***AUTOCONTINGENCIES: SUPPRESSIVE AND ACCELERATIVE
EFFECTS OF PAIRS OF SHOCKS SUPERIMPOSED ON A
POSITIVELY REINFORCED OPERANT BASELINE***

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Previous research has shown that unsignaled shock may accelerate positively reinforced operant responding if each shock signals a subsequent shock-free period. In order to explore the boundary conditions of this effect, two experiments were performed. In Experiment 1, pairs of unsignaled shocks separated by 15, 30, 60, or 120 seconds resulted in suppressed responding during the briefest intershock interval, and in accelerated responding during the longer intervals. When the second shock in each pair signaled a shock-free period of at least 3 minutes, accelerated responding also followed offset of the second shock in all but the 30-second condition. In Experiment 2, the addition of a conditioned stimulus prior to each pair of shocks restored baseline responding, and eliminated accelerative control following the second shock only under the briefest intershock interval. The results are discussed in terms of the similarity between autocontingencies (shock/no-shock relations; Davis, Memmott, & Hurwitz, 1975) and recent modifications of the feature-positive procedure (e.g., Reberg & Memmott, 1979), which stress stimulus control by shock/no-shock relationships.

Key words: suppression, acceleration, autocontingencies, shock, rats

In 1975 Davis, Memmott, and Hurwitz demonstrated that unsignaled deliveries of shock superimposed on positively reinforced operant baselines could accelerate, rather than suppress, responding. Using a modified conditioned-suppression procedure, we explored the conditions under which shock/no-shock relationships termed *autocontingencies* resulted in conditioned acceleration. We found that if unsignaled shocks were separated by at least 3 min, the offset of each shock resulted in a period of accelerated responding. However, when each shock was signaled by a 30-s tone, the accelerative effects of the shock/no-shock autocontingency were virtually eliminated.

In the above experiment and in related ex-

periments demonstrating similar effects (e.g., Davis et al., 1975; Davis & MacFadden, 1978; Davis, Herrmann, & Shattuck, 1979), shock always signaled a shock-free period of at least 3 min. Thus, although a minimum 3-min shock-free period clearly provides an effective basis for an accelerative autocontingency, the question remains whether shocks separated by much shorter shock-free intervals will also result in conditioned acceleration. It seems reasonable to suppose that at some minimum intershock value, shock might act as a suppressor, rather than as an accelerator, of baseline responding.

The role of shock as a signal for subsequent shock also bears upon the emerging literature that deals with the feature-positive effect, whereby discriminations that are based upon a single distinguishing feature proceed more rapidly when that feature is presented on positive (i.e., S+) as opposed to negative trials (e.g., Jenkins & Sainsbury, 1969). Using a modification of the basic

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feature-positive procedure, Reberg and Memmott (1979) demonstrated that shock (the distinctive feature) may be a more effective signal for no shock than it is for subsequent shock. In their experiment, Reberg and Memmott superimposed either shock/shock or shock/no-shock pairings on a positively reinforced operant baseline. The effectiveness of these two predictive relations was inferred from changes in the rate and direction of the operant baseline; that is, when responding was suppressed between shocks in a pair, the inference was made that shock₁ was an adequate signal for the occurrence of shock₂. The similarity between this procedure and our autocontingency experiments stems from the fact that in both cases the primary predictive information about shock derives from the occurrence of previous shock. In short, shock₁ signals the presence or absence of shock₂.

There is reason to believe that the results of both procedures may depend upon the value of the interval between paired shocks. The following experiments explored the boundary conditions for obtaining conditioned suppression and conditioned acceleration of operant baselines when pairs of shock are superimposed.

EXPERIMENT 1

In our original research on this topic (Davis et al., 1975), we reported that shock offset resulted in substantial acceleration of food-reinforced lever pressing when each shock signaled a shock-free interval of at least 3 min. Using a modified feature-positive procedure, Bottjer and Hearst (1979) reported that a relatively brief 6-s interstimulus interval (ISI) successfully established food as a signal for subsequent food. In contrast, Reberg and Memmott (1979), using a longer (60-s) ISI value, reported that shock was an ineffective signal for subsequent shock. Collectively, these results suggest that the duration of the interstimulus interval is an important determinant of whether suppression or acceleration of

responding will result from paired presentation of events.

In the first experiment described here, we investigated various shock-free intervals in order to determine the temporal limits for obtaining conditioned suppression and conditioned acceleration. Pairs of shocks separated by intershock intervals (ISIs) of 15, 30, 60, and 120 s were superimposed upon a baseline of food-reinforced lever pressing, and the direction of rate change during the ISI was recorded. In addition, for half the subjects in each ISI condition, the time between pairs of shock (i.e., from offset of shock₂ to onset of shock₁ in the next pair) was separated by a minimum of 3 min; for the other half the minimum interval could be as brief as 9 s. This allowed us to replicate our original autocontingency manipulation and to enter both shocks of each pair into potential predictive relationships involving subsequent shock.

METHOD

Subjects

The subjects were 24 experimentally naive male hooded rats of the Long-Evans strain. (One animal, S15, died during the course of the experiment.) Each animal was reduced to approximately 80% of its free-feeding weight and was maintained at that level throughout the course of the experiment. All subjects were individually housed with water freely available in their home cages.

Apparatus

Three rodent test chambers and sound attenuating shells (Campden Instrument Co.) were used. A continuously operated exhaust fan provided ventilation and masking noise (76 dB). The lever protruded into the left side of the front wall of the chamber and required a minimum downward force of 15 g (0.15 N) for operation. In the lower center of the same wall was a food hopper into which 45-mg Noyes food pellets could be delivered.

The floor of each chamber was a grid of 16 stainless steel rods, 0.9 cm in diameter and

spaced 0.9 cm apart, through which scrambled shock could be delivered. Scrambled shocks (1 s, 0.6 mA) were delivered to the grids, walls, and lever of the chamber.

Procedure

After preliminary magazine and lever-pressing training, all rats were allowed to collect 100 pellets under a continuous reinforcement schedule. Over the next 2 days, subjects were gradually trained to respond on a variable interval (VI) 30-s schedule (range: 4 to 90-s). Variable-interval training continued for twenty 30-min sessions, by which time visual inspection of the cumulative response records revealed stable rates of responding.

After response rates had stabilized on the VI schedule, pairs of shock in which components of the pair were separated by inter-shock intervals (ISIs) of 15, 30, 60, or 120 s were superimposed on the lever-pressing baseline. Subjects were randomly assigned to one of 8 groups ($n = 3$) based on factorial combinations of ISI value (15, 30, 60, and 120 s) and on whether or not shock₂ in each pair predicted a minimum 3-min shock-free period. This minimum shock-free period defined the autocontingency (AC). Thus, half the subjects were exposed to a shock/no-shock autocontingency based upon shock₂ in each pair; the remainder were not. Subjects in $\overline{AC}$ groups were exposed to interpair intervals as brief as 9 s. The distributions of interpair intervals used for both AC and $\overline{AC}$ conditions are given in Figures 1 and 2.

Groups were identified with regard to their ISI value and to whether or not shock₂ predicted no shock under the AC. For example, Group 60-AC refers to the condition under which shocks in each pair were separated by a 60-s interval, and shock₂ predicted a minimum 3-min shock-free period. For all groups, three pairs of shocks were delivered in each 30-min session. Other than the minimum interpair interval imposed by the autocontingency, shock pairs were programmed to occur randomly during the

30-min session. This procedure remained in effect for 80 sessions.

RESULTS

Baseline

Baseline rates were derived from responding during all minutes of the session excluding the within-pair intervals and the 1-min periods following deliveries of shock₂. As indicated in Table 1, the occurrence of shock reduced baseline responding in all groups, although this effect was least pronounced under the briefest (15-s) ISI condition. The overall rate of baseline responding was not systematically affected by whether or not an autocontingency was programmed following shock₂. Also, there were no systematic fluctuations in local rates during the sessions, which permitted baseline rates to be derived from responding that occurred throughout the session, rather than from brief periods prior to each pair of shocks.

Responding During the ISI

Responding during the ISI, as well as during postshock₂ periods, was expressed in terms of conventional suppression ratios calculated from the formula $A/(A + B)$, where A refers to response rate during the designated period and B refers to baseline rate as defined above. These ratios are presented in Figure 3; the individual as well as pooled ISI response rates upon which they are based are presented in Table 1.

In general, the pattern of responding observed during the ISI was determined by ISI duration. The most extreme ISI conditions produced the most uniform behavioral results. Responding during the brief 15-s ISI was generally suppressed in both AC and $\overline{AC}$ groups. In contrast, the 120-s ISI produced consistent temporal patterning under both AC and $\overline{AC}$ conditions. As indicated in Table 1, response rates were typically accelerated during the early portion of the 120-s ISI, but progressively decreased and were ultimately suppressed in the period immediately prior to the delivery of shock₂.

Fig. 1. Distribution of fifty intershock pair intervals employed during final 25 conditioning sessions in the absence of a shock/no-shock autocontingency.

Fig. 2. Distribution of fifty intershock pair intervals employed during final 25 conditioning sessions under the shock/no-shock autocontingency.

Table 1

Individual and group response rates obtained during baseline, interstimulus interval, and postshock₂ periods, as well as suppression or acceleration ratios based upon these measures averaged over final five sessions in Experiment 1.

Condition/ Subject	Baseline Rs/min (% Preshock Rate Recovered)	Interstimulus interval (Successive 15-s Periods) ¹				ISI Suppression Ratio ²	Postshock ₂ Interval (Successive 15-s Periods) ¹				Postshock ₂ Suppression Ratio ²
15-AC											
S1	10.8 (17.1)	4.5				.29	13.0	14.4	17.6	20.3	.55
S2	9.1 (22.9)	4.3				.32	19.5	21.9	21.9	18.3	.68
S3	10.0 (15.5)	5.0				.33	21.0	15.3	18.0	15.3	.68
$\bar{X}$	10.0 (18.5)	4.6				.31	17.8	16.9	19.2	18.0	.64
15-AC											
S4	15.0 (27.9)	4.3				.22	17.6	14.7	7.7	11.5	.54
S5	10.2 (19.2)	5.6				.35	9.9	8.5	8.8	8.3	.49
S6	9.0 (19.2)	1.9				.17	4.5	4.0	6.1	4.3	.33
$\bar{X}$	11.4 (22.1)	3.9				.25	10.7	9.1	7.5	8.0	.45
30-AC											
S7	3.2 (5.8)	1.1	0.0			.26	2.9	1.6	0.8	1.3	.48
S8	7.5 (11.7)	2.1	2.1			.22	1.1	3.2	1.9	7.5	.13
S9	4.9 (9.3)	0.8	1.1			.14	1.9	1.6	2.1	4.5	.28
$\bar{X}$	5.2 (8.7)	1.3	1.1			.21	2.0	2.1	1.6	4.4	.30
30-AC											
S10	4.0 (7.2)	4.0	1.9			.50	4.5	4.0	5.9	3.7	.53
S11	2.2 (5.3)	0.8	0.9			.27	2.7	0.8	0.3	1.1	.55
S12	1.3 (2.4)	0.9	0.0			.38	2.9	0.8	1.1	0.8	.55
$\bar{X}$	2.5 (5.0)	1.9	0.9			.38	2.9	1.9	2.4	1.9	.54
60-AC											
S13	2.5 (4.8)	9.9	3.5	2.9	1.3	.80	16.3	5.9	2.4	1.9	.87
S14	2.2 (4.4)	5.6	2.1	1.6	0.5	.72	12.8	4.0	0.0	0.3	.85
$\bar{X}$	2.4 (4.6)	7.8	2.8	2.3	0.9	.76	14.6	5.0	1.2	1.1	.86
60-AC											
S16	5.4 (11.4)	1.6	1.1	0.7	0.4	.23	0.8	0.3	0.5	2.1	.13
S17	8.4 (13.6)	10.1	5.3	4.0	1.3	.55	10.6	2.9	5.6	3.7	.55
S18	4.9 (10.7)	1.1	0.3	0.3	0.3	.18	0.3	0.0	0.0	0.0	.06
$\bar{X}$	6.2 (11.9)	4.3	2.2	1.7	0.7	.32	3.9	1.1	2.0	1.9	.25
120-AC											
S19	8.3 (12.5)	10.9	10.9	10.4	7.2	.57	14.9	6.9	8.5	6.7	.64
		8.0	5.6	6.7	7.7						
S20	2.6 (5.3)	12.9	7.5	3.2	1.3	.83	16.3	7.2	5.9	2.1	.86
		1.1	1.1	3.5	2.1						
S21	4.5 (7.8)	9.3	4.5	0.8	2.7	.67	11.7	3.7	1.9	2.4	.72
		2.4	2.9	4.3	3.2						
$\bar{X}$	5.2 (8.5)	11.0	7.6	4.8	3.7	.69	14.3	5.9	5.4	3.7	.74
		3.8	3.2	4.8	4.3						
120-AC											
S22	3.4 (6.3)	5.6	3.7	1.9	1.1	.62	3.2	0.8	1.9	2.1	.48
		0.5	2.9	1.9	2.4						
S23	4.5 (7.6)	10.4	3.7	3.5	3.5	.70	12.5	9.1	4.5	6.1	.74
		1.1	3.2	2.1	1.3						
S24	6.4 (14.6)	22.1	5.3	3.2	1.6	.78	12.5	4.5	6.7	2.4	.66
		2.1	2.9	2.4	2.9						
$\bar{X}$	4.8 (9.5)	12.7	4.2	2.9	2.1	.70	9.4	4.8	4.3	3.5	.63
		1.2	3.0	2.1	2.2						

¹Calculations expressed in terms of responses per minute. ²Ratios based upon responding during initial 15-s period.

Fig. 3. Group mean suppression ratios obtained during intershock interval (ISI) and immediately following shock₂ of each shock pair during final five sessions in Experiment 1.

Nevertheless, accelerated responding that occurred early in the 120-s ISI was sufficient to result in an accelerative (i.e., $> .50$) suppression ratio value for all subjects under the 120-s condition.

Responding Following Shock₂

Table 1 and Figure 3 show the comparison of AC versus AC- conditions in the accelerative effects of the shock₂/no-shock autocontingency. Only the 15- and 60-s intershock intervals produced appreciably greater acceleration following shock₂ under the AC, and even here, there were substantial individual differences.

DISCUSSION

The duration of the ISI was a key determinant of whether the effects of paired shocks on the operant baseline were suppressive or accelerative. In those instances in which shocks were separated by relatively brief intervals (15- or 30-s), conditioned suppression typically occurred during the ISI, sug-

gesting that shock₁ acted as a signal for subsequent shock. These results are similar to those of Bottjer and Hearst (1979), who reported that food presentations separated by brief ISIs resulted in accelerated responding, indicating that food, under their procedure, had become a signal for subsequent food.

As in our previous experiments, shock separated by the relatively longer 120-s interval resulted in conditioned acceleration immediately following offset of shock (cf. Davis et al., 1975, 1979; Davis & MacFadden, 1978). These results again confirm the notion that shock may be an effective signal for no shock, provided an adequate shock-free period is programmed.

It is worth noting that both suppressive and accelerative effects must be evaluated with regard to the session baseline. Thus, for example, the suppressed rates recorded during the ISI under the 15-s condition were, in general, equal to or greater than the baseline rates obtained for animals exposed to the other conditions. Similarly, the accelerated rates recorded for the 120-s animals during the ISI were generally equal to the baseline level obtained for the 15-s animals.

If we assume that subjects did not discriminate between shock₁ and shock₂ at any but the briefest ISI, then otherwise puzzling effects at the longer ISI conditions may be explained. For example, the absence of conditioned acceleration following shock₂ in the 30-AC group may stem from generalization between shock₁ (which was suppressive) and shock₂. Similarly, evidence of conditioned acceleration following shock₂ in the 120-AC group is consistent with the subject's failure to discriminate between shock₁ (which was accelerative) and shock₂. In summary, in every case other than the 15-s ISI, the two shocks in each pair appeared to function as separate rather than paired events and resulted in the same direction and degree of behavioral control. There were instances, such as the 60-AC and 120-AC conditions, in which the animal's failure to discriminate between shocks 1 and 2 may have gone undetected because both the ISI and

postshock₂ periods resulted in acceleration. In such cases, generalization of ISI-based acceleration may have been masked by a similar effect produced by the shock₂ AC.

An important implication of the animals' failure to discriminate between shock₁ and shock₂ would be their corresponding failure to discriminate between the ISI and inter-pair interval (IPI). This would have direct bearing upon the detection of periods of relative "safety" from shock which, in turn, would directly affect baseline response rates. Such a view is consistent with the relatively lower baseline rates that occurred in all groups except those exposed to the 15-s ISI.

It is worth noting that discrimination between ISI and IPI would be enhanced by the reliable signaling of one of these periods. This is precisely what has been done in a number of recent modifications of the feature-positive procedure (e.g., Reberg & Memmott, 1979; Bottjer & Hearst, 1979). As previously noted, the modified feature-positive design bears considerable analogy to the present autocontingency procedure. However, it does differ in one important regard: The period between delivery of paired stimuli is typically signaled. A primary effect of this manipulation would be to facilitate the discrimination between the ISI and the IPI and to enhance the rate of baseline responding. This is borne out by examination of the most directly comparable groups—Reberg and Memmott's shock positive group, which received a 60-s ISI, and Group 60-AC in the present experiment. Although no autocontingency was formally programmed in the Reberg and Memmott (1979) experiment, an analysis of their procedure suggests that the postshock distribution, which was programmed on a VI 20-min schedule, approximated the shock/no-shock autocontingency manipulation used in the present experiment. Baseline rates under the signaled ISI procedure used by Reberg and Memmott were nearly eight times larger than the present rates.

It appears that with the exception of the 60-s ISI group, the predictive function of shock₂ modulated neither the direction nor

the amount of control exerted by shock₁. In one regard this result is surprising. In our original experiment on autocontingencies, we reported that the addition of a CS prior to each shock affected the degree of accelerative control resulting from the autocontingency; in short, a second predictive relation involving shock reduced the impact of the shock/no-shock relation (Davis et al., 1975). By analogy, it would not have been surprising to find that the addition of an autocontingency based on shock₂ would similarly restrict the predictive impact of shock₁. This was clearly not the case, as neither suppression (in the case of the 15- and 30-s ISIs) nor acceleration (in the case of the 120-s ISI) was affected. It is likely that this effect was over-ridden at the longer ISIs by the generalization that occurred between shock₁ and shock₂ in each pair.

EXPERIMENT 2

It has previously been reported that the addition of a Pavlovian conditioned stimulus (CS) prior to each shock attenuated the accelerative effects of a shock/no-shock autocontingency (Davis et al., 1975). The results of Experiment 1 demonstrated that neither suppressive nor accelerative control by shock₁ was attenuated when shock₂ was also in predictive relation to subsequent shock. The question remains whether control exerted by either shock₁ or shock₂ would be affected by the addition of a stimulus prior to the occurrence of each pair of shocks.

METHOD

Subjects

Subjects were the same as those used in Experiment 1. Two animals, S11 and S18, died during the course of the experiment.

Apparatus

The same test cages and equipment were used in Experiment 2 as in Experiment 1. In addition, a Mallory Sonalert (Model SC628H) was positioned directly over each test cage and produced a 1-min tone (90 dB).

Procedure

The conditions for the eight experimental groups were maintained as in Experiment 1. In addition, however, a 60-s presentation of tone preceded and terminated with the delivery of shock₁ in each pair. Signaled-shock conditions remained in effect for between 25 to 30 sessions.

RESULTS

The effects of introducing a tone prior to each pair of shocks are described in terms of three primary response measures, which are summarized in Table 2 and derive from data collected during the final five sessions.

Baseline Responding

As illustrated in Figure 4, the addition of a tone prior to each pair of shocks produced a substantial recovery of baseline responding at the shorter (15- and 30-s) ISI durations ($t[10] = 6.43$; $p < .001$). This rate increase occurred whether or not shock₂ predicted no shock under the autocontingency. In contrast, the addition of the CS had no apparent

effect on the baseline rates in either the 60-s or 120-s ISI conditions.

Conditioned Suppression

As shown in Table 2, responding during the 1-min tone prior to shock was markedly suppressed for all subjects. An analysis of variance using ISI duration and presence or absence of signal as main effects indicated that suppressive control exerted by the tone did not differ across conditions. Comparison of Figure 5 to Figure 3 suggests that the addition of the tone attenuated the difference between effects of the AC and $\overline{AC}$ conditions. Other effects of the preshock tone are expressed in terms of the previously defined $A/(A+B)$ suppression ratio and are illustrated in Figure 5.

Autocontingency Control

The effects of the tone on control exerted by both shocks in each pair are summarized in Table 2. These results are presented in a form that allows direct comparison with the

Fig. 4. Group baseline response rates obtained during the final five conditioning sessions in Experiment 1 (no CS prior to each shock pair), and in Experiment 2 (CS added prior to each shock pair).

Table 2

Individual and group response rates obtained during Experiment 1 and Experiment 2 base-lines, interstimulus interval, and postshock₂ periods, as well as suppression or acceleration ratios based upon these measures averaged over final five sessions in Experiment 2.

Condition/ Subject	Baseline Exp. 1	Rs/min Exp. 2	Tone Suppression Ratio	Interstimulus Interval (Successive 15-s Periods) ¹				ISI Suppression Ratio ²	Postshock ₂ Interval (Successive 15-s Periods) ¹				Postshock ₂ Suppression Ratio ²
15-AC													
S1	10.8	22.1	.03	13.3				.38	17.3	25.6	38.7	29.6	.44
S2	9.1	22.0	.03	3.5				.14	16.3	22.7	26.4	28.5	.43
S3	10.0	15.1	.03	4.8				.24	10.1	14.9	20.8	22.9	.40
$\bar{X}$	10.0	19.7	.03	7.2				.25	14.6	21.1	28.6	27.0	.42
15-AC													
S4	15.0	22.7	.04	6.6				.23	20.3	17.3	17.3	19.9	.47
S5	10.2	22.3	.02	13.1				.37	17.9	19.2	18.4	19.5	.45
S6	9.0	22.9	.00	6.9				.23	19.5	15.2	20.5	19.5	.46
$\bar{X}$	11.4	22.6	.02	8.9				.28	19.2	17.2	18.4	19.6	.46
30-AC													
S7	3.2	17.9	.00	7.2	4.0			.29	8.8	6.7	11.5	12.5	.33
S8	7.5	11.5	.02	2.7	4.0			.19	3.2	6.7	7.7	9.3	.22
S9	4.9	6.8	.05	4.0	4.8			.37	5.3	6.4	6.4	6.4	.44
$\bar{X}$	5.2	12.1	.02	4.6	4.3			.28	5.8	6.6	8.5	9.4	.33
30-AC													
S10	4.0	9.7	.02	5.3	2.1			.35	8.3	5.3	5.3	5.3	.46
S12	1.3	6.9	.02	2.9	1.1			.30	3.7	3.2	1.1	1.1	.35
$\bar{X}$	2.7	8.3	.02	4.1	1.6			.32	6.0	4.2	3.2	3.2	.41
60-AC													
S13	2.5	2.7	.09	18.4	0.3	0.3	3.7	.87	10.7	0.5	1.1	0.8	.80
S14	2.2	2.0	.00	5.6	1.6	0.3	1.6	.74	9.9	0.8	1.1	0.0	.83
$\bar{X}$	2.3	2.4	.04	12.0	0.9	0.3	2.6	.80	10.3	0.6	1.1	0.4	.81
60-AC													
S16	5.4	3.5	.00	4.0	2.9	2.1	2.4	.53	4.0	1.6	1.6	1.9	.53
S17	8.4	8.5	.02	14.4	11.5	9.9	8.5	.63	9.1	5.1	5.1	3.5	.52
$\bar{X}$	6.9	6.0	.01	9.2	7.2	6.0	5.5	.58	6.5	3.3	3.3	2.7	.52
120-AC													
S19	8.3	8.9	.04	12.3	14.1	11.5	11.7	.58	17.3	13.3	8.0	10.1	.66
				10.9	8.5	9.6	8.0						
S20	2.6	1.5	.08	8.3	7.2	4.5	2.4	.85	15.2	8.8	2.1	0.5	.91
				5.1	1.9	1.6	0.5						
S21	4.5	3.7	.05	6.1	8.5	5.3	5.9	.62	8.3	5.6	5.9	4.8	.69
				4.5	5.3	3.5	4.0						
$\bar{X}$	5.2	4.7	.06	8.9	9.9	7.1	6.7	.68	13.6	9.2	5.3	5.1	.75
				6.8	5.2	4.9	4.2						
120-AC													
S22	3.4	0.5	.04	5.3	6.7	6.1	1.9	.91	5.6	1.1	0.0	0.0	.92
				0.5	0.5	0.3	0.5						
S23	4.5	5.5	.06	14.9	13.4	12.3	7.2	.73	20.3	10.4	8.8	4.3	.79
				7.7	6.4	4.8	7.5						
S24	6.4	9.1	.03	15.5	6.7	3.2	2.7	.63	21.9	7.7	5.3	2.1	.71
				1.6	4.3	4.0	3.7						
$\bar{X}$	4.8	5.0	.04	15.5	12.3	9.4	5.4	.76	15.9	6.4	4.7	2.1	.81

¹Calculations expressed in terms of responses per minute.

²Ratios based upon responding during initial 15-s period.

Fig. 5. Group mean suppression ratios obtained during intershock interval (ISI) and immediately following shock₂ of each shock pair during final five sessions in Experiment 2.

results of Experiment 1, obtained prior to the addition of the tone.

Although suppressive control during the 15-s ISI was unchanged by the addition of the CS, a *t* test performed on data from the final 5 sessions for each rat (3 rats times 5 sessions) revealed that the tone virtually eliminated accelerative control previously established by shock₂ in Group 15 AC, ($t[14] = 1.92$; one-tailed $p < .05$)¹. The general level of control by both shocks in the 30-s ISI groups appeared to be unaffected by the addition of a preshock stimulus, as was control in the 60-AC group. In the 60-AC group, however, responding during the ISI was marginally increased from borderline

suppression in Experiment 1 (0.32) to borderline acceleration in Experiment 2 (0.58), although this difference was not statistically significant.

The effect of the CS on the 120-s condition was confined to the 120-AC group. These subjects showed an increase in accelerative control exerted by both shock₁ and shock₂. Although the increase during the ISI was relatively small ($t[14] = 1.57$; $p > .05$), the change following shock₂ was substantial, appearing both in absolute response rates and in ratio estimates of acceleration ($t[14] = 5.44$; $p < .01$).

DISCUSSION

The reduction of responding during the tone preceding shock replicates the basic conditioned suppression phenomenon (e.g., Blackman, 1977; Davis, 1968; Davis & Wright, 1979; Estes & Skinner, 1941).

The general elevation of baseline responding in ISI groups 15 and 30, which resulted from the addition of a preshock signal, is consistent with previous reports (e.g., Davis, Memmott, & Hurwitz, 1976). The question remains why this effect was confined to the briefer ISI conditions—that is, why the CS did not restore baseline responding under the 60- and 120-s ISIs. As previously noted, the paired shocks that occurred under the longer ISI conditions were less likely to be functionally effective as pairs. Hence, only the initial shock in each pair would have been signaled. In contrast, the CS would have served as a signal for both paired shocks under the briefer ISI conditions. The implication of this difference would be considerable for baseline responding. As Davis, Shattuck, and Wright (1981) have demonstrated, reductions in the proportion of shocks that are signaled yield corresponding reductions in baseline recovery. It would be tempting to argue that the present signaling manipulation amounted to a 100% CS-US reliability condition for ISI Groups 15 and 30, and a 50% condition for the longer ISIs. Indeed the 100% reliable signal does likely describe the 15-s condition. However, this interpretation appears inap-

¹The legitimacy of using repeated measures as data points within the *t* test is discussed by Kazdin (1982). The use of a one-tailed test in the present situation is based upon an a priori directional prediction stemming from previous research in which accelerative control by an AC was consistently reduced by the addition of a CS (e.g., Davis et al., 1975).

propriate for the 30-s ISI condition. By our own reckoning in Experiment 1, the 30-s interval was sufficiently long for shock₁ and shock₂ to be treated as separate rather than as paired events.

The loss of accelerative control by shock₂ under the 15-AC condition replicates our earlier findings, which were based on individual rather than on paired shocks. In that study, Davis et al. (1975) demonstrated that the addition of a CS prior to shock eliminated accelerative control by the shock/no-shock autocontingency. It is notable that signaling shock₁ modulated accelerative control by shock₂ only when the ISI was brief. That is, instances of accelerative control exerted by shock₂ under the 60-AC and 120-AC groups were not affected by the addition of a CS. This lends credence to our proposal that shocks were likely to be perceived as pairs under the briefest ISI, but functioned as separate events under the longer ISI durations.

GENERAL DISCUSSION

Collectively, the results of Experiments 1 and 2 demonstrate that the effects of pairs of shocks, ranging from suppression to acceleration, depend upon the duration of the time between those paired shocks. Our previous accelerative results (e.g., Davis et al., 1975), obtained when shock signaled freedom from shock for a minimum 3-min period, clearly represent a special case and are not the inevitable result of a shock-based autocontingency. The present results indicate that, depending on the ISI value, suppression as well as acceleration may result from exposure to an autocontingency procedure.

The present results bear upon behavioral control by paired events under the recently modified feature-positive procedure. This technique has resulted in conflicting results and produced a controversy surrounding the general superiority of feature-positive learning: Does exposure to a shock/shock procedure result in better discriminative learning than exposure to a shock/no-shock ar-

rangement? As previously noted, the answer to this question has been equivocal (cf. Bottjer & Hearst, 1979; Reberg & Memmott, 1979).

The present data help to resolve this conflict by focusing upon the contribution to feature-positive learning made by the autocontingency component of the procedure. Our data indicate quite clearly that a key to establishing control by paired events is the duration of the interval between them, which often goes unspecified in general procedural descriptions. In short, the distinction between feature-positive and feature-negative procedures involving paired events may be illusory; the ultimate effect of either procedure appears to depend upon the duration of the ISI. When the ISI is brief, stimulus₁ acts as a signal for stimulus₂, resulting in excitatory conditioning and, in the case of shock, conditioned suppression. In contrast, longer ISIs may result in inhibition of delay, and a range of effects from inhibition (acceleration) early in the shock/no-shock interval to excitation (suppression) later in the ISI.

The implications of this analysis for the existing literature are clear. Robust control by a feature-positive procedure requires excitatory conditioning (e.g., event₁ must signal event₂). This is likely to occur only in those cases when events 1 and 2 are separated by a brief ISI. As our data suggest, when the ISI exceeds 30 s, it is likely that some degree of inhibitory control will occur, thus attenuating excitatory control by event₁. This is precisely what Reberg and Memmott (1979) reported and found difficult to explain. In contrast, Bottjer and Hearst (1979), using an extremely brief ISI, reported sustained evidence of excitatory conditioning.

The failure in Experiment 2 to maintain postshock₂ acceleration in the 15-s condition underscores the importance of the behavioral baseline in the analysis of both conditioned suppression and conditioned acceleration. As has previously been noted (Davis, 1981; Davis et al., 1976, Hurwitz & Davis, 1983), the behavioral baseline is not

only a sensitive measure of the effects of Pavlovian procedures, but ultimately modulates the degree to which these effects may be detected using operant measures. For example, the addition of a CS prior to each shock pair produced a two-fold increase in baseline responding under the 15-s ISI condition. Previous research has shown that conditioned acceleration may be restricted to situations in which the behavioral baseline is relatively suppressed (e.g., Davis et al., 1976; Davis & McIntire, 1969). Thus, evidence of postshock₂ acceleration may have been eliminated because baseline responding had recovered to a level that prevented the detection of accelerative effects. The fact that postshock₂ responding was elevated substantially beyond the rate observed following shock₁ was incidental; its detection using a conventional $A/(A+B)$ ratio was unlikely because of a concomitant increase in the baseline, which itself may be the most revealing result of the signaling manipulation.

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