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Cardiac concomitants of feedback processing

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Abstract

This study examined the heart rate changes associated with positive and negative performance feedback in a probabilistic learning task derived from Holroyd and Coles (Psychological Review, 109 (2002) 679). In this task, subjects were presented with six stimuli and asked to respond by pressing a left versus right key. Responses were followed by positive or negative feedback. Subjects had to infer the S-R mapping rule on the basis of feedback provided to them. Two stimuli were consistently mapped onto the left versus right key (100% mapping). Two other stimuli were randomly mapped onto the keys (50% mapping) and responses to the two remaining stimuli received always positive or negative feedback (always condition). Negative feedback was associated with heart rate slowing in the 100% condition. Heart rate slowed following both positive and negative feedback in the 50% condition, but only when the previous encounter with the stimulus was followed by alternate feedback. Heart rate did not differentiate between positive and negative feedback in the always condition. The results were interpreted in support of the hypothesis assuming that heart rate slowing is elicited when performance-based expectations are violated.

Keywords: Error processing; Heart rate; Feedback; Performance monitoring; Motivation

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1. Introduction

To adjust performance appropriately to environmental demands, it is important to monitor ongoing action and to process feedback for possible errors. The monitoring of external signals from the environment permits the adjustment of response settings in order to prevent errors from recurring in the future. Error and feedback monitoring have been incorporated in various experimental and neuropsychological tasks, to the extent that participants have to adjust their behaviour to prevent errors on future trials, as for example during reversal and extinction (Rolls et al., 1994), dimensional shifting (Zelazo et al., 1996), set-shifting (Omori et al., 1999) and the Wisconsin Card Sorting task (Heaton et al., 1993; Nagahama et al., 1998).

From a psychophysiological perspective, error and feedback monitoring have received increased interest since the discovery of a frontally located negative brain potential that is associated with making errors—the error-related negativity (ERN). The ERN is observed when participants make errors in choice reaction time tasks (Falkenstein et al., 1991; Gehring et al., 1993), with peak amplitude at ≈ 80 ms after the initiation of the incorrect response. When feedback alerts the individual that an erroneous response is made, a similar ERN or medial-frontal negative (MFN) component is observed that peaks ≈ 200–300 ms after the feedback (Miltner et al., 1997; Gehring and Willoughby, 2002). The ERN/MFN is assumed to reflect a mismatch between representations of the actual response and the appropriate response (Coles et al., 1995; Holroyd et al., 2002). Localization studies examining the underlying neuro-anatomical mechanisms of cognitive control have shown that the resolution of error-related conflict is centered in, or very near, to the anterior cingulate cortex (ACC) (Botvinick et al., 1999; Carter et al., 2000; Gehring and Willoughby, 2002).

Recently, it was shown that feedback monitoring is also reflected in heart rate slowing (Somsen et al., 2000; Van der Veen et al., 2000). In the study of Somsen et al. (2000), adolescents completed a computerized Wisconsin Card Sorting Task (WCST). This task requires that participants match figures onto one of four response options. The matching principles are not known beforehand. The subject must find the correct sorting rule (color, shape or number) on the basis of feedback. When the subject has sorted correctly on ten consecutive trials, the rule changes without warning and the process of rule searching starts again. Somsen et al. observed that the shift towards cardiac acceleration that usually occurs at response onset (Coles and Strayer, 1985; Somsen et al., 1985) was significantly delayed when participants received negative feedback relative to positive feedback. This cardiac slowing was larger when the feedback was unexpected (after a rule change) and for good relative to bad performers. Somsen et al. concluded that a monitoring/evaluation process must have triggered the cardiac response. The authors suggested that this heart rate slowing following negative feedback bears a functional similarity to the ERN. Thus, the heart rate deceleration occurring when individuals receive negative feedback might reflect a performance monitoring mechanism that is responsible for immediate error correction and strategic adjustments that reduce the likelihood of errors in the future (Bernstein et al., 1995; Coles et al., 1995).

The interpretation of heart rate slowing as a manifestation of error/feedback processing was further examined by Van der Veen et al. (2000) using a time-estimation task derived from Miltner et al. (1997). In this task, subjects are required to estimate a 1-s time-interval, followed by positive or negative feedback. Miltner et al. (1997) observed the occurrence of the ERN following negative feedback, which was interpreted as a manifestation of a performance monitoring mechanism that results from the mismatch between the actual response and the anticipated response. Van der Veen et al. (2000) modified this task somewhat by including a yoked-control condition, in which feedback was given with a similar 50% probability, but was now unrelated to performance. The results replicated findings of Somsen et al. (2000) by showing heart slowing following negative feedback. However, heart rate slowing to negative feedback occurred also when the feedback was not related to actual performance. This finding seems to challenge the interpretation of heart rate slowing as a manifestation of a performance monitoring mechanism. Van der Veen et al. (2000) submitted the hypothesis that heart rate deceleration following negative feedback might reflect a motivation-related detection process that occurs when incentive outcomes are worse than anticipated (see also Elliot et al., 1997).

The aim of this study was to examine to what extent heart rate slowing following negative feedback is related to the subject's expectancy. According to Somsen et al. (2000), heart rate slowing is related to the expectations derived from performance on previous trials. Van der Veen et al. (2000) proposed that heart rate slowing following feedback is related to the valence (positive versus negative) of the feedback rather than its relation to performance expectations. These competing hypotheses were examined using the probabilistic learning task that Holroyd and Coles (2002) used to assess the ERN. The experimental task required participants to press one of two buttons in response to a series of six stimuli. The participants were told to infer the stimulus-response mappings by trial-and-error, using information provided by positive or negative feedback, presented at the end of each trial. A critical aspect of the task was that the six stimuli differed in the degree to which the response was predictive of the value of the feedback. For two of these stimuli, participants could learn to control the value of feedback by acquiring the stimulus-response mapping. In this condition, the feedback was 100% valid (the '100% condition'). For two other stimuli, feedback was unrelated to the selected response, providing 50% positive and 50% negative feedback (the '50% condition'). The last two stimuli provided always-positive feedback or always-negative feedback, independent of the response (the 'always condition'). This paradigm provides the opportunity to contrast the Somsen et al. (2000) 'monitoring hypothesis' versus the Van der Veen et al. (2000) 'valence interpretation' of heart rate changes associated with feedback reinforcement.

In addition, this paradigm allows us to examine the relation between error-related cardiac slowing and the ERN, by making a comparison between the current results and the results reported by Holroyd and Coles (2002). Holroyd and Coles (2002) and Holroyd et al. (1998) have recently proposed a neurocomputational theory that accounts for effects of positive and negative feedback during reinforcement learning. According to this theory, commission of an error induces a phasic decrease in mesencephalic dopaminergic activity, when the system first detects that ongoing

events are worse than expected. The reinforcement learning signals are thought to work according to a 'temporal difference' (TD) learning algorithm (Sutton and Barto, 1998). The TD signal is assumed to give the earliest prediction of future positive or negative outcomes and is purportedly associated with the ERN. If heart rate responses to negative feedback can be seen as an equivalent of ERN, heart rate is expected to be sensitive to signals giving earliest predictions that outcomes are going to be worse than expected. The interpretation of error-signals in terms of expectancy seems, to a large extent, consistent with the monitoring hypothesis (Somsen et al., 2000).

According to the 'monitoring' hypothesis, phasic heart rate deceleration following negative feedback is related to a mismatch between the expected and the actual feedback (Somsen et al., 2000). This hypothesis assumes that a cognitive system evaluates whether the feedback can be used to adjust behaviour in order to prevent negative feedback in the future. Cardiac slowing should therefore be largest following feedback indicating an error in the 100% condition because in this condition participants can predict the feedback based on their performance. Given that the monitoring process is used to adjust performance on consecutive trials (Coles et al., 1995), heart rate slowing following an error in the 100% condition is expected to be associated with more successful performance on the next stimulus encounter. In contrast, heart rate slowing is not predicted to occur in the always condition as feedback is linked to the stimulus and not the response. Thus, under this condition, performance based violations of the feedback will not occur. Similarly, in the 50% condition, subjects cannot predict the outcome and thus heart rate is not anticipated to slow in response to negative feedback. In contrast, the 'valence hypothesis' (Van der Veen et al., 2000) suggests that heart rate slowing will occur whenever negative feedback is presented. Thus, according to this hypothesis, heart rate slowing will not differentiate between conditions.

2. Method

2.1. Participants

Twenty-one healthy adults (14 women) participated in the experiment. The participants, ranging in age from 20 to 29 (mean age = 24.5), were undergraduate students at the University of Amsterdam and received course credits for their participation, plus a performance-related bonus. All participants were healthy according to self-report and had normal or corrected-to-normal vision.

2.2. Stimuli

Stimuli were presented in color against a white background on a 17-inch computer screen placed at a distance of 100 cm from the participant. Each experimental block involved a new set of six imperative stimuli. Imperative stimuli were generated from Microsoft Office clip art, including neutral pictures of animals, buildings, etc. and

were scaled to a uniform size. A yellow or a blue square served as positive and negative feedback signals, indicating that the participant was rewarded or penalized on that trial. The stimulus-response mappings and feedback assignments were counterbalanced across participants and were fixed across the experiment. A black square was presented whenever a 600 ms response deadline was missed.

2.3. Experimental task

On each trial, the imperative signal was response terminated. The response initiated a blank screen for 1000 ms that was terminated by the 1500 ms feedback stimulus. The interval between consecutive imperative stimuli was ≈ 3 s. Participants were required to make a two-choice decision by pressing the 'z' or '/' button on a computer keyboard. To ensure that participants would make mistakes in the 100% mapping condition after the mapping was learned, participants were required to respond within 600 ms after the onset of each imperative stimulus. If they did not respond before the deadline, they received a penalty signal, indicating that they lost two cents and that they should respond faster. Trials on which the penalty signal was presented were not analyzed. On average, the penalty signal was given on 3% of the trials (ranging from 2 to 7%). Otherwise, the feedback stimulus indicated that the participant had earned (in case of a correct response) or had lost (in case of an incorrect response) 1 cent of bonus money on that trial.

The task was divided into eight blocks of 210 trials. In each block, six new imperative stimuli were presented, each of which was presented 35 times in a pseudo-random order. Participants were not informed about the stimulus-response mappings, but were told to infer the mappings by trial and error and to respond in order to increase their bonus. One of the six stimuli was mapped to the left button, so the participant received positive feedback if he/she pressed the left key and negative feedback if he/she pressed the right key. Another of the six stimuli was mapped to the right button, so that the participant received positive feedback if he/she pressed the right key and negative feedback if he/she pressed the left key. Following Holroyd and Coles (2002), these two mappings were called *100% mappings*. For two other stimuli in each block, feedback was delivered at random to the participants, independently of whether they pressed the left or the right key. As a result, the participant received positive feedback on 50% of the trials and negative feedback on the other 50% of the trials. Again following Holroyd and Coles, these mappings were called *50% mappings*. The fifth stimulus was always accompanied by positive feedback (*always correct mapping*) and the sixth stimulus was always accompanied by negative feedback (*always incorrect mapping*).

Before the experimental phase, participants received written instructions and performed a practice block of 100 trials. Participants began the task with a bonus of HFL 2.50 (= ?1.31). At the end of each block, participants were provided with information indicating the total amount of money they earned throughout the task. Bonus money was paid to the participants upon completion of the experiment.

2.4. Recordings and data reduction

During the task, the electrocardiogram (ECG) and respiration were continuously recorded. The ECG was recorded from three AgAg/CL electrodes, attached via the modified lead-2 placement. Respiration was recorded through a temperature sensor placed under the nose. The signals were amplified by a Nihon Kohden polygraph and sampled by a Keithley AD-converter at a rate of 400 Hz. The recorded inter beat intervals (IBIs) were screened for physiologically impossible readings and artefacts. These were corrected by adjusting specific parameters in the program that extracted the IBIs from the digitized ECGs. The respiration signal was used only to eliminate heart rate changes associated with gross respiratory maneuvers.

Four IBIs were selected around the feedback, that is, the concurrent IBI (IBI 0), the IBI preceding the feedback stimulus (IBI −1) and two IBIs following the feedback stimulus (IBI 1 and 2). IBI's were referred to the second IBI preceding the feedback (IBI −2). Analysis of IBI −2 showed that there were no differences between the three conditions, $F(1,160) = 0.01$, $P = 0.90$. Heart rate analyses with IBI as repeated measures factor were adjusted using Huynn–Feldt corrections to adjust for inhomogenity of the variance-covariance matrix.

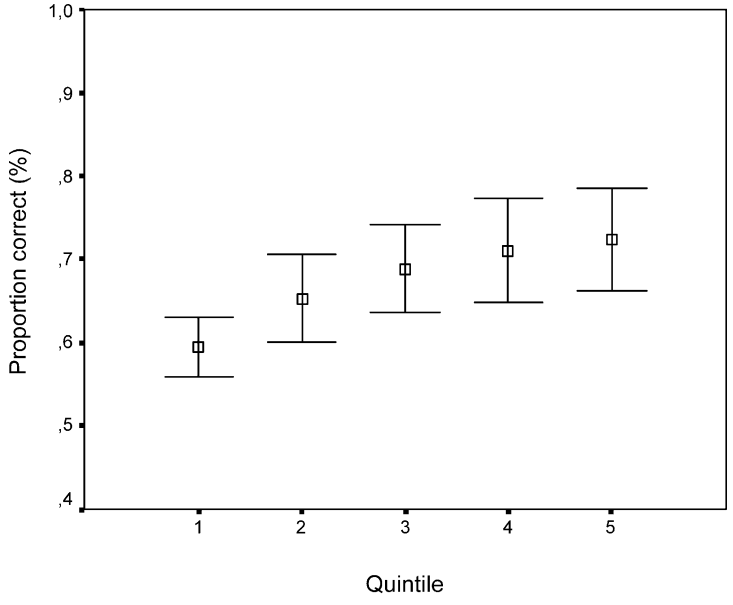

Fig. 1. Performance increase for the 100% condition as a function of task duration (in quintiles of 42 trials).

3. Results

3.1. Performance

Performance could improve in the 100% condition, because in this condition feedback was dependent on the correctness of the response. Therefore, we asked whether accuracy increased as a function of trial block quintile (42 trials) in the 100% condition. As can be seen in Fig. 1, participants learned the stimulus-response mapping rule as the task progressed, $F(4,80) = 14.01$, $P < 0.001$.

Feedback was not systematically linked to responses in the 50% condition. However, because participants may still have made use of the feedback in this condition, we examined if participants changed their response to the stimulus on the next encounter with that stimulus more frequently after receiving negative relative to positive feedback. This was evaluated by examining responses on consecutive encounters with the same stimulus. If subjects use the feedback provided to them in the 50% condition, they are likely to have maintained the same response unless negative feedback prompted them to select the alternative response. The results revealed that positive feedback led participants to select the same response on the consecutive encounter with the same stimulus more often (M = 40.6%, S.D. = 0.02) than negative feedback (M = 35.6%, S.D. = 0.02), $F(1,19) = 13.95$, $P < 0.001$.

3.2. Heart rate

The heart rate analyses are presented in two separate sections. The first set of analyses focused on differences between positive and negative feedback across conditions. The second set of analyses focused on sequential learning effects for each separate condition. All analyses reported below included a 'quintile' factor, to examine if cardiac responses changed during progression of the trial block. However, 'quintile' did not modulate any of the significant effects, therefore we will only report effects collapsed over block quintiles.

3.3. Analysis of cardiac responses evoked by positive and negative feedback

Four IBIs surrounding the feedback stimulus were submitted to repeated-measures ANOVAs to assess whether positive and negative feedback evoked different cardiac responses in the three conditions. The $3 \times 2 \times 4$ (condition $\times$ feedback $\times$ IBI) ANOVA yielded main effects of condition, $F(2,40) = 14.19$, $P < 0.001$, H-F = 0.97, feedback, $F(1,20) = 12.26$, $P < 0.005$ and IBI, $F(3,60) = 6.84$, $P < 0.005$, H-F = 0.838. There were significant interactions between condition and feedback, $F(2,40) = 3.99$, $P < 0.025$, H-F = 1.0, condition and IBI, $F(3,120) = 8.98$, $P < 0.001$, H-F = 0.604 and feedback and IBI, F (3,60) = 3.73, $P < 0.025$, H-F = 0.803. These interactions were qualified by a three-way interaction between, condition, feedback and IBI, $F(6,120) = 3.72$, $P < 0.015$, H-F = 0.533, that is plotted in Fig. 2.

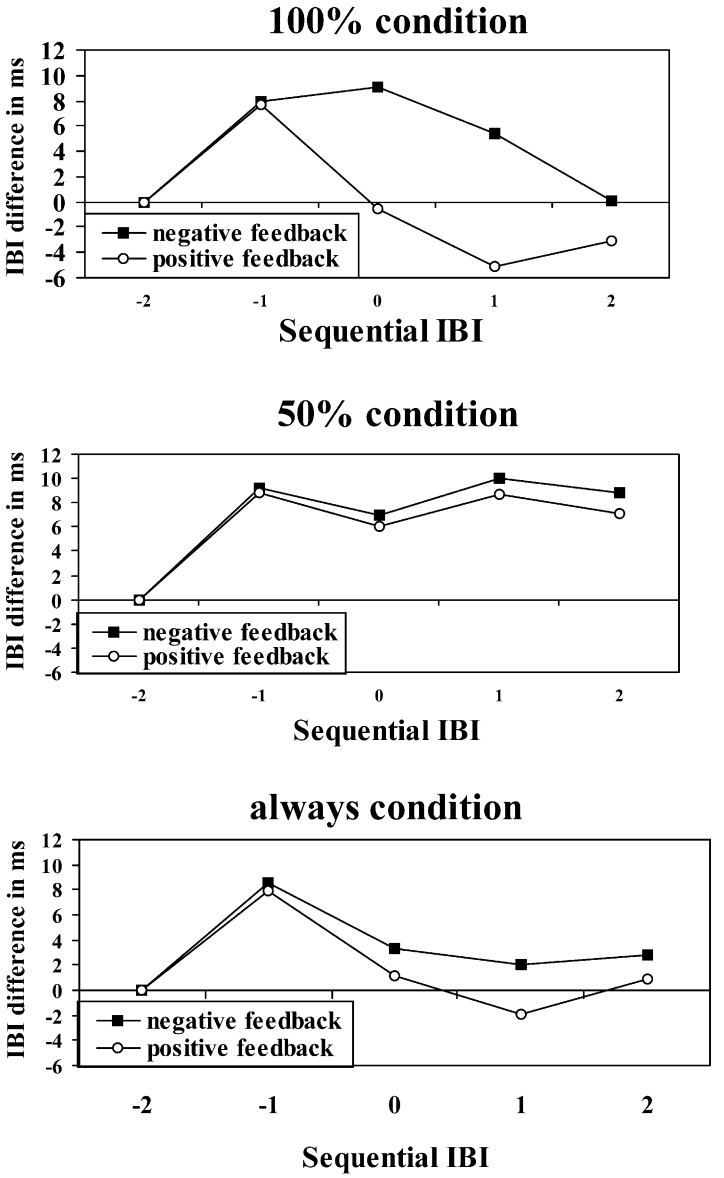

Fig. 2. Cardiac responses associated with negative and positive feedback in the 100% mapping condition, 50% mapping condition and always mapping condition.

As can be seen in Fig. 2, in the 100% condition heart rate accelerated upon correct responding, but the acceleration was delayed upon incorrect responding. These impressions were verified by a significant interaction between feedback and IBI in a follow-up simple effect analysis, $F(3,60) = 5.78$, $P < 0.01$, H-F $= 0.707$. In the 50%

condition, heart rate was delayed for both positive and negative feedback trials; the interaction between feedback and IBI was not significant, $F(3,60) = 0.35$, $P = 0.73$, H-F = 0.737. Moreover, the maximum slowing in the 50% condition has shifted from IBI 0 to IBI 1, verified statistically by an interaction between condition (100 vs. 50%) and IBI, $F(3,60) = 11.36$, $P < 0.001$, H-F = 0.732. In the always condition, heart rate accelerated immediately following the response and there was no difference between positive and negative feedback trials, $F(3,60) = 1.11$, $P = 0.35$, H-F = 0.590.

3.4. Sequential analyses

The next set of analyses focused on sequential trials in the 50 and 100% condition because in these conditions, performance based expectations could be violated by alternating feedback or performance errors. Note that for all following analyses, the consecutive encounters were separated by trials associated with a different stimulus and/or response.

Analysis of sequential data in the 50% condition should reveal if cardiac slowing is larger when predictions on the basis of the previous stimulus encounter are violated. According to this 'monitoring/violation hypothesis', heart rate slowing in the 50% condition should be larger following feedback that was different from the previous encounter, compared to feedback that was similar to the previous encounter. The data in the 50% mapping condition were averaged according to four separate conditions and focused on the *last* feedback in the sequence: negative feedback trials preceded by negative feedback trials, negative feedback trials preceded by positive feedback trials, positive feedback trials preceded by positive feedback trials and positive feedback trials preceded by negative feedback trials, based on the same stimuli and the same responses. The 2 × 2 × 4 (same/alternate × feedback valence × IBI) ANOVA resulted in a significant interaction between same/alternate and IBI, $F(3,54) = 6.99$, $P < 0.001$, H-F = 0.791. As can be seen in Fig. 3, IBIs were longer

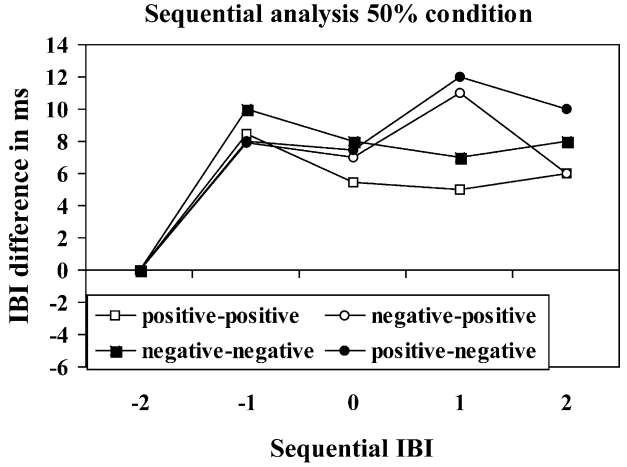

Fig. 3. Sequential effects on cardiac responses in the 50% condition (see text for explanation).

following alternating feedback compared to same feedback. In contrast, there was no significant interaction between IBI and feedback valence, $F(3,54) = 0.26$, $P = 0.79$, H-F = 0.712 and feedback valence did not modulate the same/alternate × IBI interaction, $F(3,54) = 1.10$, $P = 0.35$, H-F = 0.825.

Given the evidence for the monitoring hypothesis in the previous analysis, sequential data in the 100% condition were analyzed in order to examine if heart rate slowing following negative feedback represents the manifestation of a remedial action mechanism. Data in the 100% mapping condition were averaged according to four separate conditions and the analysis focused on the *first* feedback in the sequence. The four conditions were: negative feedback trials followed by negative feedback trials, negative feedback trials followed by positive feedback trials, positive feedback trials followed by positive feedback trials and positive feedback trials followed by negative feedback trials. According to the 'remedial action' hypothesis, heart rate slowing should be largest for negative feedback trials that are followed by a correct response. The $2 \times 2 \times 4$ (same/alternate × feedback valence × IBI) ANOVA revealed greater heart rate slowing following negative feedback compared to positive feedback, $F(3,57) = 6.75$, $P < 0.001$, H-F = 0.890. However, this effect was not modulated by the same/alternate factor ($F(3,57) = 0.53$, $P = 0.56$, H-F = 0.554), revealing that cardiac slowing was not associated with successful performance on the next encounter of that stimulus.

4. Discussion

The present study examined whether heart rate slowing following negative feedback is related to predictions derived from previous performance (Somsen et al., 2000) using the probabilistic learning task used by Holroyd and Coles (2002) to study the ERN. Accuracy measures in the 100% condition were comparable to the Holroyd and Coles (2002) results showing that participants successfully used feedback to improve their performance. Although feedback was not performance-related in the 50% condition, participants more often kept making the same response when previous feedback was positive indicating that, to some extent, they still made use of the feedback.

Analysis of cardiac responses yielded several important results. As in previous studies (Somsen et al., 2000; Van der Veen et al., 2000), negative feedback was associated with heart rate slowing in the 100% condition. Following the monitoring hypothesis (Somsen et al., 2000), it could be suggested that heart rate slowing following errors may result from a remedial error monitoring system that alerts when an error is made in order to reduce errors in the future. However, when we examined direct feedback effects on success or failure on following trials, cardiac slowing was not associated with success on the following stimulus encounter. This finding suggests that heart rate reflects a mechanism implicated in detection of failures rather than providing immediate compensation. Hajcak et al. (in press) examined the relation between the ERN, error positivity (Pe; i.e. another component associated with performance monitoring and error detection) and heart rate slowing following

errors and subsequent post error slowing. Post error slowing was presumed to provide and index of compensatory action taken to increase the likelihood of correct responses on trials subsequent to errors. Hajcak et al. found that heart rate slowed following errors when no feedback was given about performance This finding suggests that heart rate is also sensitive to the internal processes associated with performance monitoring. There was, however, no consistent relation between heart rate and subsequent post-error slowing. Interestingly, the Pe but not the ERN, was associated with post error slowing, suggesting that both the ERN and heart rate slowing following errors may be related to detection rather than compensatory processes.

A finding of the current study that seems to challenge the monitoring hypothesis is the observation that heart rate slowed following both positive and negative feedback in the 50% condition. At first glance, this finding is consistent with the findings reported previously by Van der Veen et al., (2000) who interpreted this finding in terms of the 'motivational valence' hypothesis. That is, the delay in heart rate recovery to negative feedback was interpreted to suggest that heart rate responses to feedback stimuli are manifestations of reward or punishment processing rather than indices of performance monitoring per se. However, sequential analyses of the current findings showed that heart rate slowing following positive and negative feedback in the 50% condition was only present for trials on which feedback was different from feedback on the previous encounter of the same stimulus. This result is important as it shows that the cardiac response reflects the processing of the feedback stimulus vis-à-vis performance rather than being a manifestation of its motivational valence. Heart rate slowed following the feedback only when the feedback violated predictions, either positively or negatively. These findings suggest that, in line with the Somsen et al. (2000) assumptions, performance-related cardiac slowing results from a mismatch between the anticipated outcome and the actual feedback.

Finally, consistent with the monitoring hypothesis (Somsen et al., 2000) heart rate changes were similar following positive and negative feedback in the always condition. This result is consistent with the monitoring hypothesis, because the feedback was related to the stimulus and therefore subjects could not make performance-based expectations. Thus, in this condition expectations were never violated. In contrast, the findings are inconsistent with the strict version of the valence hypothesis (Van der Veen et al., 2000) because this hypothesis suggests that heart rate should slow whenever negative feedback is given. A weaker version of the motivation valence hypothesis may suggest that the cardiac response reflects the processing of reward or punishment information provided by the feedback but only when subjects are seeking information for adjusting their performance to dynamical changes of the environment. Obviously, the weaker version of the motivational valence hypothesis is difficult to distinguish from the performance monitoring hypothesis of cardiac responses to feedback stimuli.

The cardiac responses in this study showed partly similar and partly different patterns than those observed for the ERN by Holroyd and Coles (2002). The Holroyd and Coles' neurobiological model of error processing proposes that during

reinforcement learning, dopamine signals are sent following a temporal difference (TD) learning algorithm, when the neural system first detects that the consequences of an action are better or worse than expected. Like the stimulus-locked ERN, cardiac responses did not differ for feedback in the always correct and always incorrect conditions. Additionally, like the response-locked ERN, cardiac slowing was observed subsequent to an erroneous response in the 100% mapping condition. According to the Holroyd and Coles' model, the response-locked ERN occurs after an incorrect response, because once mappings are known the response gives the first indication for TD error signal firing. Cardiac slowing after an incorrect response might be a similar manifestation of the firing of a TD error signal.

Differences between cardiac responses and ERN measures appeared when feedback was not informative. In the 50% mapping condition, Holroyd and Coles observed a feedback-locked ERN following negative feedback, whereas in this study heart rate slowed following both positive and negative feedback, but only when the feedback was different from the feedback on the previous stimulus encounter. At least two possible accounts can be invoked to explain for these apparent differences. First, the participants may have performed the experiments differently. This seems unlikely, since the procedural details and the performance results were comparable. The second possibility is that the system initiating the feedback-related ERN differs from the system regulating the feedback-related heart rate responses. Note that heart rate was responsive to changing feedback in general, whereas the ERN in Holroyd and Coles' study was sensitive only to signals providing negative feedback. This inconsistency may suggest that the feedback-related ERN is sensitive to signals alerting that the outcome of a choice is *worse* than anticipated (cf. Holroyd and Coles, 2002), whereas heart rate seems sensitive to signals alerting that feedback is *different* than anticipated (Somsen et al., 2000). The implications of these admittedly subtle differences should be subject to further investigation.

Frontal systems of the brain, including the prefrontal cortex (Stuss and Benson, 1986), the ACC (Posner and DiGirolamo, 1998) and the basal ganglia (Cummings, 1993; Holroyd and Coles, 2002) are believed to contribute to executive control and error monitoring. Based on neurobiological studies (Gehring and Knight, 2000), there is evidence suggesting that the frontal cortex plays an important role in monitoring of behaviour and errors. Using source localization analyses, the scalp distribution of negative feedback-related ERN responses was consistent with a source in or very near to the ACC, which phenomenology corresponds closely to that of response-related ERN responses (Miltner et al., 1997). Holroyd and Coles (2002) reported a similar locus and argued that the ERN is generated when a negative reinforcement-learning signal is conveyed to the ACC via the mesencephalic dopamine system. The ACC then acts on the signal to modify the task at hand. A related theoretical approach, the conflict-monitoring theory, proposes that the ERN reflects the neural response of the ACC when it detects response conflict (Botvinick et al., 1999; Carter et al., 2000). Animal studies showed that changes of heart rate were caused by electrical and chemical stimulation of the ACC (Maeda et al., 1988; Ter Horst et al., 1997; Crippa et al., 1999). Furthermore, Kubota et al. (2001) reported a significant relation between ACC activity and heart rate variability in men

(see also Gianaros et al., submitted for publication). Given the direct relationship between ACC activity and heart rate changes, it can be hypothesized that ERN effects, which reflect activity of conflict-related attentional networks including the ACC, might have interrelation with the peripheral autonomic activities.

To summarize, on the basis of a reinforcement-learning paradigm inspired by Holroyd and Coles (2002) ERN study, we have shown that heart rate slowing is sensitive to violation of performance-based expectations. The timing and responsiveness of the cardiac signal correspond well with monitoring models that are proposed in the literature to explain the ERN, and are possibly initiated by the same underlying neurological structures, e.g. the ACC (Gianaros et al., submitted and Kubota et al., 2001). The monitoring hypothesis of cardiac slowing is consistent with substantial literature reporting phasic heart rate changes in preparation of, or following informative events (Sokolov, 1963; Jennings, 1992; Jennings et al., 1997) and the strong version of the alternative hypothesis regarding motivational cardiac responses (Van der Veen et al., 2000) was disconfirmed. This study illustrates the importance of examining multiple psychophysiological signals in tasks requiring executive control and reinforcement-based learning.

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