Effects of foreperiod duration on reflexive and voluntary responses to intense noise bursts

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Abstract
The question of whether a common mechanism underlies the facilitation of voluntary and reflexive reactions by a warning stimulus was investigated in two experiments. In both studies, the foreperiod preceding an intense noise burst was manipulated within and between blocks of trials. Previous reaction time experiments have shown that individuals respond fastest at the shortest foreperiod for between-block manipulations and fastest at the longest foreperiod when foreperiod duration is varied unpredictably from trial to trial. In the present research, this pattern was found for voluntary hand-grip responses, but acoustic startle blanks were facilitated at long foreperiods for both within- and between-block manipulations. Invariance of the trisynaptic postauricular reflex across foreperiod conditions was evidence against any general activation of low-level motor pathways by warning stimuli. Analyses of nonreflexive lid movements subsequent to startle blink suggested that inhibition of spontaneous blinking during the foreperiod may have contributed to the unexpected divergence between voluntary reactions and eyeblink reflexes.

Descriptors: Alerting, Immediate arousal, Auditory attention, Brain stem, Expectancy, Orbicularis oculi, EMG

Since the time of Wundt (1880, cited in James, 1890), researchers have known that a warning stimulus facilitates voluntary reaction time (RT). Surprisingly, this latency facilitation is also found for eyelid reflexes to abrupt acoustic, cutaneous, and photic stimuli (e.g., Silverstein, Graham, & Bohlin, 1981; Zeigler, 1982), with the amount of enhancement dependent on foreperiod duration (e.g., Hackley & Graham, 1987). Is there a common mechanism?

If so, then essential characteristics that have been identified for warning effects on voluntary RT should also be observable for reflexes. An important characteristic that has been documented for speeded voluntary reactions is the contrasting effects of blocked versus variable manipulations of foreperiod duration. When foreperiod varies randomly from trial to trial, faster reactions are usually observed at the longest foreperiods due to the progressive increase in the objective probability of reaction stimulus occurrence as the foreperiod “ages” (Klepper, 1956; Niemi & Nääätänen, 1981; Requin, Brener, & Ring, 1991). For example, if a time interval greater than all but the longest possible foreperiod has passed and the reaction stimulus has still not occurred, the individual can be certain that the stimulus is imminent. On such long foreperiod trials, the participant will be well prepared at the time of stimulus arrival, and the reaction latency will be brief. By contrast, prior to stimulus arrival on the shortest foreperiod trials, all possible foreperiods are equiprobable. Temporal uncertainty is maximal, and the participant is caught in a relatively unprepared state. Consequently, reaction times during these trials tend to be long.

The opposite relationship between foreperiod duration and reaction time is usually obtained when foreperiod duration is manipulated across blocks (Woodrow, 1914; reviewed in Niemi & Nääätänen, 1981). In such cases, the participant’s ability to estimate short versus long time intervals is the main determinant of the degree of readiness. Speeded performance is poor for the longer foreperiods because these intervals are more difficult to estimate and, hence, participants cannot prepare as effectively for the reaction stimulus.

These foreperiod effects interact with reaction stimulus intensity, at least within the auditory modality (e.g., Berstein, Chu, Briggs, & Schurman, 1973; Niemi, 1979; Sanders, 1977). Specifically, there is a decrease in the slope of the RT–foreperiod function for strong as compared with weak reaction stimuli. According to the most widely accepted interpretation, moderate or intense acoustic stimuli trigger a phasic increase in immediate arousal, or alerting, that can compensate for the low degree of preparedness associated with temporal uncertainty (Posner, 1978, chap. 5; Sanders, 1977, 1980).

These well-studied RT phenomena—the Foreperiod × Intensity and Blocked Foreperiod × Variable Foreperiod interactions—were the focus of our comparison of warning effects on voluntary and reflexive reactions. Our experiments were motivated by pre-
vously documented parallels between alerting effects on voluntary responses and eyeblink reflexes. In a study by Hackley and Graham (1987), for example, both latency and magnitude of acoustically and cutaneously elicited blinks were facilitated at long relative to short foreperiods when the warning interval was changed randomly (1–4 s) within blocks of trials, similar to the typical pattern for voluntary RT.

In an experiment reported in abstract form by Putnam, Butler, and Anthony (1978; discussed briefly in chapters by Anthony [1985] and Putnam [1990]), blocked and variable foreperiod effects on the acoustic blink reflex were compared. For participants in both the blocked and variable groups, eyeblink magnitude increased linearly with foreperiod duration, which ranged from 2 to 16 s. This pattern was not what would be expected based on the reaction time literature, but two important methodological differences might have explained the discrepancy. First, because the 75-dB warning stimulus persisted throughout the duration of the foreperiod, a greater level of arousal may have built up at the longer intervals in both groups due to the dynamogenic effects of such stimuli (e.g., Graham, 1975). Second, because the participants were not assigned any explicit task during this experiment, the temporal uncertainty and preparedness relationships that underlie foreperiod effects on RT may not have been present.

The present study was undertaken to address the question of whether a common mechanism is responsible for the effects of foreperiod duration on voluntary and reflexive responses. Evidence for a common mechanism would help distinguish between two alternative hypotheses regarding the locus of warning stimulus effects on voluntary responses. Posner (1978) theorized that these effects are due to a phasic enhancement of alerting, which reduces the time needed for some central mechanism to respond to the build-up of sensory information. The central mechanism is never specified, but is said to be nonsensory, nonmotoric, attention dependent, and is responsible for certain speed–accuracy tradeoffs (pp. 130–131); hence, the central mechanism could best be characterized as a decision-level process. In contrast, Sanders (1977, 1980) explicitly hypothesized that alerting (or immediate arousal, in his terminology) influences RT at a low-level motor stage referred to as motor adjustment. Evidence in favor of this hypothesis includes the finding (Sanders, 1980) that foreperiod effects on RT interact with instructed tonic muscle tension.

To examine these alternatives, blink-elicted noise bursts were used as discriminative (Experiment 1) or imperative (Experiment 2) stimuli in voluntary performance tasks. If the pattern of results is the same for both reflexive and voluntary responses, then the common mechanism hypothesis would be supported. Because reflexes are presumably not mediated by a central decision stage, such findings would argue against Posner’s theory. In addition to eyeblink reflexes, the pinna-flexion (postauricular muscle) component of the acoustic startle reflex was also recorded. Because this reflex is generated by a short, probably trisynaptic, reflex arc (Cassella & Davis, 1986; reviewed in Hackley, Woldorff, & Hilliard, 1987), foreperiod effects that parallel those on voluntary responses would be consistent with Sanders’ hypothesis that alerting acts upon low-level motor pathways.

These experiments extend the briefly reported findings of Putnam, Butler, and Anthony (1978) in that a discrete rather than continuous warning stimulus was used, and the intensity of the reflexogenic stimulus was manipulated. Also, the required perceptual discrimination in the first experiment and speeded simple reaction in the second experiment address concerns about the lack of an explicit task in the earlier study by Putnam et al.

**EXPERIMENT 1—PERCEPTUAL DISCRIMINATION TASK**

**Methods**

**Participants**
The final sample comprised 24 adult volunteers (6 men, 18 women; aged 18–42 years) who were recruited from an introductory psychology class. Participants who normally wore glasses or contact lenses removed them during the experiment. Data from 10 additional participants were not included, either because reflexes shown in the signal averaged electromyograms failed to exceed background levels by at least a factor of two (n = 8), or because of equipment failure (n = 1) or experimenter error (n = 1).

**Apparatus and Stimuli**
Participants were seated in a lounge chair throughout the 1-hr long experimental session. Two adjacent red light-emitting diodes served as warning stimuli. The diodes were mounted at eye level 2 m in front of the participant on the wall of the dimly lit room. Reflexogenic stimuli were delivered by a Lafayette Model ANL 912 white noise generator. After amplification by a Lafayette ANL 917 amplifier/mixer, the stimuli were delivered via TDH-50p headphones. These stimuli consisted of 55-ms-long white noise bursts, with instantaneous rise times that were randomly presented at intensities of either 70 or 97 dB SPL (A). The onset asynchrony relative to the warning stimulus was 1,500, 3,000, or 6,000 ms, which are referred to as the short, medium, and long foreperiods, respectively. Warning stimulus duration was 200 ms. Nonreflexogenic target stimuli were presented simultaneously with a random 20% of the startle stimuli. These target stimuli were 65-dB tone pips with a rise time of 35 ms, a tonal frequency of 1000 Hz, and a duration of 90 ms. Thus, a target lasted 35 ms longer than the noise burst within which it was embedded. Presentation of the stimuli and acquisition of the electrophysiological data were controlled by an Intel 286-based computer.

**Procedure**
Each participant was given a brief description of the experiment before providing written informed consent, in accordance with a university-approved protocol. After electrode application, the participant was seated in the lounge chair and given several practice trials in which to learn to detect the weak tone pips. During the experiment proper, the participant fixated on the warning stimulus and maintained a silent count of the number of targets presented within each block of trials. During the interblock interval, the participant reported this count and was given feedback as to its accuracy. Like the Putnam et al. (1978) study, there was no motor task in this experiment. However, participants did need to anticipate, and attend to, the startle stimuli to detect the embedded targets.

The experiment was conducted in a single session composed of six blocks of 24 trials with intertrial intervals of 8–16 s. Interblock intervals were about 3 min, with a longer break given after the third block of trials. For the blocked foreperiod group, each participant received a unique ordering of foreperiods, with the order reversed between the first and the second halves of the experiment. For example, the order for one participant was 1,500, 3,000, 6,000, 6,000, 3,000, and 1,500 ms for Blocks 1–6, respectively. For participants in the variable foreperiod group, foreperiod duration fluctuated randomly from trial to trial within each of the six blocks. When the experimental session was completed, participants were asked to respond to a questionnaire concerning their assessments.
of task difficulty, general arousal, and ability to predict the onset of the noise-burst stimuli.

**Recordings**

Electrophysiological activity was recorded using Ag/AgCl surface electrodes and Grass EC-2 paste. Electrodes were disinfected prior to use and care was taken to avoid excessive abrasion of the skin during application. Bipolar electromyographic (EMG) recordings of the blink reflex were obtained at sites overlying the inferolateral portion of the left m. orbicularis oculi. The postauricular reflex of the right pinna was recorded with two electrodes positioned just posterior to the tendon of insertion for m. reitahens auriculium, the agonist muscle for pinna flexion. (This tendon can be identified as a wedge-shaped protrusion that is visible when the pinna is gently pulled forward.) Bipolar vertical electrooculograms (VEOGs) were recorded with electrodes placed at left superior and inferior orbital sites. The amplifier bandwidth was 10–1000 Hz (−6 dB cutoffs) for EMG and 0.01–100 Hz for VEOG. Analog-to-digital conversion was carried out at a rate of 2500 Hz, with a recording epoch beginning 30 ms prior to reflexogenic stimulus onset and lasting for a total of 175 ms. Bioelectric signals were sent from Grass model 12 amplifiers to the computer for digitization and subsequent storage on compact disks.

**Data Analyses**

Electromyographic activity was full-wave rectified and then signal averaged according to trial type, with averages time locked to reflexogenic stimulus onset. The data were collapsed across presence versus absence of target tone pipes, based on null effects for this variable in a preliminary analysis of the blink data. Trials with excessive EMG activity during the baseline period (e.g., spontaneous blinking) or excessive gape shifts away from the fixation point were excluded from these averages. Because the warning stimulus was turned off after 200 ms, there tended to be some instability in fixation during the baseline period. Overall, 13.6% of the trials were rejected; these rates were approximately equal across trial types.

The size of the signal-averaged blink EMG response was measured by the computer as the mean amplitude within a window of 25–80 ms relative to activity during the 30-ms prestimulus baseline. Poor signal-to-noise ratios in the low intensity conditions for some participants made (cursor-based) manual scoring of the response latencies necessary for blink and postauricular reflex EMG. The point at which the signal-averaged EMG activity first exceeded a level twice that of the baseline activity was scored as response onset. This analysis was done by a technician who was naive to the experimental hypotheses and by one of the authors with similar results (i.e., pattern of effects and p values were the same). VEOG measures of reflexive blinks were also subjected to the above procedures, but in this case, latency was measured by a computer program that found the largest peak and searched backward to the first point in time at which 50% of that value was reached.

Condition means for amplitude and latency measurements were subjected to repeated measures analyses of variance (ANOVAs), with a rejection region of 0.05 adopted throughout. Data were analyzed using 2 × 2 × 3 mixed design ANOVAs, with factors of group (variable or blocked), intensity (97 dB or 70 dB), and foreperiod duration (1,500, 3,000, or 6,000 ms). When analyses were performed on variables with more than two levels, the Greenhouse–Geisser adjusted p value and the epsilon correction factor are reported. However, uncorrected degrees of freedom are given to aid the interpretation of our statistical design.

**Results and Discussion**

**Performance and Phenomenological Report**

Error rates for each trial block were calculated by taking the absolute value of the difference between the number of targets reported and the actual number of targets and then dividing that value by the actual number of targets (×100%). Response bias was calculated similarly but without taking the absolute value of the result. Thus, bias values greater than 100 indicated a tendency to overestimate the number of targets, whereas values less than 100 indicated an average underestimation of the number of tone pips. Participants made fewer errors, F(5,115) = 3.30, ε = 0.67, p < .05, but did not shift their bias, F(5,115) = 2.27, ε = 0.65 (not significant), as trial blocks progressed. For participants in the blocked group, error and bias data could be examined for specific foreperiods. Using foreperiod and half session as factors, there was a significant main effect of foreperiod on bias, F(2,22) = 4.07, ε = 0.74, p < .05. Participants tended to overestimate the number of targets during blocks of trials with longer foreperiods.

Responses to a postexperimental question concerning which foreperiod allowed the best prediction of when the noise burst would occur were compared for participants in the blocked and variable conditions. As expected, participants in the blocked group tended to be more likely than participants in the variable group to report that they could predict stimulus onset more accurately at shorter foreperiods, although this trend was not statistically significant, χ²(2, n = 24) = 3.41.

**Blink Reflex**

Tables 1 and 2 present means and standard deviations for all electromyographic measures. Figure 1 graphically portrays the mean amplitudes and latencies collapsed across group, and Figure 2 shows the grand average EMG waveforms for the variable and blocked groups. The waveform for the middle foreperiod condition lies intermediate between the short and long foreperiod waveforms in this and in all other figures, but has been omitted for the sake of clarity. These signal-averaged EMGs are similar in size, latency, and morphology to those previously published for both the acoustic eyelink and postauricular reflexes (e.g., Hackley, 1993).

Analyses of blink reflex amplitude as measured between 25 and 80 ms documented a main effect of foreperiod duration, F(2,44) = 6.38, ε = 0.56, p < .02, a main effect of intensity, F(1,22) = 24.83, p < .001, and an interaction of foreperiod and intensity, F(2,44) = 3.50, ε = 0.87, p < .05, but no interactions or main effects involving blocked versus variable group. As can be seen in Figures 1 and 2, blinks were reliably larger at longer foreperiods, with this effect being greatest for the high intensity stimuli. This pattern is quite different from that observed for the latency of voluntary responses. However, the amplitude or force of voluntary responses as a function of foreperiod duration has never been studied, so far as we know.

For blink reflex latency, there was a main effect of foreperiod, F(2,44) = 4.42, ε = 0.76, p < .05, a main effect of intensity, F(1,22) = 28.35, p < .001, and an interaction of these variables, F(2,44) = 3.57, ε = 0.97, p < .05, but again, no effect of group. Blink reflex latency was facilitated at longer foreperiods, especially for weak stimuli (see Figures 1 and 2). This pattern is similar to that observed in RT studies, as discussed in the introduction, except for the absence of a Foreperiod × Group interaction. The greater foreperiod effect at high intensities for magnitude but at low intensities for latency is surprising. However, disassociations between magnitude and latency results for the blink reflex have
Table 1. Mean (SD) Reflex Magnitudes (μV) for Eyeblink and Postauricular Reflexes in Experiment 1 as a Function of Foreperiod (FP) and Intensity (dB) of the Reflexogenic Stimulus

<table>
<thead>
<tr>
<th>Reflex magnitude</th>
<th>Variable</th>
<th>Blocked</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>70 dB</td>
<td>97 dB</td>
</tr>
<tr>
<td>Blink, EMG</td>
<td>1.500</td>
<td>2.4 (2.1)</td>
</tr>
<tr>
<td></td>
<td>3,000</td>
<td>3.3 (3.2)</td>
</tr>
<tr>
<td></td>
<td>6,000</td>
<td>5.3 (5.6)</td>
</tr>
<tr>
<td>Blink, VEOG</td>
<td>1.500</td>
<td>23.8 (21.7)</td>
</tr>
<tr>
<td></td>
<td>3,000</td>
<td>32.5 (28.4)</td>
</tr>
<tr>
<td></td>
<td>6,000</td>
<td>41.5 (34.8)</td>
</tr>
<tr>
<td>Postauricular</td>
<td>1,500</td>
<td>3.1 (3.5)</td>
</tr>
<tr>
<td></td>
<td>3,000</td>
<td>3.2 (2.8)</td>
</tr>
<tr>
<td></td>
<td>6,000</td>
<td>2.4 (2.5)</td>
</tr>
</tbody>
</table>

Note: EMG = electromyogram, VEOG = vertical electrooculogram.

been found previously (e.g., Graham & Murray, 1977) and may reflect different circuits responsible for triggering the response versus controlling the response size and duration (Blumenthal & Berg, 1986).

VEOG recordings provided additional evidence regarding modulation of startle blink by foreperiod and intensity. It is now known that VEOG measures of eyelid reflex movement of the eyelid relative to the corneoretinal dipole (Antero, Hari, Katilla, Ryhanen, & Seppanen, 1985; Matsuo, Peters, & Reilly, 1975). As an index of position and movement, the VEOG is sensitive to the absence of eye movement in the tonic activity of the levator palpebrae superioris muscle (which lifts the upper eyelid) and the burst of excitation within m. orbicularis ocu. As was the case with EMG, blink VEOG magnitude (60–160 ms) was relatively enhanced for high intensity noise bursts, F(1,22) = 72.01, p < .001, and for longer foreperiods, F(2,44) = 18.67, e = 0.80, p < .001, regardless of group. Mean VEOG latency was reliably affected by intensity, F(1,22) = 11.07, p < .01, but not by foreperiod or group. The absence of a foreperiod effect and other discrepancies with respect to the EMG data may be due to our particular algorithm for measuring latency (i.e., time to reach 50% of peak amplitude). This method is commonly used in event-related potential research to measure the latency of components with poorly defined onsets, but the algorithm has the drawback that its values are determined by amplitude and latency. Because longer foreperiods were associated with larger blinks, the 50% point took longer to be reached, presumably cancelling the onset latency reduction effect.

Postauricular Reflex

For the mean amplitude of postauricular reflexes, measured from 8 to 30 ms, an effect was found only for intensity, F(1,22) = 41.41, p < .001 (see mastoid EMG in Figure 2). Similar results were obtained for onset latency, with faster responses for stronger stimuli, F(1,16) = 8.41, p < .02. No effect of foreperiod duration was obtained for this trisynaptic reflex, which suggests that variation in foreperiod length does not have a generalized effect on low-level sensory or motor structures (cf. Sanders, 1980).

The discrepancy with respect to foreperiod effects on blink underscores an important general principle: Different components of a complex reflexive behavior such as startle can exhibit distinct patterns of sensitivity to experimental manipulations. That a short-latency component, such as the postauricular reflex, would be sensitive to a narrower range of variables than the (intermediate-latency) eyeblink reflex is not surprising. Long-latency components of startle can exhibit effects not shown by the eyeblink reflex, for example, exaggerated amplitudes in individuals with

Table 2. Mean (SD) Onset Latencies (ms) for Eyeblink and Postauricular Reflexes in Experiment 1 as a Function of Foreperiod (FP) and Intensity (dB) of the Reflexogenic Stimulus

<table>
<thead>
<tr>
<th>Reflex latency</th>
<th>FP (ms)</th>
<th>70 dB</th>
<th>97 dB</th>
<th>Blocked</th>
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<tr>
<td></td>
<td></td>
<td>70 dB</td>
<td>97 dB</td>
<td>70 dB</td>
</tr>
<tr>
<td>Blink, EMG</td>
<td>1,500</td>
<td>38.4 (7.3)</td>
<td>30.0 (5.3)</td>
<td>37.0 (11.9)</td>
</tr>
<tr>
<td></td>
<td>3,000</td>
<td>35.3 (10.1)</td>
<td>31.3 (4.8)</td>
<td>33.6 (6.3)</td>
</tr>
<tr>
<td></td>
<td>6,000</td>
<td>31.6 (4.0)</td>
<td>28.8 (3.7)</td>
<td>33.4 (7.5)</td>
</tr>
<tr>
<td>Blink, VEOG</td>
<td>1,500</td>
<td>70.2 (10.3)</td>
<td>78.2 (3.6)</td>
<td>68.4 (17.3)</td>
</tr>
<tr>
<td></td>
<td>3,000</td>
<td>70.8 (5.9)</td>
<td>76.3 (4.8)</td>
<td>76.3 (9.4)</td>
</tr>
<tr>
<td></td>
<td>6,000</td>
<td>71.8 (7.4)</td>
<td>77.4 (3.3)</td>
<td>75.8 (10.2)</td>
</tr>
<tr>
<td>Postauricular</td>
<td>1,500</td>
<td>12.7 (2.2)</td>
<td>12.4 (1.8)</td>
<td>12.9 (1.6)</td>
</tr>
<tr>
<td></td>
<td>3,000</td>
<td>12.9 (1.8)</td>
<td>11.8 (1.1)</td>
<td>14.6 (4.8)</td>
</tr>
<tr>
<td></td>
<td>6,000</td>
<td>13.0 (2.7)</td>
<td>12.3 (1.3)</td>
<td>12.7 (2.2)</td>
</tr>
</tbody>
</table>

Note: EMG = electromyogram, VEOG = vertical electrooculogram.
Figure 1. Mean eye-blink electromyogram magnitude and latency as a function of acoustic stimulus intensity and foreperiod length for Experiment 1. Because the intensity and foreperiod effects did not differ across groups, the data have been collapsed across groups.

hereditary startle disease (e.g., Brown et al., 1991). Consequently, the finding of normal reflexive eyelids in this disorder does not imply, as Marsden and colleagues have argued (e.g., Brown et al., 1991), that acoustic blink is not a legitimate component of the startle reflex.

EXPERIMENT 2—SIMPLE RT TASK

Experiment 1 demonstrated that blink reflexes are facilitated at long foreperiods regardless of whether foreperiods are manipulated within or between blocks of trials, in contradistinction to the pattern typically observed in RT studies. The second experiment was performed to determine whether this typical pattern (i.e., a reversal in slope of the RT foreperiod function across blocked vs. variable conditions) could be replicated using the same stimuli as in Experiment 1. In addition to voluntary responses, blink reflexes were measured to assess whether the invariance in foreperiod duration effects for blink might be limited to situations in which no explicit motor task is required, as was the case in Experiment 1 and the study by Putnam et al. (1978).

Methods

Participants

The final sample consisted of 24 adult volunteers (10 women, 14 men, aged 18–45 years) who were recruited from an introductory psychology class with the same inclusion criteria as those in Experiment 1. There were 20 right-handed and 4 left-handed participants, according to self-report.

Apparatus and Procedures

The equipment and arrangement of the stimuli were the same as those used in Experiment 1 except that a grip dynamometer (Lafayette Model 76618), with a clamp that rendered the grip bar immovable, was used as a response device. Also, the laboratory was moved to a new building and the room illumination was slightly weaker. The procedures also were the same, except for the participants’ task. During this experiment, participants were required to make a gripping response with their preferred hand as soon as they heard the noise burst. Participants were told to grip the handle abruptly with about 20% of their maximal force. No tone pips were embedded in the white noise bursts of Experiment 2.

Recordings

Forearm EMGs associated with the hand-grip responses were recorded via two surface electrodes placed over the flexor muscles of the forearm, using the standard placement described by Lippold (1967). Blink reflexes were recorded by means of the VEOG method, with the same electrode placement as in the first experiment. The bandpass was 0.01–100 Hz for VEOG and 30–3000 Hz for EMG. Impedances were kept below 10 kΩ for periocular electrodes and below 15 kΩ for the forearm electrodes. Analog-to-digital conversion was carried out at a rate of 700 Hz, with a recording epoch beginning 30 ms prior to reaction stimulus onset.

Figure 2. Grand average reflex electromyogram (EMG) waveforms in Experiment 1 for short (1.5 s) and long (6 s) foreperiod trials with 97-dB startle stimuli for the variable group and blocked group.
Table 3. Mean (SD) Onset Latencies (ms) and Magnitudes (µV) for Eyeblink Reflexes and Voluntary Hand-Grip Reactions in Experiment 2 as a Function of Foreperiod (FP) and Intensity (dB) of the Reflexogenic Stimulus

<table>
<thead>
<tr>
<th>Variable</th>
<th>FP (ms)</th>
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<th>Blocked</th>
<th>70 dB</th>
<th>97 dB</th>
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<tr>
<td>Blink reflex magnitude, VEOG</td>
<td>1,500</td>
<td>12.5 (12.8)</td>
<td>56.6 (49.6)</td>
<td>7.9 (12.7)</td>
<td>37.2 (12.7)</td>
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<td>3,000</td>
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<td>55.4 (50.6)</td>
<td>11.0 (15.4)</td>
<td>44.8 (42.5)</td>
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<td></td>
<td>6,000</td>
<td>16.1 (18.4)</td>
<td>77.0 (62.3)</td>
<td>9.4 (8.6)</td>
<td>55.3 (52.5)</td>
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<td>Blink reflex latency, VEOG</td>
<td>1,500</td>
<td>46.3 (14.3)</td>
<td>39.1 (10.8)</td>
<td>47.3 (16.2)</td>
<td>44.5 (11.2)</td>
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<tr>
<td></td>
<td>3,000</td>
<td>45.4 (13.2)</td>
<td>38.1 (13.8)</td>
<td>51.8 (18.3)</td>
<td>41.1 (13.8)</td>
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</tr>
<tr>
<td></td>
<td>6,000</td>
<td>49.3 (12.6)</td>
<td>32.0 (11.0)</td>
<td>49.1 (10.9)</td>
<td>41.6 (11.2)</td>
<td></td>
</tr>
<tr>
<td>Forearm response magnitude</td>
<td>1,500</td>
<td>32.6 (11.3)</td>
<td>40.5 (13.2)</td>
<td>24.9 (12.0)</td>
<td>33.8 (16.8)</td>
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<td>34.6 (14.4)</td>
<td>40.3 (15.5)</td>
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<tr>
<td></td>
<td>6,000</td>
<td>34.2 (14.5)</td>
<td>38.8 (13.9)</td>
<td>28.4 (16.2)</td>
<td>35.1 (17.3)</td>
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<td>Forearm response latency</td>
<td>1,500</td>
<td>229.8 (61.5)</td>
<td>202.7 (57.1)</td>
<td>180.8 (76.4)</td>
<td>144.0 (48.0)</td>
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<td></td>
<td>3,000</td>
<td>204.7 (43.7)</td>
<td>189.6 (61.9)</td>
<td>172.6 (61.2)</td>
<td>153.8 (66.7)</td>
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</tr>
<tr>
<td></td>
<td>6,000</td>
<td>191.1 (44.2)</td>
<td>162.1 (45.1)</td>
<td>205.7 (77.2)</td>
<td>159.8 (60.6)</td>
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Note: VEOG = vertical electrooculogram.

and lasting for 701 ms poststimulus onset, long enough to capture even slow grip reactions.

Data Analysis
The reduction in the number of data analyzed was conducted in the same manner as in the first experiment. The percentage of trials rejected due to blinks in progress, suspected gaze shifts, or errors of omission in the RT task averaged 10.3%. These rates were approximately equal across trial types. Misses were defined as trials in which no response was initiated prior to 670 ms. There were only four misses across the entire experiment, so error rates were not analyzed. The electrophysiological data were analyzed using 2 × 2 × 3 mixed design ANOVAs, with factors of group (variable or blocked foreperiod), intensity (97 or 70 dB), and foreperiod duration (1,500, 3,000, or 6,000 ms).

Results and Discussion
Forearm EMG
Table 3 presents means and standard deviations for each group, intensity, and foreperiod for all electrophysiological measures. For forearm response magnitude, there was a main effect of intensity, F(1,22) = 40.0, p < .001, such that louder noise bursts apparently elicited more forceful gripping responses.

For voluntary forearm EMG onset latency, there was a main effect of intensity, F(1,22) = 31.7, p < .001, and an interaction of group with foreperiod, F(2,44) = 5.5, p < .02, τ = 0.69, as shown in Figure 3 (top panel) and Figure 4. The interaction was characterized by faster onsets at the short foreperiod for the blocked group and faster onsets at the long foreperiod for the variable group. These findings are consistent with those of many prior studies of overt reaction latency (reviewed in Niemi and Nääätänen, 1981; Requin et al., 1991; Sanders, 1977). These findings indicate that our failure to observe this pattern for the reflexes measured in Experiment 1 was due not to some idiosyncrasy of the particular stimuli or task conditions. For example, the fact that participants were at least mildly startled on each trial apparently did not interfere with their ability to develop temporal expectations and prepare for the reaction stimuli in a normal manner.

Phenomenological Report
The bottom panel of Figure 3 illustrates participants' responses to the postexperimental questionnaire item concerning which of the three foreperiods allowed them to best predict when the noise burst would occur. The reversal in slope across the two groups, S = 2, n = 24 = 87.8, p < .001, conforms to the presumed variation in temporal uncertainty (e.g., Niemi & Nääätänen, 1981; Requin et al., 1991). Note the close correspondence with voluntary EMG latencies shown in the top panel of this same figure.

Blink Reflex (VEOG)
As can be seen in Figures 5 and 6, reflexive blinks were larger at longer foreperiods, F(2,44) = 5.8, τ = 0.62, p < .02, and at the stronger intensity, F(1,22) = 35.9, p < .001. The intensity and foreperiod duration effects on amplitude interacted such that the foreperiod effect was stronger for high intensity stimuli, window 60-160 ms, F(2,44) = 5.3, τ = 0.56, p < .03, which is similar to the pattern observed in Experiment 1. For hand-scored onset latency of the reflex, there was a main effect only for intensity, F(1,20) = 11.4, p < .005, with the stronger intensity yielding quicker blinks. Automated measurement of the 50% fractional peak latencies showed a similar pattern of results. More importantly, these findings corroborated those of Experiment 1 in that the facilitation of blink reflexes at long relative to short foreperiods was independent of whether this variable was manipulated between or within blocks of trials. This pattern stands in stark contrast to the results for voluntary response latency. A serendipitous finding, based on our use of a signal-averaging epoch that extended well past the reflex, offers a possible clue toward explaining this discrepancy.

Spontaneous Posttrial Blinking
The plateau in Figure 6 follows the eyeblink reflex and extends to the end of the recording epoch at 700 ms. The mean amplitude of this plateau was greater for participants in the blocked group than those in the variable group, blocked = 68.4 µV, variable = 20.6 µV, F(1,22) = 5.7, p < .02. Inspection of the single trial data indicates that the flattened morphology is an artifact of signal averaging. This extended supraorbital positivity is actually the summation across trials of discrete eyeblinks—not much longer in
Figure 3. (Top) Mean forearm electromyogram onset latency across foreperiods in Experiment 2. (Bottom) Percentages of participants in each group who indicated, on a postexperimental questionnaire, that short, medium, or long foreperiods best allowed them to predict when the noise burst would occur. Both the subjective report and response data indicate that participants in the variable foreperiod group were more prepared for the reaction stimulus to occur at the longest foreperiod, whereas readiness to respond was more evident at the shortest foreperiod for the blocked group.

Figure 4. Signal-averaged forearm electromyogram (EMG) responses in Experiment 2 for short and long foreperiods collapsed across intensities. Consequently, longer foreperiods should be associated with larger and faster blink reflexes.

The difference between the blocked and variable groups may lie in the fact that under blocked conditions, participants could schedule their posttrial spontaneous blinks with a fair degree of accuracy. This “scheduling” produced the plateau of enhanced spontaneous blinking (Figure 6) that began less than 300 ms after reaction stimulus onset. By contrast, participants in the variable group did not know when the foreperiod would end, so they could not schedule their posttrial blinks in advance. Consequently, the suppression lasted longer, a process that resulted in the reduced posttrial spontaneous blinking seen for this group in Figure 6. Responses on a postexperimental questionnaire item support this explanation. When asked, “During the experiment, did your eyes ever feel dry, like you needed to blink?,” participants in the variable group tended to respond “yes,” $\chi^2(1, n = 12) = 5.32, p < .05$, whereas participants in the blocked group were equally likely to respond either way, $\chi^2(1, n = 12) = 1.33$, not significant.

Figure 5. Mean eyeblink magnitude in Experiment 2 as a function of startle stimulus intensity and foreperiod length collapsing across the two groups of 12 participants.
In the previous section, we postulated a need-to-blink hypothesis as a possible explanation for the finding of larger blink reflexes following longer foreperiods. This hypothesis could be tested easily in an experiment in which the participants are required to keep their eyes closed. Electromyographic blink reflexes can still be recorded with the eyes closed (Silverstein, Graham, & Calloway, 1980), but presumably the circuits responsible for suppression of spontaneous blinking would no longer be active. Persistence of the foreperiod effect under these circumstances would falsify the need-to-blink hypothesis. As an alternative hypothesis, a warning stimulus might engage diverse anticipatory processes, with distinct effects on various neural pathways, including those that mediate voluntary and reflexive responses. Lesion studies support this general hypothesis (e.g., Daum et al., 1993).

Assuming that foreperiod effects on reflexes are either absent (as in the case of the postauricular reflex) or are generated by different mechanisms than those which underlie the foreperiod–RT relationship (as in the case of blink), then the existence of a general facilitation of low-level motor pathways is not supported by the present data. This conclusion would be in disagreement with Sanders’ (1980) theory of alerting effects, although that theory was based on studies of overt response latency and was not specific about matters of neural instantiation.

Additional evidence against Sanders’ theory comes from a recent study of event-related brain potentials. Smulders (1993, chap. 3) compared lateralized readiness potentials during a choice RT task in blocks of trials with variable versus constant interstimulus intervals. There were no explicit warning signals, so each reaction stimulus essentially served as the warning stimulus for the next trial. The blocked versus variable manipulation had a 50-ms effect on the time interval from reaction stimulus onset to the onset of the lateralized readiness potential, but had no reliable effect on the time interval extending from onset of the lateralized readiness potential to onset of the movement itself.

In information-processing terms, the lateralized readiness potential is a measure of relatively early motoric processes. For example, this component is sensitive to anticipated response complexity (e.g., Hackley & Miller, 1995) and to some decision-level processes (e.g., Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988). Furthermore, a voluntary response can be aborted after the onset of lateralization but prior to the initiation of EMG activity (e.g., Miller & Hackley, 1992). The point here is that, although advocates of Sanders’ theory might argue that the “motor adjustment” stage at which foreperiod is assumed to have its effects is upstream from the level indexed by brain stem reflexes, this stage is likely to be downstream from the processes indexed by the lateralized readiness potential. Taken together, the reflex and event-related potential data provide compelling evidence against Sanders’ interpretation of foreperiod effects. By default, Posner’s (1978) decision-level interpretation is supported, but direct electrophysiological tests of this hypothesis have not yet been provided.

REFERENCES


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