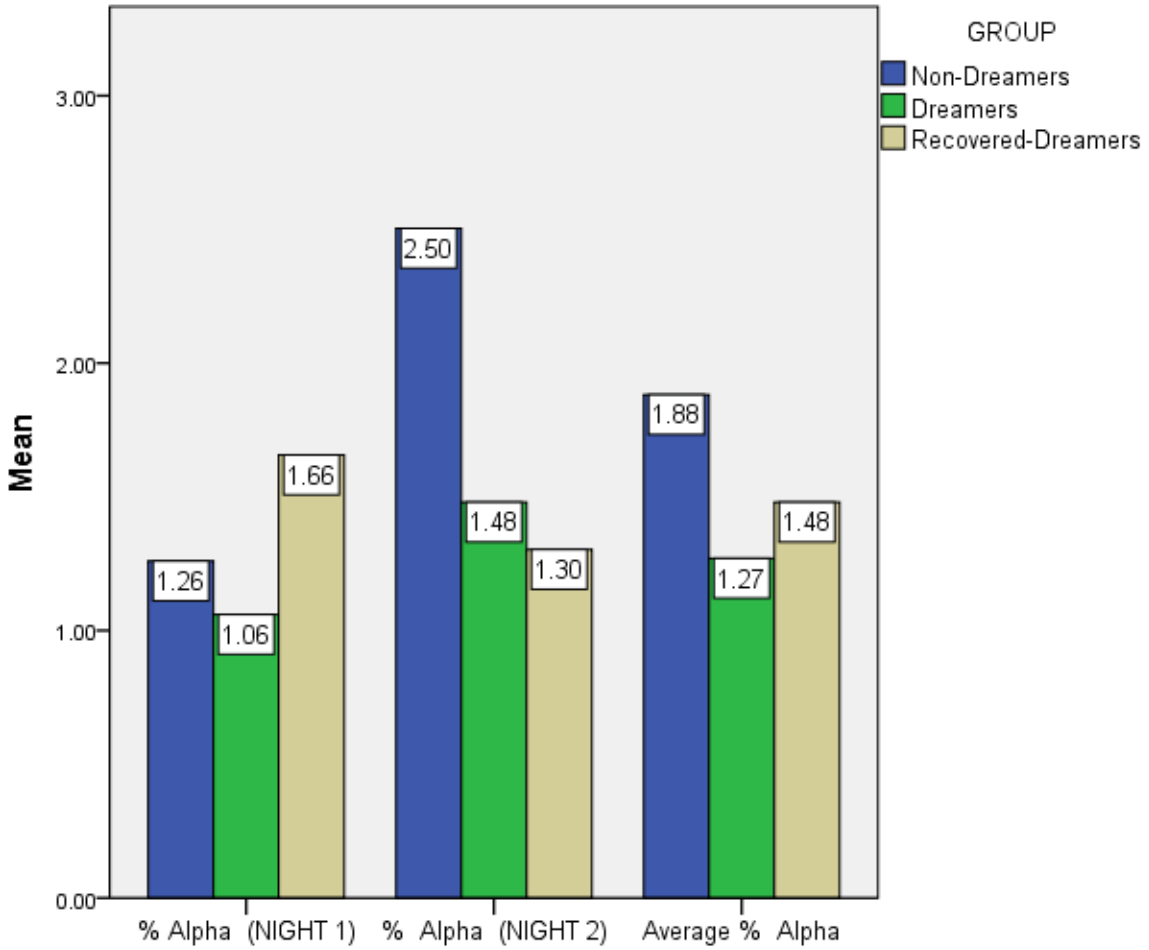

Figure 7. Percentage of Alpha activity in REM sleep

Micro-arousal Index. On the first night in the sleep laboratory, there was a large difference in the average number of micro-arousals per hour of REM sleep with the micro-arousal index for non-dreamers (M = 28.06) being twice the size of that of the dreaming (M =14.20) patients (Table 5). What is interesting to note is that recovered-dreamers presented with a higher micro-arousal index than non-dreamers (Figure 8). The difference between the latter two groups for the second night and for both nights on average was analogous to the first night- with recovered-dreamers having a slightly greater micro-arousal index than non-dreamers. Conversely, dreaming patients (M = 10.74, SD = 5.92) showed a substantially lower index of micro-arousals than non-dreaming (M = 24.09, SD = 25.84) and recovered-dreaming (M = 27.00, SD = 25.23) patients, respectively, for both nights on average (Table 5).

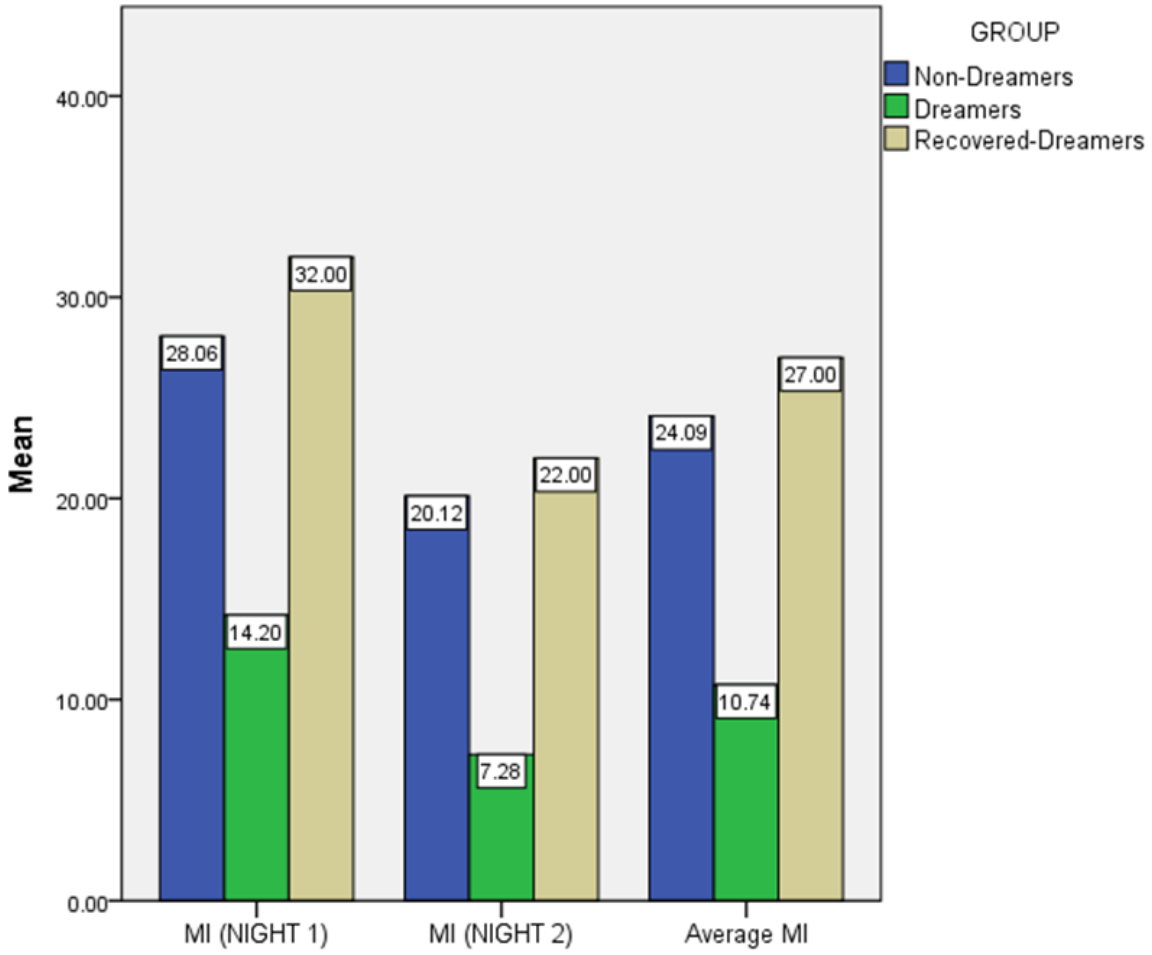

Figure 8. Micro-arousal Index in REM sleep

REM Density. Analysis of the average frequency of eye movements during REM sleep indicated substantially higher REM density for dreaming patients (M = 5.40) compared to non-dreaming patients (M = 2.90) for the first night in the sleep laboratory (Table 5). Conversely, on the second night non-dreamers had superior REM density to dreamers (Figure 9). That said, recovered-dreamers had a marginally superior REM density to non-dreamers for both nights on average and for each night separately. Furthermore, on average for both nights (Table 5) dreaming patients (M = 4.67, SD = 2.42) showed greater REM density than non-dreaming patients (M = 3.96, SD = 3.50).

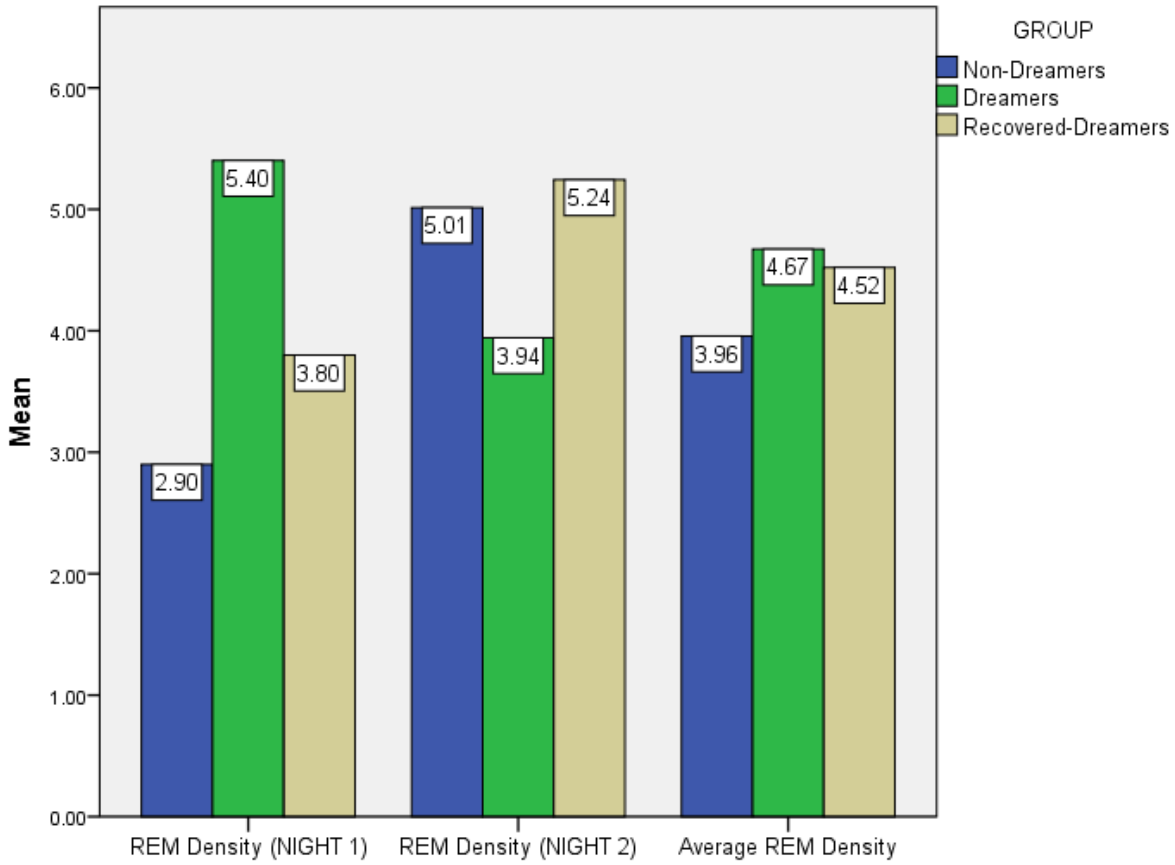

Figure 9. REM Density

Table 5.

REM Quality: Comparison of Non-dreaming, Dreaming and Recovered-dreaming Means

		Non-Dreamers	Dreamers	Recovered-Dreamers
% Alpha	Night 1	1.26	1.06	1.66
	Night 2	2.50	1.48	1.30
	Average of Both Nights	1.88	1.27	1.48
MI	Night 1	28.06	14.20	32.00
	Night 2	20.12	7.27	22.00
	Average of Both Nights	24.09	10.74	27.00
REM Density	Night 1	2.90	5.40	3.80
	Night 2	5.01	3.94	5.24
	Average of Both Nights	3.96	4.67	4.52

Note: Non-Dreamers (n=5); Dreamers (n=4); Recovered-Dreamers (n=3).

% Alpha: defined as the percentage of Alpha events in REM sleep. Calculated as Alpha/REM.

MI= Micro-arousal Index: defined as the average number of micro-arousals per hour of REM sleep time.

REM Density: defined as the frequency of eye movements during REM sleep. Calculated as REMs/REM Duration.

Analysis of Variance

Observed Effect Sizes. A post hoc power analysis was conducted using the program G*Power3 (Faul, Erdfelder, Lang & Buchner, 2007). A total sample size of 12 patients was used with alpha at the recommend 0.05 (Cohen, 1988) to calculate the achieved effect sizes from the means of a one-way analysis of variance (ANOVA) for the three groups: dreamers, non-dreamers and recovered-dreamers (Table 6). Cohen (1988) defines *f* statistics of 0.1, 0.25, and 0.4 as small, medium, and large effects, respectively.

Table 6.

Post hoc Power Analysis: Compute Achieved Effect Size from Means

		Observed Effect Size Cohen's f statistic
General Sleep Efficiency Measures	PSQI	0.30
	Sleep Efficiency Index	0.27
	Sleep Onset Latency	0.48
REM Quantity Measures	REM % SPT	0.51
	Longest REM Period	0.69
REM Quality Measures	% Alpha in REM	0.17
	Micro-arousal Index	0.34
	REM Density	0.11

Sample Size. A well designed pilot study may be used to generate information for sample size calculations (Arain et al, 2010; Osborne, 2003; Lenth, 2001; Leon, Davis & Kraemer, 2011; Shanyinde, Pickering & Weatherall, 2011; Thabane et al, 2010). However, it is important to note that these sample size estimates are based on preliminary findings from the pilot study and must be interpreted with caution. Accordingly, this study acknowledges the uncertainty surrounding estimates of effect sizes and required sample sizes for future main studies and for this reason a sample size table and graph of various values of the effect sizes are provided above (Table 7 and Figure 10). A power analysis using the G*Power3 computer program (Faul, Erdfelder, Lang & Buchner, 2007) indicated that a total sample of roughly 18 to 429 patients (6 and 143 patients per group) would be required to obtain a large (Cohen's $f = 0.8$) to small (Cohen's $f = 0.15$) effect size, respectively, using a univariate analysis of variance with alpha at 0.05 and statistical power at the recommended 0.80 (Cohen, 1988; Table 7).

Table 7.

Same size of various values of effect sizes

	α error probability = 0.05
Effect Size	Total Sample Size
0.10	966
0.15	431
0.20	244
0.25	157
0.30	110
0.35	82
0.40	63
0.45	51
0.50	42
0.55	35
0.60	30
0.65	26
0.70	23
0.75	20
0.80	18

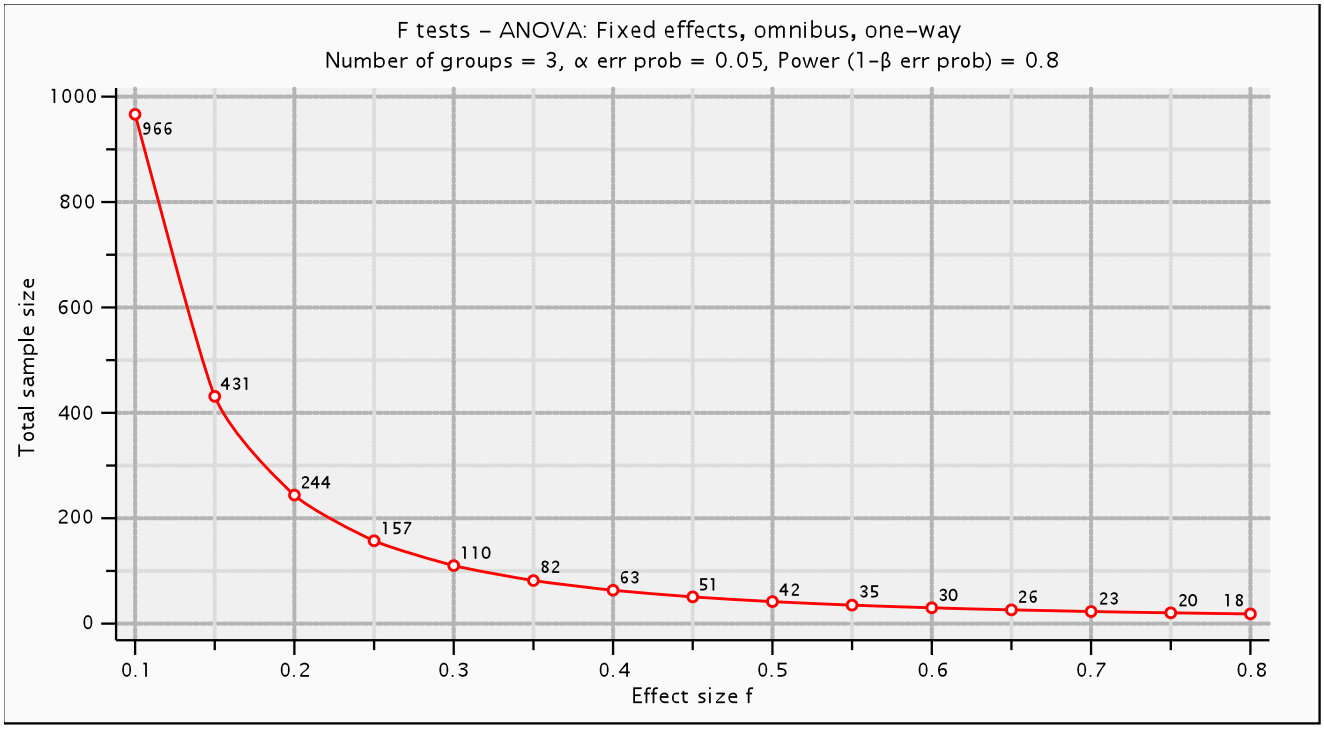

Figure 10. Recommended sample size for detecting a range of fixed effect sizes for a one-way omnibus ANOVA.

These preliminary findings give clear evidence that reduced REM quality and REM quantity were experienced by non-dreaming patients in comparison to dreaming patients. Whereas the relationship between non-dreamers and recovered-dreamers was not always clear. Although it remains undecided as to what the minimal and maximum amount of sleep interruptions characterizing disturbed sleep are (Bosselli, Parrino, Smerieri & Terzano, 1998), these findings are important as we can deduce that non-dreamers and dreamers certainly differed from each other. The comparison of general sleep efficiency between non-dreamers, dreamers and recovered dreamers did give evidence which directly supports previous findings that a reduced quality and quantity of general sleep is experienced by patients who have ceased to dream (Bischof & Bassetti, 2004; Poza & Massó, 2006; Solms, 1997). The importance of these findings in terms of the main hypothesis of this study, which claims that the lost ability to dream is associated with REM sleep quality and quantity, is discussed next.

Discussion

Despite decades of dream research, a physiological function of dreaming has not been empirically established. This pilot study hoped to address the significant gap by describing preliminary data in terms of the Freudian dream theory. In order to achieve this, this study also assessed the feasibility of conducting a future main study with regards to three broad areas: processes, resources and the scientific basis of a planned main study. These findings are discussed in the following sections of this paper.

Comparison of Non-dreaming, Dreaming and Recovered-dreaming Participants

General Sleep Efficiency.

Subjective sleep quality. Individual reports of subjective sleep quality were considered together with polysomnographic reports in a comparison of non-dreaming, recovered-dreaming and dreaming patients. However, in this pilot study, the PSQI did not reveal any differences on subjective sleep quality between the three groups. This is interesting to note, as it is demonstrates that in the face of vast objective differences in sleep architecture (discussed below), the groups did not show any dissimilarities on their subjective experience of sleep quality.

Reduced sleep efficiency index in non-dreamers. Dreamers were able to sleep more efficiently on the first night in the sleep laboratory, with a substantial 15 % larger sleep efficiency index, compared to non-dreamers. While this discrepancy was not seen on the second night, it is worth noting that a potential factor mediating the altered sleep architecture seen in the second night is the lack of sleep experienced by the non-dreamers from the first night. Sleep deprivation has been reported to result in sleep rebound (Chokroverty, 2009). In this study, patients' sleep architecture was studied for two consecutive nights. The first night intended to orientate patients to the sleep laboratory while the second night was designed to obtain a more accurate reading. Because of this, future research should consider studying patients for more than two consecutive nights to avoid the potential effects of sleep rebound.

A potential argument could be proposed that the lack of dreaming itself affects the ability to adapt to an unfamiliar sleep setting (Cameron-Dow, 2012). However, the fact that non-dreamers were unable to effectively adapt to the sleep laboratory environment due to cessation of dreaming is merely a tentative conclusion which requires further investigation to establish validity for this hypothesis.

That said other factors that have been shown to affect sleep efficiency need to be considered. The most noteworthy of these are studies demonstrating that stoke affects sleep (Bakken et al., 2011; Gottselig et al, 2002; Herman et al, 2003). A study by Bassetti and Aldrich (2001) indicated that reduced total sleep time and lowered sleep efficiency may be related to the impact that a stroke has on sleep architecture. That said, while stroke has been reported to affect sleep efficiency, this effect has mostly been shown in male patients (Bakken et al., 2011). As three of five non-dreamers were female, the argument that a cessation of dreaming may be related to disturbed sleep architecture (which is not completely explained by the effect of a stroke) is given further support.

Longer sleep onset latencies in non-dreamers. Dreamers were able to fall asleep in a normal amount of time (less than 30 minutes) while recovered-dreamers and non-dreamers spent roughly 60 minutes or more trying to fall asleep. This may be due to the tentative argument made previously, that non-dreamers may be taking longer to enter sleep due to a difficulty adapting to a new environment.

REM sleep quantity.

REM amounts in non-dreamers. Firstly, the pilot study was able to demonstrate reduced REM amounts in non-dreaming patients compare to normal or near-normal REM amounts in dreaming patients. As REM sleep typically accounts for 20-25% of total sleep time in healthy adults (Chokroverty, 2009), dreamers demonstrated near normal REM amounts with REM accounting 18.02 % of total sleep time (TST) on average of both nights, in comparison to only 10.57% for non-dreamers and 12.47% for recovered-dreamers.

Shorter REM cycles in non-dreamers. The pilot study revealed large differences in the longest mean REM period between dreamers and non-dreamers. On average over both nights, dreaming patients' longest REM period was twice as long as the duration of the non-dreaming patients' longest REM period. Average differences in the longest REM period between dreaming and non-dreaming groups may indicate differences in REM sleep maintenance as well as the appearance of more Alpha in the non-dreamers, which would be in line with the hypothesis, indicating disturbed sleep. There were no striking difference between non-dreamers and recovered-dreamers on average for both nights and for each night taken independently. It is worth noting, however, that sleep may still be disturbed in recovered-dreamers (albeit not as disturbed as non-dreamers) due to the fact that recovered-

dreamers may have reduced dream quality in comparison to dreamers. Further investigation is required to establish why non-dreamers and recovered-dreamers appear unable to sustain REM sleep. Can REM sleep not be maintained as successfully without dreaming? The trends observed in the pilot data suggest that this may be the case. Added to this, is the fact that the biggest effect sizes were observed in the pilot data for the average longest REM period. However future research is needed to establish validity for this hypothesis.

REM sleep quality.

Increased Alpha amounts in non-dreamers. Due to the limited flexibility of the data analysis software (Alice Sleepware © 5 *Respironics*) and data recording hardware (Alice © 5 *Respironics* polygraphic amplifier) used in the pilot study, only the overall Alpha power band was given for REM sleep and not the frequency- and topographic- specific Alpha activity of dream recall from both tonic and phasic REM sleep. In this pilot study, Alpha activity was shown to be marginally higher for non-dreamers compared to dreamers and recovered-dreamers respectively; with the exception of the first night in the sleep laboratory where the latter group had a superior Alpha percentage for REM sleep compare to non-dreamers. This study recommends that the data be recorded and analysed in the planned main study in such a way that Alpha activity can be assessed for occipital derivations separately. For the reason that Alpha activity suppressed over EEG occipital derivations has been shown to be positively correlated with successful dream recall in REM sleep (Bertolo et al., 2003; Hong et al., 1996) and in particular tonic REM fragments (Cantero, Atienza & Salas, 2000).

Reduced REM density in non-dreamers. This pilot study demonstrated that the frequency of eye movements during REM sleep was substantially higher for dreaming patients compare to non-dreaming patients for the first night in the sleep laboratory. Recovered-dreamers also demonstrated greater REM density than non-dreamers, but the difference was marginal. Conversely, on the second night non-dreamers had superior REM density to dreamers. However, a possible confounding factor of these results is the very low number of rapid eye movements that the non-dreamers documented on the first night in the sleep laboratory (*mean* = 65.60 in comparison to *mean* = 282.75 in dreaming patients). Thus, it is possible that the sleep architecture displayed by non-dreaming patients on the second night is a direct result of deficient rapid eye movements on the first night. This is reasonable,

given that sleep deprivation has been shown to be related to REM rebound[18] (Chokroverty, 2009).

Increased micro-arousal index in non-dreamers. Pilot data demonstrated that dreaming patients showed a substantially lower index of micro-arousals than non-dreaming and recovered-dreaming patients respectively. This indicated that the latter two groups experienced an increase of sleep disruptions on average for both nights and for the first and second night respectively. A comparison of arousals[19] for total sleep time (TST) between the three groups also revealed substantial differences in the pilot study (see Table 2). However, the EEG reports generated by Alice Sleepware © 5 *Respironics* software only provided the micro-arousal index for stage REM and not the arousal or awakening index for REM sleep. Although the pilot data only shows trends that are in line with this theory for the average number of arousals per hour of TST, it is reasonable to propose that similar trends will be observed for REM sleep. The argument can be made that the inability to sustain REM sleep may be due to more arousals occurring in this stage of sleep. For that reason, it is recommended that alternative software be used for the future planned study so that arousals can be analysed as a function of stage REM.

Feasibility

Processes.

Exclusion and inclusion criteria. The posterior cortical lesion site chosen for the pilot study was the territory of the posterior cerebral artery (PCA). For the reason that thrombotic infarctions in the PCA territory create more circumscribed damage. Based on the parietal cases studied by Solms (1997), it was predicted that the occurrence of patients with posterior cortical lesions were not extremely rare. Occipital lobe damage was also considered preferable for this study as a global cessation of dreaming has been shown in cases following a unilateral left tempero-occipital hematoma (Poza & Massó, 2006) as well as a bilateral occipital stroke (Bischof & Bassetti, 2004). Accordingly, parietal involvement was not considered a critical aspect of the lesion site, but occipital lobe damage was. Therefore the sample size of the pilot study was extremely dependent on the availability of patients referred

[18] REM rebound refers to the increased frequency and depth of REM sleep which has been shown to be a result of sleep deprivation (Chokroverty, 2009).

[19] In this pilot study, arousals were characterized as arousal phenomena occurring between 15 and 60 seconds. Arousal index is defined as the average number of arousals per hour of sleep time.

by neurological specialists at Gatesville Medic Centre, Cape Town, who met the required inclusion criteria. The fact that the planned main study's sample size will be decidedly dependent on the availability of patients with such restrictive inclusion criteria calls for reassessment of the inclusion and exclusion criteria. The middle cerebral artery (MCA) territory is the most common site in a cerebral infarction, due to the size of the territory and the direct flow from internal carotid artery into the MCA, providing the easiest path for thromboembolism (O'Sullivan & Schmitz, 2007). For this reason, it is suggested that inclusion and exclusion criteria be adjusted to include MCA stroke patients who have corresponding posterior lesions as well.

Polysomnographic recording methods. Topographically, Alpha waves display the greatest amplitude over posterior regions and in particular posterior occipital regions (Spriggs, 2002). Increased activity (Armitage et al., 1989) and reduced Alpha activity (Esposito et al., 2004) reported in REM sleep provides further evidence that REM-Alpha waveforms may be related to dream recall. Furthermore, Alpha activity suppression over specific brain areas has been typically interpreted as an activation index of those cortical regions involved in the information processing of an specific sensory modality, both in active wakefulness (Kaufman, Glanzer, Cycowicz &Williamson, 1989; Pfurtscheller & Neuper, 1992; Schupp et al., 1994) and mental imagery (Kaufman, Schwartz, Salustri & Williamson, 1990; Davidson & Schwartz, 1977). More interestingly, in a study by Cantero et al. (2000) in which the individual contribution of occipital alpha power in tonic[20] and phasic REM fragments were assessed, two variants of alpha activity were reported to have distinct functional roles during human REM sleep. The first variant, background Alpha (suppressed over occipital EEG derivations when rapid eye movements were present) may be related to the visual imagery in dreams. The second variant, bursts of spontaneous Alpha activity (which showed the same spectral features in tonic and phasic[21] REM fragments) may be functioning as a micro-arousal during REM sleep in order to facilitate a connection between this physiological state and the external world (Cantero et al., 2000).Whereas longer REM-alpha arousals (which are typically longer in duration and are complemented by changes in

[20] Tonic refers to events which are continuous and typically occur during REM sleep (Spriggs, 2002).

[21] Phasic refers to a brief event occurring during sleep (Spriggs, 2002).

the electromyogram[22] (EMG) amplitude) would engender a state shift and sleep fragmentation (Cantero & Atienza, 2000).

Accordingly, it is reasonable to expect that high occipital Alpha power may be negatively correlated with the relative degree of brain activation over occipital EEG sites during REM sleep as formerly demonstrated by other measures, such as cerebral blood flow, which are amplified in waking and REM sleep (Madsen & Vorstrup, 1991; Sakai, Meyer, Karacan, Derman, & Yamamoto, 1980). For this reason, it is suggested that the future main study investigate frequency- and topography-specific Alpha activity of dream recall from tonic and phasic REM sleep. Although Alpha activity is largely seen in the occipital regions, both central and occipital placements are recommended in order to maximize the detection of Alpha activity (Broughton, 1987). In addition, bilateral central and occipital electrodes are recommended for the main study main study in the event that an electrode becomes non-functional during the night (Broughton, 1987). It is suggested that sleep stages be visually scored per 20-s epochs according to standard criteria (Rechtschaffen & Kales, 1968). EEG artefacts can be detected by an automated artefact algorithm or alternatively upon visual inspection.

Electroencephalographic data analysis methods. Spectral analysis ought to be conducted using a Fast Fourier transformation (FFT; 10% cosine 4-s window) to yield a 0.25 Hertz bin. The Hanning (cosine) window is recommended to avoid "leakage" of spurious frequencies occurring as a result of abrupt changes in EEG signals at the beginning and the end of the EEG segments. Values above 25 Hertz should not be included in the analysis. It is suggested that REM sleep be expressed as the percentage of total sleep time per night before averaging over subjects. Alpha power spectra must be calculated during REM sleep in the frequency range from 7.5 to 13 Hertz. While the absolute power of Alpha activity was not available for analyses in the pilot study, it is recommended that the absolute power be used instead of the relative power for the main study. The reason for this, is that absolute power yields data that is more interpretable (Pivik et al., 1993). Finally, artefact free 4-s epochs are to be averaged over 20-s epochs. Furthermore, it is suggested that 20-s segments from six REM periods for each participant be quantified using spectral analysis.

[22] Recording of electrical activity produced by a muscle (Spriggs, 2002).

Resources.

Hardware and Software. The hardware used in the pilot study (Alice © 5 *Respironics* polygraphic amplifier) to capture the polysomnographic (PSG) recordings was not compatible with other software that provided the necessary functions for more advanced quantitative EEG (qEEG) analyses. The Alice Sleepware © 5 *Respironics* software used in the pilot study to compile the EEG data into PSG reports could not perform spectral analysis on selected epochs for REM sleep. Consequently, analysis of measures of REM sleep quality were limited to the data provided in the reports. As such, it is suggested that the future main study use a polysomnographic amplifier that will provide flexibility and increase ease of use and recording power for all types of EEG and polysomnographic recordings

Scientific.

Effect sizes. Analysis of the observed effect sizes (Table 6) suggests that measures of REM quantity differed substantially between the three groups. Specifically, REM as a percentage of sleep period time (SPT) as well as the average longest REM period indicated large effect sizes. One could argue that the loss of dreaming results in the inability to sustain REM sleep, hence the shorter REM periods and the inferior amount of time spent in REM sleep in comparison to dreaming patients. Therefore, further research is warranted to assess these REM quantity variables in relation to dream loss.

Sample Size. Due to the small sample size in this pilot study, tests of significance were not appropriate. Therefore, the statistical significance of these differences still needs to be established in a powered-sample size in the planned main study. Nonetheless, a comparison of the mean values of non-dreamers, dreamers and recovered-dreamers that has been done in this pilot study gives a clear indication that support for the sleep protection hypothesis may be found with a larger sample size. The following sections of this thesis will discuss the hypothesis that dreams maintain sleep in more detail.

Dreams Protect Sleep

If we return to the literature on the neural correlates of dreaming, two distinct brain regions emerged as being fundamentally involved in dream generation: the posterior cortical regions, in particular the occipital lobes, and the white matter of the ventro-mesial quadrant of the frontal lobes. Furthermore, Panksepp (1998) identified the latter region, the frontal mesial limbic system, as being the neurological system that drives behaviour in waking life.

Based on the dream model proposed by Solms (2000), it was suggested that patients with occipital damage will be unable to redirect the volitional urges arising from elevated activation of the dopaminergic MC-ML system to the posterior- cerebral- artery territory as this region was damaged. Because of this, and because these urges would normally drive one to actively take part in SEEKING behaviour (Panksepp, 1998), the logical conclusion is that non-dreaming patients showed disrupted sleep because of a failure to redirect these urges and consequently be unable to visuo-spatially construct a virtual seeking experience in the form of dreams (Solms, 2000).

Implications of these Findings

While there are distinct limitations of this pilot study, the implications of these preliminary findings for the scientific search for a physiological function of dreams is two-fold. One, despite recent developments in finding the electrophysiological correlate of dream recall, no one has yet been able to definitively link these correlates to a function of dreams. This study begins to fill this gap by providing preliminary evidence that a cessation of dreaming is associated with reduced REM sleep quality and quantity. Hence these preliminary findings are descriptively in line with the hypothesis that dreams protect sleep.

Two, since the hypothesis that dreams maintain sleep was originally proposed by Freud (1900), this pilot study has generated tentative support for Freudian dream theory. For the reason that, Freud's dream theory is the bedrock of psychoanalysis, there is immense significance in finding an empirical support for this hypothesis so as to add greater scientific credibility to the field of psychoanalysis. This should be reason to pursue the main study.

Conclusion

The hypothesis that dreams maintain sleep was explored in this pilot study using analysis of subjective sleep quality, structural neuro-imaging, and polysomnographic data in 12 patients with lesions in the PCA territory. Comparisons between the non-dreaming, dreaming, and recovered-dreaming groups revealed reduced REM sleep quantity and quality in non-dreaming patients, as well as increased Alpha activity. While the difference between non-dreamers and recovered dreamers was not as distinct, it may be that the latter group does not have extensive dreaming, or may have reduced dream quality, and therefore may still be prone to sleep disturbances albeit not as pronounced. Hence, trends were evident in the pilot data that supported the hypothesis that dream loss is related to reduced REM quality and quantity. In doing so, this pilot study provides preliminary support for the Freudian dream

theory. Hopefully, the evidence related to the validity and successful implementation of this pilot study will be used in conjunction with the subsequent recommendations to lead to a further main study and so doing establish a firm empirical basis for the function of dreaming.

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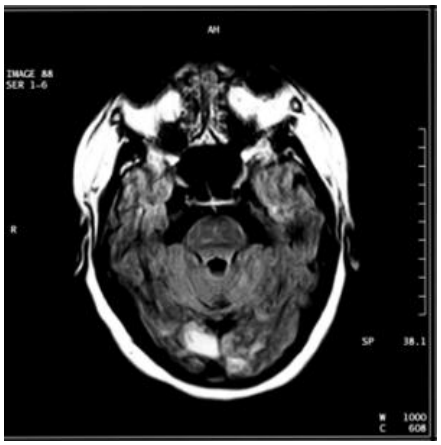
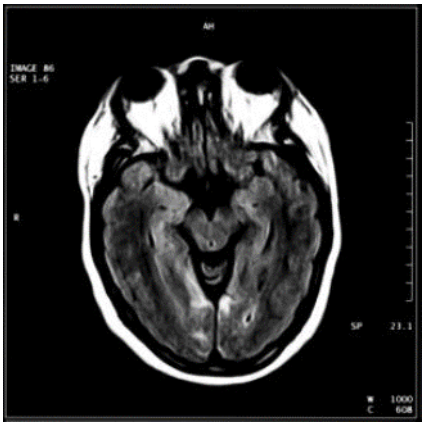
Appendix A: Magnetic Resonance Images (MRI) and Computer Tomography (CT)

Figure 1. Magnetic Resonance Images: Non-dreaming patient (#1)

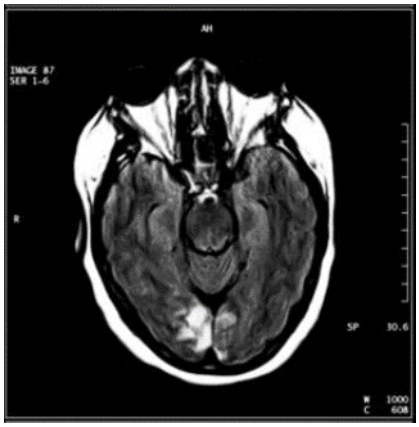
A.



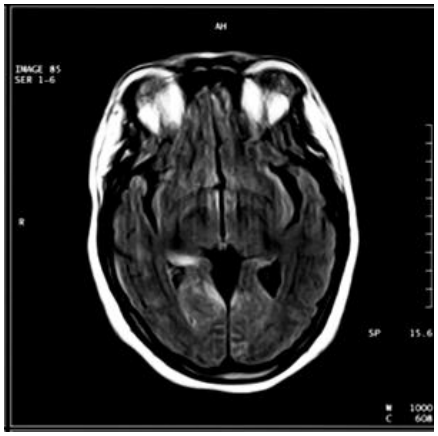
B.



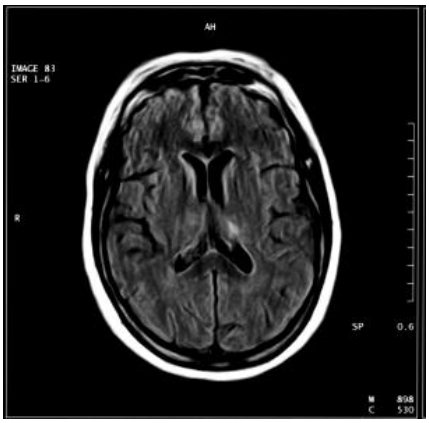
C.



D.



E.



F.

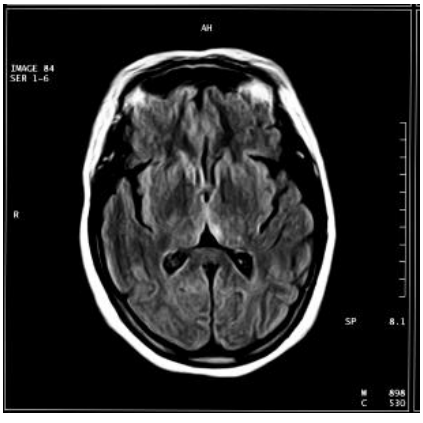
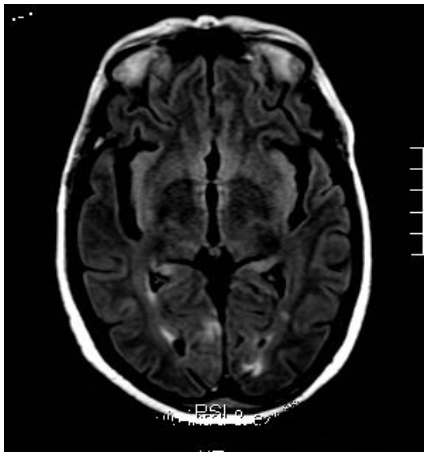
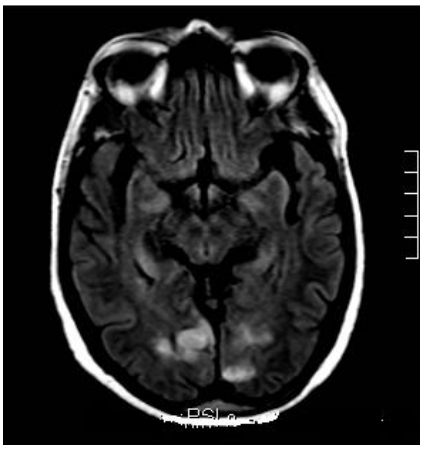


Figure 2. Magnetic Resonance Images: Non-dreaming patient (#2)

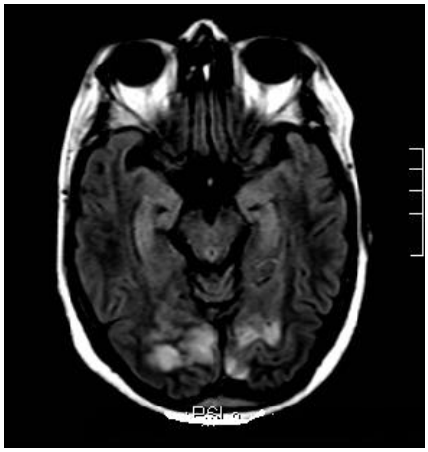
A.

B.

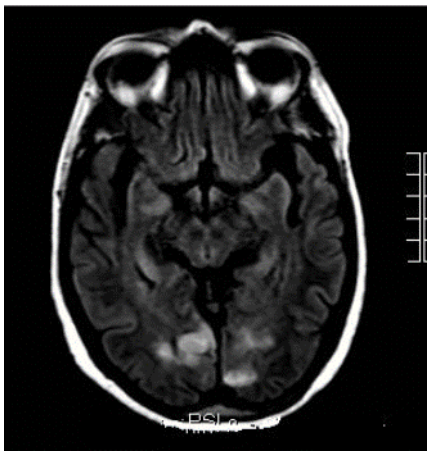




C.



D.



E.

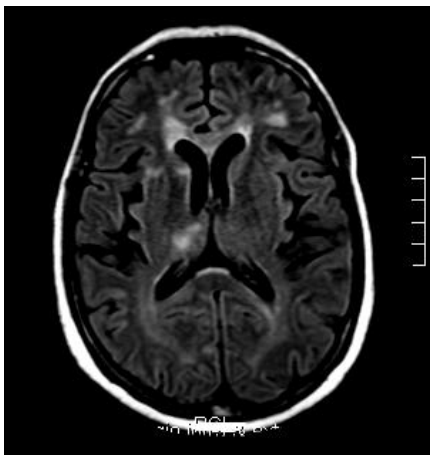
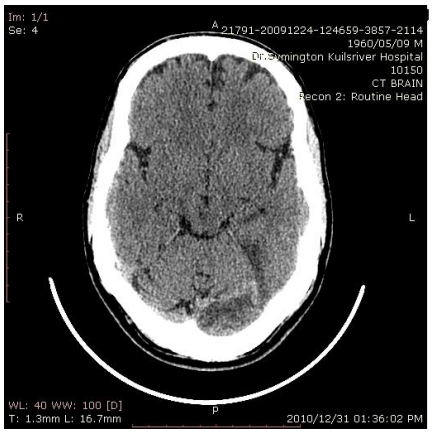
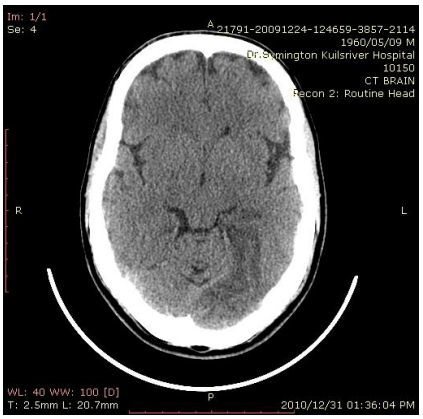


Figure 3. Computer Tomography Images: Dreaming patient (#3)

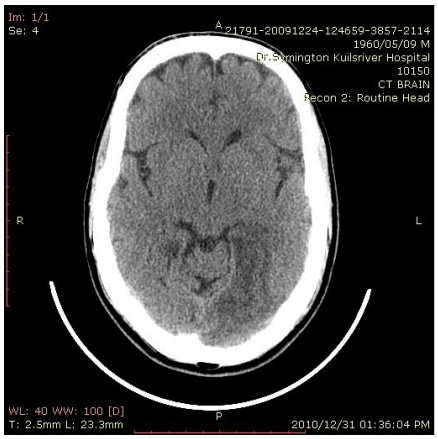
A.



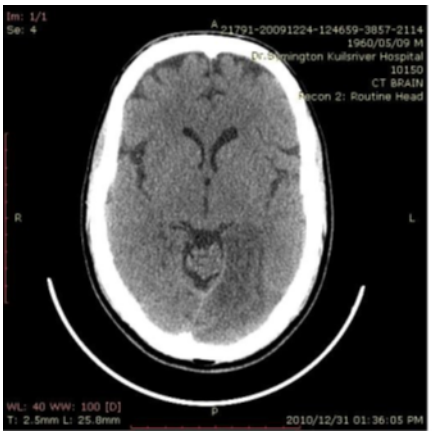
B.



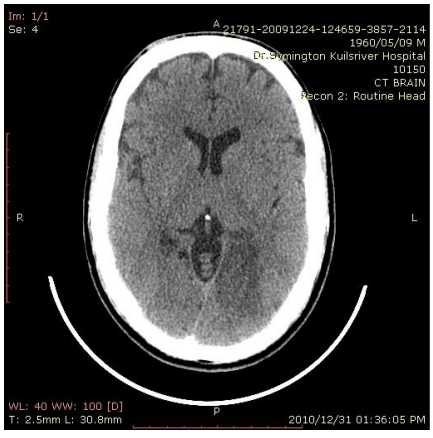
C.



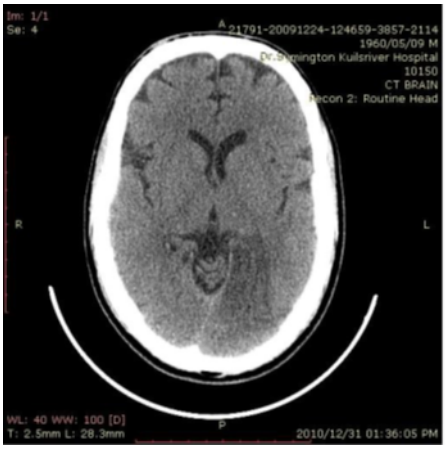
D.



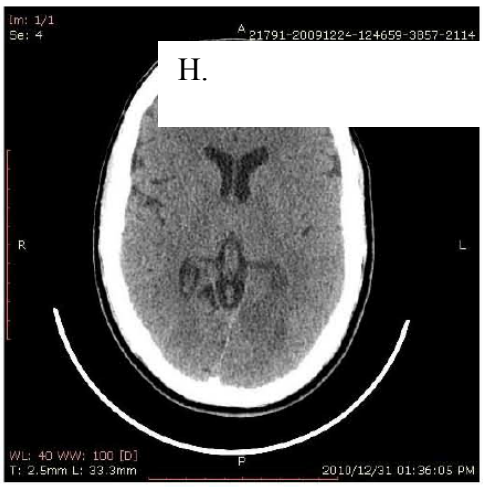
E.



F.



G.


Im: 1/1
Se: 4
A
21791-20091224-124659-3857-2114
R
L
P
WL: 40 WW: 100 [D]
T: 2.5mm L: 33.3mm
2010/12/31 01:36:05 PM

H.

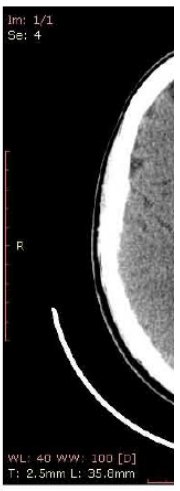
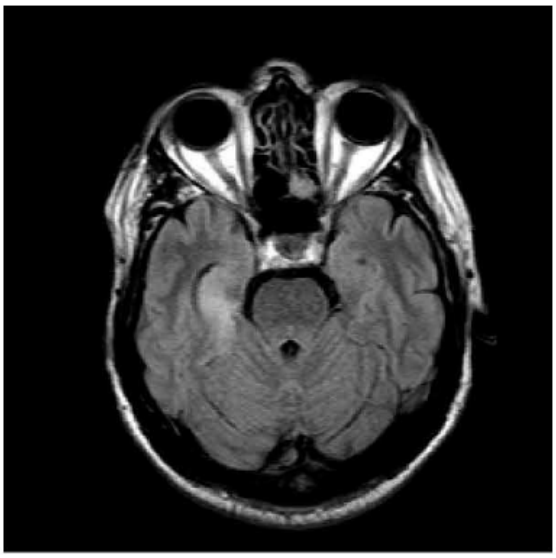
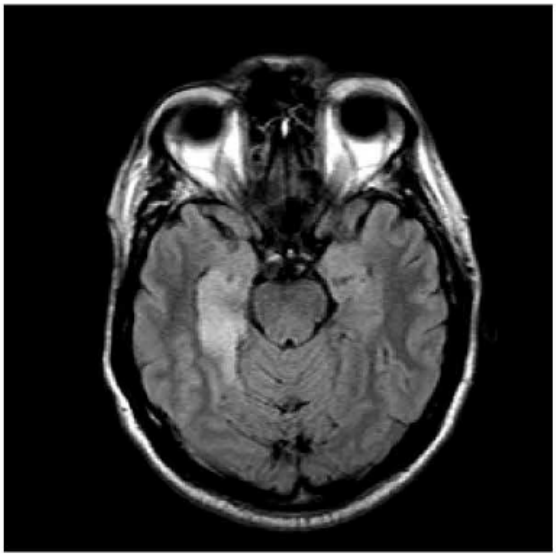

Im: 1/1
Se: 4
R
WL: 40 WW: 100 [D]
T: 2.5mm L: 35.0mm

Figure 4. Magnetic Resonance Images: Dreaming patient (#4)

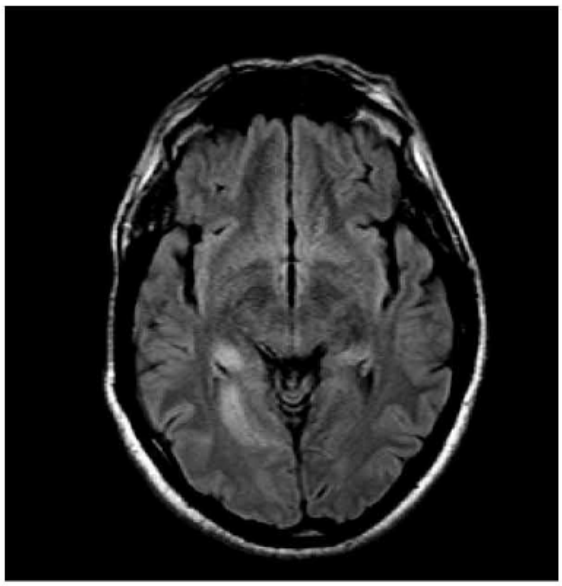
A.



B.



C.



D.

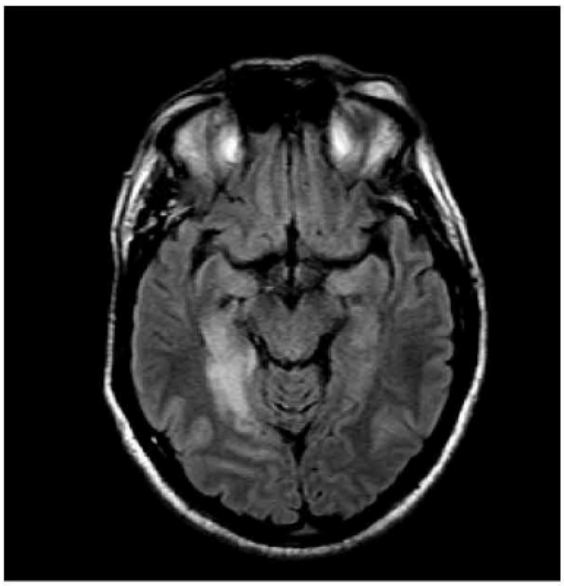
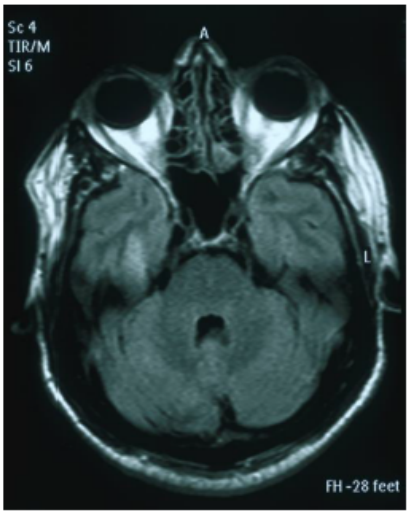
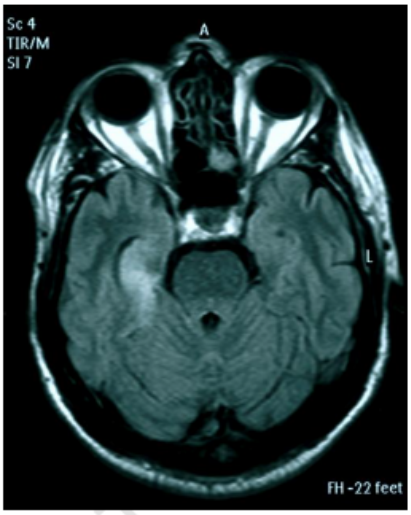


Figure 5. Magnetic Resonance Images: Dreaming patient (#5)

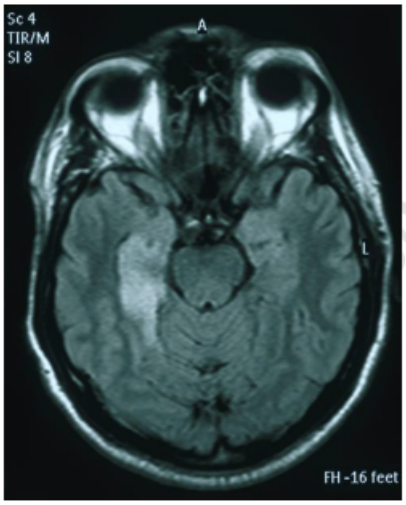
A.



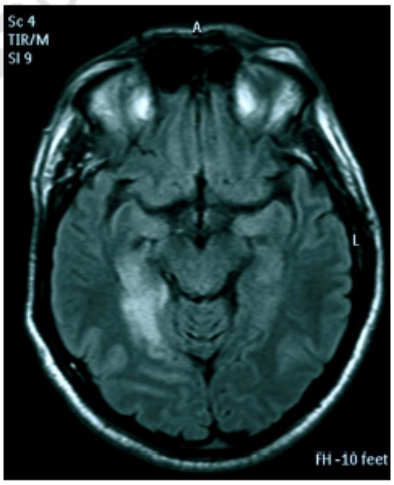
B.



C.



D.



E.

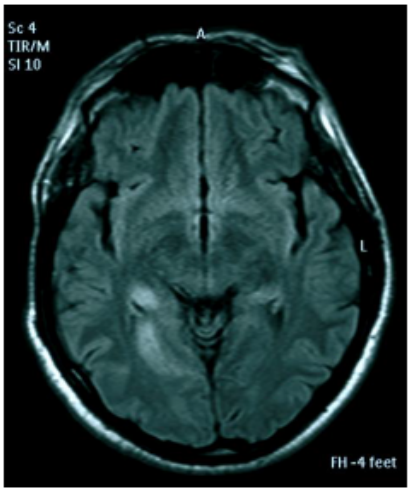
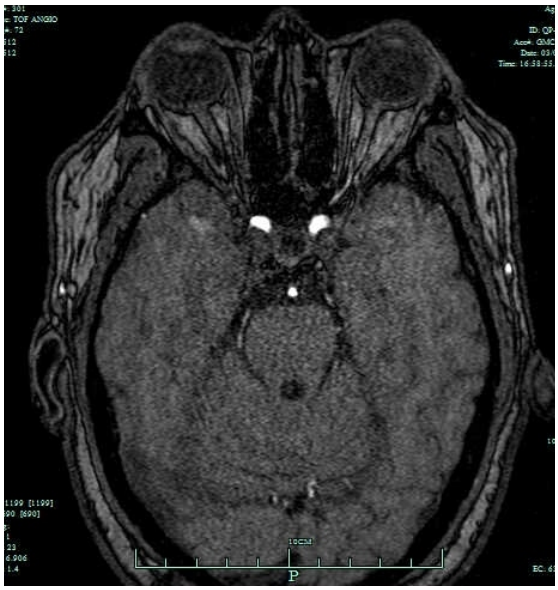
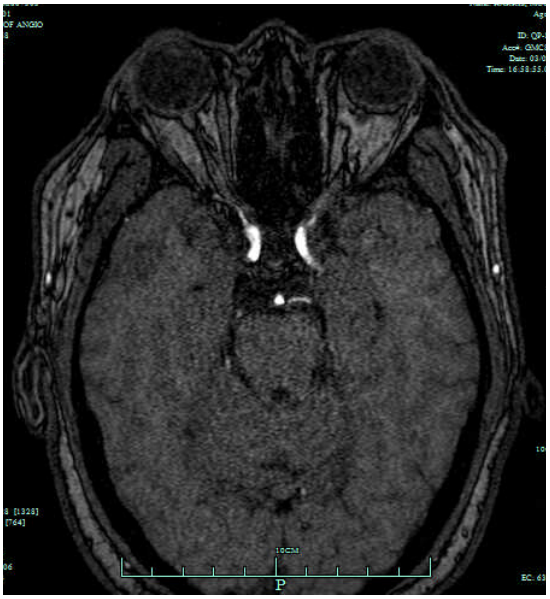


Figure 6. Magnetic Resonance Images: Non-dreaming patient (#6)

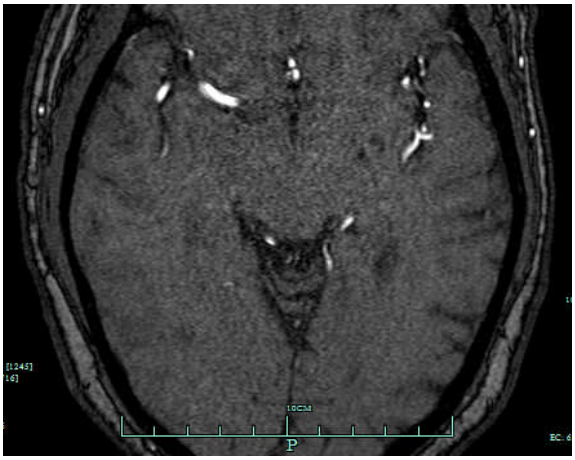
A.



B.



C.



D.

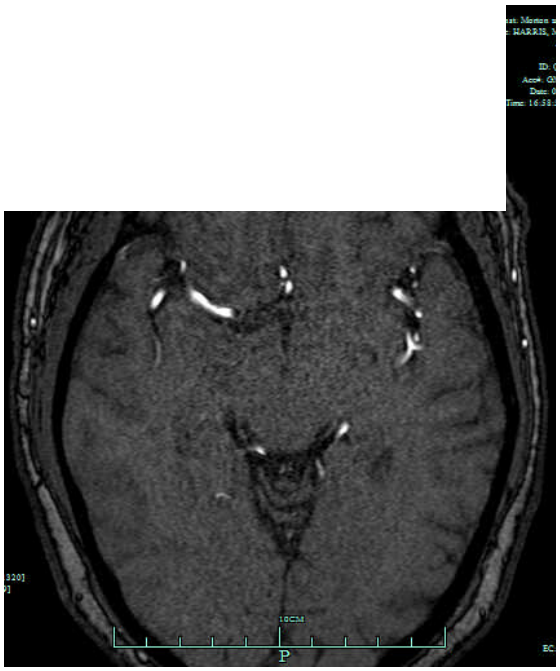
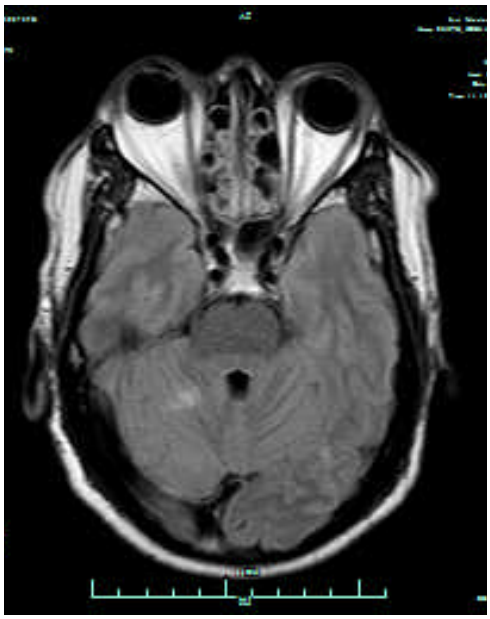
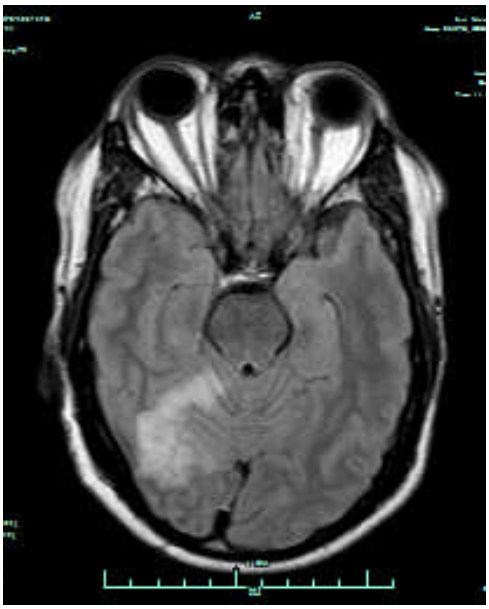


Figure 7. Magnetic Resonance Images: Recovered-dreaming patient (#7)

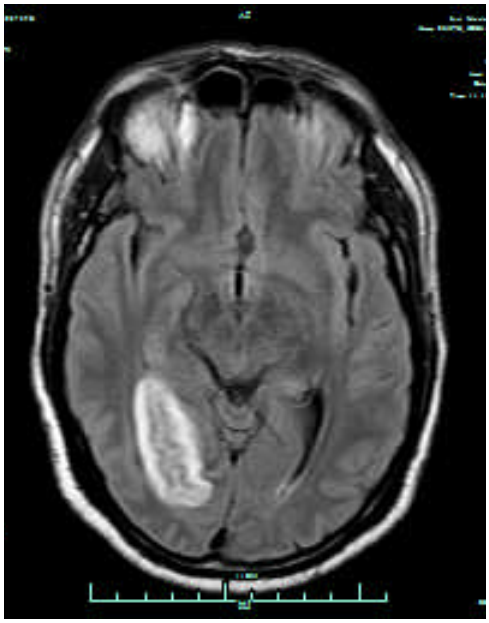
A.



B.



C.



D.

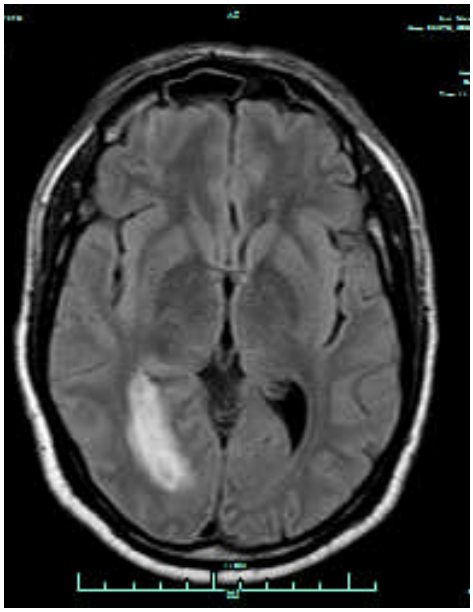
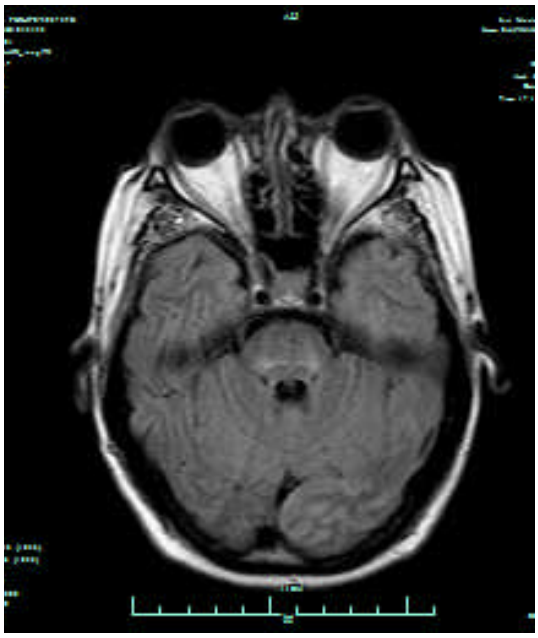
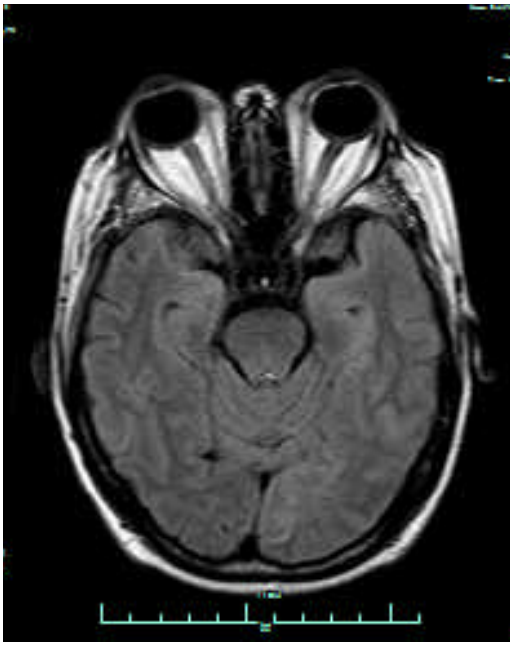


*Figure 8.*Magnetic Resonance Images: Non-dreaming patient (#8)

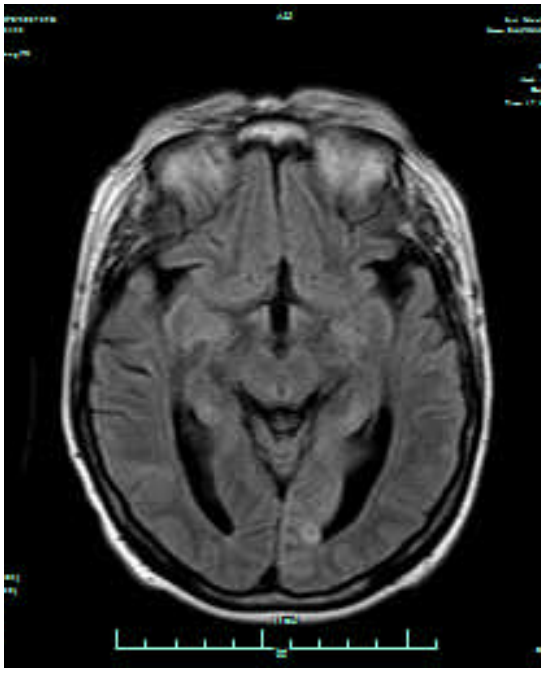
A.



B.



C.



D.

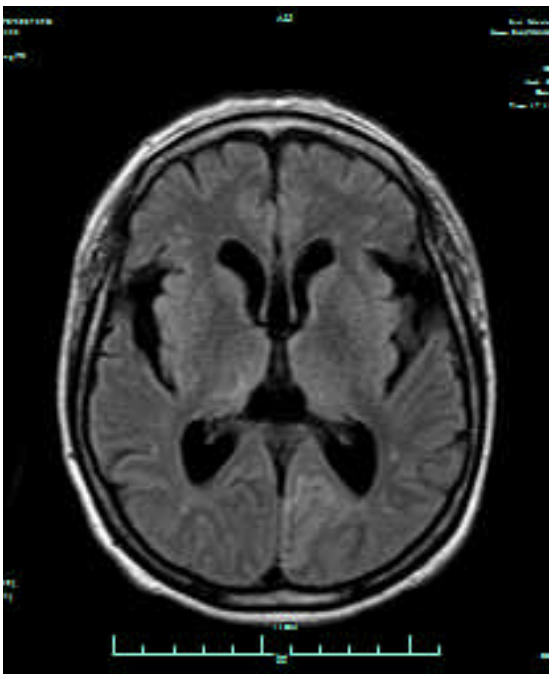
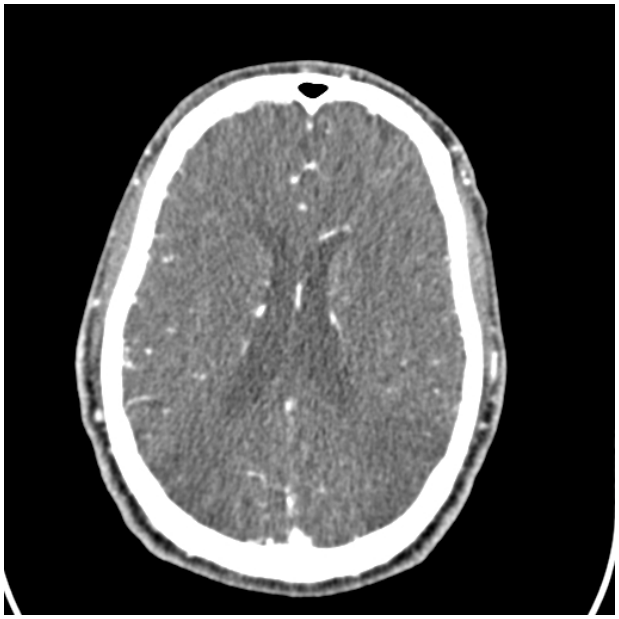
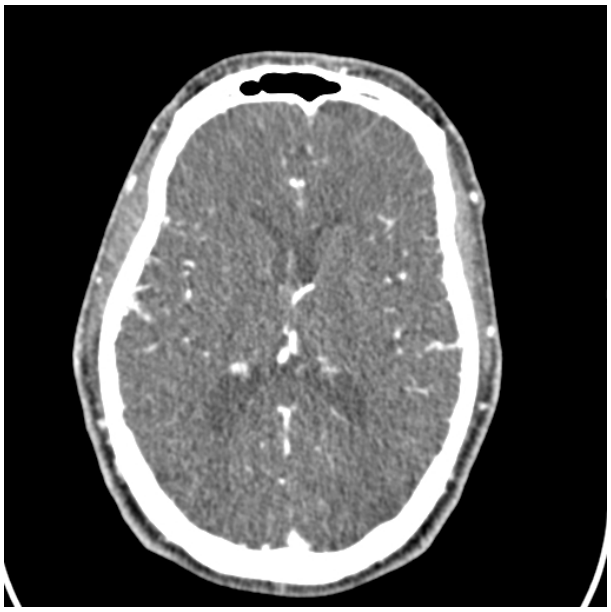


Figure 9. Computer Tomography Images: Non-dreaming patient (#9)

A.

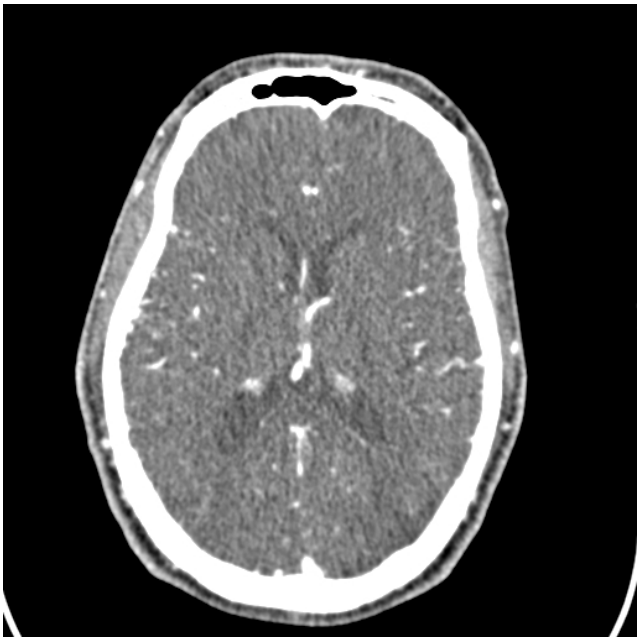


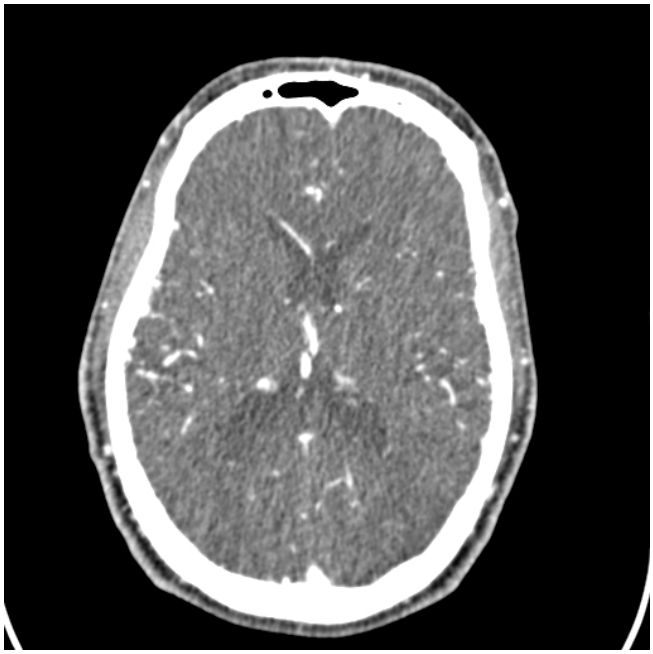
B.



C.

D.





Appendix B: Informed Consent Form

Title of research study: Do Dreams Protect Sleep? Testing the Freudian hypothesis of the function of dreams

Name of principal researcher: Catherine Cameron-Dow
Department/research group address: Psychology Department
Faculty of Humanities
University of Cape Town
Telephone: 021 650 3435
Email: cmrcat004@mail.uct.ac.za

Name of participant:

You are invited to take part in a research study for the Department of Psychology, at the University of Cape Town, in order to see whether suffering a stroke has had an effect on your dreams. Your participation is completely voluntary.

Participant's involvement:

What's involved: The study will involve spending two consecutive nights in a sleep laboratory. You will be connected to a polysomnograph, which is a simple sleep monitoring device that involves small pads being placed on different parts of your body (mainly your face and forehead). You will be asked to sleep as you usually would in your home environment. During the first night, you will be awakened twice by the researcher and asked whether you were dreaming. During the second night,

you will not be awakened, and will just be required to sleep. During both nights, your sleep cycles will be recorded using a polysomnograph.

Risks: There are no risks associated with this study. However, if you feel uncomfortable at any time, for any reason, you may withdraw from the study without any negative consequences for yourself or the study. All data will be kept confidential and will only be used for research purposes.
Benefits: There are no direct benefits for participating in this study, except for monetary compensation (discussed below) and the possibility of detecting any sleep disorders that you may have.

Payment: As you would be giving up a considerable amount of your time, you will be paid R500 for each night that you complete in the sleep laboratory. Thus, if you complete the full two nights of the study you will receive R1000.
Please sign if you have read all the information and you agree to take part in the study.

Signature of Participant: _____

Name of Participant: _____

Signature of principal researcher: _____ (name)

Date: _____