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## Cardiac and electro-cortical responses to performance feedback reflect different aspects of feedback processing

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**Cardiac and electro-cortical data were collected from participants performing a probabilistic learning task, in which stimuli were mapped to a left or a right hand response. Participants had to infer the appropriate mappings by trial-and-error, using trial-to-trial performance feedback. Negative feedback was followed by a relative deceleration of heart rate, and by an “error-related negativity” (ERN) at the scalp, suggesting that these two measures may reflect the activity of the same performance monitoring mechanism. However, we found no evidence of a correlation between the degree of cardiac deceleration and ERN amplitude. The two measures also appeared to be differentially sensitive to our task manipulations. Thus, our data suggest that cardiac deceleration and the ERN reflect different aspects of feedback processing.**

### Introduction

Performance monitoring has recently received much attention in the psychophysiological literature. This started with the discovery of a negative event-related brain potential (ERP) component directly following the commitment of an error, the error-related negativity (ERN, Gehring et al., 1993, or Ne, Falkenstein et al., 1991). The ERN is most clearly visible in response-locked ERPs, peaks almost directly after the erroneous response (80-100 ms), and is maximal at fronto-central midline electrodes. The source of the ERN is thought to be located in the anterior cingulate cortex (Carter, et al., 1998; Dehaene et al., 1994).

According to the error detection hypothesis, the ERN is a reflection of the outcome of the comparison between representations of the actual response and the appropriate response (e.g., Coles et al., 2001). The ERN is elicited when there is a mismatch between these two representations, and the amplitude of the ERN depends on the degree of mismatch. An ERN-like negativity is also elicited when subjects receive negative performance feedback (Holroyd & Coles, 2002; Miltner, Braun, & Coles, 1997; Nieuwenhuis, et al., 2002). The observation of this feedback-related negativity is not incompatible with the error detection hypothesis of the ERN, but does call for a slightly broader interpretation: Holroyd and Coles (2002) have proposed a theory that provides an integrative account of the ERNs observed following response errors and negative feedback. According to this theory, the ERN reflects a reward prediction error signal that is elicited by the monitoring system when it detects that ongoing events are suddenly worse than expected. If the response represents the earliest predictor of negative outcome, the ERN is elicited following the incorrect response. In contrast, if the feedback itself is the earliest predictor of negative outcome, the ERN occurs following negative feedback.

Cardiac responses to feedback have only recently received some attention (Somsen et al., 2000; Van der Veen et al., 2004; Crone et al., in press). In general it has been found that negative feedback is associated with larger cardiac slowing than positive feedback. Cardiac slowing following response errors has also recently been reported (Hajcak et al., 2003). The finding that cardiac measures are sensitive to performance feedback and errors suggests the possibility that these measures and the ERN reflect the operation of the same performance monitoring mechanism. The goal of the current study was to further examine cardiac responses to feedback and errors and to examine the relation between cardiac slowing and the ERN. We used a modified version of the probabilistic learning task used by Holroyd and Coles (2002; see also Crone et al., in press). The task is described in detail by Nieuwenhuis et al. (2002), who reported the ERP results of this study. Here we will report the cardiac results and relate them to the ERP results. Participants were required to learn different stimulus-response mappings using trial-to-trial performance feedback, indicating whether the chosen response was correct or incorrect. Three experimental conditions were defined in terms of the degree to which particular stimulus-response combinations were predictive of the feedback outcome. In the *100% condition*, a particular stimulus-response combination was followed by the same (positive or negative) feedback stimulus on 100% of the trials, allowing participants to learn these mappings. In the *50% condition*, the feedback stimulus was randomly chosen (50% positive, 50% negative), irrespective of the particular stimulus-response combination. Thus, the feedback was not contingent on the subject's behavior. Finally, an intermediate condition was provided by the *80% condition*; that is, a particular stimulus-response ensemble was followed by one type of feedback on 80% of the trials and by the other type of feedback on 20% of the trials.

As reported in detail by Nieuwenhuis et al. (2002), the ERN results were in accordance with the theory of Holroyd and Coles (2002). The amplitude of the response-related ERN increased as a function of the extent to which the response in a particular condition was predictive of the feedback value. That is, the response-related ERN was largest in the 100% condition, in which subjects learned to recognize errors at the time of the response, and it was absent in the 50% condition, in which negative feedback could not be foreseen at the time of the response. The feedback-related ERN showed the opposite pattern: It was largest in the 50% condition, in which the feedback itself

constituted the earliest predictor of negative outcome. If the cardiac response described above reflects the same process as the ERN, it should show a comparable differential sensitivity to errors and feedback in the three experimental conditions. This hypothesis was tested by examining cardiac responses to errors and feedback separately, and by computing separate between-subject correlations between response-related and feedback-related ERN and the cardiac responses.

### **Materials and Methods**

We limit ourselves to summarizing the most essential aspects of the employed methods. Further details of the methods can be found elsewhere (Nieuwenhuis, et al., 2002).

#### *Participants*

Sixteen young adults (8 women) participated in this experiment. The participants, ranging in age from 18 to 23 years ( $M = 20.4$ ), were undergraduate students at the University of Amsterdam and received course credits for their participation.

#### *Design and Procedure*

In each of five blocks of 300 trials, participants were presented with a set of six imperative stimuli presented in a quasi-random order. Participants were instructed to respond to each stimulus with a left or right hand response within 700 ms following the stimulus. They were not told the stimulus-response mappings in advance but were instructed to learn these on the basis of a feedback stimulus that was presented 1000 ms following the stimulus. In case of a correct response, a designated *positive feedback* stimulus indicated that the participant had earned 2 cents on that trial. In case of an incorrect response, a *negative feedback* stimulus indicated a penalty of 2 cents. A third feedback stimulus was presented in case a response missed the deadline (penalty of 4 cents). Two stimuli were consistently mapped to the left or right hand response (*100% condition*), so that participants could learn to control the value of the feedback. For two other stimuli, feedback was delivered randomly (*50% condition*), regardless of the given response. As a result, participants were rewarded on 50% of the trials and penalized on 50% of the trials. Following the same logic, the two remaining stimuli were associated with an *80% condition*. That is, one stimulus required a left button press on 80% of the trials (referred to as *valid* trials) and a right button press on 20% of the trials (*invalid* trials), and the other stimulus required a right button press on 80% of the trials (*valid*) and a left button press on 20% of the trials (*invalid*). At the end of the each block, participants were informed about the total amount of bonus money they had earned throughout the experiment.

#### *Recordings and Data Reduction*

For each participant, response-locked and feedback-locked ERP waveforms were computed separately for each condition. Difference waves were computed by subtracting the ERPs associated with positive feedback from the ERPs associated with negative feedback. The amplitude of the response-locked ERN was defined as the lowest value in the difference wave at Cz in the time window of 0-150 ms following the response. The amplitude of the feedback-locked ERN was defined as the peak value in the difference wave at Cz in the time window of 200-350 ms following feedback onset.

The ECG was derived from pre-cordial leads and sampled at 250 Hz. The R-peak occurrence times were detected with an accuracy of 4 ms and stored off-line. The series of R-peaks was checked for artifacts and corrected when necessary. Four inter-beat intervals (IBIs) were selected around the presentation of the feedback stimulus (FB-locked) and the response (R-locked): the concurrent IBI (i.e., IBI0), the IBI preceding the stimulus (IBI-1), and the two IBIs following the stimulus (IBIs 1 and 2). Cardiac responses were referenced to the fourth IBI preceding the onset of the imperative stimulus.

#### *Statistical Analysis*

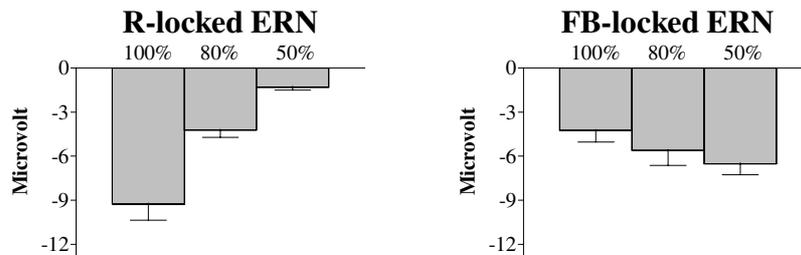
Cardiac measures were statistically evaluated with a repeated-measures analysis of variance (ANOVA) with sequential IBI (4 levels: IBI-1, IBI0, IBI1, and IBI2), type of feedback (2 levels: positive vs. negative feedback), condition (3 levels: 50%, 80%, 100%), and block half (2 levels: first half vs. second half) as within-subjects factors. Because the effects of block half were minor

and nonsystematic, statistical main and interaction terms involving this factor will not be mentioned in the Results section. Where appropriate, the statistical degrees of freedom were adjusted using the Greenhouse-Geisser correction. For all analyses, a difference was considered statistically significant when the p-value was equal or less than .05. Data from invalid trials of the 80% condition were not included in the reported analyses due to insufficient observations. Relationships between heart rate and ERN amplitude were examined by computing Pearson-moment correlations across individual participants.

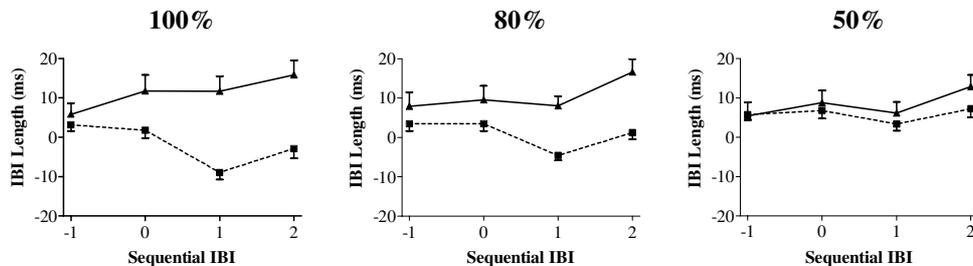
## Results

### Performance and ERN

Performance and ERN results have been described elsewhere in detail (Nieuwenhuis, et al., 2002). To summarize, accuracy was highest in the 100% condition (79% correct), intermediate in the 80% condition (73%) and at chance level in the 50% condition (49%). Amplitudes of R-locked and FB-locked ERNs are shown in Figure 1. Both were affected by condition, resulting in largest amplitudes of the R-locked ERN in the 100% condition, intermediate in the 80% condition, and lowest in the 50% condition. The opposite pattern was found for FB-locked ERN.



**Figure 1.** Response-locked and feedback-locked ERN amplitude ( $\pm$ SEM) for each of the three experimental conditions. ERN is computed as the difference wave between ERPs to positive feedback/correct response and negative feedback/error.



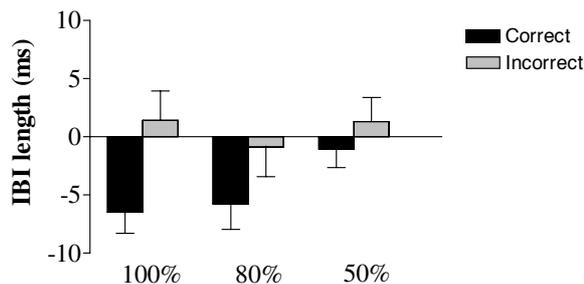
**Figure 2.** Feedback-locked cardiac responses ( $\pm$ SEM) to negative and positive feedback for each of the three experimental conditions.

### Cardiac Responses

The cardiac responses to both types of feedback are shown in Figure 2. Statistical analyses revealed a main effect of sequential IBI,  $F(1,15)=17.0$ ,  $p<.001$ , and type of feedback,  $F(1,15)=17.00$ ,  $p<.005$ . Furthermore, it was found that type of feedback and sequential IBI interacted significantly,  $F(2,31)=6.7$ ,  $p<.005$ . Condition interacted significantly with type of feedback,  $F(2,30)=3.9$ ,  $p<.05$ , with sequential IBI,  $F(2,26)=14.6$ ,  $p<.001$ , and entered in a significant three-way interaction with type of feedback and sequential IBI,  $F(3,45)=5.6$ ,  $p<.005$ . The three-way interaction effect indicates that the difference between negative and positive feedback observed was largest and

earliest in the 100% condition, smaller and later in the 80% condition, and nearly absent in the 50% condition. Post-hoc analyses showed that in the 100% condition, type of feedback affected IBIO, IB11 and IB12. In the 80% condition, only IB11 and IB12 were affected, and in the 50% condition only IB12. Moreover, separate tests for positive and negative feedback trials revealed that condition only affected the cardiac response to *positive* feedback. In the 100% condition we found the shortest IBIs following positive feedback, in the 80% condition IBIs were longer, and in the 50% condition IBIs were longest.

Because, on average, the response preceded the feedback by around 600 ms, it is possible that the FB-locked responses were slightly confounded with response-related cardiac effects. It is possible to get an estimate of these response related cardiac effects, which is not affected by feedback related processes. This can be achieved by analyzing those IBIs following the response that can *not* be influenced by the feedback. Previous research has shown that only relevant events timed during roughly the first third of the inter-beat interval can influence the length of this interval (Jennings & Wood, 1977; Somsen, van der Molen, Jennings, & Orlebeke, 1985). For R-locked cardiac responses this means that we can analyze the interval in which the response is given (IB10) and the following interval (IB11), unless the feedback stimulus is presented in the first third of this interval. Thus we computed an average of IB10 and IB11 as compared to the IB1-4 baseline (see Figure 3). An ANOVA with condition and type of feedback as within-subjects factors, revealed a significant main effect type of feedback,  $F(1,15)=4.8$ ,  $p<.05$ . This effect reflects the relatively longer IBIs following errors.



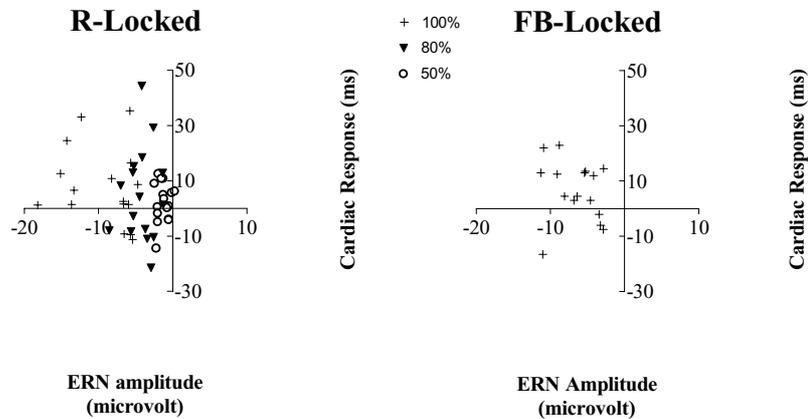
**Figure 3.** Response-locked cardiac responses ( $\pm$ SEM) to those responses that can not be influenced by feedback-locked processes (see text for details), averaged over IB10 and IB11. Correct responses are responses followed by positive feedback and incorrect responses are responses followed by negative feedback.

Based on the results of the FB-locked data, in which it was found that only cardiac responses to positive feedback were affected by condition, we performed separate analyses on positive and negative feedback with condition as factor. The analysis of positive feedback revealed an effect of condition,  $F(2,27)=7.8$ ,  $p<.005$ , showing a decrease in cardiac acceleration from the 100% to the 50% condition. The analysis of cardiac responses to negative feedback did not reveal any significant effects.

*Psychophysiological relations*

We performed a number of correlational analyses to assess the relation between IBIs and ERNs. For the R-locked responses we used that part of the cardiac response that could not be affected by FB-locked processes (see previous paragraphs), and computed a difference score between responses to negative and positive feedback. For the FB-locked responses, we only analyzed the 50% condition, which is the only condition in which all differences can be attributed to the feedback stimulus. No significant correlations between the ERN and equivalent cardiac measures were found ( $r=.23$  for FB-locked responses, and  $r=-.21$ ,  $.06$  and  $.07$  for R-locked responses in the 100%, 80% and 50% condition respectively;  $p>.1$  for all correlations). FB-locked and R-locked ERNs plotted against the equivalent cardiac responses are shown in Figure 4. This figure shows that FB-locked responses appear to show some kind of association if the outlier with a strongly

negative value for both measures is ignored. If this case is removed from the sample, the correlation between FB-locked ERN and cardiac responses reaches significance,  $r=-.604$ ,  $p=.017$ .



**Figure 4.** Cardiac responses plotted against ERN. Cardiac responses were computed as the difference between negative and positive feedback, averaged over IB10, IB11, and IB12.

Finally, the same variables were used to perform a median split analysis on the basis of the amplitude of the ERN, resulting in two groups with either a high ( $N=8$ ) or low ( $N=8$ ) ERN amplitude. These groups did not differ with respect to cardiac responses.

### Discussion

The current study examined cardiac responses to feedback and errors and the relation to electrocortical responses. Most importantly, the data point to a dissociation between electrocortical and cardiac responses to feedback and errors. The ERN showed the expected variation in amplitude that was compatible with the theory of Holroyd and Coles (2002), in which the ERN is seen as a reflection of a negative reward prediction error signal arriving in ACC. Cardiac responses were consistent with the results reported previously (Crone et al., in press; Hajcak et al., 2003; Somsen et al., 2000; van der Veen et al., 2004) by showing that negative feedback is associated with cardiac slowing relative to the heart rate response to positive feedback. Interestingly, condition only affected the response to positive feedback, which contrasts markedly with modulations of the ERN, which are mainly seen following error responses and error feedback. Combined with the general lack of association between both physiological measures, the current findings strongly suggest that electrocortical and cardiac measures reflect different aspects of performance monitoring.

The FB-locked cardiac responses were comparable to those reported by Crone et al. (in press). Negative feedback stimuli evoked the predicted enhanced and prolonged cardiac deceleration. This enhanced deceleration was visible in all three conditions but, as in the Crone et al. study, differences between cardiac responses to positive and negative feedback were largest in the 100% condition and smallest in the 50% condition. As mentioned above, the observed effects in the 50% condition can only be attributed to processing of the feedback stimulus: Participants can not distinguish between correct and incorrect responses until they have encoded the feedback. Responses in this condition can be directly compared to the results reported recently by van der Veen et al. (2004). These authors reported a very similar pattern of cardiac responses to non-informative feedback. They explained their findings in terms of motivational properties of the feedback stimulus. However, as will be argued later on the basis of our R-locked data, the current data cannot be used to substantiate this hypothesis.

The effects of type of feedback and condition on R-locked responses were comparable to the FB-locked responses. That is, heart rate slowed following incorrect responses. In addition, the shorter IBIs accompanying correct responses were shortest in the 100% condition and longest in the 50% condition. Although the sensitivity of the cardiac response to performance accuracy is consistent with previous reports (e.g., Hajcak et al. 2003; Somsen et al., 2000), it should be noted that the graded cardiac response following correct choices has not been observed in other studies. This finding seems to present a challenge to both the Crone et al. (in press) and the Van der Veen et al. (2004) interpretation of the cardiac response to performance feedback. Crone et al. (in press) proposed that heart rate slowing occurs when actual feedback is different than anticipated. According to this view, negative, not positive, feedback should differentiate between cardiac responses. Van der Veen et al. (2004) suggested that heart rate is sensitive to the valence of the feedback; that is, the heart rate response should be proportional to the reward communicated by the feedback. In the current study, however, positive feedback communicated the same amount of reward across conditions and yet conditions yielded different heart rate responses to the feedback. At this point, we have no ready explanation for the current apparent discrepancy but design differences might be important. Thus, the probabilistic learning task used by Crone et al. included an 'always' condition instead of the current 80% condition; that is, subjects always received positive feedback following one response and negative feedback to another. Van der Veen et al. used a time-estimation task not a probabilistic learning task. Further experimentation is required to assess whether these design differences impact the cardiac response in a systematic way.

Most importantly, the patterns of R-locked cardiac and cortical responses were highly similar. That is, the largest differences were found in the 100% condition and the smallest differences were found in the 50% condition. The two measures did not correlate, however. Moreover, groups with high or low ERN amplitude did not differ with respect to cardiac responses. Finally, the condition effects on the cardiac response were obtained only for correct, not incorrect, choices while this pattern was the reverse for the cortical responses. The current findings, then, suggest that cardiac and cortical responses reflect different aspects of error processing. It has been suggested (Hajcak et al., 2003) that the cardiac response to errors may reflect a process that is indexed by the Pe, a slow positive waveform that usually follows the ERN (e.g., Falkenstein et al., 1991). The ERN is typically associated with cognitive and error processes (Coles et al., 2001) whereas the Pe has been associated with affective and evaluative processes (Falkenstein et al., 2000; Nieuwenhuis et al., 2001). Interestingly, localization studies have identified the caudal part of the anterior cingulate as the neuro-electrical source of the ERN whereas its rostral part has been associated with the Pe (e.g., Bush et al., 2001). Anatomical studies indicate that the cardiovascular system implicates the rostral part of the anterior cingulate (e.g., Vogt et al., 1995) supporting the suggested link between the cardiac and Pe response to errors. Obviously, the current results provide only indirect support for the hypothesis that cardiac responses to errors and the cortical Pe reflect similar processes and share a common origin. At this point, the hypothesis is speculative and should be tested in future studies aimed at establishing a direct link between cardiac and cortical responses.

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