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Dreaming and the dorsolateral frontal lobes: Towards a better understanding of the mechanism of dreaming.

By

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Declaration

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

Signature: Signature removed Date: 9/02/2006

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Abstract

The exact mechanism of dream production is still poorly understood. Based on exploratory findings that damage to the dorsolateral prefrontal cortex does not cause changes in these patients subjective experience of their dreams (Solms, 1997), a study was conducted in order to investigate the role of this area in dream production. The dreams of seven patients with damage to the dorsolateral prefrontal cortex were compared with those of normal participants. A content analysis found no significant quantitative differences between the dreams of dorsolateral prefrontal patients and normal controls. In addition, none of the patients with damage to the dorsolateral prefrontal cortex reported any subjective changes in their dreams since falling ill. These findings are congruent with those of numerous neuroimaging studies, which indicate that the dorsolateral prefrontal cortex is deactivated during dreaming, and provide support for the theory that deactivation of the dorsolateral prefrontal cortex during sleep accounts for many of the formal features of dreams.

Keywords: Dorsolateral prefrontal cortex; mechanism of dreams; formal features of dreams.

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1. Introduction

Despite the fact that dreaming forms an integral part of the mental life of almost all humans, we still know very little about how the brain generates dreams and why they are generated. Given that dreams are entirely subjective phenomena and that research into changes in dreaming following brain injury is therefore plagued by methodological problems, the paucity of our present knowledge is perhaps not so surprising. Based on claims that damage to the dorsolateral frontal region of the brain does not cause any changes in these patients' subjective experience of their dreams (Solms, 1997), a study was done in order to investigate whether this area of the brain plays a role in the mechanism of dreaming. Thus the hypothesis investigated was that damage to the dorsolateral frontal lobes causes no significant changes in normal dream production. This was achieved by comparing the dreams of patients with damage to the dorsolateral frontal lobes with those of normal participants. The rationale of the study was that it is theoretically as important to establish which anatomical structures and neuropsychological functions are inessential to dreaming as it is to establish which structures and functions are essential. Thus the study aimed to provide empirical evidence to support key theoretical models of the dream process and thereby to contribute to the small, but fertile field of dream research.

1.1. Defining dreaming

Dreaming can be defined as "imagery that consists of sensory hallucinations, emotions, story-like or dramatic progressions, and bizarreness" (Nielsen, 2000, p. 853) experienced during sleep. However, as Nielsen (2000) points out, there is no standardized definition of dreaming, and definitions often vary from study to study. Hobson (1988) has identified five major characteristic features of dreams. These are: often strongly felt emotions, illogical content and organization, sensory impressions, an uncritical acceptance of events depicted in the dream, however bizarre, and difficulty in remembering the dream upon awakening. Attempts have been made to differentiate between ordinary dreams, more elaborate, vivid and intense forms of dreams (such as nightmares, for example) and other forms of cognitive activity, such as thinking, or the experience of vague and fragmentary impressions, during sleep (Nielsen, 2000). However, such attempts are often hampered by ill-developed coding systems that frequently lack reliability and validity (Domhoff, 1996).

At this point it is also important to note that the word "dream" can have various meanings. Thus it can refer to a subjective experience during sleep, the memory of that subjective experience upon awakening, or that which is reported to others (Domhoff, 1996). Since a dream only attains objective existence once it is reported, the present study will only deal with dreams in this sense, what Domhoff (1996) calls the "dream report" or "dream narrative." This reliance on the dream report as the only means of accessing the dream experience is probably the foremost methodological problem in dream research. Given that dreaming takes place during sleep, and active dream recall, as well as the recording of the dream, only upon awakening, the continuity between dream experiences and the beliefs about them cannot be taken for granted (Beaulieu-Prévost & Zandra, 2005a; Hobson, Pace-Schott, & Stickgold, 2000). Thus recent studies have, for example, shown that factors such as memory, personality and individual biases can influence self-reported evaluations of dream recall frequency (Beaulieu-Prévost & Zandra, 2005b), and that individual differences in dream recall frequency can influence people's beliefs regarding the content of their dreams (Beaulieu-Prévost & Zandra, 2005a). These kinds of methodological problems and their impact on the current study are discussed in more detail later. For now, we turn our attention to some of the most prominent theoretical models of the dream process.

1.2. Dreams as a function of REM sleep?

When it was discovered that humans spend about 25% of their sleeping hours in a state characterised by paradoxical cerebral activation and rapid eye movement (REM) (Asterinsky & Kleitman, 1953), it became possible to measure the physiological parameters of REM sleep and its correlation with dream reports. Findings indicating that normal subjects report dream experiences in 70 –95% of awakenings from REM sleep (Dement & Kleitman, 1957), led to the belief that REM sleep is the physiological equivalent of dreaming (Hobson & McCarley, 1977; Solms, 2000). This prompted a radical departure from Freud's psychoanalytic dream theory, which saw dreams as the product of a defensive transformation of unconscious wishes found unacceptable by consciousness (Freud, 1900/1976), and led to the development of one of the most influential theoretical models of dreaming namely; the activation-synthesis model. Developed by Hobson and colleagues, this model describe dreams as the product of the integration by forebrain mechanisms of disparate and chaotic

internal data, present due to random activation of the brain during REM sleep (Hobson & McCarley, 1977; Hobson, 1988). Hobson's model is based on the pioneering work of Jouvet (1962) whose ablation, stimulation and recording studies showed that REM sleep is controlled by pontine brain stem mechanisms. This became the central tenet of the activation-synthesis model, leading Hobson and McCarley (1977) to believe that the forebrain played an entirely passive role in the dream process and that all causal stimuli that create dream imagery arise "from the pontine brain stem and not in cognitive areas of the cerebrum" (p. 1347).

However, as Solms (2000) points out, these propositions can be questioned on the basis of several contradictory findings, the first of which is the fact that not all dreaming is correlated with REM sleep. Thus studies indicate that complex mentation can be elicited in between 43% and 50% of non-REM (NREM) awakenings (Foulkes, 1962; Nielsen, 1999). Although Hobson and colleagues do not dispute these results, they do query the extent to which the reported mentation can be called "dreams", since there seems to be qualitative differences between NREM and REM dreams (Hobson et al., 2000), with NREM dreams being more "thoughtlike" and less bizarre than REM dreams (cf. Nielsen, 2000). Despite this objection, Hobson's revised activation-synthesis model, the Activation-Input-Mode [AIM] model does take into account the fact that at least some NREM dreams are indistinguishable from REM dreams (Hobson, 1988; Rechtschaffen, 1973). And although the claim that all dreams are generated by the same brain stem mechanisms that produce REM sleep has been abandoned, the claim that pontine brain stem mechanisms generate all dreams has been retained (Hobson, 1992; Solms, 2000). The new model therefore conceptualises both REM and NREM dreams as being "a function of the physiological condition of the reciprocally interacting brain stem neuronal populations that constitute the sleep-cycle control oscillator" (Hobson, 1992, p. 228).

This shift in the theory implies, according to Solms (2000) that it should be possible to demonstrate that dreaming is eliminated due to lesions to the brain stem. This method is however problematic since lesions that are large enough to eliminate both REM and NREM usually destroy consciousness altogether (Hobson et al., 2000; Solms, 2000). This has led Solms (2000) to investigate the corollary hypothesis of the AIM model; that dreaming is not controlled by forebrain mechanisms. On the basis of his own findings, as well as an extensive review of the literature, Solms (2000) reports that of the 111 cases of focal lesions causing cessation or near cessation of

dreaming, "the lesion was localized to the forebrain – and the pontine brain stem was completely spared – in all but one case" (p. 846). Anoneria (loss of dreaming) was found to be specifically associated with lesions to the posterior convexity of the hemispheres, in or near the parieto-temporo-occipital junction - bilaterally as well as unilaterally - and to the white matter surrounding the frontal horns of the lateral ventricles bilaterally (Solms, 1997; 2000). Moreover, the REM state was found to be completely preserved in those cases in which the sleep cycle was evaluated (Solms, 2000). Solms (2000) not only contends that dreaming can be terminated by forebrain lesions which spare the REM cycle, but also that forebrain mechanisms can initiate dreams in the absence of REM sleep. As evidence for this he takes the phenomenon of nocturnal seizures, which usually take place in NREM sleep (Kellaway & Frost, 1983). A review of the literature (cf. Solms, 2000) suggests that the foci or lesion in the case of these kinds of seizures is almost always located in the temporal lobe (i.e. in the forebrain) and unsurprisingly, given the well-established connection between fear and temporal lobe seizures (Heilman, Bowers and Valenstein, 1993), often take the form of recurring nightmares. Additional findings by Penfield and colleagues (Penfield, 1938; Penfield & Rasmussen, 1955), which showed that stimulation of the temporal lobe focus could artificially reproduce these anxious experiences, strengthens Solms' (2000) argument. This led Solms (2000) to conclude that REM sleep and dreaming are most probably controlled by different brain mechanisms and are therefore doubly dissociable states. This theory therefore holds that any brain state that involves cerebral activation during sleep will simultaneously generate dreams (Solms, 2000). This position is not far removed from that of Hobson and colleagues, whose AIM model suggests that conscious states, including the experience of dreaming "show a clear-cut dependence on brain activation level" (Hobson et al., 2000, p. 40). However, whereas Solms (2000) holds that dreaming will only occur if and when the general activation of the brain activates specific dopaminergic circuits situated in the ventromesial forebrain, Hobson's AIM model maintains that the dreaming state is the direct consequence of a shift in modulatory balance from aminergic (i.e. serotonin and noreprinephrine) to cholinergic (i.e. acetylcholine) during REM sleep (Hobson et al., 2000).

Despite the debate as to whether dreams are exclusively initiated by pontine brain stem mechanisms or not, a coherent picture is starting to evolve regarding the forebrain structures involved in the dream process. The evidence for the involvement

of these structures and their implications for our understanding of the mechanism of dreaming are briefly discussed below.

1.3. The Neuroanatomy of dreaming

Most of our knowledge regarding the forebrain structures involved in the mechanism of dreaming is derived from clinico-anatomical studies (Solms, 1997) and functional neuroimaging studies (Braun et al., 1997, 1998; Franck et al., 1987; Heiss et al., 1985; Madsen et al., 1991; Maquet et al., 1996; Nofzinger et al., 1997). The remarkably consistent picture derived from these two methods (Hobson et al., 2000), has done a great deal to inform our understanding of the mechanism of dreaming. However, before discussing these results it is important to note some of the methodological limitations of both these methods. Thus clinico-anatomical studies are relatively blunt instruments when it comes to revealing mechanistic and functional details, given that they "cannot discriminate between the effects of the destruction and the disconnection and cannot target specific neuronal groups in heterogeneous regions" (Hobson et al., 2000, p. 15). Neuroimaging studies, such as those measuring cerebral blood flow, suffer from the same constraint because they measure global activation and cannot identify small but influential neuronal populations (Hobson et al, 2000). In addition, the interpretation of neuroimaging studies, such as positron emission tomography (PET), is problematic because it is impossible to determine whether increased activity is inhibitory or excitatory (Hobson et al., 2000; Solms, 1997). Other methodological constraints mentioned by Hobson et al. (2000) include small sample sizes used in some neuroimaging studies, the possibility that the functional activity of a brain area may vary with changes in its inputs, and the possibility that normal functional disconnections that occur during REM sleep may result in the same neural structures performing differing tasks depending on the state the brain is in. Despite these limitations, the findings of neuroimaging studies have been consistently replicated by several independent groups and are bolstered in their validity by the complementary findings of lesion studies (Hobson et al., 2000).

1.3.1. The effects of brain damage on dreaming

Although brain-damaged patients often exhibit preserved dreaming, their dreams are often highly abnormal (Solms, 1997). In his exploratory study of the clinico-anatomical correlates of dreaming Solms (1997) divided abnormalities in dreaming

following brain injury into two broad categories: (a) *deficits* in dreaming or dream imagery, which include cessation or restriction of visual dream-imagery and global cessation or reduction of dreaming and (b) *excesses* of dreaming or dream-imagery, which includes increased frequency and vivacity of dreaming and recurring nightmares. His research has shown that these abnormalities all involve different brain structures and varieties of neuropsychological dysfunction.

As mentioned above, "global anoneria", or total cessation of dreaming was found to be associated with either posterior cortical or deep bilateral frontal lesions. The anterior variant of global anoneria is hypothesised to be caused by a disconnection of the mediobasal frontal cortex from the brain stem and diencephalic limbic areas (Hobson et al., 2000) and is associated with adynamia and lack of volition (Solms, 1997). This implies, according to Solms (2000) that "motivational mechanisms are essential for the generation of dreams" (p. 848). In corroboration of this, a neuroimaging study reported increased activation of the caudal orbital frontal area during REM sleep in comparison to waking and NREM sleep (Braun et al, 1997). In the posterior variant, global anoneria is seen as the result of lesions in the parieto-temporo-occipital junction of either hemisphere. Right-sided lesions were found to be associated with disorders of spatial cognition, whilst left sided lesions were associated with disorders of quasi-spatial (symbolic) operations (Solms, 1997). This suggests that both symbolic quasi-spatial functions and concrete spatial functions are fundamental to the process of dreaming (Solms, 1997; 2000). This finding is supported by evidence from a neuroimaging study that showed activation of the right inferior parietal cortex during REM sleep (Maquet et al, 1996).

Solms (1997) makes a distinction between global anoneria and a second syndrome called "visual anoneria" or non-visual dreaming. This disorder, first systematically formulated by Doricchi and Violani (1992), is characterized by a full or partial loss of visual dream imagery and is associated with the inability to produce mental imagery in waking life. This syndrome is associated with bilateral lesions to medial-occipito-temporal cortex (especially areas V3, V3a, V4, but not V1, V5, or V6) (Solms, 1997; 2000). Similarly, Braun and colleagues have found activation of the extrastriate visual cortex, but not the striate visual cortex, during REM sleep (Braun et al, 1997; 1998)

In terms of excesses of dreaming and dream imagery, Solms (1997) noted that lesions to the rostral limbic system (i.e. in the anterior thalamus, basal forebrain,

anterior cingulate, and mesial frontal cortex) cause excessively vivid and frequent dreaming. This symptom-complex is associated with a breakdown in of the distinction between dreaming and waking cognition as well as other reality-monitoring deficits. According to Solms (2000) inhibition of these structures during sleep may account for the hallucinated, delusional, disorientated, and paramnestic quality of dream cognition. Neuroimaging studies indicate that the thalamus, basal forebrain, medial prefrontal and anterior cingulate areas are all selectively activated during REM sleep, but deactivated during NREM sleep (Braun et al., 1997; Maquet et al., 1996; Nofzinger et al, 1996). Given that REM dreams are generally more vivid than NREM dreams (Hobson, 1988), one would expect the opposite to be the case, if Solms' (2000) hypothesis was correct. However, although this indicates a discrepancy between the clinico-anatomical and neuroimaging findings, Hobson et al. (2000) cautions that lesions in these areas could just as well be irritative as destructive and that lesions to different areas of this functionally highly heterogeneous region could cause dramatically different effects.

The second symptom complex identified by Solms (1997), recurring nightmares, is (as discussed above) associated with discharging lesions in the medial and anterior temporal cortex and is associated with unpleasant hallucinatory experiences during waking (Solms, 1997; 2000).

These findings provide overwhelming evidence that specific forebrain structures are involved in generation of dream imagery and thus provide a great deal of support for Solms' (1997) theory. Neuroimaging findings that indicate activation of limbic and paralimbic regions, specifically the anterior and lateral hypothalamic areas, the amygdaloid complex and septal-ventral striatal areas during REM sleep (Braun et al, 1997; Maquet et al, 1996; Nofzinger et al., 1997) has also led to a revision of Hobson's original theory (Hobson & McCarley, 1977). Thus he now admits that there is a greater degree of forebrain control of both REM sleep and the dream process than he previously thought (Hobson et al., 2000).

However it is theoretically as important to establish which anatomical structures and neuropsychological functions are inessential to dreaming as it is to establish which structures and functions are essential (Solms, 1997). Thus studies have found that the heteromodal association areas (specifically the dorsolateral prefrontal and inferior parietal cortices) are deactivated during both REM and NREM sleep (Braun et al., 1997; Maquet et al., 1996). We now turn our attention to one of the areas of

the brain hypothesised to have no input in the dream process, namely the area of the dorsolateral prefrontal lobes (Solms, 1997).

1.3.2. Brain damage and unchanged dreaming

Solms' (1997) research found that normal (i.e. unchanged) dreaming correlated significantly with lesions to the dorsolateral frontal lobes. In addition, a lateralizing tendency was found, with normal dreaming being significantly higher in cases of lesions to the left hemisphere than to the right hemisphere. This led him to speculate that "the dorsolateral frontal region (and perhaps the left dorsolateral prefrontal region in particular) plays no essential role in the normal dream process" (Solms, 1997, p. 222). In corroboration of this, several $H_2{}^{15}O$ PET studies have found significant deactivation of the dorsolateral fontal cortex during both REM (Braun et al., 1997; Maquet et al., 1997) and NREM sleep (Anderson et al., 1998; Braun et al., 1997). In addition, a single photon emission computed tomography (SPECT) study conducted by Madsen and colleagues also found decreased blood flow to frontal areas during REM sleep (Madsen et al., 1991). However, these finding are contradicted by a fluorodeoxyglucose (FDG) PET study conducted by Heiss and colleagues who compared cerebral glucose uptake during dreaming versus dreamless sleep and found increased, rather than decreased, superior frontal activity during dreams (Heiss et al., 1984). A more recent FDG PET study conducted by Nofzinger and colleagues also did not find a significant decrease of glucose uptake in the dorsolateral convexity when comparing REM sleep and waking (Nofzinger et al., 1997). Methodological differences between FDG and $H_2{}^{15}O$ methods may account for this discrepancy (see Braun et al., 1997, Hobson et al., 2000 and Nofzinger et al, 1997 for discussions).

On balance, it therefore seems that the evidence points towards the deactivation of the frontal convexity during dreaming (the implication of which will be discussed below). However, it is important to note that the only lesion study in this area had a severe methodological constraint. Solms' (1997) research relied upon a very subjective method in which patients were asked whether or not their dreams have changed subsequent to neurological damage, and if so, in which ways. This method is problematic, since, as mentioned above, people's beliefs about the content of their dreams are influenced by a variety of factors (Beaulieu-Prévost & Zandra, 2005a). For example, individual differences in dream recall frequency (DRF) have been found to influence people's beliefs regarding the amount of anxiety present in their dreams.

Thus people's beliefs about the amount of anxiety present in their dreams were only related to the actual affective content of their dreams in individuals with high DRF. Conversely, in individuals with low DRF, beliefs regarding the amount of anxiety present in their dreams were related only to their current affective state (Beaulieu-Prévost & Zandra, 2005a). It may therefore be that factors such as DRF may influence people's beliefs around other aspects of their dreams, such as the vivacity of visual dream imagery, narrative complexity, or emotional intensity. The present study aimed to address this methodological problem by using more objective measures. As such, it employed the use of blind raters in order to compare the dream reports of patients with damage to the frontal convexity with those of normal participants. If the frontal convexity plays no role in the normal dream process, a content analysis should reveal no quantitative differences between the dream reports of patients with damage to that area and normal participants.

2. Theoretical framework

2.1. Dreams and the dorsolateral prefrontal cortex

In his model of the normal dream process, Solms (1997) hypothesised that inhibition of the dorsolateral frontal convexity, together with the inhibition of spinal motoneurons, during sleep serve the purpose of preventing appetitive interests, aroused due to external sensory stimulation as well as endogenous stimulation, such as REM activation, from expressing themselves in volitional motor action. As discussed above, evidence for this line of reasoning comes from his own research, which indicate that patients with damage to the dorsolateral frontal convexity (and perhaps the left dorsolateral frontal convexity in particular) report no changes in their dreams subsequent to neurological illness (Solms, 1997), and is supported by the findings of several neuroimaging studies (Anderson et al., 1998; Braun et al., 1997; Madsen et al., 1991; Maquet et al., 1997). However, it is important to note that the deactivation of the dorsolateral prefrontal cortex during dreaming is only relative, and that lesions to this area may therefore further lower the activation, thus affecting the dream content of these patients. In addition, Solms' (1997) hypothesis of a left lateralizing tendency has not been confirmed as yet. In fact, his tentative finding has been contradicted by one neuroimaging study that compared decreases in cerebral blood flow during REM sleep in the left and right frontal convexity and found no

hemispheric differences (Braun et al., 1998). Solms' model is also contradicted by Foulkes' (1978) hypothesis that the left frontal areas of the brain contribute to the narrative element of dreams. According to this view, which has since been abandoned (Foulkes, 1996), dream narratives are mediated by the same left frontal areas, which, in the production of waking narratives, provide expressive speech with an intention or plan, and aid the subsequent recoding of that plan into speech (Foulkes, 1978). The left frontal lobe is therefore seen as imbuing speech with its predicative structure and logical coherence (Luria, 1973). However, given the erratic narrative structure of most dreams, it would perhaps not be so surprising if the prefrontal cortex - the part of the brain that is most involved in creating narrative structure - is inessential to the dream process (Solms, 1997). Thus Solms (1997) proposes that the bizarre nature of most dreams can be ascribed to dorsolateral prefrontal deactivation.

Dreams are characterized by cognitive deficits in self-reflective awareness, orientation, and memory (Hobson, 1988; Hobson et al., 2000). These formal characteristics of dreams have all been ascribed to the deactivation of the frontal convexity during sleep (Braun et al., 1997; Maquet et al., 1996). Given this, we now turn our attention to a closer examination of the functions of the dorsolateral frontal lobes.

2.2. Behaviour and the frontal lobes

The exact function of the prefrontal cortex has baffled generations of researchers, leading Teuber (1964) to describe it as "a riddle". However, a large body of research (comprising both animal and human studies) has provided us with a much clearer understanding of the diverse anatomical units that make up the prefrontal cortex, their connections with other areas of the brain, as well as their functions (Damasio & Anderson, 1993; Fuster, 1989; Passingham, 1993). Thus, research has shown that damage to the prefrontal cortex causes specific neuropsychological impairments depending on the site and size of the lesion, and that there is no single "frontal lobe syndrome" as such (Damasio & Anderson, 1993; Fuster, 1989). The prefrontal cortex is usually divided into three cortical surfaces: dorsolateral, orbital, and medial, and there is some evidence of differential involvement of each of these structures in some cognitive functions (Luria, 1966/1980; Stuss & Benson, 1986). However, interpretation of these studies should be approached with caution, as the terms used to

define these areas are not always consistent and the use of a specific anatomical label does not always exclude pathology involving other areas (Stuss & Benson, 1986). In addition, results seem to vary depending on the definition of what is being measured (Stuss & Benson, 1986). Despite this, a new generation of researchers, making use of modern neuroimaging technologies, are elucidating the qualitative and quantitative differences between frontal subgroups in terms of their performance on different tasks (see for e.g. Reverberi et al., 2005a; Reverberi et al., 2005b; Simons et al., 2005), and thereby confirming Luria's (1966/1980) observation that "different parts of the frontal lobes subserve different functions" (p. 360).

Damage to the dorsolateral prefrontal cortex in humans is associated with specific disorders of attention, motility, and temporal integration (Fuster, 1989). In terms of disorders of attention, Luria (1966/1980) found that patients with damage to large portions of the dorsolateral frontal lobes showed a lowering of general awareness as illustrated by little or no interest in the environment and a general attitude of apathy. A lack of drive, called *Antriebsschwäche* by Klages (1954 cited in Fuster, 1989) seems to be the underlying deficit in these cases. This lowering of general awareness also extends to self-awareness, which is expressed by shallowness of interest, loss of self-concern and impairment in self-monitoring or the ability to self-correct (Stuss & Benson, 1986). In the case of disorders of motility, dorsolateral frontal patients may suffer from *hypokinesis*, a disorder characterized by a general decline of spontaneous motor activity, which may range from "aspontaneity" that mainly affects language and social behaviour (Fuster, 1989) to akinetic-abulic syndrome with mutism (Luria, 1966/1980). Fuster (1989) distinguishes between disorders of general spontaneous motility and disorders of goal-directed behaviour. He classifies the later as a disorder of temporal integration, associated with concreteness and a constriction of the scope and complexity of behaviour, leading to stereotyped sequences of behaviour with little regard for either the origins of the sequence or its goals (Fuster, 1989). Interestingly, Fuster (1989) also classifies defective memory (due to attentional deficits), and defective planning associated with dorsolateral prefrontal damage as disorders of temporal integration. According to his theoretical model, the cardinal function of the prefrontal cortex is the temporal organization of behaviour. Thus the functions of the prefrontal cortex are seen as interrelated and mutually supportive of each other in guiding purposive behaviour (Fuster, 1989).

According to Luria, the impairment of purposeful behaviour associated with damage to the dorsolateral frontal lobes can be seen as the product of the disruption of the regulatory role of language on behaviour (Luria & Homskaya, 1964; Luria, 1966/1980). This view therefore sees both voluntary and involuntary motor activity are preceded and guided by internalised linguistic schemata. Disruption of such schemata cause what seems to be a dissociation between knowing and doing, with patients being unable to implement their volitional intensions, despite being able to move normally (Luria & Homskaya, 1964; Luria, 1966/1980).

Both Luria's (1966/1988) and Fuster's (1989) theoretical models of the functions of the prefrontal cortex congruent with Solms' (1997) hypothesis that inhibition of the dorsolateral prefrontal convexity during dreaming will obstruct the expression of ideas into action. Thus the function of dorsolateral prefrontal deactivation during dreaming is most probably the preservation of sleep (Solms, 1997). If Solms' hypothesis is correct, lesions to this area should not disrupt the production of dreams in any way, and the dreams of these patients should therefore be indistinguishable from those of normal dreamers.

3. Research Method

3.1. Aims

The aim of the present study was to confirm Solms' (1997) observation that that damage to the dorsolateral prefrontal cortex seem to cause no significant changes in these patients' ability to produce dreams. In addition, we aimed to overcome some of the methodological problems associated with his study by making use of more objective measures.

3.2. Design

The present study made use of a natural experimental design, with dorsolateral frontal patients as the experimental group and non-brain damaged patients as the control group. Most recent dream reports were obtained from all participants and, after transcription, these were subject to a content analysis. The dreams of dorsolateral prefrontal patients were then compared with those of normal participants in order to ascertain whether there are any quantitative differences between the dreams of these two groups. Since deactivation of the dorsolateral prefrontal cortex

during sleep has been hypothesised to cause some of the striking cognitive deficits present in dreams (Braun et al., 1997; Hobson et al., 2000; Maquet et al, 1996; Solms, 2000), specific aspects of executive function and memory were evaluated in order to provide a clinical picture which would allow us to explain any differences, or lack thereof, between the dreams of dorsolateral prefrontal patients and those of control participants.

3.3. Sample

Twenty-five patients with focal frontal lobe damage as identified by computerized tomography (CT) or magnetic resonance imaging (MRI) scans were selected from the neurology and neurosurgery wards of Groote Schuur Hospital (Cape Town). Six patients were excluded on the basis of damage to mainly the mesial and orbital surfaces of the frontal lobes, rather than the frontal convexity. Four first-language Xhosa speaking patients could not converse fluently enough in English or Afrikaans to be tested in either of these languages and were therefore excluded. Three patients were discharged or referred to a long-term care facility outside the geographical area before testing could be completed and were therefore excluded. One patient passed away before testing could be completed. Four patients declined to participate. Seven patients (four females and three males) therefore participated in the study. None of these patients had a history of psychiatric disorders, substance abuse or a previous neurological disease. Three patients had damage to the right dorsolateral frontal lobe, two had damage to the left dorsolateral frontal lobe and two had bilateral damage. Seven normal volunteers (three females and four males), recruited from the orthopaedics ward constituted the control group. Controls and patients were matched for age and education (See Table 1).

Table 1
Demographic variables of patients and controls

	Dorsolateral frontal patients	Control participants
Number	7	7
Mean age (S.D.)	42 (13.55)	40 (11.01)
Mean education (S.D.)	10 (2.07)	10 (2.22)

3.4. Ethical Considerations

The study was conducted with ethical approval from the University of Cape Town's Department of Psychology and the Groote Schuur Hospital Ethics Committees. Given that the study took place in a hospital setting, the researcher was sensitive to the health of participants and those who were deemed to be too ill to participate immediately were given time to recuperate before being interviewed. Informed consent was obtained from all participants prior to the administering of any tests (see appendix B). As the study had a low risk of inflicting either emotional or physical trauma, the need for oral debriefing was deemed unnecessary. The confidentiality of participants' dream reports and test results were protected. All audio- recordings made were suitably anonymised, securely stored, made accessible only to the investigator and destroyed at the end of the project.

3.5. Instruments

The following materials and instruments were used: a) a Demographic Questionnaire, b) a Most Recent Dream report (Hall & Van de Castle, 1966 cited in Domhoff, 1996), c) the Orientation test of the Mini Mental Status Exam (MMSE) (Folstein, Folstein & McHugh, 1975), e) the Digits Forward test of the Wechsler Adult Intelligence Scale (WAIS-R) (Wechsler, 1981), f) various sub-tests of the Delis-Kaplan Executive Function System (D-KEFS) (Delis, Kaplan & Kramer, 2001), g) two tests measuring verbal and visual memory respectively: i) the Babcock Story Recall Test (Babcock, 1930 cited in Lezak, 1995) and ii) the Rey Complex Figure Test (Rey, 1941 cited in Lezak, 1995).

a) Demographic Questionnaire

The demographic questionnaire included questions regarding age, gender, home language, level of education, history of psychiatric illness, history of substance abuse, description of current medical condition and diagnosis and CT or MRI scan report, if relevant (see Appendix A).

b) Most Recent Dream Report

All participants were subjected to an in-depth interview in which they were asked to recall the *most recent dream* they have experienced *after* the onset of

neurological illness (see Appendix A). The importance of reporting the most recent dream experienced was emphasized, since people have a tendency to report recurrent dreams or nightmares, or dreams which are especially unusual (Domhoff, 1996). The importance that the dream is dated after the onset of neurological illness is self-explanatory in this case. Some participants initially claimed that they do not dream, or that they do not remember their dreams. Any reported absence of dreaming was carefully explored. Since it was not expected that any of the dorsolateral frontal convexity patients would experience anoneria, they as well as those participants who did not remember their dreams were encouraged to pay careful attention to their dreams and to try to remember as much of them as possible. These participants were reinterviewed the following day or until a most recent dream report was obtained. In addition, all participants were asked whether they had experienced any changes in their dreams since falling ill.

c) *Orientation for time and place*

Orientation for time and place was assessed using the appropriate subtests of the MMSE (Folstein, Folstein & McHugh, 1975). The test consists of five items measuring the participant's orientation for time (year, season, month, date and day of the week), as well as five items measuring orientation for place (country, province, city, hospital/building/home, and ward/clinic/floor). Orientation can be defined as "the awareness of self in relation to one's surroundings" (Lezak, 1995, p.335), and requires consistent integration of attention, memory and perception. Although preserved orientation depends on the intactness and integration of many different mental activities (Lezak, 1995), and therefore has no localizing significance by itself, one would expect impaired orientation in dorsolateral prefrontal patients given the attentional deficits commonly associated with damage to this area (Fuster, 1989).

d) *Digits Forward Test*

The Digits Forward Test of the WAIS measures attentional capacity and consists of eight pairs of random number sequences that the examiner reads aloud at the rate of one number per second (Wechsler, 1981). As mentioned above, patients with damage to the dorsolateral prefrontal cortex show marked deficits in

attention (Stuss & Benson, 1986), which often takes the form of a lowering of general awareness (Fuster, 1989). Digit span is considered to be highly vulnerable to brain damage, and is often impaired in patients with frontal lobe damage (Fuster, 1989; Stuss & Benson, 1986).

e) Subtests of the Delis-Kaplan Executive Function System (D-KEFS)

The extent of executive dysfunction was determined using the following subtests of the D-KEFS:

i. Trail Making Test (Condition 4: Number-Letter Switching)

The task requires the examinee to switch back and forth between connecting numbers and letters in sequence. It measures the examinee's ability to engage in cognitive flexibility. Cognitive flexibility is essential for higher-level skills such as multitasking, simultaneous processing, and divided attention (Delis, Kaplan & Kramer, 2001). Impaired performance on the switching condition of other versions of the Trail Making Test (i.e. as part of the Halstead-Reitan battery) has been found to be particularly sensitive to frontal lobe damage (Reitan, 1958 cited in Stuss & Benson, 1986).

ii. Word-colour Interference Test (Condition 3: Inhibition)

This test is based on the Stoop (1935) procedure and requires the examinee's to inhibit an over-learned verbal response (reading the words) in order to generate a conflicting response (naming the colour of the ink the words are written in) (Delis, Kaplan & Kramer, 2001). Poor performance of this task is seen as reflecting deficits in concentration (Lezak, 1995).

iii. Sorting Test

The D-KEFS Sorting Test was designed to isolate and measure multiple components of concept formation and problem solving abilities. The test consists of two card sets, with six cards in each, with various stimulus words and perceptual features. During Condition 1, Free Sorting, the examinee is required to sort the cards into two groups of three cards each, according to as many different categorization rules as possible (there are eight possible target sorts in total). This measures the examinee's ability to initiate problem-

solving behaviour. In addition, he/she must describe the concept he/she used to generate each sort, which allows for an assessment of the examinee's ability to explain the sorting concepts he/she used abstractly (Delis, Kaplan & Kramer, 2001). However, some examinees with impaired concept formation may also be impaired in their ability to initiate problem solving behaviour, which will lead to few incorrect description responses in Condition 1. A second condition has therefore been incorporated, the Sort Recognition condition, during which the examiner sorts the cards, and the examinee has to attempt to identity the concept used to generate the sort (Delis, Kaplan & Kramer, 2001). An early version of the test (the California Card Sorting Test) was found to be sensitive to deficits in multiple executive functions in patients with focal frontal lesions (Delis, Squire, Bihrle, & Massman, 1992). The cognitive demands made on the examinee by the D-KEFS Sorting test can be compared with those made by the more well-known Wisconsin Card Sorting Test (Delis, Kaplan and Kramer, 2001). Dorsolateral frontal patients have found to be significantly impaired on the latter task in comparison to patients with inferior frontal and posterior lesions (Milner, 1963).

iv. Tower test

The objective of this task is to move disks of various sizes across three pegs to build a designated tower in the fewest number of moves possible and to simultaneously follow a number of rules. It taps a variety of executive functions such as spatial planning, rule learning, inhibition of trial-and-error responding, and the ability to establish and maintain cognitive set (Delis, Kaplan and Kramer, 2001). The test consists of nine items of increasing level of difficulty. Patients with frontal damage have been found to be severely impaired on a very similar test, the Tower of London (Shallice, 1982 cited in Fuster, 1989).

f) Memory tests

Although patients with damage to the prefrontal cortex do not suffer from a basic incapacity to form or to retain memories, they may exhibit memory deficits because of low drive and attentional deficits (Fuster, 1989). Given finding by Cathala et al. (1983) that various graded tests of visual and verbal memory

correlated positively with frequency and informative richness of dream recall, deficits in memory posed a possible extraneous variable in dorsolateral frontal patients ability to recall their dreams. Verbal and visual memory was therefore assessed using two tests: the Babcock Story Recall test and the Rey Complex Figure Test.

i. *Babcock Story Recall Test*

In this test, a 21-unit story is used to measure immediate and delayed recall of verbal material. The original story developed by Babcock (1930 cited in Lezak, 1995) reads:

December 6/ Last week/ a river/ overflowed/ in a small town/ ten miles/ from Albany./ Water covered the streets/ and entered the houses./ Fourteen persons/ were drowned/ and 600 persons/ caught cold/ because of the dampness/ and cold weather./ In saving/ a boy/ who was caught/ under a bridge,/ a man/ cut his hands.

The test was slightly adapted to make it more appropriate for a South African situation by changing the words "miles" to "kilometres" and "Albany" to "Knysna" (see Appendix A). The test was administered according to the standardized procedure described in Lezak (1995).

ii. *Rey Complex Figure*

The Rey Complex Figure designed by Rey (1941, as cited in Lezak, 1995) aims to investigate both perceptual organization and visual memory in brain-damaged subjects. Following the standardized procedure described in Lezak (1995), subjects were asked to copy the figure, set out so that its length ran along the subject's horizontal plane, on a blank piece of paper. On completion of the copy trial the picture was removed and immediate recall was tested after a three-minute interval. Delayed recall was tested after 40 minutes of intervening tasks.

3.6. Procedure

Patients with focal frontal damage were selected from the neurosurgery and neurology wards of Groote Schuur hospital as identified by their hospital notes and CT or MRI scan report. After a senior radiologist reviewed the scans, those patients with dorsolateral damage were approached by the researcher, told what participation in the study would involve, and asked whether they would like to participate. Patients who were willing to participate were asked to give written informed consent. As many of the patients were gravely ill and tired quickly, a flexible approach was taken to testing. Thus patients were typically tested over a period of 2-3 days, with about an hour of testing taking place on each day. Testing took place in the ward, but in two cases patients were discharged before testing could be completed. These patients were followed up at home. A similar approach to testing was used with control participants, who were selected from the orthopaedics ward after being matched with frontal patients in terms of age and education and then recruited.

3.7. Scoring and Experimental measures

3.7.1. Most Recent Dream Report

The Hall/Van de Castle system of dream analysis was used in order to code and subsequently compare the dreams of dorsolateral frontal patients with those of normal controls (Hall & Van de Castle, 1966 cited in Domhoff, 1996). This system has the advantage that it is possible to achieve high inter-rater reliability with it, since it has clearly defined categories and consists of nominal scales only, and is therefore not dependent on raters making difficult comparative judgements as in the case of hierarchical scales (Domhoff, 1996). The Hall/Van de Castle system is an empirical, as opposed to theoretical scale, and consists of 10 general categories, namely:

- Characters
- Social interactions
- Activities
- Striving: Success and Failure
- Misfortunes and Good fortunes
- Emotions
- Physical surroundings: Settings and objects

- Descriptive elements
- Food and eating
- Elements from the past

The Hall/Van de Castle system is often used in order to understand the meaning of dreams in a specific population or to determine how people's dreams relate to their waking concerns (Domhoff, 1996). However, this was not the focus of the present study as the aim was to compare the overall quality, or typical 'dreamlikeness' of the dreams experienced by dorsolateral frontal patients with those experienced by normal participants. Based on the four aspects highlighted by Nielsen's (2000) definition of dreams, four content categories were created out of the various categories and subcategories of the Hall/Van de Castle coding system. These were: i) Emotions, ii) Bizarre or Unrealistic Elements, iii) Sensory Impressions, and iv) Dramatic Intensity. These are briefly discussed below.

i. Emotions

The emotion category of the Hall/Van de Castle coding system measures any explicitly stated feelings present in the dream report and consists of five subcategories: anger, apprehension, sadness, confusion, and happiness.

ii. Sensory impressions

The extent to which sensory impressions were present was measured by a summation of the categories for characters, physical surroundings, and descriptive elements.

iii. Unrealistic elements

The presence of bizarre or unrealistic elements was assessed with a separate subscale of the coding system, the Unrealistic Elements Scale (Domhoff, 1996, p. 278).

iv. Dramatic intensity

The degree of "dramatic intensity" was obtained by summing up the scores obtained on the following seven content categories: aggression, friendliness, sex, success, failure, misfortune, and good fortune (Domhoff, 1996). Both aggression and friendliness are subscales of the social interaction scale. Sex falls into a

category of its own and success and failure are both part of the striving scale. Misfortune and good fortune form one broad category.

Two blind raters rated each dream report according to these four categories after being given a small sample of dreams for training purposes. During the training session any ambiguities about coding categories were discussed and resolved. Inter-rater reliability, as established with Pearson's product moment correlation coefficient, was high. The percentage perfect agreement on the 'emotion' category was 79%, it was 99% for 'sensory impressions', 90% for 'unrealistic elements', and 94% for 'dramatic intensity'. The relatively low percentage perfect agreement on the 'emotion' category in comparison to the other categories can be explained in terms of the low frequency of emotions explicitly stated in dreams in this sample, as well as raters tendency to infer emotions from the contents of the dream despite being explicitly instructed otherwise.

3.7.2. Orientation for Time and Place

Orientation for time and place was assessed using five items each, with one point awarded for each correct answer, thereby providing a total orientation score out of ten.

3.7.3. Digit Forward Test

Digit span was established by administering 8 trials of increasing span length consisting of two items each. The test was discontinued when the participant was unable to correctly repeat back both items of any given trial. Thus digit span was determined by the trial in which the participant was able to pass at least one item. Because the Wechsler scoring system confounds information about length of span with information about the reliability of span performance (Lezak, 1995), the recommended scoring system in which the participant is awarded one point for each correct trial (Wechsler, 1981) was not used. Instead, the length of span of patients and controls were compared.

3.7.4. DK-EFS subtests

i. Trail Making Test (Condition 4: Number-Letter Switching)

In order to evaluate whether participants had a deficit in cognitive flexibility, the completion-time measure of this test was used. In addition,

three types of errors were scored. These are: sequencing errors, which occur when an examinee makes a connection within the correct set of symbols (number or letters), but connects the wrong item within that set; set-loss errors, which occur when an examinee fails to switch from one set of symbols to the other; and time-discontinue errors, which occur when an examinee fails to connect one or more items because the time limit has elapsed. These errors where summed in order to obtain a total error score, which reflects the degree of dysfunction on the task (Delis, Kaplan & Kramer, 2001).

ii. Word-colour Interference Test (Condition 3: Inhibition)

Performance on this test is measured by the time taken to complete the condition, as it reflects the examinee's ability to inhibit the more automatic task of reading words in order the name the dissonant ink colours as quickly as possible. In addition, two types of errors were scored: corrected and uncorrected errors. These errors were summed in order to obtain a total error score, which reflects the severity of an examinee's impaired performance on this test (Delis, Kaplan & Kramer, 2001).

iii. Sorting Test

Examinees' ability to sort the cards was measured by the confirmed correct sort index. This measure is based on the number of initial, correct target sorts across both card sets of Condition 1 for which the description response received a score of at least 1 point. Examinees' ability to describe their sorting concepts was measured by the free sorting description score, which is based on the sum of correct description scores across both card sets of Condition 1. The total number of correct description scores summed across both card sets on Condition 2 provided the sort recognition description score, which quantifies an examinee's ability to describe the sorting concepts or rules used by the examiner to sort the cards (Delis, Kaplan & Kramer, 2001).

iv. Tower Test

The total achievement score, which is the sum of achievement scores for all items administered, was used to establish examinees' proficiency at this task. In addition, the total number of rule violations across all items administered was divided by the total number of items administered in order to establish the Rule-Violation-Per-Item Ratio. Using this ratio corrects for varying levels of successful achievement (Delis, Kaplan & Kramer, 2001).

3.7.5. Memory Tests

i. Babcock Story Recall Test

Following the scoring instruction detailed in Lezak (1995), four points were added to the total number of units recalled on immediate recall in order to equate for the second reading of the story before the delayed recall trial. The delayed recall score was established by simply adding up the number of units recalled during this trial.

ii. Rey Complex Figure Test

During all three trials the drawing sequence was recorded by the examiner and scored according to the unit scoring system developed by Osterrieth (1944, as cited in Lezak, 1995, p. 570). The scoring system requires the examiner to consider each of the 18 units of the figure separately. The accuracy of each unit is appraised as well as its relative position within the whole design. A maximum score of 36 points can be achieved for each trial administered.

3.8. Statistical Analysis

Due to the non-parametric nature of most of the data obtained on the various measures described above, the Mann-Whitney test for two independent samples was used. In these cases, p-values were estimated using the exact method, unless otherwise stated. A t-test for two independent samples was used when appropriate (i.e. when the data was on a ratio scale). However, in most these cases the assumption

of the homogeneity of variances was violated (as tested by the Levene test). In these cases the analysis was repeated using the Mann-Whitney test, since this test does not make any a priori assumptions about the shape of the distribution (Howell, 1997). Effects were considered significant at the $p < 0.05$ level.

The dream reports of prefrontal patients and controls were compared using Domhoff's (1996) recommended method. Dream reports from different groups may differ in length, and this may in turn influence the number of elements present in the dream (Domhoff, 1996). Since any significant differences in dream length would therefore have to be compensated for during analysis, the word-count of the dream reports produced by prefrontal patients and controls were compared before establishing whether there were any quantitative differences in the dreams of the two groups. Individual differences in report length will however still affect the frequencies of dream elements (Domhoff, 1996). To control of this, general categories were converted into a percentage of the total frequency of all categories present. Because the overall aim of the study was to compare the "dream-likeness" of the dreams experienced by dorsolateral prefrontal patients with that of controls, subcategories of each general category were not analysed. Such an analysis would in any case be meaningless due to the low frequency of some categories, and the small sample size. Effect size was determined by comparing the percentage scores of each group on a specific category with each other after adjustment using Cohen's h statistic. Significant levels were determined by calculating z scores. All data was captured on Excel and analysed using the Statistica statistics package.

4. Results

4.1. Most Recent Dream Report

Consistent with Solms' (1997) findings, none of the prefrontal patients reported any subjective changes in their dreams since falling ill. Analysis of the length of the dream reports produced by patients and controls showed that the mean number of words in patients' dream reports was 83.7 whereas the mean number of words in controls' dream reports was 89.9 (Standard deviation 20.89 and 19.37 respectively). A *t* test comparing the two groups was non-significant [$t(12) = -0.57$; $p = 0.579$], indicating there were no significant difference in the length of the dream reports produced by patients and controls.

In order to determine whether there were any quantitative differences in dreams of dorsolateral frontal patients and normal controls, the number of elements present for each content category were compared. No significant difference was detected between patients and controls in terms of the number of emotions present in their dreams [h = 0.149; z = 0.279; p = 0.39]. There was also no significant difference between patients and controls in terms of dramatic intensity [h = 0.231; z = 0.432; p = 0.33]. Similarly, there was no statistical difference between patients and controls in terms of the number of sensory impressions present [h = 0.124; z = 0.232; p = 0.41]. Finally, there was also no statistical difference between patients and controls in terms of the number of unrealistic elements present in their dreams [h = 0.148; z = 0.277; p = 0.39]. No significant differences were therefore found between dosolateral frontal patients and control participants in terms of any of the content categories.

Table 2
Frequencies of sub-categories for the general category: Emotion

	Patients	**Controls**
Anger	0	0
Apprehension	2	2
Sadness	1	1
Confusion	0	1
Happiness	1	3
Total frequency	**4**	**7**

Table 3
Frequencies of sub-categories for the general category: Dramatic Intensity

	Patients	**Controls**
Agression	4	1
Friendliness	2	2
Sex	0	0
Success	0	2
Failure	1	0
Misfortune	2	0
Good Fortune	0	0
Total frequency	**9**	**5**

Table 4
Frequencies of sub-categories for the general category: Sensory Impressions

	Patients	Controls
Characters	15	14
Physical surroundings	22	35
Descriptive Elements	14	10
Total frequency	**51**	**59**

Table 5
Frequencies of sub-categories for the general category: Unrealistic Elements Scale

	Patients	Controls
Unusual Activities	0	0
Unusual Occurrences	1	1
Distorted Objects or arrangements of objects	0	0
Metamorphoses	1	0
Total frequency	**2**	**1**

Table 6
Frequencies and percentages for each major content category

	Patients		Controls	
	f	%	f	%
Emotion	4	6%	7	10%
Dramatic intensity	9	14%	5	7%
Sensory impression	51	77%	59	82%
Unusual elements	2	3%	1	1%

4.2. Orientation for Time and Place

The prefrontal group obtained an average orientation score of 6.7 as opposed to an average score of 9.9 obtained by control participants (standard deviation 2.37 and 0.38 respectively). When a t-test was performed in order to compare the orientation score of prefrontal patients and controls, the assumption of homogeneity of variances was violated [Levene $F(12) = 7.609$; $p = 0.017$]. The analysis was therefore repeated using the Mann-Whitney test, which revealed a significant difference between patients and controls [$U = 5$; $z = -2.49$; $p = 0.011$]. Prefrontal patients were therefore found to be significantly less orientated for time and place than control participants.

4.3. Digit Span Forward Test

Prefrontal patients were found to have an average digit span of 6, whereas control participants had an average digit span of 6.14 (standard deviation 0.57 and 0.69 respectively. This difference was found to be non-significant [U = 21.5; z = -0.383; p = 0.710].

4.4. Subtests of the D-KEFS

i. Trail Making Test (Condition 4: Number-Letter Switching)

No significant difference was found between patients and control participants in terms of completion time on Trail Making test [t(12) = 1.912; p = 0.08]. However, patients made significantly more errors than control participants on this test [*U* = 7; z = 2.236; p = 0.026].

ii. Word-colour Interference Test (Condition 3: Inhibition)

No significant difference was found between patients and controls in terms of completion time on the inhibition trail of the Word-Colour test [t(12) = 1.705; p = 0.114]. Similarly, the result was non-significant when the analysis was repeated using the Mann-Whitney test [*U* = 18, z = 0.83, p = 0.486]. However, patients made significantly more errors than control participants [*U* = 8.5, z = 2.044, p = 0.038] on this test.

iii. Sorting Test

Patients were able to sort significantly fewer groups of cards correctly than controls [*U* = 8; z = -2.108; p = 0.038]. They also scored significantly lower than controls in terms of their ability to describe their sorts [*U* = 3; z = -2.747; p = 0.004]. In addition patients' recognition description score was significantly lower than that of controls [*U* = 5; z = -2.492; p = 0.011].

iv. Tower test

There was no significant difference between patients and controls on the total achievement score [*U* = 10.5; z = -1.788; p = 0.073]. When a t-test was performed in order to compare the rule-violations-per-item ratio of patients and controls, the assumption of the homogeneity of variances was violated [Levene F (12) = 9.102, p =

0.011]. The analysis was therefore repeated using the Mann-Whitney test, which revealed a significant difference between patients and controls [U = 7; z = 2.236; p = 0.026]. Patients therefore had a significantly higher error ratio than controls.

Table 7
Results of DK-EFS subtests

	Patients (n =7)	**Controls (n =7)**
Trail making 4 (Completion time)	208.6 (37.11)	161.9(52.93)
Trail making 4 (Total errors)	10.6 (9.43)*	1.1(0.09)
Word-colour Inhibition (Completion time)	114.7 (45.38)	82.1(22.22)
Word-colour Inhibition (Total errors)	13.4 (15.16)*	1.9(1.95)
Sorting Test (Total confirmed correct sorts)	3.3(1.79)*	5.6(1.39)
Sorting Test (Free sorting description score)	10.0(4.62)*	21.4(6.39)
Sorting Test (Recognition description score)	12.0(7.92)*	24.6(5.38)
Tower Test (Total achievement score)	10.7(5.99)	15.0(0.58)
Tower Test (Rule violations per item ration	1.0(1.06)*	0.1(0.11)

Averages with S.D. in parentheses are reported. Underlined values are significantly different from Control Group when converted to rank sum values; *P < 0.05.

4.5. Memory Tests

i. Babcock Story Recall Test

Significant differences were found between patients and controls on both the immediate [U = 0.00; z = -3.131; p = 0.0005] and delayed recall [U = 1.00; z = -3.00; p = 0.001] trials of the Babcock Story Recall tests. This indicates that patients recalled significantly fewer units of information than controls on both trials.

ii. *Rey Complex Figure*

A significant difference was found between patients and control subjects with regard to the number of units produced on the Rey Copy trial [U = 7.5; z = -2.172; p = 0.026]. A significant difference was also found between patients and controls in terms of the number of units recalled on immediate recall [U = 9; z = - 1.981; p = 0.047], but when the more stringent exact probability was applied, this result was not found to be significant [exact p = 0.053]. No significant difference between patients and controls was found in the number of units recalled during delayed recall [U = 9.5; z = -1.917; p = 0.053].

Table 8
Results of memory tests

	Patients (n =7)	Controls (n=7)
Babcock (Immediate)	7.4(1.98)**	17.0(3.00)
Babcock (Delayed)	6.0(3.32)**	12.7(3.95)
Rey Copy	27.1(10.29)*	35.1(1.06)
Rey Immediate Recall	11.4(6.7)	18.1(2.44)
Rey Delayed recall	10.0(6.35)	16.3(2.07)

Averages with S.D. in parentheses are reported. Underlined values are significantly different from Control Group when converted to rank sum values; *P < 0.05; **P<0.01.

5. Discussion

5.1. Unchanged dreaming and damage to the dorsolateral frontal lobe

The current study replicated Solms' (1997) finding that patients with damage to the dorsolateral frontal lobes report no changes in the subjective experience of their dreams. In addition, we were able to extend his findings by showing that when a content analysis of the dream reports produced by prefrontal patients and control participants was performed, no statistical differences could be detected between the two groups on four major content categories. Moreover, damage to the dorsolateral prefrontal convexity did no seem to affect the length of the dream reports produced by these patients. These results are consistent with a number of neuroimaging studies that showed deactivation of the dorsolateral frontal lobes during both REM and non-REM sleep (Anderson et al., 1998; Braun et al., 1997; Madsen et al., 1991; Maquet et al., 1997).

Damage to the dosolateral prefrontal area was associated with a significant deficit in verbal memory, but not in visual memory. Given the finding that prefrontal patients did not produce significantly shorter dreams, or dreams with a significantly lower frequency of dream elements on any of the content categories, this seems to indicate that deficits in verbal memory do not adversely affect dream recall. However, this finding is inconsistent with that of Cathala et al. (1983) who reported positive correlations between tests of verbal memory (and visual memory) with dream frequency and informative richness of dream recall in frontal patients. This discrepancy can probably be explained in terms of methodological differences between the latter study and the present one (cf. Solms, 1997 for a critique), as well as

differences in the location of the lesion in the frontal group. The lack of statistical differences between the dreams produced by patients with damage to the prefrontal lobes and normal participants therefore indicate that damage to this area causes no significant changes in the dreams of patients with damage to this area.

5.2. Executive dysfunction and the formal features of dreams

According to Hobson (2002) dreams have a number of formal features, amongst which internally generated perceptions and emotions are the two most fundamental. In addition, dreams are characterized by a number of cognitive deficits such as a loss of self-reflective awareness, loss of orientation stability, loss of directed thought, reduction in logical reasoning, poor memory, and loss of volition (Hobson, 2002). Various researchers have hypothesised that deactivation of the dorsolateral prefrontal cortex, and other heteromodal association areas, such as the inferior parietal cortices, during dreaming may account for some of these cognitive deficits in dreams (Braun et al, 1997; Hobson et al., 2000; Maquet et al, 1996; Solms, 2000).

In the present study, damage to the dorsolateral prefrontal cortex was associated with a poor performance on a number of executive tests. Thus the dorsolateral frontal group were found to have significant difficulty in acquiring and maintaining cognitive set as evidenced by a significantly higher error score on the Trail Making Test, as well as a higher rule-violations-per item ratio than the control group on the Tower Test. The prefrontal group's high error ratio on the Tower Test also indicates rule-breaking behaviour and a deficit in the ability to use mistakes to guide future behaviour. Deficits in problem solving behaviour were noted in the prefrontal group, as illustrated by their significantly decreased ability to *initiate* problem-solving behaviour compared to controls on the Sorting Test. In addition, the prefrontal group were found to be concrete, as evidenced by defective concept formation skills on both the Free Sorting condition and Sort Recognition condition of the Sorting Test.

In our sample, attentional deficits were revealed by the prefrontal group's poor performance on the Trail Making Test and the Stroop Test. Both these tests are considered to be sensitive to frontal lobe damage, and to attentional deficits in particular (Stuss & Benson, 1986). Conversely, no significant differences were found between prefrontal patients and control participants in terms of Digit Span, another test traditionally used to assess attention (Lezak, 1995). However, it should be noted

that digit span does not consistently show decline after frontal lobe damage (Stuss & Benson, 1986).

An attentional deficit could explain our findings of significantly poorer recall of verbal material by the prefrontal group, both immediately and after a substantial delay as tested by the Babcock Story Recall Test. According to Fuster's (1989) theoretical model of frontal lobe functions, the lack of drive associated with damage to the dorsolateral prefrontal cortex may in turn affect these patients ability to direct and maintain attention. As a consequence thereof, "the patient fails to memorize the essential material for reaching conceptual and behavioural goals (Fuster, 1989, p. 136). Thus the prefrontal cortex is seen as subserving the temporary retention of information necessary for goal-directed behaviour (Fuster, 1989; Luria, 1966/1980). Fuster's (1989) theory is bolstered by recent findings that the dorsolateral prefrontal cortex is active during short-term memory tasks and during free recall (Fletcher, Frith, & Rugg, 1997; Wagner et al, 1998).

Attentional deficits could also explain why the prefrontal group was found to be significantly less orientated for time and place than the control group. The ability to be aware of oneself in relation to one's surroundings "requires consistent and reliable integration of attention, perception, and memory" (Lezak, 1995, p. 335). In frontal patients the ability to direct attention and sustain it over a period of time is impaired (Fuster, 1989; Stuss & Benson, 1986).

Could the lack of significant change in the production of dreams subsequent to damage to the dorsolateral frontal lobes be explained by these cognitive deficits associated with damage to the dorsolateral prefrontal lobes? According to both Hobson (2002) and Solms (1997) they could.

5.2.1. Orientational deficits and dream bizarreness

As shown in the current study, damage to the dorsolateral prefrontal lobes is associated with orientational deficits, which are in turn associated with attentional deficits (Stuss & Benson, 1986). In dreams, orientational instability leads to plot discontinuity, where time, place, persons and actions change without notice (Hobson, 2002; 2005). According to Hobson, plot discontinuity along with plot incongruity (the mismatching features of characters, objects, actions, or settings) and cognitive uncertainty (explicit vagueness) are the qualitative characteristics of dream bizarreness (Hobson et al., 1987). Deactivation of the dorsolateral frontal convexity

may account for these orientational deficits, and thus explain the bizarre nature of dreams to some extent (Hobson et al., 2000).

In addition to these orientational deficits, the dreaming brain has a tendency to confabulate, due to an impairment in memory (discussed in more detail below). These cognitive deficits, together with visual hallucinations, are very similar to a functional delirium. Thus Hobson (1997; 2004) suggests that the dreaming brain suffers from a psychosis, similar to that associated with drug or alcohol abuse. This position is similar to that of Solms and Turnbull (2002), who argue that "the functional anatomy of dreaming is almost identical to that of schizophrenic psychosis" (p. 213). However, Domhoff (2005) has criticized this characterization of dreaming as a psychotic state. Thus he argues that Hobson's and Solms' emphasis on dream bizarreness is misplaced and is not supported by the empirical findings on dream content. In addition, Domhoff (2005) argues that findings by Hobson and co-workers (Rittenhouse, Stickgold & Hobson, 1994) which indicate a high frequency of abrupt scene and character changes do not necessarily indicate that dreams are inherently bizarre as such events happen in waking fantasy as well. Although this may be the case, Domhoff (1995) does not consider that in waking life, unlike in dreams (and psychosis), we are able to reflect on the incongruities and impossibilities of our fantasies (Kahn & Hobson, 2005).

5.2.2. Self-reflective awareness and reduced logical reasoning

Self-reflective awareness, also called meta-awareness, can be defined as the "awareness of mental life itself" (Cicogna & Bossinelli, 2001). In other words, self-reflective awareness involves being aware of the phenomenal experience of objects or events. Self-reflective awareness can be seen as the basis of intelligent behaviour. It implies the acquisition and use of knowledge in an organized and deliberate way (Stuss & Benson, 1986). The characteristics of self-reflective awareness closely resemble those attributed to prefrontal functions, leading Stuss and Benson (1986) to hypothesise that self-awareness is the highest attribute of the frontal lobes. Impairment in self-reflective awareness can be observed in the shallowness of interest, loss of self-concern and impairment of self-monitoring commonly associated with damage to the prefrontal area (Fuster, 1989; Stuss & Benson, 1986). In the current study, these deficits were illustrated by dorsolateral frontal patients' significantly higher error ratio on the Tower Test, and by their higher error score on

the Stroop Test in comparison to controls. Although we are not claiming that self-reflective awareness can be localized exclusively to the dorsolateral prefrontal cortex, damage to this area does seem to impair these patients' ability to monitor their behaviour.

In dreams, self-reflected awareness is functionally expressed as "reality testing": the ability to reflect and recognize that one is dreaming instead of having an experience that is coming from the real world (Bosinelli, 1995; Cicogna & Bosinelli, 2001). Studies indicate that this ability is absent in 90-95% of dreams (Cicogna & Bosinelli, 2001). In addition, a recent study has shown that we suffer from a specific kind of impairment in logical reasoning whilst dreaming (Kahn & Hobson, 2005). Thus it seems that our ability to think about the dream (i.e. to detect the incongruities in the hallucinatory experience) is impaired. Although thinking within the context of the dream was not found to be common, this was found to be rational and thus similar to our waking thought (Kahn & Hobson, 2005). The finding that metacognition, 'knowing that we are hallucinating' is impaired during dreaming was attributed to deactivation of the dorsolateral prefrontal cortex and precuneus (Kahn & Hobson, 2005). Findings that metacognition is restored during lucid dreaming, has led Kahn and Hobson (2005) to speculate that the dorsolateral prefrontal cortex becomes reactivated in this state. In patients with damage to the dorsolateral prefrontal cortex such reactivation may not occur. However, these speculations still need to be validated by brain imaging studies.

5.2.3. Poor memory within the dream

As discussed above, damage to the prefrontal cortex is associated with a specific, attention related, memory impairment (Fuster, 1989). Although dream elements often appear to arise from waking events, this does not imply that episodic memory is necessarily used for dream construction (Stickgold et al., 2001). Episodic memory can be defined as "a memory of an event, recalled as an integrated whole, with the actual waking event (or "episode") replayed in one's mind" (Stickgold et al., 2001). Episodic memories are thought to be dependent on the hippocampus for their integration (Schacter & Tulving, 1994). Findings that dream elements only reflect one or two aspects of the waking experience, instead of fully integrated episodic memories (Stickgold et al., 2001), seem to indicate that dream elements are not hippocampally mediated episodic memories. Reduced cortical output by the

hippocampal formation during REM sleep, as well as the deactivation of the dorsolateral frontal lobes during both REM and non-REM may account for this phenomenon (Stickgold et al., 2001). Impairment of episodic memory during dreaming limits our access to facts that are readily available during waking life (Hobson, 2005). Thus, for example, dream characters known to us are often not represented accurately in our dreams (Khan, Pace-Scott & Hobson, 2002) and important information (such as that the character is no longer alive, for example), is inaccessible (Hobson, 2005). Reduced activation of the dorsolateral prefrontal cortex during dreaming may, in part, explain why we are unable to recall accurately in our dreams.

5.2.4. Volition and the dorsolateral prefrontal lobes

Damage to the prefrontal convexity is associated with a disorder of goal-directed behaviour (Fuster, 1989; Luria, 1966/1980). As discussed above, this disorder is seen as the consequence of a fundamental impairment in the ability to temporally organize behaviour (Fuster, 1989). Behaviourally, it manifests as concreteness, stereotypical behaviour (Fuster, 1989), and a dissociation between knowing and doing (Luria, 1966/1980). Deficits in temporal integration are readily demonstrated by neuropsychological tests such as the WCST (Fuster, 1989). In the present study dorsolateral prefrontal patients' difficulties in temporal integration were illustrated by concreteness and deficits in problem solving ability on the Sorting Test.

During dreaming, deactivation of the dorsolateral prefrontal lobes, together with the inhibition of spinal motoneurons, will prevent appetitive interests, aroused due to external sensory stimulation as well as endogenous stimulation, such as REM activation, from expressing themselves in volitional motor action (Solms, 1997). Thus deactivation of the dorsolateral prefrontal cortex during dreaming does not only account for some of the striking formal features of dreams, but also serves a very specific function. This function is presumably the preservation of sleep (Solms, 1997).

5.3. Summary and Conclusion

The present study has been able to verify Solms' (1997) observation that damage to the dorsolateral prefrontal cortex does not cause any subjective changes in these patients' dreams. In addition we have been able to show that the dreams of

dorsolateral prefrontal patients are not significantly different from those of normal controls when a content analysis of four major content categories was carried out. Our results are therefore consistent with the available clinico-anatomical (Solms, 1997) and neuroimaging evidence (Anderson et al., 1998; Braun et al., 1997; Madsen et al., 1991; Maquet et al., 1997), which point to a deactivation of the dorsolateral prefrontal cortex during dreaming. In addition, the present study has managed to avoid some of the methodological problems associated with some of these previous studies. Thus we have made use of a more objective method of comparing the dreams of patients with damage to the frontal convexity with those of normal participants than Solms (1997) did in his exploratory study. Moreover, we have avoided the possible confounding variable of conflating REM sleep with dreaming, which is often the case in neuroimaging studies (Solms, 1997).

Damage to the dorsolateral prefrontal cortex does not seem to have any significant impact on the production of dreams. A careful look at the cognitive deficits associated with damage to the dorsolateral prefrontal cortex seem to indicate that deactivation of this area during sleep may account for many of the cognitive deficits associated with dreams to some extent (Hobson et al., 2000). Thus the cognitive deficits exhibited by our sample of dorsolateral prefrontal patients would not affect the production of dreams in any of the ways assessed in this study, because deactivation of the dorsolateral prefrontal cortex (and the attending cognitive deficits) is an inherent part of the dream process. Deactivation of this area may prevent the expression of ideas into action, thereby serving the function of preserving of sleep (Solms, 1997). However, further research is necessary to confirm the relationship between the apparent cognitive deficits in dreams with specific aspects of executive dysfunction.

5.4. Limitations

The sample size of the current study was particularly small due to numerous problems encountered during the data collection phase. Issues surrounding language, and cultural variations in terms of the meaning and significance of dreams in particular, had an impact on sample size and selection. A number of Xhosa speaking participants denied having any dreams and declined, rather forcefully, to participate in the study. It was only when the researcher realized the word "dream" in Xhosa has two very different meanings, when these patients reluctance to participate was

understood. Thus the word "dream" can be translated as *iphupha*, meaning an ordinary dream, or as *umbono* meaning "vision" (or dreams from the ancestors). Having the latter experience may indicate that one has been called to be an *ukuthwasa*, a healer. Although care was taken to use the correct Xhosa word (three of the prefrontal patients and four of the control participants were Xhosa first-language speakers) to indicate the mental experience we were interested in, these kinds of cultural factors may have had an impact on the kinds of dreams which were reported. Varying degrees of fluency in English may also have affected these patients' ability to describe their mental experiences, as well as their performance on some of the tests we conducted. Although these factors should be noted, their impact on the study is not negative, since they affected both the prefrontal and the control group.

Care was taken to ensure that participants reported their most recent dream. However, although Domhoff (1996) argues that the Most Recent Dream method gives a representative sample of all dreams, Chapman and Underwood (2000) have pointed out that there is a tendency toward recalling dramatic, and therefore more memorable, dreams. Thus the study could be expanded upon by collecting a number of dreams from prefrontal patients and controls over a period of a few weeks.

The small sample size did not allow for comparisons to be made between patients with damage to the left or right dorsolateral frontal lobes. Future research could expand on our observations by making use of a larger sample, which will allow for comparisons to be made between right and left dorsolateral prefrontal patients. In addition, the small sample size excluded the possibility of making within group correlations between the scores on cognitive tests and dream content scores, for example, correlations between participants' scores on the test of verbal memory with dream length.

The present study only assessed the general dream characteristics of patients with damage to the dorsolateral prefrontal cortex and compared this with the general dream characteristics of normal participants. Future research should focus on measuring and comparing the specific cognitive deficits associated with dreaming, such as loss of self-reflective awareness, loss or orientational stability, reduction in logical reasoning, poor memory, and loss of volition in the dreams of patients with damage to the dorsolateral frontal lobes and control participants.

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Appendix A: Interview schedule

Patient ☐ **Participant Nr**_____

Control ☐

Name:	
File Number:	
Sex:	
Birth Date:	
Age:	
Marital Status:	
Kin:	
Native Language:	
Schooling:	
Handedness:	
Date of Onset:	
Date of Admission:	
Date of Discharge:	
Type of stroke/ cause of lesion:	

Psychiatric history

History of Alcohol/Drug Use

Description of Current Condition/ Medical History

Brain Scan

Rey Complex Figure Test

Units	(Copy)	(Recall)	Delayed Recall
1.			
2.			
3.			
4.			
5.			
6.			
7.			
8.			
9.			
10.			
11.			
12.			
13.			
14.			
15.			
16.			
17.			
18.			
Total			

Remarks

Babcock Story Recall

December 6/ Last week/ a river/ overflowed/ in a small town/ ten kilometres/ from Knysna./ Water covered the streets/ and entered the houses./ Fourteen persons/ were drowned/ and 600 persons/ caught cold/ because of the dampness/ and cold weather./ In saving/ a boy/ who was caught/ under a bridge,/ a man/ cut his hands.

Immediate Recall

Total Units_____

Delayed Recall

Total Units_____

Dream interview

Interview Date:_____
Date of onset of neurological illness: _____

Do you still dream at night? Yes/No

If yes:
Could you please tell me your *most recent dream*? But before you start could you tell me the date this dream occurred:_____. Was this before or after you became ill?

Could you now describe the dream exactly and in as much detail as you can remember it. Please tell me about anything you saw, heard, smelled, tasted or felt in your dream. Specifically, please describe where your dream took place and whether that place was familiar to you; give me description of the characters in your dream (people or animals) and tell me about everything which happened in your dream, no matter how strange it might have seemed. Also can you tell me of any emotions you experienced in the dream.

_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____

If No:
Do you know what a "dream" is?_____
Have you ever had a dream?_____
When did you have your last dream? (approximate date)_____
Was that before or after you fell ill?_____
Do you think that you have had dreams in the past, but did not remember them?_____
Do you think that you still dream now, but do not remember them?_____
Has your ability to remember your dreams become (a) better, (b) worse, (c) remained unchanged, or (d) are you unsure?
Do you think your dreams have changed since you became ill? Yes/No
If so, please specify in which ways.

Neuropsychological Symptoms

Symptoms	Tick	Remarks
Acalculia		
Adynamia		
Agraphia		
Alexia		
Anosognosia		
Aphasia		
Apraxia		
Achromatopsia		
Constructional Apraxia		
Disinhibition		
Disorientation		
Disturbed Problem-Solving		
Finger Agnosia		
Neglect		
Hypoarousal		
LTM Disorder		
Perseveration		
Prosopagnosia		
Right-Left Disorientation		
STM Disorder		
Topographical Agnosia		
Visual Agnosia		

Behavioural Checklist

Aspontaneity	
Adynamia	
Impersistence	
Akinetic Mutism	
Little Initiative	
Reduplicative Paramnesia	
Contaminated Consciousness	
Impulsiveness	
Distractibility	
Socially Inappropriate Behaviour	
Rule Breaking Behaviours	
Concrete Attitudes	
Disorganised Behaviour	

Concluding Remarks

Appendix B: Information Sheet

Title of the Project:
Dreaming and the dorsolateral frontal lobes: Towards a better understanding of the mechanism of dreaming.

- You are invited to participate as a subject in a psychological study. Please read this information sheet carefully and do not hesitate to ask the researcher for any additional information.

- The overall purpose of the investigation is to explore and evaluate different aspects of dreaming following brain damage.

- As a participant of the study you will be asked to recall and describe in detail some of your dreams. You will also have to complete some neuropsychological tests. Your answers may be audio-recorded.

- There are no anticipated risks involved in this research, but if you should experience mental and/or physical fatigue, or any form of psychological distress please be aware that you should inform the investigator immediately.

- It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and asked to sign a consent form. If you decide to take part you are still free to withdraw from the study at any time, without having to give a reason and without this affecting future treatment.

- The confidentiality of your answers and identity will be protected. All audio-recordings made will be suitably anonymised, securely stored, made accessible only to the investigator and destroyed and the end of the project. In discussions of the results in the research report false initials will be used to identify individual participants.

- This study is an educational project, forming part of a Masters degree in psychology and the University of Cape Town.

- The study is being reviewed by the Department of Psychology's ethics committee.

- If you have any questions regarding this study, or concerns regarding the manner in which the study was conducted or would like to be informed of the results when the study is completed, please feel free to contact the researcher or her supervisor.

Researcher: Tania Badenhorst
Tel. 021-447 8942

Supervisor: Mark Solms
Psychology Department
UCT
Cape Town

Consent Form

Title of the Project:
Dreaming and the dorsolateral frontal lobes: Towards a better understanding of the mechanism of dreaming.

Have you read the Subject Information Sheet YES/NO

Have you had an opportunity to ask questions and discuss the study? YES/NO

Have you received satisfactory answers to all of your questions? YES/NO

Have you received enough information about the study? YES/NO

Do you understand that you are free to withdraw from the study:
- at any time
- without having to give a reason for withdrawing
- and without affecting your future treatment? YES/NO

Do you understand that some of your answers in the study will be audiotaped? YES/NO

Do you consent to the confidential use of these recordings for scientific purposes? YES/NO

Have you been given a copy of the information sheet And this consent form. YES/NO

Signed_____ **Date**_____

(NAME IN BLOCK LETTERS)_____