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Parkinson's disease and the influence of the forebrain dopaminergic system
on dreaming.

by

Hetta Gouse (GSXHET002)

A minor dissertation submitted in partial fulfilment of the requirements for the award of
the
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Supervisor: Prof. Mark Solms

Department of Psychology

University of Cape Town

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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 from the work, or works, of other people has been attributed, and has been cited and referenced.

Signature: H Gouse

Date: 14.02.2004

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University of Cape Town

Abstract

The purpose of the study was to investigate a hypothesis proposed by Solms (2000) to the effect that a forebrain dopaminergic mechanism is the final common path to dreaming. It was hypothesised that sufferers of Parkinson's disease (PD) would have decreased intensity and frequency of dreams - that is that PD disease may lead to cessation or reduced dreaming, reduced duration of dreams, reduced narrative complexity of dream and a change in dream emotion - and that this will further decrease with the duration of the disease irrespective of the medication that the patient is taking. Self-report questionnaires (N=40) and the Most Recent Dream Report (Domhoff, 1996) was used for assessment. PD patients were found to have shorter as well as more pleasant dreams. A correlation ($p < .05$) was found in the PD sample between duration of illness and emotional quality of dreams and dream duration. The specific hypothesis of the study was not fully supported. However, the findings do support the suggestion that dreaming is generated by the mesocortical-mesolimbic dopamine system.

Keywords: dopamine, parkinson's disease, dreaming.

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A study was done to ascertain the nature - specifically the intensity and frequency - of dreams in Parkinson's disease (PD) patients. The hypothesis investigated was that sufferers of PD would have decreased intensity and frequency of dreams - that is, that PD may lead to cessation or reduced dreaming, reduced duration of dreams, reduced narrative complexity of dream and a change in dream emotion - and that this will further decrease with the duration of the disease irrespective of the medication that the patient is taking. The rationale of the study is to further attempt to confirm the hypothesis that a dopaminergic mechanism "is the final common path to dreaming and that the [cholinergic] brainstem oscillator that controls rapid eye movement (REM) state is just one of the many arousal triggers that can activate this [dopaminergic] forebrain mechanism" (Solms, 2000. p. 843).

1: Introduction

Before the early 1950's sleep was seen as a unitary state. In 1953 and 1955 Aserinsky and Kleitman discovered rapid-eye-movement (REM) sleep, a period of sleep characterised by physiological activation, particularly eye-movement activity. The remainder of sleep came to be known as non-REM (NREM) sleep. By the 1960's the early monistic view of sleep was discarded in favour of this dualistic REM and NREM conception of sleep. Further research made the differences between the two states of sleep more apparent. REM sleep is characterised by activation in several systems - changes in blood flow (Kanzow, Krause & Kuhnel, 1962), blood pressure (Snyder, Hobson & Goldfrank, 1963), heart rate and regularity (Snyder, Hobson, Morrison & Goldfrank, 1964), respiration (Aserinsky, 1965; Snyder et al. 1964) and brain temperature (Kawamura & Sawyer, 1965; Reite & Pegrarn, 1968). It was also found that REM correlated very highly with subjective reports of dreams (Dement & Kleitman, 1957a; 1957b) and consequently dreaming and the physiological state of REM sleep came to be equated. This equation, as will be seen, has recently been shown to be problematic.

It is currently accepted that sleep is composed of 5 cycles. The REM cycle alternates with the 4 other sleep cycles and occurs roughly every 90-100 minutes. About 25% of sleeping time in humans consists of REM sleep. Normal subjects report dreams 70 – 90% of the time on awakenings from REM sleep and 5-10% of the time on awakenings from NREM sleep (Dement & Kleitman, 1957a; 1957b; Hobson, 1988; Nielsen, 1999). These facts contributed greatly to the belief that dreaming and REM sleep are physiologically identical and that dreaming is just “an epiphenomenon of REM sleep” (Hobson, et al., 1998, p. R12; LaBruzza, 1978). Equating REM sleep with dreams also created the possibility of studying the neuronal mechanism of dreaming in animals, despite the fact that the subjective aspect of dreaming is necessarily inaccessible to the researcher and inaccessible to objective study.

Several theoretical frameworks concerning the mechanisms for sleep and dreaming have been suggested. The most authoritative contemporary theory for sleep cycle control is the reciprocal-interaction model (RIM) proposed by McCarley and Hobson in 1975. The dominant (and closely related) neurological model for dream mechanisms is the activation-synthesis model (ASM) proposed by Hobson and McCarley in 1977. Both these models rely heavily on the REM = dream equation. Recently an alternative, dopaminergic hypothesis has been proposed by Solms (1997). “This hypothesis rests on the observation that dreaming and REM sleep are doubly dissociable states. That is, dreams can occur without REM, and REM can occur without dreams – under both normal and pathological conditions” (Solms, 2001, p. 127).

1.1 Theoretical framework

1.1.1 Reciprocal interaction model (RIM)

The perception that the pontine brain stem contains the primary causal generator of dreaming and that dreaming is an epiphenomenon of REM sleep is still strong (Hobson, Stickgold & Pace-Schott, 1998). Jouvet (1962) demonstrated that the forebrain is incapable of producing REM sleep, and that it is also unnecessary for REM sleep

generation. His conclusion that REM sleep is controlled by pontine brain stem mechanisms is still central to all main contemporary models of sleep cycle control.

The RIM, as described by McCarley and Hobson (1975), is a structural and mathematical model for control of the desynchronised phase (REM) of the sleep cycle. The model suggests a reciprocal interaction between cells in the nucleus subcoeruleus and nucleus locus coeruleus (LC) and cells in the pontine gigantocellular tegmental field (GTF). The necessary and sufficient cause of rapid eye movement (REM) sleep and dreaming is the firing of cholinoceptive and/or cholinergic REM-on cells in the gigantocellular tegmental field of the pontine reticular formation (Aserinsky & Kleitman, 1953; 1955). During REM sleep the cells in the nucleus subcoeruleus and nucleus locus coeruleus are off and these aminergic (noradrenergic and serotonic) REM-off cells provide the conditions for REM-on cells to fire.

It is currently acknowledged by Hobson, (Hobson, Lydic & Baghdoyan, 1986) that their initial findings regarding this system are inadequate and that the whole network of nuclei giving effect to and contributing to this oscillatory mechanism is more widely distributed. Despite this their localization of the executive control of the REM/NREM cycle still lies narrowly within the pontine brain stem and the claim by Hobson and colleagues (1998), and that the cholinergic brainstem mechanisms cause REM sleep and dreaming, remains. The assertion that the forebrain is a passive participant in the REM state, that all the visual elements, and motor cortical events in REM sleep is induced by brain stem neurons, also still stand (Hobson, 1988; Hobson & McCarley, 1977; Solms, 2000).

1.1.2 Activation synthesis model (ASM)

The ASM for human dreaming was formulated on the basis of brain stem-lesion research done in cats. This neurocognitive theory postulates that the cholinergic brain stem actively generates dreams and that dreams are passively synthesized by the forebrain. "The activated forebrain synthesizes the dreams by fitting experimental data to information endogenously and automatically generated by reticular, vestibular, and oculomotor neurons in the pontine brain stem" (Hobson & McCarley, 1977, p. 1347).

The proposal is that these cells not only trigger dreaming but that they also determine some of the unusual and bizarre formal features of dreaming.

1.1.2.1. Psychological implications

The psychological implications of the original version of this theory were the following - a) The primary motivating force for dreaming is physiological. Thus, dreams have no psychological meaning or function, certainly not in the sense that Freudian theory proposed; b) “*Specific stimuli for the dream imagery* appear to arise intracerebrally but from the pontine brain stem and not in cognitive areas of the cerebrum” and that the forebrain is “making the best of a bad job in producing even partially coherent dream imagery from the relatively noisy signals sent up to it from the brainstem” (Hobson & McCarley, 1977, p. 1347); c) The emotional, conceptual, and perceptual structures of the forebrain involved in the elaboration of the brain stem stimulus is seen as “primarily a synthetic constructive process, rather than a distorting one as Freud presumed” (Hobson & McCarley, 1977, p. 1374); d) The forgetting of dreams is attributed to a “state dependent amnesia” since a change of state, to waking, when carefully effected can produce good recall. This is directly related to the RIM - “dream amnesia can now be modelled in a testable way as the result of a different balance between cholinergic and aminergic neuronal activity and the resulting effects on second messengers and macromolecules” (Hobson & McCarley, 1977, p. 1374). This leaves the notion of repression as an explanation for the forgetting of dreams redundant.

The clarity of dream vision is attributed to the activation of the visual system during REM sleep. It is suggested that the ponto-geniculo-occipito (PGO) waves are responsible for the improbable constructions in dreams (Hobson, 1988). The theory offers no suggestions as to what cognitive (cerebrum/forebrain) processes might be active during dreaming and maintains that the causal stimuli for dreaming is the pontine brain stem and not the cognitive areas of the cerebrum/forebrain. If we accept that REM sleep, and thus dreaming, are controlled by the pontine brain stem mechanism then we also have to concede that dreaming is not controlled by forebrain mechanisms (Solms, 2000).

1.1.3 REM ≠ Dreaming

The assumption that REM sleep, and therefore dreaming, are controlled by the pontine brainstem is problematic as a) the correlation between REM sleep and dreaming is incomplete; and b) because anticholinergic drugs and lesions on the cholinergic forebrain structures do not reduce or obliterate dreams (Solms, 2001).

a) Early laboratory studies with humans elicited dream reports, although less “dream-like” and more “thought-like” than REM dreams, from NREM awakenings (Foulkes, 1962; Foulkes & Schmidt, 1983; Foulkes & Vogel, 1965; Herman, Ellman & Roffwarg, 1978). Despite this, enough full-blown dream content was present to contradict the REM = dreaming equation. The notion that REM = dreaming was already abandoned by most dream researchers in the late 1960’s (Domhoff, 2003). It is no longer disputed that dreaming can occur in the absence of REM and REM in the absence of dreaming, but the particular frequency of dreaming in NREM is still controversial (Solms, 2000).

Foulkes (1962) found reports of complex mentation in over 50% of NREM awakenings (with 50 -70% of dream reports at dream onset). Later studies placed the average at 43% (Nielsen, 1999). This indicates that at certain points in sleep there is a high dream presence without REM. However, not all REM and NREM dreaming are qualitatively the same, which raises the question of the extent to which NREM and REM dreaming may be equated. Monroe, Rechtschaffen, Foulkes & Jensen (1965) suggest in their study that roughly 10-30% of NREM dreams cannot be distinguished from REM dreams, and even Hobson (1988) accepts the number of 5-10%. “If we adjust this conservative figure to account for the fact that NREM sleep occupies approximately 75% of total sleep time, this implies that roughly one quarter of all REM-like dreams occur outside REM sleep” (Solms, 2000. p. 845).

The rising morning phase of the diurnal rhythm is accompanied by NREM dreaming which becomes exponentially greater in the late morning (Kondo, Anthrobus, & Fein, 1989). This occurs to such an extent that sleep onset, in addition to late morning dreams, and to a lesser degree nightmares are dissociated with “cholinergic activation of an

aminergically demodulated forebrain” (Solms, 2001, p. 125). None of this can be attributed to Hobson’s theory. The ASM is therefore not sustainable.

These findings, which have recently been confirmed by morning awakenings (Antrobus, Kondo, & Reinsel, 1995; Cicogna, Natale, Occihionero, & Bosinelli, 1998) contributed to the modification of the activation-synthesis model and a retreat from the claim that the brain mechanisms that produce the REM state also produce dreams. The modified ASM has been called the AIM (activation, synthesis, input) model.

1.1.4 AIM (activation, synthesis, input)

The expanded ASM model, AIM (1. activation, 2. input 3. modulation) attributes a greater role to the forebrain in the regulation of REM sleep dreaming. This revised model takes into account all states of consciousness and not only dreaming by including two other areas that were previously neglected - input and neurochemical modulation.

1. *Activation* includes total and regional activation levels in the brain. The “global, regionally nonspecific picture of forebrain activation” has been replaced by “a preferential activation of limbic and paralimbic regions of the forebrain in REM” (Hobson, Pace-Schott, & Stickgold, 2000, p. 808). This implies that the forebrain is responsible for shaping dream plots through dream emotion rather than “playing a secondary role in dream plot instigation” (Hobson et al., 2000, p. 808).

2. *Input* “is a measure of the extent to which the brain-mind is processing external sensory data impinging upon receptors (as it is in waking) or from internal data sources (as in day-dreaming or REM sleep)” (Hobson et al., 2000, p. 832).

3. *Modulation* refers to the ratio of aminergic (i.e. serotonin and norepinephrine) to cholinergic (i.e. acetylcholine) chemical influence on the brain in each consciousness state. During waking the ratio is high. During REM it is reversed and serotonin and norepinephrine levels are almost zero (Hobson et al., 2000).

“In the full AIM model, waking is said to be characterized by high levels of brain activation, external sources of input, and aminergic neuromodulation, whereas REM is

characterized by high levels of brain activation, internal sources of input, and cholinergic modulation. NREM is characterized by low levels of brain activation, internal sources of input, and a mixture of aminergic and cholinergic modulation.” (Domhoff, 2003, p.150).

It is maintained that stimulation of the visual cortex through cholinergic neuroactivation is responsible for the visual/hallucinatory imagery of dreaming. The high level of activity in the basal ganglia is responsible for the ever present motion in dreams, and poor executive functions, including working memory, are responsible for the incongruity and discontinuity of dreams (Hobson et al., 2000). This model places less emphasis on the REM stage and NREM dreaming is attributed to subtle stimulation of the same area of the brain stem that produce REM sleep, the pontine tegmentum. Despite this change in the model the shift in theory went almost unnoticed because the notion that dreams are generated by pontine brainstem mechanisms was maintained.

b) According to the ASM, which proposes (in both its classical and modified form) that dreams are synthesised by a cholinergically activated forebrain, it is expected that there should be a decrease in the frequency of dreaming and REM like mentation with the administration of anticholinergic medications and with damage to the cholinergic forebrain structures (Solms, 2001). This does not occur: as Cartwright (1966) observed – anticholinergic drugs such as scopolamine and atropine actually intensify dreaming and dreamlike mentation – the opposite effect to what is expected according to AIM (see Ketchum et al., 1973; Perry & Perry, 1995 for review). Many clinical cases have also been reported where vividness and frequency of dreaming increase after structural damage to the basal cholinergic forebrain nuclei (see Solms, 1997, for review).

1.1.5 Pontine brainstem dream generation

The concept that dreams are generated by the pontine brainstem is further problematic as there is evidence that pontine brainstem damage does not cause loss of dreaming in humans. In domestic cats large lesions of the pontine brainstem result in the elimination of REM sleep. Twenty-six human cases with the same correlation have been reported (Adey, Bors & Porter, 1968; Chase, Moretti & Prenskey, 1968; Cummings & Greenberg, 1977; Feldman, 1971; Lavie, Pratt, Scharf, Peled & Brown, 1984; Markand & Dyken,

1976; Osorio & Daroff, 1980). Of the twenty-six cases loss of dreaming was only reported in one case (Feldman, 1971), but the location of lesion in this case was uncertain. This does not necessarily indicate that dreaming is preserved in such cases (Solms, 2000). However, there is some evidence that dreaming is maintained with pontine brain stem lesions. Solms (1997) reported clinical evidence, but not polygraphic data, for preserved dreaming in four patients with large pontine lesions.

The assumption that dreaming is controlled by brainstem mechanisms will be difficult to refute by direct brainstem lesion data because extensive brain stem lesions are required to destroy both the REM and NREM components of the oscillator hypothesised to generate dreaming. Such lesions usually leave the patient unconscious. Indirect clinicoanatomical methods may, however, be used to refute the hypothesis by demonstrating that dreaming is eliminated in individuals with forebrain lesions who have a completely intact brain stem (Solms, 2000).

1.1.6 The role of forebrain mechanisms in dream generation.

The phenomenon of subjective loss of dreaming with forebrain lesions is not a new observation. Wilbrand (1892/1996) reported the first case in 1887 – his patient, who suffered a bilateral occipitotemporal thrombosis, stopped dreaming almost completely. There are a further 109 published cases (including some in which the sleep cycle was evaluated) of cessation or near cessation of dreaming in individuals with focal forebrain lesions (Solms, 2000). Considering the widely accepted notion that REM sleep is the equivalent of dreaming, this lack of evidence for a correlation between dreaming and REM sleep is important. If the pontine brainstem is not the primary causal generator of dreaming then what causes humans to dream?

In the abovementioned cases lesions were present in two forebrain regions – a) the transitional zone between the occipital, temporal, and parietal cortex, at the back of the forebrain, in or near the region of the parieto-temporo-occipital (PTO) junction; and b) in the white matter surrounding the frontal horns of the lateral ventricles, that is, the ventro-mesial quadrant of the frontal lobe. The latter area corresponds with the region that was

targeted in modified frontal leucotomy which, in several large series resulted in a 70 – 90% occurrence of complete or nearly complete loss of dreaming (Frank, 1946, 1950; Jus, Jus, Villeneuve, Pires, Lachane, Fortier & Villeneuve, 1973; Partridge, 1950, Piehler, 1950; Schindler, 1953).

Why should dreaming be lost with lesions in the ventromesial quadrant of the frontal lobes? Solms (2000) proposed that REM sleep and dreaming are controlled by different brain mechanisms based on the discussed observation that REM sleep and dreaming are doubly dissociable states. He suggests that the mesolimbic and mesocortical dopaminergic systems play a role in dream activation.

1.2 The Dopaminergic Hypothesis

The ventromesial quadrant of the frontal lobes contains a considerable number of fibres linking frontal and limbic structures with dopaminergic cells in the ventral tegmentum. The source cells for the mesolimbic and mesocortical dopaminergic systems are situated in the ventral tegmental area of Tsai which ascend through the forebrain bundles of the lateral hypothalamus via the basal forebrain areas. They primarily terminate on the nucleus accumbens in the ventral striatum and also on the amygdala, anterior cingulate gyrus, and frontal cortex (Solms, 2000).

This system, whose circuits bring about goal-seeking behaviours and self-satisfying interactions with the world, has been described as the “seeking” or “wanting” command system of the brain (Berridge, (in press); Panksepp, 1985, 1998). This system is also thought to have been primarily targeted in prefrontal leucotomies (Panksepp, 1985).

Anti-psychotic medications, which block mesocortical-mesolimbic dopaminergic activity, also target this system (Role & Kelly, 1991) and loss of interactive interest in the world is one of the main psychological effects of the drugs (Lehmann & Hanrahan, 1954; Panksepp, 1985). It is for this reason that anti-psychotic drugs are sometime described as ‘chemical leucotomies’ (Breggin, 1980; Panksepp, 1985). This site is also thought to be principally affected by many stimulants, for example, cocaine and amphetamines (Role &

Kelly, 1991) and the positive symptoms of schizophrenia are widely linked with overactivity of this system. These symptoms can also be induced by L-dopa, amphetamines and cocaine (Bird, 1990; Kandel, 1991; Panksepp, 1998). When this system is damaged it results in disorders typically marked by reduced interest, imagination, initiation and ability to plan ahead (Panksepp, 1985) with lack of initiative (adynamia) being a regular side effect of orbitomesial prefrontal leucotomy (Brown, 1985).

There are several factors that suggest that dreaming is generated by this mesocortical-mesolimbic dopamine system. a) Following transection of the forebrain component of this circuit, dreaming ceases completely without any effect on REM sleep (Frank, 1946, 1950; Jus et al. 1973; Partridge, 1950; Piehler, 1950; Schindler, 1953; Solms, 1997). b) Adynamia is a frequent side effect of the surgical transection of this circuit and characteristically accompanies loss of dreaming after deep frontal lesions. It also (statistically) distinguishes between dreaming and non-dreaming patients with such lesions (Solms, 1997). c) Disproportionate and unusually vivid dreaming and nightmares as well as positive psychotic symptoms appear when this system is chemically activated by agents such as L-dopa (Nausieda, Weiner, Kaplan, Weber & Klawans, 1982), without affecting REM sleep (Hartman, Russ, Oldfield, Flake & Skoff, 1980). d) Blocking activity in this circuit with drugs like haloperidol results in the inhibition of excessive, unusually frequent and vivid dreaming (Sacks, 1987, 1990, 1991) and other psychotic symptoms (Solms, 2000). e) Bartholini (1976) found that women treated with sulpiride (a DA blocking agent that acts mainly on mesolimbic DA neurons) experienced a marked decrease in sexual and aggressive dream content as well as a decrease in the duration of their dreams.

2. Rationale

2.1 Dopamine

There are four major dopamine systems in the brain:

1. Extending from the hypothalamic nuclei to the median eminence. In this location dopamine inhibits the release of prolactin from the pituitary gland.

2. Extending from the substantia nigra to the striatum (nigrostriatal system). This system is associated with motor activity of the basal ganglia and is the system that is most affected by PD.
3. The mesolimbic system arises from cells in the ventral tegmental area of the ventral striatum and extends to the nucleus accumbens.
4. The mesocortical system originates from cells in the ventral tegmental area and projects to the limbic and the prefrontal cortex (Clark & Boutros, 1999). Dreams are affected by the latter two systems.

PD patients were chosen for this study because of the loss of dopaminergic cells that occur with the disease. PD is traditionally associated with degeneration of the nigrostriatal dopamine system (substantia nigra pars compacta) but recent studies show that the disease affects dopamine systems in a more widespread fashion than was previously thought. Loss of cells occurs in the pigmented substantia nigra zona compacta, which projects to the striatum. Pathology also occurs in the ventral tegmentum, the locus coeruleus, the serotonergic raphe nucleus, and the nucleus basalis of Meynert (Bradshaw & Mattingley, 1995). In the ventral tegmentum area, dopaminergic neurons are affected with deficits in the lateral hypothalamus, amygdala, and the entorhinal, cingulate, hippocampal, and frontal cortices (Gupta, 1993). Dopamine containing cell groups such as the ventral tegmental area and the peri- and retrorubral region are affected albeit less so than the substantia nigra (Agid, Ruberg, Javoy-Agid, Hirsch, Raisman-Vozaris, Vyas, Faucheux, Michel, Kastner, Blanchard, Damier, Villares & Zhang, 1993).

Theory and research at this point suggests that a very specific group of forebrain structures are involved in dreaming including the anterior and lateral hypothalamic areas, amygdaloid complex, septal-ventral striatal areas; and the infralimbic, prelimbic, orbitofrontal anterior cingulate, entorhinal insular, and occipitotemporal cortical areas (Braun, Balkin, Wesenten, Carson, Varga, Baldwin, Selbie, Belenky & Herscovitch, 1997; Maquet, Peters, Aerts, Delfiore, Degueldre, Luxon & Franck, 1996; Nofzinger, Mintun, Wiseman, Kupfer & Moore, 1997). It is also of importance to note that the length of dreams as well as the frequency and vivacity of dreams are increased with the

administration of dopaminergic agents, without affecting the frequency, intensity and duration of REM sleep in a similar manner (Hartman, et al., 1980).

Analysis of the major metabolite of dopamine, homovanillic acid, in living patients, and bioassay of the brains of deceased parkinsonian patients have demonstrated that brain dopamine is reduced by over 90 percent and often to imperceptible amounts. Thus, PD can be equated with a lack of dopamine (Kolb & Whishaw, 1980) which, in turn, we hypothesize should lead to less frequent and more impoverished dreaming.

2.2 Dream research in Parkinsonian patients

Sandyk, (1997) reports complete cessation of dreaming with the onset of PD. Chipolli, Bolzani, Massetani, Murri & Muratorio (1992, p. 522) found that the impairment of cognitive functioning due to PD did not result in an all or nothing effect but that there were quantitative differences in dreams of PD patients (on L-dopa with peripheral dopadecarboxylase inhibitors (DI)) in relation to the outputs of the “higher-level processes of dream production, reflected in the sequential and structural characteristics of such patients’ dreams”. Both Chipolli et al. (1992) and Massetani, Luchetti, Piccini, Bianchi, Maestrini and Muratorio (1986) found a higher dream recall rate in subjects on chronic L-dopa + DI and found evidence of the unusual dream phenomena (qualitatively vivid, well organised and coherent, temporally condensed, seemingly real, and often affectively neutral) as described by Sharf et al. (1978). Massetani et al. (1986, p. 69) also found that “The Parkinsonian patient’s dreams proved well-organised from a narrative point of view, closely linked to the subject’s personal experience, often involving family environment, generally lacking bizarre elements and affectively neutral”. According to Hall and Van de Castle’s scale Parkinsonian dreams differ to the normal population in the content of activity and emotivity in that emotional content and emotivity was scarce (Massetani et al. 1986).

Little attention has been paid to dream organization and dream recall in PD patients with the majority of studies focusing on the effect of L-dopa on sleep patterns, hallucinations and altered dream phenomena. The literature indicates a 30-48% prevalence of altered dream phenomenon in patients on treatment (Cipolli et al., 1992;

Goetz, Leurgans, Pappert, Raman & Stemer, 2001; Pappert, Christopher, Goetz, Prancie, Niederman, Raman & Leurgans, 1999; Scharf et al. 1997). None of the studies addressed dream phenomena before and after the onset of PD and the research only reports a *return of dreams* after the start of therapy (Kales, Ansel, Markham, Scharf, & Tan, 1971; Kendel, Beck, Wita, Hohneck, & Simmerman, 1972; Nausieda, 1987; Sandyk, 1997).

The proposed study will focus on the subjective dream experience of PD sufferers assessing the nature of their current dream experiences, how it differs to premorbid dream experience, and lastly the relationship between the frequency and intensity of dreams and the duration of PD.

3. Method

3.1. Sample

Twenty Parkinsonian patients (10 men and 10 women with a mean age of 68) were drawn from two private neurological practices according to the following criteria:

a) The presence of idiopathic PD established by means of a neurological examination, symptoms including bradykinesia, rigidity, resting tremor, and impaired postural reflexes. Subjects were not assessed according to Hoehn and Yahr's (1967) scale.

b) The absence of severe dementia and a history of previous functional psychosis. Subjects with a history of nonpsychotic psychiatric disorders or a family history of psychotic mental illness were not eliminated.

c) There was no control for current treatment for Parkinson's disease. Medications include: Carbilev, Pergolite, Triapexola, Artane, Levadopa, Selegiline, Permax, Symmetrel, Sinemet, Medopar and Pexola.

A control group consisting of 20 individuals with diabetic mellitus (DM) matched on age and gender was drawn from the diabetic ward at Groote Schuur Hospital.

All participants were fluent in English. Duration of PD since the first diagnosis ranged from 6 months to 14 years. Other illnesses: 1 PD patient reported depression that was at the time being treated with Effexor. Neither the depression nor the medication affected his dreaming. Other illnesses reported were chronic lower back pain, thyroid, psoriasis, polymyalgia rheumatica, high blood pressure, familial hypertriglyceridemia type 4, diverticulitis, hernia, spastic colon, prostate, hypertension and migraine.

3.2. Ethical Considerations

The research was conducted with ethical oversight by the Department of Psychology Ethics Committee and the Groote Schuur Ethics Committee. Participation was voluntary. Informed written consent was obtained from participants prior to questionnaire administration. To ensure confidentiality each participant was allocated a number that appeared on their questionnaire for identification. The list containing participant's names and identification numbers were kept separately from the questionnaires. All information was treated as strictly confidential.

3.3. Instruments

Self report questionnaires were administered to the participants. Survey instruments include: a) a Demographic Questionnaire, b) a Most Recent Dream report (Calvin S. Hall and Robert Van de Castle in Domhoff, 1966), and c) The Dream Interview (Solms, 1997). Questionnaires were formatted so as to require the minimum of writing to compensate for writing difficulties often experienced in PD. See Appendix A for Questionnaires.

Demographic Questionnaire: The demographic questionnaire includes age, gender, home language, level of education, date of onset of symptoms, date of diagnosis of disease, medication and dosage, other medication and illness.

Most recent dream report: The Hall/Van de Castle system instruction (which is empirical, objective, and comprehensive) was used.

“Please describe the dream exactly and as fully as you can remember it. Your report should contain, whenever possible, a description of the setting of the dream, whether it was familiar to you or not; a description of the people, their sex, age, and relationship to you; and a description of any animals that appeared in the dream. If possible, describe your feelings during the dream and whether it was pleasant or unpleasant. Be sure to tell exactly what happened during the dream to you and the other characters. Please use the space provided and additional sheets if necessary” (Domhoff, 1996).

Replication of normative findings on dream reports in the Hall/Van de Castle system provide a comparison point for studies of dream content globally and has been used in many different countries. Over 10 000 dream reports using this system have given consistent gender, developmental, and cross-cultural differences. Core findings are also stable across gender lines and cultural boundaries (Domhoff, 1997).

The Dream Interview: The dream interview is based on a questionnaire developed by Mark Solms (1997) to assess whether or not (361) patients dreams have changed since the onset of their neurological illness, and if so, how the dreams have changed. The questionnaire approaches the neuropsychology of dreams from a clinical-descriptive viewpoint as opposed to a psychometric test score. The questionnaire thus evaluates patterns of change rather than quantitative deviation from a statistical measure of normality (Solms, 1997). Variables assessed are: sleep experience; presence of dreams; dream recall; narrative complexity of dreams; emotional quality of dreams; recurring nightmares; frequency of dreams; visual imagery of dreams; the ability to distinguish between dreams and real experiences; the length of dreams; dream content; and the influence of medication on dreams. Neither validity nor reliability of the scale has been tested.

3.4. Procedure

PD Patients were contacted by phone or letter by the respective neurologists informing them of the study, what it encompasses and asking if they might be interested in participating in the study. They were notified that the researcher will be contacting them

telephonically during the next week to give them more information on the study and to assess their willingness to participate.

On telephonic contact the researcher clarified all questions regarding the study and informed the patients what was required from them in participation. They were given a choice of personal delivery and assistance in completing the questionnaire; having the questionnaires mailed to them; or having it emailed to them. Mailed questionnaires included a stamped self-addressed return envelope. The researcher made it clear to all subjects that she was available to them at all times, both telephonically and/or for a personal visit. Participants were encouraged to ask assistance from the researcher, a friend, or family member to complete the form if they had difficulty in writing so as to prevent short and incomplete dream reports. The importance of a thorough and comprehensive dream report was stressed.

DM out-patients were informed of the study by the head nurse in the ward after which they were approached by the researcher.

3.5. Data recording and capturing:

Most Recent Dream report: Three blind raters assessed the Most Recent Dream reports according to the following categories: element count; emotional quality; and narrative complexity. The following guidelines concerning each category were given to the raters:

Element count

Each new item, place or action in the dream constitutes an element. Once it has been scored it should not be scored again, e.g.

Being chased by a man / all over a house/, down corridors, into rooms and **/hiding in places/ so he won't find me.** Then **escaping out of the window/ onto the roof/ and into the garden.** When I woke up I was really afraid. (7 elements)

- In this instance corridors and rooms are covered by 'all over house' and are not counted as new elements.

- The subject's personal elaboration on the dream is not considered as part of the dream e.g. 'I was really afraid' is not part of the dream description but a subjective feedback on the emotional quality of the dream.
- Any reference to how things are in reality in relation to the dream is not a dream element e.g. **I am in a house with my sister (eldest)** who, in reality I had an argument with. **We were chatting/ when suddenly I hit her...** (3 elements). E.g. **I returned to an old friend's house** whom I worked with for one year. At first **it was the wrong house** (2 elements).
- If the subject mentions a book, movie or workplace and later mentions the title of the book/movie/workplace it should not be scored as a new element, but be considered as one element.
- In depth descriptions of an object e.g. '...a R5 coin, it was hard shiny and silver' are classed together with the original element, i.e. the R5 (1 element).

Emotional quality

Is the dream pleasant, unpleasant or neutral? Often the dreamer her/himself indicates the emotional quality of the dream. In such an event the dream should be rated as perceived by the dreamer. In the absence of that the rater should interpret the dream according to the positive and negative emotions in the dream.

Negative emotions: anger, apprehension, sadness, and confusion

Positive emotions: varying degrees of happiness.

Neutral: The absence of emotion in the dream.

With (a)1-3 = pleasant; (b)4 = neutral; (c)5-7 = unpleasant. Due to rater difficulties these were classed into categories a, b, and c.

Narrative complexity

- 1) Simple, straightforward, banal and mundane.
- 2) Bizarre, incredible, convoluted and strange.

The raters were initially given a small sample of dreams for training purposes. This sample of dreams was, as suggested by Domhoff (1996), chosen for their difficult coding decisions in order to highlight potential problem areas for the raters. Some ambiguities about coding will always exist in dream content studies and reports of a small percentage of the entire sample are generally responsible for the ambiguities. Rater agreement on most dreams is very high (Domhoff, 1996). Problems were discussed by the raters and resolved where possible.

Initially the percentage of perfect agreement for categories 'emotional quality' and 'narrative complexity' was 75%. This was mainly due to difficulties in rating the emotional quality of dreams on a likert type scale. Good inter-rater reliability is only possible when one limits the categories of emotional states and does not attempt to differentiate levels of intensity (Domhoff, 1996). Once the scale was changed from measurement to categorical data into categories of pleasant, neutral and unpleasant the percentage of perfect agreement rose to 92%.

The average correlation for element count between raters throughout the training session was $r = 0.84$. In the final exercise the correlation for element count between raters was calculated at $r = 0.84$. This score increased to $r = 0.93$ when the results of a particularly difficult dream was removed from the pool.

The Dream Interview: Data was captured in Excel and analysed using the Statistica statistics package.

3.6. Data Analysis

Most recent dream report: Chi-square and correlation was used for the Most Recent Dream Report analysis.

Demographic Questionnaire and The Dream Interview: A classical 2×2 chi-square framework was used to compare the two groups. Due to the small sample size and the rarity of some of the phenomena under study multivariate techniques could not be applied

meaningfully and thus clinical description was relied on to supplement the formal statistical analyses.

4. Results

4.1. Dream presence (N=40):

Of the 20 PD subjects three reported that they do not dream at night; two subjects were uncertain if they dream at night; and fifteen confirmed the presence of dreams. All three subjects who do not dream at night cited that 'I do not know whether they dream or not but that I cannot recall my dreams'. One reported that dream cessation occurred *after* the onset of PD and the other two reported that dream cessation occurred prior to the *diagnosis* of the disease. The two subjects who were unsure if they dreamt stated that they think that they do dream but that they can't remember their dreams. Three subjects from the control group, the DM group, reported cessation of dreaming citing 'I do not know whether I dream or not but I can't recall my dreams' when asked why they think that they do not dream. None connected the cessation of dreaming to the onset of diabetes.

	Yes	No	Unsure
PD	15	3 (1)	2 (2)
DM	17	3 (1)	-

(1) I do not know whether I dream or not but I can't recall my dreams.

(2) I dream but I can't remember my dreams.

Table 1. Presence of dreaming in PD subjects and DM subjects

4.2. The dream questionnaire:

Statistically significant results are discussed first followed by statistically insignificant but clinically noteworthy results. See table 2 for the main features of the PD sample in the dream questionnaire.

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Table 2: Main features of the PD sample in the dream questionnaire (N=20):

<i>Category</i>	<i>Classification</i>			
	<i>Excesses</i>	<i>Deficits</i>	<i>Normality</i>	<i>Normality</i>
Sleep	Better	Disrupted	Unaffected	Unsure
Dream at night	Yes	No	-	Unsure
Dream-recall	Better	Worse	Unchanged	Unsure
Narrative complexity	More simple b	Less simple	Unchanged	Unsure
Emotional quality*	More pleasant a	More unpleasant	Unchanged	Unsure
Recurring nightmares	Present	Absent	-	Unsure
Dream frequency	More	Less	Unchanged	Unsure
Visual imagery	Changed	-	Unchanged	-
Dream imagery	More vivid	Less vivid	Unchanged	Unsure
Reality confusion	Yes	No	-	-
Dream duration*	Longer	Shorter a	Unchanged	Unsure
Dream content	Recent	Remote	-	Unsure
Change in content	Yes b	No	-	Unsure
Medication	Yes	No	-	-
Day-time hallucinations	Yes	No	-	-

* Statistically significant at .05 level

a: change in this direction

b: statistically insignificant but noteworthy

4.2.1. Length of dreams:

The length of dreams in the PD group was significantly more varied than dream length in the DM group, ($\chi^2(3) = 9.11, p < .05$). In the PD group six participants reported that their dreams were unchanged; five reported shorter dreams; three reported longer dreams; and one was unsure. In the DM group fourteen subjects reported that their dreams were unchanged; and three that their dreams were longer since the onset of diabetes.

	Longer	Shorter	Unchanged	Unsure
PD	3	5	6	1
DM	3	6	14	0

Table 3: Location of the source of the significant difference in the length of dreams between PD and DM groups.

4.2.2. Emotional quality of dreams:

The PD group reported a change in the emotional quality of their dreams, ($\chi^2(3) = 8.87, p < .05$). One participant reported that dreams were more unpleasant than before the onset of PD; six reported that their dreams were more pleasant than before; and seven reported no change. In group 2 two participants indicated that dreams were more unpleasant than before; fourteen indicated unchanged dreams; and one was unsure of any change. No subjects in the DM group indicated that dreams were more pleasant.

	More pleasant	More unpleasant	Unchanged	Unsure
PD	6	1	8	0
DM	0	2	14	1

Table 4: Location of the source of the significant difference in the emotional quality of dreams between PD and DM groups.

4.2.3. Narrative complexity:

This variable was very close to significance ($\chi^2(3) = 7.31, p = .06$) and is worth mentioning considering the clinical importance of narrative complexity in dreams in this context. In the PD group four subjects reported that their dreams were **more** simple, straightforward, banal and mundane than before the onset of PD; three reported that their dreams are **less** simple and therefore more bizarre, incredible, convoluted and strange; seven reported that their dreams were unchanged in these respects; and one was unsure. In the DM group fourteen subjects reported no change; two indicated that their dreams are less simple and straight forward, and one was unsure.

	More simple	Less simple	Unchanged	Unsure
PD	4	3	7	1
DM	0	2	14	1

Table 5: Narrative complexity in dreams

4.2.4. Duration of PD in relation to emotional quality and dream duration:

A significant correlation ($p < .05$) was found in the PD sample between duration of illness and emotional quality of dreams, and duration of illness and dream duration. There was not a significant correlation between narrative complexity and duration of illness.

4.3 The Most Recent Dream Report:

Variables found to be significant or nearly significant in the questionnaire were assessed in the Most Recent Dream Report. Variables assessed were emotional quality of dreams, length of dreams, and narrative complexity of dreams.

4.3.1. Emotional quality of dreams:

Emotional quality dreams, as in the questionnaire, resulted in a significant result ($\chi^2(2) = 5.64, p < 0.5$). The direction of change falls into the 'unpleasant' category for the DM group.

	Pleasant	Neutral	Unpleasant
PD	7	20	2
DM	2	4	4

Table 7: Direction of change in emotional quality of dreams in the Most Recent Dream Report.

4.3.2. Length of dreams:

Unlike the self-report questionnaire that found that PD dreams are shorter since the onset of PD no statistically significant difference was found between word count and element count in the Most Recent Dream Report.

5. Discussion

5.1. Dream frequency and presence:

The present hypothesis derived from the dopaminergic hypothesis (Solms, 2000) which predicts that there will be a decrease in the frequency of dreams in PD patients due to the degeneration of the mesolimbic and mesocortical dopaminergic systems is not directly supported by this study. The Dopaminergic Hypothesis (Solms, 2000) suggests that the mesolimbic and mesocortical dopaminergic systems play a role in dream activation. Although this study did not find a statistically significant difference between the PD and DM groups in relation to dream presence it is of clinical interest to note that one PD patient, Mr. S.,¹ connected the cessation of dreaming to the onset of PD while two others reported that this symptom occurred prior to the onset of the *diagnosis* of the disease. In this sample the estimated mean duration of the disease before diagnosis is 1.7 years with 6 years being the longest and 0 the shortest. Although some patients reported immediate diagnosis of the disease it is unlikely due to the progressive nature of the disease. It is therefore possible that PD related dream changes could have occurred in the earlier stages of the disease without the subject connecting the two phenomena. It should also be noted (as discussed in the limitations of the study) that some PD patients declined to participate in the research because they said that they could not recall dreams. None of the DM patients connected cessation of dreaming with their disease. This question thus begs further investigation.

5.2. Length of dreams:

PD patients reported their dreams to be shorter than before. Bartholini's (1976) reported a decrease in dream duration in women treated with sulpiride, a DA blocking agent that acts mainly on mesolimbic DA neurons (there was no statistical difference in dream duration between men and women in the PD sample). Hartman et al. (1980) demonstrated that the length of dreams was increased with the administration of dopaminergic agents without affecting the frequency, intensity and duration of REM

¹ Mr S. was diagnosed with PD in 1989. He reported that he experienced some drug related nightmares for a short period some time ago but the nightmares stopped with the adjustment of his medication.

sleep in a similar manner. In light of these two findings the decrease of dream length in PD patients can be reasonably attributed to a decrease of DA in the brain.

Firstly, the decrease in dream duration possibly indicates that DA plays a role in dream activation in the sense that it is not necessarily needed to be present for dream activation per se but that it is necessary to consolidate the dream process. This does not support Hobson et al's. (2000, p.808) suggestion that the forebrain is responsible for shaping dream plots through dream emotion rather than "playing a secondary role in dream plot instigation". If the forebrain is only involved in dream emotion there is no apparent reason why damage to this system should influence dream length. However, dream activation and continuance of the dream can be plausibly linked to each other – therefore the mechanism that initiates the dream may also perpetuate the dream. The shorter dreams imply that the forebrain activation is not only involved in dream emotion (as will be seen in the discussion on emotional quality of dreams) but that it also plays a role in dream activation. Secondly, the correlation between disease duration and shorter dreams further supports the above argument and supports Solms's (2000) hypothesis that the mesocortical/mesolimbic system plays an active role in dream formation.

The subjective reporting by PD patients - that their dreams are shorter - was not supported by the raters findings on their Most Recent Dream report. One possible reason for this is that memory tends to favour emotionally salient dreams (Bednar, 2000). The overall subjective experience of dreaming might therefore be that the dreams are shorter but in reporting the most recent dream the patient might have elaborated and described dream phenomena in excessive detail. Thus the subjective experience of a shorter dream is not evident in the dream report. This confound can be overcome by collecting morning recall questionnaires of sleep laboratory dream series from all participants as opposed to only the most recent dream.

5. 3. Emotional quality of dreams:

There is a significant difference in the emotional quality of dreams in both the self-report questionnaire and the Most Recent Dream Report although the direction of change between the two differ - PD patients reported the emotional quality of their dreams to be

more pleasant than that of the DM group in the questionnaire while the raters found that the DM dreams were more unpleasant than the PD dreams. This was a surprise finding as the emotional quality of dreams in the PD sample was expected to be impoverished and affectively neutral (Massetani et al., 1986; Solms, 2000).

The questionnaire: Bartholini (1976) found a decrease in sexual and aggressive dream content in women treated with sulpiride (data on men is not available but Domhoff (1996) reports that men tend to have more sexual dreams and are more aggressive in their dreams than women. No significant difference was found between men and women for emotional quality of dreams within the PD sample). The same result can be reasonably expected from the process of DA depletion in PD. Both sexual and aggressive dream content are more often negatively experienced by the dreamer (Domhoff, 1996). A lower frequency of such elements in dreams may therefore make for less intense dreams and a more pleasant dream experience.

Another possible explanation, which is compatible with the above finding, is the interaction of PD medication on a DA depleted brain. When the mesocortical/mesolimbic dopaminergic system is blocked loss of interactive interest in the world is one of the main psychological effects² (Lehmann & Hanrahan, 1994; Panksepp, 1985). Drugs like haloperidol (which cause the inhibition of the transport mechanism of cerebral monoamines, particularly by blocking the impulse transmission in dopaminergic neurons) cause the inhibition of disproportionate, unusually frequent and vivid dreaming (Sacks, 1987, 1990, 1991) and psychotic symptoms (Solms, 2000). When L-dopa is introduced to a healthy system positive symptoms of schizophrenia are induced (Bird, 1990; Kandel, 1991; Panksepp, 1998) as well as unusually vivid dreaming and nightmares (Nausieda, Weiner, Kaplan, Weber & Klawans, 1982). PD patients who are *over medicated* on L-dopa also report the onset of nightmares (as was also reported by Mr. S.) (Cipolli et al., 1992; Goetz, Leurgans, Pappert, Raman & Stemer, 2001; Pappert, Christopher, Goetz, Prancie, Niederman, Raman & Leurgans, 1999; Scharf et al. 1997). All PD patients in this study will have a lower than normal level of DA in their system which, according to the effect of haloperidol should lead to a degree of dream inhibition. But all the PD

² It is also a side effect of PD.

patients are also currently on medication that increases DA in the system, therefore their dreams (if one considers the effects of L-dopa) may be stimulated to some degree. Therefore, if excess DA causes nightmares it is possible that in the near absence of natural DA the same medication in limited amounts may cause dreams to be experienced as more pleasant.³

The Most Recent Dream Report: Dreams from both groups were randomly divided between raters and blindly assessed in relation to each other. DM dreams were rated to be more unpleasant than PD dreams. Therefore, in relation to the PD dreams the DM dreams were unpleasant. Taking the above finding into consideration we argue that this does not indicate an abnormal unpleasantness in DM dreams but suggests a higher amount of pleasantness in the PD dream sample. These findings are therefore consistent.

5.4. Narrative Complexity:

Although not statistically significant it is of clinical interest to note the distribution of the data on this question. Of the 15 PD dreamers 7 reported a change in their dreams with the majority of dream change lying in the simpler category (see table 4). Of the 17 DM dreamers only 2 reported a change in their dreams with both reporting their dreams to be more complex. No DM patients reported their dreams to be simpler than before. This spread of data suggests a trend in the direction of dream narration in PD patients but the present study does not allow us to draw any conclusion as to what that might be. This important dream feature should be further investigated with the use of a larger sample in future studies.

5.5. General discussion:

The hypothesis of this study that PD patients will have less frequent and more impoverished dreaming as suggested by Solms is not fully supported. Nevertheless the results of this study support other aspects of the Dopaminergic hypothesis. Furthermore,

³ Dream stimulation with smaller doses of L-dopa may occur to only such a degree that sexual and aggressive dreams still tend to be more absent than in normal dreamers and therefore dreams seem more pleasant than previously as it might be without the medication.

the results of the present study are incompatible with the AIM model proposed by Hobson (1988).

It should be kept in mind that the DA system involved in PD is not exactly the same system proposed to be involved in dreams but that the two systems overlap. It is possible that PD patients are only mildly affected by the degeneration of the mesolimbic-mesocortical DA system. The decrease in dream duration in the PD sample implies that the forebrain activation of the mesolimbic-mesocortical dopamine system is not only involved in dream emotion but also plays a part in sustaining dreams and possibly dream activation. When the forebrain component of this forebrain circuit is severed dreaming may cease completely without any effect on REM sleep (Frank, 1946, 1950; Jus et al. 1973; Partridge, 1950; Piehler, 1950; Schindler, 1953; Solms, 1997). The decrease in dream length in PD patients and the cessation of dreaming in Mr. S. (whose pontine brainstem is intact) suggests that it is unlikely that the cholinergic brainstem mechanism is the cause of dreaming and that the forebrain is not just a passive participant in the REM state and synthesis of dreams. In light of the differences in dream length and emotion and the correlation between duration of PD and duration of illness and emotional quality of dreams, and duration of illness and dream duration we can not concede that dreaming is not controlled by forebrain mechanisms. The question stands that if dreaming is reliant on the cholinergic activation of an aminergically demodulated forebrain why is there evidence of cessation of dreaming and shortening of dreams in PD patients with intact brainstems (Mr. S; Sandyk, 1997)?

Adynamia, a frequent side effect of PD, often occurs after the surgical transection of the mesocortical-mesolimbic dopamine circuit and typically accompanies loss of dreaming after deep frontal lesions. It also statistically distinguishes between dreaming and non-dreaming patients with such lesions (Solms, 1997). The mesolimbic-mesocortical dopamine system in PD patients is not destroyed but the disease is recognised to reduce the levels of dopamine markedly. In such a degraded system one could therefore expect a change in dream phenomena, for example the shortening of dreams and emotional changes in dreams. One would not necessarily expect to see complete cessation of dreaming although it is possible. This is supported by Hartman et

al. (1980) who demonstrated that the length of dreams were increased with the administration of dopaminergic agents without affecting the frequency, intensity and duration of REM sleep in a similar manner.

The change in emotional quality of dreaming confirms that the DA mesolimbic-mesocortical system plays an important role in dream emotion. Bartholini's (1976) and Hartman's (1980) studies along with these results suggests specifically that the mesocortical-mesolimbic dopaminergic forebrain mechanism influences dream emotion. This motivating system whose circuits bring about goal-seeking behaviours and self-satisfying interactions with the world, (Berridge, (in press); Panksepp, 1985, 1998) and also results in disproportionate and abnormally vivid dreaming and nightmares as well as positive psychotic symptoms when chemically activated (Nausieda, Weiner, Kaplan, Weber & Klawans, 1982) without affecting REM sleep (Hartman, Russ, Oldfield, Flake & Skoff, 1980) appears to affect dream themes, specifically aggressive or sexual dream content (Bartholini, 1976), which in turn determines the degree of pleasantness experienced in the dream (Domhoff, 1996).

5.6. Limitations

PD patients often have difficulty with writing and may find completing a questionnaire stressful. Care was taken to eliminate impoverished data reporting by suggesting to subjects to make use of the researcher, a friend, or a family member to complete the questionnaire in the event that the subject had difficulty in writing. Domhoff (1996) found no difference in the quality of self-written or verbal dream reports. Care was also taken to ensure that participants reported their most recent dream. There is a tendency in subjects, when they can't remember their most recent dream, to report either a recurring dream or a vivid dream experienced during childhood. Although Domhoff (1996) argues that the Most Recent Dream method gives a representative sample of all dreams Chapman and Underwood (2000) and Mealey (2000) pointed out that there is a tendency toward recalling dramatic and therefore more memorable dreams. A further tendency toward bias exists as it is estimated that only 5-10% of all dreams are recalled (Domhoff, 1996). The limitations in this area should be noted although its impact on this study is not

negative in that despite the tendency toward recall of vivid dreams emotional quality and length of dreams are sufficiently affected to be different from a normal population.

Due to the nature of data collection a real danger of sample self selection existed. Several possible participants declined participation because they did not dream. The researcher tried to overcome this hurdle by stressing that all information on dreams, including the cessation of dreaming, was important and of interest to the study.

A cross sectional study with sample size of 40 is considered adequate for the present exploratory study. The next suggested step is a longitudinal study matched on medication and dosage with a larger sample size. Although personality and cognitive variables do not influence dream recall and reporting (Domhoff, 1996) it is suggested that a sample matched on education will be more effective.

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Appendix

1. Demographic Questionnaire
2. Most Recent Dream Report
3. The Dream Interview

University of Cape Town

*For the control group Parkinson's disease was substituted with diabetes.

The following is a series of questions about your current status as a Parkinson's disease (PD) patient. The questionnaire consists of three parts: 1) demographic details and medical history; 2) a most recent dream report; and 3) a dream assessment questionnaire.

The questionnaire is voluntary – you do not have to answer it if you do not want to. It is completely confidential.

Please answer the questionnaires in the order that they appear. Do not read the dream questionnaire before the completion of your most recent dream report.

1. How old are you?.....

2. What gender are you? (please circle the relevant sex)Male Female

3. What is your home language? (please circle your home language)

1. English
2. Afrikaans
3. isiNdebele
4. isiXhosa
5. isiZulu
6. Sepedi
7. Sesotho
8. Setswana
9. siSwati
10. Tshivenda
11. Xitsonga
12. Other

4. What level of education do you have? (please indicate tertiary education as the number of years studied)

a) Secondary _____ b) Tertiary _____

5. When, more or less, was the onset of PD symptoms? (please, if possible, specify the month and year, e.g. March 1997)

6. When was the diagnosis made? (please specify the month and year, e.g. March 1997)

7. What medication are you currently taking for PD?

8. What is the dosage?

9. Are you taking any other medication? (please circle).....Yes No
If yes, what medication are you taking?

10. Are you suffering from any other illnesses besides PD? (please circle)

Yes No

If yes, please indicate what it is?

University of Cape Town

Dream Assessment Questionnaire

The Dream Interview

This questionnaire assesses if there have been any changes in your dreams since the onset of PD as well as specific aspects of your dreams. Please take your time to complete the questionnaire.

1. Since the onset of PD, have you experienced your sleep to be

- a) better b) disrupted c) unaffected d) unsure if sleeping has been affected

2. Do you dream at night?

- a) yes b) no c) unsure

2.1. If you answered *no* to question 2 please indicate why you think you do not dream at night.

- a) I do not dream at all.
b) I dream but can't remember my dreams.
c) I do not know whether I dream or not but I can't recall my dreams.

2.2. Was this the case prior to the onset of PD?

- a) yes b) no

If you answered YES to question 2 please continue with the questionnaire.

3. How has your ability to recall dreams (morning recall) been affected by PD? Has it become

- a) better b) worse c) unchanged d) unsure

4. This question relates to the narrative complexity of your dreams. Are your dreams

- a) **More** simple, straightforward, banal and mundane than before the onset of PD?
- b) **Less** simple, straightforward, banal and mundane than before the onset of PD (i.e., more bizarre, incredible, convoluted and strange)?
- c) **Unchanged** in these respects?
- d) **Unsure**.

5. This question relates to the emotional quality of your dreams. Do you feel that your dreams are

- a) **More pleasant** than before the onset of PD?
- b) **More unpleasant** than before the onset of PD?
- c) **Unchanged** from before?
- d) **Unsure** if there are any changes?

6. This question relates to recurring nightmares. A recurring nightmare is the same nightmare dreamt on several occasions.

Are recurring nightmares

- a) present?
- b) absent?
- c) unsure.

6.1. If you responded to question 6 with *present*, did this also occur before the onset of PD?

- a) yes
- b) no
- c) unsure

7. This question relates to the frequency of your dreaming since the onset PD.

Do you have

- a) **More** frequent dreams
- b) **Less** frequent dreams
- c) **Unchanged** frequency of dreams
- d) **Unsure**

