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Electroencephalograms in Behavior Changes in Cats.

M. D. WHEATLEY, JOHN R. KNOTT, AND W. R. INGRAM.

From the Departments of Anatomy and Psychiatry, State University of Iowa.

Lesions of the ventromedial hypothalamic nuclei in cats usually are accompanied by striking behavior changes, depending upon the symmetry and completeness of these rather small lesions (Wheatley[1]). Postoperatively these animals display a malevolent attitude, are extremely difficult to handle and thereafter are quite untameable. Hyperphagia is frequently part of this syndrome and the animals may become very obese. There are no motor defects and their behavior is well-controlled and purposeful, not to be compared with the sham rage of decorticate preparations. How these small, subcortical lesions produce such effects has not been explained, although a disturbance in hypothalamo-cortical relationships is presumably involved. Since clinical observations[2-11] have indicated that a high proportion of patients showing behavior disorders may have abnormal electroencephalograms, and since Hoagland[2] observed that emotional stimulation may increase delta activity from both hypothalamus and cortex, it was thought that electroencephalographic observations on these cats might indicate some physiological change in cortical activity.

The bilateral lesions were produced by electrolysis, and in all cases of savage behavior were found to be restricted to the immediate vicinity of the ventromedial nuclei. All cats used were in good health at the time of experiment. The experiments were performed as follows: Fifteen mg of beta erythroidin hydrobromide in 0.9% NaCl solution were injected intravenously, and an intratracheal catheter inserted. Artificial respiration was maintained by positive pressure, the respirator being set to provide 27 inspirations a minute. Complete paralysis was maintained by the administration of a solution of erythroidin containing 0.5 mg per cc, given intravenously at a suitable rate. Our experience, in agreement with that of others,[12] indicates that such curarization does not significantly affect the EEG. Silver electrodes were fixed to the scalp with electrode paste and collodion. The numerical designation and position of the leads were: 1, occipital; 2, parietal (vertex); 3, frontal; 4, the common lead, was from the ear. Some of the resistance offered by skull, skin, etc., was eliminated by removal of bone from the skull areas beneath the electrode sites a week or more before the first run. Each animal was subjected to several twenty minute periods of recording. A six channel Offner electroencephalograph with a crystograph recorder was used.

Observations. Records from over a hundred experiments of this type are in general agreement with those of Clark and Ward[13] on normal cats with implanted leads and with similar preparations of our own. Normal

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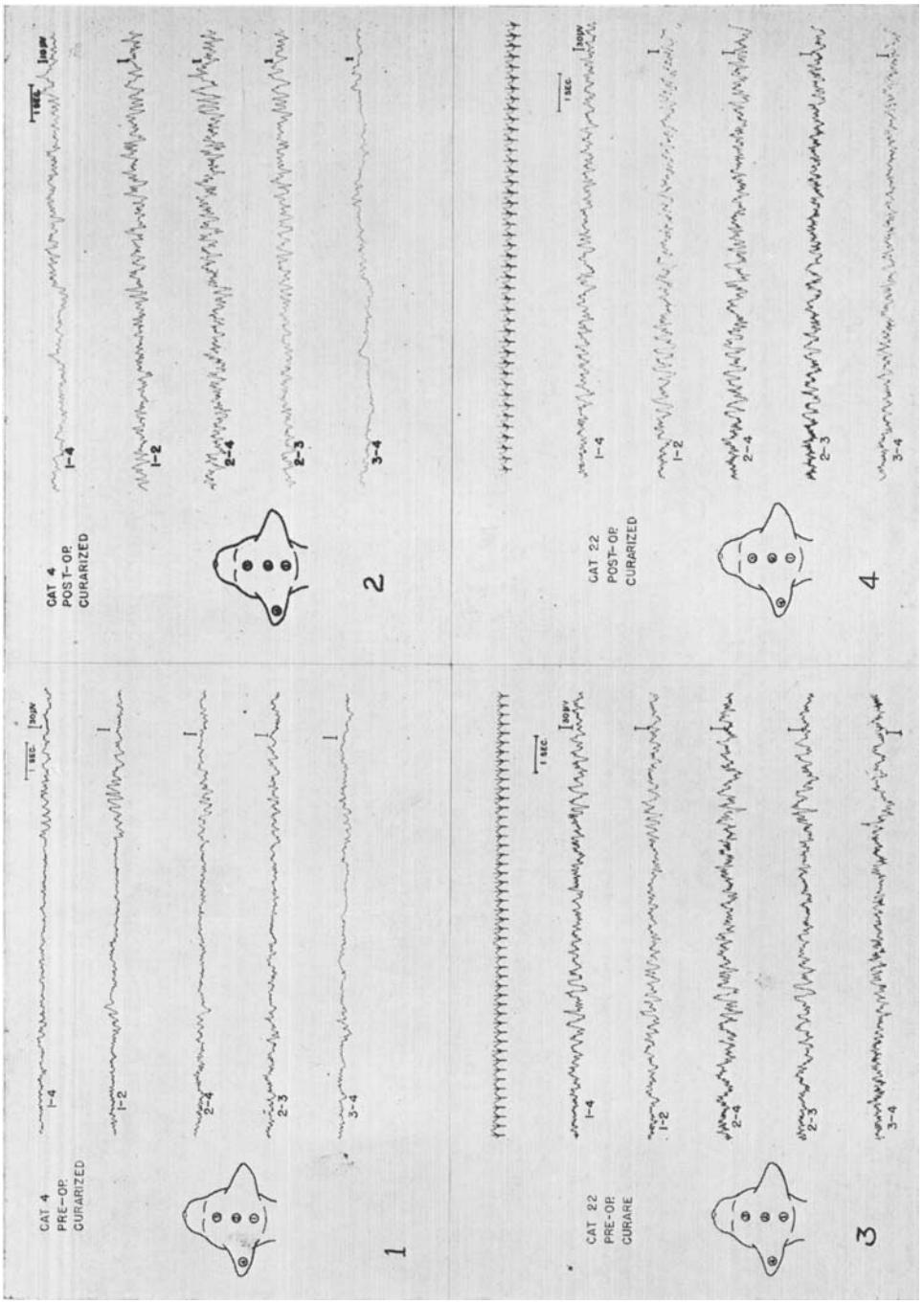

FIG. 1. Preoperative EEG of cat 4, which later became savage.
FIG. 2. Postoperative EEG of cat 4, in savage state.
FIG. 3. Preoperative EEG of cat 22, a control.
FIG. 4. Postoperative EEG of cat 22, a control.

cats show a striking variability in the EEG as to frequency, amplitude and the occurrence of random bursts of fairly uniform frequencies, even when curarized. Differences between normal curarized and non-curarized animals are relatively slight. It is of some interest that EEG patterns resembling those which accompany the appearance of sleep in cats may occur with great frequency under erythroidin. Such patterns may be seen in

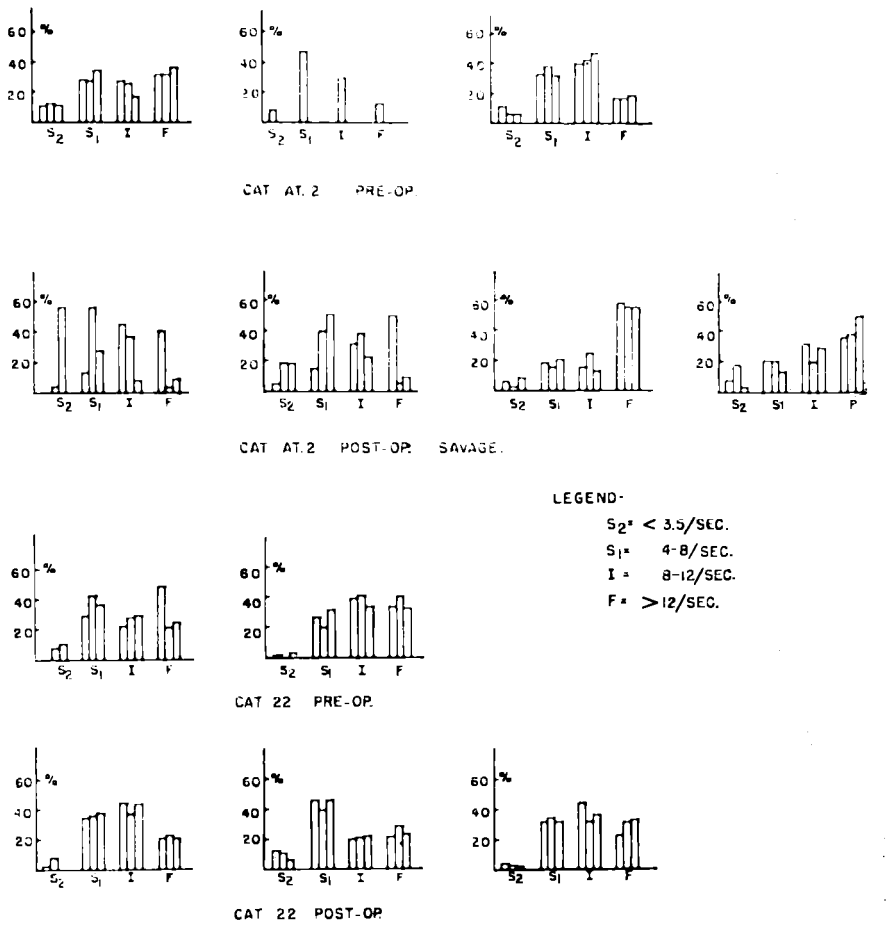

FIG. 5.

Graphic representation of frequency analyses for cat 2, savage, and cat 22, control. Each graph represents a 20-minute run. Each vertical bar indicates the percentage of a frequency band in a 30-second sample of the record.

Fig. 1 and 2, especially from the occipital and vertex leads, but less evident from the frontal monopolar lead. Sample records of normal unoperated cats are shown in Fig. 1 and 3. Fig. 2 is a record from the same cat as in Fig. 1, after the animal had become extremely savage following operation for production of hypothalamic lesions. Fig. 4 is a postoperative record from the same cat as in Fig. 3, but in this case no savageness was present.

Inspection of records from normal and savage cats and comparison of preoperative and postoperative records from savage cats disclosed no qualitative differences which were considered to be outside the normal variation. In an attempt at more precise analysis, several 30 second samples from the vertex lead of each run were measured for frequency distribution. Four frequency categories were used: (a) very slow (S_2), less than 3.5/sec.; (b) slow (S_1), 4-8/sec.; (c) intermediate (I), 8-12/sec.; (d) fast (F), more than 12/sec. Examples of the distribution patterns in 2 cats, one savage and one unoperated control are shown in Fig. 5, and a summary of the findings in normal, savage and control cats will be found in Table I. Inspection of these data leads to the conclusion that no significant difference exists. This conclusion is strengthened by the degree of variability in the same animals, pre- and postoperatively, and justifies rejection of the hypothesis that any sig-

TABLE I.
Frequency Analysis of Normal and Operated Cats.
(Lead 2-4.)

Experiments	Avg % frequency			
	S_2	S_1	I	F
29 normal (13 cats)	8.86 ± 1.98*	37.48 ± 2.36	26.18 ± 2.2	27.13 ± 2.79
14 operated controls (5 cats)	3.62 ± 0.84	34.62 ± 4.33	38.5 ± 2.76	23.34 ± 4.64
19 malevolent (5 cats)	9.43 ± 1.64	32.06 ± 2.3	29.27 ± 1.79	30.66 ± 2.74

S_2 indicates frequencies less than 3.5 per second; S_1 indicates frequencies of 4-8 per second; I indicates frequencies of 8-12 per second; F indicates frequencies greater than 12 per second.
* Standard error.

nificant trends exist after this type of hypothalamic injury.

Corticohypothalamic relationships remain physiologically and anatomically obscure. Technical factors have interfered with anatomical studies (Ingram[14]). Neuronographic studies (Ward and McCulloch[15]; Murphy and Gellhorn[16]) indicate that connections between the frontal lobe, at least, and hypothalamus are fairly extensive. The significance of these connections is not clear. According to Kennard[17] and Obrador,[18] extensive destruction of the hypothalamus suppresses cortical potentials. On the other hand, Morrison, *et al.*[19] found no relationship between hypothalamic and cortical activity, and Jasper and Droogleever-Fortuyn[20] found no generalized modification of cortical potentials upon stimulation of the hypothalamus. Laufer[21] and Lennox and Brody[22] have reported that hypothalamic lesions in human patients are associated with generalized low frequency activity. Our own results indicate that relatively small, bilateral lesions restricted to the ventromedial part of the tuber do not alter cortical activity in curarized animals, even when such lesions may be considered causally related to decided changes in behavior with outward signs of an emotional type. However, records from the cortices of such animals under more nearly "normal" circumstances, unaffected by drugs, both at rest and when displaying their characteristic rage, have not yet been secured, and such data will be an essential part of the final interpretative picture. Under aggravation, savage cats show violent autonomic discharges, which are not detectable under erythroidin. Under non-narcotized conditions Darrow's[23] theory that such discharges may be associated with changes in cortical activity may then well be put to test.

Summary. Electroencephalographic studies of cats curarized with erythroidin show no discernible differences between cortical potential patterns in normals, operated controls and cats showing "savage" behavior after production of relatively specific hypothalamic lesions.

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