Dissociated roles of the anterior cingulate cortex in reward and conflict processing as revealed by the feedback error-related negativity and N200

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1. Introduction

Reinforcement learning theory is predicated on the idea that action selection is modified by its consequences (Sutton and Barto, 1998). According to Thorndike’s law of effect, if an action is followed by a reward (positive feedback) then that action will have a greater probability of being performed again, whereas if the action is followed by a punishment (negative feedback) then that action will have a lesser probability of being performed again (Catania, 1999). Recently there has been an increased interest in the functional role of the anterior cingulate cortex (ACC) in the cognitive processes that underlie reinforcement learning. It has been suggested that the dorsal region of the ACC contributes to high-level cognitive control of motor behavior, especially by using reward-related information to guide action selection (Holroyd and Coles, 2002; Nieuwenhuis et al., 2004a; Rushworth et al., 2007; but also see Botvinick et al., 2001, 2004). Several lines of investigation support this view: for example, ACC neurons have been found to be involved in revising estimates of action values (Rushworth et al., 2007), in registering positive and negative reward prediction errors (Matsumoto et al., 2007), and in guiding voluntary choices based on the history of actions and outcomes (Holroyd and Coles, 2008; Kennerley et al., 2006; Seo and Lee, 2007). Furthermore, the ACC may have an important role in maintaining action–outcome associations when the action is probabilistically associated with an outcome (Paulus and Frank, 2006; Rushworth et al., 2004). The finding that ACC neurons respond to choice outcomes has motivated the proposal that this region composes part of a larger system for reinforcement learning, such that reinforcement learning signals, believed to be carried by the dopamine system, shape the connectivity and function of neurons in the ACC and prefrontal cortex (Brown and Braver, 2005; Doya, 2008; Seo and Lee, 2007, 2008). Specifically, it has been proposed that the ACC selects and executes goal-directed temporally extended sequences of actions according to principles of hierarchical reinforcement learning (Holroyd and Yeung, in press).

Evidence for the role of the ACC in reinforcement learning in humans comes from observations of the event-related brain potential (ERP). Over the last decade, ERP studies have revealed a negative-going deflection in the ERP that is elicited by negative but not positive feedback in human trial-and-error learning tasks (Miltner et al., 1997; for review, see Nieuwenhuis et al., 2004a). When measured as the difference between the error-related and correct-related ERPs, this “feedback ERN” (fERN) is characterized by a negative deflection at frontal-central recording sites that peaks approximately 250 ms following feedback presentation (Holroyd and Ruggleson, 2007; Miltner et al., 1997). Source localization procedures have also indicated that the fERN is produced in or near the ACC (e.g., Hewig et al., 2007; Gehring and Willoughby, 2002; Miltner et al., 2003). The evaluative system that produces the fERN appears to classify outcomes into binary categories – as events that either do, or do not, indicate that a task goal has been
achieved (Hajcak et al., 2006; Holroyd et al., 2006). Further, this system appears sensitive to either utilitarian information (monetary gains vs. losses) or performance information (correct vs. incorrect choices) depending on which aspect of the feedback is made salient to the subjects (Nieuwenhuis et al., 2004b). We have proposed that this electrophysiological signal is elicited by the impact of reinforcement learning signals carried by the midbrain dopamine system onto motor areas in the ACC, where they are utilized for the adaptive modification of behavior according to principles of reinforcement learning (Holroyd and Coles, 2002; Baker and Holroyd, 2009).

A complicating factor in the interpretation of the fERN is that its timing, polarity and scalp distribution coincides with another ERP component called the N200. The N200 has been associated with the detection of response conflict (Yeung et al., 2004), a function that is also attributed to the ACC (Botvinick et al., 2001). According to this hypothesis, response conflict is elicited by the simultaneous activation of incompatible response options, even in situations in which an actual physical response is not required (Nieuwenhuis et al., 2003). For example, in go/no-go (Nieuwenhuis et al., 2003) and oddball (Holroyd, 2004) tasks, the N200 is characterized by an increase in amplitude to the infrequently occurring stimulus (for review see Folstein and Van Petten, 2008). These morphological and functional similarities between the fERN and N200 components have raised questions about their ontological statuses, namely whether the two ERP components are different manifestations of the same underlying phenomenon (Holroyd, 2004). As a case in point, a recent comparison of the negative deflections following error feedback and infrequent oddball stimuli suggests that these ERP components are in fact the same phenomenon (Holroyd et al., 2008). This observation has motivated the proposal that the difference in fERN amplitude between reward and error trials results from a positive-going deflection, the reward positivity (Rew-P),1 elicited by reward feedback (Holroyd et al., 2008; Cohen et al., 2007).2 According to this position, the N200 is elicited by conflict associated with unexpected task-relevant events including unexpected positive feedback, but unexpected positive feedback also elicits a reward positivity that cancels out the N200 (Holroyd et al., 2008).

In line with this proposal, we recently found that substance dependent individuals who were impaired at reward learning produced a N200 to reward feedback that mirrored the N200 to non-reward feedback (Baker et al., in press), suggesting that reward feedback failed to induce the Rew-P in these individuals. Conversely, problem gamblers produced an abnormally large Rew-P to monetary gains (Hewig et al., 2010). Together, these findings help support the idea that the amplitude of the fERN is modulated by reward processing in both these cases (i.e. a relatively small Rew-P in substance dependent individuals and a large Rew-P in problem gamblers), and not by error processing.

In what follows, we present a series of empirical dissociations between the “classical” N200 and the fERN that provide further support for the idea that the two ERP components are in fact distinct phenomena. In particular, in this study we disentangle the N200 from the fERN across a series of experiments that independently manipulated their amplitudes and latencies. For the purpose of clarity, we hereafter refer to the N200 as the negative deflection following error feedback, the Rew-P as the positive deflection following reward feedback, and the fERN as the amplitude of the difference between the ERPs elicited by error and reward feedback.

2. Experiment 1

Our initial experiment was in fact motivated by a question unrelated to the N200, but rather concerned the neural source of the fERN. Even though source localization procedures have indicated that the fERN is produced in or near the ACC (e.g., Hewig et al., 2007; Gehring and Willoughby, 2002; Milner et al., 2003), its origin is still a matter of contention (e.g., van Veen et al., 2004; Nieuwenhuis et al., 2005). The inverse problem is formally insoluble but can be ameliorated by application of the “converging methods” approach in which multiple source analysis techniques are utilized to compensate for their respective weaknesses (Luck, 1999, 2005, p. 296). For this reason, we adapted to an ERP paradigm a reward-based decision-making task that was previously used in a human fMRI study (Bush et al., 2002), a human intracranial recording study (Williams et al., 2004), and a monkey intracranial recording study (Shima and Tanji, 1998). Each of these studies demonstrated differential activation of the dorsal ACC between negative and positive feedback. We reasoned that converging evidence across monkey intracranial, human intracranial, human fMRI and human ERP studies would provide relatively solid evidence that the fERN is in fact generated in the ACC. To foreshadow our results, we found that the feedback stimuli in this simple decision making task did indeed elicit the fERN, as predicted. More surprisingly, the fERN was delayed by about 100 ms compared to what is typically observed in fERN experiments, thereby exposing the N200 on reward trials.

3. Methods

3.1. Subjects

Twenty undergraduate students (12 males and 8 females, aged 18–25) were recruited from the University of Victoria Department of Psychology subject pool. All subjects had normal or corrected-to-normal vision; one participant who reported a history of a brain injury was excluded. Each received course credit as well as a monetary bonus associated with the experimental task. The amount of money depended on the probability of the reward, as described below. All subjects were asked to provide informed consent as approved by the local research ethics committee. This experiment was conducted in accordance with the ethical standards prescribed in the 1964 Declaration of Helsinki.

3.2. Task

Subjects were seated comfortably in an electromagnetically shielded booth and carried out a response selection task using a standard SRX Button Box. The stimuli were viewed from a distance of about 70 cm on a 17-in., 1024 × 768 computer monitor, and were controlled using E-Prime experiment control software (Psychological Software Tools, Pittsburgh, PA). Viewing angles subtended 3.3° horizontally and 5° vertically. Subjects were asked to rest their forearms on the flat desktop and position their hand and forearm so that the fingertips of the index fingers rested comfortably on the button box. Subjects received both written and verbal instructions that explained the procedure and that stressed the importance of correct posture while minimizing head movement and eye blinks.

The task was closely modeled after the reward-based decision-making task discussed above (Bush et al., 2002; Shima and Tanji, 1998; Williams et al., 2004). Each trial started with the display of a gray fixation cross ‘*’ on a computer screen that remained until subjects pressed a button (Fig. 1). Subjects were not under a time limit to execute a response but were encouraged to respond as quickly as possible. They were instructed to use their left index finger to press button 1 and their right index finger to press button 2 and on the first trial were asked to guess a response. Following their response a blank screen was displayed for 500 ms and immediately afterwards a feedback stimulus was presented for 500 ms. The feedback stimuli were displayed in green on a black background in the centre of the screen and were selected at random (without replacement) according to the following probabilities: On 80% of the trials (Stay-Reward condition) subjects were presented with a feedback stimulus (SSS) indicating that they received a relatively large reward (3 cents CAN) and that they should press the same button on the following trial; on 10% of the trials (Switch-Reward condition) subjects were presented with a feedback stimulus
The amplified signals were digitized at a rate of 250 samples per second using a differential amplifier with a frequency response of DC 0.017–67.5 Hz (90 dB above ground). Signals were acquired using sintered Ag/AgCl ring electrodes mounted in a nylon cup (Jasper, 1958). Signals were filtered with a 4th order digital Butterworth filter with a pass band of 10–20 Hz. For feedback, stimuli, a 1200 ms epoch of data extending from 200 ms prior to 1000 ms following feedback onset was extracted from the continuous data file for analysis. Ocular artifacts were corrected using the eye movement correction algorithm described by Gratton et al. (1983). The EEG data were re-referenced to linked mastoids electrodes. The data were baseline corrected by subtracting from each sample the mean voltage associated with that electrode during the 200 ms interval preceding stimulus onset. Trials with muscular and other artifacts were discarded using a ±100 μV level threshold and a ±50 μV step threshold as rejection criteria. The EEG data were then re-segmented by condition. For the feedback cues, epochs of 800 ms were segmented, extending from 200 ms prior to 600 ms following the onset of the feedback cue, ERPs were created for each electrode and participant by averaging the single-trial EEG according to feedback type.

3.3. Electrophysiological recordings

The electroencephalogram (EEG) was recorded using a 63 electrode montage in accordance with the extended international 10–20 system (Jasper, 1958). Signals were acquired using sintered Ag/AgCl ring electrodes mounted in a nylon electrode cap (Falk Minow Services, Herrsching) and amplified by low-noise electrode differential amplifier with a frequency response of DC 0.017–67.5 Hz (90 dB octave roll off). The amplified signals were digitized at a rate of 250 samples per second. Digitized signals were recorded to disk using Brain Vision Recorder software (Brain Products GmbH, Munich). Inter-electrode impedances were maintained below 15 kΩ. Two electrodes were also placed on the left and right mastoids (M1 and M2). During recording all activity was referenced to an overall average. The electrooculogram (EOG) was recorded for artifact correction; horizontal EOG was recorded from the sub orbit of the right eye and electrode channel Fp2.

3.4. Data analysis

Post processing and data visualization were performed using Brain Vision Analyzer software (Brain Products GmbH, Munich). The digitized signals were filtered using a 4th order digital Butterworth filter with a pass band of 10–20 Hz. For feedback stimuli, a 1200 ms epoch of data extending from 200 ms prior to 1000 ms following feedback onset was extracted from the continuous data file for analysis. Ocular artifacts were corrected using the eye movement correction algorithm described by Gratton et al. (1983). The EEG data were re-referenced to linked mastoids electrodes. The data were baseline corrected by subtracting from each sample the mean voltage associated with that electrode during the 200 ms interval preceding stimulus onset. Trials with muscular and other artifacts were discarded using a ±100 μV level threshold and a ±50 μV step threshold as rejection criteria. The EEG data were then re-segmented by condition. For the feedback cues, epochs of 800 ms were segmented, extending from 200 ms prior to 600 ms following the onset of the feedback cue, ERPs were created for each electrode and participant by averaging the single-trial EEG according to feedback type.

3.5. fERN analysis

Statistical analysis of EEG data was restricted to channel FCz, where the fERN is maximal (Holroyd and Krigolson, 2007; Miltner et al., 1997). The fERN was evaluated with a difference wave approach in which the stimulus probabilities were balanced across conditions (e.g., Holroyd et al., 2009; Holroyd and Krigolson, 2007). Specifically, for each participant a difference wave was created by subtracting the Switch-Reward ERP (10% probability) from the corresponding Switch-No-Reward ERPs (10% probability). The peak amplitude of this difference wave was obtained by detecting its maximum deflection beginning at 200 ms and extending to 400 ms following the onset of the feedback stimulus. These values were statistically tested against zero using a one sample t-test.

4. Results

Fig. 2 (top) shows the ERPs elicited by the Switch-Reward and Switch-No-Reward conditions and the corresponding difference wave recorded at channel FCz. FERN amplitude was significantly different from 0, t(19) = 11.33, p < 0.001 (M = −5.17 μV, SD = 2.04), and exhibited a frontal-central scalp distribution with a maximum at channel FCz (Fig. 2, bottom). These observations are consistent with the previous literature (e.g., Baker and Holroyd, 2009; Holroyd and Krigolson, 2007; Miltner et al., 1997). However, the fERN latency (M = 342 ms, SD = 34) occurred about 100 ms later than what is typically reported (200–300 ms).

Interestingly, inspection of Fig. 2 reveals large negative-going deflections in both ERPs during the time period of the fERN, much like the classical N200 (Polstein and Van Petten, 2008; Pritchard et al., 1991). To investigate this result further, we measured N200 amplitude base-to-peak using the algorithm described in Holroyd et al. (2003). A paired-sample t-test revealed that the Switch-Reward feedback (M = −4.3 μV) and Switch-No-Reward feedback (M = −3.8 μV) stimuli elicited N200s of about equal amplitude, t(19) = 1.04, p > .05.

5. Discussion

The purpose of Experiment 1 was to look for the source of the fERN by utilizing in an ERP experiment a task design that has previously been used in human fMRI (Bush et al., 2002), human intracranial recording (Williams et al., 2004), and monkey intracranial recording (Shima and Tanji, 1998) studies to show feedback-related activation of the ACC. Consistent with our prediction, we demonstrated that the positive and negative feedback stimuli associated with switch cues in this task elicited a fERN, providing converging evidence that the ACC is the actual source of the fERN. This inference is supported by a source localization analysis that indicated that the origin of the fERN was in the region of the ACC (data not shown; see also Hewig et al., 2007; Gehring and Willoughby, 2002; Miltner et al., 2003). However, contrary to our expectation, the fERN was generated about 100 ms after it normally occurs (Nieuwenhuis et al., 2004). Although this differ-
ence was observed in the time range of the P300, a positive-going deflection that follows the fN200 about 250 ms post-feedback (Polich and Ciardo, 2006), the frontal-central scalp distribution of the difference wave is consistent with its identification as the fERN rather than the P300 (Fig. 2, bottom) (Holroyd and Krigolson, 2007). Notably, the fERN was immediately preceded by a large negative-going deflection in both ERPs at about 250 ms following feedback onset that is reminiscent of the N200. Given our position that variance in fERN amplitude can result from modulation of N200 amplitude by the superposition of a positive-going ERP component, the Rew-P (Holroyd et al., 2008), we suggest that the Rew-P was delayed in the present task on Switch-Reward trials by about 100 ms, thereby exposing the N200. This delay may have resulted in the four experiments to account for all possible combinations of reward probabilities and feedback instructions, as indicated below.

6. Experiments 2–5

We modified the reward-based decision making task in Experiment 1 to investigate whether feedback complexity delayed the fERN in that task. Specifically, we dissociated the instruction and performance aspects of the feedback by displaying an instruction cue before the feedback stimulus (although in principle the feedback stimulus could have been presented before the instruction cue, doing so would have confounded the feedback probabilities; see Section 7). Further, reward probability and cue instructions were fully counterbalanced across tasks, yielding four separate experiments (see Section 7 and Fig. 3). In brief, each experiment was characterized by an instruction cue that occurred frequently and a second instruction cue that occurred infrequently. Further, Reward and No-Reward feedback following the infrequent instruction cue always occurred with equal probability. We predicted that the feedback stimuli following the infrequent instruction cues would elicit a fERN about 250 ms post-feedback, when it is normally observed.

7. Methods

7.1. Subjects, electrophysiological recordings and data analysis

Forty-eight undergraduate students (30 female, 18 male, ages 18–32) were recruited from the University of Victoria Department of Psychology subject pool to participate in four experiments (12 subjects per experiment). Electrophysiological recording and data analysis procedures were identical to those followed in Experiment 1 except that an electrode montage of 32 channels was used in Experiments 3–5 (as opposed to 63 channels). Note that in all experiments the fERN was determined by subtracting the Reward feedback ERP on trials following the infrequently occurring instruction cue from the No-Reward feedback ERP on trials following the infrequently occurring instruction cue. Thus, in Experiments 2 and 4, the difference wave was constructed by subtracting the Switch-Reward ERPs (10% probability) from the corresponding Switch-No-Reward ERPs (10% probability), and for Experiments 3 and 5 the difference wave was constructed by subtracting the Stay-Reward ERPs (10% probability) from the corresponding Stay-No-Reward ERPs (10% probability).

7.2. Task

We modified the reward-based decision making task in Experiment 1 such that the information provided by the feedback stimulus in that task was separated into an instruction cue followed by a feedback cue (Fig. 3). Each trial began with the display of a fixation cross in the form of a ‘+’ sign, which remained at the centre of the screen until the participant made their button choice: on the first trial they were asked to guess a response. The button choice was followed by a blank screen delay for 500 ms and then by one of two instruction cues, presented in yellow on a black background in the centre of the screen for 500 ms. The instruction cues consisted of a Stay Cue (‘<’ ‘>’) that indicated that the participant should repeat the same button choice on the following trial, and a Switch Cue (‘<’ ‘>’) that indicated that the participant should press the alternative button choice on the following trial. A blank screen was then presented for 500 ms, followed by one of two feedback cues presented in green on a black background for 500 ms. The feedback stimulus consisted of a Reward Feedback (‘$’) that indicated that the participant would receive 3 cents CAN for that trial, and a No-Reward Feedback (‘0’) that indicated that the participant would receive nothing for that trial. The feedback cue was then followed by a blank screen for 1000 ms.

The feedback stimulus probabilities vs. instruction cue probabilities were fully counterbalanced across the four experiments (Fig. 3). Across all four experiments, one instruction cue occurred frequently (on 80% of the trials) and the other instruction cue occurred infrequently (on 20% of the trials). Further, the frequently occurring instruction cue was always followed by the same feedback stimulus (on 100% of the frequent instruction cue trials, corresponding to 80% of all trials), whereas the infrequently occurring instruction stimulus was followed (at random, with replacement) half of the time by one feedback stimulus (on 50% of the infrequent instruction cue trials, corresponding to 10% of all trials) and half of the time by the other feedback stimulus (on 50% of the infrequent instruction cue trials, corresponding to 10% of all trials). Thus, in each experiment the feedback stimulus was predetermined on trials in which the frequently occurring instruction stimulus occurred but was undetermined on trials in which the infrequently occurring instruction stimulus occurred. Note that calculation of the fERN difference wave requires balanced feedback expectancies (Holroyd and Krigolson, 2007), which precluded presentation of the feedback stimuli before the instruction cues (in which...
Fig. 3. Task designs for Experiments 2–5. Note the instruction cue probabilities and feedback stimulus probabilities were fully counterbalanced across the four experiments. Solid arrows indicates the trial trajectory following a response for Frequent trials, dotted arrows indicate the trial trajectory following a response for Infrequent trials. Middle arrows depict time line for one trial.

Fig. 4 presents stimulus-locked grand average ERPs recorded at channel FCz for Experiments 2 through 5. Across all four experiments, both Reward and No-Reward Feedback on trials with the infrequently occurring instruction cue elicited a robust negative-going deflection in the ERP during the time-window of analysis. However, contrary to our prediction, in Experiments 2 and 3 this negative deflection was larger following Reward feedback than following No-Reward feedback, appearing as a positive deflection that peaked around 230–250 ms in the associated difference wave (see asterisks in Fig. 4). For Experiment 2, this positive deflection was maximal at channel FCz \((M = 4.9 \mu V, SD = 1.04)\), peaked 243 ms \((SD = 38)\) after feedback onset, and was significantly different from zero, \(t(11) = 4.54, p < 0.001\). For Experiment 3, the positive deflection was maximal at channel FCz \((M = 3.9 \mu V, SD = 1.45)\), peaked 258 ms \((SD = 35)\) after feedback onset, and was significantly different from zero, \(t(11) = 3.98, p < 0.001\).

Conversely, the ERPs in Experiments 4 and 5 exhibited a larger frontal-central negative deflection for the No-Reward condition than for the Reward condition, thus appearing as a negative deflection in the associated difference wave as predicted. For Experiment 4 this negative deflection was maximal at channel FCz \((M = -2.6 \mu V, SD = 1.84)\), peaked 241 ms \((SD = 35)\) following feedback onset, and was significantly different from zero, \(t(11) = -2.35, p < 0.01\). And for Experiment 5, the negative deflection was maximal at channel FCz \((-3.1 \mu V, SD = 1.65)\), peaked 272 ms \((SD = 41)\) following feedback onset, and was significantly different from zero, \(t(11) = -2.78, p < 0.01\).

9. Discussion

Across four experiments we separated the instruction-related and performance-related information conveyed by the feedback stimuli in Experiment 1 into separate Instruction and Feedback cues, in order to determine whether the delay in the fERN observed in Experiment 1 resulted from feedback complexity. We expected that dissociating the feedback information from the instruction information would reduce processing time and elicit a fERN with an earlier latency as is typically observed. To our surprise, the results in two of these four experiments were opposite to what we predicted. Specifically, although Experiments 4 and 5 appeared to elicit a normal FERN in the usual time range (albeit of relatively small amplitude), Experiments 2 and 3 elicited a negative-going deflection that was larger to the Reward Feedback than to the No-Reward Feedback, thereby producing a positive rather than negative deflection in the difference wave. The difference wave analysis confirmed that this positive deflection was statistically significant, was maximal at frontal-central electrodes sites, and had a peak latency at around 250 ms. Inspection of the individual ERPs in Fig. 4 revealed that a relatively large negative deflection in the ERP at around 250 ms post-feedback was elicited by the frequent Reward feedback stimulus (that occurred on 90% of the trials overall) when it followed the infrequent instruction cue (Experiments 2 and 3), and by the frequent No-Reward feedback stimulus (that occurred on 90% of the trials overall) when it followed infrequent instruction cue (Experiments 4 and 5). Thus, across all four experiments the frequently occurring outcome appeared to elicit a large negative going deflection in the ERP around 250 ms when it followed the infrequently occurring instruction stimulus.

We suggest that the functional and morphological characteristics of this negative deflection are consistent with its identification as the N200 (Holroyd, 2004). Evidently, feedback stimuli that
occurred relatively frequently overall elicited a larger N200 when they followed an infrequently occurring instruction stimulus. But why? Suggestively, in all of these experiments the instruction cue intervened between the response and feedback, a task design that to our knowledge has never been utilized previously in a fERN study. We suspected that the presentation of the instruction cues in this manner may have interfered with the normal production of the fERN by independently modulating N200 amplitude. To investigate this possibility we conducted two more experiments.

10. Experiment 6

In Experiment 6 we modified the reward based decision-making task used in Experiment 2 so that it was more similar to typical fERN tasks. The primary changes involved eliminating the fixation cue and requiring subjects to respond after rather than before the instruction cue; as a consequence, the instruction cue indicated to subjects how they should behave on the present trial rather than on the following trial. Additionally, rather than switching to the alternative response on 20% of the trials as they did in Experiment 2, subjects were told to “guess” the appropriate response on these trials (compare Figs. 3 and 5). Note that the stimulus probabilities were identical across the two experiments. We predicted that these changes would succeed in producing a normal fERN and, in doing so, point toward the critical factor underlying its disruption in the previous experiments.

11. Methods

11.1. Subjects, task, electrophysiological recordings and data analysis

Twelve undergraduate students (7 females, 5 males, ages 18–25) were recruited from the University of Victoria. Participation for this study was strictly voluntary. The task procedures were identical to Experiment 2, except that the fixation cross was eliminated and subjects were asked to select their response following presentation of the instruction cue (Fig. 5). Further, for the Stay Cue (||), which appeared on 80% of the trials, subjects were asked to repeat the response they made on the previous trial, and for the Guess Cue (< >), which appeared in 20% of the trials, subjects were instructed to guess the appropriate response. The trial ISI was reduced by 1000 ms because subjects responded directly to the instruction cue, in contrast to Experiment 2 where the instruction cue was displayed for 500 ms following the
response and then an additional blank screen was displayed for 500 ms. Recording and data analysis methods were identical to Experiment 2.

12. Results

Fig. 6 (top) presents the Reward and No-Reward ERPs recorded at channel FCz following the Guess Cue. Consistent with previous research, the fERN was clearly evident as a sharp negative deflection in the difference wave ($M = -7.24 \mu V, SE + 1.18$) that peaked 268 ms after feedback onset (Holroyd and Krigolson, 2007; Miltner et al., 1997). This difference was significantly different from zero, $t(11) = -6.89, p < 0.0001$, and exhibited a frontal-central scalp distribution with a maximum at channel FCz (Fig. 6, bottom).

13. Discussion

By modifying the task in Experiment 6 to resemble standard fERN tasks, we sought to isolate the critical factor that disrupted the fERN in the previous experiments. The main adaptations to the task consisted of eliminating the Fixation Cue, requiring that subjects respond following the Instruction Cue rather than following the Fixation Cue, instructing subjects that they were to execute the instruction on the present trial (rather than on the following trial), and instructing subjects that the infrequent instruction cue ‘< >’ required them to guess the response rather than to switch responses. These changes resulted in reducing the trial length by 1 s.

Even though the feedback probabilities in Experiments 2 and 6 were identical (Figs. 3 and 5), only Experiment 6 elicited a quintessential fERN (Figs. 4 and 6).

This result indicates that a difference between the two task designs disrupted the fERN in Experiment 2. However, because the tasks differed in multiple respects this critical factor remains undetermined. In particular, the fERN might depend on the requirement that subjects guess the response rather than switch to the alternative response, or it might depend on the instruction cue preceding the response such that the subjects responded to instructions on the present trial rather than on the previous trial. (We assume that the elimination of the fixation cue and the change in trial length were unlikely to affect the fERN). For this reason we conducted an additional experiment that was identical in design to Experiment 6 except that the infrequent instruction cue required subjects to switch to the alternative response as in Experiment 2 (Fig. 3) rather than to guess the response as in Experiment 6 (Fig. 5). In this case, the instruction cue indicated to subjects to switch their response on the present trial rather than on the subsequent trial. We predicted that if fERN production depends on guessing the response, then Experiment 7 would fail to yield a normal fERN. Alternatively, we predicted that if fERN production depends on subjects responding to instructions presented on the current trial as opposed to previous trial, then Experiment 7 would yield a normal fERN.

14. Experiment 7

15. Methods

15.1. Subjects, task, electrophysiological recordings and data analysis

Twelve undergraduate students (8 females, aged 18–23) were recruited from the University of Victoria. The task procedures were identical to those of Experiment 6 except that the infrequent instruction cue, which appeared on 20% of the trials, instructed subjects to ‘switch’ to the button that they did not choose on the previous trial rather than simply guess the response (Fig. 7). Thus, the infrequent instruction cue indicated to subjects that they should switch to the alternative response as it did in Experiment 2, but here the instruction cue indicated that the response on the present trial should be different than the response on the previous trial, whereas in Experiment 2 the instruction cue indicated that the response on the subsequent trial should be different from the response on the present trial.
following the presentation of the infrequent instruction cue subjects were asked to ‘switch’ from their choice on the previous trial (Experiment 7) instead of simply ‘guessing’ the choice (Experiment 6). Despite this change to the task instructions the feedback elicited a normal fERN (Fig. 8), indicating that the fERN can be elicited on trials irrespective of whether the instruction cues indicate exactly how to respond (i.e. to switch to the opposite response) or to guess the response, so long as the instructions pertain to the present trial rather than to the following trial.

18. General discussion

As its name suggests, the fERN is commonly associated with a negative deflection in the ERP elicited by error or negative feedback. By contrast, we have recently proposed that this negative deflection is actually the N200 (Holroyd, 2004). According to this position, unexpectedly rewarding events elicit a positive deflection in the ERP, the Rew-P, that cancels out or inhibits production of the N200 (Holroyd et al., 2008; see also, Cohen et al., 2007; Eppinger et al., 2008; Hewig et al., 2010, 2007; Potts et al., 2010, 2006; Foti et al., in press). When calculated from the difference wave, the fERN reflects the difference in the size of the N200 on error trials with a smaller or absent N200 on correct trials.

This possibility was further supported by the results of Experiment 1. Although this experiment was intended to provide converging evidence about the neural source of the fERN, to our surprise we found that the fERN was elicited approximately 100 ms after it is normally observed (Holroyd and Coles, 2002; Miltner et al., 1997). Further, this delay appears to have exposed the N200 on both Reward and No-Reward trials (see Fig. 2). We suggest that the complexity of the feedback stimuli in the task, which provided both performance and instructional information, delayed feedback processing by that amount of time (see also Mars et al., 2004). This interpretation is supported by the results of Experiments 6 and 7, which, when the instructional information was dissociated from the feedback stimuli, yielded an earlier fERN in the time period of the N200. These findings are consistent with the view that the N200 is elicited by task-relevant events in general but is suppressed by the Rew-P on trials with unexpected positive feedback (Holroyd et al., 2008).

According to the conflict monitoring theory of the ACC (Botvinick et al., 2001), the N200 is a manifestation of response conflict (Yeung et al., 2004) even in situations in which an actual physical response is not required (Nieuwenhuis et al., 2003). If this hypothesis is correct, it follows that factors that independently affect N200 amplitude should disrupt the fERN. Experiments 2–5 provide evidence of this possibility. Specifically, when the instructional information was provided after rather than before the response, such that it indicated to subjects what action to carry out on the following trial rather than on the present trial, the fERN was disrupted in two out of four experiments (Experiments 2–5; Fig. 4). Across these four experiments, a relatively large N200 was elicited by the frequent feedback stimulus when it followed the infrequent instruction cue, irrespective of the valence (Reward or No-Reward) of the feedback (Fig. 4). Note that in these instances the instruction cue intervened between the response and the feedback.

Suggestively, a recent study by Jia et al. (2007) demonstrated that when subjects were required to guess on each trial whether or not two sequentially presented visual stimuli would have the same color, wherein the second stimulus also served as feedback to the guess, perceptual conflict between the two stimuli generated a large N200 irrespective of whether the feedback indicated correct or incorrect performance (Jia et al., 2007). By contrast, the N200 was greatly reduced on correct trials associated with no perceptual conflict. These authors suggested that the N200 is sensitive not only

16. Results

Fig. 8 presents the Reward and No-Reward ERPs recorded at channel FCz following the Switch Cue. Consistent with previous research, the fERN difference wave (M = −6.73 μV, SE = 1.23) peaked 300 ms after feedback onset (Holroyd and Krigolson, 2007; Miltner et al., 1997). This value was significantly different from zero, t(11) = −5.47, p < 0.0001. Although visual inspection of the scalp map suggests that this difference wave was centrally distributed (see Fig. 8, bottom), a paired-sample t-test reveals that the values at Cz (M = −6.91 μV, SE = 1.33) and FCz were nearly identical, t(11) = −0.47, p = .65. This observation is consistent with other reports of fERN scalp distributions (Miltner et al., 1997; Holroyd et al., 2009; von Borries et al., 2010).

17. Discussion

Experiment 7 investigated whether the fERN was disrupted in Experiments 2 and 3 because in those experiments the subjects were presented with an instruction cue (that indicated how to respond on the following trial) after making a response on the present trial (on the basis of the instruction from the previous trial); or whether the fERN was disrupted because they were told to switch their response from the previous trial rather than simply to guess their response. The task design was modeled after Experiment 6 (Fig. 5), which did elicit a normal fERN (Fig. 6), except that

Fig. 8. Top: Experiment 7: ERPs for the Switch-Reward, Switch No-Reward, and associated difference wave. Data recorded at channel FCz. Gray shaded area corresponds to peak detection time window of the fERN; 0 ms corresponds to time of stimulus onset. Note that negative is plotted up by convention. Bottom: Scalp voltage maps for the maximal negative deflection in the difference wave at 300 ms following feedback onset.
to performance monitoring but also to the conflict between perceptual representations held in working memory (Jia et al., 2007). This hypothesis is consistent with evidence that N200 amplitude is exercised by mismatching stimuli in sequential matching tasks and in cue-target sequences (Folstein and Van Petten, 2007). Further, Missonnier et al. (2003) found that a positive-to-negative ERP deflection superimposed over the classical P200 and N200 was modulated in amplitude by working memory load. Taken together, these studies suggest that task demands involving high conflict and working memory loads should strongly activate the ACC, giving rise to increased N200 amplitudes.

In light of these observations, we suggest that the sequential presentation of the instruction and feedback stimuli in Experiments 2–5 of the present study may have modulated N200 amplitude in a comparable manner. Here, subjects maintained in working memory a representation of the required response for the following trial even as the feedback information was delivered for the present trial. The requirement that subjects utilize performance information for the present trial while maintaining information for a forthcoming response may have induced conflict between these representations, resulting in a larger N200. The fact that the N200 was largest when the frequently occurring feedback stimulus followed the infrequently occurring instruction stimulus suggests that conflict was greatest in this condition in particular. We see two possibilities here. First, because the frequent instruction cue predicted the frequent feedback stimulus with 100% validity, subjects likely came to expect the frequent feedback stimulus following the frequent instruction cue. Evidently, when the infrequent instruction cue occurred, subjects wrongly expected the infrequent feedback stimulus to follow. This expectation was violated when the infrequent instruction cue was in fact followed by the frequent feedback stimulus, eliciting a large N200. Second, an alternative account depends on the possibility that the conflict signal results from simultaneous activation of incompatible task representations supported by the ACC, rather than by incompatible response representations (Holroyd and Yeung, in press). In the present case, the Stay and Switch cues may have respectively activated Stay and Switch task representations in the ACC. Because the frequent feedback stimulus always followed the frequent instruction cue, a strong association may have developed between the frequent feedback stimulus and the frequent task representation. According to this account the trials with infrequent instruction cues would have activated the infrequent task representation; thus when these trials were followed by the frequent feedback stimulus, which in turn would have activated the frequent task representation because of their learned association, the result would lead to conflict at the level of task representations. In either case, irrespective of the mechanism of conflict, Experiments 6 and 7 demonstrated that when the instruction cue indicated what movement to execute on the present rather than on the subsequent trial, such that performance information associated with the present trial did not conflict with behavioral information held in working memory for the following trial, a normal fERN was observed. Taken together, these findings suggest that the variance in fERN amplitude across conditions can sometimes result more from the effect of response conflict rather than from unpredicted positive feedback. For this reason increases in ACC activity elicited by high-conflict situations could potentially disrupt the fERN, especially when measured as the difference between the N200 and Rew-P. Considering this, the specific functional role of the process indexed by the N200 in reward tasks and its interaction with the positive-going deflection that determines fERN amplitude remains to be elucidated in future studies. On the other hand, the N200 appears to reflect a variety of different negative deflections associated with different experimental manipulations (Folstein and Van Petten, 2008). It remains to be determined whether the N200 seen in the present study reflects conflict detection specifically or a cognitive processes underlying a related but different N200 component. Irrespective of the process, effects attributed to the fERN in other studies, such as those employing reversal-learning tasks, may in fact have described results related to the N200 (and its associated neurocognitive process) rather than an error-related process underlying the fERN (see e.g., Barcelo, 1999; Cohen and Ranganath, 2007; Chase et al., 2011).

In summary, these observations provide support for a recent formulation of the RL-ERN theory that holds that activity intrinsic to the ACC produces the N200, and that on trials following unpredicted rewards, the N200 is suppressed by the Rew-P. Our findings revealed two important dissociations between the fERN and N200. First, feedback stimulus complexity can in fact delay the onset of the Rew-P, thereby exposing the N200 on both Reward and No-Reward trials. Second, manipulating the degree of stimulus conflict can affect N200 amplitude independently of fERN amplitude. Thus, despite extensive spatial and temporal overlap between the N200 and the fERN, the present findings indicate that these two ERP components are dissociable. Because the Rew-P typically occurs during the time-range of the N200, a complete understanding of the fERN awaits further investigations into the specific nature of the N200 (e.g., Warren et al., 2010).

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