THE JOURNAL OF NERVOUS AND MENTAL DISEASE
Vol. 140, No. 6

THE EFFECT OF LSD ON THE SLEEP-DREAM CYCLE

AN EXPLORATORY STUDY

WILLIAM J. GREEN, M.D.[1]

In the past decade, the sleep-dream cycle in humans has been much studied. Investigations have been concerned with various aspects, including the effect of pharmacologic agents. However, a current review of approximately 1,100 references on lysergic acid diethylamide (LSD) revealed no study directed to the relationship of LSD and the sleep-dream cycle. The present paper therefore reports an initial exploration of the effect of LSD on the sleep-dream cycle.

The cyclic EEG activity which can be expected in normal individuals in a typical night of sleep has been identified. Dement and Kleitman (9) divided these patterns into four stages: 1) a low-voltage, modified alpha pattern, with irregular frequency; 2) presence of 14 cycle per second sleep spindles and occasional K-complexes with a low-voltage background; 3) random delta waves with some superimposed spindling; 4) half or more of the record dominated by large delta waves and no spindling. During sleep, Stage 1 appears in conjunction with bilaterally synchronous, conjugate, rapid vertical and horizontal eye movements (REMs) at approximately 90-minute intervals, and has come to be known as rapid eye movement periods (REMPs). The REMs do not ordinarily appear in Stages 2, 3 or 4, known as NREMPs (no rapid eye movement periods). Numerous investigators (1, 8, 10, 18, 20, 21) have reported that subjects are more likely to report dreaming when awakened from REMPs than when awakened from NREMPs. Rechtschaffen (26) found that NREM period mental activity was more like thinking than dreaming, more conceptual and more concerned with the contemporary life of the subject.

Physiologic changes have been reported (19, 20) to occur during the rapid eye movement periods (REMPs). Some of these are increased and irregular respiration, increase in blood pressure and pulse, an increased arousal threshold and a marked decrease in gross body movements. Snoring has been reported not to occur during REMPs (20), or to be extremely rare (12). Some investigators have suggested that the psychological status during dreaming may be a third state of mind when compared to wakefulness and non-dreaming sleep (*e.g.*, no rapid eye movement period). Comparison of the EEG pattern during an REMP and the awake state reveals similarities which are not present in any of the other stages of sleep. With this frame of reference, some investigators have described the REMP status as being most near wakefulness, hence "light sleep." The physiologic changes associated with REMPs have been termed to be the result of "activation" (to be discussed below). Because REMPs show a waking-like EEG pattern of cortical activity, some workers have inferred the psychological status of the two to be alike. However, this assumption may be questioned in terms of evidence that links arousal thresholds and galvanic

[1]Veterans Administration Hospital, Topeka, Kansas. This paper results from a study conducted at the VA Hospital with support by Hospital Project 331, P6-64, and was presented to the Faculty of the Menninger School of Psychiatry in partial fulfillment of the requirements for graduation. The writer wishes to express his appreciation for the advice and assistance of Dr. Howard Shevrin of the Menninger Foundation. Thanks are also extended to Dr. R. E. Reinert, Chief of Staff, and Mr. Richard B. Shackelford, Research Unit, of the Topeka Veterans Administration Hospital.

skin resistance (GSR), both of which are increased during REMPs. The GSR has been reported by numerous investigators (3, 6, 11, 27, 30, 31) to vary inversely with the state of alertness or arousal. States of relaxation are accompanied by high skin resistance levels, with a maximum being reached during sleep. Situations causing startle, attention, alertness, anticipation and apprehension are accompanied by low skin resistance. The GSR phenomenon is thought to be related to the permeability of the sweat gland lining, which is mediated by the sympathetic nervous system. The GSR seems to be inversely related to the activity of the vigilance system (reticular activating system) (31). Hawkins (16) found the basal skin resistance to be *higher* during REMPs than NREMPs and a progressive increase in basal resistance to occur, paralleling the greater amount of REMPs in the last half of sleep.

Reports in the literature (24) indicate that the principal changes in the EEG after LSD administration are in the alpha rhythm (8 to 13 cycles per second) of awake subjects, with an increase in the rate of 1 to 3 cycles per second and the appearance of beta rhythm (18 to 30 cycles per second). These changes have been observed in cortical and subcortical recordings. It has been suggested (19) that hypnotic drugs decrease the amount of total nightly dream time by increasing the depth of sleep and decreasing the amount of Stage 1 EEG. One study (15) concluded that a large dose of alcohol, in normals, reduced the amount of Stage 1 EEG associated with rapid eye movements and inferred dream time. It has been suggested that depressants to the central nervous system decrease the frequency and increase the amplitude of EEG waves, and stimulants of the central nervous system increase the frequency and decrease the amplitude of EEG waves (7). It would seem to follow that stimulants would decrease the depth of sleep and provide part of the setting in which Stage 1 EEG with rapid eye movements (REMs), and dreaming occurs. However, amphetamine has been found to inhibit the REMPs in subjects undergoing experimental dream deprivation. Oswald (25) observed that amphetamine addicts in the abstinence syndrome had an alteration of the rapid eye movement periods (REMPs). The changes that Oswald reported include the onset of REMPs as soon as 4 minutes after sleep onset as contrasted to a normal time lapse of about 70 minutes. Further, the REMPs lasted as long as 70 minutes, compared to a normal duration of about 10 minutes in the first two hours of sleep. Total nightly dream time was as high as 48 per cent; the usual amount is about 22 per cent. Normal functioning returned immediately if amphetamine was given but if not, the return to a normal cycle took 3 to 8 weeks.

LSD and related drugs are commonly called hallucinogens (4), although the abnormal mental states they produce are not always characterized by hallucinations. LSD is a semi-synthetic amide of rye ergot. It was investigated in dogs by Rothlin in 1938, but was abandoned when undesirable side-effects were found. In 1943, Albert Hoffman, a Swiss biochemist, while synthesizing LSD tartrate in an effort to produce a stimulant, accidentally inhaled a small quantity of the drug. He experienced feelings of depersonalization, visual hallucinations, body image distortion, euphoria and distortion of time perception. Subsequently, studies have identified the following pharmocologic properties of LSD:

Central action: changes in psychic activity manifested by excitation, mood changes, perceptual disturbance, hallucinations, depersonalization, and body-image distortions.

Somatomotor changes: pyramidal effects, extrapyramidal effects, ataxia, and spastic paralysis.

Autonomic nervous system activity: respiratory depression, hypotension, bradycardia or tachycardia, hyperglycemia, temperature rise, mydriasis, pilomotor reaction, lacrimation, nausea and vomiting.

Peripheral action: constriction of the smooth muscle of the uterus, vagina, bronchioles and blood vessels.

The latent period for autonomic effects of LSD is 20 minutes; the psychic effects appear in 40 minutes, reach a peak in 2 to 3 hours, and last 8 to 12 hours. The amount of LSD required to produce symptoms is minute: 60 to 200 micrograms. The effects are generally dose-related, but they are modified by the subject's personality and the setting in which it is given.

LSD may be described as ergotropic in its action in that it produces excitement and increases sympathetic activity, sensitivity to external stimuli and skeletal muscle tone. Ergotropic agents in general, including mescaline, amphetamine and ephedrine, if given in sufficiently large doses, induce behavioral changes and illusions in man. One attempt to explain the psychotomimetic effect of LSD is based on the similarity of its chemical structure to epinephrine, so that it may possibly interfere with the epinephrine cycle. The indole-like chemical structure is found in LSD, mescaline, psilocybin, serotonin, reserpine and other psychotomimetics such as hashish, ibogaine and harmine.

The effect of LSD and other psychotomimetics in producing an artificial "model psychosis" (2) in a controlled laboratory setting has provided the stimulus for a large number of LSD studies. These investigations have focused on understanding the metabolic, neurophysiologic and neuropharmacologic changes that may be involved in naturally occurring psychoses. Some understanding of the changes in nightly dream time which may be produced by LSD administration could further the knowledge of the role of dreams in functional psychoses.

DREAMS AND PSYCHOSES

Freud (14) proposed that dreams function to provide a safety valve for partial discharge of instinctual drives (wish-fulfillment) and thus preserve sleep. It therefore follows that in mental disorders postulated to have increased instinctual drive pressure there is an increased dreaming time. When psychological disorganization begins, it would be expected that the total nightly dream time will increase in order that reintegration of psychological functioning comes about through drive reduction. Fisher (13) has described a patient from whom he obtained sleep-dream data before, during and after an acute schizophrenic disorganization marked by delusions and hallucinations. The patient's baseline total nightly dream time was 28 to 30 per cent (according to Fisher, considerably higher than the 20 to 22 per cent found for normal samples), but increased rapidly to 50.1 per cent at the onset of the acute psychosis. The dream time percentage level subsequently returned to previous levels following the administration of 20 to 30 milligrams of trifluoperazine (Stelazine). Fisher assumed that instinctual drive pressure striving for discharge was markedly increased at the most acute stage, when the ego defenses were pathologically weakened, allowing the emergence of the dream cycle into the waking state in the form of delusions and hallucinations. If LSD produces a temporary intensification of instinctual drive strength similar to what appears to happen in a psychosis, it may be hypothesized that the total nightly dream time would increase following administration of LSD.

SUBJECT

The subject (*S*) was a 38-year-old Caucasian male, a patient in the alcoholic treatment program of the hospital. He had been severely alcoholic for the past five years but had no delirium tremens or psychotic episodes. He was alert, oriented,

with no memory impairment and no perceptual disorder; his measured intellectual level was in the bright normal range. He stated he had generally recalled dreaming every other night and did not experience anxiety dreams (*e.g.*, nightmares). For two weeks prior to the experiment, as well as the intervening week between the baseline and LSD sessions, *S* was asked to keep a dream log. He recalled no dream details during that time. He described being told by his wife that he frequently snored, occasionally talked in his sleep, and vaguely remembered a few episodes of sleepwalking during childhood. Examination revealed him to be free of physical and neurologic impairments, and all laboratory studies, including EKG and EEG, were within normal limits. There had been conversation among the ward patients who had been given LSD in previous experiments and also some who were currently being tested. *S* thought beforehand that he might have "some wild dreams" because he had heard that remark from a previous participant in the alcoholic treatment program.

METHOD AND TECHNIQUE

Three hundred micrograms of LSD were administered to *S* at 12 noon, once during his hospital stay as a participant in the alcoholic treatment program.[2] After receiving LSD, *S* experienced a daytime psychotomimetic reaction (rated as "three" on the scale) with distortion of reality and mental confusion. (The level scale of Chwelos *et al.* [5] for LSD reactions ranges from a level of one to six: one is characterized by resistances taking the form of running away from the drug, denial of the drug's potency, and holding onto reality; six is characterized by a psychedelic reaction with a revealing ecstatic experience, a feeling of one with God and man similar to a religious conversion experience, and the discovery of new and greater insights.) Continuous all-night EEGs were recorded, with two baseline nights (BL^1 and BL^2) in succession, followed a week later by the LSD experimental night and the two nights following (FU^1 and FU^2). The time of retiring at night and morning awakening was approximately the same for all five sessions, with the awakening time dictated by ward policy.

The procedure for monitoring EEGs and eye movements continuously through the night has been described by Dement and Kleitman (9). In the present study at least five channels of information were obtained for each session. Three eye-movement and two EEG channels were recorded. The electrode placements were prefrontal, parietal and outer canthi leads. Bipolar and ear reference EEG recordings were made. A microphone on *S*'s bed permitted continuous appraisal of his noise and sound.

The galvanic skin resistance (GSR) measurements were obtained with an Airborne Tissue Resistance Monitor, Model 152, impressing a current of eight cycles per second square wave. The two GSR electrodes were placed on the left foot. GSR readings were obtained intermittently by direct readings in K ohm values on the LSD night and the two follow-up nights (there was a technical failure on the baseline nights).

MEASURES

For the purpose of this paper only data from REMPs will be reported. The dimensions to be described are: time of onset of REMPs during the sleep sessions, per cent REMPs, actual duration of REMPs, various indicators of activity during REMPs (*e.g.*, gross body movements, vocalizations, GSR), occurrence of isolated bursts of REMPs (to be described below).

Because *S* was awakened each morning at the same time (6:00 a.m.) on all sessions, he was interrupted at different points

[2] Chotlos, J. W. and Reinert, R. E. Veterans Administration Study P11-63, Research Project in Alcoholism.

in his fourth dream cycle. It was therefore difficult to compare this last cycle for all sessions. It was decided to analyze the first three dream-cycles because they were comparable for all sessions.

RESULTS

Onset of first emergent Stage 1: The onset of the first REMP for the LSD session was approximately a half-hour later that for any of the other four sessions: 72 minutes as compared to 48, 46, 43, 45 for BL^1, BL^2, FU^1 and FU^2 respectively. Because of this delay during the LSD session, when *S* was awakened he had just begun his fourth REMP.

Per cent REMPs: The calculated dream time percentages were: 18.32 (BL^1), 21.65 (BL^2), 26.45 (LSD), 29.43 (FU^1), and 21.87 (FU^2).

Actual duration of REMPs: The average duration of REMPs in minutes was in the expected direction: 14.1 (BL^1), 19.05 (BL^2), 28.48 (LSD), 32.38 (FU^1) and 17.72 (FU^2).

Activity during REMPs: Gross body movements during REMPs were indicated by movement artifacts on the EEG and were generally related to sounds relayed over the microphone placed on *S*'s bed. Body movements were most frequent on the LSD night and least frequent during the baseline nights. The follow-up nights fell in between the two extremes. As one would expect, there was a progressive increase in the number of body movements as the dream periods became longer in duration.

Vocalizations: A variety of sounds made by *S* were recorded, such as grunts, groans, moans, snorts, snores, "uh" and "uh-uh." Snoring occurred in all REMPs during the LSD night, and in the third REMP during both follow-up nights, but in none of the baseline REMPs.[3]

Galvanic skin resistance: Technical difficulties prevented obtaining GSR readings during the baseline nights. The GSR readings were highest on the LSD night, indicating the presence of lower tissue conductance than on the follow-up nights.

[3] Snoring occurred once in the fourth REMP of BL^2 but was not included in Table 4.

TABLE 1

*Time of First Appearance of Successive REMPs**

REMP	Minutes after Sleep Onset				
	BL^1	BL^2	LSD	FU^1	FU^2
1	48	46	72	43	45
2	117	130	152	128	106
3	216	218	268	260	224

* REMP = rapid eye movement period; BL = baseline night; LSD = experimental night with LSD; FU = follow-up night.

TABLE 2

*Total Duration of Successive REMPs**

REMP	Duration in Minutes				
	BL^1	BL^2	LSD	FU^1	FU^2
1	8.66	7.00	7.80	14.83	7.50
2	21.66	13.16	27.16	33.66	27.16
3	12.00	37.00	50.50	48.66	18.50

* REMP = rapid eye movement period; BL = baseline night; LSD = experimental night with LSD; FU = follow-up night.

TABLE 3

*Gross Body Movement during a REMP**

REMP	BL^1	BL^2	LSD	FU^1	FU^2
1	0	0	2	0	1
2	0	1	4	3	0
3	1	3	15	10	7

* REMP = rapid eye movement period; BL = baseline night; LSD = experimental night with LSD; FU = follow-up night.

TABLE 4

*Vocalization during a REMP**

REMP	BL^1	BL^2	LSD	FU^1	FU^2
1	0	0	13	0	0
2	0	0	3	0	0
3	0	0	15	1	8

* REMP = rapid eye movement period; BL = baseline night; LSD = experimental night with LSD; FU = follow-up night.

TABLE 5
*G. S. R. during a REMP**

REMP	Readings in K Ohms				
	BL^1	BL^2	LSD	FU^1	FU^2
1	—	—	51.5	41.0	46.6
2	—	—	31.2	30.7	27.3
3	—	—	30.2	23.7	24.5

* REMP = rapid eye movement period; BL = baseline night; LSD = experimental night with LSD; FU = follow-up night.

TABLE 6
*Isolated Bursts of REM**

REMP	BL^1	BL^2	LSD	FU^1	FU^2
1	0	0	1	1	1
2	0	0	1	0	1
3	0	0	1	1	1

* REMP = rapid eye movement period; BL = baseline night; LSD = experimental night with LSD; FU = follow-up night.

The cited readings are mean values which were obtained by taking the first four readings of each REMP before the dreaming was interrupted temporarily by the appearance of Stage 2 EEG. The figures in Table 5 indicate the GSR at the beginning of the three REMPs but do not indicate the overall course during a whole rapid eye movement period (REMP).[4]

The G.S.R. range during a sleep-dream cycle was progressively lower during sleep, in contrast to the basal skin resistance figures reported by Hawkins *et al.* (16). These authors found the basal skin resistance to be greater in the second half of sleep, paralleling that part of sleep associated with a greater amount of dreaming.

Bursts of Stage 2: During the dream periods there were intermittent interruptions with varying amounts of Stage 2 EEG in most of the cycles of all the sessions. During the transitory Stage 2, an isolated single burst of Stage 1 lasting about ten seconds with an isolated REM was observed (Table 6). There were no other related observations at the times of the isolated bursts. The isolated single REM burst, not present on baseline nights, occurred in all LSD cycles and all but one cycle of the follow-up nights.

On both baseline nights the experimenter found *S* pleasant and cooperative, but on the LSD night he was irritable, sarcastic and suspicious. The latter behavior persisted on FU^1, but on FU^2 he again seemed pleasant and friendly.

DISCUSSION

The dream periods on the LSD night were different in a variety of respects from those on the baseline and follow-up sessions. The hypothesized increase in dream time on the LSD night occurred: a still higher percentage of dream time was present on the first follow-up session. The dream periods on the LSD night were different from all other sessions in the following ways: there was marked delay in onset of dreaming after sleep onset; increased gross body movements; increased vocalization; and higher GSR readings. The "drive discharge" hypothesis does not explain the paradoxic effect of LSD in increasing dream time at the same time that activity which is normally associated with non-REMP sleep also increases. After sleep onset, the GSR varied in a repetitive cyclic pattern during the sleep-dream cycles. The lowest GSR readings occurred in Stage 4 EEG: the highest readings were in Stage 1 EEG.

[4] During these sessions the GSR readings were high at the onset of sleep (more so on LSD) but gradually decreased to a range where the readings varied in a cyclic fashion fairly consistently. During an individual sleep-dream cycle the resistance (in K ohms) was lowest in Stage 4, with progressive increases in resistance readings as the stage of sleep went from Stage 4 to Stage 1 with REMs. There were minor fluctuations of the GSR on the meter during an REMP, with a decrease in resistance paralleling an interruption of dreaming. A burst of REMs frequently produced a small simultaneous immediate increase in the K reading. During a given REMP, it was observed on monitoring that a decreasing GSR foretold of the cessation of the REMs and the end of the dream period.

The LSD night dream time was approximately five per cent higher than the baseline sessions. The dream time increase was even more marked on FU^1, suggesting that the dream stimulus effect of LSD continued until FU^2 when the percentage returned to baseline levels. At the same time *S*'s irritability, hostility and suspiciousness, as seen on the LSD night and FU^1 night, had dissipated on FU^2 and he was as pleasant as during the baseline nights. On the LSD night *S* was awakened just after the onset of his fourth REMP, and it seems very probable that the amount of total nightly dream time would have exceeded all other sessions if he had been permitted to sleep another hour. (Usually each succeeding dream period increases in duration.)

During normal sleep there is a minimum of gross body movements detectable on the EEG during REMPs as compared to NREMP sleep. Jouvet (17) and others have reported that the desynchronized EEG pattern associated with REMPs in cats was marked by the absence of electromyographic activity of the neck muscles. In humans, evidence indicates that gross body movements frequently seem to function as transition points ending the REMP, as well as fragmenting dreams within a dream period. In this study, however, on the LSD night there were frequent gross body movements, markedly exceeding the other sessions. Next in order of frequency was FU^1, followed by FU^2, BL^2 and BL^1, with some parallel in the order of dream time percentage.

Vocalization is normally minimal or absent during REMPs, and on the baseline sessions none occurred. There was a marked amount of vocalization on the LSD night, with a variety of grunts, snorts, moans and meaningful exclamations such as "uh" and "uh-uh." Such expressions did not occur on any other session. Next in order of vocalization frequency was FU^2, and FU^1 recorded only one. Vocalization occurred with about equal frequency both with and without associated gross body movements.

Snoring in humans has been reported as rare (12) or nonexistent during REMPs. For snoring to occur it is necessary for the pharyngeal area to be relaxed or hypotonic. Some investigators have used the usual absence of snoring during REMPs to support the claim that *Ss* are then more alert (pharyngeal tonicity prevents snoring) than they are in other EEG stages of sleep (with snoring). That reasoning also implies that *Ss* cannot experience the exciting imagery of a dream and snore at the same time. In this study, snoring occurred on BL^2, but during the fourth REMP (hence not considered in the data as previously noted). Snoring occurred during all LSD REMPs and in the third REMP of both follow-up nights. This *S*'s snoring lasted varying amounts of time, some intermittent for one to two minutes, but at other times *he snored during much or all of the REMP*, with some durations of approximately 20 minutes without interruption of the snoring.

During REMPs of all sessions there were instances of temporary interruption of Stage 1 REMP with a Stage 2 EEG pattern. During the latter, an isolated burst of Stage 1, lasting approximately ten seconds with a single isolated REM, occurred during all LSD REMPs and during all but one REMP on the follow-up sessions, and not during the baseline sessions. These REM bursts appeared to be related to LSD and its probable sustained effects. This may be a manifestation of the REMP-stimulating effect of LSD, with persistence on the follow-up nights, even though the dream time percentage returned to baseline level on the second follow-up night.

In the only other study thus far reported, Muzio (22) administered small doses (10 to 30 micrograms) of LSD to young adults at bedtime. Some of his findings were very similar to those of the present study.

These were: 1) a general increase in body motility and restlessness; 2) maintenance of the periodicity of the sleep cycle; 3) appearance of REMs during Stage 2; and 4) an increase in dream time percentage on the LSD night. The methodology of the experiment and the LSD dosage were different and may be related to some differences such as the presence of occasional EEG and behavioral arousals, a marked prolongation of the second REMP in four *S*s with a subsequent decrease in the duration of the third, fourth and fifth REMPs. However, some findings in the present study on the LSD night were not found by Muzio *et al.* (22), including: 1) a progressive increase in the duration of the REMPs; and 2) a continued increase in dream time percentage on FU^1, followed by recovery to baseline levels on FU^2. The commonality of LSD in these two studies makes the comparisons striking.

In spite of the increased motoric discharge and vocalization which might be taken to mean that *S* was more aroused, GSR readings were higher. This paradox is comparable to other indices regarding the place that Stage 1 occupies along a "depth" dimension. A state of arousal during REMPs is indicated by the desynchronized EEG pattern, absence of snoring, increased irregular respiration and increased heart rate; increased arousal thresholds and GSR readings presumably indicate a "deep" state. One study (16) concluded that the increase in GSR indicated that dreaming was "deep" sleep. However, Darrow (6) suggested that disturbing conditions which occasion the release of epinephrine in the bloodstream might be associated with a decrease of the adaptive palmar sweat secretion and therefore with an increase in GSR instead of a decrease. He found that the GSR *decreased* following intravenous injection of $\frac{1}{20}$ cc. of adrenalin in one *S*.

"Excessive and disturbing excitation in which primitive automatic mechanisms are released from higher level control tends to be associated with a marked increase in blood pressure and may provide indication that there has been a departure of galvanic measurements from parallelism with higher level facilitative processes. Marked blood pressure increase in the absence of overt activity may provide indication of intracortical conflict, and, possibly of 'relative functional decortication'" (6, p. 87).

It is therefore possible, in the light of Darrow's evidence, for an increase in GSR to be associated with autonomic activation rather than a state of tranquility.

The half-hour postponement of the first REMP on the LSD night may be explained neurophysiologically on the basis of the indole-like chemical structure of LSD. This postponement may be compared to the inhibitory effect of amphetamine on REMPs (amphetamine has an indole-like chemical structure). LSD produced no dream deprivation. On the FU^1 night the continued LSD effect was accompanied by a dream time percentage which was three per cent more than the LSD night. The chemical structure in itself would not explain the other findings of increased dream time, indicators of activity during REMPs, or isolated REMP bursts.

The findings in the present study are not fully explained by either the "drive discharge" hypothesis or the "indole structure" hypothesis. Some other hypothesis may fit the findings better. For example, the direct pharmacologic effect of LSD to increase dream time may have created a special problem for this constricted and avoidant *S*. The increase in gross body movement and vocalization may have represented attempts by *S* to terminate the dream by either awakening, shifting into NREMP sleep, or acting out the drama of the dream in movements and vocalizations. Such an interpretation would assume that for this *S* dreams under the influence of LSD were particularly disturbing. The

possibility of having "some wild dreams" which might be revealing must have been threatening to the degree that he might have reacted psychologically against it for protection. The latter can be seen in his restlessness and vocalizations, perhaps a response to the dream stimulus of LSD. His stated reluctance to have "wild dreams" might also explain the postponement of the first REMP on the LSD night. In spite of the motoric discharge, the *S* continued dreaming without recourse to somnambulism and with sleep preserved. The increased gross body movements and vocalization during REMPs may have helped to achieve partial discharge of the pharmacologically intensified instinctual drives with preservation of sleep.

This *S* has been described by his wife as talking and, rarely, walking in his sleep. The experimenter's request that he keep a dream log prior to the study was apparently so threatening to *S* that he said he recalled little or no dream content for a period of two weeks. For example, at best he could only recall one dream about grade school, which he could not amplify. More striking was his inability to recall a dream when awakened in the midst of a REMP at the end of the LSD session.

The effect of LSD in a *S* with a relatively normal personality might be profoundly different from the effect on this *S*. The signs of activity during REMPs and postponement of the onset of dreaming might be replaced by minimal activity in prolonged REMPs, a much higher increase in dream time percentage, and the onset of dreaming might be soon after sleep onset, if it is assumed that the paradoxic "arousal" indicators can be accounted for by defensive activity as well as the pharmacologic effect of the drug.

This study reported here has a number of limitations. Only one *S* was used, and it is hard to generalize to a larger population. In the absence of studies dealing with the sleep-dream cycle of chronic alcoholics, it is difficult to know to what extent departures from the normal sleep-dream cycle in this *S* during the predrug (LSD) baseline sessions were related to the experimental situation, *S*'s personality, or some unidentified neurophysiologic factors. There was no effort to control for a possible placebo effect, although various double-blind LSD studies have shown that the drug has distinctive effects. It is known that this *S* had certain anticipations about the effect of LSD on dreaming. It is hard to estimate the extent to which his readiness to have "wild dreams" following the administration of LSD may have in itself disturbed the sleep-dream cycle, independent of any pharmacologic effect. Only a placebo study could answer this question conclusively. Nevertheless, the variety of effects and the surprising nature of some of these warrant closer examination.

SUMMARY

The effect of LSD on the sleep-dream cycle was studied in a man, including examination of two baseline nights, followed by a day with administration of 300 micrograms of LSD and two follow-up nights. *S* was allowed to sleep through the nights without interruption until 6 a.m. Various spontaneous responses during sleep and dreaming were noted. Galvanic skin resistance was measured on the LSD and follow-up sessions after technical failure on baseline sessions.

The anticipated increase in dream time on the LSD night occurred; a still higher percentage of dream time was present on the first follow-up session. The dream periods on the LSD night were different from all of the other sessions in the following ways: marked delay in onset of dreaming after sleep onset; increased gross body movements; increased vocalization; higher GSR readings. After sleep onset, GSR varied in a repetitive cyclic pattern during the sleep-dream cycles. The lowest GSR readings occurred with Stage 4

EEG; the highest readings were with Stage 1 EEG, the dream period.

These suggestive findings would seem to warrant validation studies.

REFERENCES

1. Aserinsky, E. and Kleitman, N. Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. Science, *118:* 273–274, 1953.
2. Bercel, N. A., Travis, L. E., Olinger, L. B. and Dreikurs, E. Model psychosis induced by LSD-25 in normals. Arch. Neurol. Psychiat., *75:* 588–611, 1956.
3. Carrau, A. B. Reliability of activation level during adaptation to stress. Science, *129:* 764, 1959.
4. Charalampous, K. D. and Kinross-Wright, J. Hallucinogens: Tools for research or therapeutic agents. Texas J. Med., *59:* 848–852, 1963.
5. Chandler, W., Blewett, D. H., Smith, C. M. and Hoffer, A. Use of lysergic acid diethylamide in the treatment of alcoholism. Quart. J. Stud. Alcohol, *20:* 577–590, 1959.
6. Darrow, C. W. The galvanic skin reflex (sweating) and blood pressure as preparatory and facilitative functions. Psychol. Bull., *33:* 73–94, 1936.
7. Davis, P. A., Gibbs, F. A., Davis, H., Jetter, W. W. and Trowbridge, L. S. The effects of alcohol upon the electroencephalogram (brain waves). Quart. J. Stud. Alcohol, *1:* 602–637, 1941.
8. Dement, W. Dream recall and eye movements during sleep in schizophrenics and normals. J. Nerv. Ment. Dis., *122:* 263–269, 1955.
9. Dement, W. and Kleitman, N. Cyclic variations in EEG during sleep and their relation to eye movements, body motility and dreaming. Electroenceph. Clin. Neurophysiol., *9:* 673–690, 1957.
10. Dement, W. and Kleitman, N. The relation of eye movements during sleep to dream activity: An objective method for the study of dreaming. J. Exp. Psychol., *53:* 339–346, 1957.
11. Duffy, E. The psychological significance of the concept of "arousal" or "activation." Psychol. Rev., *64:* 265–275, 1957.
12. Fisher, H. and Schwartz, B. A. A clinical, electroencephalographic and polygraphic study of sleep in the human adult. In Wolstenholme, G. E. W. and O'Connor, M., eds. *The Nature of Sleep*, pp. 209–231. Little, Brown, Boston, 1960.
13. Fisher, C. and Dement, W. C. Studies on the psychopathology of sleep and dream. Amer. J. Psychiat., *119:* 1160–1168, 1963.
14. Freud, S. *The Interpretation of Dreams*. Standard Edition, Vol. 4 and 5. Hogarth Press, London, 1953.
15. Graham, S. C., Webb, W. B. and Williams, R. L. Alcohol and caffeine: Effect on inferred visual dreaming. Science, *140:* 1226–1227, 1963.
16. Hawkins, D., Puryear, H., Wallace, C., Deal, W. and Thomas, E. Basal skin resistance during sleep and "dreaming." Science, *136:* 321–322, 1962.
17. Jouvet, M. Telencephalic and rhombencephalic sleep in the cat. In Wolstenholme, G. E. W. and O'Connor, M., eds. *The Nature of Sleep*, pp. 188–206. Little, Brown, Boston, 1960.
18. Kamiya, J. Behavioral, subjective and physiological aspects of drowsiness and sleep. In Fiske, D. W. and Maddi, S. R., eds. *Functions of Varied Experience*, pp. 145–174. Dorsey Press, Homewood, Illinois, 1961.
19. Kleitman, N. The nature of dreaming. In Wolstenholme, G. E. W. and O'Connor, M., eds. *The Nature of Sleep*, pp. 349–364. Little, Brown, Boston, 1960.
20. Kleitman, N. *Sleep and Wakefulness*. The University of Chicago Press, Chicago, Illinois, 1963.
21. Kleitman, N. The physiology of dreaming. In Adelson, E. T., ed. *Dreams in Contemporary Psychoanalysis*, pp. 37–57. The Society of Medical Psychoanalysts, New York, 1963.
22. Muzio, J., Roffwarg, H. and Kaufman, E. Alteration in the young adult human sleep EEG configuration resulting from d-LSD-25. Paper presented at the Association for the Psychophysiological Study of Sleep, March, 1964, Palo Alto, Calif.
23. Magoun, H. W. Caudal and cephalic influences of the brain stem reticular formation. Physiol. Rev., *30:* 459–474, 1950.
24. Monroe, R. R., Heath, R. G., Mickle, W. A. and Llewellyn, R. C. Correlation of rhinencephalic electrograms with behavior. Electroenceph. Clin. Neurophysiol., *9:* 623, 1957.
25. Oswald, I. and Thacore, V. R. Amphetamine and phenmetrazine addiction: Physiological abnormalities in the abstinence syndrome. Brit. Med. J., *2:* 427–431, 1963.
26. Rechtschaffen, A., Verdone, P. and Wheaton, J. Reports of mental activity during sleep. Canad. Psychiat. Ass. J., *8:* 409–414, 1963.
27. Schlosberg, H. Three dimensions of emotion. Psychol. Rev., *61:* 81–88, 1954.
28. Schwarz, B. E., Bickford, R. G. and Rome, H. P. Reversibility of induced psychosis with chlorpromazine. Proc. Mayo Clin., *30:* 407–417, 1955.
29. Schwarz, B. E., Sem-Jacobsen, C. W. and Petersen, M. C. Effects of LSD-25 and adrenochrome on depth electrograms in man. Arch. Neurol. Psychiat., *75:* 579–587, 1956.
30. Silverman, A. J., Cohen, S. I. and Shmavonian, B. M. Investigation of psychophysiologic relationships with skin resistance measures. J. Psychosom. Res., *4:* 65–87, 1959.
31. Wang, G. H., Stein, P. H. and Brown, V. W. Brain stem reticular system and galvanic skin reflex in acute decerebrate cats. J. Neurophysiol., *19:* 350–355, 1956.

THE JOURNAL OF NERVOU

A PSY

Sleep paralysis
of the narcolepsy
narcolepsy proper
ysis, and hypnago
however, become
that sleep paralys
isolated symptom
12, 19–23, 25–27).

To the afflicted
sleep paralysis is
Just before falli
awakens he finds l
yzed. He is aware
is unable to move
feel that the spel
could only manag
movement. Occasi
to groan and ther
of others. Sometin
ciated with hypn
people sitting on
him down, or simil
tions, visual or
generally lasts fro
minutes, although
a patient whose p
a half hours. Re
ously or when th
somebody.

The symptom w
(13, 14) in 1876 a
description includ
from faint tingling
defects of touch o
and hemiplegia (
to paralysis of th
Pfister (15) descr

[1]9 East 82nd Street
partment of Psychiat
lege-Metropolitan H
City.