handed subjects are visibly greater than those of the left lobe. Only for one left-handed subject, who claimed to be strongly ambidextrous, are the responses of the left lobe visibly greater than those of the right lobe. It is apparent in the tracings of the right-handed subjects that no consistent tendency exists for the responses of one lobe to be markedly greater than those of the other lobe.

For four subjects (top four rows) the left-lobe responses are clearly greater than those of the right lobe. In all other cases, except one (bottom row), responses of approximately equal magnitude were obtained from both lobes. For the one subject the right-lobe response was markedly greater than that of the left lobe.

These experiments indicate that greater responses to flash stimuli tend to be elicited from the right occipital lobe of left-handed subjects than from the left lobe, whereas no consistent differences were demonstrated for right-handed subjects. The experiments further indicate that the right-lobe responses, relative to those of the left, are greater for left-handed than for right-handed individuals. We conclude, therefore, that the differential amplitude of the responses of the two lobes is related to handedness. Since, however, there is considerable overlap between the two groups in the relative magnitude of the responses obtained from the two lobes, handedness cannot be predicted with certainty for a single individual on the basis of observed lobe differences. More data are needed to establish the probability of being correct when predicting handedness from such lobular differences.

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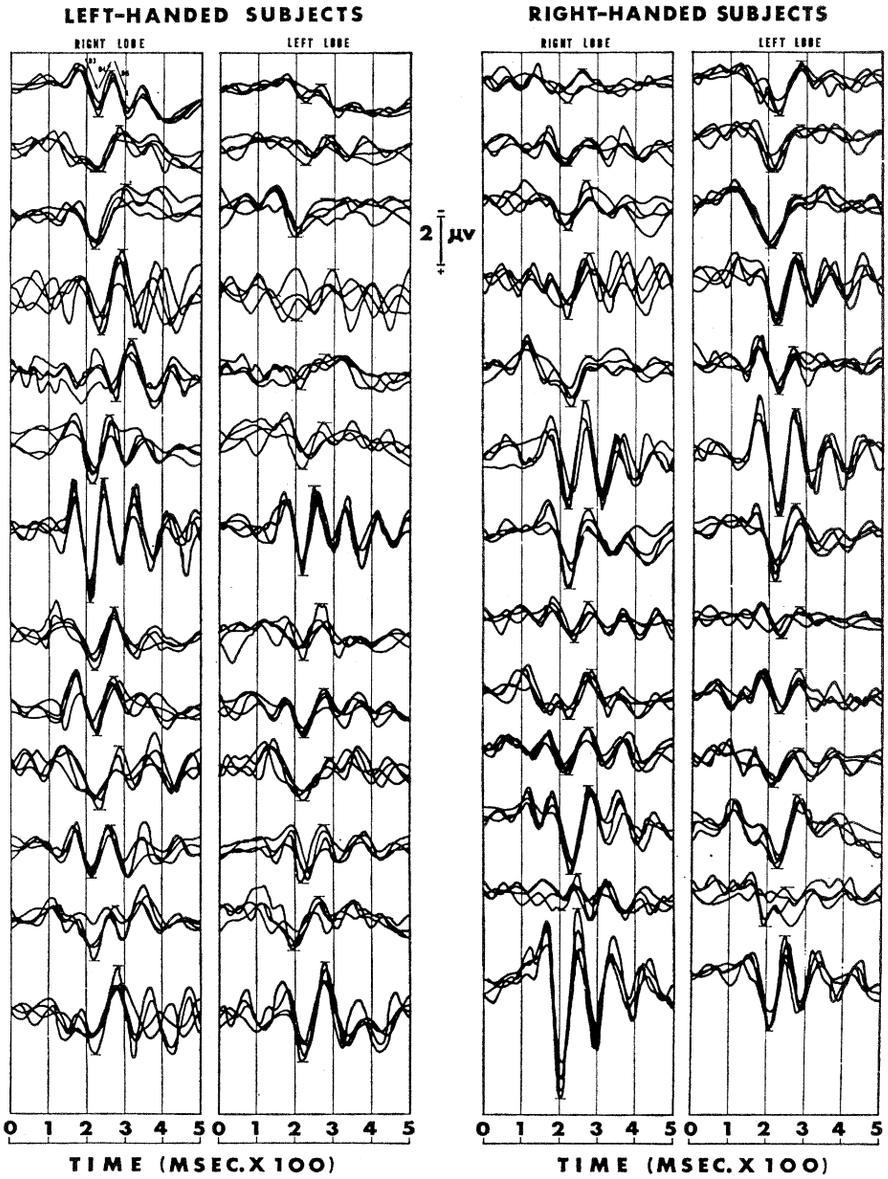

Fig. 2. Evoked cortical responses of right- and left-handed subjects to flashes presented in right and left visual fields.

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Sleep: The Effect of Electroconvulsive Shock in Cats Deprived of REM Sleep

Abstract. *Three cats were deprived of rapid-eye-movement (REM) sleep for 10 days, and three were deprived for 12 days. All cats received an electrically induced convulsion on each of the last 3 days of deprivation, as well as on the 1st recovery day just prior to sleep onset. As controls, four cats were deprived of REM sleep for 12 days and one was deprived for 10 days; the controls received no convulsions. Compensatory increases in REM sleep during recovery days were present in the convulsed animals, but were substantially lower than the recovery increases of control animals. During recovery REM sleep, convulsed cats did not display the exaggerated bursts of eye movements and body twitches seen in the nonconvulsed controls.*

A major consequence of the selective deprivation of rapid-eye-movement (REM) sleep, whether accomplished by arousals at the onset of each REM period (*1*) or by drugs (*2*), is an abrupt rise in the amount of this phase during recovery.

In a recent study (*3*), REM sleep in cats was significantly lowered following electroconvulsive shock (ECS). The continued daily reduction of REM sleep in association with one ECS per day for as long as 7 days was not followed by the usual compensatory increase above the base-line level. These results suggested that a portion of the daily requirement for REM sleep was satisfied by the convulsive experience.

If we assume that selective REM deprivation intensifies the need for REM sleep, then convulsions should have an opposite and ameliorating effect. Consequently, a reduced compensatory rebound would be expected if ECS were administered during the deprivation period. The following experiments were performed to test this possibility.

Permanent electrodes were implanted in 11 adult cats, nine male and two female, for recording of electroencephalographic (EEG), electrooculographic (EOG), and electromyographic (EMG) activity.

Three cats (E1, E2, and E3) were placed on a base-line schedule which allowed 6 hours per day for sleeping. The remaining time was spent on a continuously moving treadmill which completely prevented the occurrence of REM sleep (*4*). Several days were allowed for habituation to this routine, after which the daily sleep periods were continuously recorded for 4 to 6 consecutive days. Time spent in wakefulness, non-REM, and REM sleep was tabulated for each 6-hour session. The cats were then deprived of the REM phase for a period of 10 consecutive days. During this time, they remained on the treadmill continuously except for an occasional 2- to 3-hour period of rest recording—that is, they were removed from the treadmill and were allowed to sleep while the EMG was recorded. Throughout the rest recording they were awakened at the first sign of a drop in EMG activity. A single ECS was administered by use of a reported technique (*3*), on the 8th 9th, and 10th deprivation days, and prior to recording on the 1st recovery day. During recovery, the cats again were allowed to sleep 6 hours per day. Fourteen consecutive daily sleep periods were continuously monitored, and all other time was spent on the treadmill. Three additional cats (E4, E5, and E6) were placed on an 8-hour base-line sleep schedule and their sleep periods were recorded for 4 to 10 days. They were then deprived of REM sleep for 12 consecutive days in the same manner as the cats on the 10-day schedule. Convulsions were administered on the 10th, 11th, and 12th days of deprivation, as well as on the 1st recovery day. Recovery sleep was recorded for 6 to 12 consecutive days.

Five cats served as controls. After their baseline recordings, four (C2 through C5) were deprived of REM sleep for 12 days, and one (C1) was deprived for 10 days. These animals were allowed to recover without being convulsed.

The mean REM sleep for the three cats deprived for 10 days and convulsed was 110 minutes per day during base line and 121 minutes per day for the first 3 recovery days (Table 1). In terms of individual animals, E3 was 20 percent below his base-line mean on the first 3 recovery days, while E1 and E2 were 26 percent and 22 percent above, respectively. The control animal (C1) deprived for 10 days had a mean base-line REM time of 83 minutes, with a mean of 183 minutes for the first 3 recovery days, which represented an elevation of 120 percent.

Mean base-line REM for the cats deprived of REM for 12 days and then convulsed was 112 minutes per day. Following deprivation and convulsions, mean REM time for the first 3 recovery days was 165 minutes or 21, 50, and 60 percent above base-line for E4, E5, and E6, respectively. REM time on recovery days 1 to 3 was significantly greater than the base-line means ($P < .05$; $t = 3.5$, two-tailed t-test for correlated means). Mean recovery REM time for the 12-day controls was 223 minutes, or 94, 60, 71, and 125 percent above base line for C2, C3, C4, and C5, respectively.

Rapid-eye-movement time during recovery in all the convulsed cats was significantly lower than that observed on the corresponding recovery days for equally deprived nonconvulsed controls. ($P < .01$, $t = 4.5$ for cats deprived for 12 days; $P < .01$, $t = 3.1$ for cats deprived for 10 days.) The convulsed animals deprived for 12 days also had higher rebounds than had the convulsed cats deprived for 10 days ($P < .05$; $t = 2.1$, one tailed, t-test).

There was no sign of a delayed rebound in the convulsed animals. REM time was within the base-line range by the 5th or 6th recovery day. The latter was also true of the control animals.

The increased intensity of the phasic components of REM sleep, which accompanies lengthy episodes of REM deprivation, has been described elsewhere (*5*). The most striking and easily observed of these are the intensifica-

Table 1. Mean awake, slow-wave, and REM-sleep times of base line and first 3 recovery days of cats deprived for 10 days and convulsed (E1 to E3); 10 days and not convulsed (C1); 12 days and convulsed (E4 to E6); and 12 days and not convulsed (C2 to C5). Standard deviations are given in parentheses.

Cat	Mean time at base line (min)			Mean time after recovery (min)		
	Awake	Slow wave	REM	Awake	Slow wave	REM
			Cats on 10-day schedule			
E1	39	207	127	33	177	160
	(9)	(16)	(13)	(14)	(11)	(7)
E2	82	180	95	67	188	116
	(31)	(36)	(25)	(37)	(8)	(22)
E3	59	219	108	82	206	87
	(32)	(40)	(20)	(20)	(3)	(6)
C1	79	287	83	47	253	183
	(40)	(25)	(11)	(22)	(26)	(33)
			Cats on 12-day schedule			
E4	113	224	141	100	207	171
	(28)	(21)	(13)	(14)	(18)	(23)
E5	116	247	81	80	287	122
	(20)	(13)	(15)	(28)	(27)	(11)
E6	71	280	127	79	202	203
	(24)	(23)	(15)	(16)	(9)	(9)
C2	123	230	122	97	156	236
	(43)	(31)	(16)	(27)	(28)	(37)
C3	114	239	133	63	205	213
	(31)	(17)	(21)	(39)	(7)	(48)
C4	90	259	133	56	201	228
	(24)	(19)	(13)	(10)	(25)	(29)
C5	196	195	96	101	176	216
	(30)	(20)	(14)	(8)	(38)	(42)

tion om muscle twitches and the increase in the frequency and amplitude of individual eye movements. While exhaustive observations of these phenomena were not made in this study, it was evident that the intensification of twitching and ocular motility seen in the controls was not present in the convulsed cats. On the other hand, the EOG components of the recordings on the 1st recovery day from the convulsed cats were early identical with those obtained during base-line periods.

The data indicate that the development of the REM deprivation-compensation effect is retarded by the occurrence of convulsions during the deprivation period. In this study, our attention was focused on the comparative evaluation of compensatory changes in REM sleep, although neurophysiological and behavioral changes in the waking state also have been observed (*6*). However, such changes usually require much longer periods of REM deprivation than were used in this series of experiments. In the absence of evidence to the contrary, it seems reasonable to assume that behavioral changes would also be retarded or reversed by ECS. We can offer one incidental observation in support of this contention: hypersexual behavior, appearing on the 21st day of REM-sleep deprivation in a male cat, disappeared after several electrically induced convulsions, although the REM-sleep deprivation had not been terminated.

In view of the fact that one ECS per day in nondeprived cats only accomplished a 30- to 40-percent reduction in daily REM time (*3*), the effect of four convulsions in the present series was disproportionately large. The seizures may have been intensified by the prior deprivation. No attempt to quantify the response to ECS was made in this study, but more recently we have observed a marked prolongation of the tonic phase in mice during periods of REM-sleep deprivation (*7*).

Our results suggest a means by which electroconvulsive shock therapy may be effective in depressive psychosis. It is a clinical commonplace that severe depressions are often accompanied by profound sleep disturbances. REM-sleep deprivation appears to be an inevitable consequence of any overall loss of sleep (*8*). If the various manifestations of the REM-deprivation effect (*6, 9*) contribute to the psychotic process, then it is not unreasonable that an alleviation of this effect by electroconvulsive shock therapy may be a critical factor in the successful course of treatment (*10*).

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Solvent Contamination from Volatile Components of a Fiberglass Glove Box

Aflatoxin B_1, isolated and purified in our laboratories, showed an ultraviolet spectrum in complete accord with that reported in the literature. In ethanol, the spectrum exhibits major absorption peaks at 360, 265, and 223 mμ with absorptivity values of 21,700, 13,300, and 23,800, respectively. Some time after these initial spectra were recorded, two fiberglass glove boxes were installed. The plastic binder for the glass fibers is referred to as "polyester resin" in the manufacturer's catalog. Subsequently, all manipulation of the dry aflatoxins and preparation of aflatoxin solutions were carried out in these boxes. To prepare solutions, the crystalline material was washed into a volumetric flask with chloroform. Ultraviolet spectra of aflatoxin B_1 residues, redissolved in ethanol, from solutions prepared in this fashion showed considerable deviation from the normal spectrum. In particular, absorbance values at 223 mμ and 265 mμ showed large increases relative to the absorbance at 360 mμ.

In attempting to determine the cause of these abnormal spectra, we noticed that the abnormalities appeared when the chloroform solvent was exposed to the glove box atmosphere in an open beaker, rather than dispensed from a stoppered flask. This fact plus a persistent "plastic" odor in the boxes led to an examination of chloroform exposed to the glove box atmosphere. Beakers of chloroform, protected from dust fallout, were left in the glove boxes and in other test areas for about 18 hours. Residues from beakers of chloroform, stored within the glove boxes, showed strong ultraviolet absorptions in the 200 to 270 mμ region, with maxima at 210 to 225 mμ and 260 to 265 mμ. When small portions of these chloroform samples were added to "clean" aflatoxin solutions, the abnormal spectrum originally observed was reproduced. No such residue appeared when chloroform exposed to the atmosphere of other areas of our laboratories, including a stainless steel glove box, was evaporated. Infrared spectra of the residues obtained from chloroform exposed in the fiberglass glove boxes correlate with those of a number of the glove box components, particularly with the spectra of the phthalate esters used as plasticizers.

Therefore, if solvents such as chloroform must be handled in a fiberglass glove box, they should be exposed to the glove box atmosphere for *only* the time required for dispensing. This precaution should also protect the glove box, since we have observed that an accumulation of chloroform vapors in the box results in visible changes in the plastic.

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