Waves of the Unconscious: The Neurophysiology of *Dreamlike* Phenomena and Its Implications for the Psychodynamic Model of the Mind

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This paper reviews scientific literature on four subjective states: the dream state, the dreamy state of temporal lobe epilepsy and temporal lobe stimulation, the acute psychotic state, and the psychedelic state. Evidence is cited showing that underlying the emergence of *dreamlike* phenomena in all four states is the occurrence of high-voltage bursts of theta and slow-wave activity (2–8 Hz) in the medial temporal lobes. The medial temporal regions are recognized to play an important role in memory and emotion. In the dream state, medial temporal lobe bursts are tightly correlated with PGO waves. It has been widely speculated that PGO waves are direct neurophysiological correlates of dreaming. On a phenomenological level, the dream state, the dreamy state, the acute psychotic state, and the psychedelic state have all been viewed as conducive to the emergence of unconscious material into consciousness. An argument is made that bursts of electrical activity spreading from the medial temporal lobes to the association cortices are the primary functional correlate of discharging psychical energies, experienced on a subjective level as the emergence of unconscious material into consciousness. The implications of these findings for the scientific legitimacy of the psychodynamic model are discussed.

Keywords: unconscious; psychoanalysis; dissociation; dream; déjà vu; lysergic acid diethylamide

This paper begins with a discussion of the dream state. Most readers will be familiar with the association between rapid eye movement sleep (REM) and dreaming. REM sleep is a tonic sleep stage that contains within it phasic phenomena. Experiments in cats have shown the existence of phasic bursts from the pontine nuclei, passing through the visual nuclei of the thalamus (lateral geniculate bodies), and innervating the occipital cortex during REM sleep. These "PGO" (pontine-geniculate-occipital) waves correlate significantly with the rapid eye movements of REM sleep. Other studies in animals have also shown the existence of phasic bursts in limbic regions (amygdala and hippocampus) during REM sleep. These limbic bursts also correlate with the rapid eye movements of REM sleep. Studies in animals have confirmed that these bursts fire in synchrony with PGO waves, strongly suggesting that they are part of the same system. PGO waves have long been associated with dreaming, although our inability to record from the PGO nuclei during REM sleep has prevented us from confirming this hypothesis in humans.

Some intracranial (depth) recordings have been carried out in humans, and these have shown bursts of activity in brainstem nuclei, thalamic nuclei, and limbic regions during REM sleep. Despite the practical and ethical difficulties associated with recording from subcortical nuclei in humans, it is theoretically important that we discover a way of doing so. One way to tackle this problem is to shift our focus onto subjective states that have been compared with the dream state to see whether they contain any consistent properties. If these states, associated with *dreamlike* subjective phenomena, also show neurophysiological activity similar to that which has been recorded during REM sleep in the human depth EEG studies, then we can feel confident that this phenomena is related to the dream state.

The term "*dreamlike"* refers to phenomenological and neurophysiological properties consistent with those of the dream state. The term "*dreamlike state"* refers to the tonic *dreamlike* state of consciousness; the term "*dreamlike phenomena"* refers to the psychological and neurophysiological processes going on within the tonic state. In addition to the dream state itself, three *dreamlike* states are focused on: (1) *the dreamy state* of

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temporal lobe epilepsy and temporal lobe stimulations, (2) *the acute psychotic* state. and (3) *the psychedelic state.* A number of different *dreamlike* phenomena have been described in these states, of which visual hallucinations are probably the most immediately appealing. In addition to the visual aspect, however, this paper also focuses on a more abstract but theoretically important dimension: *the emergence of unconscious material into conscious awareness*.

Freud's work on the unconscious mind gave birth to a remarkable new psychological paradigm. Regrettably, however, psychoanalysis today occupies a relatively marginalized position within psychology. It is reasonable to suggest that the marginalization of psychoanalysis does not reflect the value of its insights but, rather, the difficulties associated with empirically verifying them. It is felt that the scientific validation of psychoanalytic concepts such as "the unconscious" is achievable. This paper works toward this goal by describing cases where psychodynamic phenomena have been identified together with the simultaneous occurrence of a characteristic neurophysiological activity.

From a classically Freudian perspective, the emergence of unconscious material into consciousness can be understood in relation to "abreaction" i.e., the discharge of psychical energies attached to previously repressed experiences. If we are bold enough to consider Jung's conception of the collective consciousness in connection to his own thoughts on psychical energy, we might also like to understand these discharges of psychical energy as events capable of traversing personal development to evoke archetypal and spiritual experiences related to our species-specific evolutionary development. A classically Freudian analysis of the phenomena is maintained throughout this paper, but this does not mean that Jung's ideas are irrelevant.

On an objective level, the paper presents a thorough review of the neurophysiological data recorded during the emergence of unconscious material in the dream state, dreamy state, acute psychotic state, and psychedelic state. This process reveals that a consistent and characteristic activity occurs in all four states during these unconscious emergences—namely, bursts of theta and slow-wave activity recorded in the medial temporal regions. It is significant that sudden discharges of activity recorded in the medial temporal regions are tightly correlated with PGO waves in sleep; it is likely that they are also a correlate of dreaming. These limbic regions are very much associated with memory and emotion. It is therefore significant that a highly characteristic activity has been sourced to these structures and correlated with the emergence of unconscious material.

The dream state

Background

From the ancient Egyptians to contemporary sleep researchers, dreaming has remained a favorite subject. So much has been written about dreams that one might assume there is little else to be said. When it comes to phenomenology, there may be an element of truth to this glib remark, but as for neurophysiology, there are an unknown number of key processes left to be elucidated, and theoretical disagreements are more colorful than ever. For thousands of years it was believed that dreams emerged from the spirit world or that they contained messages from Gods. It has only been in the last one hundred years or so that dreams have been looked at as a scientific phenomenon. Even before Freud, speculations were being made about dreams and an "unconscious" psychical realm (Abercrombie, 1830; Maury, 1861) but it was Freud who laid down the first truly progressive theory of dreaming (1900a). Freud's often misquoted premise that "the interpretation of dreams is the royal road to a knowledge of the unconscious activities of the mind" (Freud, 1900a) has remained a source of controversy for over one hundred years; and while other hypotheses have since been shown to be fallible, this central tenet has retained much of its integrity. It is a primary argument of this paper that dreams are psychodynamically meaningful and can indeed be used to inform us about the unconscious activities of the mind.

Dream psychology developed out of a long history of dream philosophy; dream physiology, on the other hand, is a relatively young discipline. Since the first known documentation of rapid eye movements in human sleep (Jacobsen, 1938), their subsequent association with dreaming (Aserinsky & Kleitman, 1953), and a characteristic electroencephalographic (EEG) output (Dement & Kleitman, 1957), there has been a dramatic increase in our knowledge of the neurophysiological and neurochemical processes involved in dreaming. Recent years have seen the development of arguments surrounding the psychological interpretation of neurophysiological data (Hobson, 2003; Solms, 2003). In 1977, Hobson and McCarley introduced their "activation-synthesis" model. This model proposed that dreaming is the result of altered brainstem activity initiated by a shift in neuromodulation away from serotonergic and noradrenergic cell firing with a reciprocal disinhibition of ascending cholinergic activity; this shift marks the onset of REM sleep. Hobson and McCarley argued that the qualitative experience of dreaming is a by-product of this ascending choliner-

gic activity. The activation-synthesis model proposed that dreaming is a bottom-up production initiated and governed by the biological processes that trigger REM sleep and is not, as Freud believed, psychologically driven. In his many writings, Hobson has championed his model as the natural successor to Freud's (Hobson, 1988, 1994, 2001, 2003, 2005).

REM = dreaming debate

The activation-synthesis model has received some revision since its original conception almost 30 years ago (Hobson, 1992). On a phenomenological level, Hobson had originally proposed that dreams have "no primary ideational, volitional, or emotional content" (Hobson & McCarley, 1977, p. 1347), a strange claim with very little intuitive appeal. Interestingly, Hobson position has since shown signs of moderation: "I view dreams as privileged communications from one part of myself (call it the unconscious if you will) to another" (Hobson, 2005, p. 83). In the face of mounting documentation of dream reports, qualitatively indistinguishable from those of REM sleep, being found in at least 10% of awakenings from non–rapid eye movement sleep (NREM) (Monroe, Rechtschaffen, Foulkes, & Jensen, 1965; Nielsen, 2003), researchers began to question the REM = dreaming hypothesis (Foulkes, 1962; 1966). To complicate matters, the REM = dreaming debate has recently been added a new dimension following neuropsychological case reports of dream ablation (but not REM ablation) after lesions to either the ventromesial quadrant of the frontal lobes or the parieto-temporo-occipital junction (Solms, 1997, 2003). The main proponent of this approach, Mark Solms, has argued that Hobson's insistence on the central importance of brainstem nuclei for the generation of dreaming must be flawed if dreaming is lost after damage to areas other than the cholinergic brainstem nuclei.

Covert REM

While the brainstem-dream-generator hypothesis continues to be debated, the confusion over NREM dream reports may be nearing a point of resolution. Increasing evidence suggests that "covert REM" occurring during NREM epochs can account for NREM dream reports (Nielsen, 2003). Nielsen defines covert REM as "any episode of NREM sleep for which some REM sleep processes are present, but for which REM sleep cannot be scored with standard criteria" (Nielsen, 2003, p. 69). The present paper embraces Nielsen's covert REM hypothesis and proposes that a neurophysiological component of REM (phasic limbic bursts), undetectable by traditional scalp electrodes, can be found not only in NREM but in waking states as well.

PGO waves

Conventional scalp electrodes are only capable of recording the synchronized oscillations of cortical cells; they reveal little about the electrical activity of underlying subcortical tissue. By the implantation of depth electrodes into the brain tissue of animals and in some cases humans, it has been possible to detect electrical activity during different behavioral states. In the early 1960s it was discovered that phasic "spikes" could be recorded simultaneously from electrodes placed in the pontine brainstem nuclei, lateral geniculate bodies, and occipital cortex of cats as they entered REM sleep; this activity was labeled PGO waves (Bizzi & Brooks, 1963; Jouvet, 1962). The highest rate of occurrence of PGO activity takes place during REM sleep (Michel, Jeannerod, Mouret, Rechtschaffen, & Jouvet, 1964; Mouret, Jeannerod, & Jouvet, 1963). Many researchers have speculated that PGO activity is the neural correlate of dreaming (e.g., Hobson, 2001; Rechtschaffen, Molinari, Wabon, & Wincor, 1970). The major problem with this hypothesis however, is the practical difficulty of recording PGO activity in humans (Hobson, 2001). Aside from early exploratory studies in the 1950s, depth electrode procedures are currently only carried out in humans for therapeutic purposes such as deep-brain stimulation or for the identification of epileptic foci. This obstacle has largely prevented the establishment of PGO in humans. A number of researchers have claimed to have recorded correlates of PGO waves using noninvasive conventional EEG (e.g., Inoue, Saha, & Musha, 1999; McCarley, Winkelman, & Duffy, 1983; Miyauchi, Takino, & Azakami, 1990), but scalp electrodes are generally not sensitive enough to pick up the very specific subcortical activity. This is particularly problematic given that subcortical activity taking place during dynamic behavioral states can appear completely different to cortical activity detected at the scalp (Brazier, 1968; Heath & Mickle, 1960).

Human PGO activity

The most direct evidence of human PGO waves and subcortical phasic events in REM sleep comes from the few depth electrode studies that have also recorded

sleep. Using depth electrodes, Freemon and Walter (1970) recorded the sleep of 8 patients: 5 with temporal lobe epilepsy, 2 with chronic schizophrenia, and 1 with an unspecified brain tumor. Phasic bursts of slow-wave activity (3 Hz) were detected in the midbrain tegmentum during REM sleep both simultaneously with eye movements and immediately preceding them. Very similar bursts of high-voltage slow waves (~3 Hz) in the central tegmentum have been recorded by Wilson and Nashold (1969) in 5 patients: 3 with intractable pain, 1 with epilepsy, and 1 with dystonic syndrome. These slow waves were immediately followed by rapid eye movements of REM sleep. Both Freemon & Walter and Wilson & Nashold reported that, on occasions, PGO-like bursts appeared outside REM sleep. PGO bursts occurring in NREM sleep stages can be considered examples of *covert REM*.

PGO-limbic bursts

Crucially, in addition to brainstem bursts, phasic activities of slow frequency (2–8 Hz) have also been recorded in limbic regions of humans (Cantero et al., 2003; Freemon & Walter, 1970; Giaquinto, 1973; Mann, Simmons, Wilson, Engel, & Bragin, 1997; Salzarulo et al., 1975; Yu, Rayport, Farison, Dennis, & Choi, 1997) and of animals (Calvo & Fernández-Guardiola, 1984; Deboer, Sanford, Ross, & Morrison, 1998; Freemon, McNew, & Adey, 1969; Hodes, Heath, & Hendley, 1952; Jacobs & McGinty, 1971; Stewart & Fox, 1991; White & Jacobs, 1975) during the rapid eye movements of REM sleep. These limbic bursts have been recorded almost exclusively from the medial temporal regions (amygdala and hippocampus) and have been found to oscillate in synchrony with PGO bursts. Synchronous firing suggests that regions are functionally related.

Findings in cats (Calvo & Fernández-Guardiola, 1984; Jacobs & McGinty, 1971; White & Jacobs, 1975), primates (Freemon, McNew, & Adey, 1969; Hodes, Heath, & Hendley, 1952; Stewart & Fox, 1991) and rats (Deboer et al., 1998) support the existence of phasic limbic bursts in REM sleep. Stimulation of the amygdala was found to increase REM sleep in rats (Smith & Miskiman, 1975), to create bursting discharges in rabbits similar to those seen in PGO-wave generation (Silvestri & Kapp, 1998), and to increase the number and density of PGO waves in cats (Calvo et al., 1987). Analysis of the neural activity marker c-fos in the brains of cats sacrificed after 3 hours of prolonged waking, NREM, and REM revealed a positive correlation between levels of c-fos labeling in the pons, septum, hippocampus, cingulate gyrus, amygdala, and nucleus accumbens and the amount of time spent in REM sleep (Sastre, Buda, Lin, & Jouvet, 2000). Sastre and colleagues proposed that the limbic system is a major target of excitatory afferents originating in the pons. In an important study in cats, Calvo and Fernández-Guardiola (1984) recorded phasic potentials occurring during REM sleep in the lateral geniculate bodies, visual cortex, anterior ventral thalamic nucleus, posterior cingulate gyrus, anterior cingulate gyrus, dorsal hippocampus, and basolateral amygdala. Calvo and Fernández-Guardiola proposed that phasic pontine activity during REM sleep propagates the limbic system.

Hippocampal theta and PGO bursts are phase locked

Increased activity in the limbic system during REM sleep is not disputed. Neuroimaging studies in both humans and animals have confirmed that these regions are especially active in REM sleep (Braun et al., 1997; Buchsbaum, Gillin, & Wu, 1989; Lydic et al., 1991; Maquet et al., 1996; Nofzinger, Mintun, Wiseman, Kupfer, & Moore, 1997). What is less certain, however, is the physiological nature of the activity. Mammalian REM sleep is characterized by rhythmic hippocampal theta waves (4–8 Hz) (Vanderwolf, 1969; Vertes & Kocsis, 1997). Hippocampal theta is the largest extracellular synchronous signal that can be recorded in the normal EEG of the mammalian brain (Vertes & Kocsis, 1997). Some researchers have based theories of REM sleep function and dreaming on hippocampal theta activity (Poe, Nitz, McNaughton, & Barnes, 2000; Sandyk, 1998; Winson, 1990), suggesting that hippocampal theta promotes memory consolidation in REM through synaptic plasticity. Researchers have investigated the relationship between hippocampal theta and PGO waves, recording a close temporal synchrony between them (Karashima, Nakao, Katayama, & Honda, 2005; Karashima et al., 2001). Experiments in rats and cats found that "theta wave instantaneously accelerated several hundred milliseconds before the negative peak of the PGO wave in both animals, and was also amplified just before PGO wave occurrence" (Karashima et al., 2005, p. 50). Cells in the central nucleus of the cat amygdala show the same accelerations related to the appearance of PGO waves (Calvo, Simon-Arceo, & Fernandez-Mas, 1996). Data showing a relationship between medial temporal lobe activity and PGO waves are supported by findings from other animal studies showing positive correlations between

hippocampal theta-wave frequency and PGO-wave density in cats (Mushiake, Kodama, Shima, Yamamoto, & Nakahama, 1988; Rowe et al., 1999) and rats (Karashima et al., 2005).

A common mechanism underlying the generation of PGO and theta waves has been proposed (Karashima et al., 2005), although it is unclear whether PGO waves drive theta generators (e.g., medial septum/vertical limb of the diagonal band of Broca, mamillary bodies, and entorhinal cortex: Vertes & Kocsis, 1997), or the other way round. It is perhaps most likely that it is a bit of both (Karashima et al., 2005). For the sake of resolving the brainstem-dream-generator/REM = dreaming hypothesis, it is important that we establish whether the pontine nuclei drive these PGO-related limbic bursts or whether the *discharge of electrical energy* from the limbic system occurs independently of brainstem control. Even if it is found that the cholinergic brainstem nuclei do initiate the medial temporal bursts that contribute to dreaming, then contrary to Hobson's activation-synthesis model, this would by no means imply that the psychological content of dreams is arbitrary and without psychodynamic significance.

The neurochemistry of PGO-limbic bursts

In cats, injections of the cholinergic agonist carbachol into the pontine area induced an immediate onset of state-independent PGO waves, with an additional increase in PGO activity during REM for a period of 10–12 days (Datta, Calvo, Quattrochi, & Hobson, 1992). More interestingly, however, microinjections of the same drug into the central nucleus of the amygdala has been found to induce prolonged enhancements of REM sleep (5 days) as well as the promotion of PGO waves occurring in NREM epochs (Calvo, Simon-Arceo, & Fernandez-Mas, 1996). Thus, it seems that cholinergic limbic (amygdala) stimulation can activate PGO activity directly without it being first initiated at the brainstem.

The PGO system appears to contain a significant cholinergic dimension. Injection of the cholinergic agonist carbachol stimulates an increase in REM and PGO activity when injected into the brainstem and the amygdala (Calvo, Simon-Arceo, & Fernandez-Mas, 1996; Datta, Calvo, Quattrochi, & Hobson, 1991). Acetylcholine release in the hippocampus of rats and cats is highest during REM sleep and is positively correlated with both frequency and power of theta waves (Keita, Frankel-Kohn, Bertrand, Lecanu, & Monmaur, 2000; Marrosu et al., 1995). However, the induction of REM sleep and PGO activity is likely not to be purely cholinergically driven. Simon-Arceo, Ramirez-Salado, and Calvo (2003) have shown that injections of vasoactive intestinal peptide (VIP) into the amygdala of cats cause an increase in REM sleep duration and intensity for a prolonged period as well as an increase in PGO activity not only during REM but also in NREM epochs (covert REM). Injections of saline did not have any effect. Additionally, researchers carrying out precise lesion studies in rats claim to have discovered glutamatergic REM-on cells in mesopontine tegmentum (Lu, Sherman, Devor, & Saper, 2006). It also seems apparent that GABA (γ-aminobutyrate) mechanisms play an essential role in the control of limbic theta oscillations (Vertes & Kocsis, 1997). Further experiments are required to establish more clearly the triggers involved in the emergence of limbic theta bursts in REM sleep as well as the relationship between PGO activity and limbic slow waves. It important to clarify whether the cholinergic pontine nuclei are necessary for the PGO-limbic bursts or whether other sources are also important, such as the major theta generators already identified in the septal region and hippocampus of animals (Vertes & Kocsis, 1997).

What are the implications of these findings?

Even though the effect came from an intervention (carbachol injected into the amygdala), the finding that PGO activity can be driven by regions higher up from the brainstem (Calvo, Simon-Arceo, & Fernandez-Mas, 1996) has implications for the activation-synthesis model of Hobson and McCarley (1977) and Solms's (2003) hypothesis that dreaming and REM sleep are controlled by different brain mechanisms. Hobson is likely to be correct in insisting on the importance of brainstem events for shifting the brain into a *dream mode*; however, his argument that ascending cholinergic activity is coalesced in an arbitrary fashion, with no psychodynamic relevance, never truly seemed to follow from the data. The remainder of this section makes the case that ascending cholinergic activity is only one component of dreaming and that a discharge of energy from the limbic regions to the association cortices is another very significant piece of the jigsaw.

It seems likely that the REM = dreaming hypothesis is destined to be surpassed in coming years by a more detailed *PGO-limbic bursts = dreaming* model, but even this model would be too crude and simplistic. We should resist the temptation to assign to dreaming one key component but, rather, understand it within the context of a number of ongoing processes. Equally, however, we should be open to the reality that some

components play a much more significant role than others.

It will be made clear in subsequent sections that the idea of a discharge of energy from the medial temporal regions to the association cortices has interesting implications for the psychodynamic model of dreaming and perhaps for the psychodynamic model itself. For the time being, however, it is important to lay down in detail the various evidence supporting the role of the medial temporal regions in dreaming.

Human PGO-limbic bursts

As already mentioned, ethical considerations prevent us from recording electrical activity in the human pontine nuclei, geniculate bodies, and occipital cortex simultaneously. Freemon and Walter (1970) recorded phasic bursts in brainstem nuclei, but the surgical procedures necessary for recording from such regions are risky and therefore rarely carried out. Depth recordings from the medial temporal regions occur more frequently, however, due to surgical procedures for the detection of epileptic foci. As already discussed, bursts of activity in the medial temporal regions are tightly correlated with PGO bursts. These limbic bursts therefore serve not only as potential correlates of PGO activity, but also as potential correlates of dreaming.

Bursts of theta and slow-wave activity have been detected in the REM sleep of humans on a number of occasions (Bodizs et al., 2001; Cantero et al., 2003; Fernández-Mendoza et al., 2006; Giaquinto, 1973; Mann et al., 1997; Ravagnati, Halgren, Babb, & Crandall, 1979; Salzarulo, 1975; Yu et al., 1997;). However, the results of these studies are confounded by the fact that most of the participants suffered from temporal lobe epilepsy. Despite the epileptic status of most of the participants, several of the researchers carrying out the recordings have maintained that their findings are representative of normal brain activity, arguing that (1) electrodes overlying known seizure-onset zones were excluded from analyses, (2) recordings from the unaffected hemisphere revealed consistent activity, (3) activity was very much determined by whether the individual was awake, in NREM, or in REM and did not correlate with seizure activity, (4) all patients showed REM sleep bursts of hippocampal theta, including individuals whose epileptic focus was not actually in the hippocampus, and (5) anticonvulsant medication is not associated with phasic limbic theta in animals or humans.

In a recent study, recording from foramen ovale electrodes in 21 patients with epilepsy, Bodizs et al. (2001) discovered bilateral delta activity (1.5–3 Hz) in the hippocampus during REM sleep epochs; a "clear association" was reported between 1.5–3 Hz oscillations and rapid eye movements in some patients. It is important to remember that not all PGO spikes have an accompanying eye movement (Ioannides et al., 2004), and this may explain why this presumed correlate of PGO was not picked up in all the patients in this study.

Human PGO-limbic bursts are not constrained to REM sleep

Recording from subdural (below the dura but not within brain tissue) electrodes on the temporal lobes and depth electrodes implanted in the cortex as well as the hippocampus of patients with drug-resistant epilepsy, Cantero et al. (2003) were able to detect phasic bursts of theta (5 Hz) in the hippocampus during REM sleep. Of the patients with electrode contacts in the hippocampus, a mean of 35 bursts were detected in NREM, compared to 896 seen in REM. If, as seems likely, limbic theta and slow-wave activity is related to PGO and dreaming, then consistent with Nielsen's covert REM hypothesis, those relatively few hippocampal bursts occurring during NREM may well account for the small percentage of REM-like dream reports that are found in NREM awakenings.

Furthermore, consistent with Nielsen's speculation that covert REM occurs frequently at sleep onset, NREM theta was particularly pronounced during transitions from sleep to waking, a finding that is backed by evidence of REM-like hippocampal slow-wave (1.5–3 Hz) activity occurring during wake–sleep transitions (Bodizs, Sverteczki, Lazar, & Halasz, 2005). Several studies have found that sleep-onset dream reports contain significantly more references to vivid hallucinatory material than do other NREM dream reports (Cicogna, Cavallero, & Bosinelli, 1991; Foulkes, 1966; Foulkes & Vogel, 1965; Lehmann, Grass, & Meier, 1995; Vogel, 1978; 1991) and this is the period associated with hypnagogic hallucinations.

Human PGO-limbic bursts in healthy participants

Using the dipole tracing, a noninvasive procedure used to estimate the spatial and temporal location of neuronal groups in the brain, Inoué, Saha, and Musha (1999)

claimed to have detected a significant theta component (6–7 Hz) in thalamic and hippocampal regions of healthy participants during REM sleep epochs. Inoué and colleagues also reported "frequent translocations of dipole positions observed from the thalamus to the hippocampus and/or the cortex or vice versa in synchrony with rapid eye movements" (p. 217). They also report phasic translocations from the pons, thalamus, and occipital cortex occurring within a very short period of less than 1 second during REM sleep, believing this to be direct evidence for the existence of PGO-like spiking activity in human REM sleep.

Magnetoencephalography (MEG) is an imaging technique used to measure changes in the brain's magnetic fields associated with neural oscillations. After initial theoretical objections (Guy et al., 1993; Sutherling, Crandall, Cahan, & Barth, 1988), it has become increasingly evident that MEG is capable of detecting activity in the human hippocampus (Hanlon et al., 2003; Nishitani, Nagamine, Fujiwara, Yazawa, & Shibasaki, 1998**;** Nishitani et al., 1999; Stephen, Ranken, Aine, Weisend, & Shih, 2005; Tesche, 1997; Tesche & Karhu, 2000; Tesche, Karhu, & Tissari, 1996). Using MEG to record the wake and sleep activity from 3 normal healthy volunteers not on any medication, Ioannides et al. (2004) discovered an orbitofrontal–amygdalo–parahippocampal–pontine sequence occurring in the last 100 ms leading up to REM saccades in REM sleep. Ioannides and colleagues propose that the frontal cortical–limbic–pontine sequence may be related to PGO activity and the emotional component of dreaming. The inclusion of the orbitofrontal cortex in this sequence will be of interest to Solms (1997, 2003), who has hypothesized that the dopaminergic, mesolimbic, "seeking system" that innervates the frontal lobes must be fully functional in order for dreaming to occur.

Finally, some very interesting preliminary findings were announced at the recent European Sleep Research Society meeting in Austria. This study showed the existence of PGO-like spiking activity in the REM sleep of nonepileptic participants (Fernández-Mendoza et al., 2006). Using depth (tetra) electrodes implanted for treatment purposes in the subthalamic nucleus of 53 Parkinson disease patients, phasic 2- to 3-Hz "subthalamic sharp wave bursts" lasting 2–8 seconds were recorded bilaterally preceding the eye saccades of REM sleep. This activity, described as "PGO-like bursts," could not be detected by scalp electrodes. Consistent with Nielsen's covert REM hypothesis, the few PGO-like bursts that occurred outside REM took place during NREM epochs preceding the onset of REM.

Testing the PGO-limbic burst = dreaming hypothesis

The increasing number of deep-brain stimulation procedures, not only in Parkinson disease patients but also patients with treatment-resistant depression [where electrodes are implanted in the (limbic) subgenual cingulate region], will provide more opportunities for the study of subcortical activity during REM sleep. Magnetoencephalography may also provide possibilities for examining the correlates of PGO and dreaming. Studies capable of recording PGO-limbic bursts during sleep should consider waking up participants whenever this activity occurs and asking them whether they were dreaming. It is vitally important for the PGO-limbic bursts = dreaming hypothesis that these awakening studies are carried out.

Most authorities would agree that a human equivalent of PGO waves probably does exist, but practical and ethical circumstances prevent us from establishing this beyond doubt. The limbic component of PGO activity is less widely appreciated than the pontine, thalamic, and occipital aspects. Some may even question whether it is necessary to concern ourselves with it. Those interested in the purely visual component of dreaming will see the incorporation of limbic activity as an unwelcome complication, but we cannot deny the evidence supporting its involvement. Functionally, the medial temporal regions are known to be important for the processing of memory and emotion (LeDoux, 1998), and the temporal cortices are known to play a significant role in visual and auditory perception. Patients with temporal lobe loss or damage often find that their dreams become considerably less narrative and lose their visual component (Kerr & Foulkes, 1981; Pena-Casanova, Roig-Rivira, Bermudez, & Tolosa-Sarro, 1985).

It seems likely that during REM and covert REM, bursts of theta and slow-wave activity in the medial temporal lobes oscillate in synchrony (at a slow frequency of less than 9 Hz) with the visual association cortices thus forming the complex imagery of dreams with their rich emotional undertones. One way to assess the plausibility of this theory is to examine states similar to dreaming and see if similar processes are going on in these. In this paper, three *dreamlike* states will be examined: the dreamy state, the acute psychotic state, and the psychedelic state. A wealth of evidence is cited to show that the most consistent neurophysiological correlate of all three *dreamlike* states is the occurrence of sudden and dramatic discharges of electrical activity in the medial temporal lobes.

In the next section, as well as continuing to address neurophysiological data, we shift our attention onto the phenomenology of *dreamlike* states. Subjective aspects of dreaming have not been addressed in any detail in this section. The main reason for this is that the neurophysiological data demand a sustained and detailed discussion. The two main phenomenological facets of dreaming that we look at in the following sections on *dreamlike* states are (1) visual hallucinations and (2) the emergence of unconscious material into consciousness. There is agreement that visual hallucinations occur in dreaming, whereas facilitated access to unconscious mental processes is still much disputed. A simple reason for this may be that people are asleep when dreaming, so real-time subjective reports are not possible. The *dreamlike* states that we are about to describe do occur in waking consciousness. Real-time descriptions of the subjective experience are therefore possible. These first-hand accounts and the interpretations of them are used here to make the case that the emergence of unconscious material does occur in these states and, furthermore, that underlying all these experiences is a consistent neurophysiological activity.

The dreamy state

The dreamy states and déjà vu

The term "dreamy state" dates back to 1876 when it was first used by the British neurologist Hughlings Jackson to refer to the dreamlike aura experienced by some of his temporal lobe epilepsy patients:

> The so-called "intellectual aura" (I call it "dreamy state") is a striking symptom. One kind of it is a "reminiscence"; a feeling many people have when in apparently good health. [Hughlings Jackson, 1888, p. 179]

One of the most common characteristics of the dreamy state is an eerie feeling of familiarity referred to as déjà vu. In *The Psychopathology of Everyday Life* Freud made the following comment on déjà vu:

> It is in my view wrong to call the feeling of having experienced something before an illusion. It is rather that at such moments something is touched on which we have already experienced once before, only we cannot consciously remember it because it has never been conscious. [Freud, 1901b]

Dreamy states are a common feature of temporal lobe epilepsy, occurring most often during the period immediately preceding a seizure. The following is an elaborate account of the gradual emergence of a dreamy state as it progresses into a seizure:

> A complete sequence of recollective thoughts, such as the following: Morning shall surpass, then noon, then we shall have evening; or, for instance, this house was built, it shall be destroyed, and another shall be built and it will be destroyed. Have these thoughts occurred before? As a matter of fact, hasn't this all happened before? Then a deep breath, followed by a rising sensation from the region of the stomach and thorax, all the above occurring very rapidly, possibly less than a quarter of a second. Simultaneously a mental darkness, i.e., a dark figure being elevated, crouched in a threatening or menacing position. The figure seems gesturing with a long sharp implement, possibly an umbrella. Accompanying this is the persistent beseeching desire for water. The above seems to be accompanied by a tenseness. Then a haze follows, as though fleecy clouds were floating by one another. Then a complete loss of consciousness. [Penfield & Perot, 1963, Case 49, p. 660]

Dreamy states induced by temporal lobe stimulation

The dreamy state is not only associated with the aura preceding temporal lobe seizures; it can also be induced by electrical stimulation of the medial temporal regions (hippocampus and amygdala) and temporal cortex (Baldwin, 1960; Barbeau et al., 2005; Bartolomei et al., 2004; Bickford, Mulder, Dodge, Svien, & Rome, 1958; Delgado, Hamlin, Higgins, & Mahl, 1956; Ferguson et al., 1969; Gloor, 1990; Halgren et al., 1978; Horowitz, Adams, & Rutkin, 1968; Penfield & Perot, 1963; Vignal et al., 2007; Wieser, 1979). Most real-time reports recorded during stimulations are highly fragmented but still offer an important insight into the curious nature of these experiences. The following account comes from one of Wilder Penfield's famous temporal lobe stimulation studies in the 1950s. Stimulation was applied to an unspecified area of the temporal cortex without warning: "Something is coming to me from somewhere. A dream. . . . I keep seeing things. I see people in this world and in that too. . . . I keep seeing things—I keep dreaming things" (Penfield & Perot, 1963, Case 66, p. 664).

The phenomenology of dreamy states

Looking more closely at the specific phenomenology of dreamy states, one of the most striking aspects

is the report of vivid recollections of long-forgotten memories. Vivid recollections or "relivings" can be seen as sitting at the extreme end of an abreactive spectrum. To clarify this point, according to Freud, when a repressed memory returns to consciousness, it rarely, if ever, returns in its original form (Freud, 1899):

> What is recorded as a mnemic image is not the relevant experience itself—in this respect the resistance gets its way; what is recorded is another psychical element closely associated with the objectionable one. [Freud, 1899]

Distortion of the original memory trace is thus seen as a defensive mechanism serving the cohesion of the ego. When the ego is threatened with a memory that does not reinforce ego cohesion, the memory is transformed so that it can be assimilated more favorably (wish-fulfillment). This is the mechanism that Freud described for screen memories (Freud, 1899) and for dreaming (1900a). As is shown below, the same mechanisms operate in dreamy states, psychotic states (wish-fulfilling delusions), and the psychedelic state. Vivid recollections are a complication to the rule, however, since rather than undergoing symbolic transformation, some dreamy-state and, as we will see later, psychedelic-state phenomena seem to circumvent ego defense mechanisms so that memories return in such detail that it is as if the original experience is being relived. The following is an example of a vivid recollection occurring after the stimulation of an area of the temporal cortex:

> I heard voices down along the river somewhere—a man's voice and a woman's voice calling . . ., it seems to be one I was visiting when I was a child. . . . Yes, I hear voices, it is late at night, around the carnival somewhere—some sort of travelling circus. . . . I just saw lots of big wagons that they use to haul animals in. [Penfield & Perot, 1963, p. 651]

Some have viewed vivid recollections as exact "playbacks," whereby original memory traces laid down at the time of the event become reactivated (Penfield, 1959). This theory may have some foundation (as will be shown later, similar experiences have been reported in the psychedelic state) but it is complicated by a number of factors. Aside from the mechanisms of symbolic transformations touched upon above, the present circumstances of the patient and his/her contemplations at the time of electrical stimulation or at the onset of a temporal lobe seizure can also have a significant impact on the material reported (Mahl, Rothenberg, Delgado, & Hamlin, 1964). This is very similar to the way that day residues affect the content of our dreams: "A dream is thinking that persists in the state of sleep" (Freud, 1900a).

The dreamy state as psychodynamic

The dreamy state has been viewed as a psychodynamic experience by a number of clinicians and researchers (Delgado et al., 1956; Epstein & Ervin, 1956; Ferguson & Rayport, 2006; Ferguson et al., 1969; Halgren, Walter, Cherlow, & Crandall, 1978; Kubie, 1953; Mahl et al., 1964; Ostow, 1952, 1954; Penfield & Perot, 1963; Rodin, Mulder, Faucett, & Bickford, 1955; Wild, 2005). In fact, it has been argued that Freud's earliest ideas on dreaming and primary-process thinking were influenced by his reading of Hughlings Jackson's work (Harrington, 1987). Freud was certainly very aware of Hughlings Jackson's publications, writing a pre-psychoanalysis monograph on aphasia based on Hughlings Jackson's ideas (Freud, 1891b), and citing him in *The Interpretation of Dreams* (1900a). In a recent article published in the *International Review of Neurobiology*, the phenomenology of dreamy states was approached from a neuroscientific and psychoanalytic perspective (Ferguson & Rayport, 2006). The authors (an esteemed psychiatrist and a neurosurgeon, respectively), with decades of experience treating patients with temporal lobe epilepsy, presented a number of case reports illustrating their belief that the dreamy state involves a release of unconscious material into conscious awareness:

> Reflected in the seizure-related behaviour may be emotional trauma of early life, negative feelings toward specific individuals because of past incidents or situations. [p. 30]

Rodin et al. (1955) conveyed a similar view:

> [During dreamy states] repression fails, the usual defence systems crumble, disturbing unconscious material erupts, anxiety mounts, and the personality structure becomes ineffective. [p. 372]

And Delgado et al. (1956):

> Electrical stimulation [of the temporal lobes] could alter the balance between drive and defence. [p. 528]

Dreamy states as qualitatively similar to dreaming

Hopefully it will become increasingly clear that the phenomenological similarities between dreaming and

dreamy states are not coincidental; not only have both been interpreted as psychodynamically meaningful, but it has also been proposed that the hallucinatory experiences of dreamy states are actually related to the hallucinatory processes involved in dreaming itself (Calvo & Fernández-Guardiola, 1984; Mahl et al., 1964). Looking again at some subjective reports, this proposition holds much intuitive appeal:

> A familiar sight danced into my mind and away again. [Penfield & Perot, 1963, Case 20, p. 633]

> Another dream. People were coming in and out and I heard boom, boom, boom. [Penfield & Perot, 1963, Case 6, p. 621]

> Someone was speaking to another. . . . It was just like a dream. [Penfield & Perot, 1963, Case 29, p. 640]

> I see the nurse. . . . It was a little dream. [Penfield & Perot, 1963, Case 32, p. 644]

> Now I hear them. . . . A little like in a dream. [Penfield & Perot, 1963, Case 2, p. 614]

> He said he heard a humming noise, sounds would fade out—"Then I start dreaming." [Penfield & Perot, 1963, Case 39, p. 653).

> Experience with cortical stimulation brings one to the conclusion that these induced mental states are like dreams. [Penfield & Rasmussen, 1952, p. 179]

There are several additional examples to those cited above of patients describing their dreamy-state experiences as "dreams" or "dreamlike" (e.g., Epstein & Ervin, 1956; Ferguson et al., 1969; Mahl et al., 1964; Vignal, Maillard, McGonigal, & Chauvel, 2007). This association with dreaming has occupied a familiar position in the discussion of dreamy states over the last 100 years or so. A number of clinicians involved with individual cases have reported that dreams described during analytic sessions often contain similar or identical elements to those described during the dreamy states of the same patients (Epstein & Ervin, 1956; Ferguson et al., 1969; Penfield & Perot, 1963).

> When the seizure content of cases 1 & 2 is compared with the dream content, it is apparent that both are similar and at times identical. [Epstein & Ervin, 1956, p. 52]

The anatomy of the dreamy state

A review of studies that have tried to pinpoint the anatomical origin of dreamy states has revealed that both medial and lateral temporal structures are involved, with a special sensitivity in the anterior hippocampus, amygdala, and superior temporal gyrus (Bancaud, Brunet-Bourgin, Chauvel, & Halgren, 1994). Stimulations of the medial temporal regions were more than ten times more likely to evoke a dreamy state than stimulations to any temporal neocortical regions (Bancaud et al., 1994). A more recent study has since shed further light on the origin of dreamy states; Bartolomei et al. (2004) analyzed the subjective reports of 24 patients receiving cortical and subcortical temporal lobe stimulations and found that stimulation of the perirhinal and entorhinal cortices was even more likely to evoke dreamy states than stimulation of either the hippocampus or the amygdala. Reviewing Penfield's cortical stimulations, visual hallucinations appeared to dominate during stimulations of the parietal, temporal occipital junction of the nondominant hemisphere as well as cortical matter overlying the amygdala and anterior hippocampus (Penfield & Perot, 1963); auditory hallucinations occurred most often after stimulation of the superior temporal gyrus, both anteriorly and posteriorly and in both hemispheres.

The neurophysiology of the dreamy state

Few studies have looked specifically at the neurophysiology of dreamy states as they occur spontaneously in association with epileptic activity. The epileptiform activity of temporal lobe epilepsy can be extremely variable, both between patients and within individuals. At the scalp, theta and delta slow-wave frequencies are often seen over the temporal region in medial temporal-seizure onset (Bien, Benninger, & Urbach, 2000; Blumenfeld et al., 2004; Pacia & Ebersole, 1997; Rodin et al., 1955; Wieser, 2004; Williamson et al., 1993). However, scalp electrodes are not always sensitive enough to detect activity preceding seizure onset; Wieser (2004) reports failure to record significant changes during the preictal aura in approximately 60% of patients. Depth electrode recordings offer a much more precise picture of subcortical activity and have managed to identify an initial slow-wave component in a large percentage of temporal lobe seizures (Bickford et al., 1956; Bragin, Wilson, Fields, Fried, & Engel, 2005; Delgado et al., 1956; Kellaway, 1956; Liberson, 1956; Wieser, 2004); this limbic slowing occurs early in the propagation of the seizure activity and is likely to be a correlate of the dreamy state:

> Very often, the aura expressed verbally by the patient was also marked in depth recording by the onset of localised abnormal activity [recorded bilaterally in the temporal lobes]. [Delgado et al., 1956, p. 529]

Recording neurophysiological activity in one patient with simple partial seizures using MEG, Oishi et al. (2003) recorded sporadic spikes of 2–2.5 Hz over the left posterior superior temporal region simultaneously with reports of visual hallucinations. As will become increasingly evident, these discharges of activity—similar in their phasic component, frequency, and temporal lobe origin to what have been recorded in the depths during REM sleep—are a consistent feature of *dreamlike* phenomena.

Neurophysiological data associated with the spontaneous occurrence of dreamy states is less conveniently recordable than data associated with induced electrical stimulations. Some very detailed investigations of dreamy states evoked by temporal lobe stimulation have been carried out in recent years (Bancaud et al., 1994; Bartolomei et al., 2004; Barbeau et al., 2005). Stimulating the medial temporal regions, Barbeau et al. (2005) conducted a coherence analysis of activity correlated with subjective reports of dreamy states. Consistent with the findings of Bartolomei et al. (2004), electrical currents applied to the perirhinal region produced reports of vivid recollections of past events and scenes. Many of these memories contained a distinct visual element, which emerged within seconds of the stimulation and appeared related to the after-discharge:

> The patient immediately said that something had materialised and that it was a neighbour going by in the street on a motorbike. He added: "I see him very often" and said that it was his brother's friend. Questioned later, he explained that he had seen a chromed part of a motor, then a black leathered boot and that he had inferred from these "distinct signs" that the person he was seeing was his brother's friend. [Barbeau et al., 2005, pp. 1333–1334]

The spreading discharge can be envisaged as a *discharge of energy* from the source of stimulation to a network of functionally related structures. Barbeau and colleagues reported that the perirhinal regions that produced dreamy states on stimulation are anterior subhippocampal structures functionally related to the ventral visual stream. Stimulations were applied at 50 Hz, the gamma frequency; however, analysis of the different frequency bands recorded during the *after-discharge* revealed that by far the most prominent frequency range was theta (4–8 Hz)—the same frequency that has been associated with PGO-limbic bursts in animals (e.g., Calvo & Fernández-Guardiola, 1984) and limbic bursts in human REM sleep (e.g., Cantero et al., 2003; Fernández-Mendoza et al., 2006).

Coherence analysis of those after-discharges that resulted in dreamy states revealed a functionally related network spreading from the site of stimulation (perirhinal region) to the entorhinal cortex, amygdala, anterior hippocampus, posterior hippocampus, and visual cortex. Functional coherence was highest in the theta frequency. Importantly, dreamy states with a visual element only occurred if there was a *spread of activity* in the theta range from the site of stimulation in the medial temporal lobes to the visual cortex. It is significant for our understanding of dreaming that electrical stimulation applied directly to the human occipital cortex produces only modest hallucinations of light and colors, whereas stimulation applied to areas of the temporal lobes produces visual hallucinations that are far more vivid and emotionally loaded (Penfield & Jasper, 1954; Penfield & Rasmussen, 1950). The implication is that a *discharge of electrical activity* is always required to flow from a source region to functionally related structures in order for *dreamlike* phenomena to materialize.

A working model

It is speculated in this paper that bursts of high-voltage theta and slow-wave activity passing between the temporal and visual association cortices is the key mechanism underlying the visual hallucinations of dreaming, the dreamy state, the acute psychotic state, and the psychedelic state. It is proposed that the latter three states all possess an aspect of REM sleep that is undetectable by conventional scalp EEG. According to these findings, it can be said that all three states are capable of displaying an important element of covert REM and that this can present itself in a waking state of consciousness as well as in sleep.

The acute psychotic state

> The madman is a waking dreamer. [Kant, 1764, p. 162, quoted in Freud, 1900a]
>
> Find out all about dreams and you will find out about insanity. [Hughlings Jackson, quoted in Freud, 1900a]

Acute psychotic states and dreaming

The popular comparison between dreaming and psychosis has a long history. Aristotle, in *On Dreams* in 350 BC, was perhaps the first to document an opinion that the hallucinations of the insane and the fantasies of dreaming are not merely similar, but may actually have a common origin. Freud dedicated a small

subchapter to the subject of "dreams and mental disease" in *The Interpretation of Dreams* (Freud, 1900a). In fact, it might be fairly argued that Freud's excitement during the writing of *The Interpretation of Dreams* was fuelled by his own expectation that "we will be working towards an explanation of the psychoses while we are endeavoring to throw some light on the mystery of dreams" (Freud, 1900a). While the psychoses have received much theoretical interest from psychoanalysis, clinically, psychoanalytic treatments have shown a limited efficacy for psychotic patients. Jacques Lacan (like Freud) actually advised against the traditional use of the couch and free association for the treatment of psychotic disorders, warning that latent psychoses might be aggravated by these techniques (Lacan, 1955–56). With regard to the *dreamlike* state, the most relevant psychotic phenomena are the positive symptoms of schizophrenia—that is, hallucinations, delusional thinking, and disturbances in the sense of identity. It is important to make clear that this paper does not propose that all psychotic symptoms meet the criteria for *dreamlike* phenomena; rather, it argues that certain symptoms, often evident only acutely (e.g., in organic delirium or during a pronounced episode), are fundamentally *dreamlike* in nature, both phenomenologically and neurophysiologically.

Visual hallucinations in acute psychotic states

One of the main complicating factors clouding the comparison of dreaming with schizophrenia is the predominance of auditory hallucinations in schizophrenia. However, it has be shown that visual hallucinations are a much more prominent feature of early-onset psychosis and acute psychotic episodes (Chapman, 1966; Fischman, 1983; Freedman & Chapman, 1973; McCabe, Fowler, Cadoret, & Winokur, 1972; Young, 1974), and cross-cultural studies have shown that visual hallucinations are much more common than auditory hallucinations among schizophrenic patients in the Middle East and Africa (Jablensky & Sartorius, 1975; Murphy, Wittkower, Fried, & Ellenberger, 1963). Opler (1956) and Al-Issa (1977) have argued that the shift in emphasis toward auditory hallucinations in schizophrenia is a relatively modern development. Finally, Fischman (1983) made the convincing argument that auditory hallucinations are a chronic feature of schizophrenia resulting from attentional bias, whereas early-onset psychosis and acute psychotic episodes are characterized by immediate and startling perceptual distortions of a primarily visual nature.

A psychodynamic explanation for acute psychotic episodes

From a psychodynamic perspective, the normal defense mechanisms appear to work abnormally in psychosis. Specifically, there is a disturbance in the mechanisms of repression with the effect that consciousness broadens to encompass aspects of the psyche that were previously unconscious.

> In a certain sense there is no unconscious in psychosis, since the unconscious is the result of repression. [Fink, 1997, p. 113]

Unlike in temporal lobe stimulations or the psychedelic state, failure to assign the acute disturbance a context seems to increase anxiety. The same anxiety is common in the psychedelic state, but assigning the experience a context and reassuring the individual that the experience is transient appears to reduce panic responses (Grof, 1980). Similarly, in the dreamy state, particularly in cases of electrical stimulation, the short-lasting application of electrical stimulation and fleeting nature of the experience means that its context is much less likely to be appraised in a delusional manner. In temporal lobe stimulations, the experience tends to begin as an unshakable *feeling* of déjà vu and occasionally develops into something more vivid and accountable:

> something . . . something . . . people's voices talking . . . a little like a dream . . . like being in a dance hall, like standing in the doorway—in a gymnasium—like at the Kenwood Highschool. [Penfield & Perot, 1963, p. 614]

The passing of unconscious material into consciousness appears to occur with more fluency in psychosis. However, what distinguishes the psychotic patient from the nonpsychotic is the difficulty that the psychotic patient has assigning the experience a context. *The patient experiencing an acute psychotic episode fails to identify the emerging material as having its basis in memory and emotion*; instead, he/she seems to either integrate the abreactive experience into a delusional belief system, as if it were directly associated with present events, or devise strategies to philosophize the material and defer its personal significance.

> The schizophrenic patient often cannot distinguish between memories and present perceptions; memories experienced with hallucinatory vividness and immediacy are sensed as perceptions of present events, and perceptions of present events may be experienced as memories from the past. [Searles, 1965, p. 305]
>
> We [patient and therapist] were sitting on benches on

> the lawn. She was in the midst of expressing an unusual upsurge of memories, laden with much feeling—in particular, with fondness and nostalgia—about the persons and places from a long forgotten era of her past, when, by a seeming effort of her will, she discontinued this line of talk, saying "do you know that woman over there who has the strange head? I think it's about to explode. What do they do—do they take people out of the pipes and then put them in someone's head like that until they mature?" [Searles, 1965, p. 400]

The temporal lobes and schizophrenia

In order to place these phenomenological descriptions into a neuroscientific context, emphasis will first be put on the functional anatomy of abnormal activity before the neurophysiological activity of *dreamlike* phenomena is described as it has been seen to operate in psychosis. It should become apparent that abnormal activity in the medial temporal structures is the most consistent correlate of *dreamlike* phenomena.

> The thesis that the psychotic picture arises from involvement of temporal lobe structures is strengthened by the fact that similar mental content often appears in the spontaneous seizure, in dream material, and in the response to electrical brain stimulation—all experiential phenomena clearly arising from this region of the brain. [Ferguson & Rayport, 2006, p. 13]

Neuroimaging studies of psychotic patients experiencing auditory hallucinations have shown increased blood flow and metabolic activity in the temporal regions (Gordon et al., 1994; Lennox, Park, Jones, & Morris, 1999; Notardonato, Gonzalez-Avilez, Van Heertum, O'Connell, & Yudd, 1989; Suzuki, Yuasa, Minabe, Murata, & Kurachi, 1993; Woodruff et al., 1995, 1997). It is important to consider how temporolimbic activity might correspond to the well-known dopamine hypothesis of schizophrenia.

The medial temporal lobe–dopamine system interaction

Anthony Grace has devised a model of schizophrenia that proposes that dopaminergic corollaries of disinhibition in the amygdala underlie the positive symptoms of this condition (Grace, 2000; Goto & Grace, 2007). Grace's model emphasizes the importance of the relative activity of different glutamatergic afferents innervating the nucleus accumbens. It is hypothesized that disruption to this equilibrium causes inappropriate, context-independent levels of valence to be ascribed to stimuli. The nucleus accumbens is a dense region of cells situated next to the septum, at the head of the caudate nucleus, where limbic structures have overlapping input with the dopaminergic system. The nucleus accumbens has been closely associated with the neurobiology of schizophrenia (Grace, 2000; Grace, Moore, & O'Donnell, 1998) and is significantly involved in regulating the activation of dopaminergic pathways to the prefrontal cortex; these pathways are thought to play a significant role in the experience of reward, pleasure, and addiction. In the pathological state of schizophrenia, Grace proposes that the balance of inputs to the nucleus accumbens is disturbed so that the amygdala exerts a dominant influence. Grace argues that disinhibited phasic input from the amygdala to the nucleus accumbens leads to an increased sensitivity of the mesolimbic dopamine system, with pathological consequences:

> In this pathological state, the amygdala not only fails to facilitate prefrontal cortical throughput, but actually competes with it for driving accumbens activity. Therefore . . . the system is biased to react exclusively based on the affective valence of the stimulus. As a result, the planned behaviour is replaced by impulsive responses based solely on the emotional state of the subject. . . . Such a condition may account for the reported flooding of emotions and the inability to discriminate relevant and irrelevant stimuli that are reported to be present in the schizophrenic patient. [Grace, 2000, p. 337]

There is currently much debate over the extent of dopamine's involvement in dreaming (see Hobson, 2003). Mark Solms believes that an intact mesolimbic dopamine system is necessary for the subjective experience of dreaming, citing reports of a loss of dreaming in patients with lesions severing this pathway (Solms, 1997, 2003). Solms supports his argument with studies documenting reports of increased positive psychotic symptoms, dream frequency, and dream vividness after treatment with the dopamine precursor L-dopa (Nausieda, Weiner, Kaplan, Weber, & Klawans, 1982; Scharf, Moskowitz, Lupton, & Klawans, 1978; Thompson & Pierce, 1999). Solms's argument is supported in part by the findings of Claude Gottesmann, who has detected maximum dopamine release in the nucleus accumbens of rats during REM sleep compared to waking and slow-wave sleep (Gottesmann, 2006). This finding is consistent with c-fos labeling in cats showing significantly greater activity in the nucleus accumbens during REM sleep than in either waking or slow-wave sleep (Sastre et al., 2000). It is also consistent with findings in rats that stimulation of the ventral subiculum of the hippocampus leads to

an approximately twofold increase in the number of phasically active dopamine neurons (Lodge & Grace, 2006).

In order to clarify the similarities between dreaming and schizophrenia, it is essential that the specific interactions between the medial temporal lobes and nucleus accumbens receive further investigation. It is also important to clarify whether sensitivity of the nucleus accumbens and mesolimbic dopamine pathways is influenced by the limbic bursts detected during REM sleep and linked with PGO waves and dreaming. Current evidence suggests that there is a connection: researchers working with rats have reported a resemblance between rhythmic activity in the nucleus accumbens of rats and hippocampal theta activity (Grace, 2000; Leung & Yim, 1993; O'Donnell & Grace, 1995). Further evidence is required to flesh-out this potentially very important line of enquiry.

The neurophysiology of acute psychotic states

Shifting our focus onto the all-important neurophysiological properties of acute psychotic states, Ishii et al. (2000), using MEG, recorded "theta wave bursts (4–8 Hz)" in the left superior temporal cortex during periods of auditory hallucinations in one nonmedicated psychotic patient. The phasic appearance of this activity correlated significantly with reports of auditory hallucinations ($p < .01$). These findings are supported by Canive et al. (1996), who recorded similar sharp and slow-wave activity bilaterally in the temporal regions of another unmedicated psychotic patient.

A number of earlier studies, many of which date back to the 1950s, have revealed the presence of theta and slow-wave bursts in the temporal and limbic regions of unmedicated psychotic patients experiencing acute visual and auditory hallucinations. During this exploratory era, the implantation of depth electrodes in human subjects was carried out much more freely than it is today (Heath & Mickle, 1960; Hodes, Heath, & Miller, 1954). Recordings of phasic slow-wave bursts in the limbic system of psychotic patients experiencing an intensification of psychotic symptoms were first described by the American neurologist Robert Heath. During a 7-year period, beginning in 1953, Heath implanted depth electrodes into the brains of human patients on 52 occasions. The appearance of high-voltage spiking, theta, and slow-wave activity in the septal region, the rostral hippocampus, and the amygdala were described in all psychotic patients experiencing an acute intensification of psychotic symptoms such as visual and auditory hallucinations (Heath & Mickle, 1960). This activity was not present in 6 nonpsychotic control subjects and was confined to the specified regions of the limbic system. Slow waves were not found in any other deep structures from which they recorded, and only on rare occasions did they transmit to the scalp:

> Our records with simultaneous recordings from the scalp, cortex, and deeper lying nuclear masses suggest that behavioural deviations correlate much more readily with activity of the deeper structures than either the cortical or scalp recordings. Actually, they indicate that a storm of activity can be brewing below with little or nothing showing at the cortical level. [Heath & Mickle, 1960, p. 233]

Electrodes were kept in place for prolonged periods—the longest being 2 years. Recordings would begin 2 weeks after implantation (recordings obtained before this period contained too much noise from the anesthesia and operative trauma to be of any value) and would take place two to three times per week on each patient. The presence of theta and slow-wave activity in the septum, hippocampus, and amygdala would occur periodically in sudden "bursts," showing strong correlation with the acute behavioral state of the patient:

> This has been a consistent observation in our last 20 patients. When the patients are disorganised acutely in their behaviour, we have noted an increase in the frequency of the spiking and slow wave, whereas during periods of relative clinical remission in psychosis, the recordings more nearly approach those obtained in nonpsychotic patients. [Heath & Mickle, 1960, p. 218]

Significantly, other researchers have recorded the same spike, theta, and slow-wave activity from the limbic regions of schizophrenic patients (Delgado & Hamlin, 1960; Hanley, Rickles, Crandall, & Walter, 1972; Heath & Leach, 1962; Kendrick & Gibbs, 1957; Monroe, 1956; Sem-Jacobsen, Petersen, Dodge, Lazarte, & Holman, 1956; Sem-Jacobsen, Petersen, Lazarte, Dodge, & Holman, 1955; Sherwood, 1962), most notably in the septal region, amygdala, and hippocampus but occasionally spreading to the frontal and parietal cortices:

> Marked changes in the activity in the temporal lobe with high voltage activity appearing in the central part of the lobe are also found. The activity starts and disappears with the hallucination episodes. [Sem-Jacobsen, 1956, p. 531]

> In a patient with a history of episodic psychotic behaviour, [depth EEG] showed paroxysmal, high amplitude theta activity in the septal region and frontal cortex when acutely psychotic. . . . During periods of remis-

> sion, this paroxysmal hypersynchronous and spiking activity would disappear. [Monroe, 1956, p. 530]
>
> . . . bursts of high-voltage waves of 3–9 Hz . . . from the middle and deeper parts of the temporal lobe . . . slow waves with a frequency of 2–5 Hz [appearing] intermittently in tracings from the parietal lobe . . . these slow waves appeared synchronously with bursts from the temporal lobe . . . during acute episodes of agitation and hallucination. [Sem-Jacobsen et al., 1956, p. 277–278]

Sherwood (1962) was another to report this phenomena, recording spike, theta, and slow waves from subcortical regions in 12 treatment-resistant psychotic patients. Patients exhibiting the most florid psychotic behaviors showed spike, theta, and slow-wave activity in the amygdala, hippocampus, and septum. Phasic spiking activity occurred "either singly, in trains, or associated with theta or delta waves" (p. 384). Sherwood described the EEG of two hallucinating patients as showing "theta waves and spikes, and short lasting subcortical evoked responses" (p. 383).

Acute psychotic episodes as qualitatively similar to LSD experiences

So far, the subjective and neurophysiological properties of the dreamy state and the acute psychotic state have been compared with those of the dream state. In the next section we review data relating to the psychedelic ("mind-manifesting": Osmond, 1952) or psychotomimetic ("psychosis mimicking": Behringer, 1927) drug LSD. The following reports are taken from psychotic patients and individuals experiencing the effects of a psychedelic drug. The phenomenological similarities are remarkable:

> My senses seemed alive, they hit me harder. Things appeared clear-cut; I noticed things I had never noticed before. [Bowers, 1968: psychotic episode]
>
> My senses became extremely acute. I could see an ant upon a tree at a great distance away. I could hear the whispering of my companions . . . far off from me. [Masters & Houston, 1966: drug experience]
>
> I have noticed that noises all seem to be louder to me than they were before. It's as if someone had turned up the volume. . . . I noticed it with most background noises. [McGhie & Chapman, 1961: psychotic episode]
>
> Sensations were acute. I heard, saw, felt, smelled, and tasted more fully than ever before. [Masters & Houston, 1966: drug experience]
>
> Colours seem to be brighter now, almost as if they are luminous. When I look around me it's like a luminous painting. [McGhie & Chapman, 1961: psychotic episode]

The psychedelic state

> In a dreamlike state, with eyes closed, I perceived an uninterrupted stream of fantastic pictures, extraordinary shapes with intense, kaleidoscopic play of colours. [Hofmann, 1980, p. 15]
>
> There are good reasons for applying the term "oneirogenic," producing dreams, to psychedelic drugs. In its imagery, emotional tone, and vagaries of thought and self-awareness, the drug trip, especially with eyes closed, resembles no other state so much as a dream. [Grinspoon & Bakalar, 1979, p. 253]

A very brief history of LSD

This section will focus most closely on the original psychedelic drug, lysergic acid diethylamide, or LSD. Discovered in 1943 by the Swiss chemist Albert Hofmann, the substance was found to have a remarkable potency (minimum psychoactive dose 25 μg), leading to speculations that related compounds might occur endogenously in the brains of schizophrenic patients. The search for an endogenous psychotomimetic proved unsuccessful, but in 1950, in the first paper on LSD published in English, Busch and Johnson made the following historical observation:

> LSD [causes] a transitory toxic state, disturbing [the] barrier of repression and permitting [the] re-examination of significant experiences of the past, which [are] sometimes relived with frightening realism. . . . LSD may enable psychotherapy to be shortened. [Busch & Johnson, 1950, p. 241]

The extent of LSD's history as a psychotherapeutic adjunct may come as a surprise to some. Between 1950 and the mid-1960s there were more than a thousand clinical papers discussing 40,000 patients, several dozen books, and six international conferences on psychedelic drug therapy (Grinspoon & Bakalar, 1979). In the mid-1960s, in reaction to increasing recreational use, the U.S. authorities began to restrict the use of LSD in scientific research (Lee & Shlain, 1985). Protestations followed not only from the many therapists using the substance in their clinical practice, but also from such powerful figures as Senator Robert Kennedy, who issued a congressional probe into the matter in 1966:

> Why if [clinical LSD projects] were worthwhile six months ago, why aren't they worthwhile now? . . . We

> keep going around and around. . . . If I could get a flat answer about that I would be happy. Is there a misunderstanding about my question? [Kennedy, quoted in Lee & Shlain, 1985, p. 93]

Exploring the history of LSD provides a fascinating insight into Cold War political tension in the United States (see Lee & Shlain, 1985; Marks, 1979), but it is the scientific data that is of most relevance here. Preconceptions about psychedelic drugs based on anything other than scientific research should be put aside when considering the data laid out in this section. The clinical efficacy of psychedelic-assisted psychotherapy is not addressed, although the interested reader is directed to the following literature, whose authors contribute balanced, informed, and critical appraisals of the LSD approach: Grinspoon and Bakalar (1979), Grof (1980), Novak (1998), and Mangini (1998).

The psychedelic state as dreamlike and psychodynamic

The phenomenology of LSD is complex but endlessly fascinating. During its years as a psychotherapeutic adjunct, the LSD experience was found to be enormously variable, depending a great deal on the individual taking the drug, his/her expectations, and the environment and the company he/she was in (Grof, 1975). Patients were often encouraged to close their eyes or wear a blindfold. In this state, all senses were significantly heightened, with the visual element especially enhanced. Closed-eye visual hallucinations took on what was often described as a "dreamlike" quality (Alnaes, 1967; Barr et al., 1972; Cohen, 1964; Grinspoon & Bakalar, 1979; Grof, 1967, 1975; Hofmann, 1980; Leuner, 1967; Merkur, 1998; Sandison, 2001). The experience was described as *dreamlike* not only in the sense of pronounced closed-eye visual hallucinations, but because these visions were almost invariably accompanied by a large amount of emotional material.

> It seems more likely that the visions seen under the drug [LSD] are projections of internalised conflicts, emotional discharges, a visual overflow from the deep pool of "primary-process" thinking. The primary process, the mental activity of the unconscious mind, is the stuff of which dreams, hypnagogic imagery, sensory deprivation and the LSD state are made. It is the fount of the psychotic's productions and the well-spring of artistic, sometimes scientific, creation. [Cohen, 1964, p. 70]

It has been proposed that the LSD experience might serve as a model for understanding the mechanisms of dreaming (Barr et al., 1972). It is telling that not only do both states show the manifestation of bizarre visual imagery, but they also contain a significant emotional dimension. During the LSD state, powerful abreactive experiences were reported (e.g., Cutner, 1959; Grof, 1967, 1975, 1980; Lewis & Sloane, 1958; Ling & Buckman, 1960; Merkur, 1998; Rolo, Krinsky, & Goldfarb, 1960; Sandison, 1954, 1957, 1963; Spencer, 1963, 1964; and countless more). Consistent with Freud's dreamwork mechanisms, the visual material would often undergo symbolic disguise. The symbolic content of the visual hallucinations were regarded by the majority of LSD therapists as the result of ego defense mechanisms attempting to screen recollection (e.g., Barr et al., 1972; Grinspoon & Bakalar, 1979; Grof, 1975, 1980; Leuner, 1967; Masters & Houston, 1966; Sandison, 1957, 2001).

> I began to see serpent's faces all over the wall, and then I saw myself as a fat, pot-bellied snake slithering away to destruction. I realised that I was destroying myself. . . . I told the doctor that there were snakes everywhere and that I was in the middle of them. I discovered that I had had a dream like that when I was a child. He asked me what it represented and I said "sex." When discussing this I was right back as a small child with moving grass all round me and I could see snakes slithering through the grass. The whole atmosphere was as it had been during sexual incidents with boys when I was six or seven. [Sandison, 2001, p. 45]

> Many of the phenomena in these [LSD] sessions could be understood in psychological and psychodynamic terms; they had a structure not dissimilar to that of dreams. [Grof, 1975, p. 19]

> As in dreams, names and things merge magically, words become suffused with the qualities of the objects they designate, puns take on great significance, and the mechanisms of condensation and displacement operate. [Grinspoon & Bakalar, 1979, p. 105]

Vivid recollections and relivings

Dynamic material emerging into consciousness during the LSD experiences did not invariably undergo symbolic transformation. In patients whose resistances were sufficiently overcome, either by an increase in dosage or by successful progression of the analytic process, vivid recollections of past scenes similar to those reported after temporal lobe stimulation would occur, bursting into consciousness with a sudden and dramatic effect (e.g., Abramson, 1956; Grof, 1975; Lienart, 1966; Sandison, 1954; Spencer, 1964):

> During the next spasm the feeling of someone behind me was stronger. I began to feel anxious about my

> legs, it was an odd abstract anxiety. . . . I think it was about then that I realised that I was reliving an incident that occurred when I was quite small, on holiday in Bournemouth. I wondered if I had gone small and was not in the least surprised to see my hand and arm quite little, the size of a child of seven or eight. [Sandison, 2001, p. 47]
>
> The least complicated psychodynamic experiences [during LSD sessions] have the form of actual relivings of emotionally highly relevant events and vivid re-enactments of traumatic or unusually pleasant memories from infancy, childhood, or later periods of life. [Grof, 1975, p. 44]

LSD states as qualitatively similar to dreamy states

Regarding the argument that *dreamlike* states share important similarities, it is interesting and important that both the *dreamlike* and recollective aspects of the LSD experience have been compared to the reports of patients experiencing the dreamy state of temporal lobe epilepsy and temporal lobe stimulation (Balestrieri, 1967; Bercel et al., 1956; Buckman, 1967; Cohen, 1964; Hollister, 1961; Schwarz, Bickford, Mulder, Rome, 1956a).

> Most of the symptoms reported [during LSD sessions] are reminiscent of the sensory seizures or psychosensory seizures commonly seen in psychomotor epilepsy, and which were reproduced by electrical stimulation by Penfield. This would suggest the primary involvement of the temporal lobe. [Bercel et al., 1956, p. 606]
>
> LSD . . . assists in the emergence of primitive thinking and apparent re-living of an instant in time, not unlike the experiences produced by the electrical stimulation of the temporal lobes. [Buckman, 1967, p. 86]
>
> LSD was administered to eight [epileptic] subjects. In five of them we observed a clear tendency to reproduce, under the effect of the drug, psychopathological phenomena which had already appeared during the spontaneous seizures (visual hallucinations, sensation of blocking of thoughts, olfactory, taste and visceral sensations, preaccessual anxiety). Our patients had the feeling that their usual ictal episode was repeated. [Balestrieri, 1967, p. 656]

LSD as a tool to research the unconscious

As will be shown shortly, the psychedelic state, like the other two *dreamlike* states discussed in this paper, possesses distinct similarities to the dream state not only in its phenomenology but in its neurophysiology. Of all the four states, it is perhaps the psychedelic state that offers the most incisive insight into the unconscious activities of the mind: (1) unlike dreaming, the LSD experience occurs in waking consciousness; (2) unlike the dreamy state, LSD phenomena occur for lengthy, sometimes torturous periods; and (3) unlike the psychotic state, LSD subjects have an unimpaired capacity for self-reflection and are almost invariably capable of recognizing the drug-induced origin of their delusional thoughts and hallucinations.

There is a wealth of literature on psychodynamic experiences in the psychedelic state. This section cannot do justice to such a large amount of information. The symbolic, abreactive, and recollective aspects have been briefly touched upon, but it should be pointed out that they are in many ways just the tip of the iceberg. A whole range of psychodynamic phenomena have been reported in the psychedelic state—from childhood regression to Jungian archetypes and even Rankian birth traumas. The argument that this material is merely fantasy, the meaningless result of toxic psychosis, or the product of overzealous suggestion would hold more weight were it not for the sheer number of consistent subjective reports and the fact that similar psychodynamic phenomena have been reported in states sharing the same characteristic neurophysiology.

> Freud once said of dreams that they were the *via regia* or royal way to study the unconscious; to an even greater degree this seems to be true for the LSD experience. [Grof, 1975, p. 217]

LSD induces REM sleep

The following sleep studies provide evidence that LSD increases REM sleep in humans. It is implied from these findings that LSD induces a covert REM state in waking.

LSD has been shown to decrease the time taken to enter REM sleep and increase the ratio of REM to NREM in humans when administered via intravenous infusion during sleep (Torda, 1968). Torda recounts that awakening the LSD participants during the fourth REM epoch resulted in reports of dreaming in every case, whereas control participants did not report dreaming with the same consistency. In a similar study in humans, LSD was administered subcutaneously just before expected sleep onset. The drug decreased the time taken to enter the first REM epoch and increased the ratio of REM to NREM from 1:3 during control nights to 1:2 during the LSD night (Toyoda, 1964). LSD administered to humans orally just before sleep onset or after being awakened 1 hour

after falling asleep, caused a significant prolongation of REM sleep epochs in 11 of 12 subjects (Muzio, Roffwarg, & Kaufman, 1966). Muzio and colleagues found that when the dose was increased in subsequent experiments, greater increases in the duration of REM sleep were seen. In connection with Nielson's covert REM hypothesis, the NREM of participants administered LSD were at times "punctuated by brief bursts of REMs accompanied by reappearances of a low voltage fast EEG and saw tooth waves [at the scalp]. These periods each lasted 2–12 sec, and then gave way to the [slow-wave sleep] pattern" (Muzio, Roffwarg, & Kaufman, 1966, p. 318).

The findings of these three studies provide good evidence that LSD induces REM sleep in the sleeping human; whether or not LSD also induces a REM-like state in the waking state is a more interesting question, however. What might be referred to as the psychedelic-induced "waking dreamer" hypothesis is an idea that has been put forward on a number of occasions in the past (e.g., Brooks, 1975; Callaway, 1988; Froment, Eskazan, & Jouvet, 1971; Hobson, 2001; Stern, Morgane, & Bronzino, 1972). Up until now, however, this issue has never been truly resolved. One reason why the idea is difficult to test is the fact that conventional scalp EEG shows a very similar output during REM sleep to that during waking (Hobson, 2001). This makes it difficult to identify anything recorded at the scalp suggestive of a crossover between waking and dreaming. Some researchers have attempted to record PGO activity during awake periods in animals administered LSD (Brooks, 1975; Froment, Eskazan, & Jouvet, 1971; Stern, Morgane, & Bronzino, 1972), but this approach has been shown to be methodologically problematic, given the difficulty of differentiating REM-sleep PGO spikes from the visual evoked responses of normal waking consciousness (Brooks, 1975). This seemingly insurmountable problem would have led to a theoretical stalemate were it not for the fact that, in the mid-1950s, a couple of depth EEG experiments involving administered LSD to humans were published.

Depth EEG recordings during the psychedelic state

In 1956, Schwarz and colleagues administered LSD and mescaline to 5 patients with depth EEG implants. Of these patients, 2 had intractable epilepsy and 3 had chronic schizophrenia (Schwarz, Sem-Jacobsen, & Petersen, 1956b). The 3 schizophrenic patients responded to both psychedelic drugs with an increase in high-voltage bursts of theta and slow-wave activity (2–5 Hz) in subcortical regions. The 2 epileptic patients showed a more varied response. Both showed a curious quietening of epileptic discharge after both drugs, but one showed the emergence of previously nonexistent spiking in the temporal lobe as well as focal high-voltage activity in the ventromedial region of the frontal lobe. The patient's psychological state during the drug reaction was described as "continuously disturbed, presumably with pronounced hallucinations" (p. 583). The second epileptic patient showed phasic spike discharges from the occipital region during the height of psychedelic reaction. One particularly relevant observation took the following form:

> That the activation in the temporal depths by mescaline and LSD-25 in one patient appeared to coincide with a period of active auditory and visual hallucinations. [Schwarz, Sem-Jacobsen, & Petersen, 1956b, p. 586]

In the second study, by Monroe, Heath, Mickle, and Llewellyn (1957), LSD and mescaline were administered to six human patients; four were schizophrenic, one showed acute psychotic symptoms and suggestions of psychomotor epilepsy, and one suffered from Parkinson's disease. Using a range of doses (50–200 μg of LSD), 14 individual drug sessions were recorded. Regular episodes of phasic, high-amplitude, theta activity described as "paroxysmal" were recorded in the septum, hippocampus, and amygdala of patients displaying the most pronounced indications of hallucinatory symptoms. Those patients that showed the strongest signs of hallucinatory behaviors exhibited the most pronounced EEG disturbances. As the author of this paper has attempted to do, Monroe and colleagues drew comparisons between the psychological and neurophysiological effects of psychedelic drugs, temporal lobe epilepsy, and acute psychotic episodes.

Like the human depth EEG sleep studies, the reliability of the activity recorded in the LSD trials might be challenged on the basis that all the patients from whom recordings were taken showed a premorbid psychopathology. It might be argued that these results cannot be regarded as indicative of the neurophysiological activity of normal healthy individuals experiencing the effects of these drugs. This criticism can, however, be contested on a number of grounds: (1) the overwhelming majority of the patients involved in both studies showed the same basic behavioral reaction to the drugs as would be seen in normal healthy volunteers; (2) the output from the depth electrodes showed distinct changes, corresponding with the intensity of the hallucinatory experiences; and (3) many animals studies

have recorded consistent subcortical activity during LSD intoxication. Studies in monkeys (Monroe & Heath, 1961) and cats (Adey, Bell, & Dennis, 1962; Brooks, 1975; Brown, 1961; Linsley, Carpenter, Killam, & Killam, 1968; Schwarz, Wakim, Bickford, & Lichtenheld, 1956c; Winters, Mori, Bauer, & Spooner, 1967) have all shown the same phasic bursts of theta and slow-wave activity in the medial temporal lobes that were seen in the human patients administered LSD. Additionally, analysis of c-fos expression in the rat brain after administration of LSD showed a five- to eightfold increase in the cell activity marker in the central nucleus of the amygdala compared to controls (Gresch, Strickland, & Sanders-Bush, 2002).

The psychedelic state and the temporal lobes

Another source of evidence that supports the role of the temporal lobes in the LSD reaction comes from Serafetinides (1965), who compared the effects of LSD in 22 patients with temporal lobe epilepsy before and after temporal lobectomy. Perceptual responses to LSD were reported to be significantly richer before the operation compared to after it. This finding is backed up by an experiment in chimpanzees that found that animals with bilateral temporal lobectomies did not show the same severe psychical disturbances evident in healthy chimps receiving equivalent doses of LSD (Baldwin, 1960). Suggestion that this diminished effect might be the result of surgical trauma or the removal of an equivalent mass of brain matter from the prefrontal cortex are undermined by the findings of Holden and Itil (1970), who administered LSD to human patients before and after frontal lobotomy and found a facilitated effect postoperatively.

Imaging studies involving LSD have never been carried out. However, using positron emission tomography (PET), Vollenweider et al. (1997) recorded the change in glucose metabolism of 10 healthy human participants after administration of psilocybin. Psilocybin—the active ingredient in magic mushrooms—has a molecular structure and pharmacology similar to that of LSD and shows cross-tolerance with LSD, and the behavioral effects of both are closely related (Abramson & Rolo, 1967; Nichols, 2004). Vollenweider and colleagues discovered the most dramatic increases in glucose metabolism (a marker of the brain activity) in the medial temporal lobes (25.3%). The authors commented that the pattern of activity seen in the drug state correlates with psychotic symptoms such as hallucinations and ego dissolution.

Summary

Taken as a whole, it should now be apparent that the neurophysiological characteristics of the psychedelic state show definite similarities to those described in the dream state, the dreamy state, and the acute psychotic state. It is believed that not only can we now say with confidence that the temporal lobes are important for the manifestation of *dreamlike* phenomena, but that it is specifically high-voltage bursts of theta and slow-wave activity in the medial temporal regions spreading to the association cortices that correlates most closely with its emergence.

Table 1 summarizes the key texts cited in this paper: those studies that support the hypothesis that the dreamy state, the acute psychotic state, and the psychedelic state share phenomenological and neurophysiological similarities to the dream state itself. It is also shown that all four states have been interpreted as psychodynamically meaningful and that this interpretation is supported by evidence of neurophysiological similarities between the four states.

Discussion

The primary argument of this paper is that the dreamy state, the acute psychotic state, and the psychedelic state all share important phenomenological and neurophysiological similarities to the dream state and can therefore be regarded as *dreamlike* with some degree of scientific legitimacy. The secondary argument is that since psychodynamic phenomena have been identified in all *dreamlike* states consistently, by different individuals, at different times, and since these phenomena appear to correlate significantly with a characteristic neurophysiology, then we can say with confidence that the phenomenology is consistent with the biology and therefore amenable to scientific investigation and eventually verification.

It is felt that the findings in this paper provide good evidence that the dreamy, acute psychotic and psychedelic states all share significant similarities with the dream state, on both a phenomenological and a neurophysiological level. There is an abundance of cases where comparisons have been drawn between *dreamlike* states and the dream state, and although these observations are subjective, their sheer numbers and the credentials from whom they have come are impressive—for example, Aristotle, Kant, Hughlings Jackson, Freud. The intuitive appeal of these comparisons has always implied the existence of a material

Table 1
Summary of the key texts cited in this paper

	Dream state	*Dreamy state*	*Acute psychotic state*	*Psychedelic state*
Subjective phenomena compared to that of the dream state; key texts	Not applicable	Hughlings Jackson, 1888; Penfield & Rasmussen, 1952; Penfield & Perot, 1963; Ravagnati et al., 1979	Aristotle, *On Dreams*; Freud, 1900a; Hobson, 2001; Gottesmann, 2005, 2006.	Cohen, 1964; Barr et al., 1972; Grof, 1975, 1980; Grinspoon & Bakalar, 1979; Hofmann, 1980; Callaway, 1988
Interpreted as psychodynamically meaningful; key texts	*The Interpretation of Dreams* (Freud, 1900a); *Dreams* (Jung, 1974)	Ostow, 1952, 1954; Kubie, 1953; Rodin et al., 1955; Epstein & Ervin, 1956; Delgado et al., 1956; Penfield & Perot, 1963; Mahl et al., 1964; Halgren et al, 1978; Ferguson et al., 1969; Ferguson & Rayport, 2006	"Case History of Schreber" (Freud, 1911c [1910]); *The Psychoses* (Lacan, 1955–56); *Collected Papers on Schizophrenia* (Searles, 1965)	*The Beyond Within* (Cohen, 1964); *The Use of LSD in Psychotherapy* (Abramson, 1967); *Realms of the Human Unconscious* (Grof, 1975); *LSD Psychotherapy* (Grof, 1980)
Reports of high-voltage theta and slow-wave activity detected in the medial temporal regions of humans	Freemon & Walter, 1970; Giaquinto, 1973; Salzarulo et al., 1975; Mann et al., 1997; Yu et al., 1997; Bodizs et al., 2001; Cantero et al., 2003	Bickford et al., 1956; Delgado et al., 1956; Kellaway, 1956; Liberson, 1956; Wieser, 2004; Bragin et al., 2005; Barbeau et al., 2005	Sem-Jacobsen et al., 1955, 1956; Monroe, 1956; Kendrick & Gibbs, 1957; Delgado & Hamlin, 1960; Heath & Mickle, 1960; Heath & Walker, 1985; Sherwood, 1962; Heath & Leach, 1962; Hanley et al., 1972	Schwarz et al., 1956b; Monroe et al., 1957

foundation; hopefully this paper has shown these intuitions to be sound.

It must be made clear that while it is believed that the psychodynamic interpretation of the *dreamlike* state is valid, it is acknowledged that this is merely a psychological interpretation. This paper could have been written from the perspective of cognitive psychology, and perhaps a cognitive approach would have some use. The concept of dissociation is both psychodynamically and cognitively relevant. The *dreamlike* state is clearly dissociative, both on a neurophysiological level (fast, low-voltage, desynchronized activity at the scalp, and high-voltage theta and slow-wave activity in the medial temporal regions) and on a phenomenological level. It is the opinion of the author, however, that the traditional cognitive approach cannot offer the same depth of insight into the phenomenology of the *dreamlike* state. The psychodynamic model has been chosen because it offers the best tools for the job and because the neurophysiological data impart a degree of scientific legitimacy to the model itself. Psychoanalysis offers an expansive view of abstract phenomena, whereas cognitive psychology generally takes a reductive approach. It is the opinion of the author that the information-processing model is a relatively sterile conception of the human psyche; its descriptions of abstract phenomena such as affect, dreaming, and *dreamlike* phenomena offer only a limited, intuitively unappealing, and plainly unsatisfactory level of explanation.

Like the spiritual encounter, the emotional impact of the *dreamlike* state exists independently of its explanation. Attempts to explain *dreamlike* states should originate from the phenomena and not be forced upon it by incompatible psychological approaches. Psychological approaches are human constructs. They operate according to a vocabulary of concepts, but these concepts merely reflect a reality; they have no existence other than as referents to phenomena, and only the phenomena can be felt to exist with any certainty. There are many examples in the psychological literature where disparate approaches offer vaguely similar interpretations of the same phenomena. To some extent, this is

the case for *dreamlike* phenomena. For example, dissociation and depersonalization can be seen as compatible to a large extent with ego dissolution. Thus, while it is believed that the psychodynamic model is the most useful model for understanding the *dreamlike* state, it would be wrong to suggest it has exclusive legitimacy. It is the phenomena that have primacy. Thus, this paper does not claim to provide evidence for the legitimacy of psychoanalytic theory. It does, however, provide evidence for neurophysiological correlates of certain phenomena of interest to psychoanalysis—for example, repression, abreaction, and psychical energy.

This paper has proposed that a characteristic neurophysiological activity invariably appears in a specific region of the forebrain during the emergence of *dreamlike* phenomena. It is acknowledged that this is only one part of a wider whole, the reality of which is considerably more complicated and ambiguous.

There are several possible future directions for the investigation of *dreamlike* phenomena. The first consideration is that contemporary empirical verification be sought for the proposition that high-voltage theta and slow-wave activity in the medial temporal regions correlates reliably with the manifestation of *dreamlike* phenomena. It is felt that the data cited in this paper provide the necessary confirmation; however, future experiments designed to test the hypothesis are very much encouraged.

In order to do this, it is necessary that aspects of the phenomenology be operationalized; collating all the different descriptions of *dreamlike* states should allow us to work toward something more testable than simply, "these states are psychodynamic." A huge number of psychoanalytic concepts have been used in reference to *dreamlike* phenomena—for example, primary-process thinking, ego dissolution, symbolism, screen memory, fantasy, repression, abreaction. Unifying these phenomena on the level of *psychical energy* might provide the necessary breakthrough. On a physiological level, the term "psychical" need only be replaced by "electrical." The following hypotheses are provided for future testing:

1. All occurrences of *dreamlike* phenomena are preceded by dissolution of the psyche's inhibitory, executive hold (the ego) over its primary processes.
2. Cortical disinhibition allows psychical energies to flow with an increased motility.
3. The defining feature of dreamlike phenomena is the discharge of psychical energy from the unconscious and its dissipation into consciousness.
4. High-voltage bursts of theta and slow-wave activity spreading from the limbic (temporal) regions to the association cortices are the primary neurophysiological correlate of the emergence of unconscious material into conscious awareness.

Contemporary ethical constraints are likely to prevent many prospective studies in this area. Grasping the practical reality of this fact should explain why such a large amount of old and rare material has been dug up. Human studies relevant to the *dreamlike* state are rare to begin with, often dating back to periods of flurried activity where conditions for ethical approval were very different from what they are today. Experiments in humans are necessary for the confirmation of the above hypotheses, because of the requirement for subjective reports. Modern imaging techniques such as magnetic resonance imaging (MRI) are capable of a high level of spatial resolution, but their temporal resolution is poor. MEG is a potentially useful tool in this respect, given its ability to detect activity at a greater depth than conventional scalp EEG. Another possible means of resolving the PGO issue in humans is to carry out more sleep recordings in patients with implanted electrodes for the purpose of deep brain stimulation (DBS). DBS techniques have undergone considerable development in recent years and are now approved in the United States for the treatment of Parkinson's disease, dystonia, and depression. It is likely that this will increase the number of opportunities for depth recordings during sleep. Exciting findings are likely to emerge from this line of research.

Modern research with psychedelic drugs may provide the necessary scientific breakthrough for psychodynamic psychology. Recent articles appearing in respected journals have recommended that modern projects be devised to look again at the clinical and experimental value of psychedelic drugs (*Lancet*, 2006; Sessa, 2005). Freud did not live long enough to voice an opinion on the psychedelic approach, but the following remark made in 1938 provides an interesting insight as to where he may have positioned himself:

> But we are here concerned with therapy only in so far as it works by psychological methods, and for the time being we have none other. The future may teach us how to exercise a direct influence, by means of particular chemical substances, upon the amounts of energy and their distribution in the apparatus of the mind. It may be that there are other still undreamt of possibilities of therapy. [Freud, quoted in Ling, 1967, p. 151]

Jung, on the other hand, like many psychoanalytic psychotherapists at the time, was rather more suspicious of

the drug-assisted approach—not because he believed psychedelic drugs could not stimulate the emergence of unconscious material, but because he believed that enough could be accessed already by the methods of conventional therapy:

> Is the LSD drug mescaline? It has indeed very curious effects—vide Aldous Huxley—of which I know far too little. I don't know either what its therapeutic value with neurotic or psychotic patients is, I only know that there is no point in wishing to know more of the collective unconscious than one gets through dreams and intuition. The more you know the greater and heavier becomes your moral burden. . . . If I once could say that I had done everything I know I had to do, then perhaps I should recognise a legitimate need to take mescaline. [Jung, 1954, p. 172]

For the future of psychoanalysis, it is advised that rather than leave psychodynamic phenomena purely to the domain of phenomenological description, we begin to make concerted efforts to correlate specific psychodynamic phenomena with neurophysiological activity. Of course, as mentioned above, opportunities to do this experimentally are limited by practical and ethical constraints; however, we should at least begin to consider the importance of this process and how it might be achieved if psychoanalysis is again to be seen as a leading force in the science of the mind.

As for dream research, an important next step is to identify the primary triggers of the characteristic wave forms that oscillate throughout the medial temporal lobes and propagate the association cortices during visual hallucinations. It also needs to be discussed, for example, what inhibitory mechanisms are shut off during the *dreamlike* state in order for limbic bursts to occur.

Finally, the pharmacological actions of LSD and related psychedelics may provide important insights into the processes of dreaming and other dreamlike states (including those that have not been discussed in this paper). As occurs in dreaming, LSD shuts of the dorsal raphe nucleus through stimulation of presynaptic serotonin 5-HT1A autoreceptors. Its principal pharmacological property, however, is its high affinity and agonist activity at the 5-HT2A receptors (Nichols, 2004). Stimulation of 5-HT2A receptors is associated with glutamate release and cortical excitation. A relinquishment of cortical inhibition may be necessary for limbic disinhibition. As was discussed in the section on acute psychotic states, bursts of activity in the medial temporal regions are associated with increased sensitivity of the mesolimbic dopamine system. Study of dreamlike states may throw some light on the still problematic area of psychosis.

Conclusion

This paper has made the case that four subjective states with similar phenomenologies also share important neurophysiological characteristics. Three of the four subjective states are described as *dreamlike*, based on their similarities to the dream state. Evidence is cited from a large number of studies showing that the behavioral manifestation of all four states is correlated with the occurrence of high-voltage theta and slow-wave activity in the medial temporal regions. Emphasis is laid on the consistent interpretation of unconscious material emerging into consciousness in all four states. Evidence that the emergence of this phenomenon correlates with a characteristic neurophysiological activity opens up the possibility of testing psychodynamic phenomena via experimental means.

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