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Timing for the Absence of a Stimulus: The Gap Paradigm Reversed

Catalin V. Buhusi and Warren H. Meck
Duke University

Contrary to data showing sensitivity to nontemporal properties of timed signals, current theories of interval timing assume that animals can use the presence or absence of a signal as equally valid cues as long as duration is the most predictive feature. Consequently, the authors examined rats' behavior when timing the absence of a visual or auditory stimulus in trace conditioning and in a "reversed" gap procedure. Memory for timing was tested by presenting the stimulus as a reversed gap into its timed absence. Results suggest that in trace conditioning (Experiment 1), rats time for the absence of a stimulus by using its offset as a time marker. As in the standard gap procedure, the insertion of a reversed gap was expected to "stop" rats' internal clock. In contrast, a reversed gap of 1-, 5-, or 15-s duration "reset" the timing process in both trace conditioning (Experiment 2) and the reversed gap procedure (Experiment 3). A direct comparison of the standard and reversed gap procedures (Experiment 4) supported these findings. Results suggest that attentional mechanisms involving the salience or content of the gap might contribute to the response rule adopted in a gap procedure.

When an ongoing timed interval is interrupted by a break or gap, animals seem to suspend their temporal processing and choose a response rule that falls between two extremes: They may restart the entire timing process, a phenomenon usually called "reset" (Church, 1978; W. A. Roberts, Cheng, & Cohen, 1989) or they may "stop" timing for the duration of the gap and resume it after the gap (Church, 1978; Meck, Church, & Olton, 1984; S. Roberts, 1981; S. Roberts & Church, 1978). For example, in a peak-interval (PI) procedure (Catania, 1970; S. Roberts, 1981), rats are trained to respond for food after a signal is on for a fixed interval (FI; e.g., 30 s). Food trials are randomly intermixed with nonfood (probe) trials, in which the signal is on for a much longer duration. The typical result is that on probe trials, after the onset of the signal, the mean response rate increases and peaks at about the moment when the reinforcement is (sometimes) presented, suggesting that rats learn the stimulus-onset–reinforcement interval. In such a temporal production experiment, memory for the timed interval may be tested by interrupting the signal (e.g., after 15 s) by a break, during which the signal is off, and observing the effect of the gap or retention interval on the response peak time. Such a procedure is usually called a (standard) gap procedure (Roberts, 1981). In the 30-s PI procedure outlined above, a peak in response rate that occurs approximately 30 s after the gap is taken as evidence for a reset. On the other hand, a peak in response rate at about 15 s after the gap is taken as evidence for a stop. Such a stop rule has been demonstrated in rats using temporal production procedures (Church, 1978; Meck et al., 1984; S. Roberts, 1981; S. Roberts & Church, 1978) at a variety of gap durations, ranging from 2 to 15 s. On the other hand, in a similar gap procedure, a 9-s gap reset the timing process in pigeons (W. A. Roberts et al., 1989), suggesting possible species differences with respect to temporal processing and prompting for an explanation that would encompass both the stop and reset phenomena.

Two explanations of the gap phenomenon have been put forward. On the basis of results obtained in rats, which seem to adopt the stop rule, Church (1978; Gibbon, Church, & Meck, 1984) proposed the stopwatch metaphor of interval timing. A "switch" process was proposed to allow timing in the presence of the timed stimulus but to stop or even reset it during a break (i.e., in its absence; for a review see Church, 1984). To continue timing after a stop, animals were assumed to temporarily retain in memory the time value at the break. On the other hand, a *memory-decay* process was proposed to account for both the stop and reset phenomena irrespective of possible attentional processes (Cabeza de Vaca, Brown, & Hemmes, 1994). According to the memory-decay hypothesis, the memory for the currently timed interval decays in the absence of the timed signal. A short gap allows for little decay, accounting for the apparent stop of the timing process. If the gap is long enough, the memory for the currently timed interval decays completely, and animals restart the entire timing process after the gap. Because both the switch and memory-decay hypotheses rely on the capacity of the animals to retain the currently timed interval over a retention period, the gap procedure is thought to test the memory for timing in normal animals (Church, 1978; S. Roberts, 1981; S. Roberts & Church, 1978) and lesioned animals (Dietrich, Allen, & Bunnell, 1997; Meck et al., 1984; Olton, Meck, & Church, 1987).

This article focuses on evaluating the importance of stimulus attributes on interval timing in a gap procedure. In our approach to this problem, we examined interval timing in rats during an empty

Catalin V. Buhusi and Warren H. Meck, Department of Psychology: Experimental, Duke University.

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Correspondence concerning this article should be addressed to Catalin V. Buhusi, Department of Psychology: Experimental, Duke University, 9 Flowers Drive, Durham, North Carolina 27708-0086. Electronic mail may be sent to catalin.buhusi@duke.edu.

interval (in the absence of a stimulus) and the effect of inserting the stimulus itself as a reversed gap during its absence. In a reversed gap procedure, all stimuli are complementary relative to the standard procedure (Figure 1). Whereas in a standard gap procedure (upper panel of Figure 1) animals are trained to time for the presence of a stimulus, in a reversed gap procedure, animals are trained to time for its absence (middle panel of Figure 1). Whereas in a standard gap procedure, the effectively timed interval is evaluated by the moment when the response rate peaks in a (probe) trial in which the stimulus is present for a duration much longer than the criterion, in a reversed gap procedure, the stimulus is absent in the probe trial. Therefore, memory for the currently timed interval is tested in a standard gap procedure by breaking the ongoing presence of the timed signal. In a reversed procedure, memory is tested by breaking the timed empty interval by the stimulus itself. We applied similar logic to trace conditioning (bottom panel of Figure 1) to study the effect of a reversed gap on interval timing during the trace. Because the switch and decay hypotheses do not differentiate between the presence or the absence of a real signal, they predict that the duration, but not the content, determines the processing of the gap (but see Treisman, 1963). In other words, a reversed gap is predicted to have a similar effect as a standard gap of equal duration.

By manipulating the length of the signal stimulus, we evaluated in Experiment 1 the interval effectively timed in trace conditioning (e.g., the stimulus-onset–reinforcement or the stimulus-offset–reinforcement interval). We evaluated in Experiment 2 the memory for timing the lack of the stimulus in trace conditioning by the introduction of the stimulus as a reversed gap during the timed empty interval. We further evaluated in Experiment 3 the memory for timing an empty interval in a reversed gap procedure. We directly compared the standard and the reversed gap procedures in Experiment 4. Under the assumption that animals use the temporal dimension irrespective of the attributes of the real signal, we expected animals to react to a reversed gap as they would to a standard one. Previous experimental results (Church, 1978; Meck et al., 1984; S. Roberts, 1981; S. Roberts & Church, 1978) have suggested that when the currently timed interval is interrupted for 15 s or less, rats adopt the stop rule. With the parameters typically used (standard and reversed gaps of 1-, 5-, and 15-s duration), we expected animals to stop timing during both the standard and the reversed gaps. In contrast, the timing process was found to be partly or totally reset by a reversed gap (Experiments 2, 3, and 4). Results are discussed in the theoretical framework of current models of interval timing and time perception.

Experiment 1: Timing the Trace

In the spirit of Morgan's (1894) canon of parsimony, one may compare the temporal processes during the lack of the stimulus in trace conditioning and in the (standard) gap procedure. If the same interval timing processes (e.g., the putative retention of the currently timed interval in memory, stopping of timing, or memory-decay processes) are assumed to be at work during the (standard) gap and during the trace, then timing would be disrupted in the latter case. For example, in a recent computational implementation of the decay hypothesis, Hopson (1999) acknowledged that although the assumption of a passive memory-decay process allows the spectral timing model (Grossberg & Schmajuk, 1989) to describe some aspects of the gap paradigm, it also hinders its ability to deal with trace conditioning. Under a strict interpretation of the switch or decay hypotheses, animals would be able to time in delay, but not in trace, conditioning, because the trace—like a (standard) gap—presumably activates the switch or decay mechanisms, altering timing (e.g., stopping or resetting it).

Indeed, although there is evidence that animals do not have difficulty conditioning to the absence of a stimulus (Kamin, 1965; Liu & Moore, 1969; Mattson & Moore, 1964; Pavlov, 1927; Schneiderman, 1966), evaluation of timing behavior in trace conditioning supports the suggestion that timing and associative strength might be orthogonal dimensions (Brown, Hemmes, & Cabeza de Vaca, 1997; Brown, Hemmes, Cabeza de Vaca, &

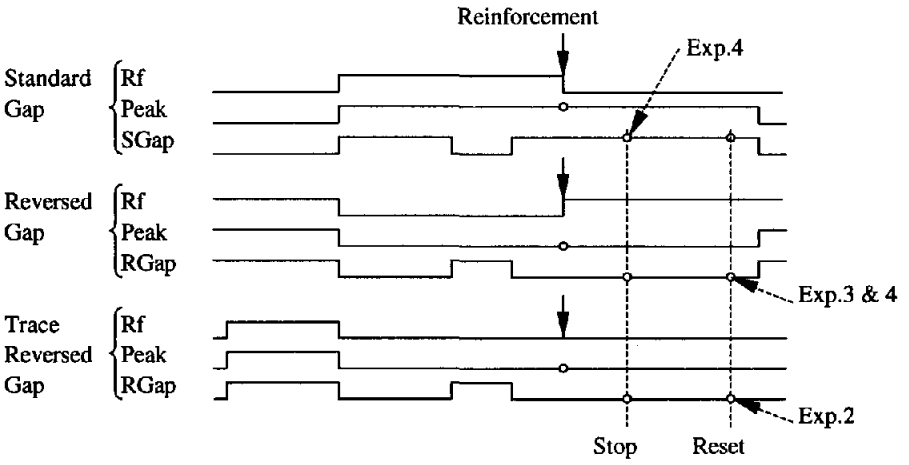

Figure 1. Reversing the gap. Top panel: standard gap procedure (Experiment 4). Middle panel: reversed gap procedure (Experiments 3 and 4). Bottom panel: a reversed gap in trace conditioning (Experiment 2). Solid arrows = moment of reinforcement; broken arrows = experimental results; Rf = reinforced trials; Peak = nonreinforced peak-interval trials; SGap = nonreinforced standard gap trials; RGap = nonreinforced reversed gap trials; open circles = response-rate peak time.

Pagano, 1993). In this line of evidence, pigeons discriminated empty intervals on the basis of their duration (Kraemer, Randall, & Brown, 1997; Mantanus, 1981; Santi, Ross, Coppa, & Coyle, 1999), but displayed a pattern of withdrawal from the key during key pecking in trace conditioning (Brown et al., 1993, 1997) and trace autoshaping (Lucas, Deich, & Wasserman, 1981). Similarly, although temporal specificity of trace conditioning was demonstrated in rats (Cole, Barnet, & Miller, 1995), the effectively timed interval was not directly evaluated. In line with Kamin (1965; Kehoe & Napier, 1991), Cole et al. suggested that in trace conditioning, rats learn the stimulus-onset–reinforcement interval but fail to bring experimental evidence in favor of this suggestion.

In Experiment 1, we evaluated (a) the timing of the response in trace conditioning in rats, (b) the time interval effectively learned, and (c) the influence of stimulus modality on interval timing, by using a PI procedure, which has been previously shown to allow for a dissociation between the rate of response and the timing of the response (S. Roberts, 1981). Rats were trained in a discrete-trials, trace-PI procedure (i.e., they received a food pellet for the first lever press after a specific interval in the absence of the stimulus [house light or white noise]). By manipulating the length of the stimulus, Experiment 1 evaluated which interval (i.e., the stimulus-onset–reinforcement interval or the stimulus-offset–reinforcement interval) was effectively timed for by rats under these conditions. If rats were to time exclusively the signal-onset–reinforcement interval, then varying the duration of the stimulus should not affect the timing of the response. On the other hand, if under these conditions, the offset were to exclusively control timing, the peak time should shift with stimulus duration but should stay fixed in respect to stimulus offset. If both cues (stimulus onset and offset) participate in controlling the interval timing process, then the behavior would vary in between the above extremes.

Materials and Method

Subjects. The subjects were 11 naive Sprague–Dawley male rats (*Rattus norvegicus*; Charles River Laboratories, Raleigh, NC), each 2-months old at the beginning of the experiment. Rats were housed in pairs in a temperature-controlled room, under a 12–12-hr light–dark cycle. Lights were on in the colony room from 7:00 a.m. to 7:00 p.m. Each daily session began at 11:00 a.m. Water was given ad lib in the home cage. The rats were maintained at 85% of their ad-lib weight by restricting access to food. Manipulations were carried out in accordance with standard procedures approved by Institutional Animal Care and Use Committee (IACUC) of Duke University.

Apparatus. The apparatus consisted of 10 standard operant boxes (Model ENV-001; Med Associates, St. Albans, VT) housed in sound-attenuating cubicles (Model ENV-019; Med Associates). Each operant box had inside dimensions of approximately 24 × 31 × 31 cm. The top, side wall, and door were 6-mm of clear plastic. The front and back walls were stainless steel, and the floor comprised 19 parallel stainless steel bars. Each box was equipped with three response levers (two retractable and one fixed; Model ENV-112; Med Associates) situated on the front wall of the box. All experimental procedures used only the left lever. According to schedule, 45-mg Noyes precision food pellets (Noyes, Lancaster, NH) were delivered in a food cup situated on the front wall, 1 cm above the grid floor, under the center lever, by a pellet dispenser (Med Associates). The stimuli used throughout experimental procedures were a 28-W, 100-mA house light mounted at the center–top of the front wall and a 78-dB white noise produced by a white noise speaker (Model ENV-225; Med Associates) mounted on the opposite wall from the levers. The intensity of the white noise was measured with a sound-level meter (Model 33-2050; Realistic Radio Shack, Ft. Worth, TX) from the center of the box.

Autoshaping. An autoshaping procedure was used during nine daily sessions to establish lever pressing for food pellets. Each lever press was rewarded on a continuous reinforcement schedule. The concurrent autoshaping procedure was such that the lever was retracted for 1 s and reinserted into the box followed by the delivery of a pellet every 60 s, independent of responding. This procedure continued for a maximum of 1 hr or until the rat had received 60 food pellets. Two of the rats failed to lever press reliably and were excluded from the experiment. The remaining rats (n = 9) were subsequently trained in a discrete-trials, trace fixed-interval (TFI) procedure.

TFI procedure. Autoshaping was followed by 15 daily sessions of a TFI schedule of reinforcement in which the stimulus (30 s in duration) was signaling the beginning of the trial (Figure 2). The first lever press 30 s after the offset of the stimulus was reinforced by the delivery of a food pellet and ended the trial. Trials were separated by a 60-s ± 30-s variable intertrial interval (ITI). Participants (n = 9) were randomly divided in two groups: light (n = 5) and noise (n = 4). The beginning of a trial was signaled by the house light in the light group and by the white noise in the noise group. TFI sessions consisted of 60 trials and were approximately 2 hr in duration.

Trace peak-interval procedure. Once TFI training was complete, rats received 14 daily sessions of a trace peak-interval (TPI) procedure (Figure 2). During these sessions, 30 reinforced TFI trials (in which the beginning was signaled by the cue assigned during TFI training) were randomly intermixed with 30 nonreinforced probe trials with similar temporal structure as the TFI trials. Trials were separated by a 60-s ± 30-s variable ITI. Each TPI session was approximately 2 hr in duration.

Testing. After the above procedures were completed, a TPI testing procedure with variable stimulus duration was conducted. During each of the five daily sessions, rats received 30 reinforced TFI trials (in which the beginning was signaled by the 30-s signal assigned during TFI training) randomly intermixed with 30 nonreinforced TFI probe trials in which the duration of the stimulus was randomly assigned at 15, 30, or 45 s (10 probe trials for each duration). Trials were separated by a 60-s ± 30-s variable ITI. Each test session was approximately 2 hr in duration.

Data collection and analysis. The paradigm was controlled through a Med Associates interface connected to a PC-compatible computer running a Med-PC software system (MED Associates, 1999). Responses were recorded in real time. Only data recorded during probe (nonreinforced) trials were used in the analyses. Additional programs were used to extract the daily mean response rates and individual peak times necessary for obtaining the performance measures described below.

Data were used to estimate the response peak time, peak rate, and precision of timing for each participant. The number of responses (in 3-s bins) was averaged daily over trials, to obtain a mean response rate for each participant. Daily mean response rate in the 60-s interval after the offset of the stimulus was fit using the Marquardt–Levenberg (Marquardt, 1963) iterative algorithm to find the coefficients (parameters) of the model that give the best fit (square-root minimization) between the equation and the data. The following generalized Gaussian+linear model was fit to the individual daily mean response rate:

$$R(t) = a \times \exp\{-.5 \times [(t - t_0)/b]^2\} + c \times (t - t_0) + d,$$

where t is the current moment (in 3-s bins), and $R(t)$ is the mean number of responses in Time Bin t. The iterative algorithm provided parameters a, b, c, d, and t_0. Parameter t_0 was used as an estimate of the daily peak time, $a + d$ was used as an estimate of the peak rate of response, and Parameter b was used as an estimate of the precision of timing. Daily estimates were submitted to repeated measures analyses of variance (ANOVAs) with session, stimulus modality, and duration as variables. Because the analyses failed to suggest a significant effect of session, data were collapsed across

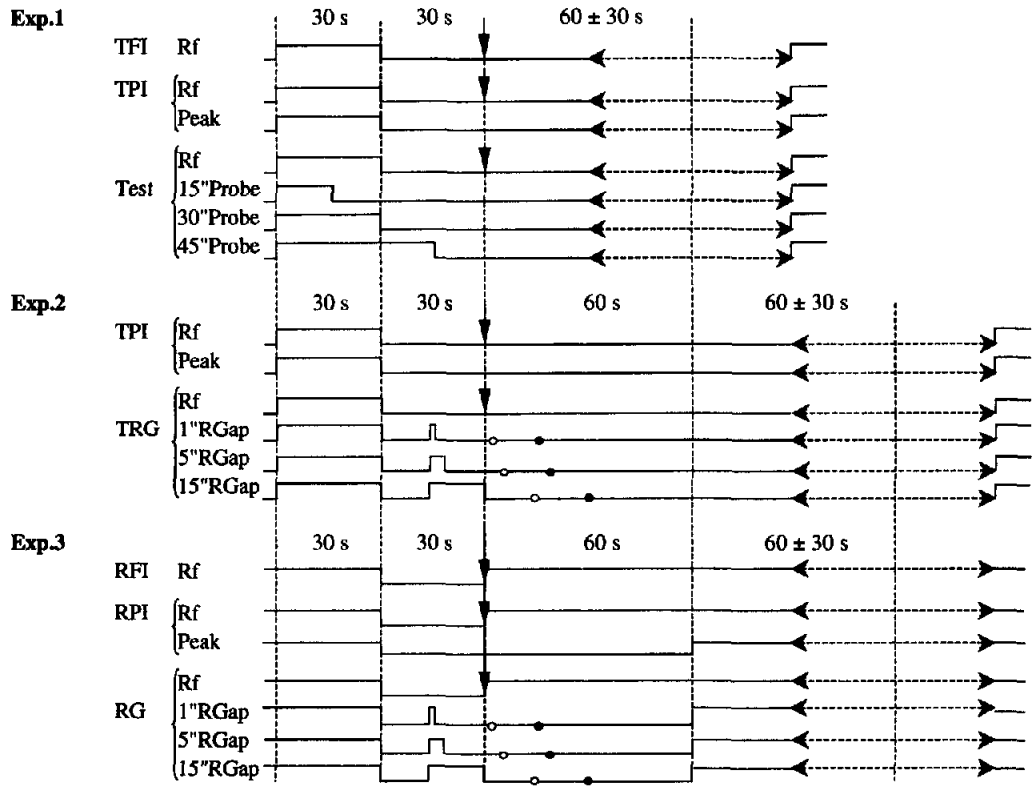

Figure 2. Details of experimental procedures used in Experiments 1–3. TFI = trace fixed-interval procedure; TPI = trace peak-interval procedure; TRG = trace reversed gap procedure; RFI = reversed fixed-interval procedure; RPI = reversed peak-interval procedure; RG = reversed gap procedure; vertical arrows = moment of reinforcement; horizontal arrows = variable intervals; Rf = reinforced trials; Peak = nonreinforced peak-interval trials; RGap = nonreinforced reversed gap trials; open circles = response rate peak time for stop rule; filled circles = response rate peak time for reset rule.

sessions and refit using the Marquardt–Levenberg algorithm. Results were submitted to repeated measures ANOVAs with stimulus modality and duration as variables.

Because of the inherent differences in response rate between rats, when averaging data over rats, the peak in the mean response rate tends to be influenced by rats with a higher response rate. Therefore, a mean percentage response rate was computed using data collapsed over sessions and is plotted in Figure 3 (Panels A, B, and C). A maximum response rate was computed for each rat, and the individual percentage maximum response-rate functions were averaged over rats. The peak time of the mean percentage response rate coincided with the mean of the individual peak times estimated using the fitting algorithm and used in statistical analyses. All statistical tests were evaluated at a significance level of .05.

Results

TPI procedure. The mean percentage maximum response rate during probe TPI trials is shown in Panel A of Figure 3. The response rate peaked at about the moment when rats were (sometimes) reinforced (i.e., 60 s after stimulus onset and 30 s after stimulus offset). The results suggest that rats learned at the moment of presentation of the reinforcement. A similar result has been reported in a classical conditioning TPI procedure using rabbit's nictitating membrane response (Smith, 1968). The result also extends the observation that response rate peaks at the moment of reinforcement in operant delay conditioning PI procedures in rats (e.g., S. Roberts, 1981).

The observation was supported by a repeated measures ANOVA with session and stimulus modality as variables, performed on the individual daily peak times from the 5 days of TPI probe trials. The analysis failed to reveal a significant effect for stimulus modality, $F(1, 7) = 0.04$, session, $F(4, 28) = 0.21$, or Session × Stimulus Modality interaction, $F(4, 28) = 1.50$. The mean peak time, 59.33 ± 1.49 s, was not found to be significantly different from the moment when rats were (sometimes) reinforced, $t(8) = 1.04$.

Testing under different stimulus durations. The mean percentage maximum response rate during probe TPI trials in which stimulus duration was randomly assigned at 15, 30, or 45 s is shown in Panels B and C of Figure 3. Data were collapsed across modalities and plotted relative to the onset of the signal in Panel B. Changes in signal duration resulted in a shift in response peak relative to its onset. When plotted relative to stimulus offset (Panel C), the mean response rates for the three stimulus durations superimposed relatively well. Response rate shifted in respect to stimulus onset, but peaked approximately 30 s from its offset, suggesting that rats used the offset-reinforcement interval as a criterion to respond to under the given experimental conditions.

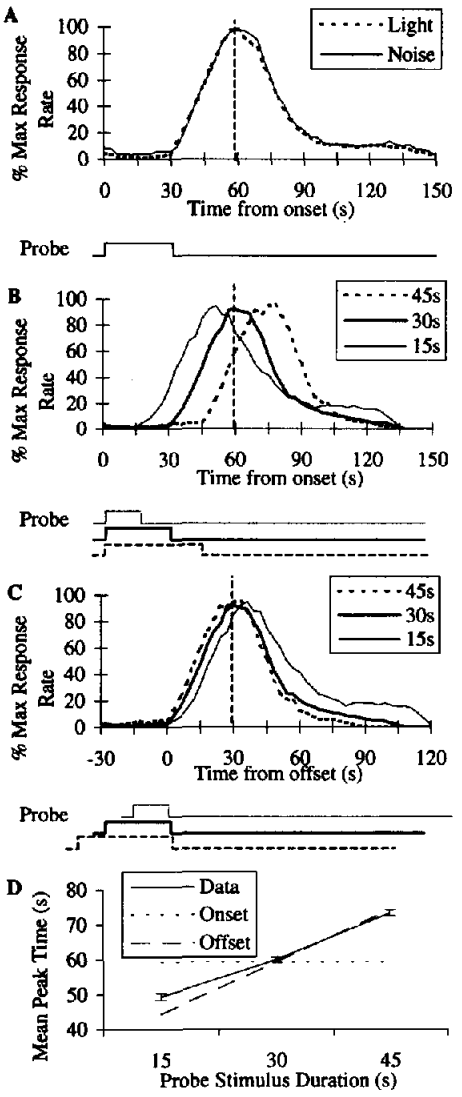

Figure 3. Timing in trace conditioning. Panel A: baseline probe trials. Panels B and C: probe trials with variable stimulus duration. Panel D: effectively timed interval. Solid line = mean peak time (± *SEM*) during probe trials with variable stimulus duration; broken lines = predicted peak time by the onset and offset of the stimulus. The diagrams under the graphs depict the probe stimuli.

The suggestion was partially supported by an analysis performed on individual daily peak times from the 5 days of TPI probe trials. A repeated measures ANOVA with session, stimulus modality, and stimulus duration as variables failed to reveal significant effects for stimulus modality, $F(1, 7) = 2.66$; and session, $F(4, 28) = 1.59$; and the Session × Modality, $F(4, 28) = 0.60$; Session × Duration, $F(8, 56) = 1.76$; and Session × Modality × Duration interactions, $F(8, 56) = 1.28$. Therefore, data were collapsed across sessions, and the parameters of the fitting equation were reestimated and resubmitted to a repeated measures ANOVA with modality and duration as variables. Stimulus duration accounted for a significant amount of variability in peak time, $F(2, 14) = 205.69$, $p < .01$. The mean peak times for the 15-, 30-, and 45-s stimulus durations were 49.35 ± 2.3, 59.94 ± 1.89, and 73.35 ± 2.05 s, respectively.

A predicted peak time was computed for each rat on the basis of the obtained peak time during the PI trials, such that if the response rate peaked sooner or later in the PI trials, it was expected to peak sooner or later in test trials. The mean predicted peak times by stimulus onset and offset, as well as the mean peak times observed in Experiment 1, are shown in Panel D of Figure 3. At both 30- and 45-s stimulus durations, the rate of response peaked at the moment predicted by the offset, $t(8) = 1.29$ for the 30-s probe, and $t(8) = 1.34$ for the 45-s probe. However, for the 15-s probe, the response rate peaked at a mean of 5.02 ± 2.75 s later than predicted by the offset, $t(8) = 4.20$. The latter result might have been due to a delay in starting the internal clock after stimulus offset (cf. S. Roberts, 1981) as well as to the use of both onset and offset as time markers. An analysis (that was based on data collapsed over sessions) of the peak rates of response in the test trials showed no effect of stimulus modality, $F(1, 7) = 1.15$, but a main effect of the duration of the signal, $F(2, 14) = 21.80$. Post hoc comparisons (Scheffe's method) of the peak response rates for the three stimulus durations suggested no differences in response rate between the trials with 30-s and 45-s stimulus duration, but a significant decrease in response for the 15-s stimulus duration. Nevertheless, a repeated measures ANOVA performed on precision parameters failed to suggest differences in precision among the three timing functions. In summary, the data generally supported the idea that under the given experimental conditions, in a trace conditioning paradigm, rats respond by using a rule that is mainly based on the offset-reinforcement duration.

Discussion

Using an operant-PI procedure in which rats were trained in trace conditioning, the results of Experiment 1 demonstrated that, irrespective of stimulus modality (auditory or visual), rats' lever pressing peaked around the moment when they were sometimes reinforced. Under the assumption that in a trace paradigm animals can use either or both the onset and offset of the signal as a time marker, Experiment 1 further evaluated the contribution of the two markers by manipulating the duration of the signal. As shown in Figure 3, variations in the length of the stimulus resulted in a shift in response peak time relative to its onset. The shift in response peak time suggests that rats used primarily the offset of the signal as a time marker. Peak time shifted in respect to stimulus onset but was located approximately 30 s after its offset (Panel C of Figure 3). A statistical analysis (summarized in Panel D of Figure 3) supports this notion for the 30-s and 45-s stimulus durations but not for the short 15-s stimulus duration for which the peak time was about 5 s later than predicted by the offset. The slight delay might have been attributed to variability in starting the internal clock corresponding to the absence of the stimulus. Indeed, there is good evidence (S. Roberts, 1981; S. Roberts & Church, 1978) that a 2- to 15-s break in the timed interval stops the timing process in rats. Therefore, the shortening of stimulus duration to 15 s might be perceived by rats as a break in the timed duration of the stimulus, so that the 5-s difference between the observed and predicted peak time might have been caused by a delay in switching from a stop–retain rule to a start rule.

Alternatively, the results obtained with the 15-s probe might have been due to the animals using both the onset and the offset as time markers. If both the onset-reinforcement and the offset-reinforcement intervals participated in controlling behavior, one would expect rats to respond better (i.e., with a higher peak rate of response) for the trained length of the stimulus than for both shorter or longer durations. A statistical analysis failed to find differences in response rate between the 30-s and 45-s probe but suggested a decrease in response rate for the 15-s probe. Taken together, the delay in peak time and the decrease in response rate in the 15-s probe support a possible contribution of the onset (or presence) of the stimulus on the response of the rats. Nevertheless, as shown in Panel C of Figure 3, when plotted relative to the offset of the stimulus, the three mean response-rate curves superimposed relatively well in absolute time units. A statistical analysis failed to find differences in precision of timing, which suggested that in all three situations animals used the same temporal criterion that was based mainly on the stimulus-offset–reinforcement interval.

The present results elucidate the durations learned in an operant trace conditioning paradigm in rats. Although trace conditioning has a long history of research (Pavlov, 1927), good timing in trace conditioning has been shown only in one classical conditioning study by Smith (1968). Using rabbit's nictitating membrane response (NMR) procedure, Smith found that the NMR peaked at the moment of reinforcement. Smith used a 50-ms tone followed 125, 250, 500, and 1,000 ms afterwards by a 1- or 4-mA periocular shock. Interestingly, in his procedure reinforced trials were randomly mixed with nonreinforced trials, making his study a PI procedure study, similar to the present experiment. Other studies, using the NMR response in rabbits (Schneiderman, 1966), salivary response in dogs (Ellison, 1964), keypecking in pigeons (Brown et al., 1993, 1997; Kraemer et al., 1997; Santi et al., 1999), and conditioned suppression of water licking in rats (Cole et al., 1995) only indirectly addressed the timing processes in the trace conditioning paradigm. The response measures, latency (Schneiderman, 1966), proximity to the key (Brown et al., 1993, 1997), and suppression of water licking (Cole et al., 1995), although allowing for the estimation of temporal specificity, failed to reveal the temporal relations learned by the animals. For example, although Cole et al. assumed that in trace conditioning, rats time the onset–reinforcement interval, our results suggested that rats are more likely to use the offset of the stimulus as a time marker and to refrain from responding in the presence of the stimulus. Similarly, Brown et al.'s (1993, 1997) procedure, although revealing a pattern of withdrawal from the response key in pigeons—a pattern that seems to have been modulated by the duration of the trial—failed to demonstrate clear stimulus–reinforcement duration learning. Furthermore, Brown et al. (1997) suggested that animals display a behavior controlled by the onset of the stimulus. Although some aspects of the control of timing might differ in classical conditioning procedures (such as the one used by Brown et al., 1993, 1997; and Cole et al., 1995) relative to the operant procedure used in our protocol, our results are more in line with a control by the offset of the stimulus.

In summary, results from Experiment 1 demonstrated timing an "empty" interval in the seconds–minutes range for rats in trace conditioning. Although interval timing seems to be controlled mainly by the offset of the stimulus, rats seem to recognize a signal of shorter duration. Results are in line with data that animals and people are able to time empty durations. Pigeons discriminate empty intervals on the basis of their duration (Kraemer et al., 1997; Santi et al., 1999), although filled durations are judged to be longer than empty ones. Similar results have been shown in humans (Allan, 1979, 1992; Thomas & Weaver, 1975). This evidence suggests possible differences between the effects of empty and filled breaks in timing. We evaluated this suggestion in Experiments 2, 3, and 4.

Experiment 2: Breaking the Trace

We evaluated memory for timing the lack of the stimulus by the introduction of the stimulus as a reversed gap halfway into the timed (stimulus-offset–reinforcement) interval in trace conditioning in Experiment 2. Under the assumption that rats use the temporal dimension in the same way during a standard and a reversed gap, we expected rats to use the same rule observed for standard gaps of equal durations. Church (1978; S. Roberts & Church, 1978) showed that rats stop timing during a (standard) gap of 2-, 4-, and 15-s durations. Similarly, S. Roberts (1981) found that a 5-s gap inserted at different locations into the timed interval, as well as a 10-s gap, shifts the peak time with a duration close to the length of the gap, suggesting the rats use a stop rule. Moreover, an investigation of the hippocampal involvement in the timing processes (Meck et al., 1984) showed that the introduction of a 5-s gap halfway into a timed 20-s interval caused a stop in the control group but a reset in the hippocampally lesioned rats. Given these results, we expected a reversed gap of 1-, 5-, or 15-s duration to stop timing in rats.

Materials and Method

Subjects and apparatus. The subjects and apparatus were the same as those used in Experiment 1.

Trace-PI procedure. Rats were retrained during seven daily sessions in a TPI procedure identical with that used in Experiment 1, with the only difference that the ITI was increased to a 120-s ± 30-s variable interval, to allow for the recording of the response rate for a longer interval (see Figure 2). TFI sessions were therefore approximately 3 hr in duration.

Trace reversed gap procedure. In the next six daily sessions, rats received 30 reinforced TFI trials randomly intermixed with 30 nonreinforced TFI reversed gap trials (10 probe trials for each reversed gap duration; see Figure 2). In each reversed gap trial, the stimulus was presented twice, for 30 s, signaling the beginning of the trial (as in the TFI trials) and also 15 s after the offset of its first presentation as a reversed gap. On the first testing day, we randomly used the following three reversed gap durations: 5, 15, and 30 s. Because we observed a reset at all three durations, in the following 5 days, we replaced the 30-s probe with a very short 1-s probe. In summary, in the 5 testing days for which data are reported here, the following three reversed gap durations were randomly used: 1, 5, and 15 s. Trials were separated by a 120-s ± 30-s variable ITI. TRG sessions were approximately 3 hr in duration.

Data collection and analysis. Data were collected and analyzed as described for Experiment 1, with the difference that the Marquardt–Levenberg algorithm used only the data collected in the 60-s interval after the offset of the reversed gap. All statistical tests were evaluated at a significance level of .05.

Results

Baseline training. The mean percentage maximum response rate during baseline trace probe trials is shown in the Panel A of

Figure 4. Response rate peaked about the moment when rats were (sometimes) reinforced (i.e., 30 s after stimulus offset). The results replicate those obtained in the baseline phase of Experiment 1. The observation was supported by a repeated measures ANOVA, with session and stimulus modality as variables performed on the individual daily peak times from the last 5 days of TPI probe trials. The analysis failed to suggest significant effects for stimulus modality, $F(1, 7) = 0.26$; session, $F(4, 28) = 2.33$; and Session × Stimulus Modality interaction, $F(4, 28) = 2.31$. The data were collapsed over sessions and the individual peak times reestimated. The mean peak time, 60.85 s ± 1.45 s, was not different from the moment when rats were (sometimes) reinforced, $t(8) = 1.34$.

Trace reversed gap procedure. The mean percentage maximum response rate during trace probe trials in which the stimulus was inserted as a reversed gap halfway into its timed absence, with a duration of 1, 5, or 15 s, is shown in the Panels B and C of Figure 4. Data were collapsed across modalities and plotted relative to the onset of the signal in Panel B. Introduction of the break resulted in a corresponding shift in peak time relative to gap offset. When plotted relative to the reversed gap offset (Panel C), the mean response rates for the three gap duration tests superimposed relatively well. Surprisingly, response rate peaked approximately 30 s from the offset of the gap, suggesting that rats used a reset rule.

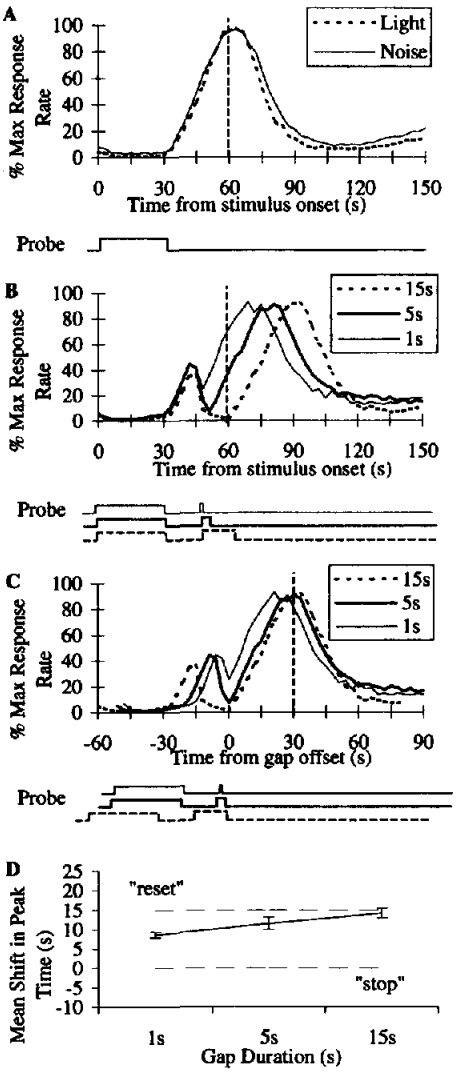

Figure 4. Timing in the trace gap procedure. Panel A: baseline probe trials. Panels B and C: reversed gap trials. Panel D: the rule used in trace gap trials. Solid line = mean shift in peak time (± *SEM*) during probe trace trials with reversed gaps; broken lines = predicted shift in peak time by stop and reset rules. The diagrams under the graphs depict the probe stimuli.

This suggestion was supported by two repeated measures ANOVAs performed on individual daily peak times from the 5 days of TPI probe trials. A repeated measures ANOVA with session, modality, and gap duration as variables failed to reveal significant effects for stimulus modality, $F(1, 7) = 0.96$; session, $F(4, 28) = 2.54$; Gap × Modality interaction, $F(2, 14) = 0.29$; Session × Modality interaction, $F(4, 28) = 2.47$; Session × Gap interaction, $F(8, 56) = 1.16$; and Session × Modality × Gap interaction, $F(8, 56) = 1.19$. Therefore, data were collapsed across sessions, the peak time reestimated, and data resubmitted to a repeated measures ANOVA with modality and gap duration variables. The gap duration, $F(2, 14) = 80.52$, was found to be significant above and beyond the modality of the stimulus. The mean peak times for the 1-, 5-, and 15-s gap duration were 70.46 s ± 1.97 s, 77.37 s ± 3.63 s, and 90.08 s ± 2.88 s, respectively.

The evaluation of the rule used by rats was based on the individual shift in peak time, computed for each rat using data collapsed over sessions. The individual shift was computed as the obtained peak time in the gap trial minus the obtained peak time of the rat in the trace PI trials minus the duration of the reversed gap. Therefore, a rat whose rate peaked sooner or later in the trace PI trials was expected to respond such that the peak time was sooner or later in the gap trials as well. A stop rule was expected to determine no shift, whereas a reset rule was expected to determine a 15-s shift. The predicted shift in peak times by stop and reset rules, as well as the mean shift in peak time observed in Experiment 2, are shown in Figure 4, Panel D. The shift in peak time was 8.61 ± 1.72 s for the 1-s gap, 11.52 ± 3.67 s for the 5-s gap, and 14.23 ± 2.78 s for the 15-s gap. The shift was not found to be different from the prediction that was based on a reset rule (15-s shift) for the 5-s gap, $t(8) = 2.19$, and the 15-s gap, $t(8) = 0.54$, but for the 1-s gap the shift was found significantly different from either the reset rule, $t(8) = 8.59$, or the stop rule, $t(8) = 11.56$. In summary, results suggested that in a trace conditioning paradigm, the introduction of the stimulus as a reversed gap halfway into its timed absence totally or partially resets timing.

An analysis of the response rate (on the basis of data collapsed over sessions) suggested a significant main effect of gap duration, $F(2, 14) = 4.52$. The mean peak rates were 43.48 ± 15.53, 46.59 ± 17.81, and 54.91 ± 21.19 responses/min for the 1-s, 5-s, and 15-s gap trials, respectively. Response rates were found to be significantly reduced relative to mean peak rate in the PI trials (59.32 ± 14.59 responses/min) in the 1-s [$t(8) = 4.07$] and 5-s [$t(8) = 2.89$] gap trials but not in the 15-s gap trials [$t(8) = 1.12$]. A post hoc comparison of the peak rate (Scheffe's method) suggested a significant difference in peak rate between the 1-s and 15-s gap trials as well as between the 5-s and 15-s gap trials. On the other hand, an analysis of the precision of timing suggested no differences between gaps.

Discussion

The results from Experiment 2 suggest that when rats time for the absence of a stimulus, the interruption of the timed absence by the stimulus itself results in a total or partial reset of the interval timing process. As shown in the Panel B of Figure 4, the response rate decreased during the reversed gap but then increased again after the gap and peaked about 30 s after the offset of the gap (Panel C of Figure 4). This suggestion was supported by a statistical analysis (summarized in Panel D of Figure 4). Rats were found to reset at the 5- and 15-s duration and partially reset at the 1-s reversed gap duration. Results are at odds with the assumption that the duration of the gap is the only criterion used by rats in the decision process. Previous results (Church, 1978; Meck et al., 1984; S. Roberts, 1981; S. Roberts & Church, 1978) established that at durations ranging from 2 to 15 s, a (standard) break prompts rats to use a stop rule. In contrast, reversed gaps of similar durations were found in Experiment 2 to prompt rats to reset the entire timing process.

The results cannot be simply attributed to rats confusing the offset of the gap with the offset of the stimulus. Results from Experiment 1 suggest that rats differentiate between the training signal and a signal shorter in duration. Whereas in Experiment 1, a shortening of the signal determined rats' response rate to peak later than that simply predicted by the offset alone, in Experiment 2 a 1-s gap determined the response rate to peak sooner than predicted simply by the offset. Taken together, the results suggest that rats do not confuse the reversed gap with the signal. Moreover, results from Experiment 2 confirm the suggestion that in trace conditioning (Experiment 1), rats learn the stimulus-offset–reinforcement interval. When a break was inserted in the trace, the peak time was shifted with an interval close to the stimulus-offset–reinforcement interval (30 s), suggesting that this is the interval that is to be considered interrupted by the gap.

Nonetheless, there is the possibility that the results might be peculiar to the trace procedure used in Experiment 2. Although partly at odds with results from Experiment 1, it is possible that in trace conditioning rats might retain in memory both the onset-reinforcement and offset-reinforcement intervals. Presumably, when the reversed gap is presented, the further increase in memory load might influence rats to free the memory resources allotted to the previous presentation of the stimulus and concentrate on the current event (i.e., on the reversed gap). According to this scenario, and in line with data from Experiment 1, the offset of the gap will control responding, and animals will respond with a rule that is similar to the reset rule. To control for such a possibility, in Experiment 3, we evaluated the effect of the reversed gap in a reversed gap procedure in which rats have to retain in memory only one time interval, as in the standard gap procedure. In fact, in Experiment 3 we used a procedure in which all stimuli were complementary to the standard gap procedure. Under the assumption that rats use the temporal dimension in the same way during a standard and a reversed gap, we expected rats to stop timing at all reversed gap durations in Experiment 3.

Experiment 3: Reversing the Gap Procedure

We evaluated memory for timing the lack of the stimulus in a reversed gap procedure in Experiment 3. The first response 30 s after the offset of the stimulus delivered a food pellet and set the stimulus back on for the duration of the ITI. In the probe trials, the stimulus was introduced as a reversed gap halfway into its timed absence. The paradigm used complementary signals relative to a standard gap procedure. Stimuli were on before rats were placed in the experimental boxes, during the ITI, and after the rats were removed from the boxes. Under these conditions and under the assumption that rats process the temporal dimension irrespective of the timed stimulus, we expected rats to use a stop rule, as suggested by experimental results with the standard gap (Church, 1978; Meck et al., 1984; S. Roberts, 1981; S. Roberts & Church, 1978). A reset at any of these durations would replicate the results of Experiment 2 and would suggest possible differences in temporal processing during the standard and reversed gaps.

Materials and Method

Subjects and apparatus. The subjects and apparatus were the same as those used in Experiment 1. However, the stimuli were interchanged between groups to minimize the transfer from previous training. Subjects had not been previously exposed to the new stimulus.

Reversed FI procedure. Rats received four daily sessions of a discrete-trials, reversed FI (RFI) schedule in which stimuli are complementary to a standard FI schedule (see Figure 2). The stimulus was presented throughout the schedule except for the timed interval, which was 30 s. During each trial, the first lever press 30 s after the offset of the stimulus was reinforced by the delivery of a food pellet, and it set the stimulus back on for the duration of the ITI. Trials were separated by a 150-s ± 30-s variable ITI. RFI sessions were approximately 3 hr in duration.

Reversed PI procedure. Once RFI training was complete, rats received six daily sessions of a reversed PI (RPI) procedure in which stimuli were complementary to a standard PI schedule (Figure 2). During each of these sessions, 30 reinforced RFI trials (in which the beginning was signaled by a 30-s lack of the stimulus) were randomly intermixed with 30 nonreinforced probe trials. During the probe trials, the stimulus remained off for a duration three times longer than the duration of the RFI criterion, before being terminated (set back on) independently of responding. Trials were separated by a 150-s ± 30-s variable ITI during which the stimulus was on. Each RPI session was approximately 3 hr in duration.

Reversed gap procedure. In each of the next five daily reversed gap sessions, rats received 30 reinforced RFI trials randomly intermixed with 30 nonreinforced gap trials (10 probe trials for each gap duration). In each gap trial, the stimulus was inserted halfway through its timed absence (i.e., 15 s after its offset; see Figure 2). The reversed gap durations were set randomly at 1, 5, and 15 s. At the termination of the gap, the stimulus was turned off for a duration that matched the duration used in the probe RPI trials and then set on independently of responding for the (variable) duration of the ITI. Trials were separated by a 150-s ± 30-s variable ITI. Reversed gap sessions were approximately 3 hr in duration.

Data collection and analysis. Data were collected and analyzed as described for Experiment 1, with the difference being that the Marquardt–Levenberg algorithm used only the data collected in the 60-s interval after the offset of the reversed gap. All statistical tests were evaluated at a significance level of .05.

Results

Baseline training. The mean percentage maximum response rate during the reversed PI probe trials is shown in Panel A of Figure 5. In both groups, the mean response rate peaked about the moment when rats were (sometimes) reinforced (i.e., 30 s after stimulus offset). The results replicated those obtained in the base-

line phases of Experiments 1 and 2 and complement those reported in the standard PI procedure (Meck et al., 1984; S. Roberts, 1981). The observation was supported by a repeated measures ANOVA, with session and stimulus modality as variables, performed on the individual daily peak times from the last 5 days of TPI probe trials. The analysis failed to suggest significant effects for stimulus modality, $F(1, 7) = 0.69$, session; $F(4, 28) = 0.99$; and the Session × Stimulus Modality interaction, $F(4, 28) = 1.56$. The data were collapsed over sessions, and the peak time was reestimated. The mean peak time, 30.98 s ± 2 s, was not found different from the moment when rats were (sometimes) reinforced, $t(8) = 1.13$. No differences in response rate or precision were found between groups.

Reversed gap procedure. The mean percentage maximum response rate during reversed gap probe trials in which the stimulus was inserted as a reversed gap halfway into its timed absence, with a duration of 1, 5, or 15 s, is shown in the Panels B and C of Figure 5. Introduction of the break resulted in a shift in peak time relative to gap offset. Data were collapsed across modalities and plotted relative to the onset of the signal in Panel B. When plotted relative to the reversed gap offset, the mean response rates for the three gap-duration tests superimposed relatively well, as shown in the Panel C of Figure 5. Response rate peaked approximately 30 s from the offset of the gap, suggesting that at all three gap durations rats used a reset rule.

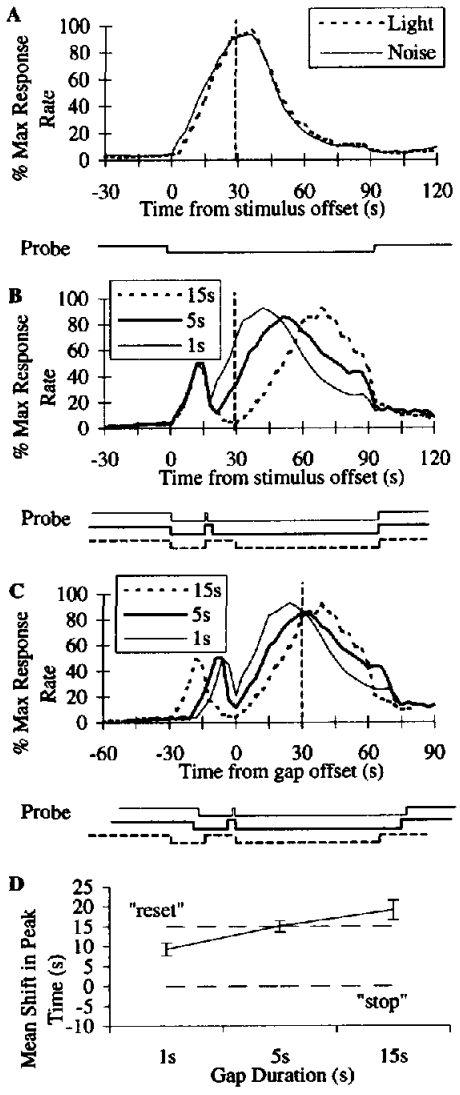

Figure 5. Timing in the gap procedure. Panel A: baseline probe trials. Panels B and C: reversed gap trials. Panel D: the rule used in reversed gap trials. Solid line = mean shift in peak time (± *SEM*) during reversed gap trials. Broken lines = predicted shift in peak time by stop and reset rules. The diagrams under the graphs depict the probe stimulus.

This suggestion was partly supported by two repeated measures ANOVAs performed on individual daily peak times from the 5 days of reversed gap probe trials. A repeated measures ANOVA with session, stimulus modality, and gap duration as variables failed to reveal significant effects for stimulus modality, $F(1, 7) = 0.19$; and session, $F(4, 28) = 2.62$; or for Gap Duration × Stimulus Modality, $F(2, 14) = 0.16$; Session × Stimulus Modality, $F(4, 28) = 0.32$; Session × Gap Duration, $F(8, 56) = 1.64$; and Session × Stimulus Modality × Gap Duration interactions, $F(8, 56) = 1.54$. Therefore, data were collapsed across sessions, the peak time reevaluated and data resubmitted to a repeated measures ANOVA with stimulus modality and gap duration as variables. The gap duration was found to be significant above and beyond the modality of the stimulus, $F(2, 14) = 97.39$. The mean peak times for the 1-, 5-, and 15-s gap durations were 41.29 s ± 3.52 s, 51.04 s ± 3.74 s, and 65.03 s ± 5.42 s, respectively.

The evaluation of the rule used by rats was based on the individual shift in peak time, computed for each rat using data collapsed over sessions. The individual shift was computed as the obtained peak time in the gap trial minus the obtained peak time of the rat in the reversed PI trials minus the duration of the reversed gap. Therefore, a rat whose rate peaked sooner or later in the reversed PI trials was expected to respond such that the peak time was sooner or later in the reversed gap trials as well. A stop rule was expected to determine no shift, whereas a reset rule was expected to determine a 15-s shift. The shift in peak time was computed for each rat, and the data were analyzed. The predicted shift by a stop and reset rule as well as the mean shift in peak time observed in Experiment 3 are shown in Panel D of Figure 5. For the 5-s and 15-s gaps, the shift was not significantly different from that predicted by a reset rule (15-s shift), $t(8) = 0.04$ for the 5-s gap, and $t(8) = 1.61$ for the 15-s gap. On the other hand, the shift by a 1-s reversed gap (9.31 s ± 3.71 s) was found different from either the reset, $t(8) = 3.53$, or the stop rule, $t(8) = 5.79$. For the 1-s reversed gap, results might be interpreted as a partial reset (see S. Roberts, 1981).

In summary, the results suggest that in a reversed gap procedure, the introduction of the stimulus as a break into its timed absence resets the timing process for 5- and 15-s gap durations. More important, a very short 1-s reversed gap is able to partially reset the timing process. The daily mean response rate in the first session of 1-s reversed gap trials in Experiment 3 as well as the Marquardt–Levenberg fit are shown for each of the rats in Figure 6. The responses are depicted relative to the 1-s gap offset. Parameter t_0 shows the estimated peak time in the 1-s gap session. A peak in response rate 15 s after gap offset was taken as evidence for a stop, whereas a peak about 30 s after gap offset was taken as evidence for a reset rule. Left panels show data for individual rats from the noise group. Except for Rat 3, all other animals seem to reset. On the other hand, in the light group (right panels) 3 rats (Rats 4, 6,

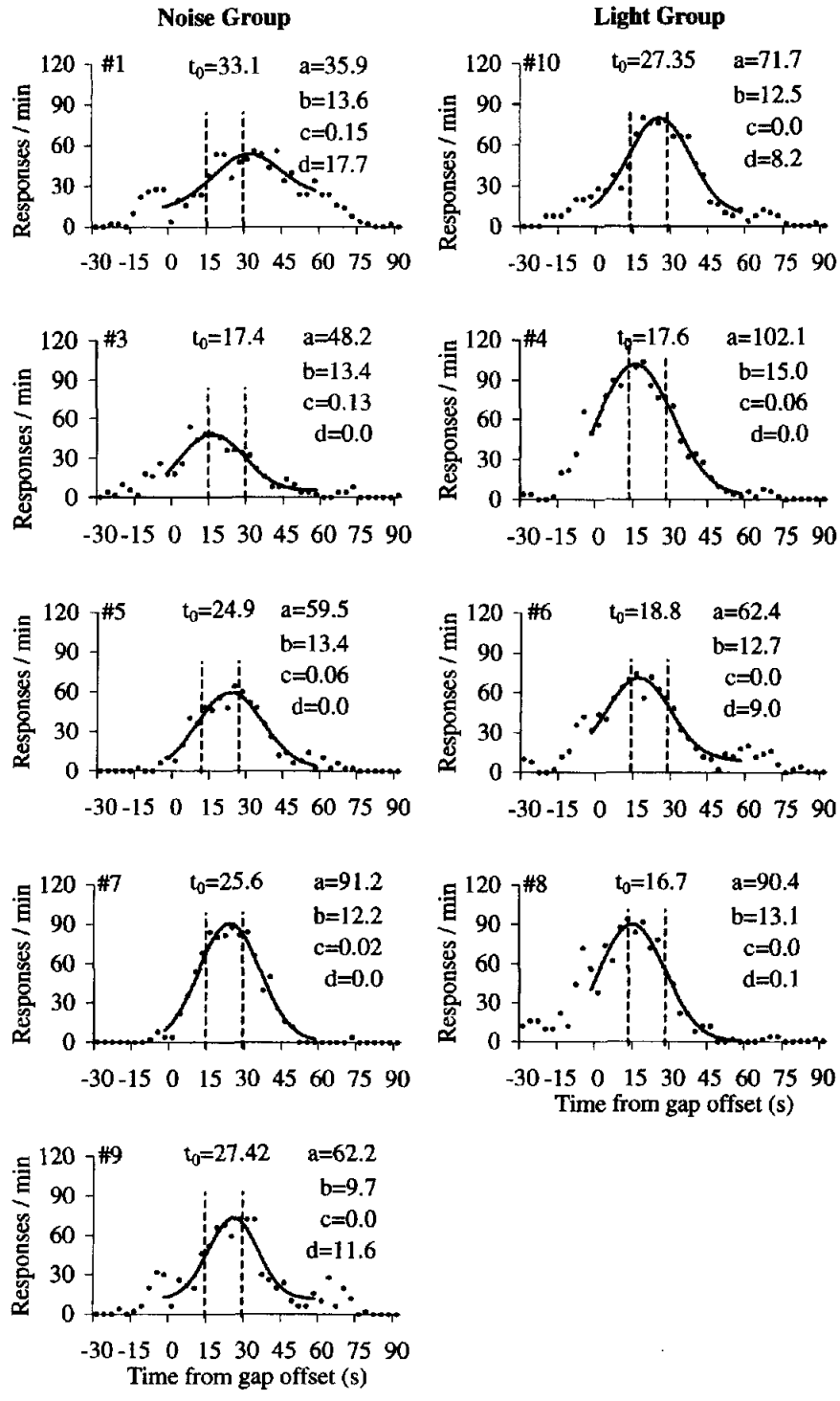

Figure 6. Obtained and fit mean response rate in the first test session for the 1-s reversed gap probe trials. Left panels: noise group (n = 5); right panels: light group (n = 4). Abscissa: time relative to the 1-s gap offset. The curve is the Marquardt–Levenberg fit for the individual mean response rate. Parameter t_0 estimates the peak time. A peak at about 15 s after gap offset is evidence for a stop (left vertical line), whereas a peak at about 30 s after gap offset is evidence for reset (right vertical line).

and 8) use the stop rule, but Rat 10 seems to reset. Taken together, 5 out of the 9 rats used a reset rule at such a short gap duration, 1 s. Results demonstrate not only that the reset mechanisms are active in rats (cf. Roberts et al., 1989) but that a reset can be completed in about a second. A similar suggestion about the rapidity of the reset mechanism was made by Church (1980).

Discussion

The results from Experiment 3 suggest that when rats time for the absence of the signal stimulus in a reversed gap procedure, the interruption of the timed absence by the stimulus results in a total or partial reset of the timing process, irrespective of stimulus (reversed gap) modality. Results obtained in this reversed gap procedure (Figure 5) complement those obtained in the (standard) gap procedure in rats (Meck et al., 1984; S. Roberts, 1981). In the reversed gap procedure, the mean response rate peaked about the moment when rats were (sometimes) reinforced, suggesting that rats time the empty interval. The interruption of the timed empty interval by the stimulus resulted in a shift in response peak that suggests the use of a reset rule (Panel D of in Figure 5). In the first session of testing, a very short 1-s reversed gap was found to be able reset the timing process in 5 out of the 9 rats (Figure 6). In summary, the results suggest that in a reversed gap procedure rats use a reset rule at gap durations as short as 5 s.

These results are worth noting for a number of reasons. First, standard breaks ranging from 2- to 15-s durations (Church, 1978; Meck et al., 1984; S. Roberts, 1981; S. Roberts & Church, 1978) prompted rats to use a stop rule. In contrast, a reversed gap of the same duration (5 s and 15 s) prompted rats to reset the entire timing process in Experiments 2 and 3. Therefore, results suggest that in a gap procedure, timing is influenced not only by the duration of the gap but by other attributes of the gap or of the timed interval. In other words, these results question the very interpretation of the data previously obtained in (standard) gap procedures as reflecting solely the timing processes involved in this paradigm.

Second, the results show that the reset of the timing process by a break in the timed interval can be obtained in rats, which were previously thought to prefer a stop rule (see, e.g., W. A. Roberts et al., 1989). Previous results supported the notion of species differences in the attitude toward a (standard) break between rats, which seemed to stop timing during a (standard) gap (Church, 1978; Meck et al., 1984; S. Roberts, 1981; S. Roberts & Church, 1978), and pigeons, which seemed to be resetters (W. A. Roberts et al., 1989). By demonstrating a reset behavior in rats, the present experiments do not endorse the notion of species differences in temporal processing, and point toward procedural differences. Some other parameters of the procedure (other than timing) seem to be taken into account by rats in their timing behavior. Such known factors in the reset process in rats are delivery of reinforcement (Staddon, 1974) and opportunity for response (Church, 1980). For example, in an estimation experiment in which rats were required to classify a sample as "short" or "long" in duration by pressing the left or right lever, the omission of the insertion of the levers was shown not to interfere with timing in subsequent trials, suggesting a reset of the clock over the ITI. Because in the cited study the minimum ITI was 2 s, Church (1980) concluded that rats are capable of resetting the clock in less than 2 s. The present results are in line with this suggestion. A short 1-s reversed gap was able to reset the timing process under present experimental conditions in half of the rats. Moreover, the present results show that the reset process can be manipulated in rats in a reversed gap procedure.

Third, results from Experiment 3 replicate those obtained in Experiment 2. Under both procedures, the introduction of a reversed gap during the empty interval prompted rats to use a reset rule. Taken together, results from Experiments 2 and 3 suggest that the reset process was not determined by details of the two procedures. For example, in Experiment 2, the reversed gap was introduced in trace conditioning, in which (a) animals might have retained two intervals in memory, (b) the content of the reversed gap (stimulus on) was different from that of the ITI (stimulus off), and (c) the stimulus was thought (see, e.g., Brown et al., 1993) to exert control over behavior as an occasion setter (Holland, 1980). On the other hand, in Experiment 3, (a) animals had to retain only the offset-reinforcement interval, (b) the content of the reversed gap (stimulus on) was similar to that of the ITI, and (c) there were no reasons to believe that behavior was controlled by occasion setters. Moreover, because in Experiments 2 and 3 the reversed gap was much shorter than the ITI, it is very unlikely that the reset was due to a confound between the gap and the ITI. Therefore, these results from Experiments 2 and 3 suggest that the memory load, the content of the gap relative to the ITI, the type of control by the stimulus, and the confound between the gap and the ITI were not likely to be determinants of the reset of the interval timing process under current experimental procedures.

Experiment 4: A Comparison of Standard and Reversed Gap Procedures

The standard and reversed gap procedures were directly compared in Experiment 4. First, because it is possible that the reset obtained in Experiment 3 might have been due to rats' previous experience in resetting (Experiment 2), Experiment 4 replicated the previous findings using naive rats. Second, a better estimate of the effectively timed interval can be obtained by having both PI probes and gap trials in the same testing phase of the experiment. Therefore, in Experiment 4, the shift in peak time was evaluated by comparing the gap trials with the PI probe trials. Most important, Experiment 4 compared the effect of standard and reversed gaps of equal durations. A failure to replicate the stop behavior in the standard procedure might be an indication of some procedural or apparatus differences among experiments conducted in different laboratories. On the other hand, one might expect rats to stop timing in the standard procedure, as suggested by previous experimental results (Church, 1978; Meck et al., 1984; S. Roberts, 1981; S. Roberts & Church, 1978) but to reset timing in the reversed procedure, as suggested by results of Experiment 3. A replication of the results obtained in Experiments 2 and 3 would support the notion of basic differences in temporal processing during the standard and reversed gaps.

The standard and reversed procedures use complementary signals (see Figure 7). Whereas in the standard group rats were trained to time for the 30-s presence of a stimulus, in the reversed group rats were trained to time for the 30-s absence of a stimulus. In the standard group the effectively timed interval was evaluated by the peak time in a (standard) probe PI trial in which the stimulus was on much longer than the criterion, but in the reversed gap

procedure, the stimulus was off in the (reversed) probe trial. Therefore, memory for the currently timed interval was tested in the standard group by breaking the ongoing presence of the timed signal. In the reversed group, memory was tested by breaking the timed empty interval by the stimulus itself. Similar to the previous design, in the reversed procedure the timed stimulus (house light) was on before rats were placed in the experimental boxes, during the ITI, and after the rats were removed from the boxes. We denote signal as the presence of the stimulus (house light) in the standard procedure and the absence of the stimulus in the reversed one.

Materials and Method

Subjects and apparatus. The subjects were 10 naive Sprague–Dawley male rats (*Rattus norvegicus*), 2 months old at the beginning of the procedure. The apparatus was the same used in Experiment 1, with the difference being that each experimental chamber was ventilated by a fan producing a 66-dB sound throughout the experimental procedures. The stimulus used throughout the procedures was the same house light used in previous experiments.

Autoshaping. An autoshaping procedure (as described for Experiment 1) was used during six daily sessions to establish lever pressing for food pellets. One rat failed to lever press reliably and was excluded from the experiment. The remaining rats (n = 9) were randomly assigned to one of two groups: standard (n = 5) or reversed (n = 4).

Standard and reversed FI procedures. Rats received 10 daily sessions of a discrete-trials, 30-s, fixed-interval schedule. The standard group received 60 standard FI (SFI) trials, whereas the reversed group received 60 RFI trials (as described for Experiment 3). In each trial, the signal was active for the timed interval; the first lever press 30 s after the beginning of the signal was reinforced by the delivery of a food pellet and terminated the signal for the duration of the ITI. Trials were separated by a 150-s ± 30-s variable ITI. Sessions were approximately 3 hr in duration.

SFI and RFI procedures. After the 30-s FI training was complete, rats received 14 daily sessions of a PI procedure: standard peak-interval (SPI) in the standard group and RPI in the reverse group. During each session, the rats received 30 reinforced FI trials randomly intermixed with 30 nonreinforced probe trials in which the signal remained active for a duration four times longer than the duration of the FI criterion, before being terminated. Trials were separated by a 150-s ± 30-s variable ITI. Each session was approximately 3 hr in duration.

Standard and reversed gap procedures. In each of the next two daily gap sessions, rats received 32 FI trials randomly intermixed with 32 nonreinforced probe trials (8 PI trials and 8 nonreinforced gap trials for each of the three gap durations). In a gap trial, the signal was interrupted halfway through its timed interval (i.e., 15 s after its beginning; see Figure 7). The gap durations were set randomly at 1, 5, and 15 s. At the termination of the gap, the stimulus was reactivated for a duration that matched the duration used in the probe PI trials and then deactivated independently of responding for the (variable) duration of the ITI. Trials were separated by a 150-s ± 30-s variable ITI. Gap sessions were approximately 3 hr 15 min in duration.

Data collection and analysis. Data were collected and analyzed as described for Experiment 1, with the difference being that the Marquardt–Levenberg algorithm used only the data collected in the 60-s interval after the termination of the gap. All statistical tests were evaluated at a significance level of .05.

Results

Data from the two test sessions were collapsed and submitted to a statistical analysis. In both groups, the mean response rate in the PI trials peaked about the moment when rats were (sometimes) reinforced (i.e., 30 s after the beginning of the signal). A one-way ANOVA with procedure as a variable failed to reveal a difference in peak time, $F(1, 7) = 0.05$. The mean peak time in the PI trials, 31.83 s ± 2.33 s from the beginning of the signal, was not found to be significantly different from the 30-s criterion, $t(8) = 1.81$. For the standard group, the results replicated those reported by S. Roberts (1981). For the reversed group, the results replicated those obtained in the baseline phase of Experiment 3.

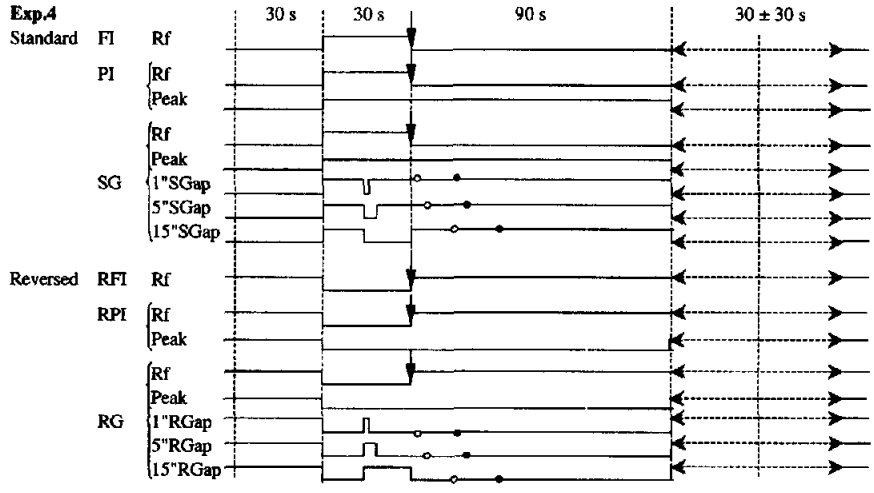

Figure 7. Details of experimental procedures used in Experiment 4. FI = standard fixed-interval procedure; RFI = reversed fixed-interval procedure; PI = standard peak-interval procedure; RPI = reversed peak-interval procedure; RG = reversed gap procedure; SG = standard gap procedure; vertical arrows = moment of reinforcement; horizontal arrows = variable intervals; Rf = reinforced trials; Peak = nonreinforced peak-interval trials; SGap = nonreinforced standard gap trials; RGap = nonreinforced reversed gap trials; open circles = response-rate peak time for stop rule; closed circles = response-rate peak time for reset rule.

The mean percentage maximum response rate during reversed gap probe trials in which the signal was interrupted by a 1-, 5-, or 15-s gap is shown in Figure 8. Panel A shows that a 1-s gap minimally affected the rate of response in the standard group (left) but shifted the peak of the response rate in the reversed group (right) in a manner that suggests a (partial) reset of timing. Similarly, Panel B shows that a 5-s gap minimally shifted the rate of response in the standard group (left) but shifted the peak of the response rate in the reversed group (right) in a manner that suggests a (total) reset of timing. However, a 15-s gap seems to have reset timing irrespective of procedure (Panel C). The results suggest marked differences in the effects of a standard and reversed gap.

This suggestion was supported by a repeated measures ANOVA performed on the individual response peak time estimated using the responses during the two gap sessions. A repeated measures ANOVA with procedure and gap duration as variables revealed a significant effect of procedure, $F(1, 7) = 19.83$; a significant effect of gap duration, $F(2, 14) = 159.69$; as well as a significant Gap Duration × Procedure interaction, $F(2, 14) = 4.48$. A shift in peak time was computed for each rat by subtracting the obtained peak time in the PI trial and the duration of the gap from the obtained peak time during the gap trials. No shift would suggest a stop rule, whereas a shift of about 15 s would suggest a reset rule. The peak-time shift was submitted to a repeated measures ANOVA that confirmed the previous analysis: a significant effect of procedure, $F(1, 7) = 25.02$; a significant effect of gap duration, $F(2, 14) = 15.92$; as well as a significant Gap Duration × Procedure interaction, $F(2, 14) = 4.48$. In the standard group, the shift was not found to be different from that predicted by a stop rule for both

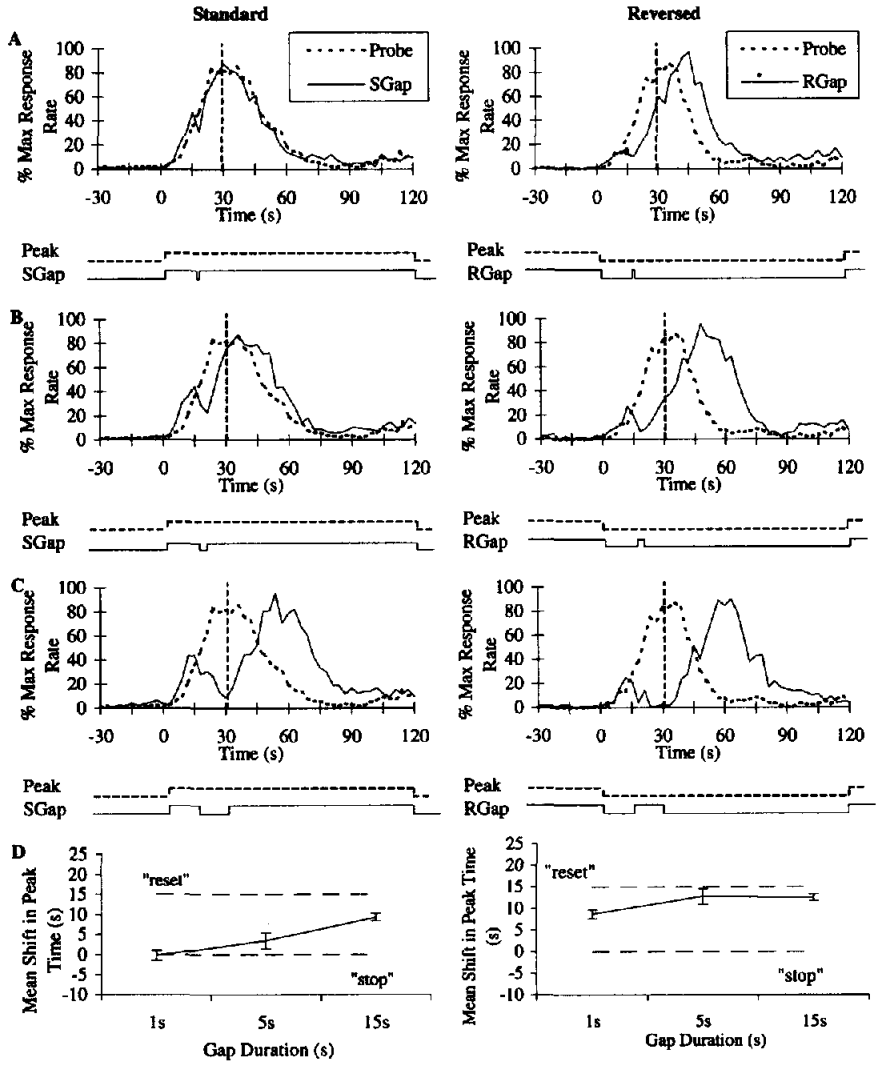

Figure 8. Standard and reversed gap procedures. Left: standard gap procedure; right: reversed gap procedure for peak-interval and 1-s gap (Panel A), 5-s gap (Panel B), and 15-s gap trials (Panel C). Panel D: the rule used in gap trials. Solid line = mean shift in peak time (± *SEM*) in gap trials; broken lines = predicted shift in peak time by stop and reset rules. The diagrams under the graphs depict the probe stimuli.

the 1-s gap, $t(4) = 0.19$, and the 5-s gap, $t(4) = 1.37$. However, the 15-s gap shifted the peak time with a duration (9.04 s ± 2.66 s) that was different from both the stop, $t(4) = 9.43$, and the reset, $t(4) = 6.21$, rules. On the other hand, in the reversed group, the shift was not found to be different from that predicted by a reset rule for both the 5-s gap, $t(3) = 1.23$, and 15-s gap, $t(3) = 3.10$. However, the 1-s gap shifted the peak time with a duration (8.71 s ± 3.08 s) that was different from both the stop, $t(3) = 9.00$, and the reset, $t(3) = 6.51$, rules.

An analysis of the peak rates of response in the four types of probe trials failed to suggest significant effects for procedure, $F(1, 7) = 0.24$; type of probe trial, $F(3, 21) = 2.27$; or the interaction, $F(3, 21) = 0.30$. Interestingly, an analysis of the precision parameter in the PI trials suggests a significant increase in precision (decrease in Parameter b) in the reversed procedure relative to the standard procedure, $F(1, 7) = 7.29$.

In summary, the results suggest a difference in processing of the gap in the standard and reversed gap procedures. The introduction of a 1-s or 5-s break into the timed signal stopped timing in the standard procedure but partially or totally reset timing in the reversed procedure. On the other hand, an increase in the duration of the gap tended to promote the reset process irrespective of procedure.

Discussion

Interval timing in the standard and reversed procedures was directly evaluated in Experiment 4. No differences in peak time were found in the PI trials. In both procedures, the mean response rate peaked about the moment when rats were (sometimes) reinforced. Interestingly, precision of timing was significantly higher in the reversed group than in the standard group, possibly reflecting an increase in variability to respond that was due to an increase in general stimulation as well as an increase in the error with which memory was sampled because of a higher load of processing in the presence of the stimulus than in its absence. Nevertheless, we failed to find differences in response rate between the two groups. The introduction of a gap halfway into the timed interval was found to depend on the quality of the timed signal. When rats timed for the presence of the stimulus, a break in the timed signal stopped timing at the 1-s and 5-s gap durations but partially reset timing at the 15-s standard gap. On the other hand, when the rats timed for the absence of a stimulus, a reversed gap of equal duration totally or partially reset the entire timing process. For example, Panel B of Figure 8 shows that a 5-s standard gap stopped timing in the standard procedure (left panel), whereas a 5-s reversed gap totally reset timing in the reversed procedure (right panel). A statistical analysis shown in Panel D of Figure 8 supports a marked difference between the effects of standard and reversed gaps of equal durations, suggesting that other aspects of the signal other than the temporal aspect were responsible for the rule adopted by the animals. Results obtained in the standard group replicated those obtained by Church (1978; Meck et al., 1984; S. Roberts, 1981; S. Roberts & Church, 1978), whereas the results obtained in the reversed group replicated the results from Experiments 2 and 3.

In the framework of the internal clock model, the classical explanation of the results obtained in gap experiments is based on the attentional switch hypothesis, which assumes that while timing for the stimulus, a gap stops the timing process (Church, 1978; Gibbon et al., 1984). On the other hand, the decay hypothesis assumes that while timing for the stimulus, a gap allows for a memory-decay process that, depending on the duration of the gap, results in a partial or total loss of the current timed interval (i.e., in a stop or reset of the interval timing process (Cabeza de Vaca et al., 1994). Note that both hypotheses apply to general timed intervals, irrespective of their nature (i.e., they are assumed to be at work during both a standard and a reversed gap). These hypotheses fail to explain the differences between the effect of standard and reversed gaps of equal durations. Results support the notion that the stop and reset behavior does not depend solely on the duration of the interrupting event.

General Discussion

One of the underlying assumptions of most theories of interval timing (Church, 1978; Church & Broadbent, 1991; Gibbon, 1977; Gibbon et al., 1984; Killeen & Fetterman, 1988; Machado, 1997; Staddon & Higa, 1999; but see Block, 1990; Treisman, 1963; Zakay & Block, 1996) is that participants are able to readily abstract from the input stimulus the temporal dimension and to tune their behavior according to this cue, irrespective of the real timed event. In other words, participants are assumed to equally use any particular stimulus as a cue for the timing process as long as duration is the most predictive feature. In contrast, evidence supports the notion that both animal and human timing is highly sensitive to properties of the timed signal (see, e.g., Meck, 1991). Auditory stimuli are judged to be longer than visual stimuli by rats and humans (Goldstone & Lhamon, 1974; Penney, Allan, Meck, & Gibbon, 1998; Penney, Gibbon, & Meck, in press); bright lights are judged to be longer than dim lights by humans (Goldstone, Lhamon, & Sechzer, 1979), pigeons (Kraemer et al., 1997), and rats (Kraemer, Brown, & Randall, 1995); and filled intervals are judged to be longer than empty intervals by humans (Allan, 1979, 1992; Thomas & Weaver, 1975) and pigeons (Mantanus, 1981). Therefore, there might be reasons to believe that when the ongoing timed interval is interrupted by a break, participants do not choose a stop or a reset rule that is based only on the duration of the gap.

We examined rats' behavior when they timed for the absence of a visual or auditory stimulus in two paradigms: trace conditioning and gap procedure. Experiment 1 examined the timing process in a trace-conditioning, PI procedure. By manipulating the length of the stimulus, Experiment 1 demonstrated that irrespective of stimulus modality, in trace conditioning rats time the stimulus-offset–reinforcement interval by relying mainly on the offset of the stimulus as a time marker. We further evaluated memory for timing the lack of the stimulus by inserting the stimulus as a reversed gap halfway into the timed (stimulus-offset–reinforcement) interval, in a trace-PI procedure (Experiment 2) and in a reversed gap procedure (Experiment 3). Under the assumption that rats use the temporal dimension in the same way during a standard and a reversed gap, and with the parameters typically used (e.g., short 1-, 5-, and 15-s durations), we expected rats to stop timing at all reversed gap durations. Results from Experiments 2 and 3 failed to confirm our predictions. Both visual and auditory reversed gaps prompted rats to reset the interval timing process at gap durations as short as 1 s. Results from Experiments 2 and 3 suggest that the memory load, the content of the gap relative to the ITI, type of

control by the stimulus, and the confound between the gap and the ITI are not likely to be determinants of the reset of the interval timing process under current experimental procedures. The hypothesis that standard and reversed gaps (of equal durations) might have very different effects on timing in rats was directly evaluated in Experiment 4. The results suggest that at the same duration of the gap, timing seems to be stopped during a standard gap but entirely reset by a reversed gap. The results support the notion that in addition to temporal information, other aspects of the timed signal affect the interval timing process. This suggestion directly contradicts current interval timing models (Church, 1978; Church & Broadbent, 1991; Gibbon, 1977; Gibbon et al., 1984; Killeen & Fetterman, 1988; Machado, 1997; Staddon & Higa, 1999) that simply assume that participants extract and use only timing information irrespective of the specific stimulus. The results are also at odds with the memory-decay hypothesis but only partly at odds with the attentional switch hypothesis (see Lejeune, 1998).

The results question the notion that memory processes account for all aspects of timing in a gap procedure. In Experiment 4, the interval timing process was reset by the short 5-s reversed gap, although rats in the standard procedure seemed able to retain the currently timed interval over the same period of time. Therefore, the same memory-decay process cannot explain these results, although this has been previously advocated as both a timing mechanism and an explanation for the gap phenomena. Interval timing in simple procedures was successfully fit by the assumption of multiple-scale, memory-decay processes (Staddon & Higa, 1999; Staddon, Higa, & Chelaru, 1999). Also, the effects of a standard gap were successfully fit by the assumption of memory-decay processes during the gap (Cabeza de Vaca et al., 1994). However, it is unclear how the same memory processes might account for (a) timing in both a standard gap procedure and trace conditioning (Experiment 1), (b) the stop of timing by a standard gap (Experiment 4; see also Church, 1978; Meck et al., 1984; S. Roberts, 1981; S. Roberts & Church, 1978), (c) the reset of timing by a reversed gap in trace conditioning (Experiment 2), and (d) the reset of timing in a reversed gap procedure (Experiments 3 and 4). For example, a computational implementation of the memory-decay hypothesis (Hopson, 1999) resulted in an interval timing model that was not able to address trace conditioning. Indeed, the simple assumption of a passive memory-decay process in the absence of the stimulus determines interval timing to be affected in trace conditioning, a suggestion at odds with results from Experiment 1, which demonstrated good interval timing under such conditions. Also, it is unclear how a passive memory process would account simultaneously for the stop and reset rules at equal gap durations by a standard and a reversed gap (Experiment 4). Although memory decay is able to account for some aspects of the gap phenomena, supplemental assumptions might be needed to account for the results reported here. Therefore, the gap procedure might not only test the memory for time but also the type of control that the attributes of the gap have over the interval timing mechanism. Such attributes contributing to the reset process are the salience (discriminability) of the gap and the content and filling of the gap. Such attributes might be more easily accommodated for by an attentional mechanism.

An attentional mechanism allows for differences in the effects of standard and reversed gaps by assuming that the outcome of such experiments does not merely depend on the duration of the gap but also by the attention that the participant pays to the gap—attention that depends on attributes of the gap such as salience and content (Block, 1990; Treisman, 1963; Zakay & Block, 1996). Although such mechanisms are unclear at present, a number of possibilities exists. First, the attentional switch might be directly controlled by the salience of the gap. For example, it is possible that a 5-s standard gap is an event of low salience, with little influence on interval timing because it is similar to the ITI. On the other hand, a 5-s reversed gap may be perceived as a distinct salient event that tends to reset the entire interval timing process. Second, the salience and content of the gap might change some temporal attribute of the gap, which in turn might control the attentional gate (see Lejeune, 1998). For example, it is possible that the longer the perceived duration of the gap is, the more likely the gap is to reset the interval timing processes. Indeed, dim lights are judged to be shorter in duration by humans (Goldstone et al., 1979), pigeons (Kraemer et al., 1997), and rats (Kraemer, Brown, & Randall, 1995). In other words, in contrast to a standard gap of equal duration, a reversed gap might reset the interval timing process because it is judged to be more salient and, thus, longer in duration. Similarly, the content of the gap might be a factor in a gap procedure. Filled intervals have been shown to be judged to be longer than empty intervals by humans (Allan, 1979, 1992; Thomas & Weaver, 1975) and pigeons (Mantanus, 1981). Therefore, in contrast to a standard gap of equal duration, a reversed gap might reset the interval timing process because it is filled and, thus, judged to be longer in duration. In this scenario, the longer the gap is judged, the more the reset mechanism might be activated. This proposed attentional mechanism is an active mechanism that is very different from the passive subjective shortening model (Spetch & Wilkie, 1983). Spetch and Wilkie's model assumes a passive shortening of the currently timed event during a retention interval. In their model, the longer the absolute duration of the retention interval is, the shorter the remembered duration of the timed interval is. The mechanism discussed here proposes a subjective lengthening of the retention interval with its salience or filling—lengthening that in turn would directly or indirectly (e.g., through a mechanism such as proposed by Spetch & Wilkie, 1983, or Cabeza de Vaca et al., 1994) determine a reset of the interval timing mechanism. Because current interval timing theories concentrate mainly on the time dimension but less on other attributes of the timed interval, they are less able to address such mechanisms. On the other hand, associative models of interval timing might be able to address some of these issues.

A few models aim at describing both the temporal and associative properties of conditioning involving multiple stimuli (Buhusi & Schmajuk, 1999; Desmond & Moore, 1988; Grossberg & Merrill, 1992; Moore & Choi, 1998). By relying on the assumption that the onset of a stimulus generates a set of memory traces with different temporal properties (Grossberg & Schmajuk, 1989), both Grossberg and Merrill's and Buhusi and Schmajuk's models fail to differentiate between interval timing in delay and trace conditioning. Moreover, in contrast to results from Experiment 1, their results suggest that animals learn the onset-reinforcement interval and not the offset-reinforcement interval. Also, these models do not address gap manipulations. Nonetheless, a supplemental assumption in line with the memory-decay hypothesis (Cabeza de Vaca et al., 1994), which allowed traces to decay during the absence of the signal stimulus, allowed Grossberg and Schmajuk's

model to address the (standard) gap procedure at the expense of hindering its description of trace conditioning (Hopson, 1999). On the other hand, Desmond and Moore's (1988; Moore & Choi, 1998) model assumes that both the onset and the offset of a stimulus trigger spreading activations that act independently and in parallel. The model was shown to describe aspects of the real-time NMR response in rabbits (Kehoe, Horne, Macrae, & Horne, 1993; Kehoe & Napier, 1991). This model helps explain results from Experiment 1, in that both stimulus onset and stimulus offset would control behavior. For example, in line with results from Experiment 1, the longer the duration of the stimulus is, the more the response is predicted to be controlled solely by the offset of the stimulus. However, the model by Desmond and Moore (1988; Moore & Choi, 1998) does not differentiate between the lack of the stimulus in trace and the lack of stimulus in a gap. This model agrees with a reset rule at all gap durations under both standard and reversed conditions. Thus, the model explains some of the results obtained in the present experiments but does not address either the stop results obtained in the standard gap procedures (Church, 1978; Meck et al., 1984; S. Roberts, 1981; S. Roberts & Church, 1978) or the difference between the effect of a standard and of a reversed gap on interval timing at equal gap durations (Experiment 4). Thus, current associative models of interval timing fail to address (a) interval timing in both delay and trace conditioning and (b) interval timing in both standard and reversed gap procedures.

Results presented here complement previous findings regarding the flexibility of the interval timing mechanisms. Church (1978; S. Roberts & Church, 1978) established that timing in rats has many properties of a stopwatch: It can be stopped temporarily, it times signals in different modalities and with different durations, it uses the same rate for different intervals, and times up. More important, S. Roberts and Church showed that the timing mechanism is flexible, in that rats stop timing during an unexpected break, but over sessions rats can learn to run the clock during a break. Therefore, reinforcement contingency may influence the timing rules adopted by animals. Along the same line of reasoning, Matell and Meck (1999) and Staddon (1974) suggested that reinforcement resets the clock. Data presented here (Experiment 4) further suggest that the response rule can be changed by some aspects of the timed or interrupting interval, although reinforcement contingencies are similar in both the standard and reversed gap procedures. Results from Experiment 4 support the notion of a flexible interval timing mechanism in rats. Such a flexibility in using both the stop and reset rules in pigeons was suggested by Wilkie, Saksida, Samson, and Lee (1994). Wilkie et al. reinforced pigeons for pecking four keys at different locations, in a specific order, in four 15-min consecutive intervals. When they switched the key lights off for 15-min after the interval that corresponded to the first key elapsed, the result was more responses directed at the second key, which was in line with a stop rule. On the other hand, removing the pigeons from the test chambers for 15 min resulted in more pecks directed at the first key when pigeons were returned to the test apparatus, which was in line with a reset rule. Because temporal, spatial, and contextual cues might have contributed to the results, Wilkie et al.'s data suggest that parameters of the experimental procedure such as the ITI, the break, the spatial location of the cues, and the schedule of reinforcement may crucially influence the putative timing rule adopted by animals. The present data reinforce this suggestion by bringing evidence that properties of the timed stimulus or interrupting event also contribute to the rule adopted by rats in the gap paradigm.

In summary, this article evaluated the cues controlling, and the memory for, timing for the absence of a stimulus in rats. Rats were found to time for the absence of a stimulus (visual or auditory), and the interval timing mechanism was found to be controlled by the offset of the stimulus. The presentation of the stimulus itself when rats were timing for its absence reset their internal clock, at gap durations (1 s, 5 s, and 15 s) that stopped the internal clock when rats were timing for the presence of the stimulus (in the standard gap procedure). The memory load, the content of the gap relative to the ITI, the type of control by the stimulus, and the possible confound between the reversed gap and the ITI are not likely to be determinants of the reset of the interval timing process under current experimental procedures. Results also failed to endorse a passive memory-decay process and suggested that attentional mechanisms involving the salience or content of the reversed gap might contribute to the response rule adopted by rats in a gap procedure. The differences between the effects of standard and reversed gaps on interval timing in rats were perhaps accounted for by variations in the perceived duration of the gap due to the salience (discriminability) and content (filling) of the gap.

References

Allan, L. G. (1979). The perception of time. *Perception and Psychophysics, 26,* 340–354.

Allan, L. G. (1992). The internal clock revisited. In F. Macar, V. Pouthas, & W. Friedman (Eds.), *Time, action and cognition: Towards bridging the gap* (pp. 191–202). Dordrecht, Netherlands: Kluwer.

Block, R. A. (1990). Models of psychological time. In R. A. Block (Ed.), *Cognitive models of psychological time* (pp. 1–35). Hillsdale, NJ: Erlbaum.

Brown, B. L., Hemmes, N., & Cabeza de Vaca, S. (1997). Timing of the CS–US interval by pigeons in trace and delay conditioning. *Quarterly Journal of Experimental Psychology, 50B,* 40–53.

Brown, B. L., Hemmes, N., Cabeza de Vaca, S., & Pagano, C. (1993). Sign and goal tracking during delay and trace autoshaping in pigeons. *Animal Learning and Behavior, 21,* 360–368.

Buhusi, C. V., & Schmajuk, N. A. (1999). Timing in simple conditioning and occasion setting: A neural network approach. *Behavioural Processes, 45,* 33–57.

Cabeza de Vaca, S., Brown, B. L., & Hemmes, N. S. (1994). Internal clock and memory processes in animal timing. *Journal of Experimental Psychology: Animal Behavior Processes, 20,* 184–198.

Catania, A. C. (1970). Reinforcement schedules and psychophysical judgements: A study of some temporal properties of behavior. In W. N. Schoenfeld (Ed.), *The theory of reinforcement schedules* (pp. 1–42). New York: Appleton-Century-Crofts.

Church, R. M. (1978). The internal clock. In S. H. Hulse, H. Fowler, & W. K. Honig (Eds.), *Cognitive processes in animal behavior* (pp. 277–310). Hillsdale, NJ: Erlbaum.

Church, R. M. (1980). Short-term memory for time intervals. *Learning and Motivation, 11,* 208–219.

Church, R. M. (1984). Properties of an internal clock. In J. Gibbon & L. G. Allan (Eds.), *Annals of the New York Academy of Sciences: Volume 423. Timing and time perception* (pp. 566–582). New York: New York Academy of Sciences.

Church, R. M., & Broadbent, H. A. (1991). A connectionist model of timing. In M. L. Commons, S. Grossberg, & J. E. R. Staddon (Eds.), *Neural network models of conditioning and action* (pp. 225–240). Hillsdale, NJ: Erlbaum.

Cole, R. P., Barnet, R. C., & Miller, R. R. (1995). Temporal encoding in trace conditioning. *Animal Learning and Behavior, 23,* 144–153.

Desmond, J. E., & Moore, J. W. (1988). Adaptive timing in neural networks: The conditioned response. *Biological Cybernetics, 58,* 405–415.

Dietrich, A., Allen, J. D., & Bunnell, B. N. (1997). Is the hippocampus involved in temporal discrimination and the memory for short intervals? *International Journal of Neuroscience, 90,* 255–270.

Ellison, G. D. (1964). Differential salivary conditioning to traces. *Journal of Comparative and Physiological Psychology, 57,* 373–380.

Gibbon, J. (1977). Scalar expectancy and Weber's law in animal timing. *Psychological Review, 84,* 279–325.

Gibbon, J., Church, R. M., & Meck, W. H. (1984). Scalar timing in memory. In J. Gibbon & L. G. Allan (Eds.), *Annals of the New York Academy of Sciences: Volume 423. Timing and time perception* (pp. 52–77). New York: New York Academy of Sciences.

Goldstone, S., & Lhamon, W. T. (1974). Studies of auditory–visual differences in human time judgement: I. Sounds are judged longer than lights. *Perceptual and Motor Skills, 39,* 63–82.

Goldstone, S., Lhamon, W. T., & Sechzer, J. (1979). Light-intensity and judged duration. *Bulletin of the Psychonomic Society, 12,* 83–84.

Grossberg, S., & Merrill, J. W. L. (1992). A neural network model of adaptively timed reinforcement learning and hippocampal dynamics. *Cognitive Brain Research, 1,* 3–38.

Grossberg, S., & Schmajuk, N. A. (1989). Neural dynamics of adaptive timing and temporal discrimination during associative learning. *Neural Networks, 2,* 79–102.

Holland, P. C. (1980). CS–US interval as a determinant of the form of Pavlovian appetitive conditioned responses. *Journal of Experimental Psychology: Animal Behavior Processes, 6,* 155–174.

Hopson, J. W. (1999). Gap timing and the spectral timing model. *Behavioural Processes, 45,* 23–31.

Kamin, L. J. (1965). Temporal and intensity characteristics of the conditioned stimulus. In W. F. Prosaky (Ed.), *Classical conditioning: A symposium* (pp. 118–147). New York: Appleton-Century-Crofts.

Kehoe, E. J., Horne, P. S., Macrae, M., & Horne, A. (1993). Real-time processing of serial stimuli in classical conditioning of the rabbit's nicititating membrane response. *Journal of Experimental Psychology: Animal Behavior Processes, 19,* 265–283.

Kehoe, E. J., & Napier, R. M. (1991). Real-time factors in the rabbit's nicititating membrane response to pulsed and serial conditioned stimuli. *Animal Learning and Behavior, 19,* 195–206.

Killeen, P. R., & Fetterman, J. G. (1988). A behavioral theory of timing. *Psychological Review, 95,* 274–295.

Kraemer, P. J., Brown, R. W., & Randall, C. K. (1995). Signal intensity and duration estimation in rats. *Behavioural Processes, 344,* 265–268.

Kraemer, P. J., Randall, C. K., & Brown, R. W. (1997). The influence of stimulus attributes on duration matching-to-sample in pigeons. *Animal Learning and Behavior, 25,* 148–157.

Lejeune, H. (1998). Switching or gating? The attentional challenge in cognitive models of psychological time. *Behavioural Processes, 44,* 127–145.

Liu, S. S., & Moore, J. W. (1969). Auditory differential conditioning of the rabbit nictitating membrane response: IV. Training based on stimulus offset and the effect of intertrial tone. *Psychonomic Science, 15,* 128–129.

Lucas, G. A., Deich, J. D., & Wasserman, E. A. (1981). Trace autoshaping: Acquisition, maintenance, and path dependence at long trace intervals. *Journal of Experimental Analysis of Behavior, 36,* 61–74.

Machado, A. (1997). Learning the temporal dynamics of behavior. *Psychological Review, 104,* 241–265.

Mantanus, H. (1981). Empty and filled interval discrimination by pigeons. *Behavioural Analysis Letters, 1,* 217–224.

Marquardt, D. W. (1963). An algorithm for least squares estimation of parameters. *Journal of the Society of Industrial and Applied Mathematics, 11,* 431–441.

Matell, M. S., & Meck, W. H. (1999). Reinforcement-induced within-trial resetting of an internal clock. *Behavioural Processes, 45,* 159–171.

Mattson, M., & Moore, J. W. (1964). Intertrial responding and CS intensity in classical eyelid conditioning. *Journal of Experimental Psychology, 68,* 396–401.

Meck, W. H. (1991). Modality-specific circadian rhythmicities influence mechanisms of attention and memory for interval timing. *Learning and Motivation, 22,* 153–179.

Meck, W. H., Church, R. M., & Olton, D. S. (1984). Hippocampus, time, and memory. *Behavioral Neuroscience, 98,* 3–22.

MED Associates. (1999). WMPC software, version 1.15 [Computer software]. St. Albans, VT: Author.

Moore, J. W., & Choi, J. S. (1998). Conditioned stimuli are occasion setters. In N. A. A. Schmajuk & P. C. Holland (Eds.), *Occasion setting: Associative learning and cognition in animals* (pp. 279–318). Washington, DC: American Psychological Association.

Morgan, C. L. (1894). *Introduction to comparative psychology.* London: Scott.

Olton, D. S., Meck, W. H., & Church, R. M. (1987). Separation of hippocampal and amygdaloid involvement in temporal memory dysfunctions. *Brain Research, 404,* 180–188.

Pavlov, I. P. (1927). *Conditioned reflexes.* Oxford: Oxford University Press.

Penney, T. B., Allan, L. G., Meck, W. H., & Gibbon, J. (1998). Memory mixing in duration bisection. In D. A. Rosenbaum & C. E. Collyer (Eds.), *Timing of behavior: Neural, psychological, and computational perspectives* (pp. 165–193). Cambridge, MA: MIT Press.

Penney, T. B., Gibbon, J., & Meck, W. H. (in press). Differential effects of auditory and visual signals on clock speed and temporal memory. *Journal of Experimental Psychology: Human Perception and Performance.*

Roberts, S. (1981). Isolation of an internal clock. *Journal of Experimental Psychology: Animal Behavior Processes, 7,* 242–268.

Roberts, S., & Church, R. M. (1978). Control of an internal clock. *Journal of Experimental Psychology: Animal Behavior Processes, 4,* 318–337.

Roberts, W. A., Cheng, K., & Cohen, J. S. (1989). Timing light and tone signals in pigeons. *Journal of Experimental Psychology: Animal Behavior Processes, 15,* 23–35.

Santi, A., Ross, L., Coppa, R., & Coyle, J. (1999). Pigeons' memory for empty intervals marked by visual or auditory stimuli. *Animal Learning and Behavior, 27,* 190–205.

Schneiderman, N. (1966). Interstimulus interval function of the nictitating membrane response of the rabbit under delay versus trace conditioning. *Journal of Comparative and Physiological Psychology, 62,* 397–402.

Smith, M. C. (1968). CS–US interval and US intensity in classical conditioning of the rabbit's ninctitating membrane response. *Journal of Comparative and Physiological Psychology, 66,* 679–687.

Spetch, M. L., & Wilkie, D. M. (1983). Subjective shortening: a model of pigeons memory for event duration. *Journal of Experimental Psychology: Animal Behavior Processes, 9,* 14–30.

Staddon, J. E. R. (1974). Temporal control, attention, and memory. *Psychological Review, 81,* 375–391.

Staddon, J. E. R., & Higa, J. (1999). Time and memory: Towards a pacemaker-free theory of interval timing. *Journal of Experimental Analysis of Behavior, 71,* 215–251.

Staddon, J. E. R., Higa, J., & Chelaru, I. M. (1999). Time, trace, memory. *Journal of Experimental Analysis of Behavior, 71,* 293–301.

Thomas, E. A. C., & Weaver, W. B. (1975). Cognitive processing and time perception. *Perception and Psychophysics, 17,* 363–367.

Treisman, M. (1963). Temporal discriminations and the indifference inter

val: Implications for a model of the internal clock. *Psychological Monographs, 77,* 1–31.

Wilkie, D. M., Saksida, L. M., Samson, P., & Lee, A. (1994). Properties of time–place learning by pigeons, *Columbia livia. Behavioural Processes, 31,* 39–56.

Zakay, D., & Block, R. A. (1996). The role of attention in time estimation processes. In M. A. Pastor & J. Artieda (Eds.), *Time, internal clocks and movement* (pp. 143–164). Amsterdam: Elsevier.

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