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# Repeated Acquisition and Delayed Performance as a Baseline to Assess Drug Effects on Retention in Monkeys[1]

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THOMPSON, D. M., J. MASTROPAOLO, P. J. WINSAUER AND J. M. MOERSCHBAECHER. *Repeated acquisition and delayed performance as a baseline to assess drug effects on retention in monkeys.* PHARMACOL BIOCHEM BEHAV **25**(1) 201–207, 1986.—As an extension of previous research on repeated acquisition, a new baseline was developed to assess the effects of phencyclidine on retention in patas monkeys. Each session was divided into three phases: acquisition, delay, and performance. During acquisition, the subject acquired a four-response chain (which was different each session) by responding sequentially on three keys in the presence of four geometric forms. When the acquisition criterion (20 consecutive correct responses) was met, the keylights turned off and the delay (retention interval) began. After the delay, the keylights and a white light above the keys were turned on for 10 min (performance). The white light indicated that the response chain was the same as the chain acquired before the delay. Retention of the acquired response chain, as measured by percent "savings" in errors to criterion, decreased as the delay was increased from 5 to 180 min, and this "forgetting curve" tended to shift to the left with increasing doses of phencyclidine (administered IM 5 min before the performance phase). "Overlearning" the response chain before the 180-min delay increased retention and attenuated the disruptive effects of the lower dose of phencyclidine.

Repeated acquisition Response chains Delayed performance Retention Overlearning Phencyclidine Patas monkeys

IT is well established that phencyclidine can disrupt the acquisition of complex operant behavior in nonhuman primates [11–13, 16–20]. For example, in one such experiment [18], a repeated-acquisition procedure was used in which patas monkeys acquired a different four-response chain each session by responding sequentially on three keys in the presence of four geometric forms. The response chain was maintained by food presentation under a fixed-ratio (FR) schedule. Errors produced a brief timeout but did not reset the chain. With increasing doses of phencyclidine (0.03–0.17 mg/kg, IM), the overall response rate decreased, the percent errors increased, and there was less within-session error reduction (acquisition). The results also indicated that this disruptive effect on acquisition is not a characteristic of all hallucinogenic agents. MDA (3,4-methylenedioxyamphetamine), a hallucinogen that is self-administered in nonhuman primates, had little or no effect on acquisition even at doses that produced marked decreases in overall response rate.

As an extension of previous research on repeated acquisition, a new baseline was developed in the present study to assess the effects of phencyclidine on retention in monkeys. With this baseline (repeated acquisition and delayed performance), one can determine the extent to which monkeys can "remember," after varying delays, the particular four-response chain they acquired during a given session. The chain is considered to be acquired when the subject completes five sequences with 20 consecutive correct responses. A comparison of the number of errors made before this acquisition criterion is met with the number of errors made before the same criterion is met in the post-delay performance phase indicates the degree of retention of the acquired response chain. In other words, a "savings" measure is used

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to quantify the degree of retention, as in the classic studies of human learning and memory by Ebbinghaus [5].

## METHOD

### *Subjects*

Two adult female patas monkeys served. Both subjects had experimental histories involving the repeated acquisition of response chains. The subjects were maintained at about 90% of their free-feeding weights (5.1 and 5.9 kg) on a diet consisting of Noyes banana-flavored food pellets, Purina Monkey Chow, fruit, and vitamins. The pellets were earned during the experimental sessions, and the Monkey Chow, fruit, and vitamins were provided after the last session each day. Water was continuously available.

### *Apparatus*

The apparatus has been described in detail elsewhere [18]. Briefly, each subject was housed in a primate cage with a removable response panel, which was attached to the side of the cage during the experimental session. Three response keys (press plates) were centered and aligned horizontally on the panel. An in-line projector, mounted behind each key, could project colors and geometric forms onto the key. An additional in-line projector centered above the response keys could project a white light. A yellow pilot lamp (mounted on a switch) was located above a food pellet aperture to the right of the keys. The response panels were connected to solid-state scheduling and recording equipment located in an adjacent room.

### *Procedure*

*Baseline.* Each session was divided into three phases: acquisition, delay, and performance. During *acquisition*, the white light above the response keys was off, and one of four geometric forms (horizontal line, triangle, vertical line, circle) was projected onto a red background on all three keys. The subject's task was to learn a four-response chain by pressing the correct key in the presence of each form, e.g., horizontal line—Left correct; triangle—Right correct; vertical line—Center correct; circle—Right correct. When the chain was completed, the keylights turned off and the yellow lamp over the food pellet aperture was illuminated. A press on the yellow lamp then reset the chain. The four-response chain was maintained by food presentation under an FR 5 schedule; i.e., every fifth completion of the chain produced a food pellet (500 mg) when the yellow lamp was pressed. When the subject pressed an incorrect key (e.g., the left or right key when the center key was correct), the error was followed by a 5-sec timeout. During the timeout, the keys were dark and responses were ineffective. An error did not reset the chain; i.e., the stimuli on the keys after the timeout were the same as before the timeout. The chain was considered to be acquired when the subject completed five sequences with 20 consecutive correct responses. When this acquisition criterion was met, the keylights turned off, and the *delay* (retention interval) began. After the delay, the keylights and the white light above the keys were turned on. The white light indicated that the response chain was the same as the chain acquired before the delay; i.e., the white light was a discriminative stimulus for the *performance* phase or retention test. The performance of the same response chain was signaled by the white light because in the behavioral history of the monkeys, the onset of the keylights alone at the start of the session set the occasion for the acquisition of another chain. The session was terminated after 10 min in the performance phase.

To establish a steady state of repeated acquisition, the four-response chain was changed from session to session. The chains were carefully selected to be equivalent in several ways and there were restrictions on their ordering across sessions [14]. An example of a typical set of six chains is as follows: Left-Right-Center-Right (LRCR), CLRL, LRLC, RCRL, CLCR, RCLC; the order of the associated forms was always the same: horizontal line, triangle, vertical line, circle (reinforcement). For each subject there were two sessions (a.m. and p.m.) each day (Monday through Friday).

The degree of retention of the acquired response chain was quantified using a "savings" measure (cf. [5]). Percent savings was calculated as follows: For a given response chain, the number of errors made before the acquisition criterion was met was compared to the number of errors made before the same criterion was met in the performance phase. Specifically, this comparison was calculated by subtracting the errors to criterion (ETC) in performance from the ETC in acquisition, and then expressing this difference as a percentage of the ETC in acquisition. For example, if the subject made 20 errors before the acquisition criterion was met, but made only 5 errors before the same criterion was met in performance, the percent savings would be 75; [(20–5/20) × 100]. If retention was perfect (i.e., ETC in performance=0), the percent savings would equal 100, whereas if there was no retention at all (i.e., ETC in performance=ETC in acquisition), the percent savings would equal 0. To permit comparisons with previous research using repeated-acquisition baselines, the data for each session were also analyzed in terms of the overall response rate (total responses/min, excluding timeouts) and the overall accuracy or percent errors [(errors/total responses) × 100] in acquisition and in performance. In addition to these measures based on session totals, within-session changes in responding were monitored by a cumulative recorder.

*Drug testing.* Before the drug testing began, the baseline of repeated acquisition and delayed performance was stabilized. The baseline was considered stable when the percent savings after a 5-min delay showed no systematic change from session to session. (The subjects were initially exposed to delays of 10, 30, 60, 120 and 180 sec, in that order, but the percent savings at these delays was generally about the same as that at the 5-min delay.) After baseline stabilization (approximately 10 sessions at the 5-min delay), 0.1 mg/kg of phencyclidine hydrochloride was tested. This dose was selected because previous research had shown that 0.1 mg/kg was the lowest effective dose of phencyclidine in monkeys responding in a repeated-acquisition task [18]. The drug was dissolved in saline and injected IM (*gluteus m.*) 5 min before the performance phase began. A higher dose of phencyclidine, 0.18 mg/kg, was then tested at the 5-min delay. After two determinations for both doses, the delay between acquisition and performance was increased to 30 min. The 30-min delay alternated with the 5-min delay each day; i.e., on one day the subjects were exposed to the 30-min delay in the a.m. session and to the 5-min delay in the p.m. session, whereas on the next day, the order of the two delays was reversed. Following baseline stabilization at the 30-min delay (4–6 sessions), the same testing procedure was used to assess the effects of 0.1 and 0.18 mg/kg of phencyclidine (administered IM 5 min before performance). To provide a

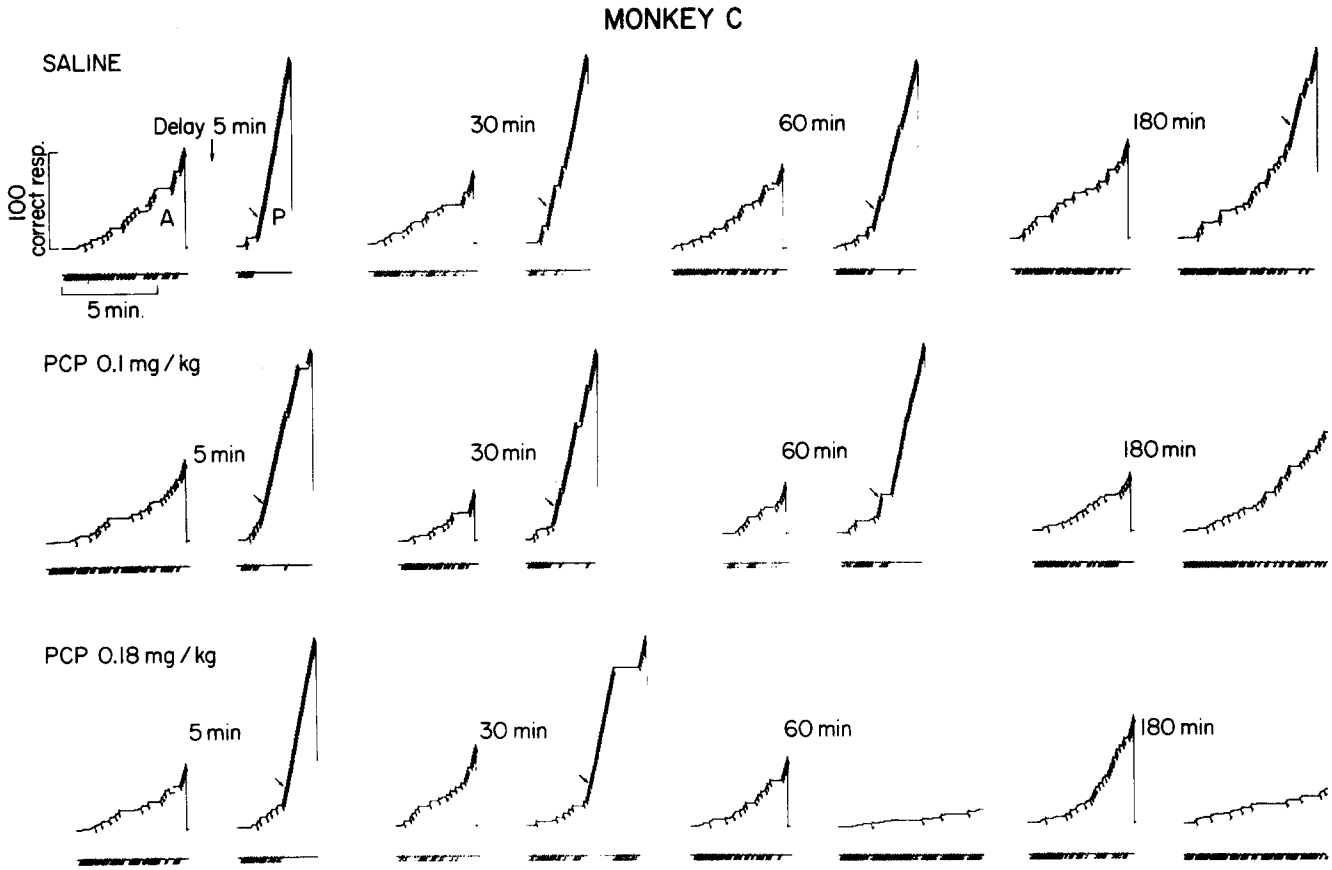


FIG. 1. Within-session effects of phencyclidine (PCP) in Monkey C. The top row of cumulative records shows data from a control session at each of four delays (5, 30, 60 and 180 min). The response pen stepped upward with each correct response and was deflected downward each time the four-response chain was completed. Errors are indicated by the event pen (below each record), which was held down during each timeout. For each session, the first record is the acquisition (A) phase and the second record is the first excursion of the response pen during the 10-min performance (P) phase. The response pen reset when the acquisition criterion (20 consecutive correct responses) was met and the small diagonal arrow indicates where the same criterion was met in performance. In each control session, saline was injected (IM) 5 min before the performance phase began. The middle row of records shows the corresponding data from sessions in which 0.1 mg/kg of phencyclidine was injected. At the 180-min delay, the criterion was never met during performance (only the first 7.5 min of performance is shown). The bottom row of records shows the effects of 0.18 mg/kg of phencyclidine at each delay. At the 60-min and 180-min delays, the criterion was never met during performance (only the first 7.5 min of performance is shown).

direct comparison, the effects of these doses were also redetermined (on different days) at the 5-min delay. The delay between acquisition and performance was then increased from 30 min to 60 min (alternating with the 5-min delay each day), and after baseline stabilization (4–6 sessions), both doses of phencyclidine were tested again using the same procedure. Finally, the delay was increased to 180 min. At this long delay, however, only one session was conducted each day. For comparison, once a week the subjects were exposed to the 5-min delay. After baseline stabilization at the 180-min delay (4–6 sessions), both doses of phencyclidine were again tested using the same injection procedure. Throughout testing, drug sessions were generally conducted on Tuesdays and Fridays, with control sessions (saline, IM 5 min before performance) occurring on Thursdays, and baseline sessions (no injections) on Mondays and Wednesdays. The volume of each injection was 0.05 ml/kg body weight.

Since Monkey C showed no retention, as measured by percent savings, at the 180-min delay under control conditions, a *probe* was conducted with this subject following the drug tests described above. In this probe, the acquisition phase continued for 5 min after the acquisition criterion (20 consecutive correct responses) had been met, thereby permitting the subject to "overlearn" the response chain before the 180-min delay began. Phencyclidine (0.1 or 0.18 mg/kg) or saline was then administered IM 5 min before the performance phase. To permit baseline recovery after each probe, the saline and drug sessions (two determinations for each dose) were conducted on Thursdays and Tuesdays, respectively.

## RESULTS

The top row of cumulative records in Fig. 1 shows data from a control (saline) session at each of four delays in Monkey C. For each session, the first record is the acquisition (A) phase and the second record is the first excursion of the response pen during the 10-min performance (P) phase. As the delay was increased from 5 min to 180 min, the number of

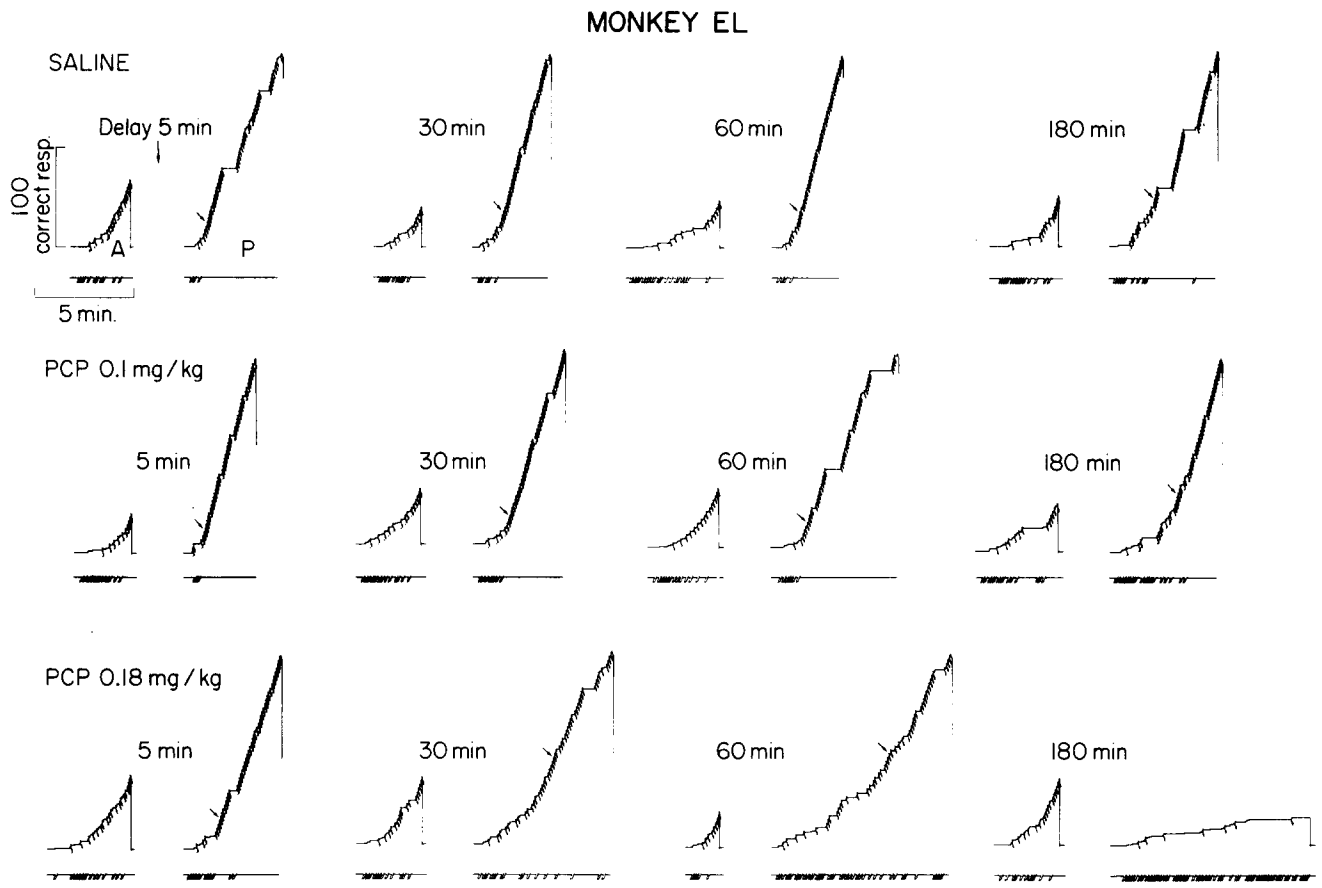


FIG. 2. Within-session effects of 0.1 and 0.18 mg/kg of phencyclidine (PCP) in Monkey EL. At the 0.18 mg/kg dose, when the delay was 180 min the criterion was never met during the 10-min performance phase. The recording details are the same as in Fig. 1.

errors in performance increased. When the delay was 180 min, Monkey C made approximately the same number of errors in performance before the criterion was met (39) as in acquisition (35), thereby indicating that there was no retention of the acquired response chain at this delay. The middle row of records shows the results obtained when 0.1 mg/kg of phencyclidine was administered 5 min before the performance phase at each delay. As can be seen, the effects of this dose of phencyclidine depended on the delay value; i.e., there were greater disruptive effects at the longer delays. At the 60-min delay, performance was approximately the same as acquisition in the number of errors to criterion (i.e., no retention occurred), whereas at the 180-min delay, the criterion was never met during the 10-min performance phase (only the first 7.5 min of performance is shown). The bottom row of records shows the effects of a higher dose of phencyclidine, 0.18 mg/kg, at each delay. At the 5-min delay, there was a small error-increasing effect but some retention occurred, whereas at the longer delays, there was no retention at all in terms of errors to criterion. In fact, at the 60-min and 180-min delays, the criterion was never met during the 10-min performance phase (only the first 7.5 min of performance is shown).

The cumulative records in Fig. 2 show the corresponding data for Monkey EL. Under control conditions, Monkey EL generally acquired the response chains more rapidly (i.e., made fewer errors) than Monkey C. Monkey EL also showed some retention at the 180-min delay, unlike Monkey C. Despite these individual differences in the control data, the dose-dependent effects of phencyclidine and the modulation of these effects by the delay value in Monkey C (Fig. 1) were generally replicated in Monkey EL. In general, at a given delay, the disruptive effects of phencyclidine on retention were greater as the dose was increased, and at a given dose, the disruptive effects were greater as the delay was increased.

Figure 3 shows the effects of phencyclidine on overall response rate in performance, percent errors in performance, and percent savings for both subjects. As the delay increased from 5 min to 180 min under control (saline) conditions, there was little or no change in overall response rate or percent errors in performance, but percent savings (i.e., retention) decreased markedly. Phencyclidine decreased overall response rate, increased percent errors, and decreased percent savings with increasing doses, but the magnitude of these effects depended on the delay value. In general, the longer the delay, the greater the effects.

Table 1 shows the effects of the "overlearning" probe; the ranges for two determinations are shown. When Monkey C "overlearned" the response chain before the 180-min delay, the percent savings was considerable after both saline and 0.1 mg/kg of phencyclidine. In marked contrast, without "overlearning" before the 180-min delay, the percent savings was zero after saline administration, and the criterion

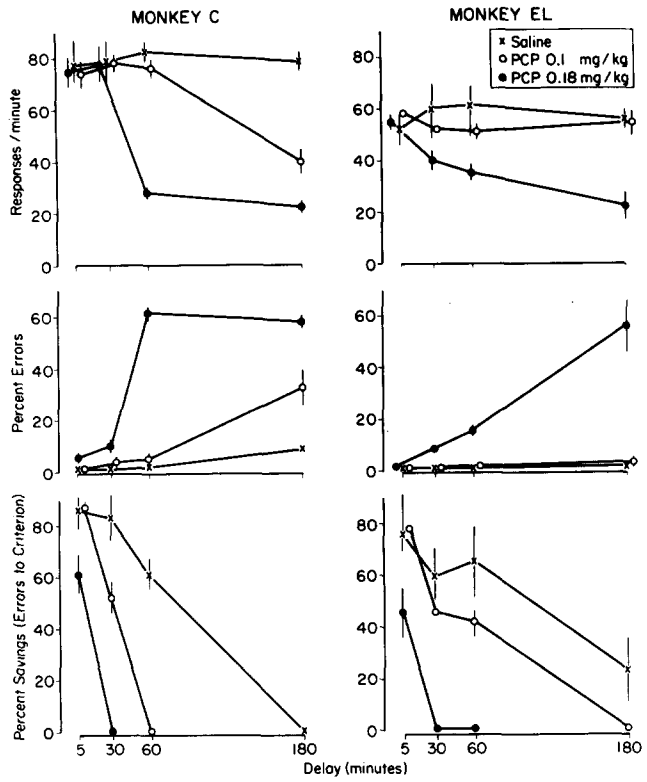


FIG. 3. Effects of phencyclidine (PCP) on overall response rate in performance, percent errors in performance, and percent savings for both subjects. The points and vertical lines indicate the mean and range for 2–3 determinations; the points without vertical lines indicate an instance in which the range is encompassed by the point. Points for percent savings have been omitted in cases where the criterion was not met in performance; points for percent savings at zero indicate that the errors to criterion in performance were equal to (or greater than) the errors to criterion in acquisition.

was never met during the 10-min performance phase after this dose of phencyclidine was administered because of the increase in percent errors. These results indicate that "overlearning" modulated both retention, as measured by percent savings, and the disruptive effects of phencyclidine. It should be noted, however, that such modulation was dose-dependent. The higher dose of phencyclidine (0.18 mg/kg) produced large error-increasing effects in performance both with and without "overlearning."

## DISCUSSION

Using a baseline of repeated acquisition and delayed performance in patas monkeys, the present research first determined the extent to which the monkeys could "remember," after varying delays, the particular four-response chain they acquired during a given session. As the delay was increased from 5 min to 180 min under control (saline) conditions, both monkeys showed a marked decrease in retention, as measured by percent savings. An interesting aspect of this methodology is the fact that considerable retention occurred at delays that are much longer than those typically studied with more conventional operant techniques, such as delayed matching to sample. When delayed matching-to-sample procedures are used with monkeys, the delays studied are usually less than 3 min (see review in [3]). The present research showed that repeated acquisition and delayed performance in monkeys is a technique that is appropriate for the study of retention at delays up to several hours.

TABLE 1

MODULATION OF DRUG EFFECTS BY "OVERLEARNING" (MONKEY C, DELAY = 180 MIN)

| | No "Overlearning" | | "Overlearning" | |
|---|---|---|---|---|
| | % Savings | % Errors (Perf.) | % Savings | % Errors (Perf.) |
| Saline | 0 | 8.8–10.0 | 67.1–81.3 | 1.7– 4.8 |
| PCP 0.1 mg/kg | —* | 25.6–40.1 | 73.1–84.6 | 1.1– 8.3 |
| PCP 0.18 mg/kg | —* | 56.2–60.0 | —* | 53.9–66.0 |

*Criterion not met in performance.

The apparent difference between the two techniques in the length of retention may be related to how long the subject is exposed to the stimuli before the delay. In delayed matching, the presentation of the sample is usually brief (a few sec or less), whereas in repeated acquisition the monkeys were exposed to the stimuli in the four-response chain for several min until the acquisition criterion was met. Previous research with rhesus monkeys has shown that accuracy in delayed matching can be increased by increasing the sample duration [4]. It is also possible that percent savings in the repeated-acquisition technique is a more sensitive measure of retention than percent correct responses in delayed matching to sample.

The present finding that retention of an acquired response chain decreased over a period of 5 to 180 min in patas monkeys with a history of repeated acquisition is similar to results obtained in rhesus monkeys with a "learning-set" history [1]. In such monkeys, retention loss for each discrimination was considerable after a delay of several minutes and was essentially complete after one hour. After a delay, a subject with a "learning-set" history seems to respond as if there were a new discrimination problem [6]. In marked contrast, in rhesus monkeys without such a history, it has been found that there is almost complete retention of discriminations at intervals up to 7 months after acquisition [1]. That both monkeys in the present study showed only moderate retention loss after one hour may be related to the fact that there was a separate discriminative stimulus (white light) for the performance phase. The performance phase (where the response chain was the same as in the acquisition phase) was signaled by the white light because in the repeated-acquisition history of the monkeys, the onset of the keylights alone at the start of the session set the occasion for the acquisition of another chain. In a sense, the white light is analogous to instructions about "what to remember" (e.g., which list of nonsense syllables) in human studies [6]. There is also an interesting parallel between the time course of retention loss in the present experiment and the "forgetting curve" obtained by Ebbinghaus in his classic studies of human learning and memory [5]. Ebbinghaus's rapid forgetting of nonsense syllables (e.g., only approximately 45% savings after a 1-hr delay) was probably due to his previous learning of other lists (i.e., "proactive interference") [6,9].

When phencyclidine was administered before the performance phase, the "forgetting curve" (i.e., percent savings as a function of the retention interval or delay) tended to shift to the left. In general, at a given dose, the disruptive effects of phencyclidine on retention were greater as the delay was increased, and at a given delay, the disruptive effects were greater as the dose was increased. The modulation of phencyclidine's effects by the delay value is not related to the time course of the drug since phencyclidine was administered at the same time (5 min before the performance phase) at each delay. Rather, the modulation would seem to indicate that repeated acquisition and delayed performance of response chains is a sensitive baseline to assess drug effects on retention in monkeys.

There appears to be only one previous study in the animal literature that has examined the effects of phencyclidine at different delay values in a retention task. McMillan [10] studied phencyclidine in pigeons responding in a delayed matching-to-sample task, with delays ranging from 1 to 8 sec. Although phencyclidine decreased matching accuracy, there was no clear evidence that retention was disrupted because the effects on accuracy did not seem to depend on the delay duration. As Heise and Milar [7] have emphasized, drug effects on retention are defined by increases in the effects with increasing delays. That phencyclidine did not disrupt retention in McMillan's experiment may be related to the fact that under control conditions, matching accuracy did not show a consistent tendency to decrease as the delay increased. In the present study, retention did decrease with increasing delays under control conditions, and phencyclidine was found to disrupt retention. This effect on retention in patas monkeys is consistent with the clinical finding that phencyclidine can produce amnesia in humans [2].

According to Heise and Milar [7], retention in operant experiments is measured by the extent to which stimulus control is maintained over delays of various durations. Based on this point of view, we conducted a probe in which the acquisition phase continued for 5 min after the acquisition criterion (20 consecutive correct responses) had been met, thereby permitting the subject (Monkey C) to "overlearn" the response chain. If the longer acquisition period establishes stronger stimulus control over the sequential responding, then one would expect an attenuation of a disruptive drug effect on retention [15]. Consistent with this interpretation, "overlearning" the response chain before the 180-min delay increased retention and attenuated the disruptive effects of the lower dose of phencyclidine (0.1 mg/kg). That the disruptive effects of the higher dose (0.18 mg/kg) were not attenuated, however, indicates that the modulation of phencyclidine's effects by "overlearning" was dose-dependent.

To permit comparisons with previous research using repeated-acquisition baselines, the data in the 10-min performance phase were also analyzed in terms of overall response rate and overall accuracy (percent errors). In both the present study, where phencyclidine was administered before the performance phase, and in previous research (e.g. [18]), where the drug was administered before acquisition, the overall response rate decreased and percent errors increased with increasing doses. In Monkey EL, who served in both experiments, the lowest dose of phencyclidine that disrupted acquisition was 0.1 mg/kg, whereas this dose had little or no effect on rate or percent errors in the performance phase (Fig. 3). Although this comparison indicates that acquisition was more sensitive than performance in detecting the rate-decreasing and error-increasing effects of phencyclidine, it should be noted that the 0.1 mg/kg dose did decrease retention (percent savings), except at the 5-min delay. At 0.17 mg/kg, phencyclidine produced large rate-decreasing and error-increasing effects in acquisition, and these large effects were also seen in Monkey EL at approximately the same dose (0.18 mg/kg) in the performance phase, but only at the longest delay (180 min). Moreover, the within-session effects of 0.18 mg/kg of phencyclidine at the 180-min delay (Fig. 2) are quite similar to the disruptive effects on acquisition [18], thereby suggesting that Monkey EL was responding during the performance phase under these conditions as if there were a new response chain to be acquired.

In previous research (e.g. [18]), repeated acquisition has been compared to a "performance" condition in which the four-response chain was the same from session to session. Procedurally, this type of performance is different from the performance phase in the present study, where the response chain was the same as in the acquisition phase. This distinction may be characterized as between-session vs. within-session performance. In general, between-session performance seems to be less sensitive to the error-increasing effects of phencyclidine than within-session performance, especially at the longer delays. One would expect this difference in sensitivity to drug effects because between-session performance is essentially an extension of the "overlearning" probe in establishing strong stimulus control.

In summary, the methodology used in the present experiment is a novel approach to the assessment of drug effects on retention. In contrast to delayed matching to sample, the baseline of repeated acquisition and delayed performance is appropriate for the study of retention at delays up to several hours. The new baseline was shown to be sensitive to the disruptive effects of phencyclidine on retention in patas monkeys and it has the potential to detect drug-induced "enhancement" of retention at the longer delays. In addition, the present baseline may be useful for investigating whether a drug affects "storage" or "retrieval" processes [8] by varying the time of drug administration during a long retention interval.

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