mental functions (including speech)" (*10*, pp. 87–88).

The ability to recall and sing familiar songs suggests that the right hemisphere plays an equal or greater role in musical memory and in the neuromotor processes of singing. The increasing comprehension of spoken words and use of propositional speech were also reported in the only two similar cases of left hemispherectomy for glioma and one for seizures of late onset in adults (*11*). The consistent findings indicate that, in addition to comprehension of speech, the adult right hemisphere alone is capable of more than the automatic primitive utterances first described by Jackson.

The ability to discriminate color, solve abstract and concrete mathematical problems, engage in purposeful movements with the left hand, and to perform at a nearly normal level in nonlanguage tests of "higher" mental functions indicates either that these functions are not exclusively or predominantly "localized" in the adult dominant hemisphere, or that, following removal of this hemisphere, the right hemisphere has the capacity to amplify previously smaller contributions to these functions. Although a spastic right hemiplegia and right hemianopsia have shown no improvement during the first 6 months following surgery, this case indicates that adult man may survive the removal of a dominant hemisphere that has matured normally until the development of an otherwise incurable and inevitably fatal glioma.

AARON SMITH
Nebraska Psychiatric Institute,
602 South 44th Avenue,
Omaha, Nebraska 68105
C. W. BURKLUND
Veterans Administration
Hospital, Omaha

References and Notes

1. O. L. Zangwill, *Cerebral Dominance and Its Relation to Psychological Function* (Oliver and Boyd, Edinburgh, 1960).
2. A. L. Benton, *Can. Psychol.* **6a**, 332 (1965).
3. W. E. Dandy, *J. Amer. Med. Assoc.* **90**, 823 (1928).
4. R. A. Krynauw, *J. Neurol. Neurosurg. Psychiat.* **13**, 243 (1950).
5. A. S. Obrador, *Cerebral Localization and Organization*, G. Schaltenbrand and C. N. Woolsey, Eds. (Univ. of Wisconsin Press, Madison, 1964), pp. 133–154.
6. W. J. Gardner, L. J. Karnosh, C. C. McClure, Jr., A. K. Gardner, *Brain* **78**, 487 (1955).
7. R. Zollinger, *Arch. Neurol. Psychiat.* **34**, 1055 (1935).
8. H. G. Crockett and N. M. Estridge, *Bull. Los Angeles Neurol. Soc.* **16**, 71 (1951).
9. J. H. Jackson, *Selected Writings of John Hughlings Jackson* (Basic Books, New York, 1958), vol. 2.
10. A. R. Luria, *Higher Cortical Functions in Man* (Basic Books, New York, 1966).
11. L. A. French, D. R. Johnson, G. H. Adkins, *J. Lancet* **81**, 58 (1961).
12. Supported by PHS research grant HD-00370. We thank the patient (E.C.), Drs. S. J. Dutch, R. J. Ellingson, J. McBeath, J. Wiley, N. Timmons, and many colleagues at the Omaha Veterans Hospital for their cooperation; and D. Moran for assistance in extensive testing.

25 July 1966

"Dream Deprivation": Effects on Dream Content

Abstract. *Dream content elicited following the selective deprivation of rapid-eye-movement sleep was intensified compared to that elicited under nondeprivation conditions. This effect was observed both for repressers and for sensitizers, but was significant only for repressers. On nondeprivation nights, the dream periods of sensitizers were shorter and their dreams more intense than those of repressers.*

The finding (*1*) that dreaming is associated with a random, low-voltage electroencephalogram (EEG) pattern (ascending EEG stage 1) in conjunction with intermittent bursts of conjugate rapid eye movements (REMs), and that this pattern recurs four or five times during a typical night's sleep (*2*), has prompted renewed interest in the possible functional significance of dreaming. Experimental attempts to assess the importance of dreaming for human subjects have employed a method of dream deprivation, in which subjects are awakened at the onset of each episode of ascending stage 1 sleep and are thus prevented from experiencing the organismic state in which dreaming occurs. Early studies employing this method found progressively decreasing latencies to the recurrence of dream periods on deprivation nights, a compensatory increase in this stage of sleep on uninterrupted recovery nights following the deprivation treatment, and increased anxiety, irritability, and appetite during the deprivation period. These results were interpreted as a "buildup of a pressure to dream" (*3*).

More recently, however, failure to observe the reported behavioral changes with human subjects (*4, 5*) and demonstration of the sleep-cycle effects of dream deprivation in intact and chronic pontile cats (*6*) have led to the interpretation that the supposed effects of the deprivation of a psychological experience are actually the effects of the deprivation of a physiological stage of sleep with some unknown biological significance (*7*). While it may ultimately prove impossible to distinguish the effects of the deprivation of this stage of sleep from the effects of dream deprivation, the present study shows that the nature of dream content, as well as of the sleep cycle, is affected by the selective deprivation of ascending EEG stage 1. In particular, the content of a dream elicited following such deprivation is intensified as a result of this manipulation.

Each of twenty male subjects slept in quiet, darkened laboratory rooms for two nonconsecutive nights. Ten of the subjects were among low scorers (repressers, those who react to threatening stimuli with avoidance defenses such as denial) and ten among high scorers (sensitizers, those who react to threatening stimuli with approach defenses such as intellectualization) on Byrne's Repression-Sensitization Scale (*8*) of the Minnesota Multiphasic Personality Inventory (MMPI). The subjects selected represented the top and bottom fifth of the distribution of test scores of male volunteers from an introductory psychology class.

Continuous EEG and electro-oculogram (EOG) recordings were taken during the night. Electromyogram recordings were also taken from the submental region, as a sharp drop in tonus of neck and chin muscles generally precedes the onset of ascending EEG stage 1 by a minute or so and can be used as a signal of such onset (*5, 9*). Ascending EEG stage 1 onset was determined by the appearance of a random, low-voltage EEG pattern accompanied by rapid eye movements, or a sudden drop in submental muscle tonus, or both. On experimental nights, the first four REM periods were interrupted at the first sign of ascending stage 1 onset. On control nights, four awakenings were made during non-REM sleep so as not to interfere with the occurrence of REM sleep. The fifth REM period on experimental nights and the third one on control nights were allowed to proceed for 5 minutes, after which awakenings were made for retrieval of dream content. Half of the repressers and half of the sensitizers, selected at random, were run in the order experimental-control, while the remaining subjects were run in the reverse order.

The elimination of REM sleep and the progressive shortening of the periods between "attempts at" REM sleep on experimental nights necessitated the use of the third, rather than the fifth, REM period on control nights to achieve rough comparability of sleep time preceding (and time of night of) the content-retrieval awakenings on the two nights. Even so, control-night content awakenings were almost invariably made later in the night and after more sleep (68.6 minutes more, on the average) than were the comparable experimental night awakenings. This situation worked against the hypothesis of increased dream intensity following dream deprivation, since it has been shown that dream intensity increases over the course of the night (*10*). While the experimental design may also seem in some sense to favor the hypothesis under test, in that a "later" REM period is used on experimental than control nights (fifth versus third), it should be noted that experimental-night content awakenings were elicited from the first sustained episode of REM sleep on that night, the other four REM periods having been terminated by an awakening at the first sign of their onset. Initial control-night REM episodes, on the other hand, were uninterrupted, and the control-night content awakenings were thus made during the third sustained episode of REM sleep on that night.

On deprivation and pseudo-deprivation awakenings, the light was turned on in the subject's room, and the experimenter entered and took readings of strength of grip from a dynamometer which the subject was to squeeze four times from a prone position. Subjects had been told that the purpose of the study was the investigation of associations among stage of sleep, strength of grip, and mental content, and were thus unaware that the deprivation of REM sleep was the actual purpose underlying awakenings on which dynamometer readings were taken on experimental nights.

That the experimental-night awakenings produced a deprivation effect on the sleep cycle is evident in the briefer intervals of non-REM sleep between the onset of REM episodes on experimental than on control nights (Table 1). The dynamometer technique of producing complete arousal rules out the possibility that such results reflect incomplete arousal rather than the effects of selective sleep deprivation.

Table 1. Effect of deprivation awakenings on length of inter-REM intervals. The intervals are measured in minutes.

Inter-REM intervals				
Interval 0–1	Interval 1–2	Interval 2–3	Interval 3–4	Interval 4–5
Experimental night				
91.85	60.85	18.15	21.45	19.22
Control night				
90.05	80.35	66.11		
Probability				
n.s.*	.005	.001		

* Not significant.

The dreamlike fantasy (Df) scale of Foulkes, Spear, and Symonds (*11*) was used in all analyses of the content-awakening interviews. This is an eight-point scale with a value of 0 assigned to "No recall, feels mind was blank," and a value of 7 assigned to "Recall of content which was perceptual, hallucinatory, and bizarre." Two judges, both completely naive with respect to subject or treatment variables, rated the dream reports with high agreement (reliabilities of .88 and .95, respectively, for experimental- and control-night reports). Two-rater averages were used in all analyses of Df ratings.

The effects of deprivation awakenings on dream content are summarized in Table 2. Considering all subjects, it is evident that dreams were more intense on experimental nights than on control nights. This intensification of the dreaming process was observed for 12 of the 16 subjects who had different Df scores on experimental and control nights and was statistically significant (*12*). Four subjects showed no change in Df ratings from control to experimental nights, and four a decrease in dream intensity following deprivation. Although an equal number (six) of repressers and sensitizers had higher Df scores on the experimental night than on the control night, the deprivation effects were significant only for repressers, whose experimental-versus control-night mean difference in Df ratings was roughly three times as large as that for sensitizers.

Table 2. Effect of deprivation awakenings on dream content as rated on the 8-point dreamlike fantasy scale (Df ratings of 0 to 7). R, repressers; S, sensitizers; E, experimental; C, control; *p*, probability.

Repressers	Sensitizers	*p* (R vs. S)	All subjects
Both nights			
5.60	5.78	.10	5.69
Experimental night			
6.50	6.10	n.s.*	6.30
Control night			
4.70	5.45	.06	5.08
E vs. C(p)			
.05	n.s.*		.025

* Not significant.

Examination of the EOG patterns during REM periods preceding content-retrieval awakenings further revealed that a larger percentage of 2½-second intervals of such periods contained one or more eye movements on experimental nights than on control nights (28.3 percent versus 21.7 percent, respectively, $p<.025$). This effect also was significant for repressers (25.3 percent on experimental nights versus 18.5 percent on control nights, $p<.025$) but not for sensitizers (31.3 percent on experimental nights versus 24.9 percent on control nights; difference not significant). The close correspondence between differences in prewakening eye movement frequency and differences in the intensity of subsequently reported dreams parallels earlier findings that active dreams are preceded by more eye movement than are passive ones (*13*).

Our sensitizer subjects scored high on MMPI pathology scales (for example, an average score three standard deviations above the general population mean on the MMPI schizophrenia scale), while the repressers had essentially normal MMPI profiles. The sensitizers had, in the absence of the deprivation treatment, more intense dream content than did the repressers—a duplication of Foulkes and Rechtschaffen's (*14*) observation that high MMPI-scoring subjects have dreams of greater intensity than do more normal subjects. Sensitizers also had, in the absence of deprivation, briefer REM periods than did repressers (mean summed control-night REM time for the first two REM periods was 45.6 minutes for repressers and 33.0 minutes for sensitizers, $p<.025$)—a duplication of Monroe's (*15*) observation that high MMPI-scoring subjects have briefer REM periods than do more normal subjects. These two results provide an additional strand of evidence in support of the hypothesis that lessened amounts of dreaming are associated with an intensification of the dreaming process.

These results suggest that the term "dream deprivation," as more clinically oriented investigators have suspected all along (*16*), may still have some validity. Interference with the stage of sleep in which dreaming occurs, either by experimental manipulation or by spontaneously generated reductions in

REM sleep, does seem to increase subsequent dream intensity, that is, the selective deprivation of ascending EEG stage 1 has immediate experiential, as well as the heretofore observed sleep-cycle, effects.

These results are also consonant with the Freudian conception of dreaming as a safety valve, which played a large role in earlier interpretations of the effects of dream deprivation. The dream content of sensitizers, whose safety valve is somehow always partially capped, and that of repressers under the capping effects of experimental deprivation, both exhibit intensification. In general, our results seem to indicate that the less a person dreams, the more intense are his dreams (*17*).

TERRY PIVIK
DAVID FOULKES

Department of Psychology,
University of Wyoming,
Laramie 82071

References and Notes

1. E. Aserinsky and N. Kleitman, *J. Appl. Physiol.* **8**, 1 (195 → W. Dement and N. Kleitman, *J. Exp. Psychol.* **53**, 339 (1957).
2. W. Dement and N. Kleitman, *Electroencephalog. Clin. Neurophysiol.* **9**, 673 (1957).
3. W. Dement, *Science* **131**, 1705 (1960).
4. F. Snyder, *Archiv. Gen. Psychiat.* **8**, 381 (1963).
5. A. Kales, F. Hoedemaker, A. Jacobson, E. Lichtenstein, *Nature* **204**, 1331 (1964).
6. M. Jouvet, in *Aspects Anatomo-Fonctionnels de la Physiologie du Sommeil* (Centre National de la Recherche Scientifique, Paris, 1965), pp. 397–449.
7. W. Dement, in *ibid.*, pp. 571–611.
8. D. Byrne, *Psychol. Rep.* **13**, 323 (1963).
9. R. J. Berger, *Science* **134**, 840 (1961).
10. D. Foulkes, *The Psychology of Sleep* (Scribner, New York, 1966), pp. 92–95.
11. ———, *ibid.*, pp. 189–190.
12. All probability values reported in this paper are one-tail, since the alternative to the null hypothesis is, in every case, specific as to direction. Tests of sleep-cycle effects of deprivation and of represser-sensitizer differences in both sleep cycle and dream content are attempted replications of earlier published findings. In addition, results in the same direction as our finding of increased dream intensity following "dream deprivation" were obtained in an unpublished study by Rechtschaffen and Foulkes, but their results, obtained under somewhat different conditions than ours, did not attain statistical significance. Student's *t*-test was used to evaluate sleep-cycle and eye-movement effects, and the Wilcoxen matched-pairs signed-ranks test for the dream content effects of the deprivation variable. The Wilcoxen rank-sum test was employed in evaluating represser-sensitizer differences in dream context.
13. R. Berger and I. Oswald, *Science* **137**, 601 (1962).
14. D. Foulkes and A. Rechtschaffen, *Percept. Motor Skills* **19**, 983 (1964).
15. L. J. Monroe, unpublished thesis, University of Chicago, (1965).
16. C. Fisher, *J. Amer. Psychoanal. Assoc.* **13**, 271 (1965).
17. Supported by grant GS-860 from NSF to D.F. Based on a thesis submitted by T.P. to the department of psychology, University of Wyoming, in partial fulfillment of the requirements for the degree of Master of Arts. We thank Ethel Meginness and John Ahrens for rating dream reports, and the latter for performing the eye-movement counts.

9 May 1966

"Copulation-Reward Site" in the Posterior Hypothalamus

Abstract. *Posterior hypothalamic self-stimulation of male rats, in which monopolar, platinum electrodes had been bilaterally implanted, increased after systemic injection of testosterone. Constant stimulation to the same site elicited immediate copulation with estrous female rats. During constant stimulation, males would press a bar to open a door for access to females. Even after ejaculation, males continued to open the door and to display sexual activity until stimulation terminated. Posterior hypothalamic stimulation is like normal sexual stimulation; it is rewarding, the reward varies with the amount of the sex hormone, and it elicits motivated copulation.*

The reward of self-stimulation of the brain has been linked to normal appetitive behavior, particularly feeding. Self-stimulation of the lateral hypothalamus increases during food deprivation (*1*) and decreases after food intake (*2*). Stimulation of the same site elicits voracious feeding (*1, 2*). Even if a learned response is required to obtain food, the rat responds as soon as stimulation begins (*3*). Thus lateral hypothalamic stimulation not only elicits rewards that vary with food intake but it also motivates feeding.

On the basis of the discovery of a "feeding-reward site," we decided to look for a "copulation-reward site." Olds (*4*) found that in some areas of the brain self-stimulation decreased after castration and increased after testosterone replacement. Herberg (*5*) confirmed this for a specific site in the posterior hypothalamus where self-stimulation elicited ejaculation. If somewhere in this posterior region self-stimulation is related to mating, in the same way that lateral hypothalamic self-stimulation is related to feeding, stimulation should motivate an animal to copulate. We have explored this possibility and now report the results.

The subjects were ten sexually experienced male Sherman rats with monopolar, platinum electrodes (0.023 cm outside diameter) bilaterally implanted in the posterior hypothalamus (*6*). The implants were made perpendicular to the skull, 4.5 mm anterior to the intra-aural line, 1.2 to 1.5 mm lateral to the midline, and 8.5 mm below the surface of the cortex.

Females for mating tests were brought into constant behavioral estrus by subcutaneous placement of two 26-gauge stainless steel tubes coated at the tips with estradiol (*7*).

The electrical stimulus was a train of 100 cy/sec, monophasic, negative, 0.1-millisecond square pulses passed through an isolation transformer to exclude direct current and produce a biphasic wave form. The intensities used were between 0.1 and 0.2 ma. All tests were conducted in a 46-cm, circular, opaque, chamber with a glass floor and an underview mirror. For self-stimulation, a lever was mounted on the chamber wall. Each time this lever was pressed it triggered a 0.5-second train of stimulation.

Because rats are most likely to be sexually active at night, dim red illumination was used for observation of behavior during the dark period of a reverse day-night cycle. Brain stimulation tests began a week after the implantation of electrodes. The male rat to be tested was allowed 30 minutes to become acclimated to the observation cage before a female rat was placed in the cage. The male then received continuous brain stimulation in 3-minute periods alternated with 3-minute periods when there was no stimulation. Mounts, intromissions, and ejaculations were recorded on an event recorder.

In these tests copulation became stimulus-bound, that is, the males copulated repeatedly when stimulation was on but seldom when it was off. At the end of one test session the male was resting far from the female while the stimulus was left off for 12 minutes; when the stimulus was again turned on, the male began copulating within 10 seconds (Fig. 1). Even after ejaculating, the male remained sexually excited, often sniffing, nudging, and, in several instances, mounting the female until stimulation terminated (Fig. 1). The other rats behaved similarly (see Fig. 2).

Unlike copulation elicited from the anterior hypothalamus (*8*), the overall frequency of copulation during stimulation was not unusually high. The striking fact is the extent to which stimulation controls copulatory behavior (Figs. 1 and 2). At the beginning of a test session the animals sometimes copulated whether stimulated or not, but after one or two stimulation periods they rarely copulated unless stimulated.

Stimulus-bound copulation was a per-