Do Dreams Protect Sleep? Testing the Freudian Hypothesis of the Function of Dreams

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DECLARATION

Compulsory Declaration: This work has not been previously submitted in whole, or in part for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation for the work, or works, of other people has been attributed, and has been cited and referenced.

Signature: _____ Date: _____

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ABSTRACT

A review of the literature indicates that a physiological function for dreaming has not yet been empirically established. Based on recent findings regarding the neural correlates of dreaming, this study tested the Freudian hypothesis that dreams protect sleep. In order to do this, sleep architecture in patients who had experienced dream loss as a result of thrombotic stroke in the region of the posterior cerebral arteries was compared with that of patients with the same pathology who had not experienced dream loss. Using medical records, structural neuro-imaging, clinical interviews, neuropsychological testing, analysis of subjective sleep quality, and polysomnographic data collected over two consecutive nights in the sleep laboratory, two non-dreaming and three dreaming cases were studied. Analysis of the individual case studies indicates that sleep was disrupted in both non-dreaming cases. In addition, mean comparisons of the non-dreaming and dreaming patients indicates that the quantity of sleep was greatly reduced in the non-dreaming patients on the first night in the sleep laboratory, and that sleep was substantially more disturbed in the non-dreaming patients on both nights, demonstrated by an increase in the number and frequency of arousals and micro-arousals. These results provide preliminary support for the hypothesis that dreams protect sleep.

Keywords: dreaming; dream loss; disturbed sleep; efficiency of sleep; neural correlates of dreaming; occipital stroke; polysomnographic data

LITERATURE REVIEW

The discovery of REM sleep in 1953 (Aserinsky & Kleitman, 1953) prompted an explosion of research into sleep and dreaming. REM sleep was found to be characterised by bursts of rapid eye movement with accompanying loss of muscle tone and high levels of cerebral cortical activation that are similar to that of the waking state. Further investigation revealed a high correlation of REM sleep and dreaming as approximately 80% of awakenings from REM sleep elicited positive dream reports while only 10% of awakenings from non-REM sleep elicited positive dream reports (Dement & Kleitman, 1957). In light of this, REM sleep and dreams were conflated as being one and the same process.

In the wake of this discovery, many different theories emerged that attempted to explain why people dream. It was theorised that dreams have an integrative function for organising arousal information (Breger, 1967). Through a process of 'unlearning', it was hypothesised that dreams function to remove undesirable cell interactions in the cerebral cortex (Crick & Mitchison, 1983). Dreams were even theorised to be simulations of threatening events in order to rehearse threat avoidance behaviours (Revonsuo, 2000).

Some even theorised that dreams do not have a function. Decades of research has focused on Hobson's 'activation-synthesis hypothesis' that equates the dream experience to the naturally occurring phenomena of REM sleep (Hobson & McCarley, 1977). This model proposed that dreams are meaningless representations that are passively synthesised in the forebrain in an attempt to organise the surge of inputs that are actively generated by the cholinergic pontine brainstem mechanisms that drive REM sleep (Hobson *et al*, 2000).

However, none of these theories have provided empirical evidence for a function for dreams (Solms & Malcolm-Smith, 2009). Furthermore, the discovery that REM sleep and dreams are controlled by separate brain mechanisms (Solms, 2000) cast serious doubt on the premise for many of these theories. In the light of abundant evidence that demonstrated dreams and REM to be doubly dissociable states (Bischof & Bassetti, 2004; Poza & Marti Massó, 2006; Foulkes, 1962; Foulkes & Vogel, 1965; Jus *et* al, 1973; Solms, 2000), it is necessary to separate scientific exploration for a function of dreams from theories of the function of REM sleep. Thus, analysis of the distinct brain mechanisms of dreaming, separate from those that drive REM sleep processes, provides new opportunities that shed light on the function of dreams.

Evidence for a 'dream system' in the brain

In the last few decades, there have been overwhelming attempts to establish the neural correlates of dreaming. Through this research, several converging lines of evidence have indicated two areas of the brain that have been shown to play a vital role in dreaming: posterior cortical regions and the white matter of the ventro-mesial quadrant of the frontal lobes.

Posterior cortical lesions

Lesion studies have indicated that areas of the posterior cortex are involved in the generation of dreams. However, precise localization of the lesion responsible for loss off dreaming has not been conclusively established.

Solms (1997) studied 361 patients suffering from a range of neurological illnesses. Using clinical interviews, CT, and MRI scanning, correlates between cessation of dreaming and specific lesion sites were established. It was established that the cessation of dreaming was not due to memory disorder. However, even if the patients had experienced memory impairment, a range of studies have confirmed that there is no empirical evidence to support the view that impaired memory can provide a sufficient explanation for a total lack of awareness of dream experience (Yu, 2007). With this in mind, Solms identified that damage in the region of the parieto-temporo-occipital (PTO) junction was one of the two sites that led to a complete cessation of dreaming. Solms concluded that the inferior parietal lobule was the critical aspect of this lesion site.

While damage to the PTO junction was shown to result in loss of dreaming, subsequent meta-analysis of Solms's findings indicated that lateral occipito-temporal lesions, not inferior parietal lobule lesions, were the critical damage responsible for loss of dreaming (Yu, 2001).

Further evidence that posterior cortical regions are involved in the generation of dreams was provided by a neuropsychological study that assessed dreams in eight patients with posterior lesions (Yu, 2006). One of these patients experienced a complete cessation of dreams. While this indicated that damage to this area could lead to a cessation of dreams, it also indicated that patients with lesions in this area do not always experience dream loss.

Thus, lesions studies have indicated posterior cortical regions to be involved in the generation of dreams, but precise localization of lesion site has not been established. However, two recent case studies have provided comprehensive data that documented that damage to the medial occipital lobes led to complete cessation of dreaming.

Bischof and Bassetti (2004) published the first case of complete cessation of dreaming with full clinical, neuropsychological, neuroimaging, and polysomnographic documentation. Their case study was a single case where bilateral occipital stroke led to a cessation of dreams. Similarly, Poza & Massó (2006) published a single case where cessation of dreams occurred following a unilateral left tempero-occipital hematoma.

While posterior cortical regions, in particular the occipital lobes, have therefore been shown to be central to the experience of dreams, it seems reasonable to assume that the loss of dream experience in such cases would be attributable to loss of the capacity to perceptually represent dream imagery. Solms (1997) reviewed closely related findings and presented evidence for this view. Solms's view was that damage to the second area, the white matter of the ventro-mesial quadrant of the frontal lobes, would disrupt dreaming due to a loss of motivational capacity.

Ventro-mesial frontal lesions

Lesion studies showed that bilateral damage to the ventro-mesial frontal white matter led to a complete cessation of dreaming (Solms, 1997). As with the posterior lesion site, this conclusion was based on his own clinico-anatomical observations in addition to his review of the world literature.

Other evidence for a 'dream system' in the brain

Further evidence that deep frontal limbic structures, as well as posterior cortical regions, are centrally involved in the generation of dreams was provided by PET studies. Braun *et al*. (1997) measured regional cerebral blood (rCBF) flow throughout the sleep-wake cycle. It was concluded that areas of the brainstem, thalamus, and basal forebrain were highly activated during REM sleep. In particular, it was emphasized that basal ganglia activation was increased during REM sleep. Increased activation was also reported in the insula, cingulate, and mesial temporal cortices. rCFB was shown to be significantly higher in the visual association areas of the fusiform and lateral occipital gyri during REM sleep.

Similarly, Nofzinger *et al*, (1997) measured regional cerebral glucose utilization in the forebrain during REM sleep using PET scans. The authors reported high levels of activation in the lateral hypothalamic area, septal area, ventral striatum, infralimbic cortex, prelimbic and orbitofrontal cortex and the anterior cingulate cortex. The frontotemporal operculum, insula, parahippocampal gyrus and left amygdala were also activated. Thus, the authors argued that basal forebrain-hypothalamic motivational and reward mechanisms were highly activated during REM sleep.

Maquet *et al.* (2006) used PET scans to measure rCBF during REM sleep. rCBF was shown to be positively correlated with REM sleep in the pontine tegmentum, left thalamus, both amygdaloid complexes, anterior cingulate cortex and right parietal operculum. Given that REM sleep awakenings resulted in positive dream reports, the authors argued that this distribution of rCBF might have reflected dreaming activity. In particular, they argued that amygdalo cingulate coactivation might have accounted for the emotional and affective aspects of dreams.

Thus, PET studies have confirmed that both posterior cortical and frontal limbic structures are highly activated during REM sleep. Given that REM sleep and dreams are known to be highly correlated (Dement & Kleitman, 1957), PET studies have given evidence to support the view based on lesion studies that both of these regions are involved in the generation of dreams.

Pharmacological studies also supported the view that the ventro-mesial quadrant of the frontal lobes is involved in the generation of dreams. Most notably, Hartmann *et al.* (1980) found that L-Dopa intensified the bizarreness, emotionality and vivacity of dreaming. This supported earlier findings that chronic levodopa therapy, which increases forebrain dopamine concentrations, was responsible for the occurrence of new dream phenomena (Sharf *et al,* 1978).

Similarly, Parkinson's disease, which is known to result in depletion of dopamine in certain forebrain structures, was shown to be associated with a complete cessation of dreaming (Sandyk, 1997). At the same time, Sandyk reported that treatment with extracranial applications of AC pulsed electromagnetic fields enhanced DA activation in the mesolimbic system in a Parkinson's patient, and that this activation restored dreaming.

In addition, Yu (2007) studied frequencies of dream experience in 55 psychiatric patients on dopamine antagonist therapies. He observed that 30% of the subjects experienced a significantly lowered frequency of dream recall. While this was only a modest result, it did support the findings discussed above to the effect that a dopaminergic system is responsible for the generation of dreams.

Single cell recordings from dopaminergic neurons in the ventral tegmental area gave further evidence that the ventro-mesial frontal dopamine pathways were involved in the generation of dreams. Dahan *et al.* (2007) measured the firing pattern of dopamine neurons in the ventral tegmental area of 12 unanesthetized head restrained rats over their sleep-wake cycle. Prominent burst firing of dopamine cells during REM sleep was observed. The most significant finding of their study, however, was the close similarity between dopamine

activation in REM sleep and dopamine activation during rewarding experiences during wakefulness such as feeding. The authors suggested that this indicated an internal generation of rewarding experiences during sleep. Further investigation of this link between dopamine reward activation during REM sleep and during wakefulness will be discussed at length in the following sections of this dissertation.

Lastly, a recent micro-dialysis study has provided further evidence for the view that frontal dopamine pathways are involved in the generation of dreams. Using intracerebral microdialysis from freely moving rats, Léna *et al.* (2005) measured the concentration of selected neurotransmitters across the sleep-wake cycle. Dopamine concentrations were found to be significantly higher in the medial prefrontal cortex and nucleus accumbens in REM sleep in comparison to slow-wave sleep.

Thus, the notion of a 'dream system' in the brain is now firmly established. Two distinct areas have emerged that are implicated in the generation of dreams: posterior cortical areas, in particular the occipital lobes, and the dopaminergic pathways of the ventro-mesial frontal white matter. Based on these findings, Solms (2000) proposed a model of how dreams are generated in the brain. His argument, supported by the findings of the neural correlates of dreaming that were reviewed above, was that dreams were generated by volitional urges that arose from the increase of activation in the dopaminergic circuit in the frontal limbic region, and that these urges were redirected towards posterior cortical regions where they were represented in their virtual form as dreams.

In addition to being involved with the generation of dreams, there is also abundant evidence that the frontal-limbic dopaminergic system is involved in the generation of hallucinations (Gottesmann, 2004; Manni, 2005). Thus, a connection was made between dream experience and hallucinatory experience in waking life. This dopaminergic system in the brain, however, was not solely shown to be involved in dreams and hallucinations; it is also known to be a highly active system during normal motivated activity in waking life.

The 'SEEKING system'

Panksepp (1998), in extensively studying mammalian emotions, identified a system in the brain that motivates and drives mammalian behaviour. This system he called "the SEEKING system" (pg. 144). The 'SEEKING system' was identified as the basic neuronal circuit that drives people to search, investigate, and interact with the environment. Simply put, it is the system that "leads organisms to eagerly pursue the fruits of their environment" (pg. 145).

Through a series of extensive studies on this system, Panksepp identified this system to be anatomically comprised of the dopaminergic circuits of the lateral hypothalamic corridor. Specifically, this circuit was shown to involve the ventrolateral regions of the diencephalon and the medial forebrain bundle of the lateral hypothalamus. The core of this system was identified as the dopaminergic circuits which extended from the ventral tegmental area to the nucleus accumbens (the ventral basal ganglia), areas of the frontal cortex, and amygdala.

Thus, from the literature reviewed above, it is clear that the 'SEEKING system' that Panksepp identified is the frontal mesial limbic system that was shown to be involved in the generation of dreams. As this system has thus been shown to be activated in both dreams and with 'SEEKING' behaviour while awake, a paradox is created. At first glance, it seems impossible that one is able to remain asleep while a system that would usually motivate one to actively engage in SEEKING behaviour is being so highly activated. The logical conclusion drawn from this seeming paradox then is that dreams function as a virtual SEEKING activity. Thus, the hypothesis drawn from these findings is that dreams protect sleep by creating a virtual reality where motivational behaviour can be carried out without the need to wake up. This protection is important as sleep deprivation was shown to have a range of short-term and long-term consequences including impaired attention and concentration, coronary heart disease, high blood pressure, and depression (Chokroverty, 2010). In this sense, it is theorised in this study that dreams provide an adaptive advantage as they allow one to remain asleep despite the overwhelming surge of neuronal activity that is normally associated with REM sleep and with active 'SEEKING' in waking life.

We, however, are not the first to make this claim.

Freud's theory of dreams

Sigmund Freud (1900), in *The Interpretation of Dreams*, outlined an extensive theory of dream function that closely resembles the above hypothesis, derived from recent findings regarding the neural mechanisms of dreaming. His theory was that dreams are part of a

process of wish-fulfilment. Through a fulfilment of unconscious desires and wishes, of which the desire to remain asleep is the most pertinent, a dream's normal function, Freud argued, was one of a "guardian of sleep" (pg. 580). This theory was summarised in Freud's later work *A Metapsychological Supplement to the Theory of Dreams* (1917):

> "A dream tells us that something was going on which tended to interrupt sleep, and it enables us to understand in what way it has been possible to fend off this interruption. The final outcome is that the sleeper has dreamt and is able to go on sleeping; the internal demand which was striving to occupy him has been replaced by an external experience, whose demand has been disposed of" (pg. 223).

Thus, Freud argued that the dream is generated by wishes stemming from appetitive or 'libidinal' urges (Solms, 2000) which is indirectly supported by the previously discussed findings that dreams were shown to be generated by the reward driven mesolimbic system. However, more importantly, Freud argued that the dream functions to fulfil these wishes, thereby allowing the dreamer to remain asleep.

As Freud's dream theory is one of the fundamentals upon which psychoanalysis is based, finding an empirical basis for his hypothesis would add greater scientific credibility to the field of psychoanalysis.

However, there are those who have argued that psychoanalytic theories are not capable of scientific investigation. In *Conjectures and Refutations* (1963), Karl Popper famously considered psychoanalysis a "pseudo-science" (pg. 33). His argument, which has become the benchmark of scientific investigation, is that theories are only testable in so far as they are falsifiable. Psychoanalytic theories, he argued, do not fulfil this criterion as "there was no conceivable human behaviour which could contradict them" (pg. 37).

Advances in the field of neuropsychology, however, have opened up the possibility of falsifying a fundamental psychoanalytic theory. Since the earliest reports of dream loss following brain damage more than 100 years ago (Charcot, 1883; Wilbrand, 1887), it is widely acknowledged that dream loss can occurr as a result of neurological disease (Bischof & Bassetti, 2004; Doricchi & Violani, 1992; Dumont *et* al., 2007; Epstein, 1979; Poza & Massó, 2006; Solms, 1997). Thus, based on the literature reviewed above, the discovery of patients who do not dream has provided an opportunity to test a fundamental psychoanalytic theory.

The literature reviewed above indicates that posterior cortical regions and the white matter of the ventro-mesial quadrant of the frontal lobes was shown to be the two regions

involved in the generation of dreams. Based on Solms's (2000) model of dream generation, it can be theorised that patients who have dream loss as a result of damage to posterior regions, would be unable to divert the surge of inputs that are generated by increased activation of the dopaminergic circuits in the basal forebrain pathways that has been shown in REM sleep. As these inputs from increased activation of the ventro-mesial frontal whiter matter would not be able to be diverted, it can be theorised that patients with dream loss as a result of posterior lesions would show difficulties maintaining sleep. Therefore, it can be argued that by testing the quality of sleep in patients who had experienced dream loss as a result of posterior lesions, in particular occipital lesions, it would be possible to test the hypothesis that dreams protect sleep.

By doing so, it would be possible to test to test the Freudian dream theory that dreams function as guardians of sleep. Some have argued that it is this interaction of neuropsychological investigation with psychoanalytic hypotheses that finally brings hope to the search for a function of dreams (Ruby, 2011). Recent empirical finding have already provided support for this method of investigation.

Quality of sleep in non-dreamers

Bischof and Bassetti (2004) published a case study of a patient who had ceased dreaming after bilateral occipital stroke. Their patient reported cessation of dreams from three days after the stroke and only experienced another dream 14 weeks later. Four all-night polysomnographics were performed: five days after the stroke, three weeks after stroke, four weeks after stroke, and six weeks after the stroke. During the last polysomnograph, the patient was awakened five minutes after the onset of the first REM period and 10, 15, and 20 minutes into the following REM periods to confirm that no dreams were occurring. Bischof and Bassetti discovered that the patient documented normal REM amounts, REM density, and REM latency. However, they also reported, without apparently noticing its potential significance, that the patient experienced a decrease in sleep quality. This abnormal sleep architecture they called "sleep maintenance insomnia" (pg. 585). This is consistent with our previously discussed hypothesis that dreams function as guardians of sleep as it demonstrated how loss of dreaming from occipital lesions resulted in a decreased efficiency of sleep.

Similarly, Poza and Massó (2006) recently reported a case of a 24-year old man who experienced total cessation of dreaming following a unilateral left tempero-occipital hematoma that resulted from a cerebral arteriovenous malformation (AVM). Polysomnographics revealed normal REM sleep characteristics. However, the authors

reported that the patient complained of poor quality of sleep following the neurological damage. Their report was as follows:

> "After the brain injury the patient started complaining about his bad quality of sleep. He spent many hours in bed, seemingly asleep, but used to wake up with the feeling of not sleeping much. However, there were no sign of hypersomnia during the day. To improve the quality of sleep, lorazepam and trazodona were administered with no further improvement. After a while the patient was able to explain that his problem was a lack of dreaming and not a difficulty to sleep" (pg. 153).

This gives support for the hypothesis that dreams protect sleep by demonstrating how loss of dreaming from occipital lesions resulted in decreased efficiency of sleep.

Further support for this hypothesis was also provided by a preliminary study conducted by Solms (1997). His study compared subjective reports, in patients with focal neurological lesions, on whether they experienced dreams or not, with subjective reports on their quality of sleep. A significantly lower level of quality of sleep was reported by patients who did not dream. These findings indicated the possibility that objective measures of a reduced quality of sleep would be found when recording the sleep patterns of patients who had suffered dream loss as a result of posterior lesion.

Therefore, a review of the current literature on dream research indicates that a function of dreams has not been empirically established. Based on the findings of the neural mechanisms of dreaming, it was theorised in this study that by testing the efficiency of sleep in patients who had experienced dream loss as a result of posterior lesions, in particular occipital lesions, but whose frontal limbic structures were preserved, it would be possible to test the hypothesis that dreams protect sleep. By this method, it was theorised in this study that it would be possible to test the Freudian hypothesis that dreams protect sleep.

RATIONALE FOR RESEARCH, AIMS, AND HYPOTHESES

The literature reviewed above indicates a significant gap in dream research as empirical evidence for a function for dreams had not been established. Despite recent developments in finding the neural correlates of the dream process, no one has been able to link these correlates to a function of dreams. This study aimed to fill this gap by *testing the hypothesis that dreams protect sleep*. By doing so, this study hoped to contribute to the search for *a scientific explanation for why dreams occur*.

At the same time, this study *tested the Freudian dream theory*. Freud's theory that dreams are the 'guardians of sleep' is identical to the hypothesis that was concluded based on the recent findings of the neural mechanisms of dreaming. As such, this study not only has the potential to contribute towards establishing an empirical function for dreams, but it also provides an opportunity to show that Popper was overly pessimistic about the prospect of testing psychoanalytic theory.

In order to achieve these aims, this study looked at patients who did not dream as a result of occipital lobe damage to see if their sleep was disrupted. The reason behind this aim was as follows: Patients with loss of dreaming due to posterior cortical lesions would be unable to divert the frontal-limbic motivational urges that occur during sleep in the form of perceptual imagery. The posterior cortical lesion site selected for this purpose was the territory of the posterior cerebral artery (PCA), for the reason that Bischof and Bassetti (2004) and Poza & Massó (2006) had observed sleep maintenance insomnia and subjectively disrupted sleep in such cases with damage to this site. Also, their cases are the most comprehensively studies to data.

Thus, in order to test whether dreams protect sleep, this research tested the following hypothesis:

> H_0: Patients who have cessation of dreams following posterior cerebral artery (PCA) stroke, but who have intact brain arousal and motivational systems, will show reduced efficiency of sleep compared to patients with PCA stroke who do dream.

DESIGN AND METHODS

Design

A quantitative multi-case study was designed to test the hypothesis that dreams protect sleep. Data was collected for neurological patients who did not dream. In addition, data was collected for neurological patients who did still dream. Each patient was analysed as a separate case study in order to understand the relationship between preservation and lack of dreaming with quality of sleep. In addition, the efficiency of sleep was compared between non-dreaming patients and dreaming patients.

Setting

The study was conducted in the sleep laboratory at the Cape Sleep Centre, Gatesville Medical Centre, Cape Town. Neuropsychological examination took place at the Cape Sleep Centre, Gatesville Medical Centre, Cape Town and at the neurology ward at Groote Schuur Hospital, Cape Town. Permission for conducting this study was obtained from both institutions.

Participants

All participants were selected by referral from neurological specialists at Gatesville Medical Centre. Seven participants were chosen, while two were excluded as their neuropathology did not match the requirements for this study. All participants had suffered thrombotic infarctions in the posterior cerebral artery (PCA) territory and had corresponding occipital damage. Participants were divided into non-dreamers and dreamers in order to compare sleep efficiency.

Non-dreaming patients (Quasi-experimental group)

The non-dreaming patients consisted of patients who had experienced thrombotic strokes and who had corresponding occipital lesions. Thrombotic strokes were considered preferable for this study as they create more circumscribed damage. Strokes were focal and circumscribed. A strict inclusion criterion was that patients did not dream since their stroke. This was detected by patients' subjective dream accounts and was confirmed in the sleep laboratory by awakening patients during REM sleep and asking them whether or not they were dreaming. A further strict inclusion criterion was that patients documented normal REM cycles, which was also confirmed in the sleep laboratory. Exclusion criteria included the presence of any other sleep or neurological disorder that might have confounded the results, or the use of any medications that could have affected sleep architecture.

Participants were selected by referral from neurological specialists at Gatesville Medical Centre, Cape Town. Due to this method of selection, sample size was highly dependent on the availability of patients with the correct lesions for this study. It was predicted, based on the parietal cases studied by Solms (1997) that the occurrence of such patients was not extremely rare. However, as this study required patients with occipital lobe damage, only two participants met the required inclusion criteria for non-dreaming patients and were analysed in this study.

Dreaming patients (Control group)

The dreaming patients consisted of patients who had experienced thrombotic strokes in the posterior cerebral artery (PCA) territory who had corresponding occipital lesions. Strokes were focal and circumscribed. This method of selection was used to control for variables associated with the experience of neurological damage and corresponding treatments as stroke has been shown to have an impact on sleep quality (Chokroverty & Montagna, 2009) which has resulted in lowered total sleep time and lowered sleep efficiency (Bassetti & Aldrich, 2001). Patients in the control group were also selected from a similar age bracket to patients in the quasi-experimental group to control for the effects that age has been shown to have on sleep quality (Redline *et al.*, 2004). The strict inclusion criterion was that patients subjectively reported normal dreams. This was confirmed in the sleep laboratory by awakening patients during REM sleep and asking them whether they were dreaming or not. Exclusion criteria included the presence of any other sleep or neurological disorder that might have confounded the results, or the use of any medications that could have affected sleep architecture.

Participants were selected by referral from neurological specialists at Gatesville Medical Centre, Cape Town. Due to this method of selection, sample size was dependent on the availability of patients with the correct lesions for this study. Three participants met the required inclusion criteria for the dreaming patients and were analysed in this study.

Materials

Case history

For each patient, a comprehensive case history was taken from the patient's medical records and from clinical interviews. Neurology reports were used to determine the onset, type, and severity of cerebral accident which was further supported by MRI scans, CT scans, and reports. Medical records also provided detailed reports of clinical and neurological symptoms. Clinical interviews were used to screen patients' subjective dream reports and to gain information regarding patients' subjective sleep efficiency. All case information was duplicated in this study in accordance with the APA guidelines for confidentiality and anonymity (American Psychiatric Association, 2005). As such, information was not distorted in any way, but all identifying information was omitted.

Dream recall

The presence, or lack thereof, of dreams since cerebral accident was initially identified by attending physicians. In clinical interviews, patients were asked to give a subjective account of their dreams, or lack thereof, since their stroke. These two methods of investigation were used to screen patients in order to divide them into non-dreaming patients or dreaming patients. The presence, or lack thereof, of dreams was further confirmed by conducting nocturnal REM-sleep interviews during the first night in the sleep laboratory according to sleep stage measured by polysomnograph recordings. Patients were awakened 10 minutes after the onset of the second REM period and 15 minutes after the onset of the third REM period, or were interviewed after spontaneous awakenings during REM sleep. Interviews consisted of brief questions regarding whether or not patients were dreaming and what had been going through their minds.

Neuropsychological investigation

A range of neuropsychological tests were used in this study, which focused on higher visual and spatial perception, visual and verbal short-term memory, and visual and verbal long-term memory. These domains were tested as they involve the necessary functions for intact dream recall and as they are the domains that have traditionally been studied in relation to loss of dreaming. Thus, neuropsychological testing was used to ensure that the necessary functions for dream recall were intact, and to test the cognitive functions that are usually associated with loss of dreaming. The neuropsychological tests that were used in this study adhered to standardised testing manuals and administration and scoring was conducted according to the guidelines of these manuals. In addition, the tests that were used are used daily on an on-going basis in the clinical practice of the neuropsychologists at Groote Schuur Hospital as

part of the Groote Schuur Neuro-Cognitive Screening Battery (Mosdell, Balchin, & Ameen, 2010).

Visuo-spatial perception

Visuo-spatial perception was tested using subtests from Luria's Neuropsychological Investigation that assesses higher visual perception and integration (Christensen, 1974). In particular, Luria's Complex Visual Scenes were focused on. The Boston Naming Test was also used (Kaplan, Goodglass & Weintraub, 2001) which additionally assessed naming.

Constructional praxis

Constructional praxis and perceptual organization was assessed using the WAIS-III Blocks (The Psychological Corporation, 1997) and the Rey-Osterrieth Complex Figure (ROCF; Rey, 1941; Osterrieth, 1944).

Visual and verbal short-term memory

The Digit Span subtest of the WAIS-III was used to assess audio-verbal short-term memory, and visual short-term memory was assessed with Corsi's Blocks (WMS-III; Wechsler, 1997).

Visual and verbal long-term memory

Verbal long-term memory was assessed using the Babcock Story (Babcock & Levy, 1930). Long-term visual memory was assessed using the ROCF which consists of a copy trial, an immediate recall trial, and a delayed recall trial. Long-term visual memory was further assessed by Benton's Visual Retention Test (Sivan, 1992). In addition, patient's ability to revisualize images from memory (Lezak, 1995) was assessed with the Bicycle Drawing Test (BDT), the South African flag, a canary, revisualization of the internal layout of the patient's house, and revisualization of the internal layout of the sleep laboratory approximately one month after the sleep study.

Subjective sleep quality

The Pittsburgh Sleep Quality Index (PSQI) was used to assess subjective sleep quality (Buysse *et al*, 1989). The PSQI is a self-rated questionnaire which assesses sleep quality and disturbances over a 1-month time period. The index generated a global score of sleep quality, as well as seven component scores: 1) subjective sleep quality, 2) sleep latency, 3) sleep duration, 4) habitual sleep efficiency, 5) sleep disturbances, 6) use of sleeping medication, and 7) daytime dysfunction.

Polysomnographic measures

The polysomnographic (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 used in this study, which included: electroencephalogram (EEG; 4 leads, 2 channels), electrooculogram (EOG; 2 channels), and the submental electromyogram (EMG; chin and leg). Chest and abdominal strain gauges, snore microphone, positional marking and finger pulse oximetry were also included. Sleep stages were visually scored for 30-s epochs by a certified polysomnographic technologist according to AASM standard criteria (Hirshkowitz & Sharafkhaneh, 2009). Standard definitions for sleep macrostructure measurements are provided in Appendix A.

In order to measure quantity of sleep, the sleep parameters that were included for analysis were: 1) total sleep time (TST), which records the number of minutes spent in sleep; and 2) percentage of time spent in sleep (% sleep), which records the percentage of time spent in sleep from lights off to lights on.

In order to measure quality of sleep, the sleep parameters that were included for analysis were: 1) total number of awakenings (W); awakening index (WI), which measures the average number of awakenings per hour of sleep time; total number of arousals (A); arousal index (AI), which measures the average number of arousals per hour of sleep time; total number of micro-arousals (M); and micro-arousal index (MI), which measures the average number of micro-arousals per hour of sleep time. Arousal phenomena is characterised by an abrupt shift in EEG frequency that includes alpha, beta, or theta waves (Chokroverty, 2009). In this study, awakenings were defined as arousal phenomena lasting longer than 60 seconds, arousals were defined as arousal phenomena lasting between 15 and 60 seconds, and micro-arousals were defined as arousal phenomena lasting less than 15 seconds.

Additional sleep parameters such as respiratory events, heart rate, leg movements, and snore events were analysed in order to fully understand any sleep disturbances.

Procedure

The identical procedure was applied to the non-dreaming and dreaming patients. Permission from attending physicians and informed consent from each participant was obtained before any data was collected. Participants were informed that the purpose of the study was to document the effects on sleep and dreaming of cerebral vascular accidents, in the hope of advancing our understanding of the function of dreaming. It was made clear to each participant that their participation was voluntary and that they may withdraw from the study at any time. Patient's medical files were analysed in order to determine the onset, type, and severity of cerebral accident and to provide detailed summaries of clinical and neurological

symptoms. Age, sex, and time since stroke were also recorded. MRI scans, CT scans, and accompanying reports were further analysed to confirm lesion site.

The study consisted of three primary components: 1) neuropsychological assessment, 2) subjective analysis of sleep quality and 3) sleep study.

Neuropsychological assessment

Neuropsychological testing took place at the neurology ward at Groote Schuur Hospital. Assessment took place in a quiet, distraction-free room. Patients' subjective reports on dreaming and sleep since onset of stroke were recorded. A range of neuropsychological tests, aimed at testing higher visual and spatial perception, visual and verbal short-term memory, and visual and verbal long-term memory were administered.

Subjective analysis of sleep quality

Subjective reports of sleep quality were obtained during clinical neuropsychological assessment at the neurology ward at Groote Schuur Hospital. In addition, the Pittsburgh Sleep Quality Index (PSQI) was administered to assess subjective sleep quality (Buysse *et al,* 1989).

Sleep study

The sleep study took place over two consecutive nights at the Cape Sleep Centre, Gatesville Medical Centre, Cape Town. Participants were restricted in the use of caffeine-containing liquids and other stimulants. The first night functioned as an orientation night and a confirmation of basic sleep/dream activity. The second night functioned as the experimental night. During both nights, the participants were monitored by the principal researcher and a qualified sleep laboratory nurse, and the polysomnograhic recordings were monitored by a qualified sleep technologist.

First night

The first night functioned as an orientation night to allow participants to adjust to sleeping in a sleep laboratory and to familiarize themselves with the sleep laboratory process. Participants were connected to a polysomnograph and simply asked to sleep as they would normally at home. Nocturnal REM-sleep interviews were conducted to confirm the presence, or lack thereof, of dreaming. Detailed polysomnographics were also recorded to confirm that participants in both groups were experiencing normal REM sleep cycles.

Second night

Participants were once again connected to a polysomnograph and were asked to sleep as they would normally at home. Participants were not awaked by the researcher during the night.

Detailed polysomnographics were recorded in order to measure the quality and quantity of sleep.

In the morning, patients were debriefed and were asked whether they felt that their quality of sleep in the sleep laboratory was similar to their quality of sleep at home. This was done to eliminate the confounding role that a sleep laboratory setting may have on the efficiency of sleep. Patients were thanked for their participation in the study and were compensated in accordance with the participation agreement (see below).

Data Analysis

Each participant was analysed as a separate case study. Detailed information regarding neurological damage, neuropsychological functioning, subjective sleep quality, and sleep data was analysed according to the dependent variable of presence, or lack thereof, of dreams. In addition, data collected for the non-dreaming patients was compared to data collected for the dreaming patients.

Neuropsychological assessment

Neuropsychological was used to provide confirmation that the necessary functions for dream recall were intact, and to test the cognitive functions that have traditionally been associated with loss of dreaming. Scoring of neuropsychological tests was done according to standard scoring procedures outlined by each test manual. Conclusions of neuropsychological functioning were based on performance on tests and overall clinical impression.

Subjective analysis of sleep quality

The Pittsburgh Sleep Quality Index (PSQI) was scored according to the standard scoring procedures outlined by the test manual.

Polysomnographic measures

The polysomnographic recordings were manually analysed and scored at the Cape Sleep Centre, Gatesville Medical Centre, Cape Town by a qualified sleep technologist according to standard sleep guidelines as set by the AASM. Thereafter, the data was compiled into a comprehensive PSG sleep report using Alice © 5 *Respironics* software.

Ethical Considerations

This study 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 granted by the Psychology Department's Research Ethics Committee at UCT as well the Faculty of Health Sciences

Research Ethics Committee at UCT. Data was only collected after ethical approval had been obtained. In addition, consent from the Cape Sleep Centre, Gatesville Medical Centre and the neurology department at Groote Schuur Hospital was obtained before their facilities were utilized.

Informed consent was obtained in writing from each participant before any data was collected (see Appendix B.). The consent form outlined that participation was voluntary, that participants were able to withdraw at any time without negative consequences for themselves, and that all data would be kept confidential and would only be used for research purposes. The consent form also obtained consent for participant's medical folders to be reviewed and for their medical information to be used confidentially.

There were no major risks associated with the administration of the study. However, if participants were uncomfortable at any time it was reiterated to them that they were able to withdraw from the study at any time. Participants were also made aware that there were no direct benefits for participating in this study other than the benefit of identification of sleep disorder if present.

Participants received a monetary compensation of R500 per night spent in the sleep laboratory (approximately £50 or $75).

RESULTS

Case Studies

Case 1: Mrs A

Date of birth: 01/07/1945
Date of CVA: 25/02/2006
Date of sleep study: 25/08/2010 and 26/08/2010

History of stroke

Mrs A, a right handed female, presented to Gatesville Medical Centre, Cape Town, on 25 February 2006 with a history of a sudden onset of blindness and severe headache accompanied by severe dizziness, and pain at the back of the neck.

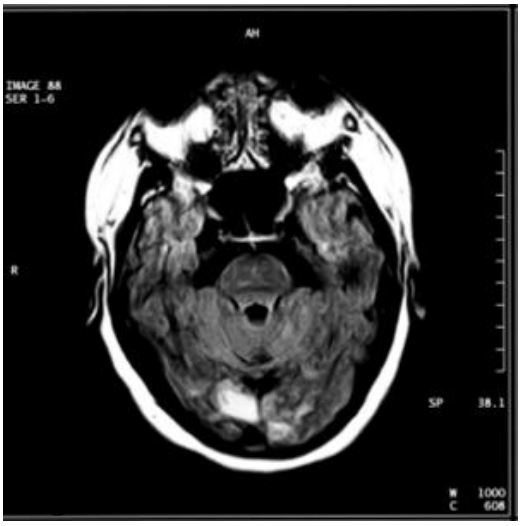
Initial work-up revealed hypothyroidism, micro voltages on ECG, and a thickened myocardium suggestive of an infiltrative process.

Neurological examination five days after initial presentation was performed as Mrs A remained in a confusional state. Neurological examination revealed an abnormal mental state characterized by an axial amnesia with forced confabulatory features, disorientation to time, executive dysfunction characterized by difficulties with complexity and problem solving, and labile mood. Of note was the absence of visual agnosia or aphasia, and general judgement was preserved. Neurological examination further revealed cranial nerve dysfunction with a left superior quadrantanopia, large and small fibre sensory neuropathy, and a mild ataxic gait. No other focal localizing signs were noted. Mrs A was also found to be photophobic, while blurred vision remained a consistent complaint. She emerged from a confusional state and regained the ability to lay down new memories approximately seven days after the initial onset of symptoms.

MRI, taken 14 days after initial onset of symptoms, demonstrated acute infarcts in both occipital lobes (larger on the right) and the right cerebellar hemisphere (Figure 1). High signal was noted on T2 and FLAIR sequences in the thalami bilaterally. MRA revealed a tight basilar stenosis at the confluence of the proximal basilar and vertebral arteries. CT angiogram showed extracranial vasculature to be normal, with no features of dissection noted. Gradient echo sequences showed no evidence of haemorrhage.

Figure 1. Magnetic Resonance Images (FLAIR sequence): Case 1

A



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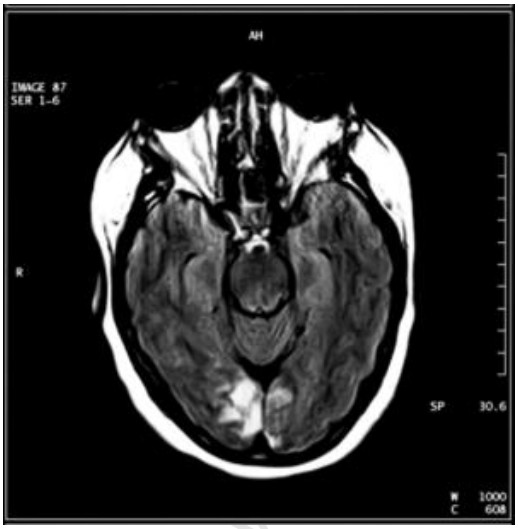

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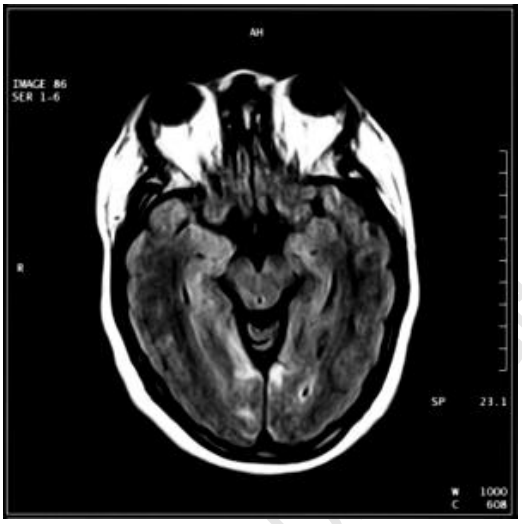

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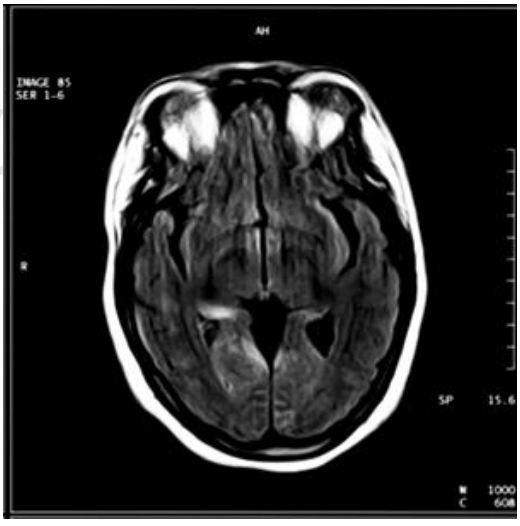

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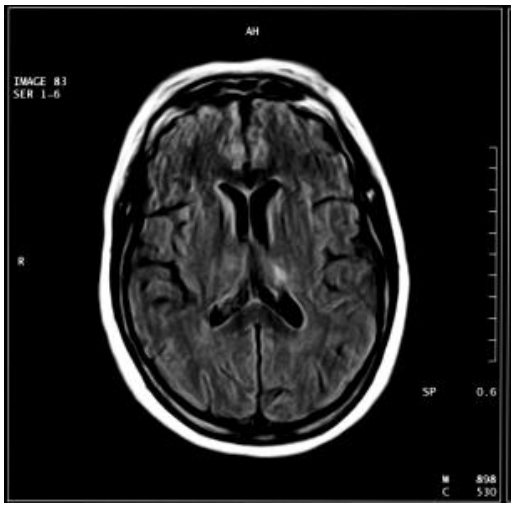

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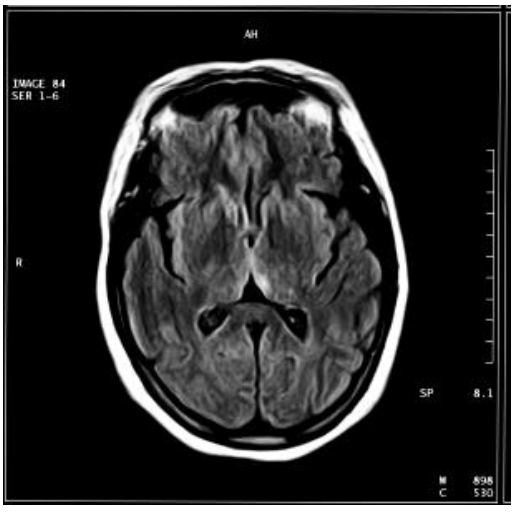


Clinical assessment and scanning techniques thus revealed that Mrs A had suffered bilateral thrombotic infarction in the PCA territory, affecting occipital lobes and thalami.

Neuropsychological assessment

Neuropsychological examination (Table 1) demonstrated that visuo-spatial perception was intact with normal performance on Luria's tests of higher visual perception and integration. Performance on the Boston Naming Test was good (38/40 answers correct).

Mild constructional apraxia was noted, with a poor copy of the Rey-Osterrieth Complex Figure (25/36), and a loss of the overall gestalt. Performance on the WAIS III Blocks was marginally better with 5 consecutive answers correct.

Performance on the Digit span test and Corsi's blocks showed normal visual and verbal short-term memory.

Tests of visual and verbal long-term memory showed a mild new learning deficit. However, revisualization of the sleep laboratory, the sleep technician, the South African flag, a canary, and her husband's face were all excellent, showing revisualization to be fundamentally intact.

In particular, Mrs A was able to recall the sleep laboratory procedure in accurate detail. The following transcript indicates Mrs A's memory of the sleep laboratory procedure recorded one month after the sleep study:

> "I thought…I didn't absolutely hear him say Gatesville. That's why we came to Groote 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."

In addition, Mrs A was able to accurately revisualize the sleep laboratory room where the sleep study had taken place. The following transcript indicates Mrs A's memory of the room in sleep laboratory recorded one month after the sleep study:

> "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."

Therefore, neuropsychological assessment indicated that Mrs A had mild constructional apraxia and a mild new learning deficit. In light of good performance on tests of visuo-spatial perception, short-term memory, and revisualization, as well as overall clinical impression, it was concluded that Mrs A had functions necessary for dream recall intact. In particular, accurate recall of the sleep laboratory procedure and room indicated that Mrs A had the necessary cognitive capacity for dream recall.

Table 1.

Neuropsychological Testing: Case 1

Test	*Score*
Luria's Complex Visual Scenes	8/11
Boston Naming Test	38/40
Rey-Osterrieth Complex Figure	
Copy	25/36
Immediate Recall	4.5/36
WAIS-III Blocks	1-5 correct
Digit Span	
Forwards	6
Backwards	3
Corsi's Blocks	
Forwards	5
Backwards	5
Benton's Visual Retention Test	6/7
Babcock Story	
Trial A	7/21
Trial B	8/21
Revisualization	
South African Flag	normal
Canary	normal
Husband's Face	normal
Sleep Laboratory	normal
Bicycle Drawing Test	normal

Dream recall

Mrs A reported that she had not been able to dream since her stroke. This remained consistent over multiple clinical interviews. She reported that before her stroke she dreamt regularly, especially when she had worries, but that she had not dreamt at all since her stroke. This indicated that complete cessation of dreaming had occurred as a direct result of her stroke, and that dreaming had ceased for 4 years, 5 months since her stroke.

This was confirmed in the sleep laboratory by conducting nocturnal REM-sleep interviews from unprompted awakenings during the 1st and 2nd REM periods. During both interviews, Mrs A reported that she had not been dreaming or experiencing any thoughts or mentations.

On a follow-up interview (4 years, 7 months post stroke; 1 month post sleep study), Mrs A reported that she had experienced dream-like mentation two weeks prior, but was unsure of whether or not it was actually a dream. The following transcript indicates Mrs A's recall of dream-like mentation:

"Mrs A:	I did have a dream but it wasn't really a dream.
Researcher:	When did this happen?
Mrs A:	A couple of weeks ago. My aunt came to me in the dream because my brother-in-law is dying of cancer now.
Researcher:	How do you know it was a dream?
Mrs A:	Because I saw her. To me it meant that she is coming to tell us that my brother-in-law, who is passing away, that his time is near. [This was explained further]
Researcher:	Why do you say it's not actually a dream?
Mrs A:	Because the only thing I remember was seeing my aunt. Then I just had a feeling I had to go to my sister. [Explained what happened when she went to her sister]
Researcher:	Did you just think about her, or feel her presence, or did you see her?
Mrs A:	No, I actually saw her.
Researcher:	How much of her did you see?
Mrs A:	I just saw her face.
Researcher:	What happened next?
Mrs A:	I didn't wake up. The next morning I just remembered that I saw her.
Researcher:	So do you think it was a dream?

Mrs A: No. It wasn't a dream.

Researcher: Please clarify.

Mrs A: Because nothing happened in it. It was just her face. There was no story".

This indicated that Mrs A may have begun to recover dream function at that stage.

PSQI

The results of the Pittsburgh Sleep Quality Index (Table 2) indicated that Mrs A reported that she subjectively experienced a good quality of sleep. Only minor difficulties were noted with subjective sleep quality, sleep latency, sleep disturbances, and day time dysfunction.

Table 2.

Pittsburgh Sleep Quality Index: Case 1

Component	*Score (range 0-3)*	*Score (range 0-21)*
Subjective sleep quality	1	
Sleep latency	1	
Sleep duration	0	
Habitual sleep efficiency	0	
Sleep disturbances	1	
Use of sleeping medication	0	
Day time dysfunction	1	
Global PSQI score		4

Note: Each individual component score has a possible range of 0-3. For each component score, a score of '0' indicates no difficulty, while a score of '3' indicates severe difficulty. The global PSQI score is a summation of the seven component scores and has a posssible range of 0-21. A score of '0' indicates no difficulty, while a score of '21' indicates severe difficulties in all areas.

Sleep study findings

As confirmed by PSG recordings, Mrs A documented REM sleep periods on both nights in the sleep laboratory (Table 3). On the first night, 2 REM periods were documented, creating 2 sleep cycles. Nocturnal REM-sleep interviews as well as unfamiliarity to the sleep laboratory environment could have accounted for the small amount of REM on the 1st night (only 5.7% of total sleep time). 3 REM periods were documented on the second night, creating 3 sleep cycles. REM sleep thus accounted for 20.5% of total sleep time on the second night. REM sleep normally accounts for 20-25% of total sleep time in healthy adults (Chokroverty, 2009), thus showing that Mrs A was able to achieve normal REM amounts on the second night.

Table 3.

REM Cycles: Case 1

		Total	*Cycle*	*REM start*
Night 1	*REM 1*	2.0	308.5	04:33:59
	REM 2	8.0	66.5	05:34:29
Night 2	*REM 1*	12.0	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

A full sleep hypnogram for the first night (Figure 2) and a full sleep hypnogram for the second night (Figure 3) demonstrates the distribution of sleep stages, stage shifts, and distribution of awakenings on each night in the sleep laboratory.

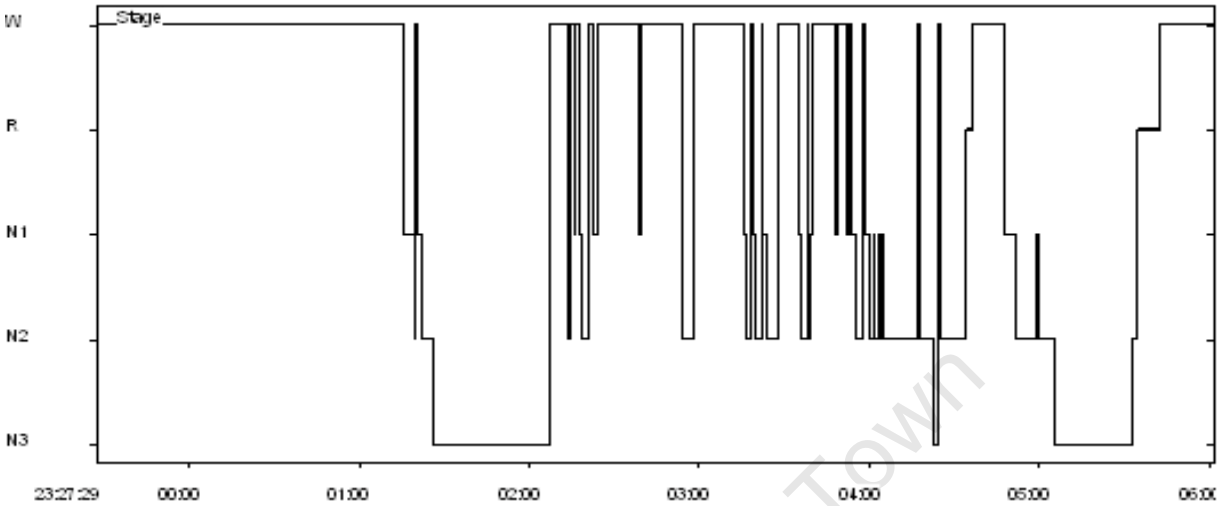


Figure 2. Sleep hypnogram for the first night (Case 1), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

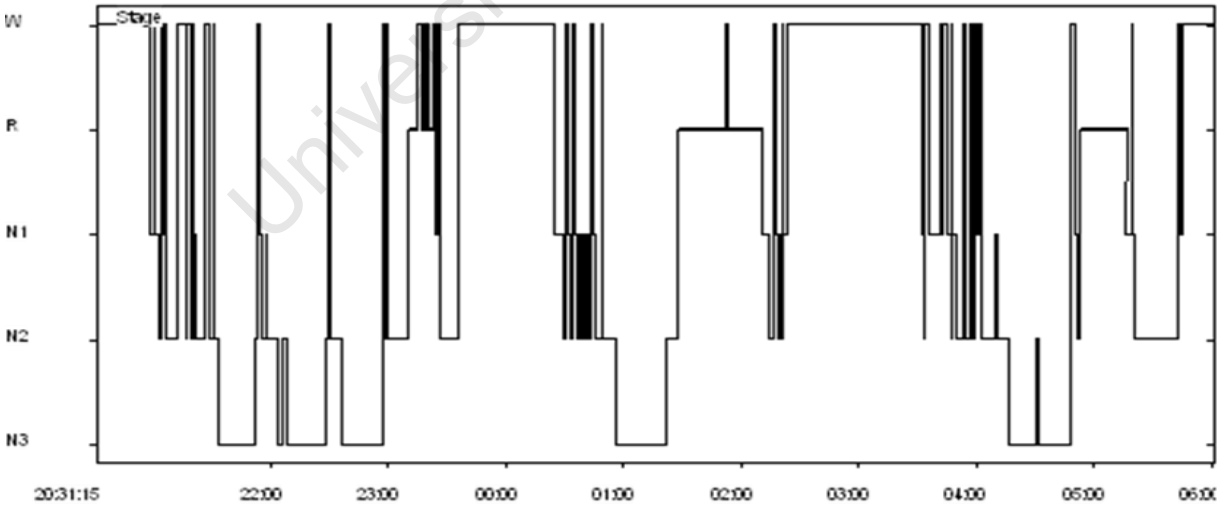


Figure 3. Sleep hypnogram for the second night (Case 1), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

Of significance, was the number of stage shifts on the first night (70) and the number of stage shifts on the second night (132). This indicated general difficulties in entering and maintaining sleep stages.

The efficiency of sleep was analysed according to sleep quantity and sleep quality.

Sleep quantity

The sleep parameters included to assess sleep quantity were total sleep time and the percentage of time spent in sleep (Table 4). Of significance, was the difficulty that Mrs A had sleeping on the first night with a total sleep time of only 175 minutes. This, however, could have been accounted for by the nocturnal REM-sleep interviews, as well as unfamiliarity to the sleep laboratory environment.

Table 4.

Sleep Quantity: Case 1

	TST	*% Sleep*
Night 1	175.00	44.42
Night 2	369.00	64.77

Note: TST = The total number of minutes spent in sleep. % Sleep = The percentage of time spent in sleep from lights off to lights on.

Sleep quality

Mrs A displayed disturbed sleep on both nights in the sleep laboratory (Table 5). A substantial increase in the number and frequency of sleep disturbances was indicated on the second night in the sleep laboratory. All sleep quality parameters indicated that Mrs A had substantial difficulties maintaining sleep on both nights, while sleep disturbances were more prevalent on the second night.

Table 5.

Sleep Quality: Case 1

	Awakenings	*WI*	*Arousals*	*AI*	*Micro-Arousals*	*MI*
Night 1	19.00	6.52	77.00	26.40	56.00	19.21
Night 2	39.00	6.34	244.00	39.70	176.00	28.62

Note: WI = Awkening Index: defined as the average number of awaknings per hour of sleep
AI = Arousal Index: defined as the average number of arousals per hour of sleep time
MI = Micro-arousal Index: defined as the average number of micro-arousals per hour of sleep time

Analysis of additional sleep parameters on the first night indicated that three arousals were associated with leg movements, six arousals were associated with snore events, and that 39 arousals were associated with respiratory events. The number of arousals associated with respiratory events on the first night indicated that Mrs A had an Apnea + Hypopnea Index (AHI) of 7.9, which, according to AASM standard criteria, was considered to be mild sleep disordered breathing (Benbir & Guilleminault, 2007).

Analysis of additional sleep parameters on the second night indicated that 1 arousal was associated with leg movements, and that 147 arousals were associated with respiratory events. The number of arousals associated with respiratory events on the second night indicated that Mrs A had an Apnea + Hypopnea Index (AHI) of 14.5, which, according to AASM standard criteria, was considered to be mild to moderate sleep disordered breathing (Benbir & Guilleminault, 2007).

Case 2: Mrs C

Date of birth: 10/01/1949
Date of CVA: 02/07/2011
Date of sleep study: 12/07/2011, 13/07/2011 and 29/09/2011

History of stroke

Mrs C, a right handed female, presented to Gatesville Medical Centre, Cape Town, on 7 July 2011 with a history of parasthesiae and numbness in her left upper limb, as well as an episode where she was dragging her left lower limb. Her family reported a six day history of confusion that had persisted and worsened over the following days. She also complained that vision had been affected.

This episode followed a motor vehicle accident three weeks prior, where she had injured her leg and fractured her patella. No head trauma was noted. After undergoing surgery on her leg, she had developed severe hypertension and diabetes mellitus.

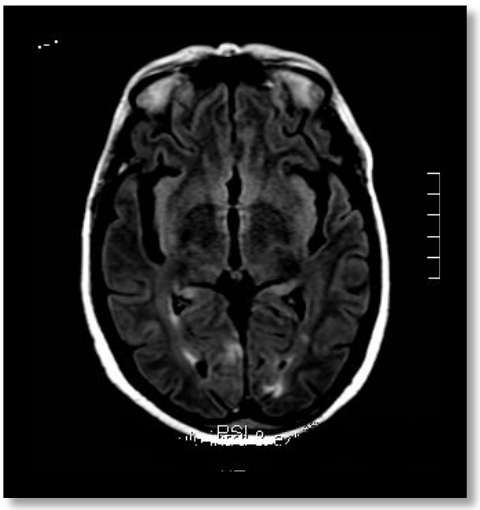
On neurological examination, she additionally complained of a headache that seemed to emanate from the region of the left orbit and radiate to the vertex of the left head. The family further reported substantial confusion and poor memory.

Neurological examination showed an abnormal mental state with disorientation to day and month, difficulties reading, and poor immediate recall. She displayed normal attention and no evidence of aphasia. Cranial nerve examination was normal. Power could not be tested in the right lower limb as the leg was bandaged and strapped, but power was normal on all other limbs. Sensation, coordination, gait, and fine motor movements were all normal.

MRI, taken on 7 July 2011, demonstrated acute infarcts in the right basal ganglia, the right thalamus, and both occipital poles (worse on the right) (Figure 4). MRA revealed atheromatous disease of the anterior and posterior intracranial circulation, with very tenuos vertebral arteries bilaterally, as well as stenosis in the basilar artery.

Figure 4. Magnetic Resonance Images (FLAIR sequence): Case 2

A



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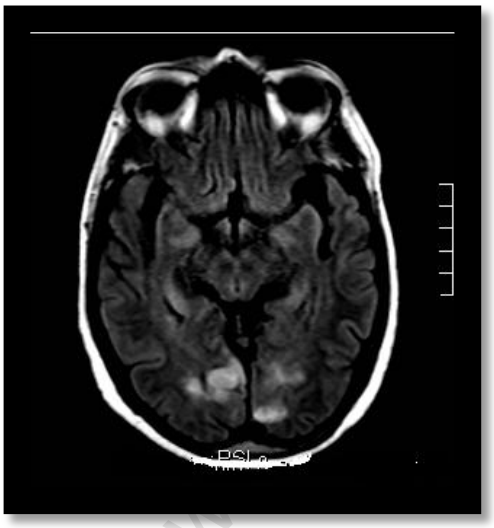

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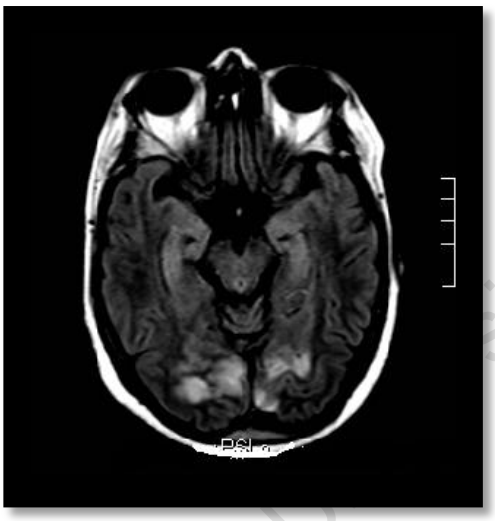

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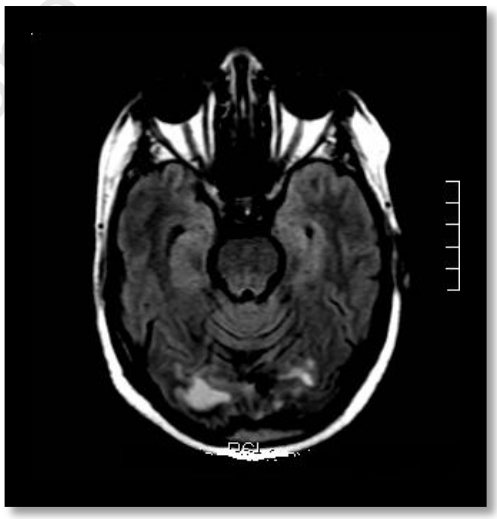

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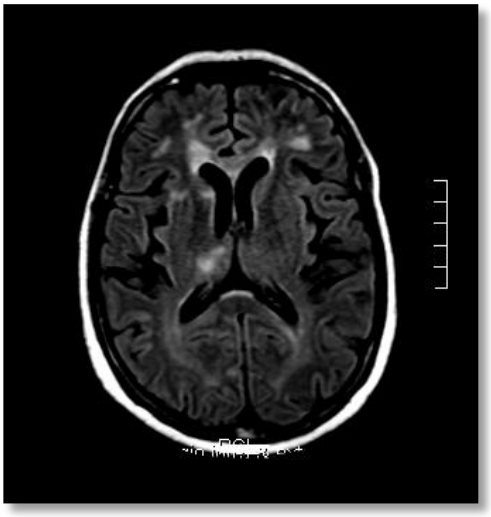


Clinical examination and investigation thus revealed that Mrs C had suffered bilateral thrombotic infarction in the PCA territory, with acute infarcts in the right basal ganglia, right thalamus, and both occipital poles.

Neuropsychological assessment

Neuropsychological examination revealed palinopsia, complex visual hallucinations, tongue and lip anaesthesia, and photophobia, all of a transient nature.

Neuropsychological testing (Table 6) indicated that visuo-spatial perception was mildly impaired with very mild apperceptive visual agnosia evident on Luria's Neuropsychological Investigation, and slight apperceptive errors on the Boston Naming Test. Naming was shown to be intact.

Constructional praxis was shown to be intact with a good copy of the Rey-Osterrieth Complex Figure and adequate performance on the WAIS III Blocks.

Performance on the Digit span test and Corsi's blocks showed normal visual and verbal short-term memory.

No visual and verbal long-term memory deficits were noted, and revisualization was intact. In particular, Mrs C was able to accurately recount the sleep lab experience, including details of the sleep laboratory nurse, the layout of the room, and the procedure of attaching the polysomnograph leads one month after the sleep study.

Therefore, neuropsychological assessment indicated that Mrs C had a very mild apperceptive visual agnosia. Overall performance on all tests as well as clinical impression concluded that Mrs C had intact functions necessary for dream recall. In particular, accurate recall of the sleep laboratory procedure and room indicated that Mrs C had the necessary cognitive capacity for dream recall.

Table 6.

Neuropsychological Testing: Case 2

Test	*Score*
Luria's Complex Visual Scenes	9/11
Boston Naming Test	46/60
Rey-Osterrieth Complex Figure	
Copy	32/36
Immediate Recall	15/36
WAIS-III Blocks	Raw score = 24, *z* score = 8
Digit Span	
Forwards	5
Backwards	6
Corsi's Blocks	
Forwards	6
Backwards	4
Benton's Visual Retention Test	6/7
Babcock Story	
Trial A	7/21
Trial B	12/21
Revisualization	
South African Flag	normal
Canary	normal
Patient's House	normal
Sleep Laboratory	normal

Dream recall

Mrs C reported that she had not been able to remember any dreams since her stroke. This remained consistent over two clinical interviews, 2 months and 3 months post stroke. She reported that before her stroke she had dreamt frequently indicating that complete cessation of dreaming had occurred as a direct result of her stroke.

This was confirmed in the sleep laboratory. Nocturnal REM sleep interviews could not be performed on the first night in the sleep laboratory as Mrs C did not enter REM sleep on that night. However, lack of dream recall in the morning and from unprompted awakenings during the 1st and 2nd night in the sleep laboratory supported that she was, in fact, not dreaming. In addition, a third sleep study night was specifically designed to confirm the lack of dreaming. Whilst normal REM was achieved on the third night, no positive dream reports were obtained.

PSQI

The results of the Pittsburgh Sleep Quality Index (Table 7) indicated that Mrs C reported that she subjectively experienced a good quality of sleep. Mild difficulties were noted with sleep latency and day time dysfunction, moderate difficulties were noted with sleep duration and sleep disturbances, and severe difficulties were noted with habitual sleep efficiency. The global PSQI score (9) indicated moderate difficulties.

Table 7.

Pittsburgh Sleep Quality Index: Case 2

Component	*Score (range 0-3)*	*Score (range 0-21)*
Subjective sleep quality	0	
Sleep latency	1	
Sleep duration	2	
Habitual sleep efficiency	3	
Sleep disturbances	2	
Use of sleeping medication	0	
Day time dysfunction	1	
Global PSQI score		9

Note: Each individual component score has a possible range of 0-3. For each component score, a score of '0' indicates no difficulty, while a score of '3' indicates severe difficulty. The global PSQI score is a summation of the seven component scores and has a posssible range of 0-21. A score of '0' indicates no difficulty, while a score of '21' indicates severe difficulties in all areas.

Sleep study findings

Polysomnographic recordings confirmed that Mrs C did not demonstrate REM on the 1st night in the sleep laboratory, and that she demonstrated 3 REM periods on the second night. On the second night, REM accounted for 18.1% of total sleep time. REM sleep normally accounts for 20-25% of total sleep time in healthy adults (Chokroverty, 2009), indicating that Mrs C experienced slightly less than the expected amount of REM. Because of the absence of REM on the 1st night, an additional night in the sleep laboratory was recorded, on which Mrs C demonstrated 2 REM periods. REM accounted for 15.2% of total sleep time on the third night, lower than the expected amount for healthy adults. Table 8 indicates each REM period on night 2 and night 3.

Table 8.

REM Cycles: Case 2

		Total	*Cycle*	*REM start*
Night 2	*REM 1*	32.0	122.0	22:32:27
	REM 2	30.0	209.0	02:03:27
	REM 3	28.5	107.5	03:52:27
Night 3	*REM 1*	23.0	242.5	02:31:43
	REM 2	19.5	103.0	04:18:13

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

A full sleep hypnogram for the first night (Figure 5), a full sleep hypnogram for the second night (Figure 6), and a full sleep hypnogram for the third night (Figure 7) demonstrate the distribution of sleep stages, stage shifts, and distribution of awakenings on each night in the sleep laboratory.

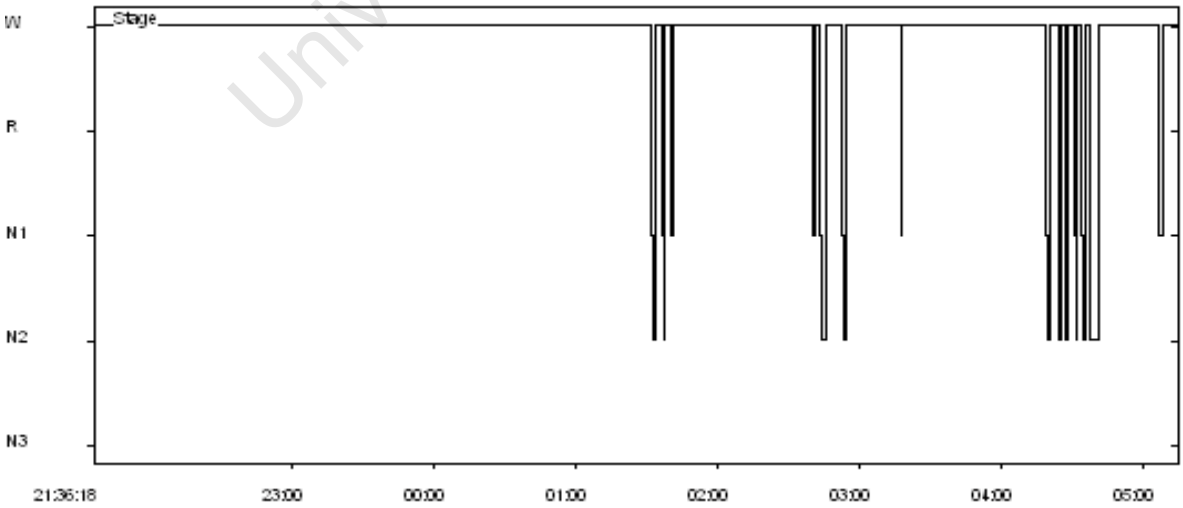


Figure 5. Sleep hypnogram for the first night (Case 2), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

Figure 6. Sleep hypnogram for the second night (Case 2), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

Figure 7. Sleep hypnogram for the third night (Case 2), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

The efficiency of sleep was analysed according to sleep quantity and sleep quality.

Sleep quantity

The sleep parameters included to assess sleep quantity were total sleep time and the percentage of time spent in sleep (Table 9). Of significance, was the almost total lack of sleep on the first night (23 minutes spent in sleep). This was partially accounted for by unfamiliarity with the sleep laboratory environment. Furthermore, increased sleep efficiency on the second night (97.85 % of time from lights on to lights off spent in sleep), was seen as a direct result of sleep deprivation from the previous night.

Table 9.

Sleep Quantity: Case 2

	TST	*% Sleep*
Night 1	23.00	5.02
Night 2	501.00	97.85
Night 3	280.00	70.50

Note: TST = The total number of minutes spent in sleep. % Sleep = The percentage of time spent in sleep from lights off to lights on.

Sleep quality

Mrs C had substantially disturbed sleep on the first and second night. Table 10 outlines specific sleep disturbances on all three nights. Of note, was the substantial frequency of awakenings on the first night (awakening index = 36.55), and the substantial frequency of arousals (arousal index = 39.10). While the number and frequency of awakenings were not substantial on the second night, the number and frequency of arousals (arousal index = 88.60) and micro-arousals (micro-arousal index = 85.27) indicated severe difficulties with maintaining sleep.

Table 10.

Sleep Quality: Case 2

	Awakenings	*WI*	*Arousals*	*AI*	*Micro-Arousals*	*MI*
Night 1	14.00	36.55	15.00	39.10	9.00	23.50
Night 2	7.00	0.84	740.00	88.60	712.00	85.27
Night 3	23.00	4.93	117.00	25.10	94.00	20.10

Note: WI = Awkening Index: defined as the average number of awaknings per hour of sleep
AI = Arousal Index: defined as the average number of arousals per hour of sleep time
MI = Micro-arousal Index: defined as the average number of micro-arousals per hour of sleep time

Analysis of additional sleep parameters on the first night indicated that ten arousals were associated with snore events and that no arousals were associated with respiratory events. This indicated that Mrs C had an Apnea + Hypopnea Index (AHI) of 0.00 on the first night, which indicated a complete lack of sleep disordered breathing.

Analysis of additional sleep parameters on the second night indicated that 624 of the arousals were associated with respiratory events. The number of arousals associated with respiratory events indicated that Mrs C had an Apnea + Hypopnea Index (AHI) of 68.6, which, according to AASM standard criteria, was considered to be severe sleep disordered breathing (Benbir & Guilleminault, 2007).

Analysis of additional sleep parameters on the third indicated that 24 arousals were associated with leg movements, six arousals were associated with snore events, and 34 arousals were associated with respiratory events. The amount of respiratory events indicated that Mrs C had an Apnea + Hypopnea Index (AHI) of only 2.6 on the third night, well within the normal range.

Case 3: Mr R

Date of birth: 09/05/1960
Date of CVA: 23/12/2010
Date of sleep study: 27/06/2011 and 28/06/2011

History of stroke

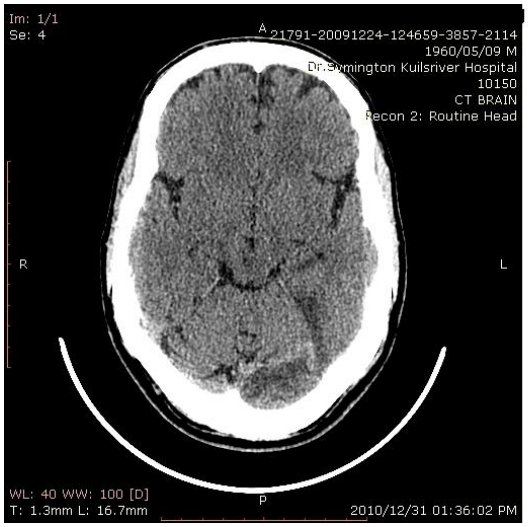
Mr R, a right handed male, presented to Kuils River Hospital, Cape Town, on 23 December 2010 with severe headache in the left frontal area and complaints of forgetfulness. He had an extensive medical history of rheumatic fever, mitral stenosis and hypertension. In addition, he was on warfarin for treatment of atrial fibrillation.

CT brain taken eight days after initial presentation revealed a left posterior cerebral artery infarction, most likely of subacute duration (Figure 8). The infarction involved the occipital lobe on the left-hand side towards its inferior portion, with no evidence of the temporal lobes being affected. Mild localized brain swelling was noted, while there was no significant mass effect and no evidence of haemorrhagic transformation. CT revealed a hypodense area in the posterior medial part of the right temporal lobe, that represented either an old small infarction or a deep sulcus. No significant calcifications of the terminal portions of the vertebral arteries or of the basilar artery were seen.

Subsequent neurological examination at Gatesville Medical Centre, Cape Town, revealed persistent headache, blurred vision and a poor cognitive state characterized by forgetfulness and personality changes.

Figure 8. Computed Tomography: Case 3

A



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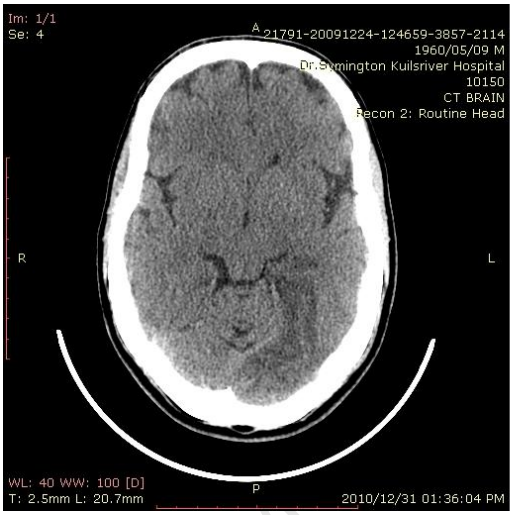

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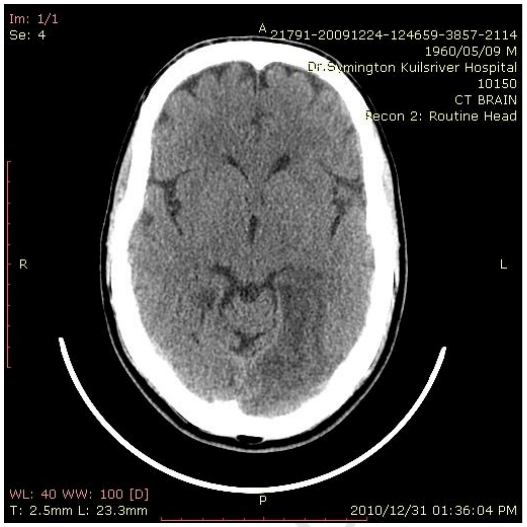

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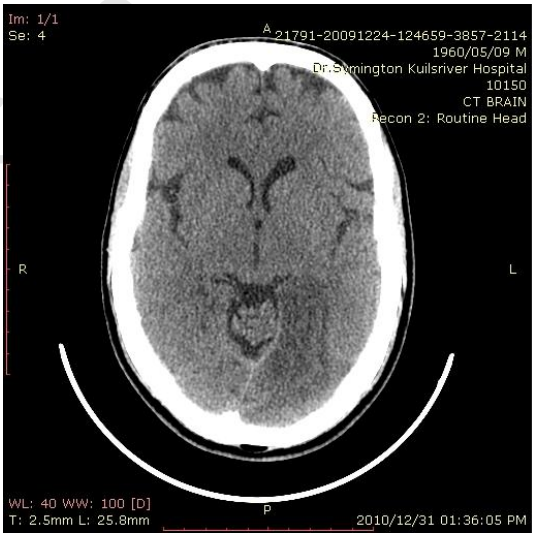

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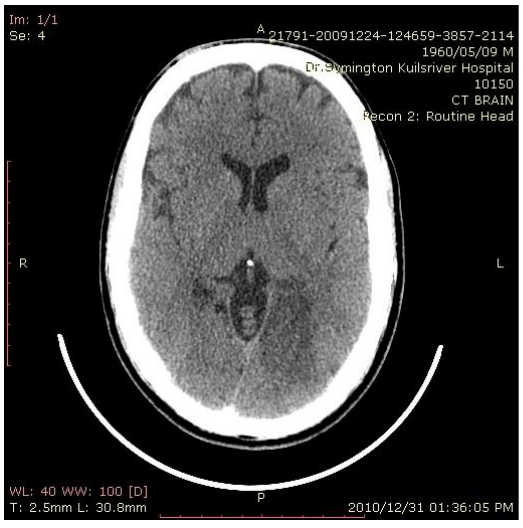

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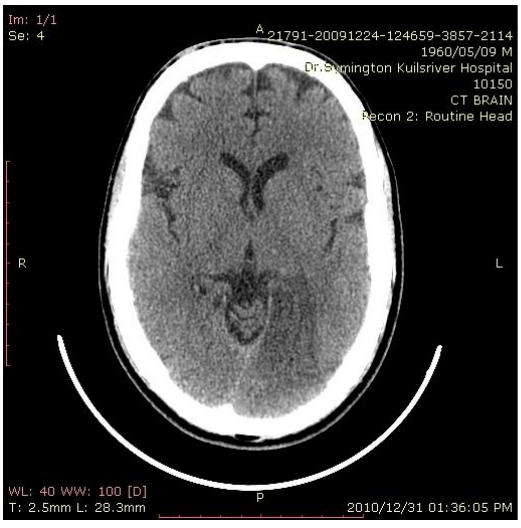

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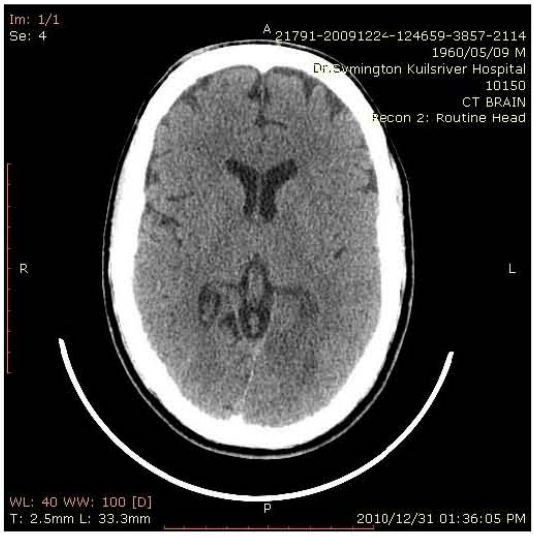
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Dr.Symington Kuilsriver Hospital
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CT BRAIN
Recon 2: Routine Head
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L
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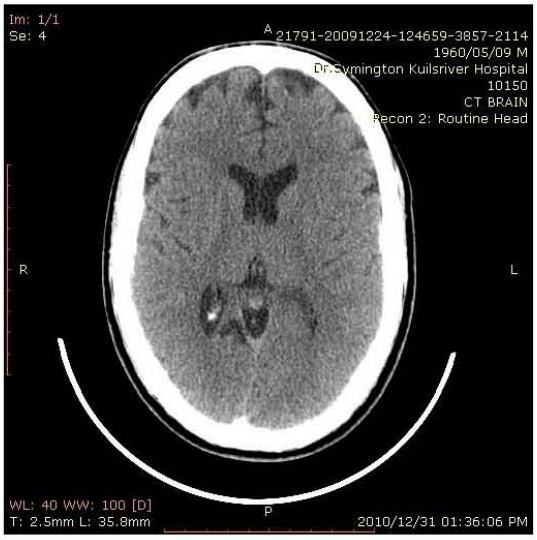
H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CT BRAIN
Recon 2: Routine Head
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Clinical examination and scanning techniques thus revealed that Mr R had suffered thrombotic infarction in the PCA territory, affecting the left occipital lobe.

Neuropsychological assessment

Neuropsychological examination (Table 11) indicated that visuo-spatial perception was intact. Performance on the Boston Naming Test was adequate, and did not display any naming deficits.

Performance on the Rey-Osterrieth Complex Figure and WAIS-III Blocks indicated that constructional praxis was normal.

Verbal and visual short-term memory were within normal range on the Digit span test and Corsi's blocks.

Testing of long-term memory showed that Mr R did not display any major verbal or visual memory deficits, while immediate recall of the Rey-Osterrieth Complex Figure was poor. Revisualization of the South African flag, a canary, the patient's own house, and the sleep laboratory was good. In particular, Mr R was able to accurately recall the room in the sleep laboratory two months after the sleep study, including details of the layout of the room and of the polysomnograph leads.

Therefore, neuropsychological assessment did not reveal any major cognitive deficits. Based on overall performance on testing as well as clinical impression, it was concluded that Mr R had intact functions necessary for dream recall.

Table 11.

Neuropsychological Testing: Case 3

Test	*Score*
Luria's Complex Visual Scenes	9/11
Boston Naming Test	33/60
Rey-Osterrieth Complex Figure	
Copy	27/36
Immediate Recall	6/36
WAIS-III Blocks	raw score = 16, *z* score = 5
Digit Span	
Forwards	5
Backwards	3
Corsi's Blocks	
Forwards	5
Backwards	4
Benton's Visual Retention Test	3/7
Babcock Story	
Trial A	4/21
Trial B	7/21
Revisualization	
South African Flag	normal
Canary	normal
Patient's House	normal
Sleep Laboratory	normal

Dream recall

Mr R reported that he dreamt regularly and that this had not been affected by his stroke. Nocturnal REM-sleep interviews were performed during the first night in the sleep laboratory during the second and third REM periods. A positive dream report was elicited from the second awakening. In addition, Mr R reported in the morning that could remember dreaming a few time during the night.

PSQI

Table 12.

Pittsburgh Sleep Quality Index: Case 3

Component	*Score (range 0-3)*	*Score (range 0-21)*
Subjective sleep quality	1	
Sleep latency	3	
Sleep duration	1	
Habitual sleep efficiency	1	
Sleep disturbances	1	
Use of sleeping medication	0	
Day time dysfunction	1	
Global PSQI score		8

Note: Each individual component score has a possible range of 0-3. For each component score, a score of '0' indicates no difficulty, while a score of '3' indicates severe difficulty. The global PSQI score is a summation of the seven component scores and has a posssible range of 0-21. A score of '0' indicates no difficulty, while a score of '21' indicates severe difficulties in all areas.

The results of the Pittsburgh Sleep Quality Index (Table 12) indicated that Mr R subjectively reported a moderately good quality of sleep. Mild difficulties were noted with subjective sleep quality, sleep duration, habitual sleep efficiency, sleep disturbances and day time dysfunction, while severe difficulty was noted with sleep latency.

Sleep study findings

Polysomnographic recordings confirmed that Mr R documented REM sleep on both nights in the sleep laboratory (Table 13). On the first night, five REM periods were documented, creating five sleep cycles. Despite nocturnal REM-sleep interviews and unfamiliarity with the sleep laboratory, REM accounted for 30.5% of total sleep time on the first night, greater than the expected amount of 20-25% in healthy adults (Chokroverty, 2009). On the second night, Mr R documented three REM periods, and the amount of REM accounted for 24.8% of total sleep time (within normal range).

Table 13.

REM Cycles: Case 3

		Total	*Cycle*	*REM start*
Night 1	*REM 1*	46.0	140.0	21:27:33
	REM 2	2.0	98.0	23:49:33
	REM 3	9.0	112.0	01:34:33
	REM 4	15.5	101.5	03:09:33
	REM 5	28.5	114.5	04:51:03
Night 2	*REM 1*	26.5	104.0	21:29:35
	REM 2	32.0	173.5	00:17:35
	REM 3	68.0	195.5	02:57:05

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

Full sleep hypnograms for the first night (Figure 9) and the second night (Figure 10) demonstrate the distribution of sleep stages, stage shifts, and distribution of awakenings.

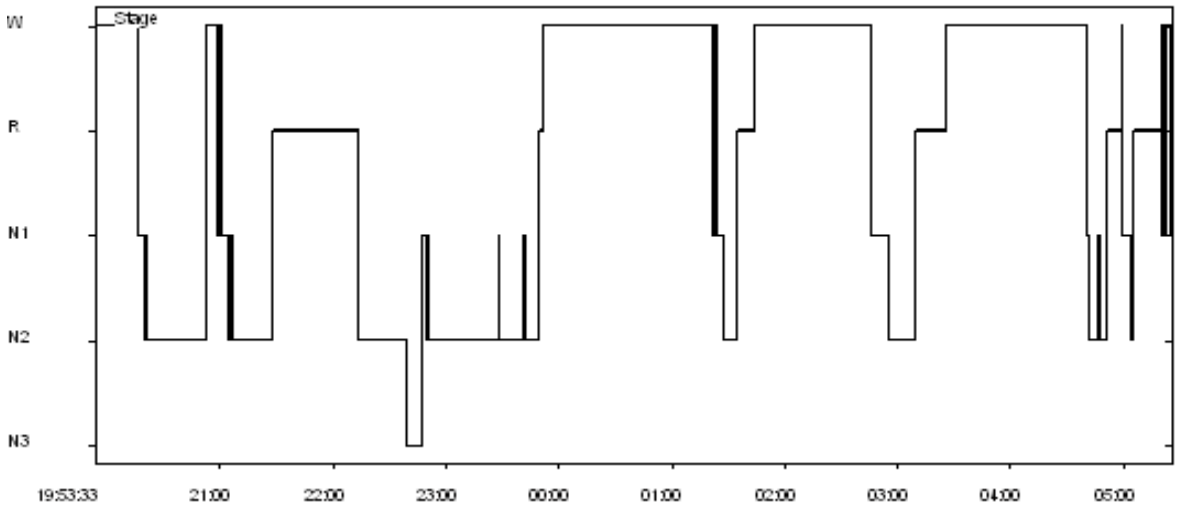


Figure 9. Sleep hypnogram for the first night (Case 3), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

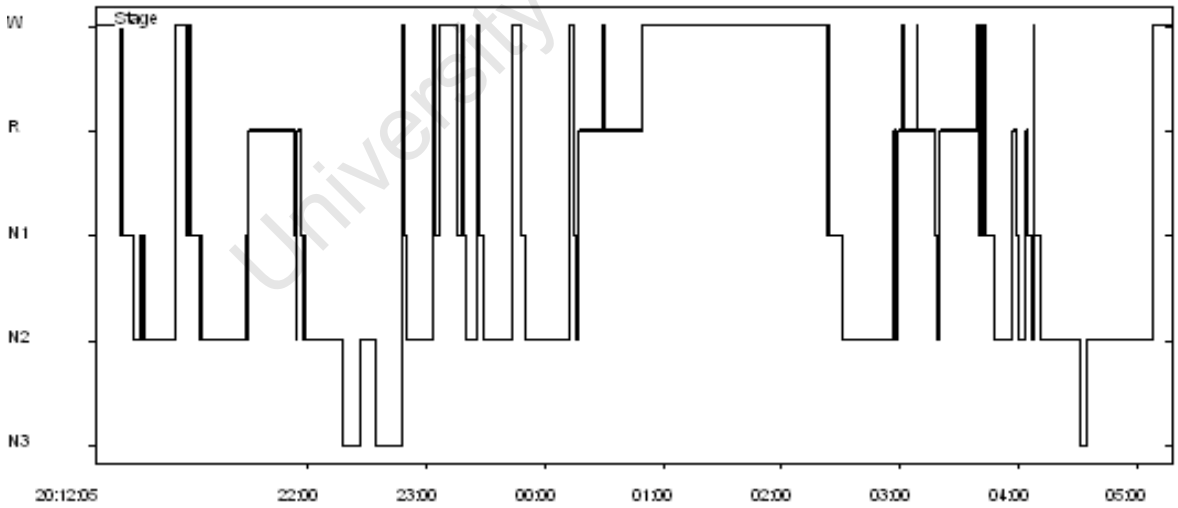


Figure 10. Sleep hypnogram for the second night (Case 3), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

The efficiency of sleep was analysed according to sleep quantity and sleep quality.

Sleep quantity

The sleep parameters included to assess sleep quantity were total sleep time and the percentage of time spent in sleep (Table 14). The decrease in percentage of time spent in sleep on the first night (54.37%) could be a reflection of disturbance from nocturnal REM sleep interviews as well as unfamiliarity with the sleep laboratory environment. However, no remarkable results were noted.

Table 14.

Sleep Quantity: Case 3

	TST	*% Sleep*
Night 1	311.00	54.37
Night 2	400.50	73.35

Note: TST = The total number of minutes spent in sleep. % Sleep = The percentage of time spent in sleep from lights off to lights on.

Sleep quality

Analysis of sleep parameters that assessed sleep quality (Table 15) did not indicate any substantial sleep disturbances on the first night and the second night. Instead, the results indicated mild disturbances across all parameters on both nights.

Table 15.

Sleep Quality: Case 3

	Awakenings	*WI*	*Arousals*	*AI*	*Micro-arousals*	*MI*
Night 1	11.00	2.12	164.00	31.60	103.00	19.87
Night 2	19.00	2.85	148.00	22.20	93.00	13.93

Note: WI = Awkening Index: defined as the average number of awaknings per hour of sleep
AI = Arousal Index: defined as the average number of arousals per hour of sleep time
MI = Micro-arousal Index: defined as the average number of micro-arousals per hour of sleep time

Analysis of additional sleep parameters on the first night indicated that 41 arousals were associated with snore events and that 106 arousals were associated with respiratory events. The number of arousals associated with respiratory events indicated that Mr R had an Apnea

+ Hypopnea Index (AHI) of 9.3, which, according to AASM standard criteria, was considered to be mild sleep disordered breathing (Benbir & Guilleminault, 2007).

Analysis of additional sleep parameters on the second night indicated that one arousal was associated with leg movements, 16 with snore events, and 69 with respiratory events. The number of arousals associated with respiratory events indicated that Mr R had an Apnea + Hypopnea Index (AHI) of 2.8, which, according to AASM standard criteria, was not considered to be sleep disordered breathing (Benbir & Guilleminault, 2007).

Case 4: Mr D

Date of birth: 30/08/1943
Date of CVA: 11/02/2010
Date of sleep study: 18/04/2011 and 19/04/2011

History of stroke

Mr D, a right handed male, presented to Gatesville Medical Centre, Cape Town, on 11 February 2010 with an acute history of imbalance, slurred speech, severe vertigo, and a possible loss of consciousness. Mr D had an extensive medical history of hypertension, diabetes, and ischaemic heart disease for which he had had coronary artery bypass surgery.

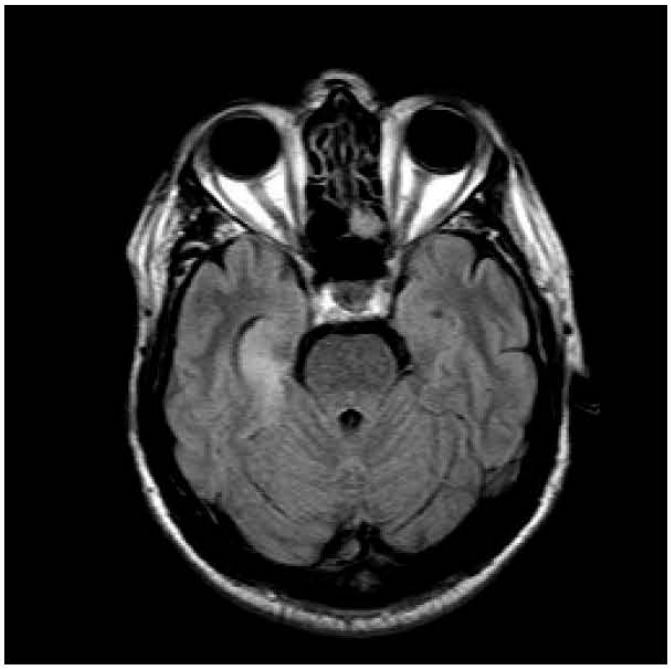
MRI, taken 5 days after initial presentation, revealed an abrupt occlusion of the right PCA at the junctions of the P1 and P2 segments (Figure 11). T2, FLAIR, and DWI showed hyperintense signal change in the right uncus, parahippocampal gyrus, and hippocampus. No significant brain swelling was noted, and no evidence of haemorrhage was found.

Subsequent neurological examination also reported topographical disorientation and forgetfulness while driving.

Clinical examination and scanning techniques thus revealed that Mr D had suffered a thrombotic infarction in the PCA territory situated in the right posterior cerebral artery at the junction of the P1 and P2 segments that resulted in abnormal parrahippocampal and hippocampal gyruses.

Figure 11. Magnetic Resonance Images (FLAIR sequence): Case 4

A



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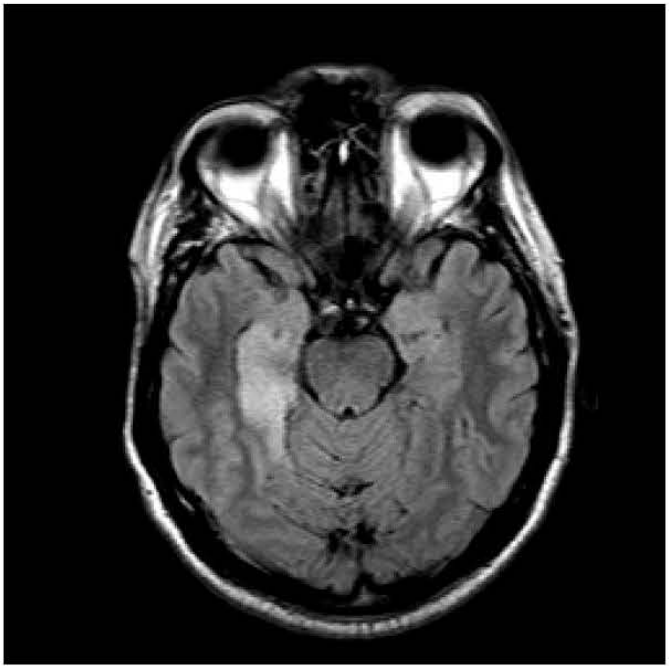

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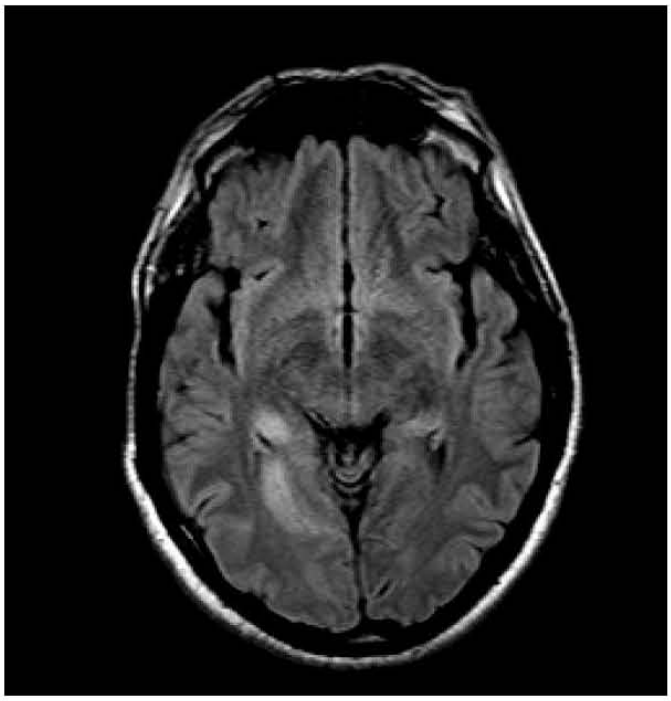

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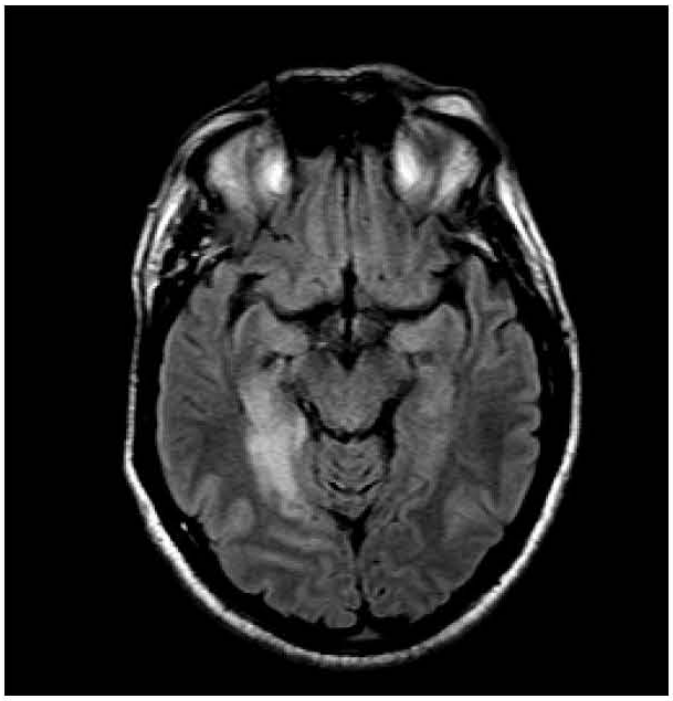


Neuropsychological assessment

Neuropsychological examination (Table 16) indicated that visuo-spatial perception was intact, except for some face recognition difficulties. Place recognition difficulties were also noted, but these were explained by the fact that the patient had topographical amnesia.

Constructional praxis was preserved, demonstrated by a very good copy of the Rey-Osterrieth Complex Figure.

Verbal and visual short-term memory were intact.

Performance on the Babcock Story indicated that verbal long-term memory was preserved. Poor immediate recall on the Rey-Osterrieth Complex Figure indicated a mild visual long-term memory deficit. However, Mr D was able to revisualize the South African flag and the plan of his house in detail.

Therefore, neuropsychological assessment revealed no major cognitive deficits, except for a mild visual long-term memory deficit. Based on the overall performance on testing as well as clinical impression, it was concluded that Mr D had intact functions necessary for dream recall.

Table 16.

Neuropsychological Testing: Case 4

Test	*Score*
Rey-Osterrieth Complex Figure	
Copy	32/36
Immediate Recall	9/36
Digit Span	
Forwards	6
Babcock Story	
Trial A	8/21
Trial B	12/21
Revisualization	
South African Flag	normal
Patient's House	normal

Dream recall

Mr D reported that he had slept well since his stroke and did not feel that his sleep had been affected in any way. He reported that he dreamt regularly, and that this had not been affected by his stroke. He was able to recall a particularly vivid dream that had occurred 1 month prior to clinical interviewing and was able to recall descriptive details of characters in the dream. He was also able to recall a dream he had experienced then night prior to clinical interviewing. The following transcript indicates Mr D's recall of the dream he had experienced the night before clinical interviewing:

> "Last night, I dreamt that intruders tried to enter our home. I was helpless to do anything about it. Finally, they couldn't enter. I saw the intruder climbing over the wall, and entering the yard".

During the first night in the sleep laboratory, nocturnal REM-sleep interviews were conducted by waking Mr D during the second and third REM periods. Both awakenings elicited positive dream reports. In addition, Mr D was able to recall one of those dreams in the morning, as well as an additional dream that he had during the night.

PSQI

The results of the Pittsburgh Sleep Quality Index (Table 17) indicated that Mr D subjectively reported a moderately good quality of sleep. Mild difficulties were noted with subjective sleep quality, sleep latency, sleep duration, sleep disturbances, use of sleeping medication, and day time dysfunction. Severe difficulties were noted with habitual sleep efficiency.

Table 17.

Pittsburgh Sleep Quality Index: Case 4

Component	*Score (range 0-3)*	*Score (range 0-21)*
Subjective sleep quality	1	
Sleep latency	1	
Sleep duration	1	
Habitual sleep efficiency	3	
Sleep disturbances	1	
Use of sleeping medication	1	
Day time dysfunction	1	
Global PSQI score		9

Note: Each individual component score has a possible range of 0-3. For each component score, a score of '0' indicates no difficulty, while a score of '3' indicates severe difficulty. The global PSQI score is a summation of the seven component scores and has a posssible range of 0-21. A score of '0' indicates no difficulty, while a score of '21' indicates severe difficulties in all areas.

Sleep study findings

As confirmed by PSG recordings, Mr D documented REM on both nights in the sleep laboratory (Table 18). On the first night, two REM periods were documented creating two REM cycles. The amount of REM on the first night accounted for 13% of total sleep time. As REM sleep normally accounts for 20-25% of total sleep time in healthy adults (Chokroverty, 2009), a lack of REM was evident on the first night. However, this could be accounted for by the nocturnal REM sleep interviews that were conducted as well as unfamiliarity with the sleep laboratory environment. On the second night, four REM periods were documented. The amount of REM on the second night accounted for 17.9% of total sleep time, slightly less than the expected amount for healthy adults.

Table 18.

REM Cycles: Case 4

		Total	*Cycle*	*REM start*
Night 1	REM 1	31.0	198.5	23:35:42
	REM 2	10.0	168.5	02:45:12
Night 2	REM 1	6.0	105.0	21:58:43
	REM 2	7.5	82.0	23:19:13
	REM 3	13.0	77.0	00:30:43
	REM 4	35.0	239.5	04:08:13

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

A full sleep hypnogram for the first night (Figure 12) and a full sleep hypnogram for the second night (Figure 13) demonstrate the distribution of sleep stages, stage shifts, and distribution of awakenings.

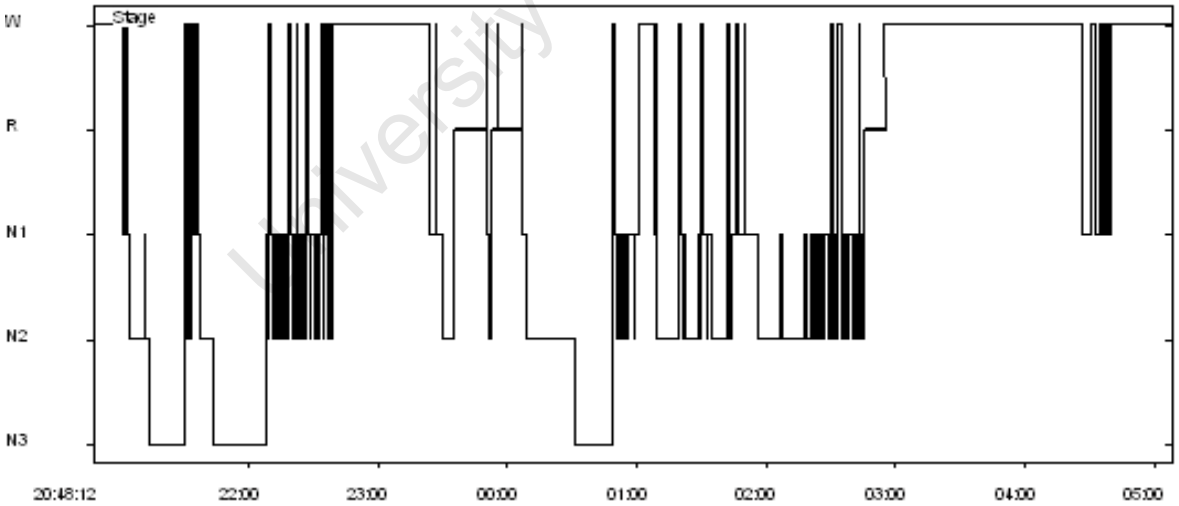


Figure 12. Sleep hypnogram for the first night (Case 4), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

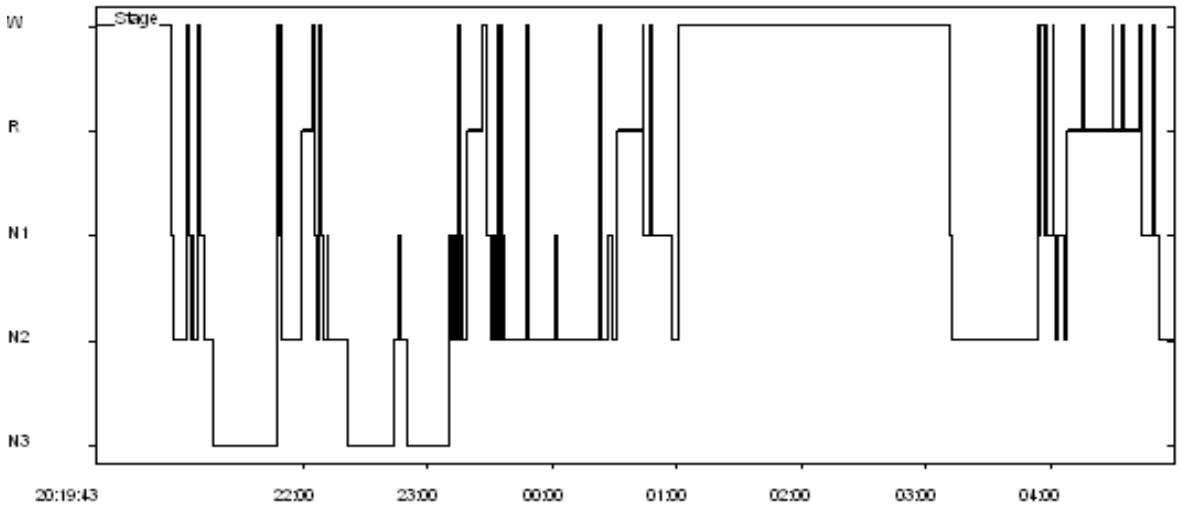


Figure 13. Sleep hypnogram for the second night (Case 4), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

The efficiency of sleep was analysed according to sleep quantity and sleep quality.

Sleep quantity

The sleep parameters included to assess sleep quantity were total sleep time and the percentage of time spent in sleep (Table 19). The relatively low percentage of time spent in sleep on the first night (59.1%) could have been a reflection of disturbance from nocturnal REM sleep interviews as well as unfamiliarity with the sleep laboratory environment. However, the percentage of time spent in sleep on the first night was very similar to the second night when no nocturnal interviews were conducted. Overall, for sleep quantity, no remarkable results were noted.

Table 19.

Sleep Quantity: Case 4

	TST	*% Sleep*
Night 1	295.50	59.10
Night 2	335.50	64.52

Note: TST = The total number of minutes spent in sleep. % Sleep = The percentage of time spent in sleep from lights off to lights on.

Sleep quality

Analysis of sleep parameters that assessed sleep quality (Table 20) did not indicate any substantial sleep disturbances on the first night and the second night. Instead, the results indicated mild disturbances across all parameters on both nights.

Table 20.

Sleep Quality: Case 4

	Awakenings	*WI*	*Arousals*	*AI*	*Micro-arousals*	*MI*
Night 1	34.00	6.90	141.00	28.60	123.00	24.97
Night 2	25.00	4.47	149.00	26.60	119.00	21.28

Note: WI = Awkening Index: defined as the average number of awaknings per hour of sleep
AI = Arousal Index: defined as the average number of arousals per hour of sleep time
MI = Micro-arousal Index: defined as the average number of micro-arousals per hour of sleep time

Analysis of additional sleep parameters on the first night indicated that two arousals were associated with leg movements, 18 arousals were associated with snore events, and that 83 arousals were associated with respiratory events. The number of arousals associated with respiratory events indicated that Mr D had an Apnea + Hypopnea Index (AHI) of 18.3 on the first night, which, according to AASM standard criteria, was considered to be moderate sleep disordered breathing (Benbir & Guilleminault, 2007).

Analysis of additional sleep parameters on the second night indicated that one arousal was associated with leg movements, 32 with snore events, and 78 with respiratory events. The number of arousals associated with respiratory events indicated that Mr D had an Apnea + Hypopnea Index (AHI) of 13.4 on the second night, which, according to AASM standard criteria, was considered to be mild to moderate sleep disordered breathing (Benbir & Guilleminault, 2007).

Case 5: Mrs J

Date of birth: 12/09/1965
Date of CVA: 21/03/2007
Date of sleep study: 20/07/2011 and 21/07/2011

History of stroke

Mrs J, a right handed female, presented to Gatesville Medical Centre, Cape Town, on 21 March 2007 with sudden severe headache, dizziness, vertigo, and light-headedness. She reported blurry vision in both eyes that had made reading difficult. Previous significant medical history included a possible panic attack in December 2006 where she had collapsed and lost consciousness for a few seconds as well as surgery to remove a left breast lump three years prior.

Neurological examination revealed a normal mental state. Cranial nerve examination showed a right homonymous hemianopia. Examination of the motor system showed normal power, tone, and reflexes. Sensation, coordination, gait, and fine motor movements were all normal.

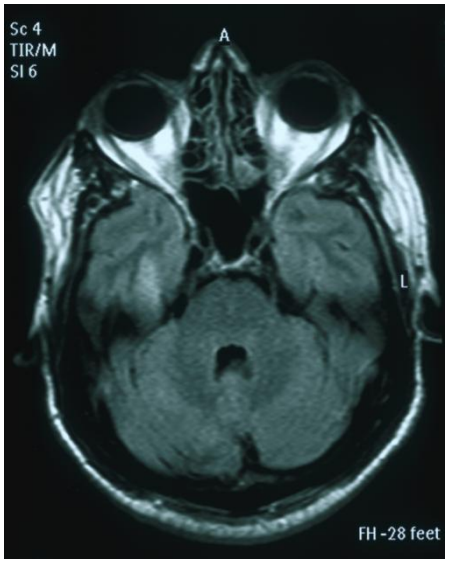
MRI revealed an infarct in the left occipital lobe (Figure 14). MRA showed no evidence of intracranial tumor and no evidence of vertebral artery dissection. An aneurysm arising from the right carotid siphon lateral to the origin of the right ophthalmic artery was noted.

Subsequent neurological examination also documented a decline in memory function, and persistent difficulties with vision and balance.

Clinical examination and scanning techniques thus revealed that Mrs J had suffered thrombotic infarction of the PCA territory that had resulted in damage to the left occipital lobe.

Figure 14. Magnetic Resonance Imaging (FLAIR sequence): Case 5

A



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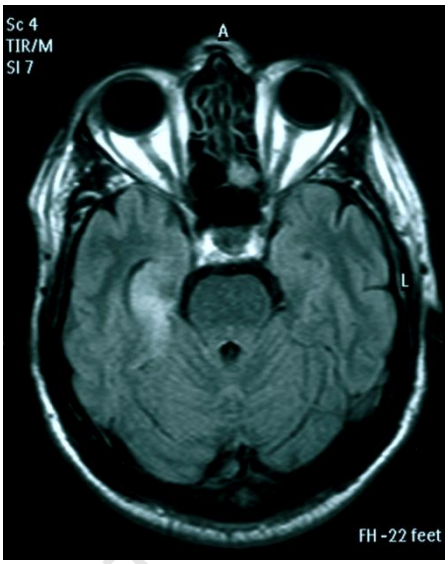

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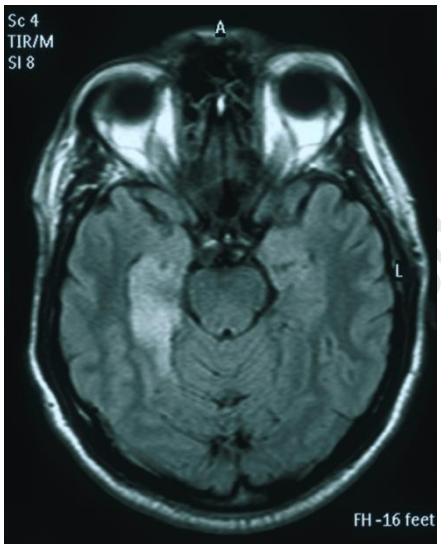

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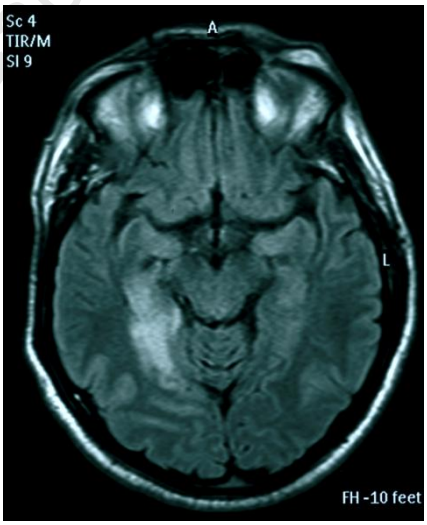

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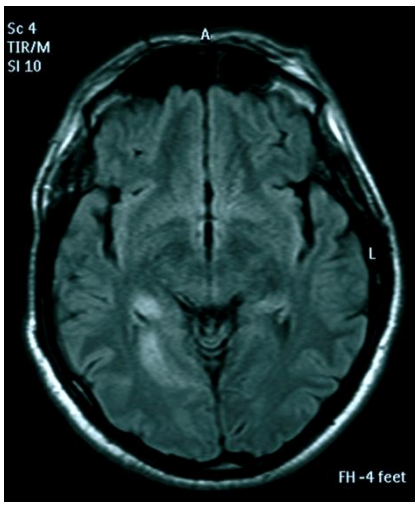


Neuropsychological assessment

On neuropsychological examination, Mrs J displayed a lower right quadrantanopia.

Neuropsychological assessment (Table 21) demonstrated that visuo-spatial perception was intact on Luria's Neuropsychological Investigation. However, performance was poor on the Boston Naming Test, which, combined with the quadrantanopia, supported the difficulties that the patient had had with reading. This did not, however, reflect an aphasic problem as the performance lacked language errors.

Constructional praxis was shown to be intact.

Performance on the Digit span test and Corsi's Blocks indicated mild impairment in visual and verbal short-term memory.

Intact verbal long-term memory was demonstrated by performance on the Babcock Story. However, visual long-term memory was demonstrated to be affected by poor immediate recall on the Rey-Osterrieth Complex Figure and a poor performance on Benton's Visual Retention test. Despite this, Mrs J was able to revisualize the South African flag, a canary, her own house, and the sleep laboratory in great detail. The following transcript indicated Mrs J's memory of the room in sleep laboratory recorded one month after the sleep study:

> "It was a tiny room. There was a window on the right, and there was glass. There was a cupboard on the left. The bed was in the centre. There was a little table".

Therefore, neuropsychological assessment revealed that Mrs J displayed mild difficulties with visuo-spatial perception, mild difficulties with short-term memory, and mild difficulties with visual long-term memory. No outstanding deficits were noted. Based on the performance on testing, as well as clinical impression, it was concluded that Mrs J had intact functions necessary for dream recall.

Table 21.

Neuropsychological Testing: Case 5

Test	*Score*
Luria's Complex Visual Scenes	10/11
Boston Naming Test	31/60
WAIS-III Blocks	raw score = 20, *z* score = 6
Digit Span	
Forwards	7
Backwards	4
Corsi's Blocks	
Forwards	6
Benton's Visual Retention Test	2/7
Babcock Story	
Trial A	7/21
Trial B	10/21
Revisualization	
South African Flag	normal
Canary	normal
Patient's House	normal
Sleep Laboratory	normal

Dream recall

Mrs J reported that she was a frequent dreamer, and that this had not been affected by her stroke. Three nocturnal REM sleep interviews were performed during the first night in the sleep laboratory; one from a spontaneous awakening during the second REM period, as well as from waking the patient during the second and third REM periods. From these awakenings, two positive dream reports were obtained.

PSQI

The results of the Pittsburgh Sleep Quality Index (Table 22) indicated that Mrs J subjectively reported a good quality of sleep. Mild difficulties were noted with subjective sleep quality, while mild to moderate difficulties were noted with sleep duration, sleep disturbances and daytime dysfunction.

Table 22.

Pittsburgh Sleep Quality Index: Case 5

Component	*Score (range 0-3)*	*Score (range 0-21)*
Subjective sleep quality	1	
Sleep latency	0	
Sleep duration	2	
Habitual sleep efficiency	0	
Sleep disturbances	2	
Use of sleeping medication	0	
Day time dysfunction	2	
Global PSQI score		7

Note: Each individual component score has a possible range of 0-3. For each component score, a score of '0' indicates no difficulty, while a score of '3' indicates severe difficulty. The global PSQI score is a summation of the seven component scores and has a posssible range of 0-21. A score of '0' indicates no difficulty, while a score of '21' indicates severe difficulties in all areas.

Sleep study findings

As confirmed by PSG recordings, Mrs J documented REM on both nights in the sleep laboratory (Table 23). On the first night, three REM periods were documented creating three REM cycles. The amount of REM on the first night accounted for 14% of total sleep time. As REM sleep normally accounts for 20-25% of total sleep time in healthy adults (Chokroverty, 2009), a lack of REM was evident on the first night. However, this was accounted for by the nocturnal REM sleep interviews that were conducted as well as unfamiliarity with the sleep laboratory environment. On the second night, four REM periods were documented. The

amount of REM on the second night accounted for 18.73% of total sleep time, slightly less than the expected amount for healthy adults.

Table 23.

REM Cycles: Case 5

		Total	*Cycle*	*REM start*
Night 1	*REM 1*	13.0	214.5	01:09:47
	REM 2	8.5	106.0	03:00:17
	REM 3	50.0	96.5	03:55:17
Night 2	*REM 1*	11.0	145.0	23:07:17
	REM 2	29.0	58.5	00:47:47
	REM 3	9.5	104.0	02:41:17
	REM 4	20.0	80.0	03:50:47

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

A full sleep hypnogram for the first night (Figure 15) and a full hypnogram for the second night (Figure 16) demonstrate the distribution of sleep stages, stage shifts, and distribution of awakenings.

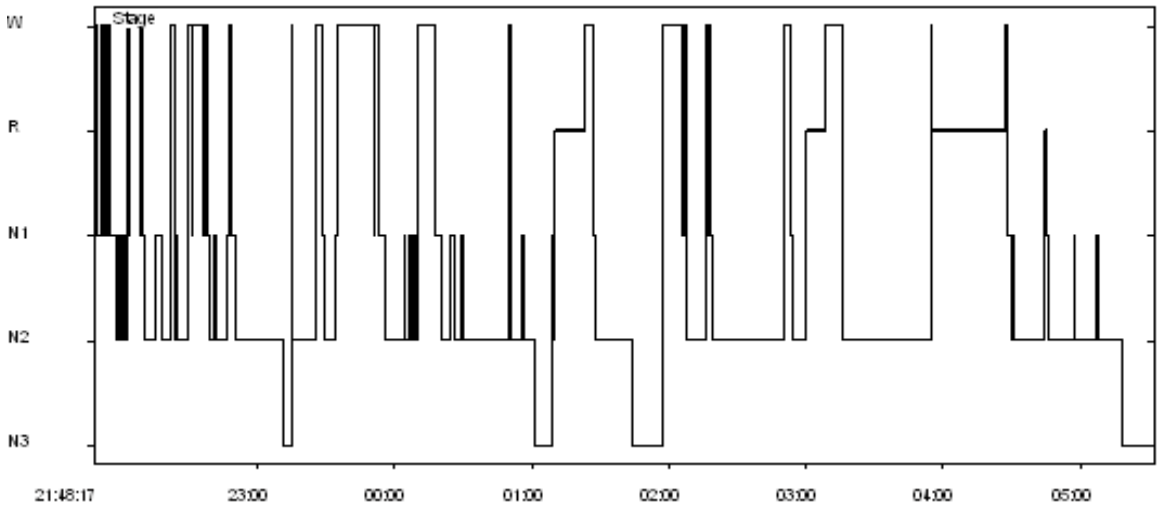


Figure 15. Sleep hypnogram for the first night (Case 5), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

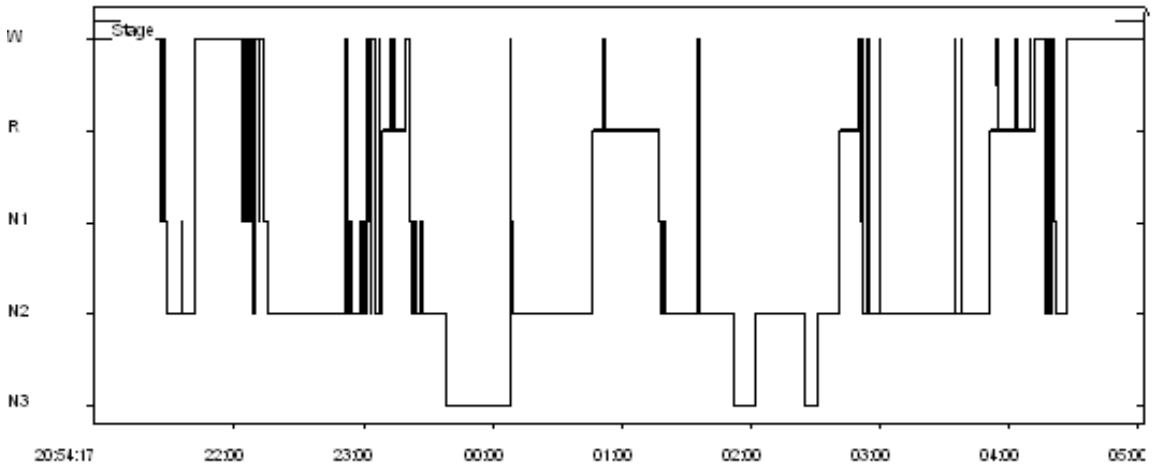


Figure 16. Sleep hypnogram for the second night (Case 5), derived from EEG recordings. Horizontal axis indicates time in hours. Vertical Axis indicates sleep stages. W = waking; R = REM sleep; N1 = stage 1 sleep; N2 = stage 2 sleep; N3 = stage 3 sleep.

The efficiency of sleep was analysed according to sleep quantity and sleep quality.

Sleep quantity

The sleep parameters included to assess sleep quantity were total sleep time and the percentage of time spent in sleep (Table 24). Mrs J was able to maintain a high percentage of sleep over both nights. As such, for sleep quantity, no remarkable results were noted.

Table 24.

Sleep Quantity: Case 5

	TST	*% Sleep*
Night 1	388.50	83.70
Night 2	371.00	76.00

Note: TST = The total number of minutes spent in sleep. % Sleep = The percentage of time spent in sleep from lights off to lights on.

Sleep quality

Analysis of sleep parameters that assessed sleep quality (Table 25) did not indicate any substantial sleep disturbances on the first night and the second night. Instead, the results indicated mild disturbances across all parameters on both nights.

Table 25.

Sleep Quality: Case 5

	Awakenings	*WI*	*Arousals*	*AI*	*Micro-arousals*	*MI*
Night 1	27.00	4.17	74.00	11.40	65.00	10.04
Night 2	32.00	5.18	78.00	12.60	65.00	10.51

Note: WI = Awkening Index: defined as the average number of awaknings per hour of sleep
AI = Arousal Index: defined as the average number of arousals per hour of sleep time
MI = Micro-arousal Index: defined as the average number of micro-arousals per hour of sleep time

Analysis of additional sleep parameters on the first night indicated that two arousals were associated with leg movements, 22 arousals were associated with snore events, and one arousal was associated with respiratory events. The number of arousals associated with respiratory events indicated that Mrs J had an Apnea + Hypopnea Index (AHI) of 0.00 on the

first night, which indicated a complete lack of sleep disordered breathing (Benbir & Guilleminault, 2007).

Analysis of additional sleep parameters on the second night indicated that 20 arousals were associated with snore events, and none with respiratory events. The number of arousals associated with respiratory events indicated that Mrs J had an Apnea + Hypopnea Index (AHI) of 0.00, which indicated a complete lack of sleep disordered breathing (Benbir & Guilleminault, 2007).

Comparison of Dreaming Cases and Non-Dreaming Cases

1. Neuropsychological Assessment

Neuropsychological tests scores were documented for all the non-dreaming cases and all the dreaming cases (Table 26).

Table 26.

Neuropsychological Testing

	Non-Dreaming Cases		*Dreaming Cases*		
Test	*Case 1*	*Case 2*	*Case 3*	*Case 4*	*Case 5*
Luria's Complex Visual Scenes	8/11	9/11	9/11		10/11
Boston Naming Test	38/40	46/60	33/60		31/60
Rey-Osterrieth Complex Figure					
Copy	25/36	32/36	27/36	32/36	
Immediate Recall	4.5/36	15/36	6/36	9/36	
WAIS-III Blocks	1-5 correct	*z* score = 8	*z* score = 5		*z* score = 6
Digit Span					
Forwards	6	5	5	6	7
Backwards	3	6	3		4
Corsi's Blocks					
Forwards	5	6	5		6
Backwards	5	4	4		
Benton's Visual Retention Test	6/7	6/7	3/7		2/7
Babcock Story					
Trial A	7/21	7/21	4/21	8/21	7/21
Trial B	8/21	12/21	7/21	12/21	10/21
Revisualization					
South African Flag	normal	normal	normal	normal	normal
Canary	normal	normal	normal		normal
Patient's House		normal	normal	normal	normal
Husband's Face	normal				
Sleep Laboratory	normal	normal	normal		normal
Bicycle Drawing Test	normal				

Where possible, scores for both the non-dreaming and dreaming cases were combined and means were established. Table 27 shows the comparison of these mean scores. Due to variability of assessment battery, the mean values expressed in Table 27 were composed of the available scores for the non-dreaming and dreaming cases for each test.

Table 27.

Neuropsychological Test Means

Test	*Overall*	*Non-Dreamers*	*Dreamers*
Luria's Complex Visual Scenes	9.00	*8.50*	*9.50*
Boston Naming Test	41.75	51.30	32.00
Rey-Osterrieth Complex Figure			
Copy	29.00	28.50	29.50
Immediate Recall	8.63	9.75	7.50
WAIS-III Blocks	20.00	24.00	18.00
Digit Span			
Forwards	5.80	5.50	6.00
Backwards	4.00	4.50	3.50
Corsi's Blocks			
Forwards	5.50	5.50	5.50
Backwards	4.33	4.50	4.00
Benton's Visual Retention Test	4.25	6.00	2.50
Babcock Story			
Trial A	6.60	7.00	6.33
Trial B	9.80	10.00	9.66

Note: Overall (n = 5), Non-Dreaming Group (n = 2), Dreaming Group (n = 3).
Test score ranges: Luria (0-11); BNT (0-60); ROCF (0-36); WAIS-III Blocks (0-68); Digit Span, Forwards (0-),Backwards (0-); Corsi's Blocks, Forwards (0-), Backwards (0-); BVRT (0-10); Babcock (0-21).

Neuropsychological assessment did not reveal any striking differences between the non-dreaming and dreaming cases. However, tests results did indicate that the non-dreaming cases performed substantially better on the Boston Naming Test and on Benton's Visual Retention Test. The difference on Benton's Visual Retention Test was important as it indicated memory loss was greater for the dreaming cases in comparison to the non-dreaming cases. This was also demonstrated with visual long-term memory as the non-dreaming cases performed marginally better on the immediate recall trial of the Rey-Osterrieth Complex Figure in comparison to the dreaming cases.

2. Pittsburgh Sleep Quality Index

Table 28.

Pittsburgh Sleep Quality Index: Test Means

	Mean Scores		
Component	*Overall*	*Non-Dreamers*	*Dreamers*
Subjective Sleep Quality	0.8	0.5	1.0
Sleep Latency	1.2	1.0	1.3
Sleep duration	1.2	1.0	1.3
Habitual sleep efficiency	1.4	1.5	1.3
sleep disturbances	1.4	1.5	1.3
Use of sleeping medication	0.2	0.0	0.3
Day time dysfunction	1.2	1.0	1.3
Global PSQI score	7.4	6.5	8.0

*Note: Overall (*n = *5); Non-Dreamers (n* = 2); Dreamers (*n* = 3).

Note: Each individual component score has a possible range of 0-3. For each component score, a score of '0' indicates no difficulty, while a score of '3' indicates severe difficulty. The global PSQI score is a summation of the seven component scores and has a posssible range of 0-21. A score of '0' indicates no difficulty, while a score of '21' indicates severe difficulties in all areas.

Analysis of the Pittsburgh Sleep Quality Index did not reveal any striking differences between the non-dreaming and dreaming cases. The dreaming cases reported, on average, slightly more difficulties overall than the non-dreaming cases.

3. The Efficiency of Sleep

The efficiency of sleep was analysed according to sleep quantity and sleep quality.

Sleep quantity

On the first night in the sleep laboratory, the non-dreaming cases spent much less time in sleep than the dreaming cases (Table 29). In addition, the non-dreaming cases spent much less time as a percentage of time from lights off to lights on in sleep on the first night in comparison to the dreaming cases.

Conversely, the non-dreaming cases spent more time in sleep on the second night than the dreaming cases. In addition, the non-dreaming cases spent more time as a percentage of time from lights off to lights on in sleep on the second night in comparison to the dreaming cases.

On average over both nights in the sleep laboratory, the non-dreaming cases spent less time in sleep than the dreaming cases. In addition, the non-dreaming cases spent less time as a percentage of time from lights off to lights on in sleep on both nights in comparison to the dreaming cases.

Table 29.

Sleep Quantity: Comparison of Non-dreaming and Dreaming Means

		Overall	*Non-Dreamers*	*Dreamers*
Night 1	*TST*	238.60	99.00	331.66
	% Sleep	49.32	24.72	65.72
Night 2	*TST*	395.40	435.00	369.00
	% Sleep	75.30	81.31	71.29
Average of Both Nights	*TST*	317.00	267.00	350.33
	% Sleep	62.31	53.02	68.51

Note: Overall (n = 5); Non-Dreamers (n = 2); Dreamers (n = 3).

TST = Total number of minutes spent in sleep. % Sleep = The percentage of time spent in sleep from lights off to lights on. All scores represent means.

Total sleep time

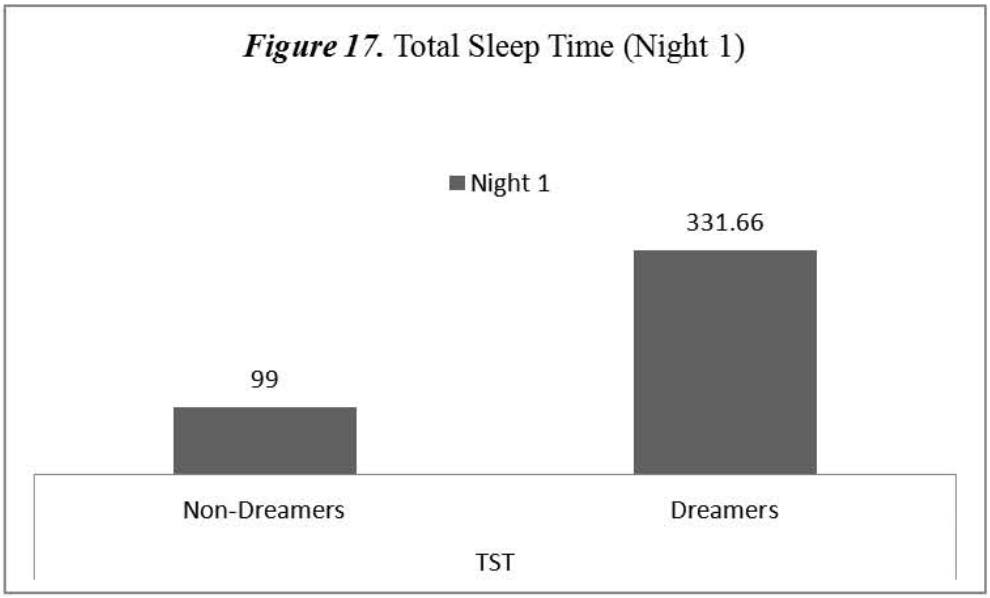


Figure 17. Total Sleep Time (Night 1)

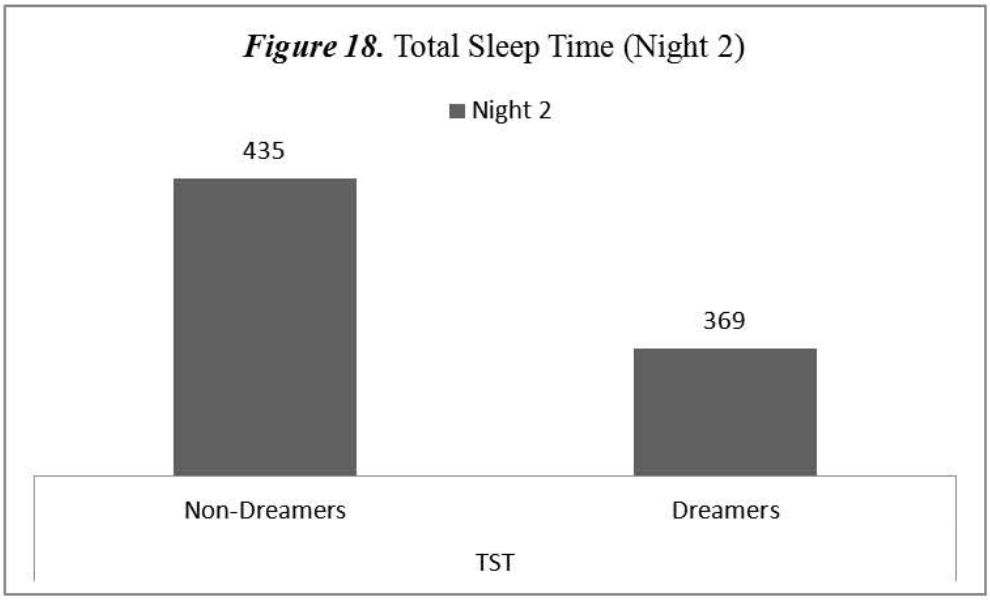


Figure 18. Total Sleep Time (Night 2)

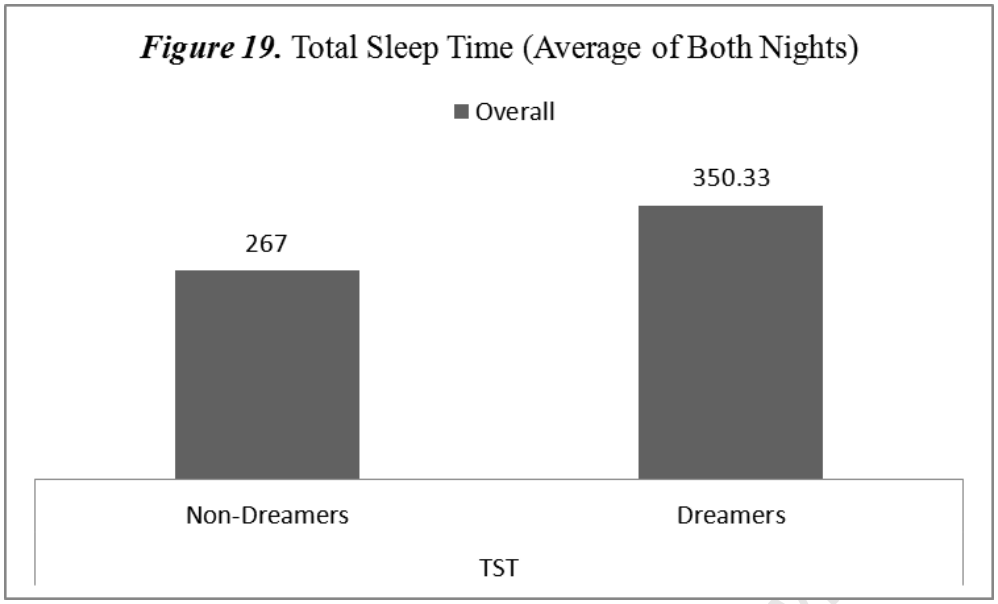


Figure 19. Total Sleep Time (Average of Both Nights)

Analysis of total sleep time thus indicated that on average over both nights (Figure 19), the non-dreaming cases spent less time in sleep in comparison to the dreaming cases. However, when each night was looked at individually, the non-dreaming cases spent much less time in sleep in comparison to the dreaming cases on the first night (Figure 17), and the non-dreaming cases conversely spent more time in sleep than the dreaming cases on the second night (Figure 18).

Percentage of time spent in sleep

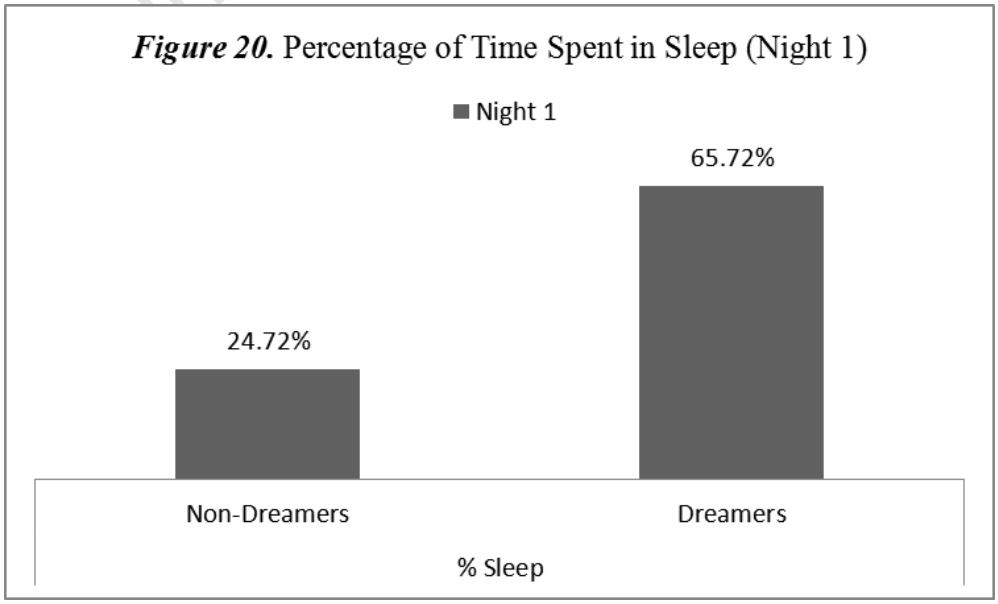


Figure 20. Percentage of Time Spent in Sleep (Night 1)

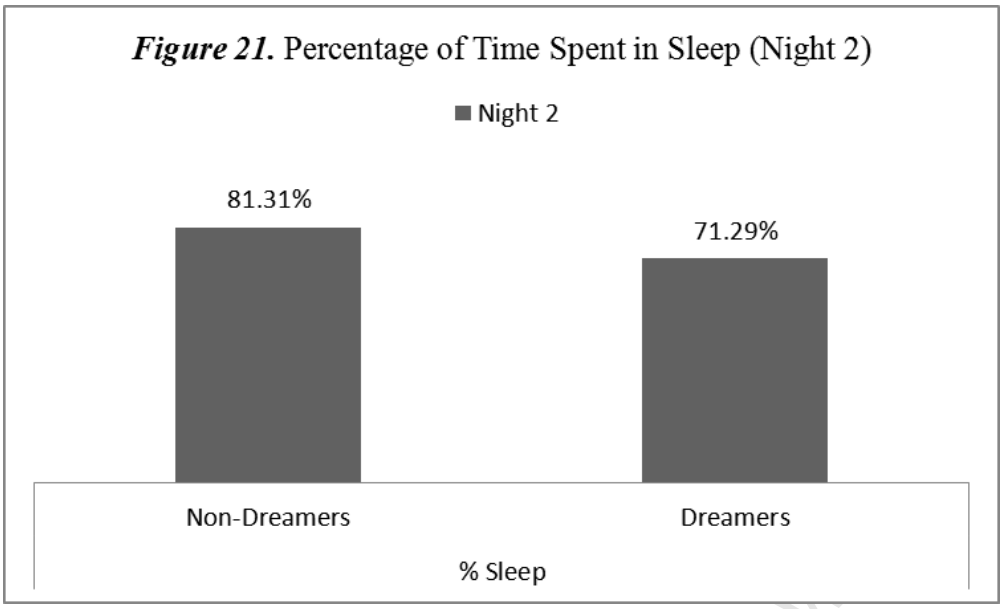


Figure 21. Percentage of Time Spent in Sleep (Night 2)

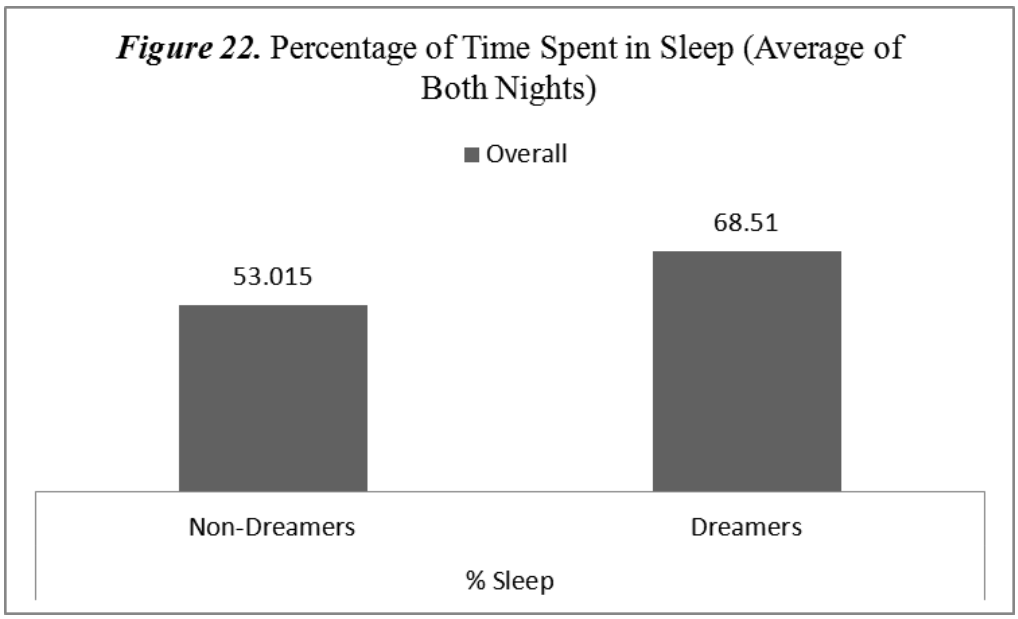


Figure 22. Percentage of Time Spent in Sleep (Average of Both Nights)

Analysis of percentage of time spent in sleep from lights off to lights on thus indicated that on average over both nights (Figure 22), the non-dreaming cases spent a lower percentage of time in sleep in comparison to the dreaming cases. However, when each night was looked at individually, the non-dreaming cases spent a much lower percentage of time in sleep in comparison to the dreaming cases on the first night (Figure 20), and the non-dreaming cases conversely spent a higher percentage of time in sleep than the dreaming cases on the second night (Figure 21).

Sleep quality

On the first night in the sleep laboratory, the non-dreamers documented slightly more sleep disturbances that the dreamers (Table 30). This was not evident in the number of awakenings, arousals, and micro-arousals as the dreaming cases showed a higher number of all three of these parameters. This was accounted for by the substantial difference in amount of time spent in sleep on the first night between the non-dreaming cases and the dreaming cases. It was, however, evident in the percentage of awakenings, arousals, and micro-arousals per hour of sleep time as the non-dreaming cases documented a slightly higher percentage for all three of these parameters.

On the second night in the sleep laboratory, the non-dreaming cases' sleep was substantially more disturbed than the dreaming cases. While little difference was seen between the non-dreaming and dreaming cases on the amount of awakenings and percentage of awakenings per hour of sleep time, a substantial difference was seen between the non-dreaming and dreaming cases on the amount of arousals and micro-arousals as well as the percentage of arousals and micro-arousals per hour of sleep time.

On average over both nights in the sleep laboratory, with the exception of the amount of awakenings, the non-dreaming cases displayed substantially more sleep disturbances on all parameters of sleep quality.

Table 30.

Sleep Quality: Comparison of Group Means

		Overall	*Non-Dreamers*	*Dreamers*
Night 1	*Awakenings*	21.00	16.50	24.00
	WI	11.25	21.54	4.40
	Arousals	94.20	46.00	126.33
	AI	27.42	32.75	23.86
	Micro-arousals	70.60	32.50	97.00
	MI	19.52	21.36	18.29
Night 2	*Awakenings*	24.40	23.00	25.33
	WI	3.94	3.59	4.16
	Arousals	271.80	492.00	125.00
	AI	37.94	64.15	20.46
	Micro-arousals	233.00	444.00	92.33
	MI	31.92	56.95	15.24
Average of Both Nights	*Awakenings*	22.70	19.75	24.66
	WI	7.59	12.56	4.28
	Arousals	183.00	269.00	125.66
	AI	32.68	48.45	22.17
	Micro-arousals	152.10	238.25	94.66
	MI	25.72	39.15	16.76

Note: Overall (n = 5); Non-Dreamers (n = 2); Dreamers (n = 3)

WI = Awkening Index: defined as the average number of awaknings per hour of sleep time

AI = Arousal Index: defined as the average number of arousals per hour of sleep time

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

Amount of Awakenings

Analysis of the amount of awakenings revealed that the non-dreaming cases had slightly less awakenings that the dreaming cases on the first night, the second night, and on average over both nights in the sleep laboratory.

Awakening Index

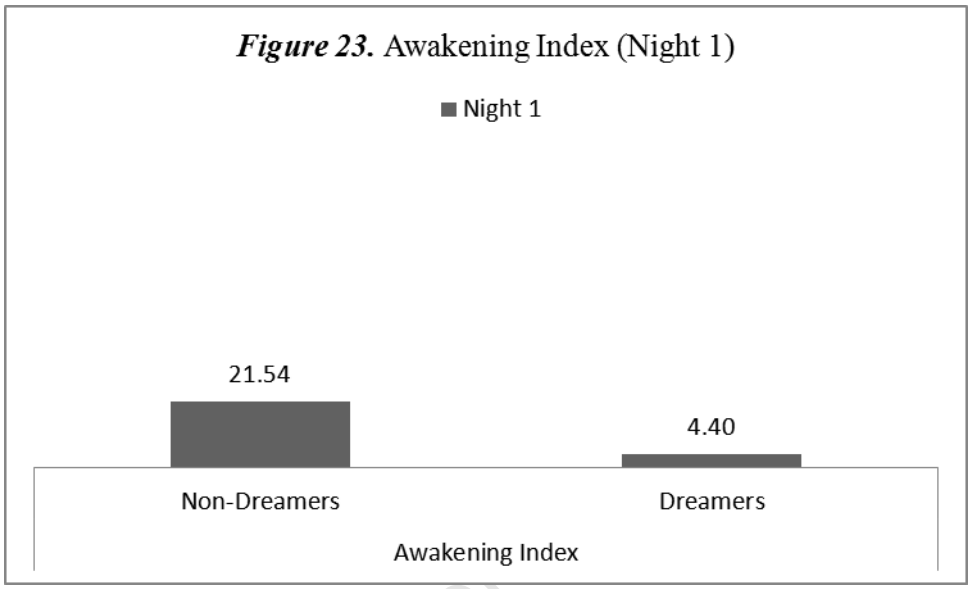


Figure 23. Awakening Index (Night 1)

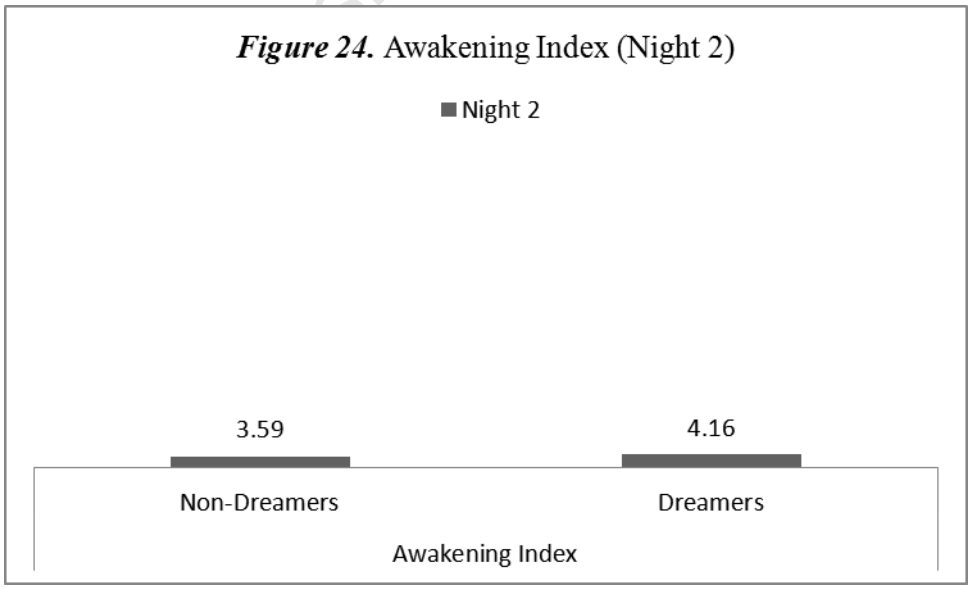
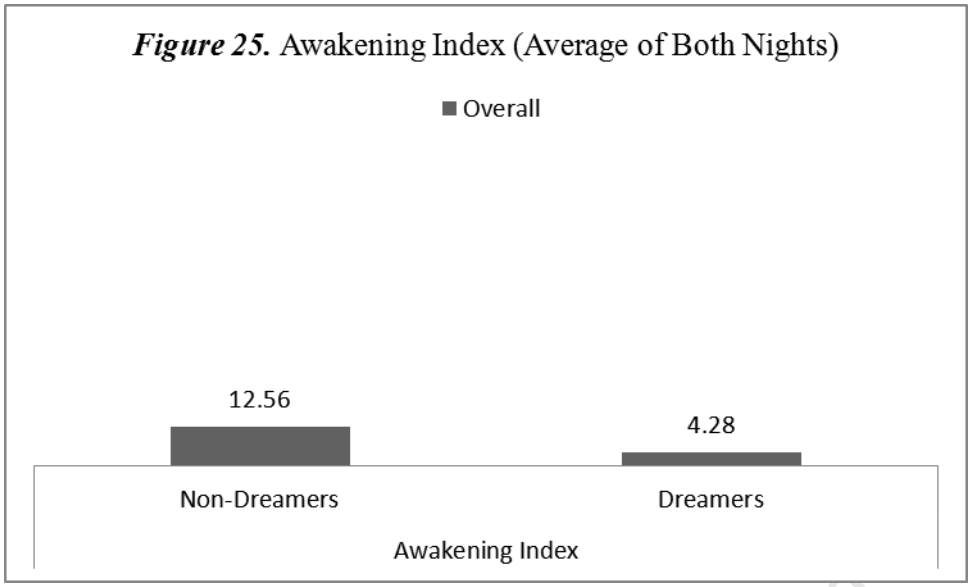


Figure 24. Awakening Index (Night 2)

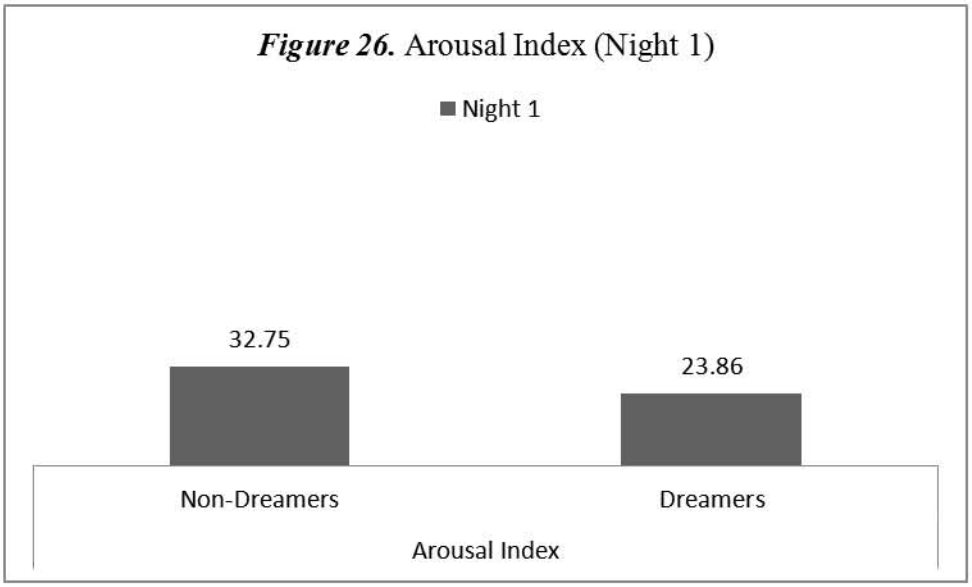


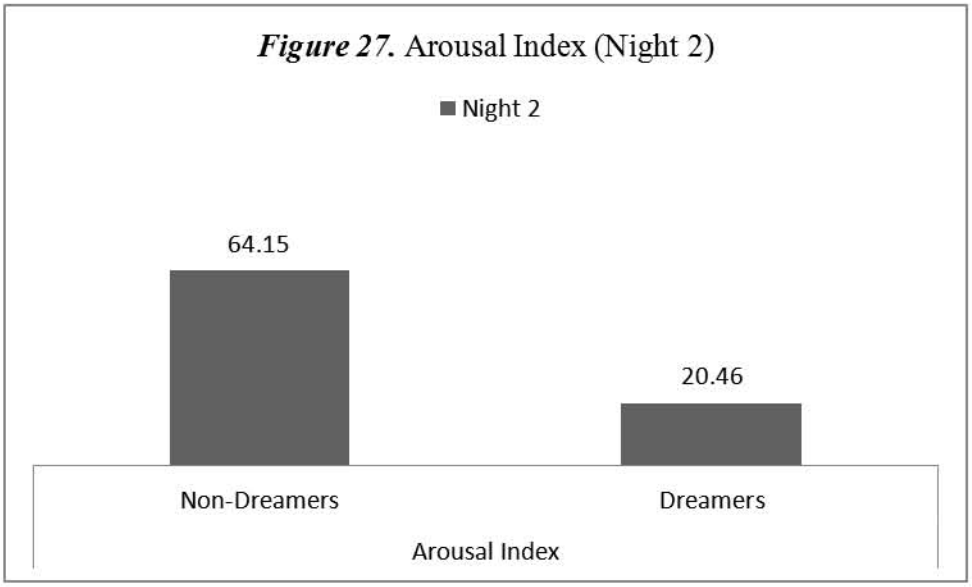
Analysis of the percentage of awakenings per hour of sleep time thus indicated that on average over both nights in the sleep laboratory (Figure 25), the non-dreaming cases had a higher percentage of awakenings than the dreaming cases. However, analysis of each night separately indicated that non-dreaming cases had a substantially higher percentage of awakenings on the first night in comparison to the dreaming cases (Figure 23), but that there was very little difference between the non-dreaming and dreaming cases on the second night (Figure 24).

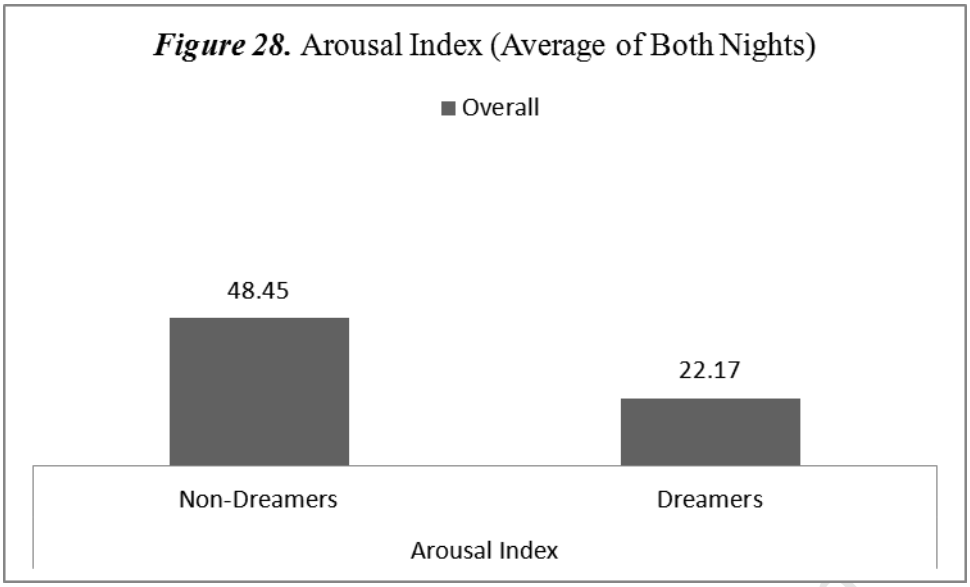
Amount of Arousals

Analysis of the amount of arousals indicated that on average over both nights in the sleep laboratory, the non-dreaming cases had a higher number of arousals than the dreaming cases. However, analysis of each night separately indicated that the non-dreaming cases had a substantially higher number of arousals on the second night in comparison to the dreaming cases, but that the dreaming cases had a higher number of arousals on the first night.

Arousal Index







Analysis of the percentage of arousals per hour of sleep time thus indicated that on average over both nights in the sleep laboratory (Figure 28), the non-dreaming cases had a substantially higher percentage of arousals than the dreaming cases. Analysis of each night separately indicated that the non-dreaming cases had a substantially higher percentage of arousals on the first night (Figure 26) and on the second night (Figure 27) in comparison to the dreaming cases, with the greatest difference indicated on the second night.

Amount of Micro-Arousals

Analysis of the amount of micro-arousals indicated that on average over both nights in the sleep laboratory, the non-dreaming cases had a substantially higher number of micro-arousals than the dreaming cases. However, analysis of each night separately indicated that the non-dreaming cases had a substantially higher number of micro-arousals on the second night in comparison to the dreaming cases, but that the dreaming cases had a substantially higher number of micro-arousals arousals on the first night.

Micro-Arousal Index

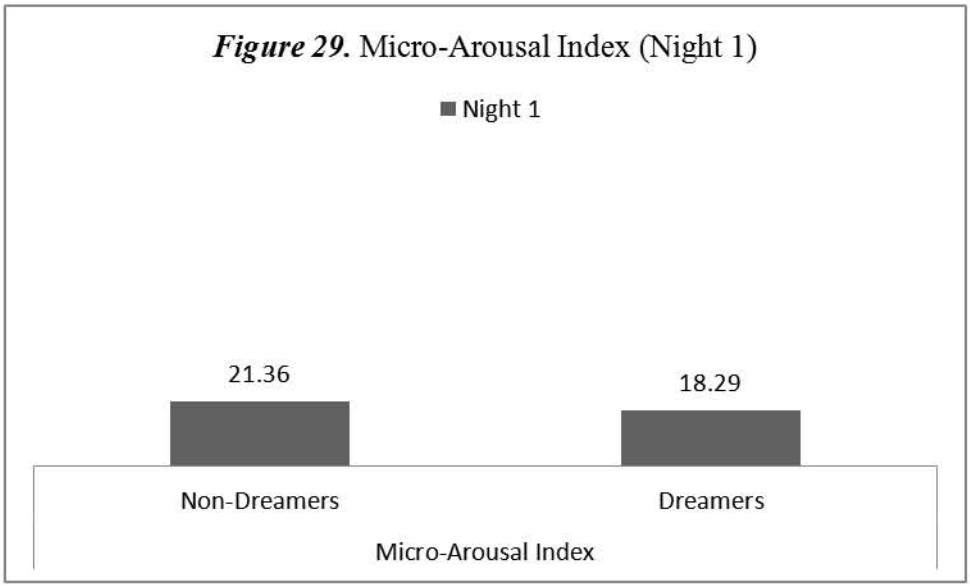


Figure 29. Micro-Arousal Index (Night 1)

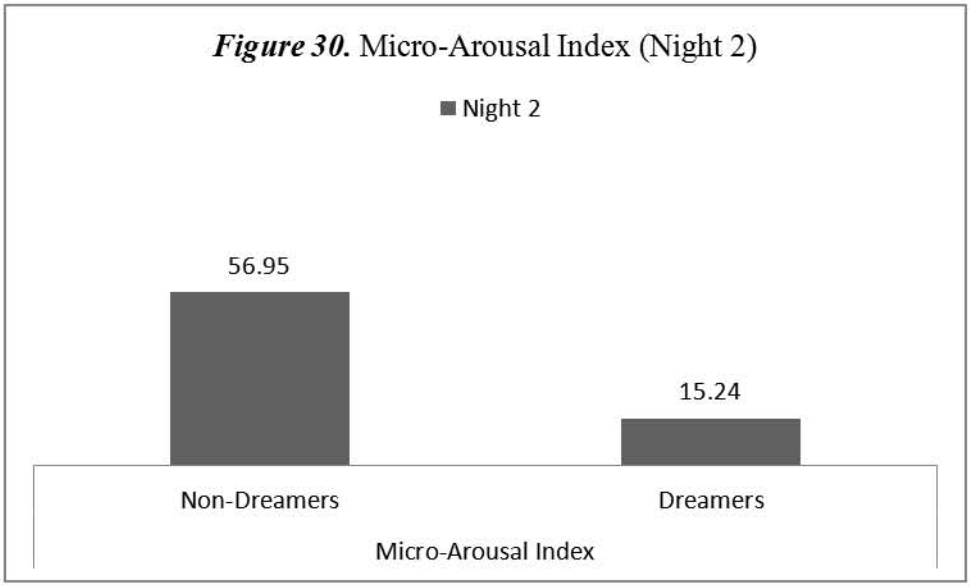
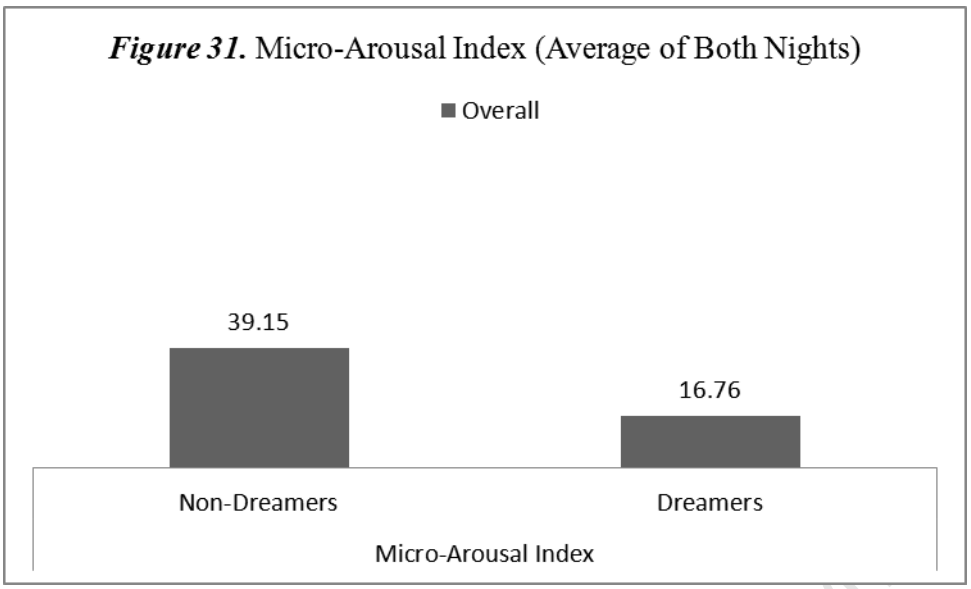


Figure 30. Micro-Arousal Index (Night 2)



Analysis of the percentage of micro-arousals per hour of sleep time thus indicated that the non-dreaming cases had a higher percentage of micro-arousals on the first night (Figure 29), on the second night (Figure 30), and over both nights (Figure 31). The most substantial difference is the percentage of micro-arousals between the non-dreamers and the dreamers was evident on the second night.

DISCUSSION

This study aimed to address the issue that despite decades of research, a function of dreams has yet to be empirically established. Specifically, this study aimed to contribute to the search for a function of dreams by testing the hypothesis that dreams protect sleep. As this is the same theory that had been previously hypothesized by Freud, this study aimed to test the Freudian dream theory. In order to achieve this, this study recorded the sleep patterns of neurological patients who did not dream to see if their sleep was disrupted in comparison to neurological patients who did dream. Findings from case study analyses and comparisons between dreaming and non-dreaming patients have provided preliminary support for the hypothesis that dreams protect sleep. These findings are discussed in the following sections of this paper.

Findings from case studies

One of the particular aims of this study was to replicate the previously published findings that a reduced quality of sleep was experienced by non-dreamers (Bischof & Bassetti, 2004; Poza & Massó, 2006; Solms, 1997). Bishof and Basseti's case, and Poza and Massó's case, are the only two comprehensively reported cases of dream loss following a lesion in the occipital lobes that resulted in subsequent reduced quality of sleep. This study was able to add to that body of data by providing two more comprehensive case studies (Case 1 and Case 2) that demonstrated a reduced efficiency of sleep in occipital stroke patients who had ceased dreaming. These two case analyses replicate the previous findings in the following ways:

1. *Normal REM amounts in non-dreamers*

Firstly, this study was able to demonstrate that normal or near-normal REM amounts could be achieved in patients who had lost the ability to dream. Like Bishof and Bassetti's case (2004) and Poza and Massó's case (2006), Case 1 and Case 2 in this study confirmed the presence of REM in patients that had lost the ability to dream. With Case 1, REM sleep accounted for 5.7% of total sleep time on the first night, and 20.5% of total sleep time on the second night. As REM sleep normally accounts for 20-25% of total sleep time in healthy adults (Chokroverty, 2009), this demonstrated that Case 1 was able to achieve normal REM amounts on the second night. Similarly, while Case 2 did not document REM on the first night, REM accounted for 18.1% of total sleep time on the second night and 15.2% of total sleep time on the third night. While this was marginally lower than the expected result (20-

25%), it did still demonstrate a substantial amount of REM that was similar to that expected of normal healthy adults.

These findings are significant in light of the discovery that REM sleep and dreams are controlled by separate brain mechanisms (Solms, 2000). With the use of subjective accounts of dream loss, clinical interviews, and nocturnal REM sleep interviews, complete cessation of dreaming in Case 1 and Case 2 was confirmed. In addition, neuropsychological assessment of both cases was able to confirm that both patients possessed the cognitive capacity for dream recall, confirming that cessation of dreaming was not a product of memory loss. In particular, both patients were able to accurately recall the internal layout of the sleep laboratory in detail one month after the sleep study, which indicated that long-term memory was intact at the time of the sleep study. As such, this study provides evidence that is able to contribute to the expanding body of knowledge that dreams and REM are doubly dissociable states (Bischof & Bassetti, 2004; Poza & Marti Massó, 2006; Foulkes, 1962; Foulkes & Vogel, 1965; Jus *et* al, 1973; Solms, 2000).

Furthermore, these findings are significant for scientific research into the function of dreams. Most theories of dream function, in particular the activation-synthesis theory (Hobson & McCarley, 1977), are based on the mistaken conflation of REM sleep and dreams. Thus, the finding that cessation of dreams can occur while REM sleep is preserved, highlights the necessity to separate scientific exploration of the function of dreams from theories of the function of REM sleep.

2. Cessation of dreams as a result of occipital damage

Secondly, this study was able to replicate previous findings by demonstrating that posterior cerebral artery infarction, which resulted in corresponding damage to the occipital lobes, resulted in loss of dreaming. Bischof and Bassetti (2004) published a single case of complete cessation of dreaming following bilateral occipital stroke, and Poza & Massó (2006) published a single case where cessation of dreams occurred following a unilateral left tempero-occipital hematoma. Similarly, the non-dreaming cases in this study (Case 1 and Case 2), both demonstrated occipital lobe damage. These findings are significant in light of the fact that precise localization of the posterior lesion responsible for loss off dreaming has not been conclusively established. However, it is also important to note that while Case 1 and Case 2 in this study demonstrated occipital lobe damage, both of these non-dreaming patients also displayed thalamic damage, bilaterally and unilaterally respectively. This did not occur in Case 3, 4, and 5, the dreaming cases. Further inspection of Bischof and Bassetti's non-dreaming case also indicated that acute infarction of the right posterolateral thalamus was

reported. Thus, these findings indicate that the thalamus may play a role in the generation of dreams. This is consistent with neuro-imaging studies that found the thalamus to be highly activated during REM sleep (Braun *et al*, 1997; Maquet *et al*, 2006). Further research, however, is necessary to establish whether thalamic damage alone can lead to cessation of dreaming.

3. *Polysomnographic recordings of non-dreaming sleep*

To date, Bishof and Basseti's case (2004), and Poza and Massó's case (2006), are the only two documented cases of total dream loss that include full clinical, neuropsychological, neuroimaging and polysomnographic data. Furthermore, the aim of these studies was to demonstrate that regular REM cycles could occur with complete cessation of dreams. While reduced sleep efficiency was mentioned, it was not the main focus of these studies. Thus, this study contributes to the understanding of dreams and sleep by, for the first time, specifically focusing on the relationship between loss of dreaming and disturbed sleep.

4. *Reduced efficiency of sleep in non-dreamers*

Lastly, the findings of this study replicate Bishof and Basseti's case (2004), and Poza and Massó's case (2006) by demonstrating disrupted sleep architecture in non-dreaming patients. Case 1 and Case 2 both demonstrated extreme difficulties with adapting to the sleep laboratory environment with a total sleep time on the first night of only 175 minutes and 23 minutes respectively. While nocturnal REM sleep interviews and unfamiliarity to the sleep laboratory environment are possible confounding factors, it is unlikely that they accounted for the lack of sleep on the first night completely. In addition, Case 3, Case 4, and Case 5 were able to adapt much more easily to the sleep laboratory environment (total sleep time of 311, 295.5, and 388.5 minutes on the first night respectively). Thus, the argument drawn from these findings is that lack of dreaming may affect the ability to adapt to an unfamiliar sleep setting. This supports the hypothesis that dreams protect sleep by showing non-dreaming sleep to be more vulnerable to external factors. By doing so, this gives further credibility to the argument set forth by Portas *et al.* (2000) that the sleeping brain may be able to, in fact, process external stimuli and detect meaningful events. To explain this, if external stimuli can be incorporated into sleeping mentation and affect dream content, it is reasonable to assume that these stimuli would be more prevalent in an unfamiliar setting with unfamiliar stimuli. Thus, the fact that the non-dreaming cases were unable to maintain sleep on the first night, in an unfamiliar setting, gives support for the hypothesis that dreams protect sleep by demonstrating the inability of the non-dreaming patients to divert these stimuli, thus resulting in substantially reduced overall sleep time.

Furthermore, the findings from the case studies that were analysed in this study demonstrated increased sleep disturbances in both non-dreaming patients. Polysomnographic recordings demonstrated disrupted sleep for Case 1 on both nights in the sleep laboratory. On the first night, mild disturbances were noted demonstrated by the number and frequency of awakenings, arousals, and micro-arousals. On the second night, mild disturbances were noted by the number and frequency of awakenings, while severe sleep disturbances were noted by the number and frequency of arousals and micro-arousals. In addition, 132 stage shifts were noted during the second night, indicating general difficulties in entering and maintaining sleep stages.

Similarly, Case 2 showed substantially disturbed sleep on both nights. On the first night, a substantial frequency of awakenings was noted. On the second night, substantially high levels in the number and frequency of arousals and micro-arousals were recorded. This indicated difficulties maintaining sleep on both the first and second night in the sleep laboratory.

These findings are consistent with the polysomnographic data published by Bishof and Basseti (2004) that showed abnormal sleep architecture in a non-dreaming patient which they called "sleep maintenance insomnia" (pg. 585). In addition, this directly supports the hypothesis that dreams protect sleep by demonstrating how cessation of dreaming resulted in disrupted sleep architecture.

Additional considerations of case study analyses

While the findings from the case studies do show support for the hypothesis that dreams protect sleep, there are certain methodological considerations and additional factors that need to be taken into account. These include the following:

1. *What is normal sleep?*

In order to fully understand the findings of the individual case studies presented in this study, it is necessary to understand how these findings differ from normal sleep architecture. Consensus on the minimal and maximum amount of sleep interruptions that characterize disturbed sleep has not been reached (Boselli *et al*, 1998). Thus, while certain sleep parameter means have been published (Chokroverty, 2009), and while there has been a move to try to establish norms for all other parameters (Boselli *et al*, 1998; Vock *et al*, 2002), agreement on the boundary between disturbed and undisturbed sleep is still under debate.

2. *Other factors that can affect sleep*

In addition to the difficulty due to lack of agreement over what constitutes normal sleep, there are other factors that have been shown to affect sleep efficiency that need to be taken into account. Foremost of these is abundant evidence that stoke affects sleep (Bakken *et al*, 2011; Gottselig *et al*, 2002; Hermann *et al*, 2003; and Vock *et al*, 2002) that can result in lowered total sleep time and lowered sleep efficiency (Bassetti & Aldrich, 2001) and increased awakenings (Giubilei *et al*, 1992). In addition, stroke-related treatments have also been shown to reduce sleep efficiency (Chokroverty & Montagna, 2009). One of the dominant arguments of the mechanism by which stroke reduces sleep efficiency, is based on the finding that hemispheric stroke patients have significantly reduced spindle activity during sleep. The theory is that spindle oscillations are involved in blocking sensory input to the cortex during sleep, thus a reduction in spindle activity can contribute to a reduction in sleep continuity (Gottselig *et al*, 2002). While specific spindle activities were not the focus of this study, it is worthwhile to mention that both Case 1 and Case 2 had substantially less spindle activity than the dreaming cases, particularly during non-REM sleep. However, the relationship of spindle activity and cognition is not known. Further research is still required in this area. Thus, while it is problematic to analyse Case 1 and Case 2 results at face value, as a substantial amount of the sleep disturbances that were shown may possibly be accounted for by the normal effects that stroke has on sleep architecture, there is no conclusive evidence to suggest that the sleep disturbances demonstrated by the non-dreaming cases in this study can be better accounted for by the effects of stroke.

Nonetheless, because stroke has been shown to affect sleep architecture, it is necessary to compare the results of non-dreaming patients with the results of stroke patients who are still able to dream. Case 2, Case 3, and Case 4 in this study provide such an opportunity and the results of this comparison will be discussed at length in the following sections of this dissertation. What can be noted, however, is that while stroke has been shown to affect sleep efficiency, this effect has been shown to be most noted with male patients (Bakken *et al*, 2011). As both of the non-dreaming cases in this study were female, this further supports that argument that the disrupted sleep architecture that was observed cannot be entirely explained by simply the effect that stroke may have had on sleep quality.

A second additional factor that has been shown to affect sleep architecture is age (Boselli *et al*, 1998; Redline *et al*, 2004). In particular, studies have shown that normal aging increases the number of arousals throughout sleep (Boselli *et al*, 1998). Both Case 1 (65 years old at time of sleep study) and Case 2 (62 years old at time of sleep study) would fall into the

age bracket where sleep is most affected. As such, it is possible that a certain amount of the non-dreaming patients sleep disruptions could be accounted for by the normal effects that aging has on sleep. Because of this, it is necessary to compare the results for Case 1 and Case 2, with stroke patients who fall in a similar age bracket. Due to relatively rare prevalence of such patients, it was not possible in this study to obtain exact age matched controls. However, Case 3 (51 years old at time of sleep study), Case 4 (67 years at time of sleep study), and Case 5 (45 years old at time of sleep study) all fall into the age bracket where sleep can be effected by normal aging.

A third, and possibly most significant, factor is the effect that sleep disordered breathing can have on sleep architecture. Previous studies have indicated that roughly 50-70% of stroke patients have sleep disordered breathing, mainly obstructive sleep apnoea (Bassetti *et al*, 2006; Hermann *et al*, 2003). An apnoea is defined as a cessation of breathing for 10 or more seconds (McNamara *et al*, 1993). General consensus seems to regard an Apnea + Hypopnea Index (AHI), i.e. the number of apneic and hypopneic events per hour of sleep time, of ≥ 10 to be pathological (Bassetti *et al*, 2006; Hermann *et al*, 2003). However, it has also been argued that an index of ten events per hour is only pathological if accompanied by day time sleepiness (Benbir & Guilleminault, 2007).

All five cases in this study showed inconsistencies in the amount of apneic and hypopneic events over the different nights in the sleep laboratory. For the non-dreaming cases, Case 1 had Apnea + Hypopnea Index (AHI) of 7,9 on the first night, and 14.5 on the second night. Case 2 had an AHI of 0.0 on the first night, 68.6 on the second night, and 2.6 on the third night. For the dreaming cases, Case 3 had an AHI of 9.3 on the first night, and 2.8 on the second night. Case 4 had an AHI of 18.3 on the first night, and 13.4 on the second night. Case 5 had and AHI of 0.00 on both nights in the sleep laboratory. In addition, all five patients did not report day time sleepiness at the time of the sleep study.

Therefore, it is probable that sleep disordered breathing did have an effect on sleep architecture for all cases (except Case 5), but it is unlikely that this better accounted for the level of sleep disruptions that were present. Nonetheless, it would be preferable to exclude patients with sleep disordered breathing in future analyses to rule this out as a confounding factor. However, due to the high prevalence of sleep disordered breathing in stroke patients (50-70%; Bassetti *et al*, 2006; Hermann *et al*, 2003), this may prove extremely difficult. However, as sleep-disordered breathing was present in both the non-dreaming and dreaming cases in this study, the comparison of sleep disturbances between the non-dreaming and dreaming cases controls for the possible effect that sleep-disordered breathing has on their

sleep architecture. The results of this comparison will be discussed at length in the following sections of this dissertation.

A fourth factor that also needs to be taken into account is the effect that sleep deprivation on the first night may have had on sleep architecture on the second night. This study was specifically designed to orientate patients to the sleep laboratory by studying them for two consecutive nights in the sleep laboratory. By doing so, the first night would function as an orientation night allowing a more accurate reading of the patients sleep architecture to be obtained on the second night. However, for Case 1 and Case 2, the fact that they may have been more vulnerable to an unfamiliar sleep environment simply because they could not dream (discussed above), produces a confounding factor for this study. Lack of sleep has been shown to result in sleep rebound, especially REM rebound (Chokroverty, 2009). Thus, it is possible that the sleep architecture displayed by Case 1 and Case 2 on the second night is a direct result of sleep deprivation on the first night. Because of this, future analyses should possibly study patients for more than two consecutive nights in order to rule out these effects.

Therefore, the results of the case study analyses in this study do give support for the hypothesis that dreams protect sleep by demonstrating how loss of dreaming reduced the efficiency of sleep in Case 1 and Case 2. However, because of additional factors discussed above, i.e. the difficulty in understanding what constitutes abnormal sleep, the effect that stroke, age, and sleep disordered breathing may have had on the results, and the possible confounding factor of sleep rebound, it is necessary to compare the results from the non-dreaming patients, with dreaming controls that have also experienced a stroke. This analysis was provided in this study by the dreaming cases (Case 3, 4, and 5).

Findings from comparison of non-dreaming and dreaming patients

In order to fully understand the findings from the non-dreaming cases, it was necessary to compare these findings with patients with similar pathologies that were able to dream. Thus, the results from the two non-dreaming cases (Case 1 and Case 2) were combined and means were established. These were then compared to the mean scores of Case 3, Case 4, and Case 5. This comparison supported the hypothesis that dreams protect sleep which will be discussed in the following sections of this study. However, in order to compare the findings from the non-dreaming and dreaming patients, it was first necessary to establish that all cases demonstrated similar pathologies and similar neuropsychological profiles, and it was necessary to gain an understanding of patients' subjective sleep quality.

1. *Pathological findings*

In order to establish differences between the two groups, it was first necessary to establish that both groups consisted of patients who had similar pathologies in order to control for the effect that stroke can have on sleep efficiency (discussed above). From the individual case study analyses, it is clear that all patients had suffered thrombotic infarction in the PCA territory that had resulted in corresponding occipital lesions. However, one striking difference is that both cases in the quasi-experimental group had suffered bilateral infarction, while all three cases in the control group had unilateral lesions. This raises an interesting debate over whether bilateral hemispheric stroke could result in greater reduction in sleep quality compared to unilateral hemispheric stroke. To our knowledge, no study to date has analysed the effect of specific lesion location on sleep architecture. However, Bassetti *et al.* (2006) did note that severity and topography of stroke did not affect the presence or severity of sleep disordered breathing in hemispheric stroke patients. Thus, while this pathological difference between the two groups needs to be kept in mind, there is no evidence to suggest that bilateral damage would result in a greater reduction of the efficiency of sleep than unilateral damage.

2. *Neuropsychological profiles*

Another important factor that needed to be controlled for when comparing the non-dreaming and dreaming patients, was the neuropsychological and cognitive abilities of the individual cases. In particular, neuropsychological assessment was important in order to establish that individual patients had the cognitive capacity necessary for dream recall, and to test the cognitive functions that have traditionally been associated with loss of dreaming. In this study it was concluded that all participants possessed the cognitive capacity for dream recall. Comparison of neuropsychological test means of the non-dreaming and the dreaming patients did not reveal strikingly different neuropsychological profiles. However, of note was the fact that the dreaming patients performed marginally worse than the non-dreaming patients on all subtests (except those that tested short-term memory), and performed most notably worse on tests of long-term memory. This result further supports the argument that lack of dream recall is not a product of memory loss (Solms, 1997; Yu, 2007) as the cases in the study who could not recall dreams, were in fact the patients that displayed better memory function. Furthermore, all five cases in this study were able to accurately recall the internal layout of the sleep laboratory one month after the sleep study. This provides clear evidence that lack of dream recall in Case 1 and Case 2 was not a product of memory loss.

3. *Subjective sleep quality*

A third factor that needed to be taken into consideration when comparing the non-dreaming and dreaming patients, was how the individual patients subjectively experienced quality of sleep. In a preliminary study conducted by Solms (1997), patient's subjective reports on whether they experienced dreams or not, was compared with subjective reports on their quality of sleep. A significantly lower level of subjective sleep quality was reported by patients who reported loss of dreaming. However, in this study, using the Pittsburgh Sleep Quality Index (Buysse *et al*, 1989), no difference was found between the non-dreaming and dreaming patients on subjective sleep quality. This is interesting to note, as it shows that despite vast objective differences in sleep architecture (discussed below), the groups did not differ on their subjective experience of sleep quality. Subjective reports of sleep quality, however, are not ideal for scientific investigation, and therefore the following sections of this paper will focus on objective measures gained from polysomnographic recordings.

Therefore, from these findings, it is clear that the non-dreaming and dreaming patients did not differ substantially from each other in terms of pathology, cognitive functioning, or subjective understanding of sleep quality that could in any way confound the results of this study. With this in mind, the findings from polysomnographic recordings show clear support for the hypothesis that dreams protect sleep by demonstrating reduced efficiency of sleep in the non-dreaming patients in comparison to the dreaming patients. Efficiency of sleep was analysed in terms of sleep quantity and sleep quality.

Sleep study findings

Sleep quantity

While this study had hypothesized that sleep quantity would be reduced in the quasi-experimental group in comparison to the control group, the results did not indicate direct support for this hypothesis. Polysomnographic recordings showed that on the second night in the sleep laboratory, the non-dreaming patients actually spent more time in sleep on average than the dreaming patients (435 minutes in comparison to 369 minutes). In addition, the non-dreaming patients spent more time as a percentage of the time from lights off to lights on in sleep compared to the dreaming patients (81.31% in comparison to 71.29%).

However, a possible confounding factor of these results, as discussed in the individual case analyses, is the lack of sleep that the non-dreaming documented on the first night in the sleep laboratory (*mean* = 99 minutes). Thus, it is possible that the sleep architecture displayed by the non-dreaming patients on the second night is a direct result of sleep deprivation on the first night. As the dreaming patients were able to sleep more effectively on the first night

(*mean* = 331.66 minutes), the same effect on the second night was not seen. As such, it is impossible in this study to compare sleep quantity between the two groups on the second night. This also indicates a potential flaw in the design of this study, suggesting that more than two consecutive nights in the sleep laboratory would be necessary for future analyses.

However, the findings from the first night should also not be ignored. The fact that the non-dreaming patients slept for substantially less time than the dreaming patients on the first night is in itself very interesting. As discussed in the discussion of the case analyses, a potential argument that could be proposed is that lack of dreaming itself may, in fact, affect the ability to adapt to an unfamiliar sleep setting. In this way, it can be argued that the lack of sleep displayed by the non-dreaming patients on the first night, and consequently the increase of overall sleep time on the second night, does support the hypothesis that dreams protect sleep by showing how the non-dreaming patients were unable to adapt effectively to the sleep laboratory environment because of loss of dreaming. This, however, is merely a tentative conclusion based on the observations made in this study. Further investigation is required to establish validity of this hypothesis.

Sleep quality

While comparison of the quantity of sleep between the non-dreaming and dreaming patients leaves doubt as to whether the non-dreaming patients were experiencing reduced efficiency of sleep, the findings in terms of sleep quality are much more conclusive. Bosselli *et al*. (1998) argue that disturbed sleep is characterized by fragmentation, frequent interruptions, and lack of continuity. All of this was evident in the non-dreamer's sleep during both nights in the sleep laboratory in comparison to the dreamers.

Analysis of the amount of awakenings revealed that the non-dreaming cases had slightly less awakenings that the dreaming cases on the first night, the second night, and on average over both nights in the sleep laboratory (19.75 in comparison to 24.66). Conversely, analysis of the percentage of awakenings per hour of sleep time indicated that on average over both nights in the sleep laboratory, the non-dreaming cases had a higher percentage of awakenings than the dreaming cases (awakening index of 12.56 in comparison to 4.28). However, analysis of each night separately indicated that the non-dreaming cases had a substantially higher percentage of awakenings on the first night in comparison to the dreaming cases (awakening index of 21.54 in comparison to 4.40), but that there was very little difference between the non-dreaming and dreaming cases on the second night. Thus, analysis of the number and frequency of awakenings over both nights in the sleep laboratory

indicated that the non-dreaming patients showed an increased frequency of awakenings on average over both nights and on the first night in comparison to the dreaming patients.

More noticeably, the non-dreaming patients displayed an increase of sleep disruptions in comparison to the dreaming patients evident in the number and frequency of arousals. Analysis of the amount of arousals indicated that on average over both nights in the sleep laboratory, the non-dreaming patients had a higher number of arousals than the dreaming cases (269 in comparison to 125.66). Analysis of each night separately indicated that the non-dreaming cases had a substantially higher number of arousals on the second night in comparison to the dreaming cases (492 in comparison to 125), but that the dreaming cases had a higher number of arousals on the first night (126.33 in comparison to 46). The finding that the dreaming cases had a substantially higher number of arousals on the first night can be accounted for by the substantial difference in amount of time spent in sleep on the first night between the non-dreaming cases and the dreaming cases. Therefore, a more valid comparison can be made by looking at the percentage of arousals per hour of sleep time.

Analysis of the percentage of arousals per hour of sleep time indicated that on average over both nights in the sleep laboratory, the non-dreaming cases had a substantially higher percentage of arousals than the dreaming cases (arousal index of 48.45 in comparison to 22.17). Analysis of each night separately indicated that the non-dreaming cases had a substantially higher percentage of arousals on the first night and on the second night in comparison to the dreaming cases, with the greatest difference indicated on the second night (arousal index of 64.15 in comparison to 20.46). Thus, while it is problematic to compare the non-dreaming and dreaming patients on the number of arousals, comparison of the percentage of arousals per hour of sleep time gives clear evidence that the non-dreaming patients displayed an increase of sleep disruptions in comparison to the dreaming patients on both nights in the sleep laboratory.

Similarly, the non-dreaming patients displayed an increase of sleep disruptions in comparison to the dreaming patients evident in the number and frequency of micro-arousals. Analysis of the amount of micro-arousals indicated that on average over both nights in the sleep laboratory, the non-dreaming cases had a substantially higher number of micro-arousals than the dreaming cases (238.25 in comparison to 94.66). Analysis of each night separately indicated that the non-dreaming cases had a substantially higher number of micro-arousals on the second night in comparison to the dreaming cases (444 in comparison to 92.33), but that the dreaming cases had a substantially higher number of micro-arousals arousals on the first night (97 in comparison to 32.5). In the same way as the number of arousals, the finding that

the dreaming cases had a substantially higher number of micro-arousals on the first night can be accounted for by the substantial difference in amount of time spent in sleep on the first night between the non-dreaming cases and the dreaming cases. Therefore, a more valid comparison can be made by looking at the percentage of micro-arousals per hour of sleep time.

Analysis of the percentage of micro-arousals per hour of sleep time indicated that the non-dreaming cases had a higher percentage of micro-arousals on the first night, on the second night, and on average over both nights (micro-arousal index of 39.15 in comparison to 16.76). The most substantial difference in the percentage of micro-arousals between the non-dreamers and the dreamers was evident on the second night (micro-arousal index of 56.95 in comparison to 15.24). Thus, comparison of the percentage of micro-arousals per hour of sleep time gives clear evidence that the non-dreaming patients displayed an increase of sleep disruptions in comparison to the dreaming patients on both nights in the sleep laboratory.

Therefore, these findings give clear evidence that reduced quality of sleep was experienced by the non-dreaming patients in comparison to the dreaming patients. This is particularly important as even if it is undecided what the minimal and maximum amount of sleep interruptions that characterize disturbed sleep are (Boselli *et al*, 1998), we can say that the non-dreaming and dreaming patients certainly differed from each other.

Therefore, while comparison of sleep quantity between the non-dreaming and dreaming patients did not reveal clear evidence for the hypothesis that dreams protect sleep, comparison of sleep quality between the non-dreaming and dreaming patients did give evidence that the non-dreaming patients experienced increased sleep disruptions in comparison to the dreaming patients. This directly supports previous findings that a reduced quality of sleep is experienced by patients who have lost the ability to dream (Bischof & Bassetti, 2004; Poza & Massó, 2006; Solms, 1997). This supports the main hypothesis proposed by this study, namely that dreams protect sleep.

However, these finding also need to be interpreted with caution. Due to relatively small number of cases in this study, tests of statistical significance could not be performed between the non-dreaming and dreaming patients. Thus, while it is clear that the non-dreaming patients did experience a reduced quality of sleep in comparison to the dreaming patients, the statistical significance of these differences has not been established. As such, further research is needed in this area with larger numbers of cases so that significance testing can be performed. Nonetheless, comparison of the mean values of the non-dreaming and dreaming patients that has been done in this study, give a good indication that support for the

hypothesis that dreams protect sleep will be found with a larger number of cases. The following sections of this dissertation will discuss the hypothesis that dreams protect sleep in more detail.

Dreams protect sleep

If we return to the literature on the neural correlates of dreaming, two distinct areas emerged that are involved in the generation of dreams. Firstly, abundant evidence showed that posterior cortical areas, in particular the occipital lobes, are essential for the generation of dreams (Bischof & Bassetti, 2004; Poza & Massó, 2006; Solms, 1997; and Yu, 2006). Secondly, abundant evidence showed that ventromesial frontal white matter is essential for the generation of dreams (Braun *et al*, 1997; Dahan *et al*, 2007; Hartmann *et al*, 1980; Jus *et al*, 1973; Léna *et al*, 2005; Maquet *et al*, 2006; Nofzinger *et al*, 1997; Sandyk *et al*, 1997; Sharf *et al*, 1978; Solms, 1997; and Yu, 2007). In addition, Panksepp (1998) identified the second area, the frontal mesial limbic system, to be the system in the brain that motivates and drives behaviour in waking life.

Based on this dichotomy, and based on the dream model proposed by Solms (2000), i.e. that dreams are generated by volitional urges that arise from the increase of activation in the dopaminergic circuits in the basal forebrain pathways, and that these urges are redirected towards posterior cortices where they are represented virtually as dreams, the findings of this study show clear support that dreams protect sleep.

To explain this further, the two non-dreaming cases in this study (Case 1 and Case 2) both had posterior cerebral artery stroke with corresponding occipital damage that had resulted in subsequent loss of dreaming. If we are to apply Solms's (2000) model of the generation of dreams to these cases, it can be seen that loss of dreaming in these cases arose from damage to the area of the brain where dreams are represented. In addition, as damage from stroke in these two cases did not extend further than the PCA territory, it can be assumed that the area of the brain that drives dream generation (ventromesial frontal white matter) was intact for both patients. Thus, the volitional urges arising from increased activation in the dopaminergic circuits in the basal forebrain pathways could not be redirected to the PCA territory as this area was damaged. Because of this, and because these urges would usually motivate one to actively engage in SEEKING behaviour (Panksepp, 1998), the logical conclusion is that these cases showed disrupted sleep because of a failure to redirect these urges into virtual seeking activity, i.e. dreams.

This is exactly what the polysomnographic recordings of the non-dreaming cases revealed. In comparison to the dreaming cases, the cases who could not dream showed reduced efficiency of sleep, particular with increased frequencies of arousals and micro-arousals. This provides the first empirical support for the hypothesis that dreams protect sleep.

Implications of these findings

While the limitations of this study are obvious, the implications of its findings are significant to the scientific quest to establish the function of dreams in two ways. Firstly, despite recent developments in finding the neural correlates of the dream process, no one has yet been able to definitively link these correlates to a function of dreams. This study begins to fill this gap by providing preliminary evidence that supports the hypothesis that dreams protect sleep. By doing so, this study contributes to the exploration on the question of why people dream that has been pondered for centuries.

Secondly, as the hypothesis that dreams protect sleep was originally proposed by Freud (1900), this study has provided tentative support for the Freudian dream theory. As Freud's dream theory is the foundation upon which the whole of psychoanalysis is based, finding an empirical support for his hypothesis adds greater scientific credibility to the field of psychoanalysis. This is significant as Freudian dream theory was largely disregarded as a credible scientific theory after the discovery of REM sleep (Aserinsky & Kleitman, 1953) and the research that followed this discovery. In addition, there are those who would argue that psychoanalytic theories are not amenable to scientific investigation at all. In *Conjectures and Refutations* (1963), Karl Popper famously considered psychoanalysis a "pseudo-science" (pg. 33). His argument, which has become the benchmark of scientific investigation, is that theories are only testable in so far as they are falsifiable. Psychoanalytic theories, he argued, did not fulfil this criterion as "there was no conceivable human behaviour which could contradict them" (pg. 37).

This study has shown that Popper was perhaps overly pessimistic about the prospect of testing psychoanalytic theory. As the hypothesis that dreams protect sleep, the hypothesis tested in this study, is a foundational hypothesis proposed by Freud, this study provides a model whereby a fundamental psychoanalytic theory has been testing according to the principles of falsification that Popper advocated. While further research is still needed in this area, and these results can only be viewed as preliminary findings, this study does show that scientific investigation is feasible to test the Freudian hypothesis that dreams function as a

"guardian of sleep" (Freud, 1990; pg. 580). In light of this fact alone, it is not entirely reasonable to describe psychoanalysis as a 'psuedo-science'.

Conclusion

The hypothesis that dreams protect sleep was investigated in this study using medical records, structural neuro-imaging, clinical interviews, neuropsychological testing, analysis of subjective sleep quality, and polysomnographic data in five patients with lesions in the PCA territory. Individual case analyses revealed that sleep was disrupted in the non-dreaming cases. In addition, comparisons were performed between the dreaming and non-dreaming patients. These comparisons revealed a reduced efficiency of sleep in non-dreaming patients compared to dreaming patients, particularly by demonstrating a greatly increased frequency of arousals and micro-arousals in patients with loss of dreaming. While methodological limitations need to be considered, this study provides preliminary support for the hypothesis that dreams protect sleep. In doing so, this study contributes to the scientific search for a function of dreams, as well as providing preliminary support for the Freudian dream theory. Hopefully these findings will lead to further scientific research in the field of dream science. By doing so, it may be possible in the near future to find a firm empirical basis for the function of dreams.

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 concurrent phenomena during sleep. *Science, 118,* 273–274.

Babcock, H., & Levy, L. (1930). An experiment in the measurement of mental deterioration. *Archives of Psychology, 117,* 105-107.

Bakken, L. N., Lee, K .A., Kim, H .S., Finset, A., & Lerdal, A. (2011). Sleep-wake patterns during the acute phase after first-ever stroke. *Stroke Research and Treatment, 2011,* 7 pages.

Bassetti, C. L., & Aldrich, M. S. (2001). Sleep electroencephalogram changes in acute hemispheric stroke. *Sleep Medicine, 2,* 185-194.

Bassetti, C. L., Milanova, M., & Gugger, M. (2006). Sleep disordered breathing and acute ischemic stroke: Diagnosis, risk factors, treatment evolution, and long-term clinical outcome. *Stroke, 37,* 967-972.

Benbir, G., & Guilleminault, C. (2007). Obstructive and nonobstructive sleep apnea: The neurobiological perspective. In A. Culebras (Ed.), *Sleep disorders and neurologic disease* (2nd ed) (pp. 227-300). New York, NY: Informa.

Bischof, M., & Bassetti, C. L. (2004). Total dream loss: A distinct neuropsychological dysfunction after bilateral PCA stroke. *Annals of Neurology, 56,* 583-586.

Bosselli, M., Parrino, L., Smerieri, A., & Terzano, M. G. (1998). Effect of age on EEG arousals in normal sleep. *Sleep, 21,* 361-367.

Braun, A. R., Balkin, T. J., Wesensten, N. J., Carson, R. E., Varga, M., Baldwin, P., *et al.* (1997). Regional cerebral blood flow throughout the sleep-wake cycle: An $H_2{}^{15}0$ PET study. *Brain, 120,* 1173-1197.

Breger, L. (1967). Function of dreams. *Journal of Abnormal Psychology, 72,* 1-28.

Buysse, D. J., Reynolds III, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatric Research, 28,* 193-213.

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. (2010). Overview of sleep and sleep disorders. *Indian Journal of Medical*

Research, 131, 126-140.

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.

Crick, F., & Mitchison, G. (1983). The function of dream sleep. *Nature, 304,* 111-114.

Christensen, A. L. (1974). *Luria's neuropsychological investigation.* Copenhagen, DK: Munksgaard.

Dahan, L., Astier, B., Vautrelle, N., Urbain, N., Koscis, B., & Chouvet, G. (2007). Prominent burst firing of dopaminergic neurons in the ventral tegmental area during paradoxical sleep. *Neuropsychopharmacology, 32,* 1232-1241.

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.

Doricchi, F., & Violani, C. (1992). Dream recall in brain damaged patients: A contribution to the neuropsychology of dreaming through a review of the literature. In J. Antrobus & M. Bertini (Eds), *The neuropsychology of sleep and dreaming* (pp. 99-140). Hillsdale, NJ: Lawrence Erlbaum Associates.

Dumont, M., Braun, C. M. J, & Guimond, A. (2007). Dreaming and unilateral brain lesions: A multiple lesion case analysis. *Dreaming, 17,* 20-34.

Epstein, A. W. (1979). Effect of certain cerebral hemispheric diseases on dreaming. *Biological Psychiatry, 14,* 77-93.

Freud, S. (1961).The interpretation of dreams. In J. Strachey (Ed. and Trans.), *Standard edition of the complete psychological works of Sigmund Freud* (Vols. 4–5). London: Hogarth Press. (Original work published 1900)

Freud, S. (1961). A metapsychological supplement to the theory of dreams. In J. Strachey (Ed. and Trans.), *Standard edition of the complete psychological works of Sigmund Freud* (Vol. 14). London: Hogarth Press. (Original work published 1917)

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.

Giubilei, F., Iannilli, M., Vitale, A., Pierallini, A., Sacchetti, M. L., Antonini, G., & Fiescki, B. (1992). Sleep patterns in acute hemispheric stroke. *Acta Neurologica Scandinavica, 86,* 567-571.

Gottesmann, C. (2004). Brain inhibitory mechanisms involved in basic and higher integrated sleep processes. *Brain Research Reviews, 45,* 230-249.

Gottselig, J. M., Bassetti, C. L., & Achermann, P. (2002). Power and coherence of sleep spindle frequency activity following hemispheric stroke. *Brain, 125,* 373-383.

Hartmann, E., Russ, D., Oldfield, M., Falke, R., & Skoff, B. (1980). Dream content: Effects of L-DOPA. *Sleep Research, 9,* 153.

Hermann, D. M., Siccoli, M., & Bassetti, C. L. (2003). Sleep-wake disorders and stroke. *Schweizer Archiv für Neurologie und Psychiatrie, 154,* 369-373.

Hirshkowitz, M., & Sharafakhaneh, 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., & McCarley, R. W. (1977). The brain as a dream state generator: An activation-synthesis hypothesis of the dream process. *The 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.

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.

Léna, I., Parrot, S., Deschaux, O., Muffat-Joly, S., Sauvinet, B., Renaud, M. –F., *et al.* (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.

Manni, R. (2005). Rapid eye movement sleep, non-rapid eye movement sleep, dreams, and hallucinations. *Current Psychiatric Reports, 7,* 196-200.

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,* 163-166.

McNamara, S. G., Grunstein, R. R., & Sullivan, C. E. (1993). Obstructive sleep apnoea. *Thorax, 48,* 754-764.

Mosdell, J., Balchin, R., & Ameen, O. (2010). Adaptation of aphasic tests for neurocognitive screening in South Africa. *South African Journal of Psychology, 40,* 250-261.

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.

Osterrieth, P. A. (1944). Le test 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.* New York: Oxford University Press.

Popper, K. R. (1963). *Conjectures and Refutations: The Growth of Scientific Knowledge.* London: Routledge & Kegan Paul Limited.

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 tempero-occipital brain injury. *Neurologia, 21,* 152-154.

Redline, S., Kirchner, H. L., Quan, S. F., Gottlieb, D. J., Kapur, V., & Newman, A. (2004). The effects of age, sex, ethnicity, and sleep-disordered breathing on sleep architecture. *Archives of Internal Medicine, 164,* 406-418.

Revonsuo, A. (2000). The reinterpretation of dreams: An evolutionary hypothesis of the function of dreaming. *Behavioural and Brain Sciences, 23,* 793-1121

Rey, A. (1941). L'examen psychologique dan les cas d'encephalophaie. *Archives de Psychologie, 28,* 286-340.

Ruby, P. M. (2011). Experimental research on dreaming: State of the art and neuropsychoanalytic perspectives. *Frontiers in Psychology, 2,* 1-10.

Sandyk, R. (1997). Treatment with weak electromagnetic fields restores dream recall in a parkinsonian patient. *Internationsal Journal of Neuroscience, 90,* 75-86.

Sivan, A. B. (1992). *Benton Visual Retention Test* (5th ed.). San Antonio, Tex.: The Psychological Corporation.

Solms, M. (2000). Dreaming and REM sleep are controlled by different brain mechanisms. *Behavioral and Brain Sciences, 23,* 793-1121.

Solms, M. (1997). *The neuropsychology of dreams*: A clinic-anatomical study. Mahwah, NJ: Lawrence Erlbaum.

Solms, M., Kaplan-Solms, K., & Brown, (1996). Wilbrand's case of "mind-blindness". In Code, C., Wallesch, C-W., Joanette, Y., & Lecours, A. R. (1996). Erlhaum, UK: Psychology Press.

Solms, M., & Malcolm-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.

Sharf, B., Moskovitz, C., Lupton, M. D. & Klawans, H. L. (1978) Dream phenomena induced by chronic levodopa therapy. *Journal of Neural Transmission, 43,* 143–51.

Vock, J., Achermann, P., Bischof, M., Milanova, M., Müller, C., nirkko, A., Roth, C., & Bassetti, C. L. (2002). Evolution of sleep and sleep EEG after hemispheric stroke. *Journal of Sleep Research, 11,* 331-338.

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.

Yu, C. K. (2006). Memory loss is not equal to loss of dream experience: A clinicoanatomical study of dreaming in patients with posterior brain lesions. *Neuropsychoanalysis, 8,* 191-198.

Appendix A: Standard Definitions for Sleep Macrostructure 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 totals 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.

Appendix B: Informed Consent Form

Informed Consent Form

Title of research study: Do dreams protect sleep?
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 from the Department of Psychology at the University of Cape Town in order to see whether your stroke has had an effect on your dreams. Your participation is completely voluntary.

Participant's involvement:
What's involved: Your medical folders will be examined and their information may be used. However, complete confidentiality will be maintained and your name will not be used.

The study will involve two consecutive nights in a sleep laboratory. You will be connected to a polysomnograph which is a simple device that involves small pads being placed on different parts of your body. You will be asked to sleep as you would normally at home. During the first night, you will be awakened twice by the researcher and asked whether you were dreaming. During both nights, your sleep cycles will be recorded.

Risks: There are no risks associated with this study, except the minor risk of falling out of bed. However, if you feel uncomfortable at any time you may withdraw from the study without any negative consequences for yourself. 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 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.

Name of Participant: _____

Signature of Participant: _____

Date: _____

Name of principal researcher: _____

Signature of principal researcher: _____

Date: _____