period of extinction, the rats continued to run in response to the buzzer with a high degree of consistency until a point was reached where, relatively suddenly, no such responses were made. Inasmuch as the individual Vincent curves have essentially the same form found in the average curves, and inasmuch as the raw data reveal the same sudden termination of the conditioned response, the genuineness of the phenomenon seems well attested.

The extinction curves presented by Kleitman and Crisler and by Switzer are based on the magnitude of the conditioned response and not upon its frequency. Does the present conditioned locomotor response suffer a diminution in magnitude during the period of extinction so that the rat finally and gradually reaches a point where no movement is made to the buzzer? The present apparatus with its circular pathway permitted runs varying in magnitude from zero to an indefinitely large value. An examination of the records, however, shows that, although the lengths of the runs varied, there was no trend toward shorter and shorter runs. A curve relating the magnitude of the response to the amount of elapsed time would, therefore, have essentially the same form as the present curves, based on frequency of response. The suggestion is therefore apparent, from a comparison of the various curves found in the literature, that there is no one type of curve for extinction. Rather the character of the curve will depend upon many factors, including the type of response conditioned.

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DISSOCIATION OF THE PYRAMIDAL AND EXTRAPYRAMIDAL FUNCTIONS OF THE FRONTAL LOBE

SECTION of one pyramidal tract below its last large supra-segmental connection with the pons produces in cats a syndrome of deficit describable either as the specific reactions lost or impaired, or as a depression of phasic activity in general; or as a loss of excitation at the final moto-neurones in the cord. Spasticity, or other evidence of release, is absent. Stimulation of the motor cortex either immediately or months after such a section demonstrates the preservation within that cortex of inhibition, effective on tonic or clonic states present in the limbs, together with the abolition of the familiar motor function. An extrapyramidal type of motor activity is at the same time uncovered.

Repetition of this lesion in rhesus monkeys produces a similar, though graver syndrome of deficit, again without spasticity. And again, stimulation of the cortex brings inhibition to bear on activity present in the limbs after destruction of the characteristic motor responses of the precentral gyrus. In the monkey, however, this inhibition is not confined, as in the cat, to the immediate motor region but is exercised by motor, premotor and prefrontal cortex, and from the second of these, most vigorously. From this region, for instance, tonic closure of the fingers into a grasp is most easily released. Moreover, although the fine type of movement characteristic of stimulation of the precentral gyrus is totally abolished by the lesion, the so-called adversive movements survive, and can be elicited not only from the premotor region but from the precentral and postcentral gyri, as well. Furthermore, after section of one or both pyramidal tracts at the level of the trapezoid body, with or without time for degeneration, epileptiform convulsions are easily set in train by stimulation of the motor, premotor and prefrontal regions, and even of spots in the parietal lobe. These involve all four limbs and face, show typical progression, tonic and clonic phases and after-exhaustion, resembling in essentials the clinical Jacksonian seizure.

One may conclude, therefore, that pyramidal and extra-pyramidal functions of the motor cortex and adjacent regions may be dissociated by pyramid section. The rapidly executed, fairly discrete movements, long familiar from cortical stimulation, are thus demonstrated to be mediated exclusively by the cortico-spinal tract. On the other hand, the integrity of this tract is not necessary to the exhibition of the larger movements called adversive, nor to the exercise of the very important inhibitory function of the cortex. Consequently, these, together with the epileptiform convulsions elicited, represent extrapyramidal activities of the cerebral cortex.

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THE NATURE OF THE BARBITURATE–PICROTOXIN ANTAGONISM[1]

MALONEY, Fitch and Tatum[2] and Maloney and Tatum[3] have shown that picrotoxin is a very effective antidote in acute barbiturate poisoning. We have had ample occasion to confirm their data in animals poisoned with large doses of intravenously administered barbiturates. Quoting only extreme cases, the results in Table 1 were obtained:

To a number of dogs and rabbits, we administered the minimum anesthetic doses of different barbiturates, waited for the onset of anesthesia and then recorded

[1] From the Department of Pharmacology and Materia Medica, Georgetown University, School of Medicine, Washington, D. C.

[2] Maloney, Fitch and Tatum, *Jour. Pharmacol. Exper. Therap.*, 41: 465, 1931.

[3] Maloney and Tatum, *Jour. Pharmacol. Exper. Therap.*, 44: 337, 1932.