conditions than it was under spontaneous conditions (Fig. 4). A small percentage of visual cortical cells are not primarily responsive to light but either respond weakly to light or respond to other stimuli (6). Such cells would not be represented well by mean excitability under driven conditions and would better represent the mean when all cells of the area are spontaneously active. Under driven conditions, cells with primarily visual function are dominantly responsive to visual afferents not shared by such diffusely projecting or nonprimary afferent cells. In contrast, under spontaneous conditions, these two different cell populations may have more active afferents in common.

Regardless of conditions, almost all cells remained significantly correlated with the slow-wave amplitude. These experimentally produced changes are a matter of magnitude of congruence under the specific conditions.

Congruence is an estimate of how well the mean represents a randomly sampled cell, and it provides an estimate of the relation of the momentary excitability of a given cell to that of the entire population. Congruence in our view is an index of neural homogeneity, and it represents how low the population variance around the mean excitability is. When congruence is low, one may assume that the variance of the cell population is high, and that neural homogeneity of the population is low. Thus, on the reasonable assumption that the cells studied here constituted a random sample, the common excitability or neural homogeneity can be estimated from the relation of a single cell to the accompanying slow activity from the same electrode.

This measure of congruence represents a new measure of neural activity of brain which is neither slow-wave nor spike, evoked potential nor integrated activity, but which derives instead from a second-order interaction of spike and wave. Such estimates of homogeneity can be accurately made on the basis of samples taken for as little as 1 to 2 minutes, or even a few seconds, depending only upon spike rate. This advantage raises the status of the relation of spike probability to wave amplitude from a static biophysical datum computed after the fact to a dynamic functional variable which may be evaluated moment to moment.

In pharmacological, behavioral, and other site-of-action studies, if one plots momentary correlation values simultaneously for a number of sites, the changing configuration of local structural homogeneity may be observed and will provide information about the relative internal communality of excitability or consistency of local neural function (7).

STEPHEN S. FOX

ROBERT J. NORMAN

Department of Psychology University of Iowa, Iowa City

## References and Notes

- 1. S. Fox and J. O'Brien, Science 147, 888 (1965).
- 2. S. Fox, J. Liebeskind, J. O'Brien, H. Dingle, in Symposium on Limbic Structure and Func-tion, T. Tokizane and J. P. Schade, Eds. (Elsevier, Amsterdam, 1967).
- (Elsevier, Amsterdam, 1967). W. R. Adey, in Reticular Formation of the Brain, H. H. Jasper, L. D. Proctor, R. S. Knighton, W. C. Noshay, R. T. Costello, Eds. (Little, Brown, Boston, 1958); C. Li, H. Mc-Lennon, H. H. Jasper, Science 116, 656 (1952); C. Li, M. Cullen, H. H. Jasper, J. Neurophysiol. 19, 111 (1956); C. Li, J. Cell. Comp. Physiol. 61, 165 (1963); L. Widen and C. Ajmone-Marsan, Arch. Ital. Biol. 98, 248 (1960); K. Von Euler, J. Green, G. F. Ricci, Acta Physiol. Scand. 42, 87 (1958); C. Dunlon, W. R. Adey, K. F. Killam M. C. Dunlop, W. R. Adey, K. F. Killam, M.

Brazier, Fed. Proc. 18, 38 (1959); J. Buch-wald, E. Halas, S. Schramm, Electroen-cephalog. Clin. Neurophysiol. 21, 227 (1966); R. Jung, Electroencephalog. Clin. Neuro-physiol. Suppl. 4, 57 (1953); F. L. Gerstein and N. Kiang, Exp. Neurol. 10, 1 (1964); W. and N. Klang, *Exp. Neurol.* 10, 1 (1964); W.
R. Adey, in *Progress in Physiological Psychology*, E. Stellar and J. Sprague, Eds. (Academic Press, New York, 1966).
D. Purpura, *Int. Rev. Neurobiol.* 1, 47 (1967).

- 4. (1959).
- We acknowledge earlier, related formulations 5. of E. R. John [E. R. John, D. S. Ruchkin, J. Villegas, *Science* 141, 429 (1963); ——, *Ann. N.Y., Acad. Sci.* 122, 362 (1964); D. S. Ruchkin, J. Villegas, E. R. John, *Ann. N.Y. Acad. Sci.* 115, 799 (1964)], who, with co-workers, developed concepts of functional correspondence based on cross correlations of correspondence based on cross correlations of evoked-potential wave shapes. John's formu-lation was developed to relate changes between brain areas at the same bioelectric level. Our concept of congruence applies to changes within brain structures and its power from measures at different bioeleclevels. tric
- 6. P. Buser and P. Borenstein, Electroenceph-P. Buser and P. Borenstein, Electroenceph-alog. Clin. Neurophysiol. Suppl. 6, 89 (1956); P. Buser and M. Imbert, in Sensory Com-munication, W. Rosenblith, Ed. (Wiley, New York, 1961), p. 607; R. Jung, *ibid.*, p. 627; A. Fessard, *ibid.*, p. 585. Supported by PHS grant MH11834. We thank M. Dietrich, systems programmer for the laboratory
- 7. the laboratory.

**Extinction Following Intracranial Reward:** The Effect of Delay between Acquisition and Extinction

28 December 1967

Abstract. Rats trained to press a bar for intracranial reinforcement gave as many responses during extinction as did water-reinforced controls, when extinction came immediately after an acquisition session. However, the experimental animals gave fewer responses in extinction than water-reinforced animals when extinction was delayed for 1 hour after acquisition. The activity level of the experimental animals was high immediately after acquisition but declined markedly over the delay period, which suggests that resistance to extinction after intracranial reinforcement is primarily a function of activity level.

Since the demonstration that electrical stimulation of the brain can serve as a reward, a number of investigators have concluded that intracranial reinforcement (ICR) and conventional reinforcement under appetitive drives may have different effects on behavior (1). The most noteworthy of these differences is the repeated demonstration that responses maintained by ICR extinguish faster than those maintained by food or water reinforcement (2). This abrupt cessation of responding when reinforcement is withheld is generally taken as the most convincing evidence that ICR is essentially different from conventional reinforcers (3).

Recently, however, it has been suggested that differences in response topography between ICR and conventional reinforcement may account for these reported differences in resistance to extinction (4). In the typical ICR situation, reinforcement is administered immediately when the subject presses a lever, while conventional reinforcement is made available at a chute or drinking nozzle some distance from the lever. Gibson et al. (4) showed that when the form of the response made for ICR and conventional reinforcement was made similar, the usual difference in resistance to extinction was not observed. They conclude that ICR is not, therefore, essentially different from conventional reinforcers.

This study is open to criticism, however. All the subjects, including those scheduled for ICR, were deprived of food during both acquisition and extinction. Since subjects had electrode implants in the lateral hypothalamus, where electrical stimulation can elicit eating (5), and since it has been shown that hunger increases the number of responses emitted during extinction of an ICR habit (6), the generality of the results of Gibson et al. must remain in question.

Howarth and Deutsch (7) have proposed that extinction following ICR is due simply to drive decay and is, there-

Table 1. Mean number of responses in the 30-minute extinction session.

Reinforcement	Zero delay	One-hour delay
Water	90.9	95.6
ICR	92.5	30.8

fore, a function of time since reinforcement. Our aim was to compare extinction following ICR and conventional reinforcement at two times: immediately after reinforcement, when any drive from ICR would remain high, and after 1 hour, when drive decay might be assumed to have occurred. Our experiment employed four groups of ten rats each. Two groups were adapted to a 23-hour water-deprivation schedule for 10 days prior to the start of the experiment, and two groups were fitted with permanently implanted, acrylic-insulated, stainless steel, bilateral, monopolar electrodes with the 0.5-mm stimulating tip aimed at the medial forebrain bundle (8), an indifferent electrode being provided by a wire loop placed over the skull. A source of 50-cycle a-c of 50 to 70  $\mu$ a, individually adjusted for each rat, provided the ICR which was delivered through one electrode and the indifferent electrode.

The experimental chamber was a Plexiglas box which was 23 cm high and had floor dimensions of 40 by 25 cm. The floor incorporated a stabilimeter with a pendulum transducer (9) from which contacts, via a sensitometer circuit, could be recorded on a counter and cumulative recorder. To make response topography between ICR and water groups more similar, the stainless steel lever (2.5 by 4.5 by 1.5 cm) incorporated a drinking cup (1 cm in diameter by 0.5 cm deep) on its upper surface. Water was supplied by a pump (controlled by Grason-Stadler programing equipment) which delivered 0.25 ml in 0.5 second after operation of the lever. Operation of the same lever could alternatively deliver brain stimulation for 0.5 second; but, to make the situations even more closely comparable, a timer was included which imposed a 0.5-second delay between closing the lever microswitch and the commencement of the 0.5-second period of brain stimulation.

Each animal was given 10 days of acquisition training in which it made a total of 1000 continuously reinforced responses. For the first 8 days, sessions were of 100 responses; on day 9, of 150 responses; and on day 10, of 50 responses. Immediately after the acquisition trials on the tenth day, two groups, one given ICR and the other waterreinforced, were given a 30-minute extinction session during which reinforcement was withheld. Each animal in the remaining two groups, after acquisition trials on the tenth day, was returned to its individual home cage, which was then placed on the stabilimeter platform for a period of 1 hour. During this delay period, the animal's activity was recorded. The animal was then placed back in the test chamber for a 30minute extinction period.

Response rates during acquisition did not differ significantly among the four groups. Table 1 shows the mean number of responses made during extinction. It is clear that animals in the ICR group in which extinction was delayed gave fewer responses in extinction than those in the other three groups. Analysis of variance indicated that the interaction between types of reinforcement (ICR versus water) and delay (zero delay versus 1-hour delay) was significant (P < .001); tests of simple effects (10) showed further that ICR and waterreinforced groups differed significantly (P < .001) for the 1-hour delay but not when there was no delay, and conversely that the effect of delay was significant (P < .001) for the ICR groups but not for the water-reinforced groups.

Of the two delayed-extinction groups, the ICR animals were markedly the more active. Figure 1 compares the mean number of stabilimeter transducer contacts (in 5-minute blocks) for the ten ICR subjects and the ten water-reinforced subjects during the 1-hour delay period. The decline in the activity of the ICR subjects and their significantly faster extinction after a rest period contrast strongly with the lower stable rate of activity and slower rate of extinction of the water-reinforced animals.

Considered in conjunction, these data suggest that the level of activity of animals reinforced by ICR is an important determinant of resistance to extinction and a significant factor to consider in interpreting differences between behavior maintained by ICR and by conventional reinforcement. Real qualitative differences between the two reinforcement conditions only become apparent when the difference in activity levels is controlled. These data also suggest that the apparent similarity between extinction functions for ICR and sugar water reinforcement found by Gibson et al. may have resulted from heightened activity of ICR subjects immedi-



Fig. 1. Activity scores: mean number of stabilimeter transducer contacts in 5minute blocks during the delay period.

ately after acquisition. Some support for the generality of the results of our study comes from a recent report by Gallistel (11), who showed that rats that receive ICR run faster in a runway after a decrease and slower after an increase in intertrial interval. This was not found in rats rewarded with water.

Our results are thus consistent with a theory such as that proposed by Deutsch (3), which assumes a drive-decay concept to account for the decline in response rate following nonreinforcement of behavior previously maintained by ICR.

## DAVID QUARTERMAIN

Rockefeller University, New York DONALD WEBSTER

University of Auckland, Auckland, New Zealand

## References and Notes

- 1. G. A. Kimble, Hilgard and Marquis' Condi-tioning and Learning (Appleton-Century-Crofts, New York, 1961), p. 263; C. T. Mor-gan, Physiological Psychology (McGraw-Hill, J. Olds, in Nebraska Symposium on Motiva-
- Jones, Ed. (Univ. of Nebraska Press, Lincoln, 1955), vol. 3, p. 73; J. P. Seward, A. Uyeda, J. Olds, J. Comp. Physiol. Psychol. 52, 294 (1959).
- 52, 294 (1959). J. A. Deutsch, The Structural Basis of Be-havior (Univ. of Chicago Press, Chicago, 1960); —— and D. Deutsch, Physiological Psychology (Dorsey, Homewood, Ill., 1966). W. E. Gibson, L. D. Reid, M. Sakai, P. B. Porter, Science 148, 1357 (1965); S. E. Glick-man and B. B. Schiff, Psychol. Rev. 74, 81 (1967)
- E, E. Coons, M. Levak, N. E. Miller, Science 5. **150**, 1320 (1965). J. A. Deutsch a
- J. A. Deutsch and L. DiCara, J. Comp. Physiol. Psychol. 63, 344 (1967).
  C. I. Howarth and J. A. Deutsch, Science 137, 35 (1962) 6. 7.
- 137, 35 (1962). 8.
- Expressed in terms of Krieg stereotaxic co-ordinates 0.8 mm posterior to bregma, and 1.7 mm lateral and 8.2 mm ventral to top of skull. Subsequent histological examination re-vealed that the electrode tips were located in the medial forebrain bundle. J. D. Davis and G. D. Ellison, J. Exp. Anal.
- Behav. 7, 117 (1964)
- 10. B. J. Winer, Statistical Principles in Experimental Design (McGraw-Hill, New York, 1962), p. 232. C. R. Gallistel, *Psychonom. Sci.* 9, 167 11.
- (1967) 12.
- Supported by F.F.R.P. grant 65.317 to D.Q. We thank Neal Miller and M. C. Corballis for critically reading the manuscript.

29 January 1968