Duration variability of the intracranially reinforced barpressing response in the rat*

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The study was designed to examine the intracranially reinforced (ICR) response duration in the rat. Twenty-two animals were implanted with hypothalamic stimulating electrodes. In addition to these, four animals had electrodes in the septal rewarding area, and six in the cortex. Animals were divided into six groups. Each group was trained with a different reinforcement schedule. Barpressing response for the ICR was recorded on paper and magnetic tape. Analysis of data was performed by a Linc 8 computer. Results indicated the response to be a highly sensitive parameter to various stimulus conditions. The scope of response duration observed and its variability suggested a role of centrally generated feedback in the emission of the barpress. An attempt was made to interpret the data along a model of information processing, without reference to so-called motivational and reinforcing properties of the ICR stimulus.

Several reasons led us to believe that measuring the intracranially reinforced (ICR) response duration would provide pertinent indices for a better understanding of the nature of self-stimulating behavior. It is a well-accepted principle that the smaller the variability of the dependent variable measured, the more likely it is that the given behavior is regulated by a simpler mechanism. On this basis, for instance, it was concluded that the licking response of a drinking rat is under on-off control, where various dipsogenic stimuli did not affect the uniformity of the interresponse time, but only the duration of the behavioral sequence (Corbit & Luschei, 1969). Accordingly, the ICR response duration variability would provide some indication to what extent the response could be considered a triggered (i.e., all-or-nothing) event, and to what extent it is affected by assumed feedback information. Obviously, the question arises as to why the response duration would be a more elucidating dependent variable in exploring this particular problem than the response rate, the most frequently used measure of the self-stimulating behavior. A part of the answer is in the operant theory itself. In giving the reasons why the response rate should be the only datum "relevant to the 'learning process,'" and presumably behavior, Skinner (1950) described operant behavior as "essentially an emissive phenomenon," where the only reference for any given response is the time elapsed since the preceding response. For that reason, the response is regarded as a "point in time" occurrence which "has no duration." The time needed to execute a response is, of course, recognized as present

in the behavioral sequence, but it is deemed irrelevant for understanding the behavior. Extensive literature in the area of operant behavior confirms the potential value of such an approach.

The argument we wish to offer here is that the physiological psychologist, accepting the above operant principle, imposes upon himself limitations that are not warranted by the nature of the phenomenon being investigated. For the purpose of illustration, assume that two different schedules of reinforcement generate responses of two different durations. An operant interpretation most likely would propose that the given schedules differentially reinforce two durations. It is our belief that the physiological psychologist cannot be satisfied with such an explanation. We know the emission of a barpressing response to be a highly complex chain of events, with measurable correlates on cellular (Evarts, 1966, 1968), electroencephalographic (Bates, 1951; Gilden, Vaughan, & Costa, 1966), myographic (Evarts, 1967), and behavioral (Notterman & Mintz, 1965) levels. In the case of the ICR response, where the stimulus parameters and the locus of stimulation are better defined than in most other behaviors, treatment of the response as a process instead of as an event seems to be even more justified. One measure of a response as a process approach, therefore, would be its duration.

In order to establish the mode of ICR response variability, we used stimulus conditions derived not only from conventional schedules of operant behavior research, but also from self-stimulation studies which compared hypothalamic and septal brain reinforcing areas (Hodos & Valenstein, 1962; Olds, 1962), as well as those which explored interaction between reinforcing and nonreinforcing brain areas (Olds & Olds, 1962; Routtenberg & Olds, 1966).

METHOD

Subjects

Twenty-two male Holtzman rats, 100-110 days old at the time

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Fig. 1. A sample of response pattern in the animal trained with FI 240-msec reinforcement schedule. The upper trace is an analog recording of transducer output, where each depression of the bar is represented by an upward movement of the pen. The response marker for the same response sequence is shown on the lower trace, where high-amplitude spikes indicate reinforced responses, and low-amplitude spikes, the nonreinforced ones. Calibration: 27 g and 1 sec.

of surgery, were used. All animals were housed in individual cages with free access to food and water throughout the experiment.

Electrodes and Electrical Stimulus

Bipolar electrodes were made of 0.2-mm stainless steel wire, and coated with epoxylite. Electrical stimuli were generated by Tektronix Type 160 units and consisted of biphasic square pulses, 2 msec per pulse pair at 100 Hz. Stimulus trains of such pulses were 100 msec long. Timing and control of stimulus trains were performed by digital circuits. Current intensity of the stimulus train was continuously monitored with an oscilloscope.

Procedure

Brain stimulating electrodes were implanted under sodium pentobarbital anesthesia (44 mg/kg) according to the following schedule: (a) Twelve animals were implanted with a single electrode aimed at the posterolateral hypothalamus. Stereotaxic coordinates used were 5 mm posterior to bregma (A-P), 1.7 mm lateral from midline (L), and 7.6 mm vertical from the surface of cortex (D). (b) In addition to a hypothalamic electrode as above, four animals were implanted with a second contralaterally placed electrode aimed at the septal area (A-P = 0 mm, L = 1 mm, D = 4.2 mm). (c) In addition to a hypothalamic electrode as in previous groups, six animals were implanted with a second electrode aimed at the cortex of the opposite hemisphere (A-P = -5 mm, L = 2 mm, D = 1 mm).

Animals implanted with the hypothalamic electrode alone and those with cortical electrodes as well were divided into three (N = 4 each) and two (N = 3) groups, respectively. Assignment of animals to groups with different training reinforcement schedules was done on the basis of the response rate during the first training session with hypothalamic reinforcement. Though the rate at that time is lower than the terminal level, it may serve as an index for comparison of Ss.

There were six groups of animals. The first three groups were control for the second three groups, where septal and cortical stimuli were used. The six reinforcement schedules used in the training of each respective group were as follows: (I) Continuous reinforcement schedule, CRF, where every response was followed by a 100-msec reinforcing stimulus in the lateral hypothalamus. This schedule represented a principal control both for the remaining two control groups and the three experimental groups. (II) In a FI 240-msec schedule, delivery of the reinforcement was followed by a 140-msec interval during which emitted responses were not reinforced. This schedule was used primarily as a control for Group V, where the hypothalamic stimulus was followed by a cortical one. Also, it generates a large

number of nonreinforced responses and thus provides indices for the effect of the electrical stimulus upon the emitted response. (III) Delayed reinforcement schedule consisting of a 140-msec pause and a 100-msec train which followed onset of each response; it served as a control for Group VI. (IV) A continuous reinforcement schedule with concurrent septal training where animals were trained for 10 min with hypothalamic, and 10 min with septal, reinforcement. From session to session, the order of placements varied randomly. (V) Double-stimulus schedule, where the reinforcing hypothalamic stimulus was followed by a 100-msec-long stimulus delivered in the cortex. The intertime between stimulus trains was 40 msec. (VI) Reverse double-stimulus schedule, which was similar to the previous one, except that the order of stimuli was reversed; the first stimulus train was delivered to the cortical, and the second to the hypothalamic, placement.

During the first training session with hypothalamic reinforcement, a higher current intensity was used in order to initiate the barpressing response. The current intensity was lowered 20-30 microA during every succeeding session as long as the response rate did not decline from the rate in the preceding training session. In this manner, the terminal level of stimulus intensity for all hypothalamic loci reached between the 7th and 10th training session ranged from 75 to 200 microA peak-to-peak, and for septal loci, from 160 to 220 microA peak-to-peak. Intensity criterion for the cortical stimulus was that it did not maintain self-stimulating behavior and did not produce any visible aversive effects when the stimulus was at the same current level as the reinforcement. Although an attempt was made to use these current intensities, in the course of training with cortical stimuli seizures were observed. Reduction of current intensity eliminated their recurrence. Terminal level of intensities used for cortical stimulation ranged from 40 to 100 microA peak-to-peak.

Each group of animals was trained for 12 sessions 20 min long, with one reinforcement schedule (except animals with septal electrodes). After training was completed, tests with several nontraining schedules were performed.

The animals' response was the barpress. A bar, 4 x 5 cm in size, protruding into the Skinner box, was attached to the Statham (Model P23AC) pressure transducer for recording of response emission and to a standard microswitch for triggering the stimulus. The transducer output was fed into a Grass 5D polygraph, and responses were recorded both on paper (Fig. 1) and magnetic tape (Precision Instruments recorder). The response was defined as any barpress exceeding the criterion. The criterion was arbitrarily set at 27 g of static equivalent pressure. At this criterion pressure, the switch was tripped and the electrical stimulus was delivered irrespective of the length of the response. Response duration was measured from the point where the criterion pressure was reached, until it fell below this level, and it was expressed in milliseconds. Data were processed by a Linc 8 computer (Digital Equipment Corporation). The transducer output was sampled at 500 Hz. Responses were classified according to their duration, and distributions were plotted by a Calcomp incremental plotter.

At the end of the experiment, all animals were sacrificed, perfused, and brains sectioned by a frozen technique. Staining was performed with Perl stain for iron deposits and Phloxin fast green (gallocyanine) for fibers and nuclear groups.

RESULTS

Of the 22 animals, 2 did not complete training procedures (1 each from Groups IV and VI). Findings on loci of electrical stimulation are summarized in Fig. 2. Since the number of animals in each group was relatively small, no attempt was made to correlate the electrode

Fig. 2. Coronal sections of the rat brain from König and Klippel's (1963) atlas. Electrode placements are indicated with the black dot. Numerals refer to S number.

placement with training condition and response duration recorded.

In general, results of the ICR response duration measurements clearly indicated that the shortest responses were emitted by animals trained on a continuous reinforcement schedule (Group I). Any interference with the continuous ICR contingency caused an increase in response duration (Fig. 3, Fig. 4, Table 1).

Considering the first three schedules of reinforcement separately, since they did not involve stimulation of other brain sites, results could be summarized as follows. Animals trained with the CRF schedule established their response distribution means between 120 and 180 msec, and those trained with FI had their means at durations from 140 to 200 msec. Animals trained with the delayed reinforcement schedule had their response duration means at 260 and 300 msec. The overall difference in reinforced response duration between these three groups is found to be significant: Kruskal-Wallis test, $H = 6.14$, $p < .049$ (all statistical tests are from Bradley, 1968).

It was observed that with animals trained with the FI schedule and the delayed reinforcement schedule, response duration decreased when the animals were tested with the continuous reinforcement schedule. The decrease in response duration was on the order of 10 to 30 msec and 20 to 80 msec, respectively (Wilcoxon signed-rank test, $W+ = 0$, $p < .0078$).

In most animals, irrespective of the reinforcement schedule with which they were trained, the FI 240-msec schedule generated a number of nonreinforced responses. In some cases, this number would reach 35% of all emitted responses. In eight animals, it was found that nonreinforced responses were shorter than reinforced responses. The differences ranged from 10 to 60 msec (Wilcoxon signed-rank test, $W+ = 0$, $p < .0039$). Exceptions to this finding were two animals which did not emit nonreinforced responses, and a third one

Fig. 3. Mean response duration for all six groups of animals. Stippled columns refer to training reinforcement schedule for each group. Nonstippled columns refer to duration of nonreinforced responses (Groups II and III), duration of septally reinforced responses (Group IV), and duration of responses with hypothalamic reinforcement following removal of cortical stimulus (Groups V and VI).

Fig. 4. Illustration of response duration distributions with six schedules of hypothalamic reinforcement. In Groups II and III, reinforced responses are presented with a heavy line. The number of responses and current intensity levels for each distribution are as follows: I = 457 reinforced responses (R+), 100 microA; II = 496 R+ and 135 nonreinforced responses (R-), 100 microA; III = 528 R+ and 82 R-, 150 microA; IV = 342 R+, 100 microA; V = 582 R+, 120 microA hypothalamic and 60 microA cortical stimulus; VI = 412 R+, 140 microA hypothalamic and 70 microA cortical stimulus.

(No. 9, Group I) which displayed an aversive retraction from the bar following each reinforcement (nonreinforced responses were longer).

Out of four animals in Group IV, the ones trained both with hypothalamic and septal rewarding electrodes, one animal did not self-stimulate for hypothalamic reinforcement. The three animals which did reached maximal response rate with the hypothalamic stimulus during the fifth and sixth training sessions. In the following sessions, the rate gradually decreased, and at the end of the experiment, the response rate was approximately 30% lower than the maximal rate observed. Also, hypothalamically reinforced responses were considerably longer than responses in animals trained with the hypothalamic stimulus alone (Group I). Both of these observations suggest possible influence of the parallel training with the septal reinforcement.

Although this study deals primarily with the hypothalamic reinforcement, a difference between the hypothalamic and septal reinforcement should be mentioned. Namely, when the animals from Groups I, II,

and IV were tested with the delayed reinforcement schedule, where delay was 140 msec long, increase in response duration ranged from 5 to 39 msec. In the case of the septally reinforced response, tests with the delayed reinforcement schedule generated responses 123 msec longer than those observed with the continuous reinforcement schedule. Thus, with septal loci, increases in duration of response almost completely account for the delay of the reinforcing stimulus, suggesting a higher degree of interdependence between the stimulus and the barpressing response.

Groups V and VI were characterized by the longest responses, mean durations being 267 and 343 msec, respectively. When compared with their respective controls, i.e., Groups II and III, the effect of cortical stimulation is clearly distinguishable. Thus, animals from Group V emitted responses approximately 75 msec longer than those of animals from Group II (Wilcoxon rank-sum test, $W_n = 6$, $p < .05$). In the case of Groups III and VI, the difference was around 60 msec (because Group VI had only two animals, statistical test could not

Table 1
Response Duration Means (Milliseconds) for Animals from All Experimental Groups*

Group I		Group II		Group III		Group IV**		Group V		Group VI†	
S	Mean	S	Mean	S	Mean	S	Mean	S	Mean	S	Mean
9	125.64	2	186.35	11	292.14	4	344.38	7	302.09	6	332.16
17	163.70	14	203.57	15	260.37	20	286.43	8	247.17	10	355.31
23	179.99	21	144.88	16	296.94	27	195.43	31	253.29		
28	146.18	29	189.46								

*S denotes subject number. Duration mean refers to reinforced responses only.

**Response duration mean was calculated from duration classes of 40 through 600 msec. Responses longer than 600 msec could not be taken into account. Percentage of unaccounted responses for Ss 4, 20, and 27 are 10.62, 12.78, and 8.62, respectively.

†Percentage of unaccounted responses for Ss 6 and 10 are 36.12 and 40.70, respectively.

yield but $p < .1$). Also, in the tests where the cortical stimulus was omitted, i.e., Group V was tested with FI 240 msec and Group VI with the delayed reinforcement schedule (Fig. 3), response duration distributions indicated significant shortening of the barpressing response (Wilcoxon signed-rank test, $W^+ = 0$, $p < .0313$).

DISCUSSION

This study was undertaken in order to assess whether ICR response duration could be experimentally modified, and, if so, whether the observed modification of the motor output might provide information about self-stimulating behavior. The temporal aspects of motor output were measured, not by techniques of response rate or interresponse time, but by measurement of the response duration itself. It was done under various stimulus conditions, including delay of reinforcement, concurrent training with another brain reinforcing locus, and a response-contingent nonreinforcing cortical stimulus.

In order to interpret the observed changes in the ICR response duration, it is important to visualize the sequence of events in the course of the response emission. From the resting position, the bar had to be depressed with a weight equivalent to 27 g of static pressure. In this instance, the microswitch attached to the bar is triggered and, in all but Group III (delayed reinforcement), onset of electric stimulus occurs. In most cases, the animal's pressure upon the bar continues to rise for another 20 to 30 g (see Fig. 1). At a certain point, maximal pressure is reached, and reversal of paw action takes place, resulting in release of the bar.

The most important observation is the difference in response durations between FI 240 msec and delayed reinforcement schedules (Groups II and III). Both stimulus conditions provide an equal probability of reinforcement. The longer response duration recorded with the delayed reinforcement indicates that the animal is using the information provided by the stimulus, i.e., central feedback, in determining how long the bar is going to be "held" depressed and when it is going to be released. In order to accomplish this, we are fully justified in assuming the animals' utilization of specifiable neuronal and muscular entities, and that changes in duration of the response execution reflect on the duration of these neuromuscular processes. Thus, in the case of delayed reinforcement, it would be insufficient to interpret the longer response as a result of a simple 140-msec reinforcement delay, which if subtracted from the response duration leaves no basis for differentiation of the FI and delay schedules. Rather, the longer response with the latter schedule indicates that the animal has means for detecting the reinforcing stimulus; it can prolong the activation of paw extensor muscles and, once the reinforcing stimulus is delivered, release the bar (i.e., activate flexor muscles).

A similar, although more complex, chain of events may account for the difference in response duration in animals trained with the continuous reinforcement schedule, without and with concurrent septal stimulation (Groups I and IV). In this case (Group IV), septal training increased duration of the hypothalamically reinforced response. While there remain a number of problems yet to be resolved, observation clearly suggests the hypothalamic stimulus processing to be subject to some form of change due to training with another reinforcing stimulus. On the basis of response rate, we may surmise the effectiveness of the hypothalamic stimulus to have been reduced by septal training.

In the case of training with the cortical stimulus (Groups V and VI), there is strong evidence that cortical stimulation, although behaviorally "neutral," significantly increased the response duration. More than in previous findings, the effect of cortical stimulation could be interpreted along the test-operate-test-exit (TOTE) sequence proposed by Miller, Galanter, and Pribram (1960) and Pribram (1967). The model postulates a time delay in processing afferent input, during which its qualities are being tested against memory-stored (i.e., "expected") values, and its relevance to the organism is being appraised. It is implied that the time delay between input and output is proportional to the incongruity between input and "expected" values. Thus, an interpretation could be suggested that the ICR response duration increase due to cortical stimulation reflects an increased incongruity of the stimulus in comparison with the stimulus condition where the hypothalamic reinforcement is given alone as in the CRF schedule (Group I).

The different durations of reinforced and nonreinforced responses with the FI schedule may suggest the minimum time necessary for the stimulus information processing to be in the neighborhood of 50 msec. Although we may assume the presence of several confounding variables which preclude accurate extraction of this datum, to account for the time difference, i.e., a 50-msec longer "hold" on the bar concurrent with stimulus delivery, a process has to be postulated.

In conclusion, positive findings of this study are that the motor output in the ICR response sequence can be differentially modified by various stimulus conditions. The output modifiability was interpreted as a result of centrally generated feedback information, participating in mediation of the electrical stimulus delivery and motor response execution. Here, two questions remain to be answered. First, granted the feedback hypothesis is acceptable, where do we proceed next? Second, what is the relationship between the feedback hypothesis and the frequently referred-to concepts of reinforcing and motivational excitations in the ICR situation?

Concerning the first question, the very possibility of measuring the processes involved in response generation

and execution directly represents a large area to be explored. To begin with, the feedback processes could be better estimated if some physical constraints upon behavior were used, thus limiting the number of "whole animal" free movements involved in the response emission. Also, response duration is but one of the possible parameters of the barpressing sequence. Interresponse time and force could be additional attributes of the processes involved. Correlation between these various measures may well indicate the degree to which the response per se is a unitary event, i.e., whether changes in one parameter are accompanied by changes in other parameters as well. Establishment of these technical and parametric questions would enable the researcher to explore such independent variables as anatomical distribution of given feedback dimensions, effect of various pharmacological and physiological conditions on behavior of the feedback, and so on.

In response to the second major question, on relationship between the proposed central feedback, on the one hand, and reinforcing and motivational mechanisms, on the other, there are very few points in common that could be found at the present time. The principal reason for this is that most models and interpretations of the intracranial reinforcement rely heavily on concepts of drive and reward as derived from behavioral experiments, and great efforts were made to identify drive and reward mediating fibers (Olds, Allan, & Briese, 1971; Deutsch, 1964; Deutsch & Howarth, 1963; Gallistel, 1973). Aside from the fact that even at the behavioral level these concepts cannot be considered transsituational (Premack, 1965, 1971), a substantial body of data on so-called stimulus-bound behaviors contradicts a direct relationship between electrical stimulation and the observed effects (Valenstein, Cox, & Kakolewski, 1969), and efforts to replicate behavioral measuring of refractory periods of various fiber systems was not successful (Milner, 1970, p. 398).

In short, we cannot say why the animal self-stimulates, and the motor output analysis is of little help in answering this question at the present. It is our belief, however, that we can postulate that every behaviorally definable response is preceded by a finite number of neuromuscular processes. These processes are reflected in the motor output containing the given response, but not necessarily on its rate of emission. Fine-grain analysis of the motor output, therefore, should give us better understanding of these processes. In this context, the intracranial reinforcement should be treated as sensory information, without reference to the motivational and reinforcing properties of structures stimulated.

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