Differentiation between protective reflexes: Cardiac defense and startle

Abstract

Rise time and duration are two parametric characteristics of the eliciting stimulus frequently used to differentiate among psychophysiological reflexes. The present research varied the duration (study 1) and rise time (study 2) of an intense acoustic stimulus to dissociate cardiac defense and cardiac startle using the eyeblink response as the external criterion of startle. In each study, 100 participants were presented with five white noise stimuli of 105 dB under one of five duration (50, 100, 250, 500, and 1000 ms) or rise time (0, 24, 48, 96, and 240 ms) conditions. Cardiac defense was affected by stimulus duration, present only in the 500- and 1000-ms conditions, but not by stimulus rise time, present in all rise time conditions. Rise time affected blink startle, but did not selectively alter the short latency accelerative component of the heart rate response, thus questioning whether it reflects startle.

Descriptors: Cardiac defense, Startle, Stimulus rise time, Stimulus duration, Eyeblink, Heart rate

Modulation of physiological reflexes, such as startle, by psychological factors has become one of the most frequently used procedures in the study of attention and emotion in humans. Research in this area has mainly focused on one component of motor startle: the eyeblink response. Similar studies on other protective reflexes, such as cardiac defense, are rare. However, examination of the pattern of covariation among protective reflexes, both autonomic and somatic, may help to clarify their functional significance and advance knowledge on their complex modulation by attentional and/or emotional factors (see Acevedo et al., 2005; Hillman, Hsiao-Wecksler, & Rosengren, 2005; Kofler, Müller, Reggiani, & Valls-Solé, 2001; Turpin, Schaefer, & Boucsein, 1999). Such a comparison requires first clarification of the stimulus-response characteristics of the protective reflexes under examination.

Cardiac reflexes have been a focus of psychophysiological interest since the seminal work of Graham and Clifton (1966) on the differentiation between orienting, startle, and defense. According to Graham’s classical model (1979), stimulus intensity, duration, and rise time are the key stimulus parameters to differentiate cardiac orienting (a heart rate deceleration), startle (a heart rate acceleration with peak amplitude between 1 and 3 s after stimulus onset), and defense (a heart rate acceleration with peak amplitude between 3 and 6 s after stimulus onset). Intensity differentiates between orienting (low intensity), on one hand, and startle and defense (high intensity), on the other. Duration and rise time differentiate between startle (shorter duration and rise time) and defense (longer duration and rise time). In Graham’s model, some response characteristics are also key factors to differentiate among cardiac reflexes. In addition to response direction and latency, habituation is said to differentiate orienting and startle (both showing fast habituation) from defense (no habituation or sensitization).

Graham’s model, however, has been the subject of continuous debate and reformulation (Barry & Maltzman, 1985; Cook & Turpin, 1997; Graham, 1997; Graham & Hackley, 1991; Öhman, Hamm, & Hugdahl, 2000; Turpin et al., 1999; Vossel & Zimmer, 1992). One of the critical issues concerns the separation of the cardiac components of startle and defense. Turpin and Siddle (1978, 1983) were the first to question two basic assumptions of Graham’s model: (a) that cardiac defense is a heart rate acceleration peaking between 3 and 6 s after stimulus onset; and (b) that the response shows no habituation. They described a complex pattern of heart rate changes to intense auditory stimulation with two distinct accelerative components, one acceleration of short latency (peak around second 4) and a second acceleration of long latency (peak around second 35). This response pattern showed a rapid habituation, the second acceleration almost disappearing after the first stimulus presentation.

Subsequent research has confirmed Turpin and Siddle’s findings and advanced knowledge about the parametric characteristics and the physiological mediation of the multiphasic cardiac response. It has been shown, for instance, that (a) the response pattern also shows decelerative components after each acceler-
Cardiac defense and startle (Vila and Fernández, 1989); (b) the pattern is elicited by unexpected intense acoustic and electro-cutaneous stimulation, but not by visual stimulation, matched in intensity (Vila, Fernández, & Godoy, 1992); (c) there are marked individual and gender differences (men show a higher long latency acceleration and a lower short latency acceleration than women); (Eves & Gruzelier, 1984; Vila et al., 1992); and (d) the physiological mediating mechanism of the short latency acceleration/deceleration are mainly vagal whereas those of the long latency acceleration/deceleration are mainly sympathetic (Fernández & Vila, 1989; Reyes, Godoy, & Vila, 1993).

Turpin (1986) suggested that the pattern of successive accelerations might reflect the combination of both startle and defense, with the short latency acceleration reflecting startle and the long latency acceleration reflecting defense. Turpin et al. (1999), in the context of a larger study, examined the effect of manipulating the duration (1 s vs. 5 s) and rise time (3 ms vs. 200 ms) of an intense white noise (100 dB) on the eyeblink response (recorded using a video camera) and on the short latency acceleration of the heart rate response within the first 10 s after stimulus onset. They found that faster rise time produced more frequent eyeblinks and a larger heart rate acceleration within seconds 1–2 after stimulus onset, supporting Graham’s model of cardiac startle. However, contrary to Graham’s model, heart rate accelerations measured early (within seconds 1–2) and late (within seconds 3–6) in the short latency time window habituated with stimulus repetition. Turpin et al. (1999) interpreted these data as supporting the notion that the short latency heart rate acceleration to an intense stimulus represents the cardiac component of the startle reflex with two subcomponents: an early one sensitive to stimulus rise time and a late one sensitive to stimulus intensity.

In this study, no data were reported concerning the long latency heart rate acceleration, identified by Turpin as cardiac defense.

The aim of the present studies was to assess the effects of a wider range of stimulus durations (first study) and rise times (second study) on the cardiac responses in the short and long latency windows. The focus of both studies was to test Graham’s and Turpin’s conceptualizations of cardiac startle and cardiac defense using the eyeblink response—recorded through electromyography of the orbicularis oculi muscle—as the external criterion for startle. Both studies presented intense white noise (105 dB) with an interstimulus interval of 96–100 s after a rest period of 10 min. Study 1 manipulated stimulus duration in a between-group design (50, 100, 250, 500, or 1000 ms) maintaining constant stimulus rise and fall times (0 ms). Study 2 manipulated stimulus rise time in a between-group design (0, 24, 48, 96, or 240 ms) maintaining constant the total energy of the stimulus by increasing its base duration (1000 ms) by one-third of stimulus rise time, according to Dallos and Olsen (1964).

It was hypothesized that duration (over 50 ms) should affect cardiac defense but not cardiac startle. According to Graham’s model, longer durations should accentuate the second component of the short latency cardiac response by moving its peak from 1–2 s to 3–6 s. According to Turpin’s model, longer durations should accentuate the long latency heart rate acceleration with peak between 20 and 40 s. On the other hand, stimulus rise time was hypothesized to affect both cardiac startle and defense. According to Graham’s model, longer rise times should decrease the first component (startle) and accentuate the second component (defense) of the short latency cardiac acceleration. According to Turpin’s model, longer rise times should decrease the magnitude of the short latency acceleration (startle) but not the amplitude of the long latency acceleration (defense). Given the rapid habituation of the cardiac response, these effects were specially predicted for the first stimulus presentation. As regards eyeblink startle, results parallel to cardiac startle were predicted: no effects of stimulus duration but clear effects of stimulus rise time.

STUDY 1
Method
Participants
One hundred students, 50 female, with a mean age of 20.37 years (standard deviation = 2.54, range 17–34 years old) volunteered participation. All were students of psychology at the University of Granada and participated in exchange for course credit. None of the participants had auditory or visual deficits or cardiovascular problems and none were under pharmacological treatment. Participants were allocated at random to one of five stimulus duration conditions (50, 100, 250, 500, and 1000 ms) with the restriction that each group contained equal numbers of women and men. (Analyses including the factor Gender were conducted, but are not reported as they did not reveal results pertinent to the present question. The results are available upon request from the last author.)

Apparatus
The following equipment was used: (a) a Grass polygraph, model Rps 7c, with a 7P4 preamplifier-cardio-tachometer to record the electrocardiogram and the heart rate at lead II, using standard Beckman electrodes, and a 7P3C preamplifier-integrator to record raw and integrated EMG from the orbicularis oculi region beneath the left eye using miniature Beckman electrodes; (b) a Coulbourn audio system (modules S81-02, S84-04, S82-24, and S22-18) to generate the white noise (105 dB and instantaneous rise time) and present it binaurally through earphones (Telephonic TDH Model- 49), the intensity of the sound being calibrated with a sound pressure meter (Bruel & Kjaer, model 2235) and an artificial ear (Bruel & Kjaer, model 4153); and (c) an Advantech card (model PCL812PG) with a 12 bit analogue-to-digital converter and with digital input–output functions, run by a Pentium 2 computer, to control the experimental session through the VPM program (Cook, 1994).

Dependent Variables
Cardiac Defense Response
Response pattern. Weighted averaged second-by-second heart rate was obtained from the cardio-tachometer data using the lag-correction procedure outlined by Reyes and Vila (1998). Then the 80 heart rate values following onset of each auditory stimulus were expressed in terms of difference scores with respect to a baseline level (15 s before each trial). To facilitate the statistical analysis, we followed the same procedure used in previous studies: For each participant, the 80 second-by-second heart rate values of each trial were reduced to 10 heart rate values corresponding to the medians of 10 progressively longer intervals: two of 3 s, two of 5 s, three of 7 s, and three of 13 s (Vila et al., 1992). This procedure allows reduction of the cardiac response without altering the response form: a clear pattern of heart rate changes with progressively longer accelerative and decelerative components.
Early and late components of short latency acceleration. Following Turpin et al. (1999), we also scored maximum acceleration within seconds 1–2 (early component) and seconds 3–6 (late component) of short latency acceleration.

Eyeblink Startle

Response magnitude. The raw EMG signal was recorded and amplified using a frequency band of 10–1000 Hz and integrated using a time constant of 75 ms. Sampling rate was set at 1000 Hz between 500 ms before and 1000 ms after stimulus onset. The startle reflex magnitude was defined as the difference in microvolts between the peak of the integrated response and the response onset initiated between 20 and 100 ms after stimulus onset. On trials with no detectable peak, magnitude was scored as zero.

Response onset latency. Response onset latency was measured in milliseconds. On trials with no detectable peak, latencies were scored as missing.

Procedure

Each participant attended a single laboratory session that lasted approximately 60 min. Upon arrival, the participant was seated in an armchair, received information about the experimental session, and signed the informed consent form. The participant was informed that the purpose of the experiment was to record physiological data while she or he was trying to relax during several minutes followed by the presentation of brief loud noises. Then, an interview was conducted in order to ascertain age, visual or auditory deficits, health, and pharmacological treatment. After the specific instructions were read, the skin under the electrodes was cleaned and the electrodes, filled with electrolyte paste, attached, the ground electrode being placed on the right leg. Then the physiological recording was checked, the earphones placed, and the participant left alone in a semi-darkened room. The psychophysiological test consisted of the sequence described above: 10-min rest period followed, without warning, by five presentations of a white noise of 105 dB, instantaneous rise and fall time, and duration depending on the experimental group. Recording started 15 s before each noise presentation and continued for 80 s. Intertrial intervals with no recording varied randomly between 1 and 5 s, finishing with an interval of 120 s after the last trial. During the test, participants were asked to breathe normally and maintain their eyes open looking at a fixation point located at a distance of 3 m in front of the participant’s eye level. After the test, the experimenter removed the earphones and the electrodes and the participant was debriefed and given the credits for his/her participation.

Statistical Analysis

For each dependent variable, data were analyzed using mixed-between-group with repeated-measures ANOVAs applying the Greenhouse-Geisser epsilon correction for the repeated-measures factors. The between-group factor was Duration and the repeated measures factors were Trials (the five stimulus presentations) and, for the analysis of cardiac response patterns, Time (the 10 HR medians). Analysis of significant interaction effects were performed following Keppel’s (1991) procedure. First, we identified the levels of the interacting factors explaining the significant effects (simple effects analysis). Then, if more than two groups were involved, multiple pairwise comparisons were performed using Tukey’s test. When habituation effects were detected (Trials), the analysis always tested the presence of significant Duration effects in the first trial according to the hypothesis. The level of significance was set at .05 for all analyses.

Results

Cardiac Defense

Response pattern. The Duration (5) × Trials (5) × Time (10) ANOVA, with one between-group factor, Duration, and two repeated-measures factors, Trials and Time, yielded two main effects—Duration, F(4,95) = 3.88, p = .006, and Time, F(9,855) = 33.0, p < .0001—and three interaction effects—Duration × Trials, F(16,380) = 2.72, p = .002, Time × Trials, F(36,3420) = 12.98, p < .0001, and Duration × Trials × Time, F(144,3420) = 1.66, p = .001.

Figure 1A displays the response pattern seen on the first trial as a function of Duration, and Figure 1B–F display the changes in response pattern across trials in each of the five duration conditions. In response to the first stimulus presentation, all groups showed a short latency acceleration with a maximum at median 1 (seconds 1–3) followed by a deceleration. The long latency acceleration with maximum peak at median 7 (seconds 31–37) was only evident in the two groups with longer durations (500 and 1000 ms). All groups show evidence of habituation with stimulus repetition. Analysis of the Duration × Trials × Time interaction yielded significant Duration × Time interactions only on trials 1, F(36,855) = 2.07, p = .005, and 2, F(36,855) = 2.04, p = .002.

On trial 1, significant Duration effects were found in medians 1, F(4,95) = 3.52, p = .01, and 2, F(4,95) = 2.61, p = .04, corresponding to the short latency acceleration, and 5, F(4,95) = 3.10, p = .02, 6, F(4,95) = 6.03, p = .0002, 7, F(4,95) = 6.59, p = .0001, and 8, F(4,95) = 5.89, p = .0003, corresponding to the long latency acceleration. Pairwise comparisons showed a significant increase for medians 1 and 2 in the two longest durations (500 and 1000 ms), compared to the shortest duration (50 ms). For medians 6, 7, and 8, the significant increases were in the two long durations (500 and 1000 ms), compared to each of the three short durations (50, 100, and 250 ms).

On trial 2, significant Duration effects were found for medians 2, F(4,95) = 5.09, p = .0009, and 8, F(4,95) = 2.92, p = .03. Pairwise comparisons showed significant increases for median 2 in the longest duration (1000 ms) compared to the two shortest durations (50 and 100 ms), and for median 8 in the 500 ms compared to the 100 ms duration.

Early and late components of short latency acceleration. The Duration (5) × Trials (5) ANOVA on the early component yielded a significant main effect for Trials, F(4,360) = 6.13, p < .0001. No main or interaction effect of Duration was found. The first component was larger on trial 1 (mean = 5.8 bpm) than on the rest of the trials (mean range = 2.8–3.8 bpm). The Duration (5) × Trials (5) ANOVA of the late component yielded significant effects for Duration, F(4,95) = 3.28, p = .015, and Trials, F(4,380) = 30.96, p < .0001. This component was larger on trials 1 (mean = 11.82 bpm) and 2 (mean = 7.36 bpm) than on the rest of the trials (mean range = 5.22–5.87 bpm). Post hoc analysis for the duration effect showed significant differences only between the 500 ms (mean = 9.45 bpm) and the 50 ms (mean = 4.80 bpm) durations.

Eyeblink Startle

Response magnitude. The Duration (5) × Trials (5) ANOVA yielded no significant main or interaction effects, all Fs < 0.46, p > .87. Figure 2 displays the magnitude of the eyeblink response across trials for each duration group. As can be observed, there was no clear pattern of changes in response magnitude as a
function of stimulus duration. The lack of habituation (in only five trials) is also evident in Figure 2.

Discussion

The manipulation of the duration of a white noise of 105 dB and instantaneous rise time, within the range of 50–1000 ms had a marked effect on the heart rate response. Analyses of the cardiac response pattern showed that duration affected both the short and the long latency accelerations. On the first trial, the short latency acceleration was greatest for the two longest durations (500 and 1000 ms) and smallest for the shortest duration (50 ms), with the intermediate durations showing intermediate amplitudes. In all conditions, the peak amplitude of the short latency acceleration occurred in median 1 (seconds 1–3). All groups also showed a second decelerative component after the long latency acceleration (in the two groups with the

Figure 1. Cardiac response as a function of stimulus duration and trials. Each point corresponds to the 10 medians along the 80-s poststimulus period.
longest durations) or after the return of the short latency deceleration toward the baseline level (in the three groups with the shortest durations). Habituation was evident for both the short and the long latency accelerations, the speed of habituation being faster for the long latency acceleration. The analysis of specific components of the short latency acceleration confirmed the above findings and, in addition, showed that (a) there was no significant duration effect on the early component (seconds 1–2) of the short latency acceleration and (b) the significant duration effects were all concentrated on the late component of the short latency acceleration (seconds 3–6).

These results not only confirm the characteristics of cardiac defense reported in previous studies, but also have implications for the distinction between cardiac startle and defense as proposed by Graham (1979) and Turpin (1986). Both suggested that, with intense stimuli of instantaneous rise time, all durations would elicit cardiac startle (a habituating acceleration between 1–2 s) whereas longer duration would increase the probability of observing cardiac defense (a nonhabituating acceleration between 3–6 s, according to Graham, and a habituating acceleration around second 35, according to Turpin). Our results clearly support Turpin’s proposal, that the long latency component reflects cardiac defense. The results are inconsistent, however, with the interpretations of the short latency component offered by either Graham or Turpin. The finding that stimulus duration affects the late component of the short latency acceleration seems consistent with Graham’s proposal that it may reflect defense. However, contrary to Graham’s proposal, this component displayed habituation. Turpin et al. (1999) interpret the short latency acceleration as part of the startle reflex rather than part of the defense reflex based on the observation that this component habituates. However, this interpretation seems also questionable, as the second index of startle, the blink reflex, failed to show similar habituation across trials.

As predicted, duration had no effect on eyeblink startle, suggesting that a startle reflex was present in all stimulus duration conditions (all conditions had instantaneous rise time and a minimum of 50 ms duration). Eyeblinks did not differ across duration groups with no evidence of habituation, a finding that is inconsistent with Turpin et al.’s interpretation of the short latency cardiac acceleration as startle. However, no firm conclusion can be drawn as to the interpretation of this cardiac acceleration because no experimental manipulation of startle was performed in this study, and it may be possible that different component of the startle reflex differ in their susceptibility to habituation. The second study was specifically designed to manipulate the elicitation of the startle response by variation of stimulus rise time.

STUDY 2

Method

Participants

The participants in this study were 100 student volunteers (50 female) who had not participated in Study 1. Their mean age was 21.88 (standard deviation = 3.68, range: 17–41 years). All were students of psychology at the University of Granada and participated in exchange for course credit. The selection criteria were the same as in Study 1.

Design and Procedures

Participants were allocated at random to one of five stimulus rise time conditions (0, 24, 48, 96, and 240 ms) with the restriction of equal numbers of men and women in each group. The stimulus was the same as in the previous study: a white noise of 105 dB intensity. The base duration of the stimulus was 1000 ms. This base duration was increased, in order to maintain constant total stimulus energy, according to Dallos and Olsen’s (1964) formula:

\[ T = r/3 + P, \]

where \( T \) represents total energy, \( r \) represents rise time, and \( P \) represents base duration. Stimulus fall time was maintained constant at 0 ms for all conditions. This was achieved by experimentally controlling the output gate of the stimulus generator (Coulbourn audio system) and closing the output gate while the stimulus was presented at peak intensity. All other methodological aspects of Study 2 replicated Study 1.

Results

Cardiac Defense

Response pattern. The Rise time (5) × Trials (5) × Time (10) ANOVA, with one between-group factor, Rise time, and two repeated-measures factors, Trials and Time, yielded two main effects—Trials, \( F(4,380) = 8.58, p < .0001 \), and Time, \( F(9,855) = 30.12, p < .0001 \)—and one interaction effect—Trials × Time, \( F(36,3420) = 14.21, p < .0001 \). Rise time did not show any significant effect.

Analysis of the Trials × Time interaction (see Figure 3A) revealed significant habituation effects in medians 1, \( F(4,396) = 13.29, p < .0001 \), and 2, \( F(4,396) = 20.92, p < .0001 \), corresponding to the short latency acceleration, median 4, \( F(4,396) = 3.07, p = .03 \), corresponding to the first deceleration, medians 5, \( F(4,396) = 7.21, p < .0001 \), 6, \( F(4,396) = 18.56, p < .0001 \), 7, \( F(4,396) = 25.64, p < .0001 \), and 8, \( F(4,396) = 8.40, p < .0001 \), corresponding to the long latency acceleration, and median 10, \( F(4,396) = 9.09, p < .0001 \), corresponding to the second deceleration.

Given these habituation effects, we performed an independent analysis on the first trial in order to confirm the lack of rise time effects on the response pattern. The Rise time (5) × Time (10) ANOVA yielded a main effect for Time, \( F(9,855) = 32.62, p < .0001 \), but the other factor, and the interaction were not significant, \( F < .8, ps > .77 \) (see Figure 3B).
Early and late components of short latency acceleration. The Rise Time \((5) \times \text{Trials} (5)\) ANOVA of the early component yielded a significant main effect for Trials, \(F(4,380) = 7.62, p < .0001\). Amplitude was greater on trial 1 (mean = 7.36 bpm) than on the remaining trials (mean range: 2.98–4.46 bpm). Similarly, the analysis of the late component yielded a main effect for Trials, \(F(4,380) = 40.78, p < .0001\). Amplitude was greater on trial 1 (15.64 bpm) than on the remaining trials (mean range: 6.3–8.0 bpm). The specific test of rise time effects on trial 1 revealed no significant effects for the early, \(F(4,95) = 0.21, p = .93\) nor the late, \(F(4,95) = 0.50, p = .74\), component.

**Eyeblink Startle**

Response magnitude. The Rise Time \((5) \times \text{Trials} (5)\) ANOVA yielded a significant main effect for Rise Time, \(F(4,95) = 7.11, p = .003\). None of the other main and interaction effects were significant. Figure 4 displays the magnitude of the eyeblink response as a function of Rise Time and Trials. The magnitude of the response decreased linearly, after 24 ms, with increasing rise times. No evidence of habituation was observed within each rise time group. Pairwise comparisons showed significant differences between the longest rise time (240 ms) and all other groups, and between the 48 and the 0 ms rise times.

Discussion

The manipulation of the rise time of a white noise of 105 dB, within the range of 0–240 ms, maintaining constant its total energy (equivalent to 1000 ms duration with instantaneous rise–fall time) did not have any significant effect on the cardiac response. All rise time groups reproduced on the first trial the typical cardiac response pattern observed in Study 1: a short latency acceleration followed by a deceleration and a long latency acceleration followed by a second deceleration. Peak amplitude of short latency acceleration occurred in medians 1 (seconds 1–3) or 2 (seconds 4–6). Peak amplitude of long latency acceleration occurred in all groups in median 7 (seconds 31–37). Habituation was evident for all cardiac components, the speed of habituation being slower for the short latency acceleration. Analysis of the specific parameters of the short latency acceleration confirmed the absence of rise time effects in the early and late components.

The present results do not confirm the predictions, derived from Graham’s and Turpin’s models, concerning elicitation of cardiac defense in response to the first stimulus. It was predicted that longer rise times would decrease and delay the amplitude of the short latency acceleration—the cardiac component sensitive to startle—not affecting either its late component (Graham, 1979; Turpin et al., 1999) or the long latency acceleration (Turpin, 1986; Turpin et al., 1999). Whereas, as predicted, rise time did affect the eyeblink response, suggesting a reduction in the motor component of startle with increasing rise time, no effect was observed in the cardiac response. Therefore, neither Graham’s nor Turpin’s hypothesis concerning the short latency acceleration were confirmed in this study. Turpin’s prediction concerning the long latency acceleration was, however, confirmed: Manipulation of rise time did not reduce the amplitude of the long latency accelerative component.

The differences between the present and Turpin et al.’s (1999) results concerning the effects of rise time may reflect three major methodological differences: the control of stimulus total energy, averaging across trials, and interstimulus interval. It is possible that the rise time effect reported by Turpin et al. (1999) using a white noise of similar duration to ours (1000 ms) might be due to
differences in total energy of the stimulus. In that study, the duration of the stimulus was not increased as a function of increasing rise time (from 5 ms to 200 ms). Therefore, the greater energy of the shorter rise time condition could explain why it elicited greater amplitude. Two further differences are that data from single trials were analyzed in the present study, whereas in Turpin et al.’s study analyses were performed on blocks of three trials, and the interstimulus interval was also longer in the present study (98 s vs. 45 s as average). A reanalysis of our results limited to the 0 ms and 240 ms rise time groups—the two closest to Turpin et al.’s—showed a general tendency to display a smaller early component amplitude in later trials with increasing rise time, as predicted by both Graham and Turpin. Regarding the interstimulus interval, an interval shorter than 80 s may significantly affect the response pattern, because the second acceleration–deceleration may not be fully recovered, producing superposition of responses.

The absence of a rise time effect on the cardiac response on the first trial questions its subdivision into startle and defense components as proposed by Graham or Turpin. The short and long latency cardiac accelerations were not affected by rise time, and the full response pattern was present even when the eyeblink was absent or markedly reduced. Thus, it seems difficult to associate the short latency acceleration (with its two subcomponents) with startle and the long latency acceleration with defense, as suggested by Turpin et al. (1999), or to associate the early subcomponent of the short latency acceleration with startle and its late subcomponent with defense, as suggested by Graham (1979). Rather, it seems that both the short and the long latency accelerations (with their decelerative components) form part of the defensive reaction to an unexpected intense noise of sufficient duration.

The functional significance of this complex response pattern is still a subject of debate. Based on the cascade model of defense proposed by Lang and colleagues (Lang, Bradley, & Cuthbert, 1997; Lang, Davis, & Öhman, 2000), we have suggested (Vila, Fernández, Pegalajar, & Sánchez, 1997; Pérez, Fernández, Vila, & Turpin, 2000) that cardiac defense reflects the combination of both attentional and motivational processes: The short latency acceleration–deceleration represents an attentional component of the cardiac defense response, aimed at detection and analysis of the potential danger, and the long latency acceleration–deceleration represents its motivational component, aimed at active defense such as fight or flight. This sequential process interpretation of cardiac defense is similar in many respects to that proposed by Turpin (1986), Cook and Turpin (1997), and Turpin et al. (1999), who suggested that the short latency acceleration reflects the operation of an attentional protective system linked to startle (disengagement from ongoing activity) and the long latency acceleration reflects the operation of a motivational protective system (the fight and flight response).

The findings of our two studies have additional methodological implications concerning comparison between autonomic (cardiac) and motor (eyeblink) reflexes. Both reflexes can be elicited by intense acoustic stimulation, but the parametric characteristics of the eliciting stimulus seem to be different. The pattern of cardiac defense requires an eliciting stimulus of long duration (around 500 ms and over) but no specific rise time. In contrast, eyeblink startle relies on very short stimulus rise times, and therefore be elicited with either short or long duration stimuli (50 ms and over). By manipulating the duration and rise time of the stimulus it is possible to evoke (a) eyeblink alone (using stimulus of short duration and short rise time), (b) cardiac defense alone (using stimulus of long duration and long rise time), and (c) both eyeblink and cardiac defense (using stimulus of long duration and short rise time).

Cardiac defense, however, may also be affected by factors other than stimulus duration and rise time. In Turpin et al.’s (1999, p. 445) study, no data on the long latency acceleration were reported because “provisional inspection of the raw data revealed no evidence of long-latency responses.” Methodological factors that have been demonstrated to reduce the probability of the long latency acceleration include high level of room illumination, lower stimulus intensity, shorter resting period prior to the first trial, shorter interstimulus interval, predictability of the stimulus, and unbalanced gender of participants (see Fernández & Vila, 1989; Robles, Fernández, Pérez, & Vila, 1994; Sánchez, Fernández, López, & Vila, 2002; Vila et al., 1992). Some of these factors could affect the observation of the long latency acceleration in the study of Turpin et al. (1999).

In conclusion, the differential effects of stimulus duration (between 50 and 1000 ms) and rise time (between 0 and 240 ms) on the elicitation of the cardiac response by an intense white noise question the proposal that some cardiac components of this response are part of the startle reflex (as indexed by the eyeblink response). Rather, eyeblink and cardiac response seem to be independent. The cardiac response shows a complex pattern that is markedly affected by stimulus duration (between 50 and 1000 ms) but not by stimulus rise time (between 0 and 240 ms). The opposite is true for the eyeblink response. However, by use of appropriate stimulus parameters (long stimulus duration and short rise time) both types of reflexes can be simultaneously elicited allowing comparative studies on their simultaneous or independent modulation by attentional and/or emotional factors.

REFERENCES


Bart, R. J., & Maltzman, I. (1985). Heart rate deceleration is not an orienting reflex; heart rate acceleration is not a defense reflex. Paylo-


(Received January 18, 2005; Accepted August 30, 2005)