from extinction during the transition from CRF to the schedule (*12*).

Accordingly, it is not BSR per se that makes schedule control difficult, but its property of being applied in the last link of a one-link chain. Other applied reinforcers pose similar problems in rats and ducklings (*13*) as well as humans (*14*). Carlisle (*15*) found that rats pressing to turn on a heat lamp reinforcer in a cold chamber performed well on a CRF schedule, but poorly on an intermittent schedule unless a chaining procedure was used.

Whereas most of the experiments since 1958 in which the older electrode and plug assembly was used remain valid, conclusions from those in which information about BSR onset was varied should be questioned, because the information would act as an S^D for postural adjustment leading to increased reinforcement. Some examples of those experiments follow: Stein's (*16*) conclusion that stimulus need not be an S^D for it to be a conditioned reinforcer. The finding by Steiner *et al.* (*17*) that a pattern of BSR which rats produce in a self-stimulation test is aversive when "played back" independent of behavior; Faircloth's (*18*) conclusion that BSR is more reinforcing when it is self-initiated; Bollinger and Gerall's (*19*) finding of a decrease in brain-electrode impedance associated with the acquisition of self-stimulation; and my conclusion with LoLordo (*20*) that rats prefer signaled to unsignaled BSR.

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2. The "anomalous" nature of behavior reinforced by BSR has been reviewed by G. Mogenson and J. Cioe, in *Handbook of Operant Behavior*, W. K. Honig and J. E. R. Staddon, Eds. (Prentice-Hall, Englewood Cliffs, N.J., 1977), pp. 570–595; see also (*3*, *4*).
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6. The artifact would also be manifest with a constant-current stimulator since the artifact entails electrical discontinuity.
7. M. Sidman, J. V. Brady, J. J. Boren, D. G. Conrad, A. Schulman, *Science* **122**, 830 (1955).
8. S. S. Pliskoff, J. E. Wright, T. D. Hawkins, *J. Exp. Anal. Behav.* **8**, 75 (1965).
9. Beninger *et al.* (*1*) assert that the REE does not occur, but they used RI 45-second as the maintenance schedule rather than the CRF with which the "anomaly" was first described [J. Olds and P. Milner, *J. Comp. Physiol. Psychol.* **47**, 419 (1954)]. Such intermittency would produce substantial resistance to extinction, especially in comparison to their control animals which were never trained to press the lever.
10. B. F. Skinner, *Proc. Natl. Acad. Sci., U.S.A.* **20**, 234 (1934).
11. W. E. Gibson, L. D. Reid, M. Sakai, P. B. Porter, *Science* **148**, 1357 (1965).
12. Instead of the two-lever chain (*8*), A. Brown and J. A. Trowill [*Psychol. Rep.* **26**, 699 (1970)] used a single lever and gave five response-contingent BSR's when reinforcement was due; the first BSR was an S^D in a chain to press for those that follow. An alternative to the chaining method is J. P. Huston's reinforcement reduction method for FR [*Science* **159**, 444 (1968)] or the practice of making the transition from CRF to schedule in very small steps (*4*); both protect the operant from extinction during transition.
13. P. De Paulo, thesis, Bryn Mawr College (1977). Ducklings pecked a key to present a visual imprinted stimulus, but behavior was erratic when an intermittent schedule was imposed.
14. B. A. Mathews, E. Shimoff, A. C. Catania, T. Sagvolden, *J. Exp. Anal. Behav.* **27**, 453 (1977); K. W. Spence, *Science* **140**, 1224 (1963).
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Cantor's comment disposes of one puzzling discrepancy between his data and ours (*1*). Apparently when he eliminated an electrical artifact he too found that signaled brain stimulation reinforcement (BSR) was no better than unsignaled BSR in maintaining lean interval schedules of reinforcement. Signaling BSR does, nevertheless, appear to overcome the overnight decrement to some extent (*2*).

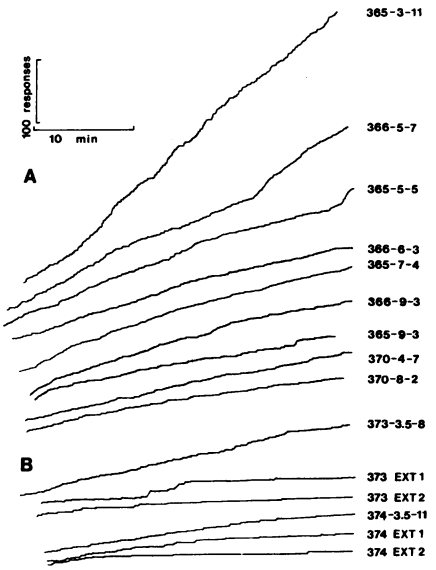

Fig. 1. (A) Representative cumulative records for three rats responding at various RI schedule values; (B) the records from one RI session and two extinction sessions (*EXT 1* and *EXT 2*) for two rats. The three numbers after each record indicate the rat, the RI value, and the number of BSR's that were delivered during that session. [Reprinted with permission from Beninger *et al.* (*3*)].

We take issue with Cantor's assertion (his reference 7) that the use of a constant-current stimulator is no protection against the artifact that invalidated his results. His figure 1 shows the contact resistance of his plug rising to a peak value of about 100 kilohms. Any constant-current stimulator able to deliver up to 40 Volts would be able to overcome that resistance. Furthermore, the oxide or dirt film that is usually responsible for poor contact in these plugs can withstand only a few volts, so that as the stimulator voltage rises to overcome the higher resistance it breaks down the film and restores good contact. Our experience in monitoring current from a constant-current source is that unless the contact is completely broken, in which case the animal gets no stimulation under any circumstances, the current is held constant, with variation of only a few percent.

Cantor's main criticism of our experiment is that because its results are out of line with those of several previous experiments, they must therefore be wrong. He notes that the highest rates we reported at a random interval (RI) 45-second schedule are much lower than the rate he obtained at a variable interval (VI) 2-minute schedule with the rat in his experiment, and speculates that we were thereby discouraged from testing leaner schedules. We have published data from just such a test (*3*). Eight rats were trained on progressively leaner RI schedules for response-contiguous BSR. All rats continued to respond up to or beyond RI 3-minute schedules, with one rat performing on a schedule as lean as RI 10 minutes. Representative cumulative response curves both during RI and extinction sessions are shown in Fig. 1. Absolute rates depend on individual characteristics of subjects and apparatus and cannot be compared from one experiment to another. However, the response rates are precisely determined by the reinforcement schedule, as is clearly the case in this experiment. Spontaneous recovery from extinction is shown from the first to the second session. These results confirm and extend those presented earlier (*1*).

We were surprised at the difference between our results and those of others (*4*). The difference does not depend on a gradual transition from high to low rates of reinforcement; we obtained the same results when naïve rats were trained for several days on continuous reinforcement and switched immediately to an RI 45-second schedule.

Environmental control was tighter in our experiment than in most earlier stud-

ies. Rats were tested always at the same time each day, in closed, sound-insulated chambers, with continuous white masking noise. There were thus no distractions to interfere with the performance of the task. Another feature that may have influenced the result is that the rat's first response in the session was usually reinforced.

Contrast effect may have contributed to the widespread impression that BSR results in rapid extinction. After watching a rat respond at 100 per minute an experimenter may regard a rate of 5 per minute as extinction, which it clearly is not. Suboptimal electrode placement or parameters of stimulation may account for other failures to achieve good BSR with lean schedules. Sidman *et al.* (*4*), for example, used septal and caudate electrodes.

Our results do not necessarily imply that chaining of responses would not produce further improvement. As pointed out by Cantor, however, most of the reported chaining experiments are confounded by presentation of multiple BSR's at the end of each chain. Cantor's own experiment, which he now claims to be an example of chaining, uses only single BSR's, but, in common with others he has presented no data from unchained control experiments. It may be that such a control is impossible. On any intermittent schedule of reinforcement the unreinforced responses are links in a chain leading to the reinforced response. This applies even to the data of Sidman *et al.* (*4*), the primary source of the belief that performance on lean schedules is impossible with BSR.

The most obvious difference between food reinforcement and BSR is not in degree of chaining but in the fact that food-deprived animals have a mechanism for motivating food-getting behavior while there is no such clearly recognizable motivation for BSR. In a deprived animal expectation of food is readily aroused because it is already being facilitated by hunger. At the beginning of a session, expectation of BSR should be more comparable to expectation of food in a satiated animal. Satiated animals will initiate responding for food (*4*) but quickly discover that food is no longer reinforcing and stop responding. An animal initiating responses for BSR, on the other hand, will have its expectation of reward confirmed and will continue to respond. In fact, it will probably do so with increased vigor because the association of BSR with the situational cues benefits from a recency effect.

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Computed Tomography Scans of Alcoholics: Cerebral Atrophy?

Carlen *et al.* (*1*) have provided us with a stimulating bit of evidence for reversible cerebral atrophy among four of eight alcoholic patients. However, many questions can be raised about their data whose answers would provide an altogether different conclusion.

Reversible atrophy was reported to have occurred only in those patients who remained abstinent from the time the first scan was obtained until the second scan was completed. However, we performed statistical analyses (Student's *t*-test, paired comparisons) of their data and found that the size of the ventricles was not significantly different when measured at an average of about 1 month after the last drink (scan 1) than at an average of about 1 year's abstinence (scan 2). Second, since the authors did not present data for a nonalcoholic control group it is impossible to determine if the ventricular size noted was abnormal even on the first scan. Also, a nonalcoholic control group was not measured twice so we do not know if their computed tomography (CT) method was reliable.

We recently completed a study of 15 alcoholics using CT scans and psychological test performance as indicators of brain pathology (*2*). We found only one case in 15 that was clearly abnormal, even though our subjects had been heavy consumers of alcohol for an average of 15 years. Before it may be concluded that brain damage is reversible upon repeated scanning, as Carlen *et al.* did, it must first be demonstrated that the scans are abnormal initially. From our own findings, we would expect at most only one of these alcoholics to have had abnormal scans upon initial scanning, if they were randomly selected. Other factors such as liver pathology (*3*) and selection of patients because of persistent neurological deficits warranting CT scans on clinical grounds (*4*) greatly increase the number of alcoholics having abnormal scans.

Morphological changes may not correlate with functional changes. For instance, we found many deficits in neuropsychological functioning among our alcoholics who had been abstinent for an average of 1 year, even when the CT scan was normal. Extensive neuronal loss within a circumscribed area must be sustained by the brain in order to detect structural changes by CT scans. Extensive neurophysiological and biochemical alterations can occur among individuals with scans that appear normal, while abnormal scans occasionally are seen among asymptomatic individuals. The combined use of both neuropsychological assessments and CT scans would appear to provide the best estimate of brain pathology.

While Carlen *et al.* noted smaller sulci in repeated scans in some of their abstinent alcoholics, we believe the most likely explanation is measurement error. The authors have stated that the average sulcus measured 1 mm with a measurement error of ± 0.25 mm. We have noted, however, that measurement from the Polaroid print, as done by Carlen and colleagues, introduces around a 3.6-fold increase in the error of measurement because of minification of the print (*5*). This means the average error would be 1 mm, or as large as the average sulcus.

We (*2*, *5*) have used the computer printout from the scanner for each slice to determine the total area of the ventricles, sulci, and inner table of the brain. The perimeters can be traced with a transparency and accurately measured with a hand planimeter. From these measurements the ventricle/brain index can be calculated. The advantage of this method is that a volumetric assessment of the ventricles can be made that is relatively independent of the particular cut taken, and the varying size of the brain table can be taken into account. Also, measurement of small structures such as sulci can be accurately determined without a large minification error.

While Carlen *et al.* speculate the axonal sprouting and regrowth of support-

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Brain Stimulation Reinforcement: Implications of an Electrode Artifact

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