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Error-related electromyographic activity over the corrugator supercilii is associated with neural performance monitoring

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Abstract

Emerging research in social and affective neuroscience has implicated a role for affect and motivation in performance monitoring and cognitive control. No study, however, has investigated whether facial electromyography (EMG) over the corrugator supercilii—a measure associated with negative affect and the exertion of effort—is related to neural performance monitoring. Here, we explored these potential relationships by simultaneously measuring the error-related negativity, error positivity (Pe), and facial EMG over the corrugator supercilii muscle during a punished, inhibitory control task. We found evidence for increased facial EMG activity over the corrugator immediately following error responses, and this activity was related to the Pe for both between- and within-subject analyses. These results are consistent with the idea that early, avoidance-motivated processes are associated with performance monitoring, and that such processes may also be related to orienting toward errors, the emergence of error awareness, or both.

Descriptors: Affect, Effort, Performance monitoring, Cognitive control, Corrugator, ERN, Pe

Performance monitoring features prominently in everyday life. Driving vehicles, operating machinery, and controlling air traffic are just a few examples of situations where individuals must continually monitor and adjust their behavior in order to avoid adverse consequences. While research in the past two decades has dramatically advanced our understanding of the cognitive and neural underpinnings of these control processes, emerging research has also begun to describe the role of emotion, motivation, and peripheral nervous system activation in performance monitoring (Inzlicht, Bartholow, & Hirsh, 2015). One recent study suggested that error processing triggers changes in corrugator supercilii activity (Lindström, Mattsson-Mårn, Golkar, & Olsson, 2013), a facial muscle associated with negative affect (Cacioppo, Petty, Losch, & Kim, 1986), and the exertion of effort (van Boxtel & Jessurun, 1993). Here, we ask if these transient, error-related increases in electromyographic (EMG) activity over the corrugator supercilii are associated with the neurophysiological and behavioral correlates of cognitive control.

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Performance Monitoring

Performance monitoring encompasses the physiological, psychological, and behavioral processes that evaluate and respond to discrepancies and failures in goal-directed behavior (Botvinick, Braver, Barch, Carter, & Cohen, 2001). When dominant response tendencies conflict with one's goals, or produce substantial differences between anticipated and actual outcomes, cognitive control is recruited to override, restrain, or inhibit those tendencies in current or subsequent behavior (Botvinick et al., 2001; Carver & Scheier, 1990; Holroyd & Coles, 2002; Miyake et al., 2000). This kind of control is present in both laboratory reaction-time tasks, such as the Stroop task (Botvinick et al., 2001; Braver, 2012), and also during everyday self-regulatory behavior, such as restraining one's desire to eat unhealthy food (Nederkoorn, Houben, Hofmann, Roefs, & Jansen, 2010). Unsurprisingly, control has been shown to confer benefits in multiple real-life domains, including health, academic performance, well-being, stress regulation, longevity, financial stability, and relationship satisfaction (Baumeister, Heatherton, & Tice, 1994; Compton et al., 2008; Duckworth & Seligman, 2005; Hirsch & Inzlicht, 2010; Moffitt et al., 2011).

To investigate the neural processes that support performance monitoring and cognitive control on the order of milliseconds, research has focused on two ERPs that follow errors: the error-related negativity (ERN) and the error positivity (Pe). The ERN presents as a sharp negative deflection in the response-locked ERP that occurs at frontocentral sites approximately 50–100 ms after mistakes (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993). Conversely, the Pe is a broader positive deflection that occurs around 200-400 ms

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following errors over more parietal electrodes, resembling a P300 or P3b response to an error (Leuthold & Sommer, 1999; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). Converging evidence suggests that the ERN is generated in caudal regions of the medial frontal cortex (Dehaene, Posner, & Tucker, 1994; Pourtois et al., 2010; Van Veen & Carter, 2002). Conversely, while activity originating from this area is also thought to contribute to the Pe (Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004; van Boxtel, van der Molen, & Jennings, 2005; Van Veen & Carter, 2002), researchers have expressed caution over localizing this broadly distributed component to a single source (e.g., Ridderinkhof, Ramautar, & Wijnen, 2009).

While both the ERN and Pe have become well established in the ERP literature, the precise functional significance of these components remains unclear. For example, the ERN has been accounted for by a number of computational theories proposing that it reflects conflict monitoring (Yeung, Botvinick, & Cohen, 2004), reinforcement learning (Holroyd & Coles, 2002), or a calculation of error likelihood (Brown & Braver, 2005).

Additionally, in line with suggestions that neural performance monitoring and affective processes are only minimally decomposable (Inzlicht et al., 2015; Koban & Pourtois, 2014; Shackman et al., 2011), recent proposals have also suggested that the ERN represents the affective or motivational significance of errors (Aarts, De Houwer, & Pourtois, 2013; Proudfit, Inzlicht, & Mennin, 2013; Saunders, Milyavskaya, & Inzlicht, 2015). Indeed, the ERN is larger in anxious psychopathologies (Weinberg, Riesel, & Hajcak, 2012), increases as a function of motivational traits associated with greater sensitivity to aversive stimuli (Nash, Inzlicht, & McGregor, 2012), and predicts faster categorizations of negatively valenced words than positively valenced ones following errors (Aarts et al., 2013). Conversely, the ERN is smaller when established regulators of emotion, such as alcohol consumption (Bartholow, Henry, Lust, Saults, & Wood, 2012), cognitive reappraisal (Hobson, Saunders, Al-Khindi, & Inzlicht, 2014), and the misattribution of arousal (Inzlicht & Al-Khindi, 2012) attenuate affective processing during task performance. These findings imply that early performance monitoring processes—as represented by the ERN—at least partially reflect the negative valence of erroneous actions (Aarts et al., 2013; Inzlicht et al., 2015).

While the Pe has been subjected to less direct empirical investigation than the ERN, the component has been related to several aspects of error monitoring, including error awareness, behavioral adjustments, and the affective processing of mistakes (Overbeek et al., 2005). As a number of studies have indicated that the Pe is reduced to errors that are not consciously perceived (e.g., Endrass, Reuter, & Kathmann, 2007; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; O'Connell et al., 2007; Steinhauser & Yeung, 2010), the role of the Pe as a marker of error awareness remains the most consistently supported account of its functional significance (Wessel, 2012). The Pe has also been compared to the P3b (Leuthold & Sommer, 1999), a stimulus-locked component elicited by motivationally significant stimuli (cf. Nieuwenhuis, Aston-Jones, & Cohen, 2005). As such, the Pe is larger to more salient mistakes (Leuthold & Sommer, 1999), Pe amplitude is positively correlated with that of the P3 (Davies, Segalowitz, Dywan, & Pailing, 2001), and the Pe covaries with experimental manipulations that induce parametric variation in the P3 (Ridderinkhof et al., 2009). Thus, converging evidence suggests that the Pe reflects the motivational significance of consciously perceived errors.

In addition to the ERN and Pe, error-related engagement of the autonomic nervous systems has been observed, including heart rate deceleration (Crone et al., 2003; Fiehler, Ullsperger, Grigutsch, & von Cramon, 2004; van der Veen, Nieuwenhuis, Crone, & van der Molen, 2004), pupil dilation (Wessel, Danielmeier, & Ullsperger, 2011), and increased skin conductance (Critchley, Tang, Glaser, Butterworth, & Dolan, 2005; Hajcak, McDonald, & Simons, 2003). These reactions are most commonly associated with arousal and orienting toward motivationally relevant stimuli (Sokolov, 1963). In line with this suggestion, error-related autonomic responses are reduced to unaware errors (O'Connell et al., 2007; O'Keeffe, Dockree, & Robertson, 2004; Wessell et al., 2011) and are positively correlated with the amplitude of the Pe (Hajcak et al., 2003). Thus, researchers have suggested that error-related autonomic activity, among other sources of error-related information, may be involved in processes that engender error awareness (Hajcak et al., 2003; Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010; Wessel et al., 2011).

Corrugator Supercilii Reactivity

Most relevant to the current investigation, a recent EMG study has suggested that error-related engagement of the corrugator supercilii muscle is increased within the first 100 ms following errors (Lindström et al., 2013). These findings provide an interesting complement to previous reports of error-related activity in the peripheral nervous system because the corrugator has been consistently associated with the expression and experience of negative affect. For example, facial EMG over the corrugator increases when individuals express anger and fear (Burrows, 2008; Burrows, Waller, & Parr, 2009), when they experience pain (Prkachin, 1992), and when they passively view aversive stimuli or unpleasant scenes (Cacioppo et al., 1986; Lang, Greenwald, Bradley, & Hamm, 1993; Larsen, Norris, & Cacioppo, 2003).

Researchers have also found that EMG responses over the corrugator are related to self-reported effort during physical and mental tasks (de Morree & Marcora, 2010; van Boxtel & Jessurun, 1993; Waterink & van Boxtel, 1994). The putative role of the corrugator both in negative affect and the exertion of effort at first might seem hard to reconcile within a single psychological theory. Recently, however, it has been postulated that mental effort is itself aversive (Botvinick, 2007; Kool, McGuire, Rosen, & Botvinick, 2010; McGuire & Botvinick, 2010). This suggests that activity over the corrugator may represent different aspects of avoidance motivation, to the extent that both negative emotion and effort are common to behavior motivated by avoidance.

In the study by Lindström et al. (2013), increases in facial EMG over the corrugator were correlated with the greater slowing of responses following errors (cf. Rabbitt & Rodgers, 1977), putatively indicative of increased response caution after mistakes (Botvinick et al., 2001; but see Notebaert et al., 2009). This finding provides preliminary evidence suggesting that error-related corrugator activity may serve as a marker of performance monitoring. However, no study to date has explicitly investigated the relationship between facial EMG activity over the corrugator and neural performance monitoring. Thus, in the present study, we sought to do this by simultaneously measuring facial EMG and error-related ERPs during a punished cognitive control task.

Given the mounting evidence that negative affect, motivation, and peripheral arousal are involved in neural performance monitoring, we predicted that increased error-related EMG activity over the corrugator (cEMG) would be associated with larger error-

related ERPs. This notion is supported by anatomical, physiological, and psychological studies of the medial frontal cortex and of performance monitoring. For example, previous work suggests that the medial frontal cortex innervates the corrugator supercilii through afferent connections with the facial motor nucleus of the brain stem (Morecraft, Stilwell-Morecraft, & Rossing, 2004), and that anterior midcingulate cortex may act as a hub that integrates information about pain, negative reinforcement, and cognitive control demands to bias behavior away from undesirable outcomes (Shackman et al., 2011).

It may be hypothesized that the ERN specifically will be associated with error-related cEMG due to their temporal and functional similarities. More precisely, both the ERN and error-related cEMG activity peak within 100 ms of error responses, and both are associated with activity in the medial prefrontal cortex. Moreover, the ERN and cEMG share a common association with both negative affect and the exertion of effort. For example, larger ERNs predict faster reaction times when categorizing negatively valenced words immediately following errors, but not positively valenced words (Aarts et al., 2013), and larger ERNs have been found when task performance is motivated through incentives or punishments (e.g., Legault & Inzlicht, 2013; Pailing & Segalowitz, 2004; Riesel, Weinberg, Endrass, Kathmann, & Hajcak, 2012). These findings suggest that early performance-monitoring processes track the affective valence or motivational significance of task-related actions. Consequently, error-related cEMG may reflect these early processes.

It may also be the case that error-related cEMG is associated with the amplitude of the Pe. Much like the Pe, cEMG following errors could signal the emergence of error awareness or represent orienting toward errors. Further, if error awareness emerges as a result of the accumulation of error-related information (Ullsperger et al., 2010), error-related cEMG could reflect early affective or motivational processes that contribute to this compound error signal. Thus, in either case, it could also be hypothesized that increased cEMG to errors—like other error-related increases in peripheral activity—will correlate with larger Pe amplitudes.

Method

Participants

Seventy-two undergraduate students at the University of Toronto Scarborough participated in return for course credits. All participants had normal or correct-to-normal vision and provided informed consent. Five participants were excluded from all analyses due to either equipment malfunction (one participant) or software malfunction (four participants). In total, 67 participants were included in the data (45 female; mean age 19.1, $SD = 1.6$).

Procedure

Electrophysiological data were measured via EEG and facial EMG while participants completed a two-choice speeded inhibitory control task. In this task, participants responded using keys on a DirectIN PCB keyboard (Empirisoft, New York, NY) in response to two stimuli, the letters *M* and *W*. The presentation probability for each stimulus was asymmetric, giving a correspondingly asymmetric response ratio of 80:20. This manipulation was based on the standard go/no-go task (Simmonds, Pekar, & Mostofsky, 2008), where the high probability target induces a prepotent tendency to respond, which has to be inhibited for low probability targets. Our task modifies the task demands of this manipulation by requiring participants to make an alternative and infrequent response to low-probability stimuli, rather than withholding a response on the no-go trial.

On each trial, participants were required to press the Z key when they saw the frequent M stimulus (low-conflict), and to press the/key when they saw the infrequent W stimulus (high-conflict). The low- and high-conflict stimuli were presented in a yellow or purple font depending upon punishment condition (detailed below). Each trial began with a fixation cross presented for 600 ms, followed by either a low- or high-conflict stimulus that remained on screen until the participant responded or until a maximum of 1,500 ms had passed. Responses were followed by 400 ms of a blank screen before the fixation cross for the next trial appeared, providing a total response to target interval of 1,000 ms.

During the task, performance-contingent punishment varied in intensity between blocks. A punishment manipulation was included in our task paradigm for several reasons: (a) to increase variability in our data by offering circumstances that would produce different amounts of negative affect, with the expectation that this would also vary cEMG accordingly (Lindström et al., 2013); (b) to see if cEMG would be larger in response to a punishment paradigm that has been shown to increase ERN amplitudes (Riesel et al., 2012); and (c) to increase the ecological validity of our task, as failing to adequately monitor performance in real life can sometimes have adverse consequences. For all blocks, participants had a 50% chance to be presented with a 3500 Hz pure tone after making a mistake. Tones were presented from desktop speakers located approximately 3 ft. in front of the participant. The intensity of the sound was manipulated blockwise and within participants, where the punished condition corresponded to a volume of 95 dB (roughly equivalent to a motorcycle engine at 5 m) and the unpunished condition to a volume of 20 dB (roughly equivalent to whispered conversation). The tone lasted 1,000 ms and was followed by an additional delay of 1,500 ms, 500 ms longer than trials without a tone. This punishment manipulation has been previously shown to alter both electrophysiological and behavioral measures of performance monitoring (Riesel et al., 2012).

Participants first completed a practice block of 20 trials without punishment, and then completed 12 normal blocks of 70 trials each (840 trials in total). Of these 12 blocks, six were punished and six were unpunished. Participants alternated between punished and unpunished blocks in sets of three, such that after completing three consecutive blocks in one punishment condition, they then completed three blocks of the other condition, repeating this process until all 12 blocks were completed. Participants were informed whether the set would be punished or unpunished prior to the onset of every block: "During this block you will sometimes be presented with [LOUD/QUIET] sounds when you press the wrong key." The color of the stimuli corresponded with the punishment condition, in order to remind participants whether a current block was punished or unpunished. Both the order of the conditions and the color of the stimuli in relation to the punishment condition were counterbalanced across participants.

After the inhibitory control task, participants completed a demographics questionnaire detailing their age, gender, and number of years speaking English, as well as a number of personality questionnaires not analyzed for the present study.

Behavioral Analyses

We measured choice error rate and reaction time (RT) on correct trials, with the hypothesis that high-conflict trials would produce

more error responses and slower reaction times than low-conflict trials. These results ensure that our task paradigm provides an effective manipulation of conflict level (i.e., that high-conflict trials initiate cognitive control processes).[1]

Electrophysiological Processing

Continuous EEG activity was measured over the cortical midline sites (Fz, FCz, Cz, CPz, Pz, Oz) using 6 Ag/AgCl electrodes embedded in a stretch Lycra cap (Electro-Cap International, Eaton, OH). Vertical electrooculography (VEOG) was recorded using a supra-to-suborbital bipolar montage placed around the right eye. Continuous EMG activity over the left corrugator supercilii muscle was recorded with two miniature EMG Ag/AgCl electrodes (Cacioppo et al., 1986). Both EEG and EMG activity were amplified using an ANT Refa8 TMSi (Advanced Neuro Technology, Enschede, The Netherlands) device. The continuous EEG signal was grounded to the forehead and referenced online to the average of all electrodes.

Offline, the EEG signal was rereferenced to the average of the two bilaterally placed ear lobe electrodes. Of the two electrodes placed over the corrugator supercilii, the one placed more laterally on the face served as a reference for the medial electrode (cf. Cacioppo, Tassinary, & Berntson, 2007). The continuous EMG signal shared the same forehead ground as the continuous EEG signal. Impedances were kept below 5 kΩ for all recordings. Recordings were digitized for the first 19 participants at 512 Hz using Advanced Source Analysis 4.7.11 software. The sampling rate was increased to 1024 Hz for the remaining 53 participants in order to obtain greater temporal resolution for the EMG signal. Prior to filtering and analysis, the data for these first 19 participants were subsequently upsampled offline to 1024 Hz using a spline interpolation procedure in BrainVision Analyzer 2.0 (Brain Products GmbH, Gilching, Germany).

Both EMG and EEG were corrected offline for VEOG artifacts (Gratton, Coles, & Donchin, 1983). EEG data were then digitally filtered between 0.1 and 30 Hz (fast Fourier transform implemented, 24 dB, zero phase-shift Butterworth filter). Automatic procedures were then used to reject EEG artifacts according to the following criteria: voltage steps of more than 25 μV between sample points, a voltage difference of 150 μV within 150-ms intervals, voltages above 100 μV and below -100 μV, and a maximum voltage difference of less than 0.50 μV within 100-ms intervals. The raw EMG signal was filtered offline using a 28–500 Hz IIR bandpass and 60 Hz notch filter, and then smoothed and rectified using a moving average procedure with a time constant of 20 ms (Cacioppo et al., 2007). Artifact rejection for EMG precluded voltages above 100 μV and below -100 μV. For EEG, artifact intervals were rejected from individual channels in each trial.

Epochs commenced 200 ms before the response, and lasted for 1,200 ms. Data for these epochs were averaged within participants independently as a function of trial type (correct and error response) and punishment condition (punished and unpunished), producing four averages for each participant. Prior to dividing averages into time bins, EMG data was standardized (i.e., z transformed) across the entire averaged epoch (Schacht, Nigbur, & Sommer, 2009). EMG averages were then divided into 100-ms time bins, and within-participant values below or exceeding three standard deviations from the within-participant means of each time bin were removed to reduce the impact of outliers.

The ERN and the correct-related negativity (CRN) were each defined as the mean EEG activity between 0 and 100 ms postresponse at the frontocentral midline electrode (FCz) for error and correct trials, respectively. The Pe and its equivalent correct-trial ERP were operationalized as the mean amplitude 200 to 400 ms following the response at the posterior electrode, Pz. Error- and correct-related facial EMG over the corrugator were divided into eight 100-ms time bins starting from 200 ms preresponse and ending at 600 ms postresponse, which encompasses the time course of the ERN and Pe. The signals for EEG and EMG were baseline corrected by subtracting the average voltage 200 ms to 100 ms before the response. In addition to these traditional operationalizations of ERPs and EMG, we also conducted analyses on their corresponding difference waves, denoted as ΔERN (ERN minus CRN), ΔPe (error Pe minus correct Pe, and ΔcEMG (error cEMG minus correct cEMG).

Statistical calculations involving error-related data were based on no fewer than five artifact-free trials (Olvet & Hajcak, 2009). For ERP error data, eight participants in the punished condition and one participant in the unpunished condition had their error data excluded for not meeting this criterion. Additionally, three more participants had just their ERN data removed from both punishment conditions due to intractably high artifact percentages for the FCz electrode. For EMG error data, seven participants in the punished condition and six participants in the unpunished condition had their error data excluded. The average number of errors for the remaining ERP data included in the analyses was 19.15 ($SD = 11.90$, range = 5 to 55), and 24.42 ($SD = 13.66$, range = 5 to 71) for punished and unpunished conditions, respectively. For EMG data, these averages were 19.40 ($SD = 12.22$, range = 5 to 55) and 24.51 ($SD = 13.67$, range = 5 to 71) for the punished and unpunished conditions, respectively. No ERP or EMG correct-related data within the punished or unpunished conditions were excluded.

In order to account for missing data in the distribution of certain variables (e.g., error rate), multilevel models were calculated in SPSS (v22) for all primary analyses, and all main and interaction effects were evaluated using Type III analyses of variance (ANOVAs). The MIXED function in SPSS was used with a restricted maximum likelihood method for fitting. Model construction was driven by parsimony, where models were aimed at addressing hypotheses rather than to maximize explained variance. An unstructured correlations covariance matrix was used to estimate a random intercept for each participant for all fixed effects. For a set of within-participant median splits, we also calculated one-way, repeated measures ANOVAs with Type III sums of squares using the GLM function in SPSS.

For all statistical calculations, effect sizes were denoted using either semipartial R^2 (R^2_β; Edwards, Muller, Wolfinger, Qaqish, & Schabenberger, 2008) or partial η^2 ($\eta_p{}^2$; Cohen, 1973). In order to reduce the incidence of Type I error from multiple comparisons, tests for different dependent variables were corrected using a false discovery rate procedure (Benjamini & Hochberg, 1995; Benjamini & Yekutieli, 2001).

1. We also measured post-error adjustments in choice error rates and reaction time; however, due to the large number of participants whose data were excluded for not containing at least six usable post-error RT trials (40 within the punished condition, and 23 within the unpunished condition), these data were not analyzed. Specifically, about 50% of post-error trials were not usable because they immediately followed a punishment tone. Trials following a punishment tone have timings that are unequal to those that do not follow a tone, and trial timings have been shown to vary post-error slowing effects (Jentzsch & Dudschig, 2009). Further, the general effect of punishment on behavior and physiology is confounded by the immediate effect of a punishment tone on a given trial, necessitating the exclusion of trials that immediately follow punishment tones.

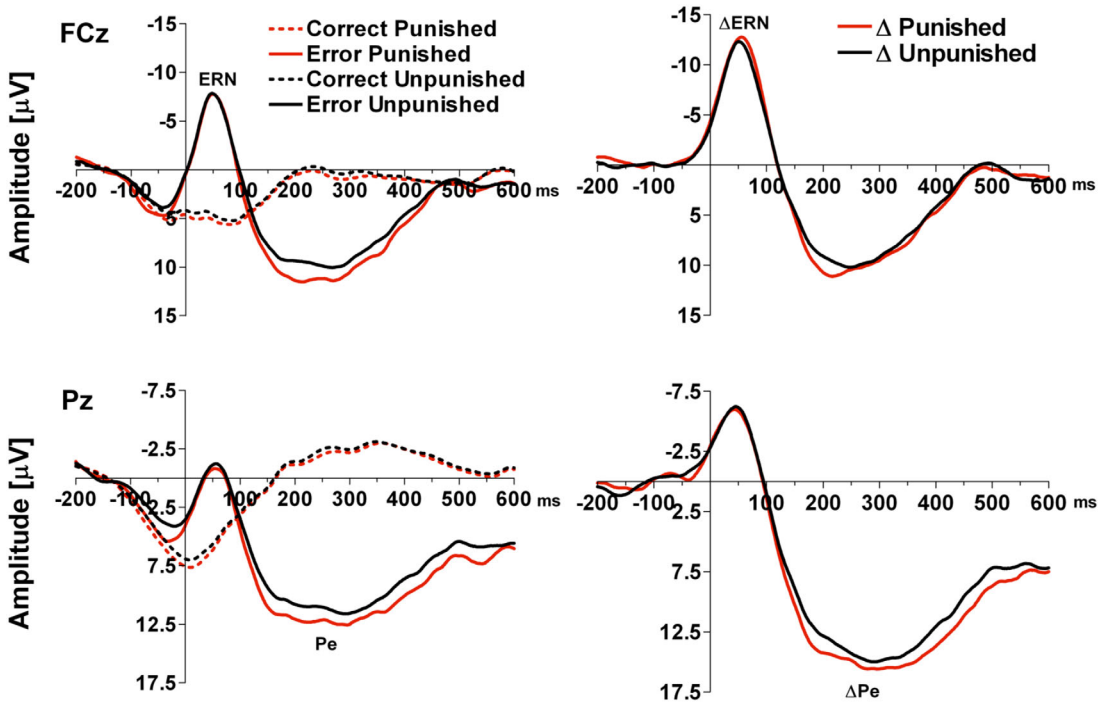

Figure 1. Error- and correct-related ERP amplitudes in the punished and unpunished conditions over the frontocentral (FCz) and posterior (Pz) electrodes. Statistical tests used a 0–100 ms postresponse time bin for the ERN over the FCz electrode, and a 200–400 ms postresponse time bin for the Pe over the Pz electrode. Difference waves of the error- and correct-related ERPs are plotted on the right.

Results

Behavioral Data

We used factorial models to assess the effect of conflict level (low- vs. high-conflict) and punishment (unpunished vs. punished condition) on mean RT and choice error rate. Effect coding denoted the levels of each of the categorical variables. For conflict level, low-conflict trials were = −1 and high-conflict trials were = 1; for punishment, trials in the unpunished condition were = −1 and trials in the punished condition were = 1. In order to reduce the effect of outliers on behavioral data, all responses more than three standard deviations above or below the mean for each participant across cells were excluded; however, this procedure did not exclude any values from any of the behavioral data.

Mean RTs. Confirming that our manipulation of conflict level was successful, participants responded more slowly on high-conflict trials ($M = 427$ ms, $SE = 6$) than on low-conflict trials ($M = 370$ ms, $SE = 6$), ($b = -54.49$, $SE = 3.6$), $F(1,192) = 461.725$, $p < .001$, $R^2_{\beta\ =.72.}$ Conversely, neither the main effect of punishment nor the interaction between punishment and conflict level were significant, both Fs < 1, ps $> .3$, indicating that our punishment manipulation did not influence mean RTs.

Choice error rates. Participants had significantly higher error rates for high-conflict trials ($M = 10.1\%$, $SE = 0.5\%$) compared to low-conflict trials ($M = 0.6\%$, $SE = 0.5\%$), ($b = -0.09$, $SE = 0.01$), $F(1,192) = 365.452$, $p > .001$, $R^2_{\beta\ =.66,}$ further indicating that greater behavioral interference occurred on high-conflict compared to low-conflict trials. As with the RT results, we did not observe a significant main effect of punishment level or Conflict Level × Punishment interaction, Fs < 2.5, ps $> .16$, on choice error rates.

In summary, although our paradigm was effective in manipulating conflict in terms of RTs and error rates, these punishment results fail to replicate previous findings that punishment reduces error rates and increases RTs on correct trials.

Psychophysiological Data

We used factorial models to assess the effect of trial type (correct vs. error) and punishment (unpunished vs. punished) on ERP amplitudes, and the effect of trial type, punishment, and time (seven 100-ms time bins, from −100 preresponse to 600 ms postresponse) on facial EMG activity over the corrugator supercilii. Identical analyses were also performed for difference waves, except in these models only punishment was included as a fixed factor. Categorical variables were effect coded such that correct trials = −1 and error trials = 1, and trials in the unpunished condition = −1 and trials in the punished condition = 1. Because difference waves cannot be calculated for participants with missing error data, the sample size for the difference wave analyses were smaller than for traditional analyses: $n = 60$ for ΔERN, $n = 65$ for ΔPe, and $n = 63$ for ΔcEMG.

ERN and ΔERN (0–100 ms). Error trials were associated with substantially more negative amplitudes ($M = -4.34$ μV, $SE = 0.5$) compared to correct trials ($M = 4.97$ μV, $SE = 0.5$), indicating the presence of a robust ERN, ($b = 9.23$, $SE = 0.71$), $F(1,183.837) = 350.638$, $p > .001$, $R^2_\beta = .66$ (see Figure 1). Contrary to our predictions, we observed no significant main effect of punishment, nor a Punishment × Trial Type interaction, Fs < 1.6, ps $> .20$. In line with our ERN results, ΔERN was less than zero,

Table 1. *Standardized Means for Postresponse cEMG Activity, by Time Bin*

	Response						
	Errors		Corrects				
Time bin (ms)	Mean	*SE*	Mean	*SE*	*F*	*p*	R^2_β
0–100	0.33	0.08	−0.13	0.08	25.757	<.001	0.12
100–200	0.24	0.09	−0.07	0.09	10.106	.002	0.05
200–300	−0.08	0.1	−0.35	0.10	9.863	.002	0.05
300–400	−0.32	0.08	−0.32	0.07	0.001	.991	< 0.01
400–500	−0.25	0.06	−0.19	0.06	1.129	.289	< 0.01
500–600	−0.1	0.06	−0.05	0.05	1.314	.253	< 0.01

$M = -9.27$, $SE = 0.73$, 95% CI (−7.82, −10.72), and there was no significant effect of punishment, $F < 1$, $p > .9$.

Pe and ΔPe (200–400 ms). In the time course of the Pe, error trials were associated with substantially more positive amplitudes ($M = 11.48$ μV, $SE = 0.56$) compared to correct trials ($M = -2.40$ μV, $SE = 0.55$) ($b = -14.54$, $SE = 0.80$), $F(1,193.054) = 613.174$, $p < .001$, $R^2_{\beta\ =.76,}$ indicating the presence of a robust Pe component (see Figure 1). Like the ERN, Pe amplitude was not influenced by the punishment condition or Punishment × Trial Type interaction, both Fs < 1, ps > .3. In line with our Pe results, ΔPe was significantly more positive than zero, $M = 13.95$, $SE = 0.84$, 95% CI (12.27, 15.63), and the effect of punishment was not significant, $F = 3.636$, $p = .061$.

cEMG and ΔcEMG. A single 2 × 2 × 7 model (Trial Type × Punishment × Time, respectively) revealed a significant main effect of trial type, ($b = 0.05$, $SE = 0.14$), $F(1,1656.865) = 16.328$, $p < .001$, $R^2_{\beta\ =.01,}$ such that cEMG was higher for errors ($M = 0.01$ μV, $SE = 0.03$) than for correct responses ($M = -0.14$ μV, $SE = 0.03$). There was also a significant main effect of time, $F(6,1635.524) = 17.165$, $p < .001$, $R^2_{\beta\ =.06,}$ such that cEMG was higher for earlier time bins compared to later time bins; see Table 1. There was no significant main effect of punishment, $F < 1$, $p = .938$. There was also a significant interaction between trial type and time, $F(6,1635.522) = 4.647$, $p < .001$, $R^2_{\beta\ =.02,}$ but no significant interaction between punishment and time, nor a significant three-way interaction between trial type, punishment, and time, both Fs < 1, ps > .8. Simple effects analyses of the interaction between trial type and time revealed that three postresponse bins (0–100, 100–200, and 200–300 ms) showed greater EMG activity for error trials than correct trials, all Fs > 9.5, all ps < .003, all $R^2_{\beta\ >.05}$, see Table 1 and Figure 2. There was no significant difference between error and correct trials for the −100–0 ms preresponse time bin, $F(1,183.782) = 2.440$, $p = .120$, and no significant differences for time bins after 300 ms, all Fs < 3, all ps > .2. For all bins in which there was a significant effect of trial type, there was no effect of punishment or a significant Trial Type × Punishment interaction (all Fs < 1, all ps > .3).

In line with our cEMG results, ΔcEMG had a significant main effect of time, $F(6,749.967) = 6.024$, $p < .001$, $R^2_{\beta\ =.05,}$ but no significant main effect of punishment, $F < 1$, $p = .337$, nor a significant interaction between time and punishment, $F < 1$, $p = .885$. The means for the first three postresponse time bins of ΔcEMG were greater than zero (see Table 2). Neither the preresponse time bin, $M = 0.13$, $SE = 0.09$, 95% CI (−0.06, 0.31), nor any time bin after 300 ms, was more or less than zero.

These results replicate previous findings that facial EMG over the corrugator increases within 100 ms of errors (Lindström et al., 2013). Interestingly, unlike this prior investigation, our results indicate that errors produce increased EMG activity compared to correct responses up to 300 ms after the response. However, consistent with our behavioral results, we did not find robust effects of punishment on either error-related ERPs or cEMG, suggesting that our punishment manipulation was not effective in altering either the behavioral or psychophysiological correlates of control.

Between-Subjects Relationships Between ERPs, Behavior, and Facial EMG

For brevity, we limited further analyses of cEMG data to the time bins where facial EMG over the corrugator was significantly increased for errors compared to correct responses (i.e., 0–100, 100–200, and 200–300 ms postresponse). As our punishment condition did not significantly influence any of our behavioral or physiological measures, we removed this factor from further analyses.

As with our previous results, we used factorial models to assess the effect of trial type (correct/error) on physiology, effect coding correct responses = −1 and error responses = 1. For the ERN and cEMG, ERP amplitudes in the 0–100 ms bin were added to three models as a predictor of each of the three 100-ms postresponse

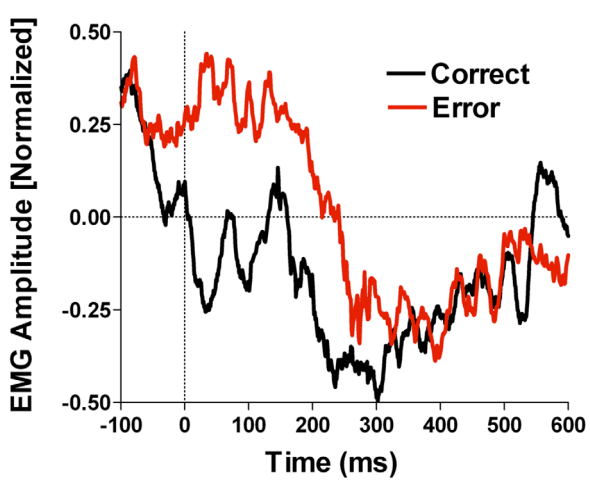

Figure 2. Time-course of the main effect of trial type (correct/error) on normalized EMG over the corrugator. cEMG activity was enhanced between 0 and 300 ms postresponse.

Table 2. *Means of the Difference Wave for Postresponse cEMG Activity, by Time Bin*

	Difference (error − correct)		95% CI	
Time bin (ms)	Mean	*SE*	LB	UB
0–100	0.47	0.09	0.29	0.66
100–200	0.33	0.09	0.15	0.51
200–300	0.25	0.09	0.07	0.43
300–400	−0.01	0.09	−0.19	0.18
400–500	−0.09	0.09	−0.27	0.10
500–600	−0.10	0.09	−0.28	0.09

Note. LB = lower-bound; UB = upper-bound.

cEMG bins, reflecting the shorter duration of the ERN compared to cEMG. Conversely, for cEMG and the Pe, cEMG in the three 100-ms postresponse bins were added to three individual models as predictors of the Pe, reflecting the earlier onset of cEMG (0–300 ms) compared to the Pe (200–400 ms).

The ERN and cEMG. After correcting for multiple comparisons, there were no significant effects of ERN amplitudes, nor any significant Trial Type × ERN amplitude interactions, for any of the three 100-ms postresponse bins, all Fs < 4.7, ps > .03, on cEMG.[2] These results were similar for the ΔERN and the three 100-ms postresponse ΔcEMG bins, all Fs < 1, all ps > 0.3.

cEMG and the Pe. There was no significant main effect of cEMG, nor a significant Trial Type × cEMG interaction for the 0–100 and 100–200 ms postresponse bins on Pe amplitude (all main effects and interactions, Fs < 2.9, ps > .09). There was also no significant main effect of cEMG for the 200–300 ms postresponse bin on Pe amplitude, $F = 3.272$, $p = .072$. However, there was a significant Trial Type × cEMG interaction for this time bin, ($b = -2.39$, $SE = 0.76$), $F(1,211.754) = 9.857$, $p = .002$, $R^2_\beta = .04$ (see Figure 3); simple effects analyses indicated that cEMG was positively correlated with Pe amplitudes, and that this relationship was unique to error responses ($b = 1.98$, $SE = 0.67$, $t = 2.971$, $p = .003$) and not correct ones ($b = -0.414$, $SE = 0.47$, $t = -0.882$, $p = .379$). These results show that ERP amplitudes were positively correlated with cEMG between 200 and 300 ms, and that this relationship was unique to error trials, but not to correct trials. This finding, further illustrated in Figure 4, depicts differences in Pe amplitude between two groups of participants with the 20 highest and 20 lowest cEMG responses in the 200–300 ms time bin.

The results for tests of the ΔcEMG and ΔPe were somewhat similar to those of the trial type data. There was no significant effect of ΔcEMG on the ΔPe for either 0–100 ms postresponse bin

2. Prior to correcting for multiple comparisons, there was a significant trial type and ERP interaction on cEMG for the 100–200 ms bin, ($b = 0.05$, $SE = 0.02$), $F(1,221.891) = 4.626$, $p = .033$ (adjusted $p = .099$). Simple effects of this interaction revealed that for correct responses, ERP amplitudes were positively correlated with cEMG, $b = 0.04$, $SE = 0.02$, $t = 1.957$, $p = .052$. Conversely, for error responses, ERP amplitudes were negatively correlated with cEMG, but this relationship was not statistically significant, $b = -0.01$, $SE = 0.02$, $t = -0.850$, $p = .369$. These findings suggest that cEMG may be positively correlated with the CRN, although the effect was only marginally significant. Conversely, our results do not suggest that cEMG at 100–200 ms is correlated with the ERN.

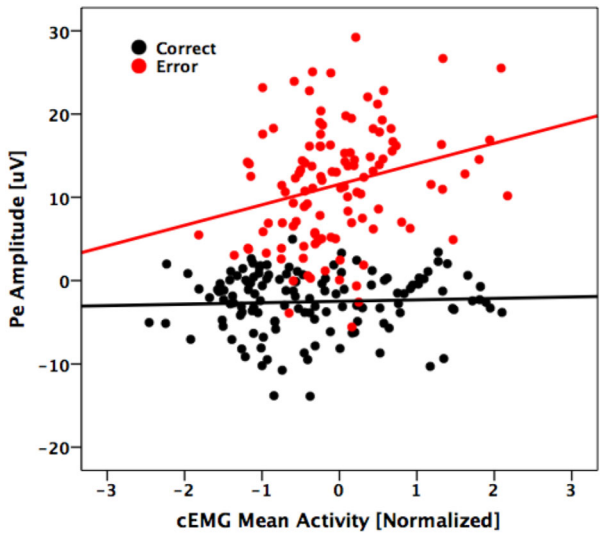

Figure 3. Correlation between error- and correct-related Pe amplitude over the Pz electrode and normalized cEMG in the 200–300 ms time bin.

($b = 1.02$, $SE = 0.59$), $F(1,116.693) = 2.995$, $p = .086$, or 200–300 ms postresponse bin ($b = 0.92$, $SE = 0.66$), $F(1,116.122) = 1.951$, $p = .165$. However, there was a significant effect for the 100–200 ms time bin ($b = 1.49$, $SE = 0.59$), $F(1,115.113) = 6.290$, $p = 0.014$, $R^2_{\beta\ =.05,}$ showing that ΔERP amplitudes for this time bin were positively correlated with cEMG.

cEMG from 100–300 ms and the Pe. As the 100–200 and 200–300 ms cEMG time bins have close temporal proximity and because they both had the same positive direction in their relationship to amplitudes of the ΔPe, we chose to conduct two additional tests by averaging these two bins together for both the cEMG and ΔcEMG data to see if their relationships with the Pe and ΔPe would hold, respectively. These tests were primarily conducted to

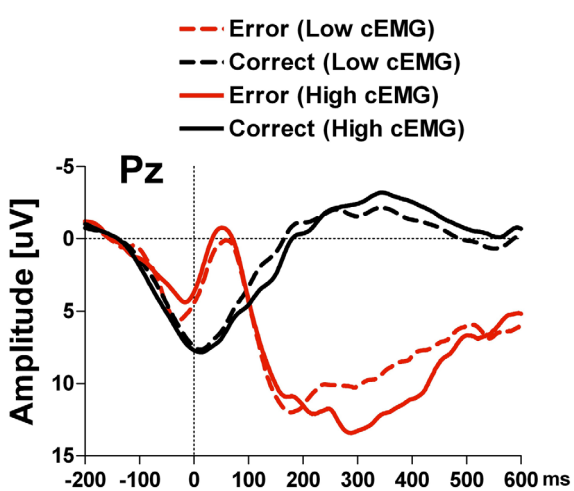

Figure 4. Between-subjects tertiary split of error- and correct-related ERP amplitudes over the poster electrode (Pz), depicting the error-related Pe (red) and its correct-related counterpart (black) between 200–400 ms. Participants with 20 highest (High cEMG) and 20 lowest (Low cEMG) error-related cEMG activity in the 200–300 ms time bin are depicted.

address the discrepancy in time between the trial type and difference wave data, as cEMG was related to the Pe at 200–300 ms, but ΔcEMG was related to the ΔPe at 100–200 ms.

For the trial type data, and after correcting for multiple comparisons, there was no significant main effect of this 100–300 ms cEMG time bin on the Pe, ($b = 2.77$, $SE = 0.93$), $F(1,235.752) = 3.978$, $p = .047$, adjusted $p = .108$, $R^2_{\beta\ =.02}$. However, there was a significant interaction between trial type and cEMG for this time bin ($b = -3.32$, $SE = 1.04$), $F(1,216.223) = 10.174$, $p = .002$, $R^2_{\beta\ =.04}$. Simple effects of this interaction again revealed that there was a significant positive relationship between cEMG and the Pe, and that this relationship was specific to error responses ($b = 2.77$, $SE = 0.93$, $t = 2.983$, $p = .003$) and not correct ones ($b = -0.55$, $SE = 0.55$, $t = -0.989$, $p = .324$). An analysis of the ΔcEMG and the ΔPe for this combined 100–300 ms time bin produced similar results as the trial type data. There was a significant effect of ΔcEMG on the ΔPe ($b = 1.80$, $SE = 0.75$), $F(1,115.008) = 5.795$, $p = .018$, $R^2_{\beta\ =.05,}$ such that the increasing difference between correct and error responses in cEMG was positively correlated with the increasing difference between correct and error responses for the Pe.

Together, these results suggest that cEMG is positively correlated with Pe amplitudes between 100–300 ms after the response, and that this relationship is unique to error responses and not correct responses.

cEMG and Behavior. Based on previous physiological results, we used factorial models to assess effects of trial type and EMG on our behavioral measures of control. We limited this analysis to the only behavioral measure of control for which we had usable data: the effect of cEMG on choice error rate. For choice error rate, we effect coded low-conflict trials as −1 and high-conflict trials as 1.

There were no main effects of cEMG on choice error rate, nor any trial type and cEMG interactions, for any of the three postresponse time bins of interest, Fs < 1.2, ps $> .3$. These results show that, while our task was successful in generating error-related cEMG activity, this activity was not related to choice error rate.

Within-Subject Relationships Between ERPs and Facial EMG

In the models described previously, we found evidence that cEMG on error trials was related to the Pe for between-subjects analyses. These analyses found that participants with larger error-related cEMG had larger Pe amplitudes. In a second set of analyses, we aimed to confirm these results within subjects (i.e., within a single participant). More specifically, are error trials with larger cEMG responses accompanied by larger Pe amplitudes, and smaller cEMG responses by smaller Pe amplitudes? Our between-subjects analyses also failed to find a relationship between ERN amplitudes and cEMG on error trials. As within-subject analyses can detect effects at the level of the individual that fail to emerge at the level of the group, we elected to also analyze the relationship between the ERN and cEMG on error trials.

In order to do this, we conducted additional within-participant median splits of ERP amplitudes based on low and high cEMG activity for the first three postresponse time bins where we found significant main effects of trial type (0–100, 100–200, and 200–300 ms). First, we exported the cEMG activity and ERP amplitudes for each individual error trial within a participant, such that each trial contained an ERP amplitude matched with a normalized cEMG response. Next, we calculated median cEMG responses for each participant, and then split ERP amplitudes for each participant into two equal-sized sets of trials determined by whether a trial's cEMG response was below or above the participant's median. Trials within each set were then averaged together, producing two means for each participant: mean ERP amplitude for low cEMG responses and mean ERP amplitude for high cEMG responses. These means were then compared with three repeated measures ANOVAs for each ERP (i.e., the ERN and the Pe), one for each time bin (0–100, 100–200, and 200–300 ms). Only participants with at least 14 errors (7 for low cEMG, and 7 for high cEMG) and without missing ERP data from the previous between-subject tests were included in these analyses ($n = 49$ for the Pe; $n = 48$ for the ERN). As in previous analyses, results were corrected for Type I error using a false discovery rate procedure (Benjamini & Hochberg, 1995).

The ERN and cEMG. For the 0–100 ms time bin, we found that ERN amplitudes on error trials with high cEMG ($M = -3.09$ μV, $SE = 0.84$) were significantly more negative than ERN amplitudes on error trials with low cEMG ($M = -1.40$ μV, $SE = 0.87$), $F(1,47) = 4.736$, $p = .035$, $\eta_p^2 = .09$. However, this result was no longer significant after correcting for multiple comparisons, adjusted $p = .105$. For the 100–200 and 200–300 ms time bins, ERN amplitudes for low and high cEMG were not significantly different from one another, both Fs < 1, both ps $> .600$. Thus, while we found evidence that error trials with higher cEMG were associated with more negative ERN amplitudes, this result should be interpreted with caution, as it was not robust after correction for multiple comparisons.

cEMG and the Pe. For the 0–100 time bin, Pe amplitudes for low and high cEMG were not significantly different from one another, $F(1,48) = 2.753$, $p = .104$. For the 100–200 ms time bin, Pe amplitudes on error trials with high cEMG ($M = 12.49$ μV, $SE = 1.04$) were significantly more positive than Pe amplitudes on error trials with low cEMG ($M = 11.08$ μV, $SE = 1.09$), $F(1,48) = 6.195$, $p = .016$, $\eta_p^2 = .11$. For the 200–300 ms time bin, Pe amplitudes for low and high cEMG were not significantly different from one another, $F(1,48) = 3.264$, $p = .077$. Confirming the between-subject analyses, these findings provide evidence that cEMG on error trials is positively associated with Pe amplitudes. More specifically, they suggest that, on average, error trials with higher cEMG are concomitant with higher Pe amplitudes, and error trials with lower cEMG coincide with lower Pe amplitudes.

Discussion

The present study explored the relationships between error-related ERPs, facial EMG over the corrugator supercilii, and cognitive control. Our primary objectives were to replicate previous findings that cEMG is sensitive to performance errors, and to relate this peripheral error-monitoring signal to neural performance monitoring and inhibitory control. Replicating one previous study (Lindström et al., 2013), we found that facial EMG activity over the corrugator supercilii was increased to erroneous relative to correct actions within 100 ms of the response. Interestingly, we also found that this difference extended as far as 300 ms after the response, and that time bins of cEMG between 100 and 300 ms after an error were positively related to Pe amplitudes for both between- and within-subject analyses. In contrast to this robust relationship between cEMG and the Pe, however, cEMG on error trials was not significantly related to ERN amplitudes in

between-subjects analyses, and was no longer significant after correcting for multiple comparisons in within-subject analyses. cEMG was also not related to the behavioral implementation of control.

While previous work has investigated relationships between error-related engagement of the peripheral nervous system (e.g., skin conductance, heart rate deceleration) and neural monitoring, this is the first study to precisely explore the relationship between error-related cEMG and ERPs. Our findings show that error-related cEMG is related to the Pe, and may not be consistently related to the ERN. As cEMG has been associated with both negative emotions (Cacioppo et al., 1986) and the exertion of effort (van Boxtel & Jessurun, 1993), our results are consistent with the notion that performance monitoring processes are linked to the affective or motivational significance of errors (Aarts et al., 2013; Inzlicht et al., 2015), particularly within the time course of the Pe. However, as cEMG has been associated with a variety of processes involving affect and motivation, the functional significance of error-related cEMG remains unclear. Further research will be necessary to determine what cEMG represents during error responses, and what its relationships to the ERN and Pe may signify.

Nevertheless, our findings are consistent with other research indicating that error-related engagement of the peripheral nervous system—including skin conductance, heart rate deceleration, and pupil dilation—covaries with the amplitude of the Pe, but not necessarily the ERN (Hajcak et al., 2003; O'Connell et al., 2007; Wessel et al., 2011). Importantly, error-related autonomic arousal has been interpreted as an orienting response to the error (Hajcak et al., 2003). Consequently, the positive association between Pe and cEMG suggests that facial EMG over the corrugator may signify orienting toward motivationally important events (i.e., mistakes) or represent processes that contribute to such orienting. These explanations are supported by proposals that the Pe shares functional and topographical similarities with the P3b, a stimulus-locked ERP sensitive to the motivational relevance of stimuli (see Leuthold & Sommer, 1999; Overbeek et al., 2005; Ridderinkhof et al., 2009).

Given the common finding that Pe amplitude is associated with error awareness (e.g., Endrass, Franke, & Kathmann, 2005; Endrass et al., 2007; Nieuwenhuis et al., 2001), our findings also suggest an interesting possibility: processes reflected in corrugator activity might be involved in or represent the generation of error awareness. Recently, Ullsperger et al. (2010) suggested that the subjective awareness of an error might arise when the accumulation of error-related signals becomes sufficiently strong to differentiate erroneous actions from noise. Evidence contributing to this compound error signal may consist of a wide variety of pre- and postresponse information, including stimulus features, response conflict, an efference copy, proprioception, interoception, and sensory input. Subsequent accrual of this information may be reflected in amplitude of the Pe (Wessel et al., 2011). Thus, processes reflected by cEMG may serve as yet another source of information that accumulates during the generation of error awareness. However, as the latency of cEMG and the Pe are overlapping, it is equally probable that error-related cEMG may reflect the emergence of attentional orienting or error awareness, rather than contributing to its generation.

Although we found that error-related cEMG was associated with the amplitude of the ERN for within-subject analyses, this finding was no longer significant after correcting for multiple comparisons. Further, we found no relationship between the ERN and error-related cEMG for our between-subjects analyses. This is an unexpected result, given the extensive research linking activity in the medial frontal cortex and cognitive control with negative affect (see Shackman et al., 2011), and consistent findings that error-related cEMG activity and the ERN share temporal and functional similarities (see also Lindstrom et al., 2013). However, as the ERN peaks rapidly after the response, its amplitude may be based on relatively limited sources of information, such as response conflict arising from ongoing stimulus evaluation (Yeung et al., 2004) or reward prediction error signals (Holroyd & Coles, 2002). Conversely, the Pe occurs later and is potentially associated with a wider array of error-related signals, such as proprioceptive feedback and sensory action effects (Ullsperger et al., 2010). Thus, the relative richness of error-related information reflected in the Pe might explain why this component, rather than the ERN, has a stronger and more consistent association with error-related cEMG.

Limitations and Future Directions

The present findings should be interpreted in light of a number of limitations. First, our results regarding cEMG and error-related ERPs are correlational in nature. Therefore, we cannot determine whether processes that support both cEMG and ERPs are causally related to one another, or if confounding factors may account for their relationship. Secondly, the inefficacy of our punishment manipulation in varying any metric of control in our study suggests that our punishment was not powerful enough to provide a substantive source of avoidance motivation. However, the effect of punishment on physiology and behavior has not always been consistent, and studies using similar punishment methodologies have found punishment effects (e.g., Riesel et al., 2012; Saunders et al., 2015) while others have not (e.g., Stürmer, Nigbur, Schacht, & Sommer, 2011). Thirdly, our data do not allow for a straightforward characterization of the functional significance of cEMG, which has previously been associated with a variety of nonisomorphic processes related to avoidance motivation. Future studies should attempt to disentangle the cognitive, affective, and motivational factors that may give rise to error-related cEMG, such as through a combination of affective priming (see Aarts et al., 2013) and self-reported effort.

Importantly, however, our findings are generative, providing several new directions for future research. What kinds of cognitive, motivational, and affective manipulations influence the magnitude of error-related facial EMG over the corrugator? Does cEMG covary with individual differences related to neural performance monitoring? For example, increased ERN amplitudes are commonly reported in anxious psychopathologies (Weinberg et al., 2012). Future research could use error-related cEMG as a further measure of altered performance monitoring in clinical samples. Finally, in light of the observed association between error-related cEMG and Pe amplitude, it may be the case that the processes cEMG represents are involved in orienting to error responses, the emergence of error awareness, or both. Thus, future research could determine if error-related cEMG—akin to the Pe—is reduced to unaware errors.

Conclusions

Taken together, our study confirms the presence of error-related facial EMG activity over the corrugator supercilii during an inhibitory control task, and shows that this activity correlates with

the Pe, an error-related potential most commonly associated with error awareness. These findings further suggest that error-monitoring processes are concomitant with rapid and transient changes in affect or motivation, and that these processes may be involved in orienting toward errors or in the emergence of error awareness.

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