Cardiac Orienting and Startle Blink Modification in Novel and Signal Situations

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ABSTRACT

Prior work has shown that reflex blinking can be facilitated by directing attention to the reflex stimulus. It was assumed that if facilitation were due to sensory enhancement, blinking would also be facilitated by novelty-induced orienting prior to the reflex-eliciting stimulus. The assumption was tested by establishing an expectancy for weak tactile stimulation to be followed by an acoustic reflex stimulus and then introducing, without announcement, trials on which weak acoustic or visual stimuli occurred and were followed by the reflex stimulus. Interspersed control trials of the reflex stimulus alone provided baseline data. Novel trials did produce the expected facilitation of reflex latency and magnitude. Similar effects were obtained when the same stimuli were presented to a different group of subjects with instructions to judge duration of the reflex stimulus on control trials and when warned by the weak acoustic and visual stimuli. In both experiments, blink latency but not magnitude was facilitated by the irrelevant tactile stimulus, thus supporting previous suggestions of a dissociation between these measures of reflex strength. A third group of subjects, receiving identical stimulation, was instructed to attend to the weak acoustic and visual lead stimuli but to perform no task. Attending to the lead stimuli did not produce facilitation of either blink magnitude or latency.

DESCRIPTORS: Attention, Heart rate, Novelty, Orienting, Reflex blink, Startle.

The acoustic blink reflex of human subjects has recently been shown to be sensitive to selective attentional manipulations. Under conditions eliciting heart rate deceleration prior to the occurrence of a startling stimulus, blink magnitude varied depending on the stimulus modality to which attention was directed. When a warning stimulus directed attention to the reflex-eliciting stimulus, blinks were larger than in the unwarmed condition; when attention was directed to concurrent stimulation in another modality, blinks were smaller than control blinks (Bohlin & Graham, 1977; Silverstein, Graham, & Bohlin, 1981). Similar findings have been reported by Anthony (1981), Anthony, Butler, and Putnam (1978), and Putnam and Meiss (1980). In studies in which cardiac deceleration has not been present during the interval between a brief lead stimulus and a reflex stimulus, changes in blink magnitude have not occurred (Bloch, 1972; Brown, 1975; Graham, 1975).

The existence of reflex facilitation concomitant with cardiac deceleration is compatible with the assumption that cardiac deceleration reflects an orienting process whose primary function is to enhance stimulus intake (Graham, 1979). As developed by Sokolov (1963), orienting theory emphasized the generalized orientation reaction which affected the sensitivity of all sensory systems or "analyzers," as well as the sensory channel of the stimulus eliciting the orienting. However, Sokolov also described a localized orienting response which produced "selective adjustment" of analyzers. The

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localized reaction is seen especially clearly in signal situations involving voluntary attention (p. 264). Thus, in the studies cited above where heart rate deceleration was produced by warning of a task stimulus to follow, the selective blink effects may be interpreted in terms of localized orienting.

While selectivity in blink effects is therefore compatible with orienting theory, effects of the manipulation which virtually defines orienting—the introduction of novelty—have never been investigated. If the mechanism underlying the blink effects is enhancement of sensory channels to which an orienting/attentional process is directed, then using a novel, non-signal stimulus to elicit a generalized orienting reaction should also produce blink enhancement. In addition, the enhancement should be non-selective, i.e., blink facilitation should be independent of the modality of the novel stimulus. The main purpose of the present research was to test these hypotheses.

A second purpose was to determine whether or not an imperative or “command” signal, i.e., a significant stimulus itself commanding activity or attention rather than warning of a significant stimulus to follow, could affect a subsequently-elicited blink and whether, if so, its effects would be localized to the modality of the signal. Sokolov (1963) ascribed signal properties both to command and to warning stimuli, but the direction of any localized orienting elicited by the two types of signal might be expected to differ. While warning stimuli, whether through learning or instructions, should localize orienting to the anticipated stimulus, the localized orienting elicited by a command stimulus would presumably be directed to the signal itself.

Blink latency was also of interest. In the studies cited above the selective effects of sensory modality on blink magnitude contrasted with a non-selective facilitating process that affected reflex latency. The latency of blinking was shorter with warning stimuli, even when magnitude was inhibited. Latency must, therefore, have been determined by a process independent of the direction of attention; Silverstein et al. (1981) suggested that the process might involve a generalized activation of motor systems. The present research provided an opportunity to examine latency in situations having different attentional and motor requirements.

Blink-modifying effects of orienting elicited by the three types of stimuli—warning signals, novel non-signals, and command signals—were measured in successive experiments. The experiments employed identical stimulation and differed only in the role assigned to stimuli by instructions. The critical set of stimuli, i.e., those serving as either warning, novel, or command stimuli, were infrequent acoustically and visually presented stimuli which led an acoustic blink-eliciting stimulus by 2 sec. The critical trials were interspersed among infrequent control trials of reflex stimulation alone and frequent irrelevant trials of tactile stimuli preceding reflex stimuli by 2 sec. The irrelevant trials were introduced so that, in the Novelty experiment, the unannounced critical trial would be unexpected or “novel” and thus elicit orienting. The presence of orienting was indexed by decreases in cardiac rate. Irrelevant trials also provided a second kind of control, for effects due solely to pairing of weak lead and intense reflex stimulus, and were not expected to facilitate either latency or magnitude (Graham, 1975).

In the initial experiment, the critical stimulus signal subjects to judge the duration of the following reflex stimulus. The purpose of the experiment was to verify that previous facilitation effects could be replicated with the more complex stimulus configuration of the present series. It was expected that blink would be facilitated both by acoustic and visual warning signals since both stimuli directed attention to the reflex stimulus. The experiment did not test directly for selective warning signal effects by varying modality of the reflex stimulus. The second, novelty, experiment was also expected to produce blink facilitation by both modalities of critical stimuli, not because they anticipated the acoustic-reflex stimulus but because novel stimuli should elicit generalized orienting (Sokolov, 1963). In the third experiment, the critical (command) stimuli were described as important stimuli to which attention should be paid so that biological responses would be maximized. If orienting evoked in this way could persist through the 2-sec interval before reflex stimulus presentation, its effects would presumably be modality specific.

GENERAL METHOD

Subjects

Sixteen introductory psychology students served as subjects in each experiment and received extra credit for their participation. All 48 subjects met criteria of normal auditory, cardiac, and respiratory function as judged from a pre-experimental questionnaire. Median age was 19, ranging from 18 to 28, and number of males was 16, ranging from 5 to 6 per experiment. Fourteen additional subjects were replaced because of equipment failure (n = 2), experimenter error (n = 1), failure to meet predetermined criteria for acceptable trials (n = 7), failure to adhere to task instructions (n = 3), and reported aversiveness of tactile stimulation (n = 1).

Apparatus

Sessions were conducted with the subjects seated in an IAC model 1203A sound-attenuated chamber (ambient noise level 31dB(A) re 20 μN/m²), isolated from the experimenter and way communication and subjects were stimulated with PDP12 computer generated broad-band noise generators which were gated with lock-in audiotape and model 350D Stadler model 3:1 were delivered by Sound intensity model 2203 sound artificial ear and through the earphone. Source for the startle stimulus were the electrotet wire-cathode, forceps controlled by an the subject’s head was positioned in an ambient illuminance which the fixation to 0.4060 foot-lambert. The subject and visual and visual visual stimuli limits matched were not subjects.

Two responses electrocardiogram recorded by an of the rule of the the Beckman biopotential equipment was led through a system which provided wave. The comp wave pulses, to the after the startle stimuli was received 68 s for each half of the movement proper (4 h included one control stimulus occurred.
ed an acoustic blink; critical trials were in-control trials of reflex or irrelevant trials of stimuli by 2 sec. The so that, in the Novinskis' critical stimulus order and thus eliciting was indexed by bivalent trials also pro-
perimenter and equipment. An intercom allowed two-way communication between chamber and control room, and subjects were also visible on closed-circuit television.

Stimulus delivery and timing were controlled by a PDP12 computer. The auditory lead and startle stimuli were broad-band white-noise bursts, from Grason-Stadler noise generators (models 453C and 901B, respectively), which were gated on and off nearly instantaneously by Ionix audiogates. They were led through Hewlett-Packard model 350D attenuators to a mixer and a Grason-Stadler model 1288 1-watt audio power amplifier, and were delivered binaurally to TDH49 circumaural phones. Sound intensity was calibrated with a Bruel and Kjaer model 2203 sound-level meter coupled to a type 4153 artificial ear and type 4134 (1.25 cm) condenser microphone. Sound pressure levels were 104dB(A) per ear for the startle stimulus and 65dB(A) per ear for the auditory lead stimulus.

The electrotactile lead stimuli were generated by a Wisconsin Medical Electronics Laboratory isolated, constant-current stimulator which delivered 60 Hz, .6 mA stimulation through Beckman biopotential electrodes secured to the ventral surface of the left forearm. The dorsal electrode was centrally located 10 cm proximal to the wrist and the second electrode, 2 cm (center to center) proximal to the first.

Visual lead stimuli were produced by an Ionix cold catherode fluorescent (CCF) lamp, with 2-msec rise time, controlled by an Ionix 6196 driver unit. The CCF was located approximately .3 m behind and .6 m above the subject's head and provided a brief, homogeneous change in ambient illumination such that luminance of the wall on which the fixation point was located increased from 3045 to 4060 foot-lamberts as measured by a Pritchard photometer. The subjective intensity of auditory, electrotactile, and visual lead stimuli was equated by means of a method of limits matching procedure using four observers who were not subjects in the main experiments.

Two response measures, eyelid movement and the electrocardiogram, were amplified and continuously recorded by an Offner Type R Dynograph and were sampled by the PDP12 computer. Samples of lid position were taken every msec for 250 msec following each lead and startle stimulus. A Conarc 85153 micro-torque potentiometer, whose output fed a modified Beckman type 9803 strain gauge coupler, detected position through mechanical coupling to the lid. The electrocardiogram, detected by Beckman biopotential electrodes in a horizontal II placement, was led through a filter/Schmitt trigger/one-shot combination which provided shaped pulses coincident with R-wave. The computer timed intervals between these R-wave pulses, to the nearest msec, for 5 sec before to 9 sec after the startle stimulus.

Procedure

As diagrammed in Figure 1, subjects in each experiment received 68 trials consisting of a familiarization set before each half of the session and trials making up the experiment proper (4 blocks of 7 trials in each half). A block included one control trial in which the white noise startle stimulus occurred without warning, five trials in which on-
measured responses beginning within a window 21–120 msec after reflex stimulus onset and discarded trials on which the lid was partially closed or in movement at the time of stimulus presentation (see Graham, Putnam, & Leavitt, 1975 for further details). Discarded trials were not replaced; they averaged 41.9% of all trials and did not differ among conditions and experiments. No-response trials, averaging 3.1%, also did not differ among conditions and experiments. They were assigned a score of zero for analyses of magnitude and were not employed in latency analyses. Heart rate, expressed as number of whole and fractional beats per minute (bpm) per 200-msec period (Graham, 1978), was analyzed for eleven 200-msec periods: on control trials, the 2200 msec preceded the reflex-elicting stimulus; on lead trials, 200 msec preceded lead-stimulus onset and 2000 msec covered the interval between lead and reflex-stimulus onsets.

For each measure and each subject, data consisted of averages within session halves of the 4 critical trials, the 4 control trials not in the familiarization set, and 8 irrelevant trials. The irrelevant trials were preselected, 2 from each block, to have the same mean trial position as control and critical trials. Thus, three main effects and all interactions involving them were estimated for blink measures: the within-subject effect of trial type, the within-subject effect of the first half of the session (4 blocks) versus the second half (4 blocks), and the between-subject effect of order of receiving auditory and visual stimuli, i.e., whether in the first or second half of the session. The interaction of halves and order of auditory and visual stimuli thus estimated effects pertaining to critical stimulus modality. Heart rate analyses estimated, in addition, the main effect of the eleven 200-msec periods, the linear trend over periods, and the interactions of periods with the effects above.

It had been planned to divide the trial type effects into two orthogonal comparisons: 1) critical trials versus the average of the two types of control conditions (startle-alone, no-lead control trials and irrelevant, lead-control trials), and 2) startle-alone versus irrelevant trials as a test of the assumption that they could both be called controls. Because the assumption proved invalid for latency and it appeared desirable to use parallel analyses for all measures, the three pairwise contrasts of trial type (and interactions involving trial type) were made—critical versus control, critical versus irrelevant, and control versus irrelevant. Alpha levels were lowered to .025 to compensate for the additional comparisons. A second assumption, that visual and auditory lead stimuli could both be called critical stimuli, i.e., would not produce significant differences in response, proved to be valid for all three response measures. The assumption was tested by the specific comparison of responses to auditory versus visual stimuli: there were no significant differences using either the overall order × halves error term or the specific critical-trials order × halves error term.

**WARNING SIGNAL EXPERIMENT**

Critical stimuli in this experiment served as warning signals of a discrimination task to be performed to the following reflex stimulus. Subjects were instructed to judge whether reflex noise bursts were short or long, except when they were preceded by tactile lead stimuli. It was explicitly pointed out that the visual and auditory lead stimuli would allow preparation for listening to the duration of the noise burst occurring 2 sec later. The task was unspeeded and subjects announced their judgments via intercom following each target presentation. No practice or feedback was given but the nature and frequency of stimulation to be delivered was described in detail prior to the experiment and the experimenter questioned subjects to be sure that the task was clearly understood.

**Results**

**Blink Response**

Blink magnitude was significantly modified in the expected direction as a function of trial type. As Figure 2 (upper) illustrates and Table 1 confirms, blinks were larger on critical stimulus than on control or irrelevant trials, whereas control and irrelevant trials did not differ from one another. The only significant interaction was critical stimulus type × order of trials. Figure 2 illustrates the significant difference in magnitude and latency as a function of stimulus lead (critical vs. irrelevant) and of order of presentation (exp. 1 vs. 3).

![Figure 2. Blink magnitude and latency differences from control for irrelevant lead and critical lead trials. Experiments 1, Warning Signal experiment: mean control magnitude = 4.93 mm; latency = 70.3 msec. Experiment 2, Novelty experiment: mean control magnitude = 4.36 mm; latency = 71.6 msec. Experiment 3, Command Signal experiment: mean control magnitude = 7.66 mm; latency = 66.9 msec. Asterisks indicate significance levels for difference of trial type from control (**, ***, ****, *****), respectively, p-values less than .025, .01, .005, and .001.]

Heart rate c reflex stimulatory changes were s 140 = 4.88, p = 0.42 (linear p
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LATENCY

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Latency differences from all lead trials. Experiment 2: control magnitude . 36 mm: latency 71.6 msec experiment: mean latency 66.9 msec. As for difference of trial type respectively, p-values less

<table>
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<tr>
<th>Source</th>
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<th>Novelty Signal Experiment</th>
<th>Command Signal Experiment</th>
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</table>

Latency

- Critical: critical trials; Con: control trials; Irrel: irrelevant tactile trials; Aud: auditory critical trials; Vis: Visual critical trials.

**...*** indicate, respectively, p-values less than .025, .01, .005, .001.

other significant effect on blink magnitude was habituation from first to second half of the session.

Blink latency was also affected by trial type, but the pattern of effects was different from that observed for magnitude (Table 1). Figure 2 (upper) shows that while blink was faster on the critical warned trials than on unwarmed control trials, blinks on irrelevant lead trials were faster as well. This was reflected in the significant difference between the warned lead trials and the control trials and between irrelevant lead trials and control trials.

Heart Rate Response

Heart rate changes during the 2 sec preceding reflex stimulus are shown in Figure 3A. Overall, changes were significant (200-msec periods $F(10/140) = 4.88, p<.001$) and generally linearly decelerating (linear periods $F(1/14) = 7.86, p<.025$), but they did not interact with trial type in the manner expected. Although the overall periods × trial type effect was significant ($F(20/280) = 3.21, p<.001$), there was no difference among trial types in the deceleratory linear trend. However, trial type effects differed between the first and second halves of the session. These differential changes were reflected in significant interactions both with periods and with the linear trend: type × half × periods $F(20/280) = 5.22, p<.001$; type × half × linear periods $F(2/28) = 7.22, p<.005$. Of particular interest, the differential effect in the two experiment halves was significant when critical trials were compared with control trials ($F(1/28) = 14.36, p<.001$) but was not significant for control versus irrelevant trials. Inspection of heart rate curves (Figure 3A) indicates that during the first half of the experiment heart rate on critical trials decelerated during the first 600 msec and then accelerated during the remainder of the interval, whereas in the second half, heart rate decelerated monotonically during the whole interval. In both halves, the heart rate deceleration in the first 600 msec was significantly below heart rate during the preceding second ($t$-ratios at 600 msec = 3.56 and 2.78, for first and second halves, respectively). No other effects, including critical stimulus modality or interactions with modality, were significant.

Task Performance

As intended, the duration discrimination was of near-threshold difficulty. Judgments were 73% correct on control trials, 70% correct with auditory warning signals, and 70% correct with visual warnings. Differences between warned and unwarned conditions were insignificant, presumably due to the small amount of practice; there were only 4 trials with each of the two warning stimuli.
Discussion

Blink effects in the present experiment replicated, in a more complex design, the previous findings (Bohlin & Graham, 1977) that both magnitude and latency of a reflex blink may be facilitated when a lead stimulus signals a sensory task involving the reflex-eliciting stimulus. The subsequent conclusion of Silverstein et al. (1981) that magnitude and latency were dissociated was also supported. The irrelevant-lead trials had no effect on magnitude, but produced significant latency facilitation.

In designing the present experiment, it was assumed that any facilitation of blink magnitude would be due to orienting/attending in anticipation of the reflex stimulus and that orienting would be reflected in decelerative heart rate changes. Therefore, facilitation of blink magnitude should not occur unless a decelerative heart rate response is elicited by the critical warning stimuli. However, the present results showed a dissociation between heart rate changes and blink facilitation: Although deceleration lasted only 600 msec in the first half of the session, blink was equally facilitated in both halves.

The dissociation is compatible with our presuppositions only if it can be assumed that deceleration-acceleration in the first half was the net result of two processes: an orienting process producing deceleration and a second process giving rise to acceleratory changes which masked the continuing orienting. The monotonic deceleration in the second half of the session would result from disappearance of the second process during the course of the experiment. Accelerative heart rate changes have been shown to be produced by muscular tension (e.g., Chase, Graham, & Graham, 1968; Obrist, Howard, Lawler, Galosy, Meyers, & Gaebelein, 1974) and by cognitive activity such as memory search, rehearsal, and decision making (e.g., Coles & Duncan-Johnson, 1975; Jennings, 1975; Lacey & Lacey, 1974). In view of the complex design and relatively few critical stimuli, it is not unlikely that such processes occurred during lead intervals in the beginning of the experiment but dissipated as the subjects became more familiar with the task. In any case, the dissociation suggests that any relationship between blink and heart rate is not due to direct effects of heart rate on blink but to mediation by a common process.

NON-SIGNAL NOVELTY EXPERIMENT

The critical stimuli in this experiment were not mentioned in the instructions to subjects and were therefore “novel” when they occurred. There was no task and subjects were told only that the experiment concerned physiological responses to various kinds of stimulation. The noise and tactile stimuli were described in detail, as well as the fact that tactile stimuli would always be followed by noise stimuli.

Results

Blink Responses

Trial type again affected blink magnitude and latency (Figure 2, Table 1). Critical trials produced blinks that were significantly larger than control blinks and insignificantly larger than irrelevant blinks. The two types of control trials (critical and irrelevant) did not differ from one another. In contrast to magnitude, the significant latency effect was due to facilitation by both types of lead stimulus trials (critical and irrelevant). Latencies on the critical novel and irrelevant trials did not differ from one another but each differed significantly from the no-lead control. As previously, magnitude but not latency habituated over session halves. No other effects were significant (Table 1).

Heart Rate Response

Cardiac changes, illustrated in Figure 3B, showed the expected deceleration in the lead interval. Not only were the main effects of periods and the decelerative linear trend over periods significant (periods $F(10/140) = 42.88, p < .001$, and linear periods $F(1/14) = 67.34, p < .001$), but both effects interacted with trial type ($F(20/280) = 6.35, p < .01$ and $F(2.28) = 10.79, p < .001$, respectively). The deceleration was larger on the critical novel than on either control or irrelevant trials ($F(2.128) = 21.58 and 5.80, p < .001$ and $< .025$, respectively) and the control and irrelevant trials did not differ from one another. Heart rate response was not affected by order, session half, or modality of the novel stimuli, or by interactions involving them.

Discussion

The facilitation of blink magnitude and latency following novel stimuli supported our hypothesis that novelty-induced orienting, by enhancing the reflex stimulus, would enhance the reflex. Further, in keeping with the concept that novel stimuli induce generalized orienting, there was no effect of modality of the critical stimuli. As expected, novel stimulation produced heart rate deceleration, an index of orienting. That the deceleration was not due solely to pairing a lead and reflex stimulus is indicated by the significantly greater deceleration on novel-lead than on irrelevant-lead trials.

COMMAND SIGNAL EXPERIMENT

In the third experiment, the critical stimuli were intended to command the subject's attention. There was no overt task and instructions did not mention stimulus pairing was described as a logical response. The light flanker ignored import response difference attention on uli. They were i memorize any o
stimulus pairing. The purpose of the experiment was described as determining differences in the biological response to important and unimportant stimuli. The light flashes and soft noise stimuli were designated important and subjects were told that response differences could be maximized by focusing attention on the qualities of the important stimuli. They were instructed not to rehearse, count, or memorize any of the stimuli.

Results

Blink Responses

Directing attention to the critical stimuli did not have any significant effect on blink magnitude and only blinks on irrelevant trials showed significantly faster latencies (Figure 2, Table 1).

Heart Rate Responses

Although blink magnitude was not facilitated by attention directed to the critical lead stimulus, heart rate did decelerate significantly (Figure 3C). Main effects of periods, $F(10/140) = 14.35, p < .001$, and linear periods, $F(1/14) = 13.92, p < .001$, interacted with trial type ($F(20/280) = 3.75, p < .001$ and $F(2/28) = 4.54, p < .025$, respectively). Further, the linear trend was significantly greater on critical than on control trials, $F(1/28) = 9.08, p < .01$. No other factors influenced the response.

Discussion

The occurrence of heart rate deceleration following the critical stimuli is in keeping with Sokolov's idea (1963) that command signals evoke orienting. It is clear from the blink magnitude data, however, that if a command signal can have enhancing effects on subsequently occurring stimuli, such effects do not last as long as those produced by novelty or by warning of a sensory task. Evidence for latency facilitation by critical stimuli was also lacking, despite the fact that irrelevant stimuli continued to be followed by a shorter latency reflex. Had latency been influenced only by a generalized process, both types of lead stimuli should have produced facilitation.

GENERAL DISCUSSION

The main purpose of the present research was to determine whether or not the reflex blink enhancement, occurring when a stimulus warns of a task involving a blink-eliciting stimulus (Boblin & Graham, 1977), could also be produced by a prior novel stimulus when there was no task. The findings were positive, suggesting that blink magnitude facilitation does not require anticipation of a specific informative stimulus, but may also occur as the result of a generalized orienting reaction which presumably affects all sensory analysers (Sokolov, 1963) and does not depend on the modality of the novel stimulus. It might be argued that subjects, although un instructed, nonetheless anticipated the reflex stimulus when they received the novel stimulus, because the reflex stimulus had previously been paired with the irrelevant tactile stimuli. Opposed to this argument is the absence of any effect of the irrelevant stimuli on blink magnitude or heart rate, indicating that pairing alone is not sufficient to develop the kind of anticipation, presumably a sensory-enhancing attention, that increases blink size.

The demonstration that blink size is increased both with orienting and directed attention suggests that the reflex is facilitated because the stimulus input side of the reflex is enhanced and not because of a generalized activating or motor readiness system. The fact that it is heart rate deceleration and not acceleration which is generally associated with blink magnitude modifications also implicates an orienting/attentional as opposed to an activation process; it is heart rate acceleration which characterizes an activation system (Malmö, 1959). Previous demonstrations that facilitatory changes in blink size can be eliminated or reversed if attention is directed away from the blink-eliciting stimulus (Silverstein et al., 1981) are further evidence of orienting/attentional effects. Although it is not clear what stance orienting theory takes with regard to whether unselected analysers are not facilitated or are actively inhibited, and attentional theories are divided on the question (e.g., Shiffrin & Schneider, 1977), activation theory makes no provision for either type of selective effect.

A second purpose of the present research was to determine whether or not attention directed to the lead stimulus itself could affect a reflex elicited 2 sec later. Although the occurrence of heart rate deceleration in the interstimulus interval supports Sokolov's contention (1963) that orienting is elicited by command stimuli, the blink findings were negative and thus could not distinguish between hypotheses of generalized versus localized orienting. Considered from a selective attention framework, the blink magnitude results are consistent with the view that stimuli are enhanced or reduced depending on the direction of an ongoing attentional process and are not affected by attentional processing that has been completed. If subjects in the Command experiment followed directions not to rehearse, count, or memorize stimuli, attentional processing would not be required to last beyond the presumably brief time it takes to process a 20-msec input. Because heart rate decelerations lasted for the full 2 sec, it would have to be assumed that the peripheral index of a central process may outlast the central activity.

The simple assumption that attentional process-
ing of the critical stimuli is completed before the reflex stimulus occurs is not, however, sufficient to account for the lack of latency facilitation by the critical command stimuli. Latency has been facilitated by lead stimuli in past experiments whether or not magnitude was affected (Silverstein et al., 1981) and the irrelevant stimuli in the present experiments produced latency facilitation in the absence of any magnitude effect. It would appear, therefore, that attention directed to the first stimulus has residual effects on the latency of response to the reflex stimulus. It is possible that completing a required task leads to a temporary reduction in activation.

The present research was also concerned with the question of latency-magnitude dissociation. Such a dissociation has been reported previously for very short lead-interval conditions, both with the whole body startle reflex of rats (e.g., Hoffman & Wible, 1970; Stitt, Hoffman, & Marsh, 1976) and the blink reflex of humans (Graham & Murray, 1977), and also for long lead intervals when lead stimuli directly the attention of human subjects away from a blink-eliciting stimulus (Anthony, 1981; Silverstein et al., 1981). The suggestion, proposed by Silverstein et al., that latency facilitation reflects a generalized activation of motor systems by stimuli warning of the need for a motor output was not supported by the present data. Latency was facilitated both in the Warning Stimulus experiment where an unspeeded motor act was explicitly required, and in the Novelty experiment where motor readiness was not explicitly required. If latency change reflects motor system activation, at least it is not a function of explicit motor requirements.

The present studies did confirm, however, that latency facilitation at long lead intervals can occur in the absence of magnitude change and showed, further, that the two measures of reflex strength are differentially affected by stimulus and instructional manipulations. Stimulus repetition, which led to significant habituation of blink magnitude in all experiments, had no reliable effect on latency. Simple pairing of an "irrelevant" tactile lead stimulus with the reflex stimulus also had no effect on blink magnitude but consistently shortened latency. Whether or not this effect is unique to tactile stimuli remains to be determined, but previous work has shown that, at least under some circumstances, tactile lead stimuli can produce both facilitation and inhibition of blink magnitude and the magnitude and latency effects are dissociated (Anthony, 1981; Graham, 1980). Finally, latency may be more affected than magnitude by modality of the critical stimuli. Interactions involving modality were significant only for latency in the Warning Signal experiment, although the pattern of relatively greater facilitation following visual than acoustic stimuli could be observed in all experiments and for both magnitude and latency. Such a consistent pattern might be due to imperfect psychophysical matching, to the fact that the acoustic lead stimulus was in the same modality as the reflex stimulus, or to inherent differences in the "alerting" capacity of acoustic and visual stimuli (Posner, 1978).

Magnitude and latency of a reflex usually change together. In seeking an explanation of why they do not in the situations studied, three points constrain explanation. First, the evidence suggests that two processes must be involved because latency and magnitude are differentially affected by manipulations; second, because onset latency reflects the time at which a response is initiated, the process determining latency must produce its effect before the process determining magnitude; third, the process determining latency probably occurs at a relatively low level of the nervous system consistent with its resistance to habituation, insensitivity to information, and sensitivity to inherent stimulus properties, i.e., latency.

The general principles of a dual mechanism model, proposed by Graham and Murray (1977) to account for latency-magnitude dissociation at short lead intervals, appear capable of also encompassing the long-interval effects. Graham and Murray proposed that stimuli activate both a relatively direct and a relatively indirect pathway to the pontine-medullary startle center (Gendelman & Davis, 1979; Szabo & Hazafi, 1965). When lead stimuli elicit activity in the direct pathway, they presumably prime that pathway so that a subsequent stimulus is more effective. Because the direct pathway is, by definition, the shortest or fastest pathway, it would thus determine the initiation (i.e., onset latency) of the reflex unless the pathway were blocked or attenuated. It is assumed that the direct path has a relatively small effect on reflex magnitude or acts as a gate and has no effect, and that magnitude depends primarily on earlier-arriving activity in the indirect pathway. Because activity in the indirect path is assumed to reach higher centers before feeding back to the startle center, it could be enhanced or reduced by processes such as attention. If the indirect path fed back at multiple levels, processes other than attention could also modify the reflex (Graham, 1975).

In applying the dual-mechanism model to long-interval effects, it is necessary to assume a mechanism, such as stimulus-induced reverberating circuits in reticular formation, for maintaining priming because, otherwise, priming of a direct brainstem path would probably decay in less than 2 sec. Presumably, high levels of tonic activation could prolong and low levels could shorten such periods of reverberating activity. If so, a temporary reduction in

both magnitude and latency might be due to the fact that the same magnitude of acoustic and visual stimuli occasionally change, and that they do not usually produce the same latency, and are by manipulation, reflect the processes of the nervous system and directly affect the processes of the peripheral nervous system. The process described here, the process described by the term reflex, is a relatively independent process that is not sensitive to the activity of the central nervous system. The central nervous system, in turn, is relatively independent of the activity of the peripheral nervous system.

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