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# OPERANT CONTROL OF RESPONSE LATENCY IN MONKEYS: EVIDENCE FOR A CENTRAL EXPLANATION[1]

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Two monkeys (*M. mulatta*) were trained to press a telegraph key after onset of a tone and release it quickly in response to a subsequent light or click stimulus occurring after a variable interval. After training first with a fixed time limit on response latency for key release and then with a continuously adjusting limit, reaction time to click was 160 msec and to light, about 200 msec. Temporal contingencies or "payoff bands" were then introduced which reinforced only responses with latencies which fell between two limits 50 msec apart. Feedback was given as to whether each latency was too slow, within the band, or too fast. A trained monkey could precisely center its latency distribution on any 50 msec-wide payoff band located from 200 to 600 msec after the stimulus, with from 60 to 80% of its responses achieving reinforcement. Distribution statistics were comparable to those of trained human subjects. Because such precise timing might be accomplished by a peripheral adjustment, such as changing the manner of holding the key, latency of electromyographic activation was measured in participating arm muscles in one monkey. Electromyographic activation preceded key release by a constant interval, regardless of response latency, indicating a more central mechanism for timing of brief intervals.

Reaction time, especially in animal research, has often been investigated as if it were a function of the stimulus parameters alone. However, there is considerable evidence from work with humans and some from animal studies that reaction time or response latency is under the control of reinforcement contingencies.

Stebbins and Lanson (1961, 1962) developed a technique for measuring reaction time in rats using a trial paradigm similar to that used in human experiments on simple reaction time. It consisted of a repeated sequence of intertrial interval (ITI), warning stimulus ($S^W$), response to $S^W$ (key-press), foreperiod (fixed or variable), reaction stimulus ($S^D$), response to $S^D$ (key release), reinforcement. In order to insure that the animal was attending to $S^W$ and was ready for $S^D$, an overt response to $S^W$, not generally used in human experiments, was required to initiate the foreperiod. It was found necessary to vary the foreperiod to insure that the animals were reacting to $S^D$ and not timing the key-release response from the key-press to $S^W$. Such a variable $S^W$ to $S^D$ foreperiod is usually used in human experiments on simple reaction time.

Using a similar method on monkeys, Stebbins and Miller (1964) found that reaction time for a strong signal light or tone stimulus was 300 to 400 msec. Compared to human simple reaction times of 140 msec for moderately loud tones and 180 to 200 msec for light onset (Woodworth and Schlosberg, 1954, p. 16), these monkey reaction times were very long. Stebbins and Miller showed that not reinforcing responses with latencies longer than an arbitrary limit of 0.4 to 0.5 sec reduced these reaction times by about 50 msec. Miller, Glickstein, and Stebbins (1966) reported that further gradual day-to-day reduction of the limit to 250 msec could decrease median reaction time to light to 170 msec for one monkey and to 210 msec for another. Attempts to reduce the limit still further resulted in erratic responding and an increased number of anticipatory responses. These results suggested that

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reaction time could be controlled by temporal reinforcement contingencies, at least in the direction of shortening it.

With human subjects, Snodgrass, Luce, and Galanter (1967) differentially reinforced auditory reaction times which fell within specified temporal limits or "payoff bands". For example, within one session a subject might be paid money only for response latencies which fell between 144 and 164 msec and fined for latencies outside this band. Trial-by-trial feedback was given as to whether each response latency was too short, too long, or in the band. Fixed foreperiods of 2 sec were used, but if the subject attempted to time from $S^W$ and ignore $S^D$, the variance in response latencies would be much too great to permit very many successes in hitting a 20 msec-wide band. Six 20 msec-wide bands were used, located between 85 and 205 msec following the onset of $S^D$. After three practice sessions, which included all six bands in blocks of about 80 trials each, the subjects were able to produce for 500 test trials on any single band a mean reaction time which matched the center of the band. Thus, the human subjects were able to locate sharply peaked reaction time distributions anywhere between 105 and 205 msec, depending on reinforcement contingencies. They were also able to produce mean reaction times of less than 105 msec; however, the variabilities sharply increased, indicating that some of the responses were being timed from $S^W$ and not $S^D$.

Snodgrass *et al.* also studied time estimation of longer intervals from 600 to 5000 msec, using 360 msec-wide payoff bands. The actual response latencies were displayed to the subject as feedback. They found that the subjects could match their mean response latencies to the center of each band with standard deviations of about 10% of the mean. These longer time intervals are of the order of interresponse times (IRT) of animals on differential-reinforcement-of-low-rate (DRL) schedules.

Experiments with animals contain many examples of timing of long intervals, of a few seconds or more. Blough (1963), using pigeons, found that the adjustment of overall rate in accordance with a DRL schedule was accomplished by decreasing the frequency of brief IRTs (300 to 400 msec) while increasing the duration of long IRTs to exceed the pause required by the schedule. Migler (1964), working with rats, reinforced brief delays in switching between two keys in the presence of one stimulus and only long delays in the presence of another, establishing two separate timing distributions within the same experiment. Stimuli intermediate to the two training stimuli were responded to not by a shift of the IRT distribution to a new location but by different proportions of IRTs from the two training distributions. Reynolds (1966) found that pigeons could discriminate among IRTs of different durations, even though they might not be emitting many IRTs reinforced by the schedule. Thus, there is ample evidence that animals can both emit and discriminate time intervals of several seconds, but there is no work on estimation of intervals of less than 1 sec. Laties, Weiss, Clark, and Reynolds (1965) proposed overt behavior chains as one basis for timing. They found that a rat under the control of a DRL schedule displayed behavior during the pauses not seen under other schedules.

In the present experiment, precise time estimation in monkeys was investigated in the region between fast reaction time, about 200 msec for light and 160 msec for click, and 600 msec, using a simple reaction time paradigm with 50 msec-wide payoff bands.

Such precise, rapid timing probably could not be accomplished by overt behavior chains of the type described by Laties *et al.*, but it is possible that the animal could delay its response accurately by making a peripheral adjustment, such as grasping the key more firmly so that it took longer to release it, or otherwise changing hand position, as suggested by Snodgrass *et al.* If the timing were done by such a peripheral adjustment, the difference in time between the onset of the electromyographic (EMG) activity in the arm muscles participating in the key-release response and the time of occurrence of the key release itself would increase with increased release latency. On the other hand, if the timing were accomplished more centrally, the difference in time between the onset of the EMG and the key release would remain constant. That is, EMG onset would be delayed by the same amount of time as the reaction time was increased. To investigate these possibilities, EMG recordings were made on one monkey from biceps and triceps in the upper arm, muscle groups which were important in snatching the hand back during rapid key release.

## METHOD

### *Subjects*

Two male monkeys (*Macaca mulatta*), one adolescent (W—3 kgm) and one adult (C—7 kgm), were maintained at 80 to 90% of free-feeding weight. They were food deprived for 22 hr before each session and were given supplemental feeding 30 min after the end of each session. The monkeys were kept in home cages and placed in primate restraining chairs only for the daily experimental sessions (Barrow, Luschei, Nathan, and Saslow, 1966). Sessions were from 150 to 600 trials long or about 1 to 1.5 hr.

### *Apparatus, Training, and Trial Events*

By a shaping procedure similar to that of Stebbins (1966) and Stebbins and Miller (1964), the subjects were trained in a darkened audiometric testing chamber (IAC, model 400A) to await the onset of a clearly audible 1000 Hz tone ($S^W$), then to press a telegraph key, and hold it down through a variable 0.5- to 2.5-sec foreperiod, releasing it after the occurrence of $S^D$, which could be either a light flash or, in some experiments, a loud click simultaneous with the onset of the light flash. The light flash was 100 msec long and was produced by turning on an NE-40 neon bulb mounted behind a 1-cm hole located 6 in. from the monkey's right eye when facing forward. The 1-msec condenser-discharge click was delivered through a speaker mounted above and in front of the animal. Simultaneous click and light were used instead of click alone because the animal tended to change orientation during long sessions without the light flashes.

Key release in the presence of $S^D$ was reinforced by a 190 mg sweetened banana- or strawberry-flavored pellet. The intertrial interval (ITI) was set at between 6 and 10 sec. Releases during a foreperiod (anticipations) and responses during an ITI started a new ITI. Trials, stimulus events, and reinforcement contingencies were programmed automatically by a system of DigiBit logic located outside the experimental chamber. Tests with both humans and monkeys indicated no detectable switching sounds. Continuous low-level white noise masked extraneous sounds from the room.

The telegraph key was positioned on the right side at waist height. The monkeys were observed to press consistently with the right hand and to release the key with a massive jerk of the whole arm. Response latencies for key release to $S^D$ onset were measured in milliseconds by a Hewlett-Packard electronic counter and printed out by a Hewlett-Packard digital recorder. Latencies were then transferred to IBM punched cards for computer analyses.

### *Titrated Upper Limit*[3]

Once the animal was reliably waiting for $S^W$ before pressing the bar and then releasing the bar after $S^D$, a 1-sec upper limit on release latencies was introduced (Stebbins and Miller, 1964). A key release with latency longer than this limit terminated the trial without reinforcement. After long response latencies had been eliminated by this procedure, the upper limit was automatically titrated so that whenever the animal's release preceded the limit and was reinforced, the upper limit was reduced by 10 msec for the next trial, but when the release followed the limit and was not reinforced, the limit was increased by 20 msec. After two to three weeks on this schedule, the newly trained animal gave a stable distribution of brief latencies. Under a titrated limit procedure, a trained animal would quickly establish and maintain such a distribution, which could be shifted easily in response to changes in stimulus parameters. Performance of a trained animal under this titrated limit procedure was taken as its minimum reaction time for each set of stimulus conditions. Variable foreperiods, randomly selected from a uniform 0.5- to 2.5-sec distribution, were always used with the titrated limit procedure. A fixed lower limit of 100 msec for light $S^D$ and 80 msec for click (with light) $S^D$ was used in the present experiments to help reduce anticipations. Releases with latencies less than this lower limit were not reinforced.

### *Payoff Bands*

On a payoff band, the key release was reinforced only when its latency fell between two fixed time limits, for example, only if the reaction time were longer than 300 msec (lower

[3]Developed in collaboration with E. Luschei, Dept. of Physiology and Biophysics, University of Washington.

limit) and less than 350 msec (upper limit). Responses outside the band terminated the trial without reinforcement. Latencies shorter than the lower limit were signaled by a blink of the houselight; those falling within the band, by feeder noises and delivery of a pellet; those longer than the upper limit, by a low-intensity, high-pitched tone. Thus the animal was given feedback information as to whether it was too fast, on target, or too slow. Most payoff-band experiments used fixed foreperiods of 1 sec; however, a few sessions were run with variable foreperiods.

*Procedure*

Both animals were highly trained, with about one year of exposure to the titrated upper limit in a variety of experiments. Monkey W made only one or two anticipations per session, and almost no very long latencies. Monkey C occasionally made deviant response (anticipations and very long latencies) on as many as 10% of the trials. For most of the sessions reported here, Monkey C's deviant responses were less than 2% of the total.

Using light $S^D$, both animals performed with the titrated limit for several days at the beginning of this experiment. Then they were shifted through a series of 50 msec-wide payoff bands out to about 0.5 sec and then back to faster bands. Monkey W was returned to the titrated limit using click (with light) as $S^D$ and again shifted from fast to slower payoff bands.

The precise shaping procedures used to establish payoff-band performance differed for the two animals. Monkey W was generally shifted to an overlapping or adjacent band so that only a session or two was necessary to shape the response latency distribution to the new location. Monkey C was usually shifted to a non-overlapping band and spent a week or so on each new band before the response latency distribution centered on the new band. Shifting Monkey C's latency distribution was facilitated by first widening the new band to 100 msec and then decreasing it to the standard 50 msec width over one to three sessions. Details of sequencing of bands are given with the appropriate graphs in the Results section below.

*Recording of EMGs*

While Monkey W was treated with Sernylan (phencyclidine hydrochloride) and additional local anesthetic, modified wound clips with attached female plugs were clipped into the skin overlying biceps on the front of the right arm and triceps on the back. Such electrodes have a life of two to four weeks.

Electromyographic activity (EMG) in right arm biceps or triceps was recorded either for entire sessions or for blocks of 50 to 100 trials taken from the latter halves of some of Monkey W's sessions. When EMGs were being measured, the animal was further restrained by breast plates which forced it to sit facing forward and permitted use of the right arm only. Such additional restraint did not affect response latencies.

EMGs were monitored on an oscilloscope, using two Tektronix 122 low-level preamplifiers. For later data analysis, the amplified EMG voltage was rectified by a bridge circuit, converted to FM and recorded trial-by-trial on magnetic tape, with the addition of stimulus and response markers. The taped activity could be converted back to AM and processed by different devices to obtain estimates of EMG burst latency and duration.

*Determination of EMG Latency and Duration*

For trial-by-trial latency measures, the $S^D$ onset marker started the counter which was then stopped when the EMG burst activated a Schmitt trigger, the resulting latency for EMG onset being printed on paper tape. A number of different Schmitt trigger criterion levels were tried and the one which produced minimum variability for the block of trials while still being set close to baseline activity was used. The selection of this trigger level was not too difficult, since the EMG was reduced in both biceps and triceps during the foreperiod and the final EMG activation in response to $S^D$ started abruptly as a large burst. Median EMG onset latency and semi-interquartile range were determined for each block of trials.

A more convenient way of estimating average EMG latency and duration for a block of trials was to sum the rectified EMG voltage trial-by-trial in a Mnemetron Computer of Average Transients (CAT). The taped activity was summed either in a forward direction, triggering each trial from the stimulus marker, or in a backward direction, triggering each trial from the response marker as the tape was

run backwards. In forward summing, events time-locked to the stimulus are emphasized; in backward summing, events time-locked to the response. Each resulting curve of summed activity was traced out by an x-y plotter. An example of a forward-summed voltage record for a block of 100 trials is given in Fig. 1. Average EMG latency for the forward sum was measured as the latency of the half-amplitude point between baseline and the height of the sum at the median key-release latency. EMG burst duration for the forward sum was then estimated as the median release latency minus the EMG latency. Average EMG duration for the backward sum was measured as the time from the response marker to the half-amplitude point between the maximum height of the summed EMG activity and the baseline preceding EMG activation. EMG latency for the backward sum was then estimated as the difference between median response latency and measured EMG duration.

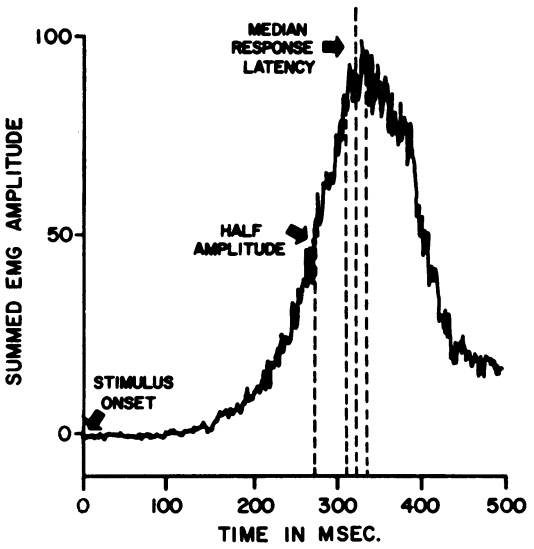


Fig. 1. Rectified biceps EMG activity forward summed from the stimulus markers for a 100-trial sample. Median response latency for the sample is indicated, bracketed by its twenty-fifth and seventy-fifth percentiles. EMG latency was determined as the latency of the half-amplitude point between the baseline and the amplitude of the summed EMG voltage at the median response latency. Summed EMG voltage is given in arbitrary linear units.

## RESULTS

### *Response Latency as a Function of Payoff Band Location*

*Data processing.* Medians and semi-interquartile ranges (SIQR) were used as distribution statistics because they are much less affected than parametric statistics by occasional long response latencies. Measures were based on distributions of an entire day's responses, excluding 10 initial warm-up trials. Session lengths were from 150 to 500 trials for Monkey W and 400 to 600 for Monkey C. In Fig. 2 and 3, medians, bracketed by twenty-fifth and seventy-fifth percentiles, are plotted as a function of the centers of 50 msec-wide payoff bands, measured in milliseconds from onset of $S^D$. Distribution statistics for sessions under the titrated limit procedure are shown for comparison.

*Light flash as $S^D$.* Monkey W (Fig. 2A) had a light reaction time of 190 msec after extended practice under the titrated limit, and 215 msec when returned to the titrated limit for a few days between payoff-band experiments. Starting at a 190- to 240-msec band centered at 215 msec, the animal was shifted to a new overlapping or adjacent band each day; thus, the points plotted in Fig. 2A are for first-day performance on each band with the exception of the 350- to 400-msec band. Four days of practice were given on this band because of its separation from the other bands; the statistics presented are from the fifth day. The points centered at 225 and 245 msec were obtained as the animal was gradually shifted back down toward its minimum latency. The data appear irregular since the animal was usually being shifted to a new band each day.

Monkey C (Fig. 2B) had a light reaction time of 210 msec after extended practice under the titrated limit. After a six months' rest it was returned to the titrated limit for three days and had a reaction time of 225 msec at the beginning of this payoff-band experiment. Payoff bands were non-overlapping. When shifting to a new payoff-band center, the band was widened to 100 msec and gradually decreased to 50 msec over one to three days. Then one week of practice was given before each session plotted in Fig. 2B. The animal was shifted successively from a 200- to 250-msec band up to 400 to 450 msec and then back down to 250 to 300 msec.

*Click (with light) as $S^D$.* Monkey W (Fig. 3A) had a click reaction time of 160 msec after one week of practice under the titrated limit. The animal was shifted upwards from a band

of 170-220 to 645-695, spending at least two days on each band. Statistics in Fig. 3A are for the second day of practice on each band except for the ones for the 375 msec-centered band which are for the fourth day and the 475 msec-centered band which are for the sixth. Performance on the longest band would have improved if practice had been continued past the second day.

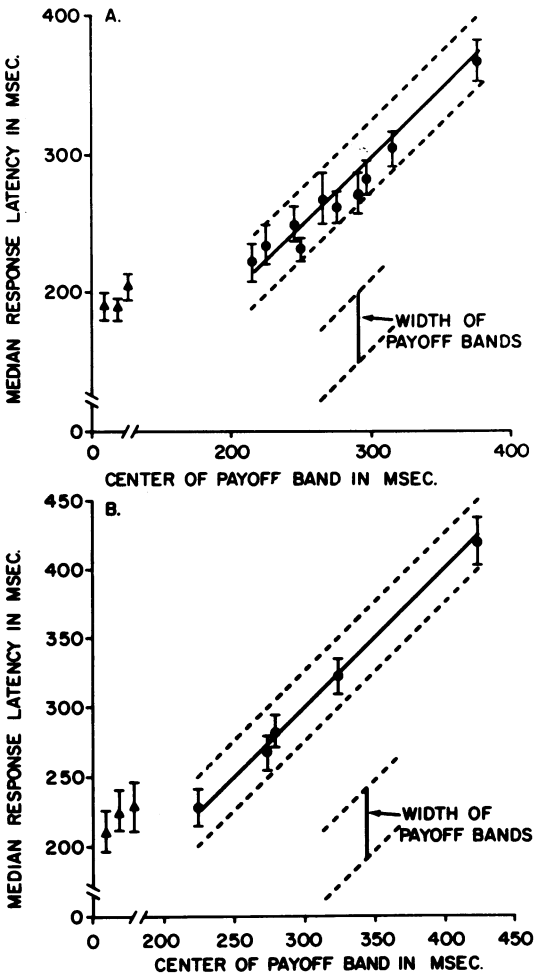


Fig. 2. Median response latency as a function of center of 50 msec-wide payoff band for the light $S^D$. Medians are bracketed by their twenty-fifth and seventy-fifth percentiles. Triangles represent comparison performance under the titrated limit (abscissa scale not relevant). The diagonal line passes through the centers of the payoff bands. (A) Monkey W. Each point comes from the first day on a new band except the one for 375 msec which comes from the fifth day. Each point is based on 150 to 400 responses. (B) Monkey C. Each point comes from at least the seventh day on each band and is based on 400 to 600 responses.

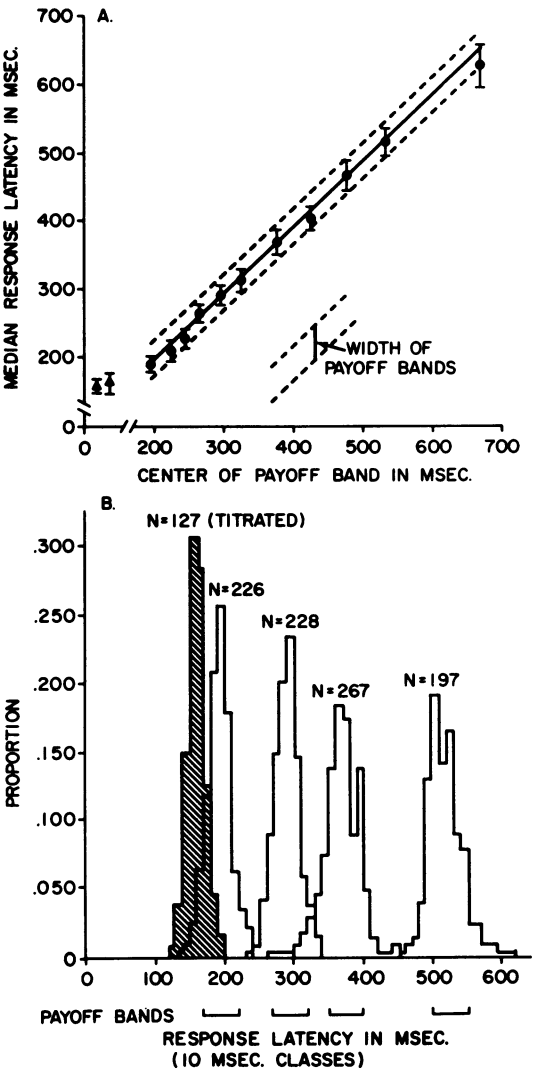


Fig. 3. Response latencies for the click (with light) $S^D$ for Monkey W. (A) Median response latency as a function of center of 50 msec-wide payoff band. Medians are bracketed by their twenty-fifth and seventy-fifth percentiles. Triangles represent comparison performance under the titrated limit (abscissa scale not relevant). The diagonal line passes through the centers of the payoff bands. Subject had at least one day's prior experience on each band. Each point is based on 150 to 500 responses. (B) Representative response latency histograms for the titrated limit and four payoff bands, corresponding to selected points in Fig. 3A. For each distribution, the proportions of response latencies falling within each 10-msec class are given to equalize for the different sample sizes. Variable foreperiods were used with the titrated (cross-hatched), 270 to 320 and 350 to 400-msec payoff bands. Fixed foreperiods of 1 sec were used with the 170 to 220 and 500 to 550-msec bands.

*General characteristics of response latency distributions.* Representative response latency histograms corresponding to some of the points in Fig. 3A are given in Fig. 3B. The proportion rather than the frequency of response latencies in each 10-msec class is used to equalize the different sample sizes. The histograms are narrow, usually unimodal and symmetrical, and sharply peaked. As band center is increased, they broaden and become less sharply peaked.

The semi-interquartile ranges tend to increase proportionately with increased median response latency, remaining about 5% of the median. The animals were able to place 80% of their responses within the fastest band and 60 to 70% in the bands centered near 400 msec. For the slowest band (645 to 695 msec) only about 35% of the responses were within the band (see Fig. 3A).

*Role of foreperiod.* Variable foreperiods (0.5 to 2.5 sec) were used with the titrated limit to control anticipation, whereas a fixed 1-sec foreperiod was generally used with payoff bands in the belief that this would aid timing. Variable foreperiods used during acquisition of a new latency distribution did seem to disrupt performance; however, if they were introduced in the latter half of a session, or after the animal had practiced for a few days on a band, they had little effect. In Fig. 3B, the "titrated", 270 to 320 and 350 to 400-msec bands were all done with variable foreperiods, while the 170 to 220 and 500 to 550-msec bands were done with the more usual 1-sec fixed foreperiod. In Table 1, samples from the latter half of various days' performances, taken during EMG recording sessions, show no consistent differences attributable to fixed *vs.* variable foreperiods between response latency medians or SIQRs for the same band. These findings were replicated on Monkey C.

## *EMG Latency and Duration*

*Agreement of EMG measures.* For several of Monkey W's sessions, two or three measures of EMG latency were obtained. The possible measures were the median EMG onset latency calculated from the trial-by-trial latencies obtained with the Schmitt trigger, the average EMG latency measurement made from the forward summed EMG voltage record, and the EMG latency calculated from the measurement of the average pre-release EMG duration obtained from the backward sum. Table 1 indicates the agreement in the estimations of average EMG latency made from the three measurement procedures. Figure 4C shows the worst discrepancies obtained between estimates of EMG duration (release latency minus EMG latency) obtained in these experiments. Values were generally within 10 msec of one another, indicating that any one of the measures was a good estimator of average EMG latency and duration. Therefore, in the rest of Fig. 4, only the half-amplitude latency and duration estimates from the forward sums are presented.

*EMG latency and duration as a function of response latency.* Figures 4A-C show that as re-

Table 1

Comparison of response latency and biceps EMG latency measures for Monkey W., click (with light) stimulus (entries in msec).

| Payoff Band | Foreperiod | N | Correlation Response vs. EMG Latency* | Medians | | ½ Amplitude Summed EMG Activity | | Semi-interquartiles | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Response Latency | EMG Latency* | Forward Sum | Back Sum | Response Latency | EMG Latency* |
| Titrated | Variable | 153 | — | 182 | — | 123 | 121 | 8.9 | — |
| 300-350 | Fixed 1 sec | 100 | 0.70 | 322 | 272 | 270 | 275 | 20.0 | 27.4 |
| 300-350 | Variable | 100 | 0.62 | 321 | 270 | 273 | 268 | 13.2 | 25.4 |
| 400-450 | Fixed 1 sec | 105 | 0.71 | 430 | 351 | 357 | 367 | 29.1 | 33.5 |
| 400-450 | Fixed 1 sec | 50 | 0.78 | 425 | 368 | 367 | 358 | 9.5 | 11.5 |
| 400-450 | Variable | 51 | 0.85 | 427 | 371 | 370 | 361 | 12.7 | 15.7 |
| 500-550 | Fixed 1 sec | 100 | 0.88 | 517 | 463 | 460 | 464 | 15.2 | 16.8 |
| 500-550 | Variable | 102 | 0.75 | 525 | 467 | 463 | 472 | 17.1 | 22.6 |
| 550-620 | Fixed 1 sec | 101 | 0.81 | 595 | 554 | 550 | 555 | 21.0 | 27.8 |

*Trial-by-trial values obtained with Schmitt trigger.

sponse latency is increased by payoff bands, EMG latency increases by the same amount while pre-response EMG duration remains constant. Figures 4A and 4B are both for click (with light) as $S^D$. Biceps activation duration (Fig. 4A) is constant at about 50 msec while triceps activation time (Fig. 4B) is constant at 30 msec. Figure 4C is for biceps with light as $S^D$. Again, the EMG activation duration is constant at about 60 msec.

The trial-by-trial response and EMG latency correlations are given in Table 1 for some of Monkey W's click (with light) sessions. These range between 0.62 and 0.88.

## DISCUSSION

It is clear from Fig. 2 and 3 that it is possible to control precisely, by the use of payoff bands, the location of a reaction time distribution anywhere between response latencies of the order of minimum reaction times and latencies of 600 msec. Earlier work (Stebbins and Miller, 1964) indicated that without time pressure, the monkey reaction time distribution to strong signal light or sound falls between 300 and 400 msec. This is as fast as the animal will perform just to get the food quickly. An upper limit, giving differential reinforcement of brief latencies, is necessary to get fast responding which approaches the human latencies achieved with verbal instructions. Use of the titrated upper limit in the present experiment produced brief reaction times of 190 to 210 msec for light and 160 msec for click, which compare well with human results. By use of the payoff-band technique the reaction time distribution can be shifted up or down without altering its sharply peaked characteristics.

The response latency distributions obtained on monkeys in this experiment compare well with those obtained on humans by Snodgrass *et al.* (1967) and with those commonly obtained in experiments on human simple reaction time. Monkey response latency distributions had semi-interquartile ranges of about 5% of the median, which would correspond to standard deviations of less than 10% of the mean, assuming normal distributions. The measure of variability in this experiment, the semi-interquartile range, thus increases proportionately with the measure of central tendency. The results of Snodgrass *et al.* also imply

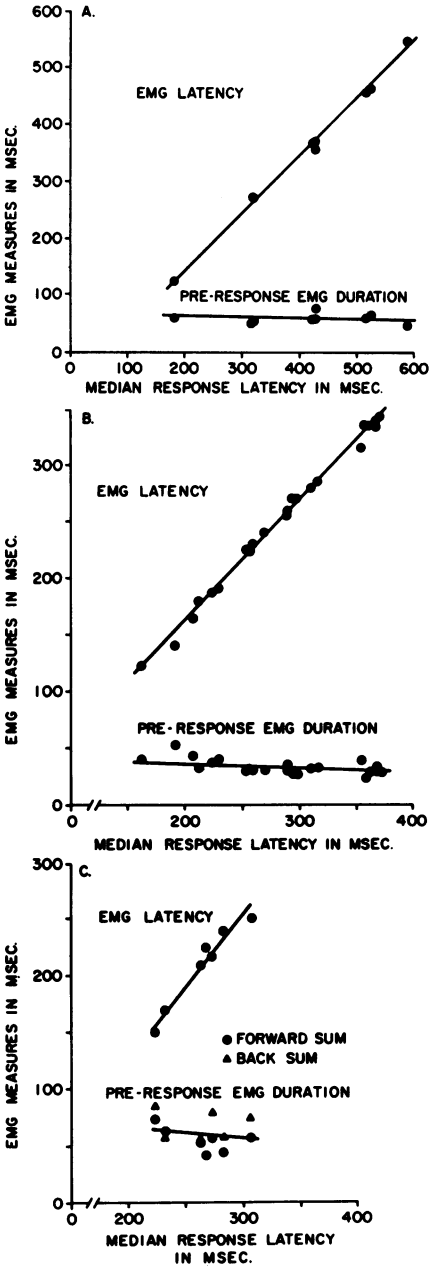


Fig. 4. Right arm EMG latency and duration as a function of right arm response latency for Monkey W. Estimates of EMG measures are based on latency for one-half amplitude point of rectified EMG activity, forward summed from stimulus onset for a block of trials. (A) Biceps EMG with click (with light) as $S^D$. Sample size was 50 to 100 trials. (B) Triceps EMG with click (with light) as $S^D$. Sample size was 150 to 350 trials. (C) Biceps EMG with light as $S^D$. Sample size was 150 to 400 trials. Backward summed data are shown for comparison.

that variability increases with the length of the interval being timed, for their subjects were able to place 60% of their responses in the 125 to 144-msec band, but only 30 to 40% in the 185 to 204-msec band.

Snodgrass *et al.* used fixed foreperiods only. The present experiment found that, for a given band, changing from fixed to variable foreperiods did not greatly change the variability or location of the latency distribution, indicating that the subjects were indeed timing from $S^D$. The semi-interquartile range for timing a 1200-msec interval should be about 60 msec if the SIQR stays 5% of the median. When an animal was giving 200-msec reaction times to $S^D$, which could be interpreted as 1200-msec time estimations from $S^W$ if the foreperiod were fixed, its SIQR was only 10 msec. In a payoff-band experiment, it is more profitable to time from $S^D$ than $S^W$ as long as the band is not too close to $S^D$. The human subjects of Snodgrass *et al.* could place only 10% of their responses in the 84 to 104-msec band, presumably because they were timing at least some of their responses from $S^W$, which was 2 sec away. Thus, narrow payoff bands can be used to prevent time estimation from the warning stimulus as an alternative to the traditional use of variable foreperiods.

Humans and monkeys differ in that it takes considerably longer to shift a monkey to a new band. The human subjects of Snodgrass *et al.* were told when they were being shifted and what the new band was. They could adopt appropriate temporal searching behaviors to locate the new band. The monkeys could discover the need to shift only by the change in density of reinforcement and feedback cues. There was no immediate way of informing them where the new band was located. Near the end of the experiment, Monkey W appeared to be shifting more easily, even to nonoverlapping bands, and to have developed searching techniques for locating a new band. Shifting Monkey C remained a painful process of extinguishing one set of response latencies and slowly building up another distribution.

The question remains of possible mechanisms for rapid precise timing in both monkeys and humans. The concept of overt mediating behavior chains was rejected because of the briefness of the intervals being timed. However, changing hand position or manner of grasping the key as a means of accurately delaying responses deserved serious consideration as a possible peripheral explanation. Measurements of human forearm EMGs in a key-release reaction time experiment by Botwinick and Thompson (1966) indicated that as reaction time was increased 70 msec by changing the foreperiod structure, the EMG latency was correspondingly increased. Pre-response EMG duration remained constant at 40 to 50 msec. In the present experiment, for muscles in the upper arm important in the key-release response, pre-response EMG duration remained constant at 30 msec for triceps and 50 to 60 msec for biceps over a range of response latencies from under 200 msec to over 0.5 sec. Since onset of EMG activation is delayed by exactly the same amount as response latency is increased in a trained responder, this indicates that the timing was centrally controlled. Of course, intervals of several seconds, such as have been used in earlier animal experiments involving response timing, may involve either peripheral adjustments or mediating behavior chains. However, indications from this experiment are that, at least up to 600 msec, response timing can be controlled by a central delay mechanism which emits intervals with variability proportional to the length of the interval being timed.

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