

Dream Function: Exploring the possibility that dreams protect sleep

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ABSTRACT

A review of current literature shows that a physiological function for dreaming has not yet been empirically established. Recent developments have resulted in the identification of the neural correlates of dreaming; however, these correlates are yet to be linked with a function for dreaming. Based on the literature, it is reasonable to propose that dreams may function to protect sleep by diverting the disruptive physiological arousal events that naturally occur during sleep. To test this hypothesis, a single-case of dream loss due to bilateral ischemic infarction in the occipital lobes was studied using polysomnographic recordings. The quality and quantity of the patient's sleep was analysed over two nights in the sleep laboratory in order to verify whether her sleep was significantly more disturbed than the established norms. As predicted, results showed that sleep was significantly more disturbed than the norms, specifically with regard to non-REM sleep stages 1 and 2. The ways in which dreaming may contribute to the protection of sleep are discussed in light of this finding.

Keywords: dreaming; dream loss; bilateral occipital lesions; disturbed sleep; polysomnograph

DREAM FUNCTION: EXPLORING THE POSSIBILITY THAT DREAMS PROTECT SLEEP

There are many theories that propose a physiological function for dreams; however, due to methodological difficulties, no dream theory to date has been empirically proven (Solms & Malcolm-Smith, 2009). How do you study objectively the most subjective of human experiences? Due to a lack of scientific investigation in this field, numerous speculative dream theories currently exist. Some theories claim that dreaming serves an adaptive evolutionary function (Revonsuo, 2000), while others believe that it helps to consolidate emotional memories during sleep (Breger, 1967). A few theories even propose that dreaming serves no function at all (Crick, 1983; Hobson, Pace-Schott & Stickgold, 2000); and there are those who believe that dreaming performs the very fundamental function of protecting sleep (Freud, 1953; Solms, 1997, 2000). However, unproven dream theories divide the field of dream science, rather than contributing to its advancement. It is therefore important that these dream theories be empirically validated. This study hopes to contribute towards uncovering a sound physiological function for dreams by empirically testing whether dreams protect sleep.

Dreaming as an Epiphenomenon of REM Sleep

In the 1950s it was established that the sleep cycle consists of various phases, one of which is rapid eye movement (REM) sleep. Rapid eye movement sleep is characterised by bursts of rapid eye movement, accompanied by specific physiological changes that include a loss of muscle tone (atonia) and cerebral cortical activation similar to that of the waking state (Aserinsky & Kleitman, 1953). Among other things, this discovery acted as an impetus for further dream research, which subsequently showed that dreaming and REM are highly correlated (Aserinsky & Kleitman, 1955; Dement & Kleitman, 1957). As a result, Aserinsky and Kleitman (1953) immediately conflated the two phenomena by stating that dreaming and REM were identical states.

Following this, Hobson & McCarley (1977) further deduced that dreams are merely an epiphenomenon of physiological REM processes, and therefore serve no independent function. This explanatory model of dreaming is referred to as the *activation-synthesis model*, and proposes that the forebrain structures that generate dreams are *activated* by cholinergic brainstem mechanisms (that generate REM sleep), causing meaningless representations (such

as thoughts, feelings and images) to be passively *synthesised*. In this way, the forebrain makes “the best of a bad job in producing even partially coherent dream imagery from the relatively noisy signals sent up from the brainstem” (Hobson & McCarley, 1977, p. 1347). This view of dreaming dominated sleep and dream research for decades. However, the assumption that dreaming and REM sleep are identical, and that both are generated by a single brainstem mechanism, has since been challenged by contemporary neuropsychological evidence (Bishoff & Bassetti, 2004; Poza, & Martí Massó, 2006; Solms, 1997).

The Neural Correlates of Dreaming

In order to understand how dreaming and REM sleep are doubly dissociable, it is firstly necessary to understand the neural mechanisms that have been found to be essential to the generation of dreams. Extensive clinico-anatomical lesion studies by Solms (1997) have identified the parieto-temporo-occipital (PTO) junction and the white matter of the ventro-mesial quadrant of the frontal lobes as being crucially involved in the dream state, as damage to either one of these regions results in a total cessation of dreaming. Furthermore, several neuroimaging studies have confirmed that these regions are highly activated during REM sleep (Braun et al., 1997; Dang-Vu et al., 2005; Maquet et al., 1996; Nofzinger et al., 1997). These studies repeatedly illustrated that the dreaming brain involves an especially specific group of forebrain structures; namely, the dopaminergic mesocortical-mesolimbic (MC-ML) system.

Dreaming is generated by the Dopaminergic Mesocortical-Mesolimbic System

The dopaminergic MC-ML system is defined as the “system [that] is formed by dopamine neurons located in the ventral tegmental area...which project to the nucleus accumbens, prefrontal cortex, septum, amygdale, and hippocampus” (Dahan et al., 2007, p. 1232). Converging lines of evidence indicate that this region is crucial to dream production. Firstly, when the dopaminergic pathway that runs through the MC-ML system is transacted, in the surgical procedure known as *modified prefrontal leucotomy*, cessation of dreaming occurs (Jus et al., 1973; see Solms, 1997, for review). Secondly, L-Dopa—a drug that specifically stimulates dopamine in this region—has been found to intensify the bizarreness, emotionality and vivacity of dreaming, while the REM cycle remains unaltered (Hartmann et al., 1980). Thirdly, a cessation of dream recall has been associated with Parkinson’s disease, which is usually the result of an insidious depletion of dopamine in various forebrain regions (Sandyk, 1997). Fourthly, it has been reported that there is an increase in dopamine release in

humans during REM sleep, within the MC-ML system (Gottesmann, 2004). Finally, recent studies based on the internal measurement of dopamine (through microdialysis and single cell recordings) during the sleep-wake cycle in rats, found a substantial increase in dopamine cell activity and terminal release during REM sleep (Dahan et al., 2007; Lena et al., 2005).

In summary, the overall consensus between the clinico-anatomical studies on the one hand, and the neuroimaging and in vivo studies on the other, suggest that the dopaminergic MC-ML system is responsible for the neurogenesis of dreams.

REM Sleep and Dreaming are Doubly Dissociable States

As previously mentioned, there is a substantial amount of evidence in support of the fact that dreaming and REM sleep are doubly dissociable states. A number of case studies provide evidence for dream cessation occurring while the REM cycle has remained entirely unaltered (Benson & Greenberg, 1969; Bishof & Bassetti, 2004; Brown, 1972; Jus et al., 1973, Poza & Martí Massó, 2006). In other words, the patients ceased to dream while still experiencing entirely normal REM sleep states. Moreover, patients who have suffered lesions to their pontine brainstems, resulting in the elimination of their REM sleep cycles, have still reported experiencing dreams (Solms, 1997).

Despite such evidence, many researchers dispute the fact that dreaming and REM actually are doubly dissociable states, because these two phenomena frequently co-occur. In response to this, Solms (2000) explains that although dreaming is highly *correlated* with REM sleep, this correlation is not due to cholinergic brainstem mechanisms *generating* dreams, but rather because cholinergic activation provides sufficient cerebral stimulation during sleep for dreaming to occur — dreaming can result from any such cerebral activation.

To illustrate this point, Solms (1997, 2000a) draws attention to the occurrence of nightmares during non-REM (NREM) sleep in patients experiencing partial seizures. Furthermore, there is a substantial amount of literature documenting dreaming outside of REM sleep, when cholinergic brainstem mechanisms are silent. Furthermore, numerous studies have documented that some form of dream reports can be obtained from all stages of NREM sleep at rates of up to 50-75% (Foulkes, 1962; Foulkes & Vogel, 1965; Suzuki et al., 2004). Moreover, dream reports from NREM sleep that are *indistinguishable* from those during REM, have been reported between 5-10% (Hobson, 1988) and 10-30% (Monroe et al., 1965, as cited in Rechtschaffen, 1973) of the time. In particular, Foulkes and Vogel (1965) reported that participants awoken at sleep onset and during N1 sleep often report vivid

“dream-like” hallucinatory images with kinaesthetic features, as well as static visual imagery throughout the NREM sleep stages.

Recently, functional neuroimaging results have provided further evidence for dream activity during NREM. For example, Hofle et al. (1997) used positron emission tomography (PET) imaging techniques to investigate the changes in regional cerebral blood flow (rCBF) during the progression from relaxed wakefulness through slow wave sleep (SWS). The changes were examined as a function of spindle and delta EEG activities that progressed during NREM sleep. Hofle et al. reported that delta activity and rCBF covaried positively in the primary and secondary visual cortex and the secondary auditory cortex (BA 22). Indeed, this activation is similar to the activation recorded when subjects are asked to lay with their eyes closed and imagine sounds or images (Hofle et al., 1997). In addition, positive covariation between delta activity and rCBF was also found in the left inferior parietal lobule (BA 40). In light of their results, Hofle et al. (1997) hypothesised that the visual and secondary auditory cortex may reveal a possible substrate for dream-like mentation during NREM sleep. These results complement the evidence reviewed above, as the activation of the left inferior parietal lobule corresponds with Solms’s (1997, 2000a, 2000b) clinico-anatomical lesion findings.

Furthermore, a functional magnetic imaging (*fMRI*) study by Portas et al. (2000), that examined auditory processing across the sleep-wake cycle, found that neutral auditory stimuli presented during sleep resulted in activation of the auditory cortex, thalamus and caudate bilaterally; while a meaningful auditory stimulus (e.g. the participant’s name) additionally resulted in higher activation of the middle temporal gyrus and the orbitofrontal cortex bilaterally. As a result, these authors concluded that the brain was able to facilitate external stimuli during sleep. Therefore, it seems that the dreaming mind may retain subliminal contact with reality through the sensory channels, suggesting that “sensory stimuli that reach us during sleep may very well become the sources of dreams” (Freud, 1900/1953, p.23, as cited in Solms, 1997, p.136). Unfortunately, neither of these studies actually investigated whether dreaming was definitely related to the neuronal activation seen during the neuroimaging scans.

However, Takeuchi et al. (2001), in a study aimed at exploring the relationship between dreaming during induced Sleep Onset REM Periods (SOREMPs) and regular N1 sleep onset, found that dreaming was more likely to occur in NREM sleep onset when there

was increased electroencephalographic (EEG) arousals¹ and waking during this time. As a result, the authors concluded that there is a “strong relationship between NREMP Dreams and awakening in our results...we postulate that arousal processes might be related to Dream production during NREM sleep” (p. 50).

Therefore, Solms’s (1997) hypothesis, that dreaming throughout the sleep-wake cycle can be activated by various arousing stimuli, some of which are external, is supported by numerous lines of evidence. Consequently, many now agree that dreaming is neither intrinsic to, nor isomorphic with, the REM state (Feinberg, 2000; Vogel, 2000). It would appear that dreaming serves a specific function during sleep, and that it is not merely an epiphenomenon of REM processes.

Dreams Protect Sleep

As mentioned, the dopaminergic MC-ML system is responsible for generating dreams, and is a partial constituent of what Panksepp (1998) refers to as the SEEKING system. This system motivates nonspecific appetitive behaviours within all mammals, including humans. Several converging lines of evidence have already confirmed that this system is highly active during REM sleep, even more so even than during waking (Dahan et al., 2007; Gottesmann et al., 2004).

This evidence has led Solms (1997, 2000a) to hypothesise that because the dorsolateral prefrontal cortex (DLPFC) is deactivated during sleep, the volitional urges which are usually executed in this region during waking (in the form of thoughts and actions with logical consistency, structure and volition) have to be redirected. Therefore, Solms (2000a) argues that these appetitive urges emanating from the highly aroused limbic system are regressively directed toward the PTO region², where they are represented virtually as dreams.

¹ Arousals can be defined as transient phenomena that result in fragmented sleep without behavioural waking (p. 10). Specifically, an arousal can be scored during REM, N1, N2, or N3 if there is an abrupt shift in the EEG frequency that is characterised by a 3 to 14 second intrusion of alpha, beta, or theta waves (but not spindles or delta). Arousals are expressed as a number per hour (Arousal Index; AI) and in middle aged adults it is normal to have an AI of up to 10 (Chokroverty, 2009).

² The PTO junction forms an association cortex in the brain; this region is not responsible for primary sensory experiences, but participates in sensory integration and abstract thought processing (Yu, 2007). As mentioned, this region is crucial to dreaming, and damage can result in complete dream loss (Bishoff & Bassetti, 2004; Poza, & Martí Massó, 2006; Solms, 1997). Therefore, it is reasonable to assert that the PTO junction is the perceptual stage upon where dreams are ‘played out’.

Freud (1900) referred to this process as “regression” and argued that “in dreams the fabric of thought is resolved into its raw material” (as cited in Solms & Turnbull, 2002, p. 211).

In addition, given the facts that EEG arousals arise from internal and external sources throughout sleep, and that they have been shown to positively correlate with dreaming at sleep onset, we propose that dreaming may also protect sleep by actively diverting these naturally occurring arousals. To date, no study has investigated the relationship between dream loss and EEG arousals that occur throughout sleep. However, we are not the first to propose this function of dreams.

Freud’s Dream Theory. In 1900, Freud proposed that dreams “serve the purpose of prolonging sleep instead of waking up. Dreams are the guardians of sleep, and not its disturbers” (Freud, 1953, p. 223). Freud (1953) argued that dreams are part of a process of wish-fulfilment that enables the sleeper to continue sleeping because “the internal demand which was striving to occupy him has been replaced by an external experience, whose demand has been disposed of” (p. 223). The similarities between Freud’s dream theory, and the hypothesis that dreams protect sleep by acting as an outlet for neurological arousal during sleep, are striking.

Moreover, Freud’s dream theory remains a cornerstone of psychoanalysis. Consequently, there is great significance in empirically testing whether or not dreams protect sleep; firstly, in seeking to establish a physiological function for dreaming; and secondly, to challenge Popper’s (1963) conclusion that psychoanalysis is a pseudo-science that is incapable of generating falsifiable hypotheses.

Preliminary Findings

In a clinical investigation, Solms (1997) found evidence to suggest that a total loss of dreaming subjectively disrupts sleep: of 101 patients experiencing complete dream loss due to numerous brain injuries and illness affecting both the anterior frontal regions, as well as the posterior PTO region, a significant proportion claimed that their sleep had been disrupted since the onset of their injury. In contrast, the 260 controls with equivalent injuries who continued to dream had significantly less disturbed sleep. As a result, Solms (1997) concluded that “pending the outcome of objective (sleep laboratory) studies, which may or may not confirm the subjective reports, the sleep-protection theory of dreams is provisionally supported” (p. 165). However, these results were attained using clinical-bedside interviews

and are thus merely suggestive; further rigorous testing, using objective polysomnographic (PSG) data, needs to be completed in order to empirically test this hypothesis.

Furthermore, in 2004, Bishof and Bassetti were the first to report a case of complete dream cessation with full clinical, neuropsychological, neuroimaging, and polysomnographic documentation. Their patient, a 73-year-old woman, had suffered bilateral occipital-lobe damage after having a stroke (an acute ischemic infarction due to atrial fibrillation). The patient experienced total dream loss, and began to regain some of her dream recall after 14 weeks. Although the purpose of their study was to show that their patient retained her REM cycle while experiencing complete dream loss, the PSG recording incidentally revealed that the woman had sleep maintenance insomnia.³

In addition, Poza and Martí Massó (2006) recently reported the case of a man who ceased dreaming after a unilateral left temporo-occipital hematoma. The authors documented their patient's lesions and sleep patterns using neuroimaging and PSG. They reported that the man continued to have intact REM sleep cycles, but that after his brain injury he started complaining about his bad quality of sleep.

Therefore, two well documented cases that have reported disturbed sleep with a loss of dreaming exist in the literature to date. However, the authors did not consider the possibility that the disturbances in their patients' sleep could be a result of their cessation in dreaming.

³ Sleep-maintenance insomnia is the “disturbance in maintaining sleep once achieved; persistently interrupted sleep, without difficulty falling asleep.” (Chokroverty, 2009)

Aims and Hypotheses

While there has been an increased interest in the scientific exploration of dreams in the last few decades, no dream theory has yet been successfully empirically tested. Furthermore, while there have been two cases of total dream loss published (Bischoff & Bassetti, 2004; Poza & Martí Massó, 2006) with full clinical, neuropsychological, neuroimaging and polysomnographic data, the aim of these studies was to fully document a cessation of dreaming simultaneously occurring with regular REM cycles and not to report the effects of dream loss on sleep. However, both cases reported that their patients *had experienced disturbed sleep since their dream loss*.

In light of these results, this study had two primary aims: firstly, we aimed to use polysomnographic recording to comprehensively document dream loss as a distinct neuropsychological dysfunction; and secondly, to focus on the relationship between disturbed sleep and loss of dreaming. For this study, it was predicted that a patient with bilateral occipital lesions would have complete dream loss and that her sleep would subsequently be disturbed. The following hypothesis was examined:

H₁: A single participant with bilateral occipital lesions will experience significantly disturbed sleep when compared with published norms.

METHODS

Sample

Mrs P was a 65-year-old dextral female. She was a homemaker with 10 years of formal education, and no previous medical or psychiatric history. The selection criteria for the case included loss of dreaming (a complete lack of subjective dream recall), accompanied by specific posterior lesions to the PTO junction. Mrs P was the first patient found to meet these criteria. Any patients with any other sleep or neurological disorder that could confound the results, were not considered for the study. Mrs P was referred by a neurologist from Groote Schuur Hospital.

Healthy Age-Matched Controls. In order to understand specifically what effects dream loss may have on sleep, it is important to exclude natural changes in sleep that are related to age. Age has been found to have disruptive effects on sleep organisation and architecture (Chokroverty, 2009; Feinberg, 1973). For this reason, a study by Boselli et al. (1998) comprised of 10 participants (5 females, 5 males) over 60 years of age, was used as a control group. Boselli et al. recruited healthy individuals with no daytime complaints, good sleep quality and regular life habits. Moreover, the participants' sleep macrostructure was compatible with widely accepted quantitative norms (Ohayon et al., 2004).

Chronic Stroke Controls. In addition to age, stroke has been found to have an impact on the quality of sleep (Bassetti & Aldrich, 2001; Chokroverty & Montagna, 2009; Korner et al., 1986; Vock et al., 2002). In order to exclude the effects of stroke on Mrs P's sleep quality and quantity, published sleep macrostructure means for a group of patients with ischemic hemispheric lesions have been used for comparison (Vock et al., 2002). Since our case was in the post-chronic phase after stroke (>5 years), Vock et al.'s study was suitable to use for comparison.

Measures

Case History

Mrs P's case history was taken directly from her medical records, and her case information has been duplicated here in accordance with the APA guidelines for confidentiality and anonymity (American Psychiatric Association, 2005). As such, certain

identifying information has been excluded, albeit not to the extent that the information provided has been distorted in any way

Dream Recall

Mrs P was asked to give a subjective account of her dreaming, or lack thereof, since her stroke in 2006. Specifically, she was asked whether she still dreamt or if she remembered any dreams she may have had in the past 5 years. In addition, semi-spontaneous nocturnal REM sleep interviewing, during the first night in the sleep laboratory, was used to verify dream loss. This interview consisted of briefly asking Mrs P, during unprompted REM awakening, if she was dreaming, and about what was going through her mind prior to being awoken.

Neuropsychological Tests

A range of neurocognitive tests were chosen for this study, focusing primarily on higher visual and spatial perception, visual and verbal short-term memory, and visual and audio-verbal long-term memory — the necessary neuropsychological functions required for intact dream recall (Appendix A). The constellation of neuropsychological subtests and scoring systems used in this study are widely recognised and internationally established standard measures, and are being used on an ongoing basis in the daily clinical neurocognitive assessments of the neuropsychologists at Groote Schuur Hospital (Strauss, Sherman & Spreen, 2006).

Visuo-spatial perception. For the assessment of visuo-spatial perception, the subtests chosen were from Luria's Neuropsychological Investigation for higher visual perception and integration included: 1) object recognition; 2) visual recognition of letters, words and phrases; 3) calculations; 4) colours and faces; 5) language (Christensen, 1974). In addition, the Judgement of Line Orientation Test (Benton et al., 1994); Benton's Facial Recognition Test (Benton et al., 1994); and the Boston Naming Test (BNT; which doubles as a language test), were also used (Kaplan, Goodglass & Weintraub, 2001).

Constructional praxis. The WAIS-III Blocks were chosen for assessing perceptual organization and constructional praxis (The Psychological Corporation, 1997). In addition, the Rey-Osterrieth Complex Figure was also utilised for this purpose (ROCF; Rey, 1941; Osterrieth, 1944).

Short-term memory. Corsi's Blocks were used to assess visual short-term memory, whilst the Digit Span Test was used to assess audio-verbal short-term memory (WMS-III; Wechsler, 1997). Additionally, the Visual Reproduction-1 test was used to assess visual short memory (WMS-III; Wechsler, 1997).

Visual and verbal memory. The ROCF was also used to assess both immediate visual memory and long-term visual memory. The ROCF scoring system includes a copy trial, an immediate recall trial, and a delayed recall trial after approximately 30 minutes. Benton's Visual Retention was also used to assess long-term visual memory (Sivan, 1992). The Babcock Story was used for the assessment of long-term verbal memory (Babcock & Levy, 1930). In addition, The Bicycle Drawing Test (BDT), the South African flag, and a canary, were all included for the purpose of assessing the patient's ability to revisualise from memory, without the aid of a copy (Lezak, 1995).

Polysomnographic Measures

The PSG recordings were completed on a portable Alice © 5 Respironics polygraphic amplifier (Cape Sleep Centre, Gatesville Medical Centre, Cape Town). The American Association of Sleep Medicine (AASM) recommended recording montage was utilized in this study and included: electroencephalogram (EEG; 4 leads, 2 channels); electrooculogram (EOG; 2 channels); the submental electromyogram (EMG; chin and leg); as well as chest and abdominal strain gauges, snore microphone, positional marking and finger pulse oximetry. Sleep stages were visually scored for 30-s epochs by a certified polysomnographic technologist based on AASM standard criteria (Hirshkowitz & Sharafkhaneh, 2009).

Sleep macro and microstructure. The analysis of conventional sleep parameters included the total duration of the sleep stages REM and NREM (N1, N2, N3); sleep latency (SL): the interval between lights-out and the first appearance of N1 sleep that subsequently progresses to N2; REM latency (REML): the time it took to reach the first REM stage; total sleep time (TST): total time spent in N1, N2, N3 and REM; total sleep episode (TSE): the total time from sleep onset to end of sleep; wake after sleep onset (WASO): the total time spent awake from sleep onset to the end of sleep; spontaneous arousals: abrupt changes in EEG frequency that may include theta, alpha and frequencies greater than 16Hz, but not sleep spindles. In addition, each arousal must be preceded by at least 10 seconds of continuous sleep, and at least 10 seconds of intervening sleep to score a second arousal (Appendix B).

Design

An explanatory quantitative single-case study design was used to investigate whether dreams protect sleep. Mrs P's medical records were used to document the events surrounding her stroke in 2006 and her subsequent dream loss, as well as to verify her lesion localisation. The dependant variables measured in this study were *quality of sleep* and *quantity of sleep*. Specifically, quality of sleep refers to sleep efficiency, including whether sleep was fragmented; quantity of sleep refers to sleep architecture, including the time spent in each sleep stage and sleep latencies. Data were collected from full-night attended laboratory PSG recording. The first night was used as an adaption night to control for confounding affects due to the unfamiliarity of the sleep laboratory. The second night was used to measure the quality and quantity for Mrs P's sleep.

The following control measures were also taken: neuropsychological testing was used to assess Mrs P's memory and visuo-spatial ability, to ensure that she did not have a neurocognitive deficit that might account for her lack of subjective dream recall. Moreover, assessing these functions could potentially help to establish a diagnostic consistency between clinical observation and anatomical pathology. In addition, the effects of age and neurological damage caused by the stroke (apart from dream loss) were controlled for by comparing Mrs P's PSG sleep parameters with that of normative controls from the literature.

The proposed study was nested within a larger study that adhered to the ethical guidelines for research with human subjects as specified by the Health Profession Council of South Africa (HPCSA), as well as the University of Cape Town (UCT) Codes for Research. Ethical approval was obtained from the Psychology Department's Research Ethics Committee at UCT, as well the Faculty of Health Sciences Research Ethics Committee at UCT (REC. REF. 163/2010).

Data Analysis

The PSG data was manually analysed and scored by the sleep technologist at Gatesville Medical Centre sleep laboratory, according to the AASM standard sleep guidelines. Thereafter, it was compiled into a comprehensive PSG sleep report using Alice© 5 Respirationics software.

Statistical Analysis. Hypothesis testing was used to determine whether Mrs P's quality and quantity of sleep on the second sleep laboratory night was significantly different to healthy age-matched controls (Appendix C). Z-tests were used to transform the Mrs P's mean

scores into z -scores (Z_{calc}) which were then compared to the critical z -values (Z_{crit}) in order to determine whether the null hypotheses were rejected. Specifically, the critical z -values used were: $Z_{crit} = \pm 2.58$ ($\alpha = 0.01$; [two-tailed test]); $Z_{crit} = 2.32$ ($\alpha = 0.01$, [one-tailed test]; Durrheim, 2002).

Neurocognitive Tests. The neuropsychological tests were analysed by way of the *hypothetical-deductive approach*, as used by the neuropsychologist at Groote Schuur Hospital in their everyday clinical practice. Specifically, we used Mrs P's test scores to confirm or reject informal hypothesis about her neurocognitive functioning. For example, we hypothesized that Mrs P's memory would be intact, and used the scores from her tests to either accept or reject this conclusion. Scoring of the neuropsychological tests was done according to the standard procedures outlined with each test.

Procedure

The neuropsychological testing took place at Groote Schuur Hospital, where a quiet room, free of distractions, was used as an assessment setting. The sleep study was completed at the Cape Sleep Centre at Gatesville Medical Centre—an approved AASM sleep Laboratory — where the PSG recording was professionally monitored by a qualified sleep technologist.

Sleep Study

Mrs P was monitored in the Gatesville sleep laboratory for two consecutive nights. On both nights full-night PSG recordings from her were obtained. The first night was an adaptation night, to help Mrs P become familiar with the laboratory setting, as well as to confirm her basic sleep/dream activity. The second night Mrs P was left to sleep without interruption in order to determine the quality and quantity of her sleep. Mrs P was thoroughly informed of the main purpose of the study and the procedures, and a consent form was signed before data collection began (Appendix D). Furthermore, she was told that she was free to withdraw from the study at any time without consequence, should she wish to do so.

Night 1. Mrs P arrived at the Gatesville Medical Centre at approximately 22:00; she arrived late because she had mistakenly thought that the sleep study was being done at Groote Schuur Hospital, and had gone there first. After being asked to lie down in the sleep laboratory bed, electrodes were attached to her as per the 10-20 system of placement (see

measures). Mrs P was then asked to sleep as she normally would in her home environment. Polysomnograph recording started at 22:53; Lights Off (LO) occurred at 23:27; and Sleep Onset (SO) was at 01:15 the following morning. During Mrs P's two REM cycles she awoke without being prompted, and these awakenings were used as an opportunity for dream recalls: she was briefly questioned as to whether she was dreaming and then left to go back to sleep. Polysomnograph recording ended 06:01. Mrs P was asked for a subjective account of her sleep, was thanked for participating in the study, and asked to come back the following night.

Night 2. Mrs P arrived at Gatesville Medical Centre at approximately 19:00. She was asked to lie down in the sleep laboratory bed and electrodes were attached as per the previous night. She was then asked to sleep as she normally would in her home environment. Polysomnograph recording started at 20:21; LO occurred at 20:31; and SO was at 20:58. Mrs P was not interrupted or disturbed and was left to sleep as naturally as possible. Polysomnograph recording ended at 06:01 and the lights were switched on. Mrs P was debriefed and asked whether she had dreamt and whether she felt that her quality of sleep in the laboratory was comparable to the quality of her sleep at home. Finally, Mrs P was thanked for taking part in the study and compensated in accordance with her participation agreement.

RESULTS

History of stroke

In late February 2006, Mrs P was admitted to hospital following the sudden onset of blindness and a severe headache while she was shopping (history obtained from her family members). It was reported that her vision returned partially within a few minutes, but that her headache persisted and was accompanied by severe dizziness. Mrs P was put on medication as a secondary stroke prevention measure.

In early March 2006, Mrs P was sent for neurological evaluation because she remained confused. Neurologically, Mrs P was found to have the following problems: an abnormal mental state characterised by axial amnesia; persistent disorientation to time; difficulties with problem solving and complexity; and labile mood. However, neuropsychological testing indicated that no visual agnosia (inability to recognise objects/images); aphasia (difficulty in producing or comprehending, spoken or written language); impulse control; or word generation symptoms were present, and that Mrs P's general judgement was preserved. Additionally, Mrs P's report indicated that she had visual cortical dysfunction, which resulted in a left superior quadrantanopia (partial defect in her visual field). Also, a mildly ataxic gait (unstable balance) was observed, but no other focal localising signs were present. Mrs P only regained her memory 7 days after her initial stroke. She was also found to be photophobic (sensitive to light) and her blurred vision remained a consistent complaint.

Magnetic Resonance Imaging Results

Routine magnetic resonance imaging (MRI) done 14 days after Mrs P's stroke in 2006, and was used to identify structural neuropathological changes (Appendix E). Overall, the scans revealed acute infarcts in both occipital lobes (larger on the right) and right cerebellar hemisphere. In addition, a small ischemic lesion was noted in the left thalamus. Furthermore, Computed Tomography (CT) angiogram demonstrated that Mrs P's extracranial vasculature was normal, and no features of dissection were noted. Moreover, no haemorrhaging was indicated by the gradient echo sequences.

Pre-morbid dream and sleep activity

Mrs P subjectively recalled that, prior to her stroke in 2006, she experienced regular dream recall (3-4 times per week) and undisturbed sleep. She reported that she used to nap during the day when she got tired (which she still continues to do).

Dream Recall

On the first sleep laboratory night, Mrs P was interviewed twice during separate unprompted REM awakenings and asked if she was dreaming. The awakenings were semi-spontaneous and were performed when she entered a REM sleep cycle. The first semi-spontaneous interview was at 4:34 am (ninety seconds into the participant's REM cycle); and the second was at 5:42am. Mrs P reported that she was not dreaming, or experiencing any other thoughts or mentation (Appendix F).

Neuropsychological Testing

On formal neurocognitive assessment, Mrs P was oriented to person, place and time. With respect to her short-term memory, Mrs P demonstrated intact function in both the verbal and visual spheres. She had a verbal span of six digits (on the Digit Span test) and a visual span of five (for Corsi's Blocks). However, she was only able to perform adequately on the WMS-III Visual Reproduction I task, getting just the first two items correct. On two final measures of visual short-term, the Benton Visual Retention Test and the immediate recall of Luria's Complex Visual Scenes, she performed well, scoring six out of seven and full marks, respectively.

Mrs P had the intact ability to revisualise objects (a bicycle, the South African flag and a canary) from her memory. She was also able to visualise a meaningful percept in the form of her husband's face. On assessment of long-term visual memory, Mrs P struggled with the ROCF. However, she performed significantly better when doing the Luria's Complex Scenes as a test for long-term visual memory, and was able to correctly recognise all the depicted scenes shown to her after delay. As a test of verbal long-term memory, she performed adequately when given the Babcock Story, scoring 5, 7 and 8 for the respective trials.

On assessment of constructional praxis, using the WAIS-III Blocks, Mrs P performed adequately, and was able to correctly reproduce the first five designs presented in under ten seconds. Thereafter, she correctly completed up to Item 10, in progressively slower times.

She was unable to complete Items 11, 12 and 13. Additionally, on the ROCF, her performance was mildly constructionally apraxic, evident from both the overall gestalt of the copy and from her score of 25 out of 36.

Finally, with respect to her visuo-spatial perception, Mrs P performed adequately on Benton's Judgement of Line Orientation test, scoring 10 out of 14. For the visual recognition of letters, words and phrases, colours and faces, she performed normally. She displayed no difficulties with Luria's calculations. Additionally, she scored perfectly on the Benton's Facial Recognition Test. The BNT was also administered, with Mrs P scoring 38 out of 40. Finally, she was able to identify all of Luria's Complex Visual Scenes without difficulty.

Sleep Study

In the subjective morning report of the second night in the sleep laboratory, Mrs P reported that she slept well and very similarly to her when she is in her home environment.

REM sleep

As confirmed by the PSG recordings, Mrs P did experience REM sleep periods (Figure 1). During the first night in the sleep laboratory she had two REM periods (two minutes and eight minutes in length). Due to semi-spontaneous dream recalls and the unfamiliarity of the sleep laboratory, Mrs P's sleep was significantly disturbed and her REM accounted for only 5.7% of her TST.

On the second night Mrs P had three REM periods (Table 1) and therefore three sleep cycles. Moreover, an analysis by fraction indicated that REM sleep was most prominent during the second 190 minute fraction of sleep, than the third 190 minute fraction, as shown in Table 2. A simple *z*-score calculation indicated that Mrs P's REM sleep was not significantly different to healthy age-matched controls, as shown in Table 3, and amounted to 20.5% of TST.

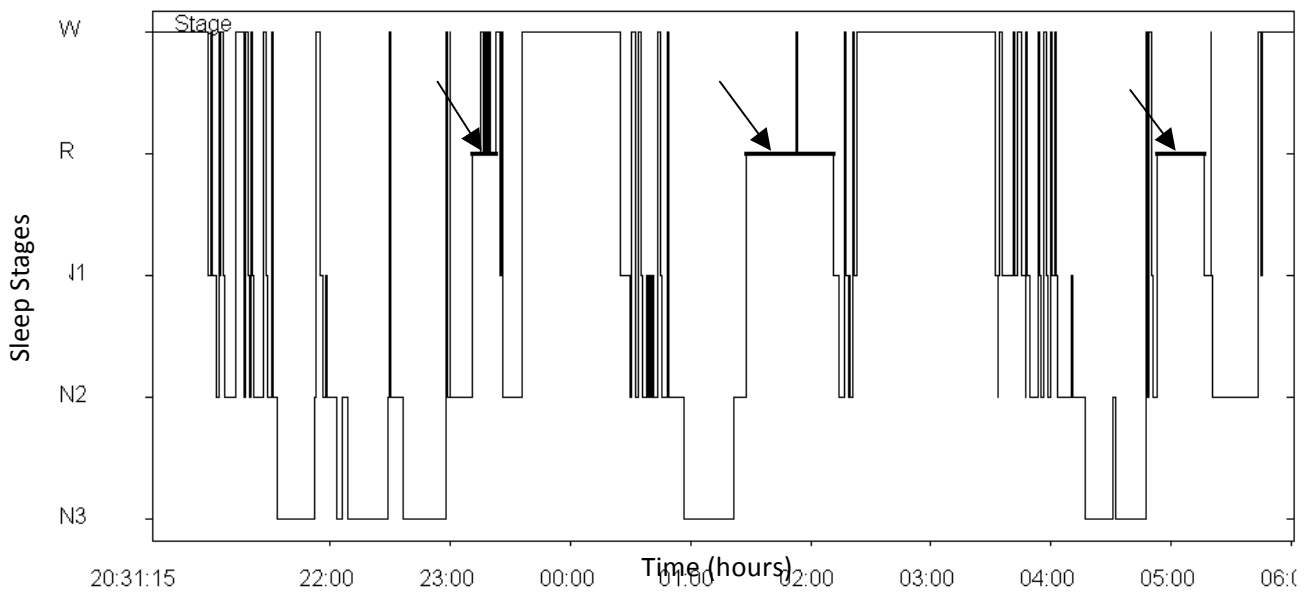


Figure 1. Sleep hypnogram for the second sleep laboratory night, derived from the EEG recordings. Horizontal axis measured in hours. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep. Arrows indicate REM sleep stages.

Table 1.

REM Cycles

	Total	Cycle	REM start
REM 1	8.5	171.5	23:10:45
REM 2	43.5	168.5	01:27:45
REM 3	24.0	185.5	04:52:45

Note. All values are measured in minutes, except REM start, which is an indication of time.

Table 2

Analysis of Sleep Stages per Fraction

	First 190 min	Second 190 min	Third 190 min
W	53.5	78.5	69.0
N3	63.0	25.0	29.0
REM	8.5	43.0	24.0

Note. All values are measured in minutes. W = waking;

N3 = stage 3 non-REM sleep; REM = rapid eye movement.

Table 3

Comparison of Sleep Parameters Between the Mrs P and Healthy Age-matched Controls

	Mrs P	Controls (n=10)	Z _{calc}
SL	27.5	12 (7)	2.21
TST	369	406 (42)	-0.88
SE	64.7	81 (8)	-2.04
WASO	173.5	68 (45)	2.34*
N1	44.5	32 (16)	0.78
N2	132	234 (31)	-3.29**
N3	117	54 (16)	3.94**
REM	75.5	86 (23)	-0.46
REML	159.5	82 (32)	2.42*

Note. All values are measured in minutes, except for SE which is a percentage. SL = Sleep Latency; TST = Total sleep time (R + N1 + N2 + N3); W = waking; WASO= Waking after sleep onset; TW = Total waking; REMS = Rapid Eye Movement Sleep; NREMS = Non-Rapid Eye Movement sleep; N1 = NREM stage 1; N2 = NREM stage 2; N3 = NREM stage 3 (Also referred to as Slow Wave Sleep [SWS]); REML = REM Latency.

*Z_{crit} = ±2.32, α = 0.01, one-tailed test; **Z_{crit} = ± 2.58, α = 0.01, two-tailed test.

Comparison of Means for Mrs P and the Normative Control Groups

Figure 2 shows a visual display of the sleep parameter means (in minutes) for Mrs P, the healthy age-matched control (HAC) group, and the chronic stroke control (CSC) group. The differences in Mrs P's sleep parameters when compared with the HAC and CSC groups were most apparent for WASO, REML, N2 and N3 sleep. Both Mrs P and the CSCs tended to have slightly less REM and more N1 than the HACs.

Waking After Sleep Onset. There was a noticeable difference between the WASO means for Mrs P, the HACs and the CSCs. Waking after sleep onset only accounted for 6.13% of the TSE (473 minutes) in the CSC group and 13.93% of the TSE (488 minutes) in the HAC group, as shown by Figure 3. However, WASO accounted for 29.9% of Mrs P's TSE (526.5 minutes).

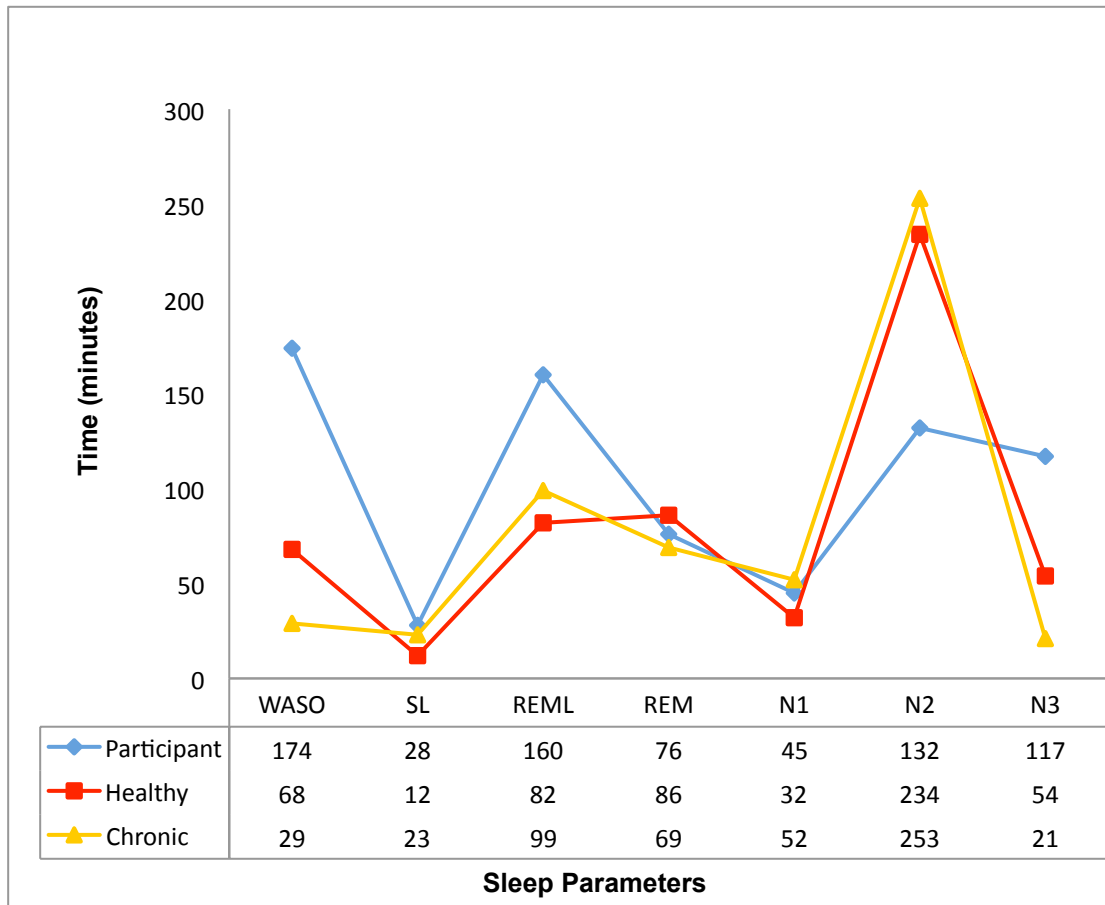


Figure 2. Comparison of Sleep Parameter means for Mrs P, the healthy age-matched controls, and the chronic stroke controls. All values are measured in minutes. WASO = waking after sleep onset; SL = sleep latency; REML = REM latency; REM = rapid eye movement; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

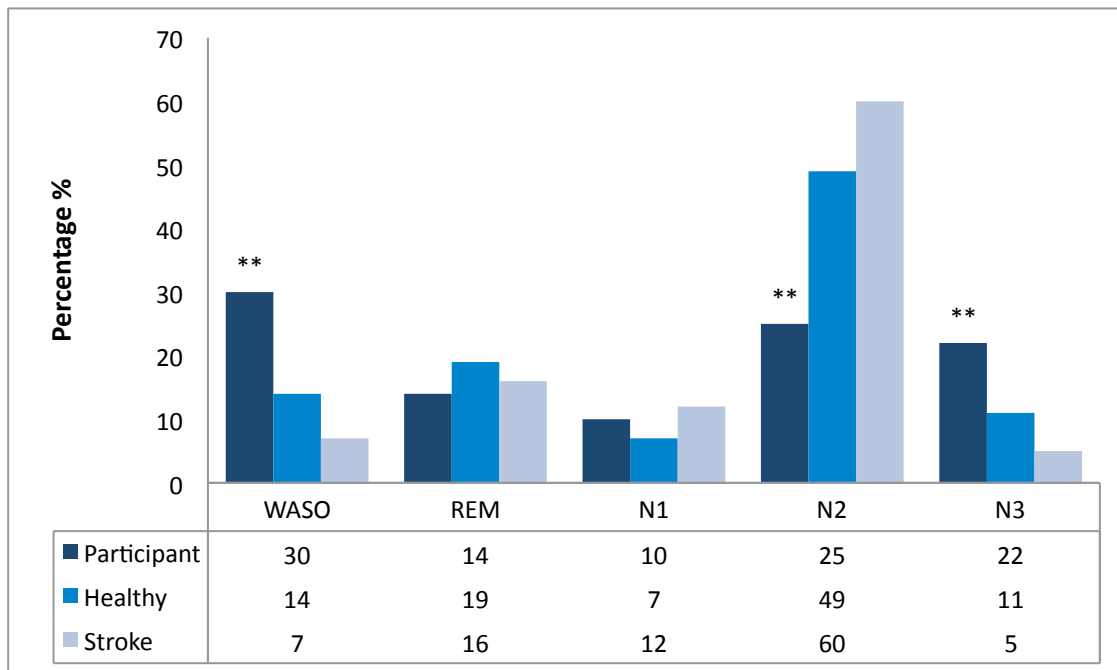


Figure 3. Sleep Stage Distributions for Mrs P and the Controls. WASO = waking after sleep onset; REM = rapid eye movement sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

** $Z_{crit} = \pm 2.58$, $\alpha = 0.01$, two-tailed test.

REM Latency. The time it took Mrs P to reach her REM 1 cycle was significantly longer than that of the HAC group and the CSC group (Table 3). Furthermore, Mrs P had 132 recorded stage shifts throughout the night (moved from one sleep stage to another) which indicates that she generally had difficulty entering or maintaining sleep stages (Figure 1).

N2 and N3 Sleep. As shown in Table 3, Mrs P's N2 and N3 sleep differed significantly from the HAC group: her N2 sleep was 102 minutes less than the HAC group and her N3 sleep was significantly increased, lasting 63 minutes longer. Moreover, the CSC group did not follow this trend and had an even greater percentage of N2 (60%) and even less N3 (5%) than the HACs (Figure 3). Therefore, Mrs P's N2 and N3 sleep stages were significantly different to both control groups, and even more so than the CSC group.

Sleep Latency and Efficiency. Mrs P's sleep latency was increased compared with HACs and she took 15.5 minutes longer for her to fall asleep. Moreover, Mrs P's decreased

sleep efficiency indicated that she spent a smaller percentage of her time in bed *asleep* (TST/TIB= 64.7%) than the HACs and the CSCs (Figure 2).

Arousals. Mrs P experienced less than half the number of spontaneous EEG arousals as HACs (Table 3). There was no discrepancy in arousals between Mrs P and the HACs during REM sleep; however, during NREM sleep Mrs P experienced significantly less arousals than the HACs (Table 3).

Table 3.

Distribution of EEG Arousals Throughout the Sleep Stages and Corresponding Arousal Indices

	Mrs P		Healthy Controls		Z_{calc}
	Arousals	AI	Arousals	AI	
TST	96	15.6	184 (38)	27.1 (3.3)	-2.32*
NREMS	60	12.3	165 (30)	30.7 (3.3)	-3.5**
REMS	19	15.1	17 (13)	11.3 (6.9)	0.15

Note. All values are measured in minutes. The values in brackets represent the standard deviations for the healthy control group. TST = Total sleep time; NREM = Non- rapid eye movement sleep; REM = rapid eye movement sleep; AI = Arousal Index: defined as the number of arousals per hour of sleep. AI = Arousals/TST; Arousals/NREMS; Arousals/REMS.

* $Z_{crit} = \pm 2.32$, $\alpha = 0.01$, one-tailed test; ** $Z_{crit} = \pm 2.58$, $\alpha = 0.01$, two-tailed test.

Furthermore, a total of 39 waking episodes and 147 respiratory arousals were recorded using PSG. Mrs P's respiratory arousals indicated that she had an Apnea + Hypopnea Index (AHI)⁴ of 14.5, which is considered to be mild to moderate sleep disordered breathing by the AASM standard criteria (Benbir & Guilleminault, 2007). However, her average blood oxygen level remained > 91% and only dropped to a minimum of 85% once, for a duration of 7 seconds, which is considered normal (Benbir & Guilleminault, 2007).

⁴ An apneic event is defined as a complete cessation of respiratory flow for at least 10 seconds. A hypopnea is a 30% or more reduction in respiratory airflow that is associated with a 4% drop in blood oxygen saturation levels. Up to five respiratory events per hour are not considered to be pathological, and even ten events per hour can be considered normal if not accompanied by day time sleepiness (Benbir & Guilleminault, 2007).

DISCUSSION

As predicted, our study confirmed preserved REM sleep in a case with deep bilateral occipital lobe damage resulting in total dream loss. Polysomnographic recording clearly indicated that Mrs P experienced REM sleep on both nights in the sleep laboratory, while dream recall established that she did in fact have complete dream loss. Therefore, Mrs P is the third comprehensively documented case of dream loss as a distinct neuropsychological dysfunction. Furthermore, the results of this case study confirmed the hypothesis: the PSG data verified that the patient's sleep was significantly more disturbed than published normative controls. Based on the previous literature, it was predicted that dreaming could protect sleep by diverting activity from the SEEKING system, as well as EEG arousals. While Mrs P did experience disturbed sleep, the results were not consistent for both of these ideas. The exact ways in which our study confirms that dreaming protects sleep are elaborated on below.

Neuropsychological Testing

The neuropsychological testing of Mrs P focused on excluding the presence of specific neurocognitive deficits which might jeopardize her ability to recall her dreams. Specifically, the basic preservation of visual and verbal short-term memory function, visual and verbal long-term memory function, and spatial cognition (visuo-spatial perception and recognition and constructional praxis), are all necessary requirements for dream recall. In addition, other more obvious neurocognitive deficits such as aphasia, also needed to be ruled out.

The neurocognitive assessment revealed that, firstly, Mrs P does not display a short-term memory deficit in either the verbal or visual sphere. This is despite her relatively poor performance on the WMS III Visual Reproduction I task, which may be accounted for by her mild constructional apraxia (see below).

Testing of Mrs P's verbal long-term memory found that although she struggled somewhat with complexity, she was clearly able to lay down new memories and retain the overall content and structure of the newly-learned material. With respect to her visual long-term memory, Mrs P proved that she did not display a major memory deficit through her ability to recall all of Luria's Complex Scenes accurately. Her performance on the Rey-Osterrieth Complex Figure (ROCF), suggested that Mrs P's visual memory was impaired. However, this test lacked construct validity due to the fact that Mrs P had primary visual

difficulties that made pen and paper tests difficult to complete. In particular, she suffered from blurred vision and photophobia, and often asked for us to close the curtains because the light ‘hurt her eyes’. Furthermore, she has a mild constructional apraxia (see below) that hindered her ability to successfully complete the ROCF test. This observation is supported by the fact that she was able to recall visual images from Luria’s Neuropsychological Investigation successfully. The real-world images depicted in Luria’s Complex Scenes were less abstract and more meaningful than the ROCF, and did not require intact higher visuo-constructional ability. Additionally, Mrs P was subsequently able to recall in some detail the room where her PSG was done, thereby adding additional confirmation that her visual long-term memory was largely intact (Appendix G). Overall, no amnesic syndrome was present.

The WAIS-III Blocks Design and the ROCF, aside from testing Mrs P’s visual short-term memory, also indicated that she had a mild constructional apraxia. This impairment may have accounted for her poor visual memory performance scores. Furthermore, Mrs P had normal visuo-spatial perception, and no prosopagnosia or agnosia was present. If she had been dreaming she would not have been incapable of recounting them due to visuo-spatial impairments. Her BNT performance also verified that she was not aphasic, further confirming that her lack of subjective dream recall was not due to an inability to verbally communicate her dreams or to understand questions/instructions given to her.

In summary, the overall neuropsychological testing of Mrs P confirmed that her dream loss was not better accounted for by short or long-term memory deficits, visuo-spatial impairments or problems with visual recognition.

Furthermore, even if Mrs P was experiencing visual memory impairment, a range of findings have confirmed that impaired memory in no way provides sufficient explanation for a total lack of awareness of dream experience (Solms 1997; Yu, 2007). Even patients with profound memory deficits are able to effectively report dream recall and awareness. The retrieval of personal dream experience through multiple channels makes it highly unlikely that dream content would be forgotten, even if there is a memory disturbance in specific modalities (e.g. visuospatial or audioverbal memory; Flexser & Tulving, 1978). Therefore, we are confident that memory loss was not a confounding factor in this study.

Dream Loss and Non- REM Sleep

Although our findings replicate those of Bischof and Bassetti (2004) and Poza and Martí Massó (2006) in terms of dream loss accompanied by disturbed sleep, neither of the aforementioned authors provided specific data as to exactly how their patients’ sleep was

disturbed. However, it is perhaps too simplistic to conclude that dreams prevent the disruption of sleep *generally*, and we propose that dreaming protects sleep in a very definite way.

Specifically, our results indicated that dream loss may have affected Mrs P's sleep architecture — which consists of the various sleep stages — in numerous ways (Appendix B). Compared with HACs, Mrs P had significantly more WASO, higher N3, and decreased N2. Further analysis ruled out the possible influences of age and general complications due to stroke: the two control groups, the healthy age-matched controls (HAC) and the chronic stroke controls (CSC), followed a similar trend with regards to their sleep stages, which differed from Mrs P. Interestingly, there was more discrepancy between the CSCs and Mrs P, than between the HACs and Mrs P, further excluding the possibility that damage due to stroke (other than dream loss) led to Mrs P's sleep disturbances. This is an important finding, considering that frequent dream mentation has been reported to occur during the N1 and N2 sleep stages, and warrants further discussion.

Looking at Mrs P's sleep hypnogram (Figure 2) it is clear that she struggled to enter N1 sleep, as well as enter and maintain N2 sleep, throughout the night: her N2 sleep is continuously fragmented and often resulted in waking. Indeed, her two longest waking periods (> 1 hour each) both emerged from disrupted N2 sleep. Moreover, after these periods of wakefulness Mrs P had difficulty going back to sleep, and increased N1 and N2 sleep fragmentation is apparent here as well.

In addition, Mrs P experienced 132 sleep stage shifts during the second night, which indicates that switching between sleep stages was problematic. Furthermore, the majority of these sleep stage shifts emerged from her N2 sleep disruptions. Mrs P's difficulty in falling asleep may also have been due to ineffective N1 sleep, as N1 is increased with inter-sleep wakefulness because it facilitates the transition from wakefulness into sleep (Hirshkowitz & Sharafkhaneh, 2009). However, Mrs P did not have increased N1 (to account for her increased WASO) when compared with the HACs. Therefore, Mrs P's continuous sleep stage shifts may have been an inability to efficiently progress between sleep stages, due to her disrupted N1 sleep. It would therefore appear that because Mrs P was unable to dream, she was unable to maintain and enter N1 and N2 sleep effectively.

Further evidence for this conclusion comes from comparing Mrs P's EEG arousals with those of the HACs. As reported by Boselli et al. (1998), elderly participants experienced substantially more EEG arousals than young and middle aged adults ($F_{(3, 36)}, p < 0.0001$) and had an average NREM arousal index (AI) of 30.7 arousals per hour. Moreover, the HACs

experienced the majority of their arousals during their N1 and N2 sleep. Indeed, of the 191 EEG arousals (on average) that the HACs had during their NREM sleep, 134 were recorded in N2 sleep; 32 during N1 sleep; and only 7 during N3 sleep. However, these arousals do not usually lead to substantially increased wakefulness; if this were the case the HACs would be expected to have more WASO than Mrs P because their NREM arousal index was significantly higher. In contrast, we found that Mrs P had a substantially *decreased* NREM arousal index of 12.3 that was accompanied by *increased* WASO. Unfortunately, Boselli et al. (1998) did not include dream reports in their study and thus did not establish whether increased arousals during sleep were related to increased dream frequency or duration. However, if we refer back to Takeuchi et al.'s (2001) findings, we have evidence that increased arousals and wakefulness positively correlate with dreaming during sleep onset and N1.

Based on our results, as well as the literature, it is reasonable to propose that dreaming is able to protect sleep by facilitating a smooth transition between the sleep stages, by actively diverting both internal and external arousals via dreams. As indicated by Mrs P's increased WASO and decreased arousals, when dream mechanisms are unable to divert both naturally occurring internal EEG arousals, as well as those caused due to external stimuli, the sleep stages are unable to progress in an uninterrupted manner and result in increased waking. This also provides further evidence for Solms's (2000a) argument that dreaming occurs throughout the sleep cycle in response to sufficient cortical activation, and not only during REM sleep.

A possibly contradicting result that was found in our study was that Mrs P had an Apnea/ Hypopnea Index (AHI) of 14.5. As mentioned, this qualifies as a mild to moderate sleep disordered breathing diagnosis according to the AASM standard criteria (Benbir & Guilleminault, 2007). However, there are a number of reasons why we believe that Mrs P's mildly disordered breathing did not account for her disturbed sleep. Firstly, severe sleep disordered breathing and obstructive sleep apnea syndrome (OSAS) has been found to affect sleep architecture by specifically reducing REM and N3 sleep, while N1 and N2 were actually *increased* (Iranzo & Santamaria, 2005). This is directly opposite to the way in which Mrs P's sleep architecture was disturbed. Moreover, sleep disordered breathing disorders usually result in a below average blood oxygen level: Mrs P's oxygen levels remained higher than 92% on average, which is considered normal (Benbir & Guilleminault, 2007). Regardless, future studies should aim at excluding sleep disordered breathing altogether, so that dream loss can be isolated as much as possible in relation to disturbed sleep.

Dream loss and REM sleep

While our results provide strong evidence for dream loss affecting N1 and N2 sleep, and specifically the transitioning between sleep stages, Mrs P's REM sleep was found to be intact and within the normal range. Specifically, she was able to maintain two fairly long REM periods; one of 24 minutes and another of 43.5 minutes without interruption. This is peculiar considering that REM sleep is the most physiologically active sleep state, and that the MC-ML dopamine system has been found to be as active, if not more active, than during waking (Dahan et al., 2007; Gottesmann, 2004). Moreover, Solms (2000a) hypothesized that dreams protect sleep by acting as an outlet for a highly active MC-ML dopamine system during sleep. Therefore, our results did not support this hypothesis; however, it is worth mentioning a few important observations that should be taken into consideration.

Mrs P's spontaneous EEG arousal index for REM sleep (AI = 15.1) was higher than the HAC group (AI = 11.3). However, unlike during NREM when these arousals led to awakenings, REM remained undisturbed. These results are again in line with those of Tukeuchi et al. (2001), who found that when they induced REM periods at sleep onset dream frequency was only altered by the amount of REM sleep during the period, and not by waking and arousals. In other words, EEG arousals during REM at sleep onset did not influence dream frequency during this period as it had during NREM sleep onset. The reason for this may be that the brain has a higher sensory threshold for external stimuli during REM. Evidence for this comes from functional neuroimaging studies, such as that by Braun et al. (1998), who found that rCBF in dorsolateral prefrontal cortex (DLPFC) and the primary visual cortices covaried negatively with REM sleep. In light of these findings they concluded that

REM sleep may represent a state in which the brain engineers selective activation of an interoceptive network, which is dissociated from primary sensory and heteromodal association areas at either end of the visual hierarchy that mediate interactions with the external world. (p. 94)

Considering this, it seems that endogenous sensory stimuli are only integrated into the dream process during NREM sleep and not during REM sleep. This has led us to the (tentative) conclusion that dreaming may serve a different function during REM sleep, as opposed to NREM sleep, when the brain is more vulnerable to disturbance and shifting between sleep stages results in arousals that can be disruptive.

However, it is important to note that Mrs P's REM was not completely normal, as her third REM period was shorter than her second (Table 1), and usually REM periods increase as the night progresses (Hirshkowitz & Sharafkhaneh, 2009). Mrs P's disorganised REM periods may be related to her inability to enter N2 sleep, when emerging from N3 sleep. Indeed, the natural progression in sleep stages during the night work as follows: N3 usually leads *briefly* into N2 and then is followed by REM (Hirshkowitz & Sharafkhaneh, 2009). Therefore, because Mrs P may have been unable to *enter* REM sleep, this may have accounted for her decreased REM throughout the night, as well as her increased REM latency (the time it took her to enter her first REM period; Table 1). However, these conclusions are only speculative and need further investigation.

One last point concerning Mrs P's REM sleep that should be noted is the fact that she was experiencing a REM rebound effect on her second night in the sleep laboratory, due to an insufficient amount of REM the previous night. REM rebound is known to cause an increased amount of REM in the night following the disturbance, and this may have made Mrs P's REM periods longer and deeper than usual (Chokroverty, 2009). This is a confounding factor that limits what we can deduce from Mrs P's REM results and is a flaw in the design of the study.

The findings of this study provide tentative evidence that dreaming is able to protect sleep by effectively diverting physiological EEG arousals that emerge from both internal and external sources, consequently aiding the successful transition between sleep stages, as well as between wakefulness and sleep.

Limitations and Future Directions

We have already referred to the fact that general brain damage due to ischemic hemispheric stroke often disrupts sleep (Bassetti & Aldrich, 2001; Chokroverty & Montagna, 2009; Korner et al., 1986; Vock et al., 2002). This is especially true during the acute (1-8 days) and subacute (9-35 days) periods after stroke. It has been reported that REM sleep is drastically reduced in the first few days after stroke and only recovers 1 – 3 weeks afterwards (Giubilei et al., 1992, as cited in Vock et al., 2002). However, since our study is only the third case of dream loss that has been comprehensively documented using PSG recording, it is reasonable to assume that none of the literature on hemispheric stroke and sleep has considered the possibility that some of the sleep disturbance in patients who have suffered stroke may be due to a loss of dreaming. Indeed, if it has been considered it has not been reported, and to our knowledge there is no literature on ischemic hemispheric stroke that

addresses this issue. Therefore, future research could explore the extent to which some of known disturbances of sleep following hemispheric stroke may be related to dream loss.

Moreover, and unlike many other dream studies, our findings highlight the importance that dreaming may have during NREM sleep. This should encourage future research to focus on dreaming outside of REM sleep. However, it is important to remember that any investigation into NREM dreaming needs to account for the fact that dreaming during this sleep stage is highly idiosyncratic between people, and does not occur as reliably as REM dreaming (Foulkes & Vogel, 1965). According to our results, this may be because NREM dreaming occurs as needed, in order to facilitate the transition of sleep, and these needs may differ drastically between people. We concede that the whole issue needs further investigation, and that our answers here are only tentative.

Additionally, because this study was unreliable with regards to our findings during REM sleep, future research will need to address whether dreaming may also have a protective function during REM sleep. However, our results indicate that a disruption in NREM may have led to increased REM latencies, and subsequently disorganised REM periods. Therefore, any disturbance in sleep that is related to dream loss may disrupt the entire balance of the sleep stages. Ultimately, it may be more productive for dream research to do away with the REM/ NREM dichotomy and study the effects that dream loss may have throughout the sleep cycle.

A limitation of this study was that we were unable to analyse a matched control. This limitation was primarily due to the scarcity of ischemic bilateral occipital lesion cases. Indeed, a control participant would have enabled more reliable conclusions to be made. However, what we have observed from Mrs P in comparison to normative controls from the literature thus far, has allowed us to propose a promising new hypothesis for future research: Dreams protect sleep by diverting external and internal arousals during NREM sleep, therefore facilitating the transition between sleep stages.

Conclusion

The hypothesis that dreams protect sleep was investigated using PSG recording in a single-case with dream loss due to bilateral occipital lesions. Mrs P was found to have significantly disturbed sleep compared to normative controls from the literature. Although most of the literature on dreaming focuses on REM sleep, Mrs P's NREM was found to be the most disturbed, especially N1 and N2. Therefore, our results point to an important function for dreaming during NREM sleep, potentially contributing to a more comprehensive

understanding of the dream process throughout the sleep-wake cycle. Furthermore, this conclusion is in line with those of other dream specialists such as Solms (1997, 2000a) and Freud (1953), and marks the first occasion in which a function for dreams has been successfully empirically tested, as well as the first time that the psychoanalytic theory of dreams has been able to be empirically tested. This will hopefully lead to further scientific research in the field of dream science.

REFERENCES

- American Psychiatric Association. (2005). *Publication manual* (5th ed.). Washington, DC: American Psychiatric Association.
- Aserinsky, E., & Kleitman, N. (1953). Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. *Science*, *118*, 273-274.
- Aserinsky, E., & Kleitman, N. (1955). Two types of ocular motility during sleep. *Journal of Applied Physiology*, *8*, 1-10.
- Babcock, H., & Levy, L. (1930). An experiment in the measurement of mental deterioration. *Archives of Psychology*, *117*, 105-107.
- Bassetti, C. L., & Aldrich, M. S. (2001). Sleep electroencephalogram changes in acute hemispheric stroke. *Sleep Medicine*, *2*, 185-194.
- Benbir, G., & Guilleminault, C. (2007). Obstructive and nonobstructive sleep apnea: The neurological perspective. In A. Culebras (Ed.), *Sleep disorders and neurologic diseases* (2nd ed) (pp. 227-300). New York, NY: Informa.
- Benson, D. F., & Greenberg, J. P. (1969). Visual form agnosia: a specific deficit in visual discrimination. *Archives of Neurology*, *20*, 82-89.
- Benton, A. L., Sivan, A. B., Hamsher, K. de S., Varney, N. R., & Spreen, O. (1994). *Contributions to neuropsychological assessment: A clinical manual* (2nd ed.). New York: Oxford University Press.
- Bischof, M., & Bassetti, C. L. (2004). Total dream loss: A distinct neuropsychological dysfunction after bilateral PCA stroke. *Annals of Neurology*, *56*, 583-586.
- Boselli, M., Parrino, L., Smerieri, A., & Terzano, M. (1998). Effect of age on EEG arousals in normal sleep. *Sleep*, *21*, 351-357.
- Braun, A. R., Balkin, T. J., Wesensten, N. J., Carson, R. E., Varga, M., Baldwin, P., ...Herscovitch, P. (1997). Regional cerebral blood flow throughout the sleep-wake cycle: An H2150 PET study. *Brain*, *120*, 1173-1197.
- Breger, L. (1967). Function of dreams. *Journal of Abnormal Psychology*, *72*, 1-28.
- Brown, J. (1972). *Aphasia, apraxia, agnosia: Clinical and theoretical aspects*. Springfield, IL: Thomas.
- Chokroverty, S. (2009). An overview of normal sleep. In S. Chokroverty (Ed.), *Sleep disorders medicine: Basic science, technical considerations and clinical aspects* (pp. 5-21). Philadelphia, PA: Saunders Elsevier.

- Chokroverty, S., & Montagna, P. (2009). Sleep, breathing, and neurologic disorders. In S. Chokroverty (Ed.), *Sleep disorders medicine: Basic science, technical considerations and clinical aspects* (pp. 436-99). Philadelphia, PA: Saunders Elsevier.
- Christensen, A.L. (1974). *Luria's neuropsychological investigation*. Copenhagen, DK: Munksgaard.
- Crick, F., & Mitchison, G. (1983). The function of dream sleep. *Nature*, *304*, 111-114.
- Dahan, L., Astier, B., Vautrelle, N., Urbain, N., Kocsis, B., & Chouvet, G. (2007). Prominent burst firing of dopaminergic neurons in the ventral tegmental area during paradoxical sleep. *Neuropsychopharmacology*, *32*, 1232-1241.
- Dang-Vu, T. T., Desseilles, M., Laureys, S., Degueldre, C., Perrin, F., Phillips, C., Maquet, P., & Peigneux, P. (2005). Cerebral correlates of delta waves during non-REM sleep revisited. *NeuroImage* *28*, 14–21.
- Dement, W., & Kleitman, N. (1957). The relation of eye movements during sleep to dream activity: An objective method for the study of dreaming. *Journal of Experimental Psychology*, *53*, 339-346.
- Durrheim, K. (2002). Hypothesis testing: the z-test. In C. Tredoux & K. Durrheim (Eds.), *Numbers, hypotheses & conclusions: A course in statistics for the social sciences* (pp. 127-141). Cape Town: UCT Press.
- Feinberg, I. (1973). Changes in sleep cycle patterns with age. *Journal of Psychiatric Research*, *10*, 283–306.
- Feinberg, I. (2000). REM sleep: Desperately seeking isomorphism. *Behavioral and Brain Sciences*, *23*, 931-934.
- Flexser, A. J., & Tulving, E. (1978). Retrieval independence in recognition and recall. *Psychological Review*, *3*, 153-171.
- Foulkes, D. (1962). Dream reports from different stages of sleep. *Journal of Abnormal and Social Psychology*, *65*, 14–25.
- Foulkes, D., & Vogel, G. (1965). Mental activity at sleep onset. *Journal of Abnormal Psychology*, *70*, 231-43.
- Freud, S. (1953). *The standard edition of the complete psychological works of Sigmund Freud*. London, UK: Penguin Books.
- Gottesmann, C. (2004). Brain inhibitory mechanisms involved in basic and higher integrated sleep processes. *Brain Research Reviews*, *45*, 230-249.
- Hartmann, E., Russ, D., Oldfield, M., Falke, R., & Skoff, B. (1980). Dream content: Effects of L-DOPA. *Sleep Research*, *9*, 153.

- Hirshkowitz, M., & Sharafakhneh, A. (2009). Clinical polysomnography and the evolution of recording and scoring technique. In S. Chokroverty (Ed.), *Sleep disorders medicine: Basic science, technical considerations and clinical aspects* (pp. 229-52). Philadelphia, PA: Saunders Elsevier.
- Hobson, J. A. (1988). *The dreaming brain*. United States: Basic Books.
- Hobson, J. A., & McCarley, R. W. (1977). The brain as a dream state generator: An activation-synthesis hypothesis of the dream process. *American Journal of Psychiatry*, *134*, 1335- 1348.
- 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*, 793–842.
- Hofle, N., Paus, T., Reutens, D., Fiset, P., Gotman, J., Evans, A. C., & Jones, B. E. (1997). Regional cerebral blood flow changes as a function of delta and spindle activity during slow wave sleep in humans. *The Journal of Neuroscience*, *17*, 4800-4808.
- Iranzo, A., & Santamaria, J. (2005). Severe obstructive sleep apnea/hypopnea mimicking REM sleep behavior disorder. *Sleep*, *28*, 203–6.
- Jus, A., Jus, K., Villeneuve, A., Pires, A., Lachance, R., Fortier, J., & Villeneuve, R. (1973). Studies on dream recall in chronic schizophrenic patients after prefrontal lobotomy. *Biological Psychiatry*, *6*, 275–293.
- Kaplan, E. F., Goodglass, H., & Weintraub, S. (2001). *The Boston Naming Test* (2nd ed.). Philadelphia: Lippincott William & Wilkins.
- Korner, E., Flooh, E., Reinhart, B., Wolf, R., Ott, E., Krenn, W., & Lechner, H. (1986). Sleep alteration in ischemic stroke. *European Neurology*, *25*, 104-10.
- Léna, I., Parrot, S., Deschaux, O., Muffat-Joly, S., Sauvinet, V., Renaud, B.,...Gottesmann, C. (2005). Variations in extracellular levels of dopamine, noradrenaline, glutamate, and aspartate across the sleep-wake cycle in the medial prefrontal cortex and nucleus accumbens of freely moving rats. *Journal of Neuroscience Research*, *81*, 891-899.
- Lezak, M. (1995). *Neuropsychological assessment* (3rd Ed.). New York, NY: Oxford University Press.
- 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*, 163–66.

- Nofzinger, E. A., Mintun, M. A., Wiseman, M. B., Kupfer, D. J., & Moore, R. Y. (1997). Forebrain activation in REM sleep: An FDG PET study. *Brain Research*, *770*, 192–201.
- Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep*, *27*, 1255-73.
- Osterrieth, P. A. (1944). Le test de copie d'une figure complex: Contribution a l'étude de la perception et de la mémoire. *Archives de Psychologie*, *30*, 286-356.
- Panksepp, J. (1998). *Affective neuroscience: The foundations of human and animal emotions*. New York, NY: Oxford University Press
- Popper, K. R. (1963). *Conjectures and refutations: The growth of scientific knowledge*. London, UK: Routledge.
- Portas, C. M., Krakow, K., Allen, P., Josephs, O., Armony, J. L., & Frith, C. D. (2000). Auditory processing across the sleep-wake cycle: Simultaneous EEG and fMRI monitoring in humans. *Neuron*, *28*, 991–999.
- Poza, J. J., & Martí Massó, J. F. (2006). Total dream loss secondary to left temporo-occipital brain injury. *Neurología*, *21*, 152-154.
- The Psychological Corporation. (1997). WAIS-III-WMS-II technical manual. San Antonio, Tex., Author.
- Rechtschaffen, A. (1973). The psychophysiology of mental activity during sleep. In F. J. McGuigan & R. A. Schnoonover (Eds.), *The psychophysiology of thinking*. New York, NY: Academic Press.
- Revonsuo, A. (2000). The reinterpretation of dreams: An evolutionary hypothesis of function of dreaming. *Behavioral and Brain Sciences*, *23*, 877–901.
- Rey, A. (1941). L'examen psychologique dan les cas d'encephalophaie. *Archives de Psychologie*, *28*, 286-340.
- Sandyk, R. (1997). Treatment with weak electromagnetic fields restores dream recall in a parkinsonian patient. *International Journal of Neuroscience*, *90*, 75–86.
- Sivan, A. B. (1992). *Benton Visual Retention Test* (5th ed.). San Antonio, Tex.: The Psychological Corporation.
- Solms, M. (1997). *The neuropsychology of dreams: A clinico-anatomical study*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Solms, M. (2000a). Dreaming and REM sleep are controlled by different brain

- mechanisms. *Behavioral and Brain Sciences*, 23, 843-850.
- Solms, M. (2000b). Forebrain mechanisms of dreaming are activated from a variety of sources. *Behavioral and Brain Sciences*, 23, 1035-1040.
- Solms, M., & Malcom-Smith, S. (2009). Dreaming in neurologic disorders. In S. Chokroverty (Ed.), *Sleep disorders medicine: Basic science, technical considerations and clinical aspects* (pp. 530-38). Philadelphia, PA: Saunders Elsevier.
- Solms, M., & Turnbull, O. (2002). *The brain and the inner world: An introduction to the neuroscience of the subjective experience*. New York, NY: Other Press.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: administration, norms, and commentary* (3rd ed.). New York: Oxford University Press.
- Takeuchi, T., Miyasita, A., Inugami, M., Yamamoto, Y. (2001). Intrinsic dreams are not produced without REM sleep mechanisms: Evidence through elicitation of sleep-onset REM periods. *Journal of Sleep Research*, 10, 43-52.
- Vock, J., Achermann, P., Bischof, M., Milanova, M., Muller, C., NirKKo, A., Roth, C., & Bassetti, C. L. (2002). Evolution of sleep and sleep EEG after hemispheric stroke. *Journal of Sleep Research*, 11, 331-8.
- Vogel, G. W. (2000). Critique of current dream theories. *Behavioral and Brain Sciences*, 23, 1014-16.
- Wechsler, D. (1997). *WMS-III administration and scoring manual*. San Antonio, Tex., The Psychological Corporation.
- Yu, C. K. (2007). A neuropharmacological study of dreaming in psychiatric patients. *Brain mechanisms of dreaming*, unpublished PhD Thesis: UCT.

Appendix A: Neuropsychological Tests

A range of neurocognitive tests were chosen for this study, focusing primarily on higher visual and spatial perception, visual and verbal short-term memory, and visual and audio-verbal long-term memory.

Visuo-spatial Perception

Luria's Neuropsychological Investigation. LNI-1 was used to assess higher visual perception and integration, and specifically included: 1) object recognition; 2) visual recognition of letters, words and phrases; 3) calculations; 4) colours and faces; 5) language (Christensen, 1974). The LNI-1 is designed not only to assess the general pattern of change in function after brain injury or damage, but to assess the neurodynamics changes underlying the change as well.

Judgement of Line Orientation Test. The JLO test was used to measure spatial perception and orientation (Benton et al., 1994). A card with 11 lines at different angles (making the shape of a semi-circle or fan) is presented to the participant, along with another card with only two lines on it. The participant is required to match the angle of the lines on a separate card with one of the 11 lines.

Facial Recognition Test. The purpose of the FRT was to assess the participant's ability to recognise unfamiliar human faces (Benton et al., 1994). The FRT uses photographs of faces (with the hair and clothing shaded out), and consists of three trials: First, the participant was required to identify a front-view photograph of a single face from a display of six front-view photographs of different faces; second, a front-view photograph had to be matched to 3 three-quarter-view photographs of the same face, that were mixed into a display of 6 three-quarter-view photographs; the third trial required the participant identify a single front-view photograph under different lighting conditions (within the photographs).

Boston Naming Test. Visual confrontation naming ability was assessed using the BNT-2 (Kaplan et al., 2001). The test is comprised of 60 line drawings and the participant is required to verbally identify each item. The line drawings are of objects that range from simple, frequently used words (e.g. comb) to words that are rare (e.g. abacus). In this study,

the BNT-2 was also used to test language ability and to ensure that the participant was not aphasic.

Constructional Praxis

WAIS-III Blocks. This subtest was chosen to assess perceptual organization and constructional praxis (Weschler, 1997a). The participant is required to use blocks to replicate models or pictures of two-colour designs.

Rey-Osterrieth Complex Figure. The ROCF test is used to assess visual-spatial constructional ability, immediate visual memory, and long-term visual memory (Rey, 1941; Osterrieth, 1944). The ROCF scoring system used was developed by Guyot and Rigault (1965) and includes scoring a copy trial, an immediate recall trial and a delayed recall trial after approximately 30 minutes. The participant was required to copy the figure as accurately as possible from the original drawing. She was required to finish in no more than 5 minutes. Immediately following the copy trial, she was required to draw the figure again from memory. A third recall trial was requested of her after a 30 minute delay.

Short-term memory

The subtests used to assess short term memory were all chosen from the Weschler Memory Scale (WMS-III; Weschler, 1997b).

Spatial Span. The Spatial Span was used to assess visual short-term memory. The subtest is comprised of two trials: First, the examiner touches a sequence of blocks and the participant is required to repeat the pattern in the same order. In the second trial, the participant is required to point to the blocks in the reverse order.

Digit Span. The Digit Span was used to test that the participant had intact audio-verbal short-term memory. The test required the participant to verbally repeat strings of digits of increasing length in the same order that she received them (forward) and the reverse order (backward).

Visual Reproduction 1. This subtest was used to assess visual short memory. During the test, the participant is shown an abstract figure for a brief amount of time (approximately

10 seconds) and is required to immediately reproduce it. The figures that the participant is required to reproduce become more complex after each successful trial.

Visual and verbal memory

Benton's Visual Retention Test. The BVRT-5 was used to assess long-term visual memory (Sivan, 1992). The test consists of 10 cards with geometric figures that increase in complexity (the last two cards include a smaller peripheral figure as well). The multiple choice version of the test was administered, where the participant had to choose the presented design from a four-card display. The multiple choice administration of this test is used to measure a participant's visual recognition.

The Babcock Story Test. This test was used for the evaluation of long-term verbal memory (Babcock, 1930; Babcock & Levy, 1940). A detailed story was narrated to the participant and she was then required to recall the details she could remember immediately after. The same story was then read a second time and the participant was again required to repeat all the story to the best of her ability.

The Bicycle Drawing Test. The BDT was included for the purpose of assessing revisualisation, and the participant was asked to draw a bicycle from memory, without the aid of a copy (Lezak, 1995).

Appendix B: Standard Definitions for Sleep Macro- and Microstructure Measurements

Sleep Macrostructure

Based on the American Association of Sleep Medicine (AASM) standard criteria, sleep is divided into two states with independent functions and controls: *Non-REM* and *REM* sleep (Chokroverty, 2009). Ideally, NREM and REM alternate in cycles that last 90-110 minutes on average. Furthermore, a sleep cycle is defined by the end of a REM period. In middle-aged adults, 4-6 sleep cycles are usually present in a normal sleep period; however, in elderly adults (>65 years) there may be as few as three sleep cycles in a sleep period of the same length. Normally, the first two sleep cycles mostly contain N3 sleep, which is reduced or absent in subsequent sleep cycles (Chokroverty, 2009). In contrast, REM sleep is increased from the first to the last sleep cycles, with the longest REM period dominating the last sleep cycle.

Non-REM Sleep. NREM sleep accounts for 75-80% of sleep time in adults. According to the AASM scoring manual, on the basis of electroencephalographic (EEG) criteria, NREM sleep can be subdivided into three stages: N1, N2 and N3. Stage 1 sleep (N1) occupies 3-8% of total sleep time; Stage 2 sleep (N2) occupies 45-55%; and Stage 3 sleep (Slow wave sleep; N3) comprises 15-20% of total sleep time. Stage 1 sleep facilitates the transition from wakefulness to sleep, and is identified when alpha rhythm diminishes to less than 50% of an epoch (30 seconds of polysomnographic recording time), that is intermixed with slower theta and beta waves. After approximately 12 minutes Stage 2 (N2) begins and lasts for about 30-60 minutes, before slow wave sleep (SWS; N3) occurs. Slow wave sleep then briefly returns to N2 sleep before progressing to REM (Chokroverty, 2009).

REM Sleep. REM sleep accounts for 20-25% of a person's total sleep time. Physiologically, it is characterised by "bursts of [rapid eye movements] in all directions... phasic swings in blood pressure and heart rate, irregular respiration, spontaneous middle ear muscle activity, myoclonic twitching of the facial and limb muscle, and tongue movements" (Chokroverty, 2009, p. 8). In addition, EEG recordings consist of a low amplitude, fast pattern of beta waves, mixed with a small amount of theta. The first REM period lasts a few minutes and then progresses to N2, which is followed by N3, before the next REM period commences.

Sleep Microstructure

The microstructure phenomena that are important to this study are arousals. Indeed, arousals can be defined as transient phenomena that result in fragmented sleep without behavioural waking (p. 10). Specifically, an arousal can be scored during REM, N1, N2, or N3 if there is an abrupt shift in the EEG frequency that is characterised by a 3 to 14 second intrusion of alpha, beta, or theta waves (but not spindles or delta). Arousals are expressed as a number per hour (Arousal Index; AI) and in middle aged adults it is normal to have an AI of up to 10 (Chokroverty, 2009). However, it should be noted that in the elderly arousals are more prevalent and an AI of 15 is considered normal (Boselli et al., 1998).

Appendix C: Statistical Null and Alternate Hypotheses

The following null and alternate hypotheses were tested using standard z-tests, in order to test whether our participant's sleep parameters were significantly different from the healthy age-matched normative control group.

H₀: the Participant will have WASO equal to healthy age-matched controls

H₁: the Participant will have increased WASO compared to healthy age-matched controls

H₀: $\mu_1 = \mu_2 = 68$

H₁: $\mu_1 < \mu_2$

H₀: the Participant will have equal REM, N1, N2, and N3 when compared to healthy controls.

H₁: the Participant will have different REM, N1, N2, and N3 when compared to healthy controls.

H₀: $\mu_1 = \mu_2 = 86$; H₀: $\mu_1 = \mu_2 = 32$; H₀: $\mu_1 = \mu_2 = 234$; H₀: $\mu_1 = \mu_2 = 54$

H₁: $\mu_1 \neq \mu_2$

H₀: the Participant will have equal arousals (during REM and NREM) compared to healthy age-matched controls.

H₁: the Participant will have unequal arousals (during REM and NREM) compared to the healthy age-matched controls.

H₀: $\mu_1 = \mu_2 = 165$; H₀: $\mu_1 = \mu_2 = 17$

H₁: $\mu_1 > \mu_2$

Appendix D: 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: _____

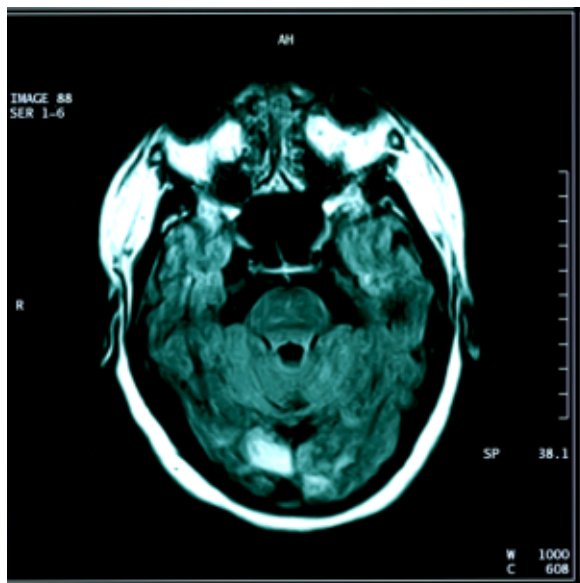
Signature of principal researcher: _____ (name)

Date: _____

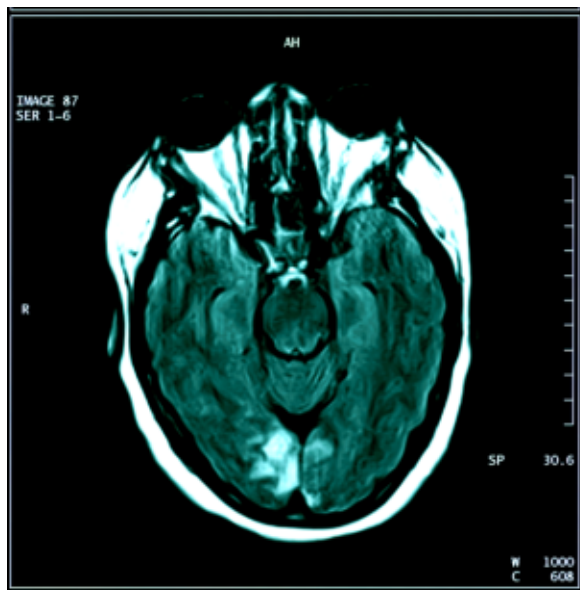
Appendix E: Magnetic Resonance Images

Figure 1. Axial magnetic resonance images (A - D) show acute infarcts in the lingual, fusiform and parahippocampal gyri bilaterally, more extensive and deep on the right. Specifically, BA 17, 18 and 27 have been affected in the left hemisphere and BA 17, 18, 19 and 27 in the right hemisphere. Images E – F also show a lacune in the posterior nucleus of the left thalamus.

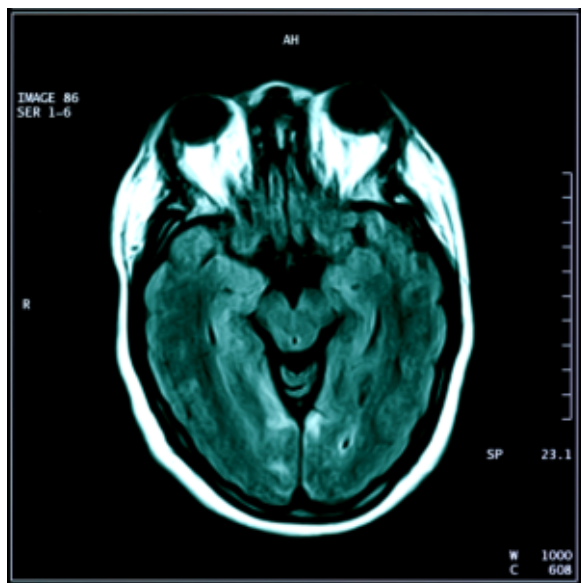
A



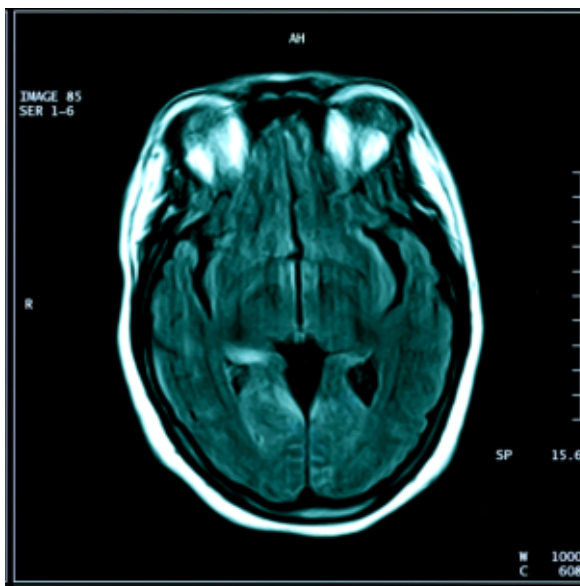
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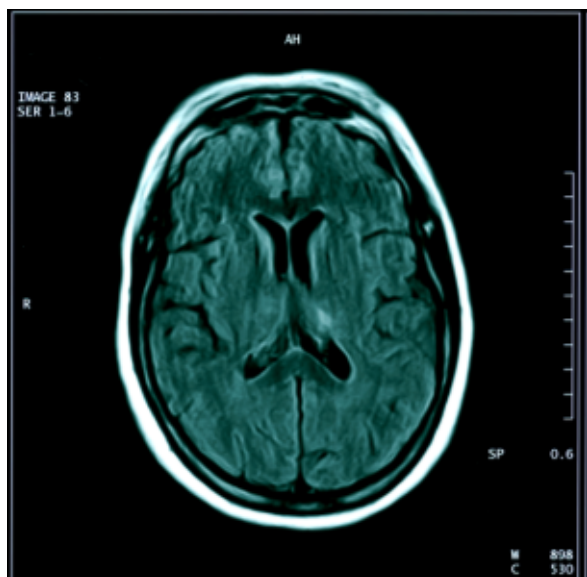
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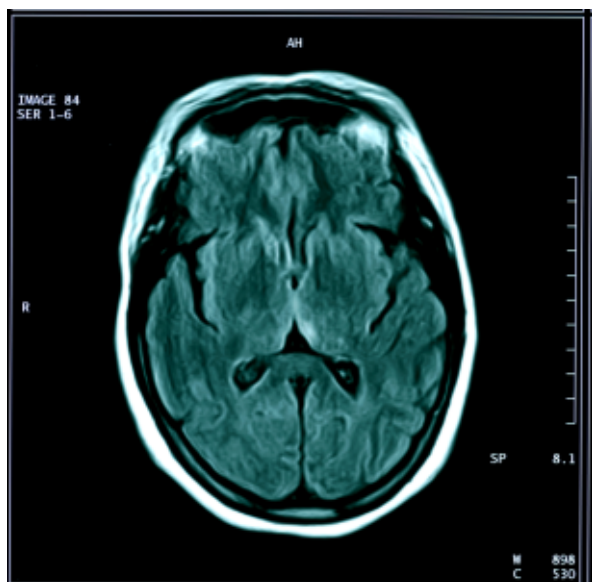
D



E



F



Appendix F: Transcripts for Subjective Dream Recall during REM

The dialogue during the first semi-spontaneous REM awakening (4:34 am) went as follows:

Researcher: "Hello."

Participant: "Mmm.."

R: "What were you dreaming about?"

P: "Hmmm....nothing."

R: "Were you dreaming?"

P: "No, uh uh."

R: "No dreams?"

P: "No."

R: "Ok, you go back to sleep."

The second semi-spontaneous awakening was at 5.42 am, and went as follows:

Researcher : "Hello."

Participant : (eyes open).

R : "What were you thinking about?"

P : (shakes head)

R : "Nothing?"

P : "Hmmm..."

R : "Were you dreaming?"

P : "(shakes head)

R : "No?"

P : "No."

R : "Ok, go back to sleep."

Appendix G: Participant's Subjective Recall of Second Sleep Laboratory Night

(Memory of first night at sleep laboratory 25/08/2010)

“I thought...I didn't absolutely hear him say Gatesville. That's why we came to Grootte Schuur. We sat here 'til 8 O'clock at the reception area there (indicates). Then I said No...Nobody's phoning, so we went home. I got into bed. Then at 10 O'clock [...husband interrupts] No it was before 10 O'clock because we got there at 10. Before 10 O'clock Brian called and said it's at Gatesville; we must come straightaway now. We can still make it. So I got up and got dressed and we went to Gatesville at 10 O'clock. Then Brian started preparing me for the sleep. He put all the gadgets on my face and my head and there was a monitor her (points to chest) and round my waist. {The monitor box was here (indicates left side)}. Then I was *supposed* to have gone to sleep. But I couldn't sleep. I usually sleep very quickly, but I just couldn't that night. Then just before Prof came to wake me up, I think I had just dozed off. I had just started to sleep that time when you came to wake me. You said I must go back to sleep, so I fell off back to sleep again. Then you woke me up again. Then I fell off to sleep again. Then I woke up at 6 O'clock when the sister came in. Then I went home.”

{key: said later}

(Description of room in sleep laboratory)

“It was just one room. With a bed and a cupboard and a window, with blinds. There was a camera in there too. It was a small room, I don't know the measurements. It was beige. Outside the room was like a ward with beds. There were three that side and three this side; about six.”

(Description of Brian- the sleep technician)

“He was from Durban. He asked such a lot of questions. He asked me how old my daughter was. Then I asked him if he was married; I was worried he was looking for a girlfriend. He was an Indian guy.”