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Is it a painful error? The effect of unpredictability and intensity of punishment on the error-related negativity, and somatosensory evoked potentials

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A B S T R A C T

We examined how predictable and unpredictable punishment intensity contingent on error commission modulated ERN amplitudes. We recorded the ERN in 35 healthy volunteers performing the Eriksen flanker task. Errors were punished with predictable nonpainful, painful or unpredictable electrical stimulation. Furthermore, we investigated trait anxiety. We observed that ERN amplitudes did not differ across conditions, nor were there significant effects of anxiety. In contrast, we found that predictable painful punishments led to smaller Error Positivity (Pe). The effects of predictability and intensity were present in Somatosensory Evoked Potentials elicited by the punishments. N1 amplitudes were increased for painful compared to nonpainful stimulation, and P2/P3 amplitudes for painful compared to nonpainful, and for unpredictable compared to predictable stimulation. We suggest that unpredictability and increased painfulness of punishments enhance the potential motivational significance of the errors, but do not potentiate ERN amplitudes beyond the ones elicited by errors punished with predictable nonpainful stimulation.

1. Introduction

To err is human. Fortunately, most of these errors are inconvenient, yet harmless. Still, on some occasions the commission of an error can have widespread implications. Researchers commonly agree that throughout evolution this has resulted in the development of a performance monitoring system that is specialized in the detection and correction of erroneous responses (Gehring, Goss, Coles, Meyer, & Donchin, 1993). Three decades ago, Falkenstein, Hohnsbein, Hoormann, and Blanke (1991) and Gehring et al. (1993) independently discovered that the commission of an error triggered a stable response-locked, frontocentral negative-going deflection in the electroencephalographic signal (EEG), peaking approximately 50 ms after the commission of the error. This response was termed Error-Related Negativity (ERN), and has since been observed after error commission in numerous cognitive tasks (e.g., Eriksen flanker tasks: Gehring et al., 1993; Go/No-Go tasks: Brázdil, Roman, Daniel, & Rektor, 2005; Stroop tasks: Hirsh & Inzlicht, 2010; etc.). The ERN is followed by another centroparietal positive-going deflection occurring 200–400 ms post-error (Falkenstein et al., 1991; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). This component is termed the Error Positivity (Pe). While the ERN reflects, according to some authors, a cognitive signal (e. g., Carter et al., 1998; Dehaene, Posner, & Tucker, 1994; Falkenstein et al., 1991; Gehring et al., 1993; (Holroyd & Coles, 2002; Yeung, Botvinick, & Cohen, 2004), it is becoming increasingly clear that it also conveys emotional and motivational aspects of the error (Hajcak, 2012; Proudfit, Inzlicht, & Mennin, 2013). Accumulating evidence has revealed stable, trait-like differences in ERN amplitudes that appear to be related to dispositional differences in threat sensitivity (reviewed in Weinberg, Dieterich, & Riesel, 2015). Furthermore, experimentally increasing the threat-value of errors has resulted in transient increases in ERN amplitudes (e.g., Meyer & Gawlowska, 2017; Riesel, Weinberg, Endrass, Kathmann, & Hajcak, 2012). In line with these findings, Hajcak (2012) proposed that, besides being cognitively salient, errors can also be conceived as endogenous threats that have the potential to put an organism in grave danger. As such, he argued that the ERN does not

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merely represent neural activity related to the detection of the error per se, but in fact reflects the first evaluation of its motivational significance, triggering both increased cognitive control and defensive responses where needed.

Still, many questions remain about which threat-related aspects of errors are captured by the ERN exactly. In the past, research has shown that the threat-value of exogenous threats (e.g. aversive electrical shocks) can be increased by making the threats unpredictable (e.g., Clark, Brown, Jones, & El-Deredy, 2008; Grillon, Baas, Lissek, Smith, & Milstein, 2004; Shankman, Robison-Andrew, Nelson, Altman, & Campbell, 2011; Wieser, Reicherts, Juravle, & von Leupoldt, 2016). Accordingly, preliminary studies on the ERN suggest that the same holds for endogenous threats such as errors. Tullett, Kay, and Inzlicht (2015) showed that ERN amplitudes were increased after participants had read about the randomness of the world compared to when they had read about the order of the world. Relatedly, Jackson, Nelson, & Proudfit (2015) found a larger ERN in the presence of unpredictable (i.e., unpredictable task-irrelevant tone sequences) compared to predictable contextual cues (i.e. predicable task-irrelevant tone sequences). Their results were furthermore replicated within adolescent- and child-samples (Speed, Jackson, Nelson, Infantolino, & Hajcak, 2017). Finally, Tan, Van den Bergh, Qiu, and von Leupoldt (2019) recently showed that experiencing unpredictable rather than predictable dyspnea (i.e., breathlessness, an inherently aversive and threatening event) while performing an interoceptive forced choice reaction time task enhanced the amplitude of the interoceptive ERN, but only when the unpredictable dyspnea condition was experienced first.

Interestingly, while all the aforementioned studies confirmed that unpredictability increased ERN amplitudes, most of them relied on task-irrelevant stimuli, and all of them exclusively examined unpredictability that was unrelated to behavioral performance, allowing contemporary conclusions to exclusively account for contextual unpredictability. In many situations, however, the unpredictability is directly related to the task at hand: It is the error itself that has unpredictable consequences.

In the current study, we examined the effects of the (un)predictability and intensity of punishing electro-cutaneous stimuli contingent on error commission on the ERN amplitude. We recorded high-density EEG in healthy volunteers while they performed an arrowhead version of the Eriksen flanker task under three conditions: Errors were either followed by (1) predictable nonpainful electrical stimulation, (2) predictable painful electrical stimulation, or (3) either nonpainful or painful electrical stimulation (unpredictable condition).

Based on the aforementioned evidence showing that punishments enhance ERN amplitudes (Meyer & Gawlowska, 2017; Riesel et al., 2012), we predicted that ERN amplitudes would be larger within the predictable painful condition compared to predictable nonpainful condition. Additionally, given that unpredictability potentiates the processing of potential threats, we predicted an even more pronounced increase for ERN amplitudes in the unpredictable, potentially painful condition compared to the predictable painful condition. As both Riesel et al. (2012) and Meyer and Gawlowska (2017) reported that participants with higher levels of trait anxiety were more susceptible to state manipulations of punishment, we furthermore hypothesized that our effects would be more pronounced in highly anxious individuals.

As a secondary analysis, we examined the effects of predictability and intensity on the Somatosensory Evoked Potentials (SEPs) elicited by the post-error electrical stimulation. Considering that electrical stimulation varied in intensity, we predicted a higher amplitude of the signal after more intense stimuli (Miltner, Johnson, Braun, & Larbig, 1989). Moreover, we also expected unpredictability to potentiate these effects. Indeed, previous studies have found increased P2 and LPC amplitudes for motivationally salient events, such as emotional (e.g., pleasant and unpleasant pictures elicited larger LPP amplitudes as compared to neutral pictures; Hajcak & Olvet, 2008), and unpredictable stimuli (e.g., unpredictable valence of pictures elicited larger P2 amplitudes as compared to predictable valence; Dieterich, Endrass, & Kathmann,

2016). Finally, we explored the effects of predictability of punishments on the Pe.

2. Methods

2.1. Participants

Forty healthy volunteers (25 females, 2 left-handed) between the ages of 18 and 40 ($M = 22.4$, $SD = 3.4$) and with normal or corrected-to-normal vision and hearing were recruited to participate in the experiment and received either course credits or a monetary compensation. Participants were recruited through the online recruitment system of the Faculty of Psychology and Educational Sciences at KU Leuven (Experiment Management System; EMS), the distribution of posters around campus, and social media. Exclusion criteria were pregnancy, past or present cardiovascular, respiratory, neurological, or psychiatric diseases, pacemakers or other electronical implants, acute or chronic pain, sleep deprivation, and regular drug/medication use. Four participants were excluded from the analyses because they made fewer than six errors in at least one condition (Olvet & Hajcak, 2009a), and one additional participant was excluded due to excessive EEG artifacts, resulting in a final sample of 35 participants (22 females, 2 left-handed) with mean age of 22.1 ($SD = 2.8$). At the start of the experiment, all participants provided written informed consent in accordance with the Social and Societal Ethics Committee (SMEC) of the KU Leuven (G-2017 11 1011) and declared in writing that none of the aforementioned exclusion criteria applied to them.

2.2. Electro-cutaneous painful and non-painful stimulation

Painful and nonpainful stimuli were square-wave electro-cutaneous pulses, generated by a DS5 bipolar constant current stimulator (Digitimer Ltd, Welwyn Garden City, UK) and delivered via a bar stimulating electrode (Digitimer Ltd, Welwyn Garden City, UK) positioned on the ventral part of the non-dominant forearm. The bar electrode was composed of two durable stainless steel disks (8 mm diameter, 30 mm spacing), filled with K-Y Jelly.

Prior to the start of the experiment, two different stimulation intensities were individually selected. A first *nonpainful* intensity was set to a level that corresponded to an innocuous sensation. A second *painful* intensity was selected to correspond to a clearly uncomfortable/painful sensation that demanded some effort to tolerate. In order to do so, both pulse amplitude and, in some participants, duration were adjusted. Specifically, participants were presented with a range of increasingly intense electrical stimulations (with 0.2 mA step increases). Calibration always started with a 0.2 mA pulse of 4 ms. At each following step, participants rated the perceived intensity on a scale from 0 (not painful at all) to 100 (worst pain imaginable), with a rating of 50 representing the painfulness threshold. Nonpainful intensities were set to a rating of about 25, while painful intensities were set to a rating of about 75. Whenever the 75 threshold was not reached by merely increasing pulse amplitude (for $N = 9$ participants), pulse duration could be extended up to maximally 10 ms. Whenever this occurred, the duration of the non-painful stimulation was matched to the duration of the painful one.

2.3. Experimental procedure

Upon their arrival at the lab, participants were comfortably seated at approximately one meter distance from a computer screen displaying some general information about the experiment. All other instructions, questionnaires, and ratings would likewise be displayed on the screen. After providing written informed consent and signing the exclusion criteria form, participants completed the questionnaires. Next, the EEG net and bar electrodes were fitted, after which the nonpainful and painful stimulation intensities were individually calibrated.

2.3.1. Flanker task

The experimental task comprised an arrowhead version of the Eriksen flanker task (Eriksen & Eriksen, 1974; Sucec, Herzog, Diest, den Bergh, & von Leupoldt, 2019), in which participants were presented with sequences of five horizontally aligned arrowheads (i.e., "<<<<<", ">>>>>", "<<><<", or "><<>>>"). Participants were instructed to respond as quickly and as accurately as possible to the direction of the central arrowhead by pressing either the left (if the central arrowhead pointed to the left) or the right (if the central arrowhead pointed to the right) mouse button with their dominant hand. The four surrounding arrowheads (i.e., the flankers) that pointed in the same (congruent trial) or opposite (incongruent trial) direction of the central arrowhead, were distractors that were to be ignored.

The task was performed under three conditions that differed with respect to the consequences of erroneous responses (Fig. 1). In all three conditions, mistakes were punished with an electrical stimulation to the non-dominant forearm. However, the intensity of the punishment (painful/nonpainful) and its predictability (predictable/unpredictable) varied across conditions. In a *predictable nonpainful* condition, mistakes were always followed by the predetermined nonpainful electrical stimulation (100 % reinforced with nonpainful stimulus); in a *predictable painful* condition mistakes were always followed by the predetermined painful electrical stimulation (100 % reinforced with painful stimulus); and in an *unpredictable* condition mistakes were always followed by an electrical stimulation that could either have the predetermined painful or nonpainful intensity (theoretically, 50 % painful and 50 % nonpainful stimuli). Participants were told at the start of each condition which one was about to start, and about the contingencies of the stimuli.

Following general instructions, participants completed a 20-trial practice block during which they received written feedback ("correct!" or "wrong!") on a trial-by-trial basis, whereas mistakes were never punished. Afterwards, self-selected stimulation intensities were reassessed and adjusted if necessary.

Each condition included four blocks of 60 trials each (30 congruent and 30 incongruent trials presented in a pseudo-random order, with at most three consecutive trials with central arrowheads pointing in the same direction and at most three consecutive trials of the same congruency type). The order of presentation of the conditions was counterbalanced across participants. Each trial started with the presentation of a white-colored fixation cross against a black background for a random duration of 600–1000 ms, followed by the presentation of the white-colored arrowhead sequence (Arial, 56) for 200 ms (see also Sucec et al., 2019). Participants could respond for 1000 ms, after which the trial was aborted and a random intertrial interval started, varying between 600 and 1000 ms. Incorrect responses were followed by electrical stimulation 200 ms after the response was given. Following each block (i.e., after every 60 trials), participants received written feedback on their performance in the previous block (in accordance with Hajcak & Foti, 2008). This manipulation was installed to maximize the total number of errors while also maximizing participants' task engagement

by encouraging either faster or more accurate responding, respectively. If participants performed below 75 %, a message emphasizing accuracy was displayed (i.e., *"Please try to be more accurate."*). If participants performed above 90 %, a message emphasizing speed was displayed (i. e., *"Please try to respond faster."*). Performances that were located between either two levels, were followed by the message: *"You're doing a great job."* At the end of each condition, participants rated the perceived stimulation intensities and their experienced level of anxiety/discomfort during that condition. Additionally, the experimenter entered the lab to reassess electrode impedances. At the end of the experiment, participants were debriefed and thanked.

2.4. Behavioral measures

2.4.1. Questionnaires

Prior to the start of the experiment, participants completed several questionnaires including the *State-Trait Anxiety Inventory – Trait* (STAI-T; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), the *Anxiety Sensitivity Index – 3* (ASI-3; Taylor et al., 2007), the *Pain Catastrophizing Scale* (PCS; Sullivan, Bishop, & Pivik, 1995), the *Intolerance of Uncertainty Scale* (IUS; Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994), the *Life Orientation Test – Revised* (LOT-R; Scheier, Carver, & Bridges, 1994), and the *Positive And Negative Affect Schedule* (PANAS; Watson, Clark, & Tellegen, 1988). These questionnaires were part of a larger prospective ongoing study, and are therefore not part of the present report. Descriptive statistics are presented in the supplementary materials. Given that the current study examined the modulatory effect of trait anxiety on ERN amplitudes as a secondary hypothesis, we did include the STAI-T in further analyses. The STAI-T is a 20-item self-report questionnaire measuring trait anxiety. Items are rated on a four-point Likert scale ranging from 1 (almost never) to 4 (almost always). Theoretically, scores can vary between 20 and 80, with higher scores corresponding to greater levels of trait anxiety. In the current sample, STAI-T scores ranged between 23 and 62 ($M = 41.89$, $SD = 9.32$), and showed excellent internal consistency (Cronbach's Alpha = .91).

2.4.2. Ratings

At the end of each condition, participants rated the intensity of the electrical stimulation they experienced during that condition. Specifically, they were asked: *"How intense was the pain during the stimulus?"*, on a scale from 0 (not painful at all) to 100 (the highest pain I could possibly imagine). A second rating probed the experienced anxiety/discomfort during that condition on a five-point Likert scale, ranging from 1 (not anxious) to 5 (extremely anxious). Specifically, they were asked: *"How uncomfortable or anxious did you feel during the previous condition?"*.

2.4.3. Performance assessment

Behavioral performance during the flanker task was assessed based

CORRECT RESPONSE ✔   **INCORRECT RESPONSE** ✖

ALL CONDITIONS

PREDICTABLE PAINFUL CONDITION

PREDICTABLE NONPAINFUL CONDITION

UNPREDICTABLE CONDITION
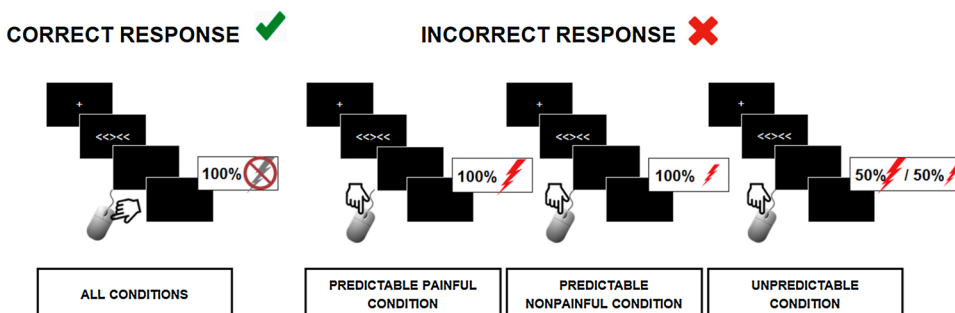
Fig. 1. Experimental Design. All trials started with the presentation of a fixation cross for a random interval (600–1000 ms), followed by the presentation of the arrowhead sequence for 200 ms. Then, the screen cleared and participants could respond for 1000 ms, after which the trial was always aborted and an intertrial interval of 600–1000 ms commenced. Whenever an erroneous response was given, an electrical stimulation was applied 200 ms post-response. The exact intensity of the stimulation varied across conditions.

on (1) error rate, (2) reaction times, and (3) post-error slowing. Error rates were computed separately for each condition as the percentage of errors that were committed in valid trials (i.e., trials on which a response was registered) of that condition. Likewise, average reaction times on correct and incorrect trials were computed for each condition. Post-error slowing following predictable, and unpredictable painful/nonpainful stimuli were quantified as the average difference in reaction time between post-error trials and the associated pre-error trials (i.e., Mean[RT $(E + 1) - RT(E−1)$] for all errors E; see Dutilh et al., 2012).

2.5. EEG recording and processing

High-density EEG was continuously recorded at 1000 Hz sampling rate, using a 129-channel system (Philips Electrical Geodesics Inc., Eugene, USA), with the vertex sensor as reference electrode (see Supplementary Fig. 1). Electrode impedances were repeatedly checked over the course of the experiment, and were kept below 50 kΩ.

Offline processing of the collected EEG signals was carried out with Letswave 6 (https://www.letswave.org/), an open source software running in Matlab (version R2018b). Raw EEG signals were first notch (48−52 Hz) and band-pass (0.1−30 Hz) filtered using 4th order Butterworth filters. Next, signals were re-referenced to an average of all channels (Cz excluded). The filtered and re-referenced data were then segmented into response-locked epochs starting 500 ms before response onset, and continuing up to 1000 ms post-response. Individual Independent Component Analyses (ICA; Jung et al., 2000) were run to identify and remove (ocular) artifacts (on average 3.8 of the 30 components removed), followed by a baseline correction over the 500 to 300 ms interval preceding the response onset (in accordance with Jackson, Nelson, & Proudfit, 2015). Furthermore, all epochs exceeding a 100 μV maximum amplitude were excluded from further analyses (18 % of the epochs).

The residual error -epochs (on average 21 epochs/condition, $SD = 14$ epochs/condition) were then averaged separately per participant and per experimental condition. Error-epochs of the unpredictable condition were additionally averaged according to punishment intensity. Next, error- and stimulation-related ERP-components were extracted.

Following a visual inspection of the data and in accordance with previous studies (Sucec et al., 2019; Tan et al., 2019), the ERN was defined as the average amplitude around FCz (electrodes 5, 6, 7, 11, 12, 106) within the first 100 ms following an erroneous response, and the Pe was defined as the mean amplitude between 120 and 200 ms after error response around the electrode PCz (electrodes: 55, 61, 62, 72, 78). We also identified the main positive deflection of the somatosensory evoked potentials (SEPs) elicited by the electrical punishment around Cz (electrodes 129, 55) within the 200–400 ms interval following stimulation (a P2/P3 component, Miltner et al., 1989). Further inspection of the data revealed potential effects of predictability and intensity at other latencies. Therefore, we also extracted an earlier negative component (N1) and a late positive complex (LPC) as a post-hoc analysis. N1 amplitudes were defined as the most negative peak around Cz (electrodes 129 and 55) within the 70–170 ms interval following stimulation. The LPC was defined as the average signal amplitude around CPz and Pz (electrodes 55 and 62) within the 400–600 ms interval following stimulation (in accordance with Juravle, Reicherts, Riechmann-Weinstein, Wieser, & von Leupoldt, 2017).

Independently, EEG preprocessing was blindly performed by another of the co-authors using Brain Electrical Source Analysis Research 6.0 (BESA GmbH, Gräfelfing, Germany). The co-author was blind to the condition, initial hypotheses, and results. The blind analysis was meant to investigate whether different methodological choices in the pre-

processing stage could influence the final results. As this blind analysis yielded the same final result for ERN, these data were not included in further analyses. Details of the blind analysis are provided in the Supplementary Material.

2.6. Statistical analyses

Depending on the outcome variable, analyses consisted of either one-way or two-way Repeated-Measures Analyses Of Variance (RM-ANOVAs), or their non-parametric equivalent (Friedman's test) performed in SPSS (version 25.0.0.1). Specifically, for outcome variables that preceded the actual punishment, and that were therefore unaffected by its intensity (i.e., error rates and ERN/Pe), analyses included only *Condition* (predictable painful, predictable nonpainful, and unpredictable) as a within-subject factor. For outcome variables that followed the punishment, and that could therefore be affected by its intensity (i.e., post-error slowing and SEP components), both *Predictability* (predictable, unpredictable) and *Intensity* (painful, nonpainful) were included as within-subject factors. Reaction time data were analyzed using a 2 (*Response*: correct, error) x 3 (*Condition*: predictable painful, predictable nonpainful, and unpredictable) RM-ANOVA. Additionally, post-hoc inspections of the grand average waveforms revealed potential effects of *Predictability* and *Intensity* on SEP components other than the P2/P3. Therefore, we exploratively analyzed the amplitude of the N1 and the LPC with an additional 2 (*Predictability*: predictable, unpredictable) x 2 (*Intensity*: painful, nonpainful) RM-ANOVA. Greenhouse-Geisser (G-G) corrections to the degrees of freedom for within-subject factors were applied when Mauchly's test of sphericity was significant, signaling that the sphericity assumption had been violated. Partial eta-squared (η_p^2) and Cohen's *d* were computed as measures of effect size for *F*-tests and *t*-tests, respectively. All significant effects ($p < .05$) were followed-up with Bonferroni-corrected post-hoc pairwise comparisons.

As a secondary analysis investigating the potential modulatory effects of trait anxiety for the behavioral and ERP results, STAI-T scores were added to the model as covariate-of-interest. Significant effects were again followed-up with Bonferroni-corrected post-hoc pairwise comparisons.

2.6.1. Control analyses

Given that gender and performance differences between conditions have been shown to influence ERN amplitudes (e.g., Gehring et al., 1993; Larson, South, & Clayson, 2011), we repeated our ERN-analyses controlling for gender, and we ran Pearson correlations (two-tailed) for all ERN amplitudes and the associated stimulation intensities (both objective and subjective), error rates, reaction times, post-error slowing, and post-condition anxiety/discomfort ratings. Bonferroni-corrections were applied to reduce the risk of type I error inflation.

Furthermore, although erroneous responses in the unpredictable condition were theoretically followed by an equal number of painful and nonpainful punishments, this might not have been the case in practice. Indeed, every error was equally likely to be punished with a painful or nonpainful electrical stimulation, irrespective of the punishment intensity on previous error-trials. To check the relative over- or under-representation of painful compared to nonpainful stimulations in the unpredictable condition, we exploratively investigated whether participants differed with respect to the proportion of painful compared to nonpainful punishments in the unpredictable condition (P/N), and whether this difference in itself might have influenced the associated ERN amplitudes. In order to do so, we correlated (Spearman, two-tailed) the ratio P/N with the ERN amplitudes of the unpredictable condition.
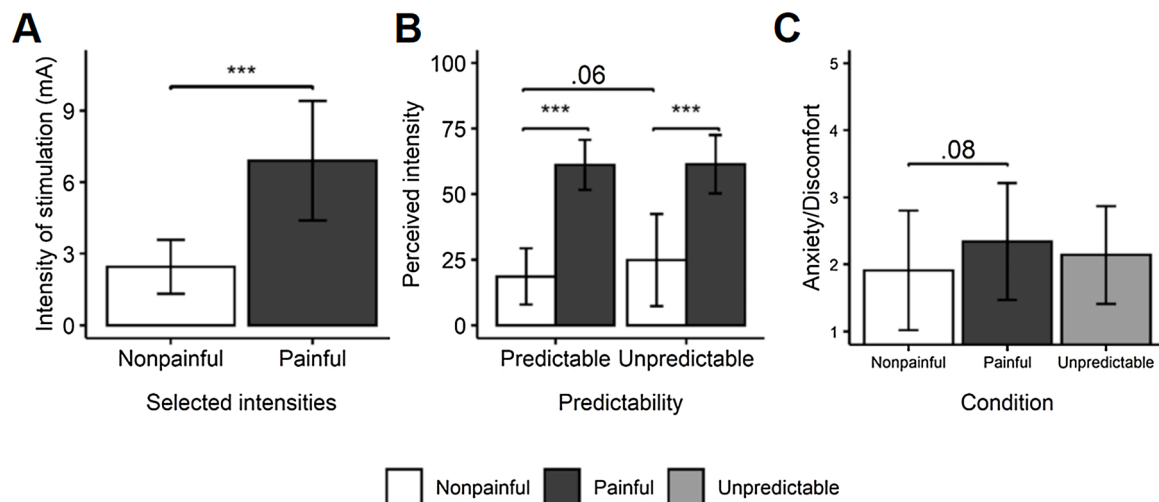
Fig. 2. Objective and perceived differences in stimulation intensity and anxiety. **A**. Mean objective intensities of the selected intensities. **B**. Mean perceived intensity of the stimulations according to Predictability and Intensity on a scale ranging from 0 (not painful at all) to 100 (worst pain imaginable). **C**. Mean perceived differences in anxiety/discomfort according to experimental condition. Error bars represent the standard deviations. *** $p < .001$, Bonferroni-corrected.

3. Results

3.1. Objective and perceived differences in stimulation intensity and anxiety

3.1.1. Intensity of stimulation

Stimulation lasted on average 5 ms ($SD = 2$ ms). Selected intensities ranged between 0.9 mA and 5.2 mA for the nonpainful stimuli ($M = 2.45$ mA, $SD = 1.13$ mA), and between 2.6 mA and 10 mA for the painful stimuli ($M = 6.90$ mA, $SD = 2.50$ mA). A paired-sample *t*-test confirmed that intensity levels differed significantly, $t(34) = 14.03$, $p < .001$, $d = 2.37$ (Fig. 2A). An Anxiety x 2 (Intensity) RM-ANCOVA revealed no effect of Anxiety (all *p*'s > .38).

Table 1

Mean (SD) of Behavioral and ERP Results by Condition or by Predictability and Intensity.

	Condition		
	Predictable painful	Predictable nonpainful	Unpredictable
Error rate (%)	9.71 (4.45)	12.03 (8.17)	11.19 (6.66)
RT error-trials (ms)	355 (42)	349 (43)	347 (37)
RT correct-trials (ms)	427 (52)	423 (39)	421 (39)
ERN (μV)	−1.23 (2.48)	−1.25 (2.47)	−1.12(2.32)
Pe (μV)	5.21 (4.14)	6.30 (4.12)	6.35 (4.55)

	Predictable		Unpredictable	
	Painful	Nonpainful	Painful	Nonpainful
Post-error slowing (ms)	27(26)	22 (29)	21 (38)	14 (39)
N1 (μV)	0.80 (6.23)	4.78 (4.52)	1.27 (5.42)	3.78 (4.46)
P2/P3 (μV)	15.85 (7.07)	12.46 (5.31)	17.09 (6.52)	14.22 (6.20)
LPC (μV)	5.59 (4.77)	4.79 (3.88)	6.69 (4.31)	5.98 (3.60)

Note. Pre-punishment measures are detailed in the upper section of the table, while post-punishment measures are detailed in the lower section. RT = Reaction Time.

3.1.2. Perceived intensity

A 2 (*Predictability*) x 2 (*Intensity*) RM-ANOVA examining the subjectively perceived intensity of the electrical stimuli presented as punishment of the errors of $n = 33$ participants[1] revealed significant main effects of *Predictability*, $F(1,32) = 5.02$, $p < .05$, $\eta_p^2 = .14$, and *Intensity*, $F(1,32) = 263.00$, $p < .001$, $\eta_p^2 = .89$, and a significant *Predictability* x *Intensity* interaction, $F(1,32) = 6.51$, $p < .05$, $\eta_p^2 = .17$ (Fig. 2B). Post-hoc pairwise comparisons indicated that participants consistently rated the painful intensity as more intense than the nonpainful intensity in both the predictable ($M_{painful} = 61.15$, $SD_{painful} = 9.56$ versus $M_{nonpainful} = 18.58$, $SD_{nonpainful} = 10.76$), $t(32) = 20.94$, $p < .001$, $d = 3.65$, and unpredictable conditions ($M_{painful} = 61.36$, $SD_{painful} = 11.13$ versus $M_{nonpainful} = 24.85$, $SD_{nonpainful} = 17.53$), $t(32) = 11.23$, $p < .001$, $d = 1.95$. Yet, post-hoc comparisons also revealed that the *Predictability* effect only trended towards significance for the nonpainful intensity following Bonferroni-correction, indicating that unpredictability only slightly increased the experienced painfulness for the nonpainful intensity, $t(32) = 2.57$, $p = .06$, $d = 0.45$, but not of the painful intensity, $t(32) = 0.21$, $p = 1.00$, $d = 0.04$. An additional *Anxiety* x 2 (*Predictability*) x 2 (*Intensity*) RM-ANCOVA revealed no effects of *Anxiety* (all *p*'s > .56).

3.1.3. Anxiety/discomfort ratings

A 3 (*Condition*) RM-ANOVA on the anxiety/discomfort ratings that we obtained at the end of each condition, showed that participants reported small differences in discomfort/anxiety depending on the experimental condition they were in (Fig. 2C), evidenced by a medium eta square value, $F(2,68) = 3.03$, $p = .055$, $\eta_p^2 = .08$. Specifically, participants experienced slightly more discomfort/anxiety during the predictable painful condition ($M = 2.34$, $SD = 0.87$), than during the predictable nonpainful condition ($M = 1.91$, $SD = 0.89$). However, the difference only trended toward significance after Bonferroni-correction, $t(34) = 2.32$, $p = .08$, $d = 0.39$. This was due to the fact that some participants (7 out of 35) reported that the nonpainful condition induced

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[1] Ratings of two participants were missing in at least one of the three conditions.
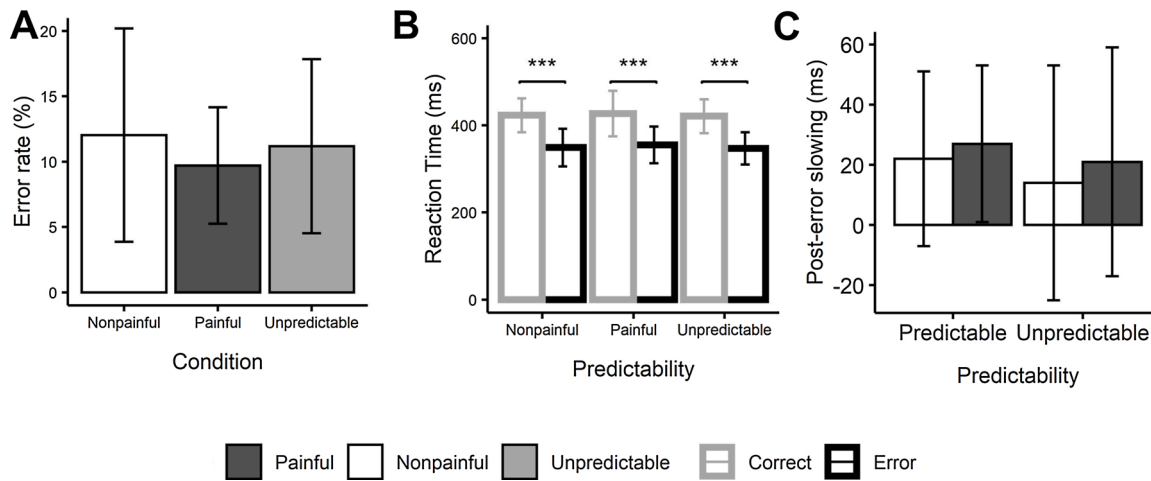
Fig. 3. Behavioral results. **A**. Mean error rate per condition. **B**. Mean reaction times per condition and response. **C**. Mean post-error slowing per stimulation predictability and intensity. Error bars represent the standard deviations. *** $p < .001$, Bonferroni-corrected.

more discomfort/anxiety than the painful condition.[2] Of these, 5 found the nonpainful condition the most uncomfortable condition. Discomfort/anxiety ratings concerning the unpredictable condition ($M = 2.14$, $SD = 0.73$) were situated in between those of both predictable conditions and did not differ significantly from either one condition, with $t(34) = 1.23$, $p = .68$, $d = 0.21$, and $t(34) = -1.31$, $p = .60$, $d = -0.22$, respectively. An *Anxiety* x 3 (*Condition*) RM-ANCOVA did not reveal any effect of trait anxiety (all p's > .42).

3.2. Flanker task: behavioral results

The behavioral performance on the flanker task was assessed based on error rate, reaction time, and post-error slowing. Mean (*SD*) results per condition (for error rates and reaction times) and per *Predictability* x *Intensity* combination (for post-error slowing) are presented in Table 1.

3.2.1. Error rate

A non-parametric Friedman test revealed that the conditions differed with respect to their error rates, $\chi^2(2) = 8.22$, $p < .05$, with lower error rates within the painful condition compared to both predictable nonpainful and unpredictable conditions (Fig. 3A) . Yet, post-hoc Wilcoxon signed-rank tests revealed that these differences did not survive Bonferroni-correction, Z's > -1.94, p's > .15.

Given that we could not include anxiety as covariate to this nonparametric test, we performed a median-split of the STAI-T in order to assign participants with scores lower or equal to the median to a low ($N = 20$), and participants with scores higher than the median to a high ($N = 15$) anxiety group. When sorted for anxiety levels, the error rate results revealed that only the low anxiety group made fewer mistakes in the painful condition (low anxiety $\chi^2(2) = 6.25$, $p < .05$, high anxiety $\chi^2(2) = 3.19$, $p = .20$), however Wilcoxon signed-rank tests again did not survive Bonferroni-correction, Z's > -2.05, $p > .12$.

3.2.2. Reaction time

A 2 (*Response*) x 3 (*Condition*) RM-ANOVA revealed that conditions did not differ statistically with respect to reaction times, as evidenced by an absent main effect of *Condition*, $F_{G-G}(1.64, 55.59) = 1.24$, $p = .30$, $\eta_p^2 = .04$ and *Condition* x *Response* interaction, $F(2,68) = 0.12$, $p = .89$, $\eta_p^2 < .01$. However, consistent with previous studies, participants responded significantly faster on error-trials compared to correct-trials in all conditions, as evidenced by a significant main effect of *Response*, $F(1,34) = 391.70$, $p < .001$, $\eta_p^2 = .92$ (Fig. 3B) . Again, there were no effects of *Anxiety* (all p's > .20), within an *Anxiety* x 2 (*Response*) x 3 (*Condition*) RM-ANCOVA.

3.2.3. Post-error slowing

A 2 (*Predictability*) x 2 (*Intensity*) RM-ANOVA showed that post-error slowing did not differ between errors that were punished with predictable and unpredictable stimulations, and errors that were punished with painful and nonpainful stimulation, as evidenced by the absence of *Predictability* effect, $F(1,34) = 1.90$, $p = .18$, $\eta_p^2 = .05$, and *Intensity* effect, $F(1,34) = 0.89$, $p = .35$, $\eta_p^2 = 0.03$, and a *Predictability* x *Intensity* interaction, $F(1,34) = 0.05$, $p = .83$, $\eta_p^2 = .001$ (Fig. 3C) . However, adding *Anxiety* as a covariate within the *Anxiety* x 2 (*Predictability*) x 2 (*Intensity*) RM-ANCOVA decreased the p-value of the *Predictability* factor, $F(1,33) = 6.65$, $p < .05$, $\eta_p^2 = .17$, suggesting that, overall, participants slowed down more following errors with a predictable punishment intensity, compared to an unpredictable punishment intensity. An additional significant *Predictability* x *Anxiety* interaction, $F(1,33) = 5.38$, $p < .05$, $\eta_p^2 = .14$, and follow-up correlational analysis revealed that the amount of post-error slowing following punishments of unpredictable intensity increased with increasing anxiety ($r_{anxiety, \, slowing \, unpredictable}(33) = .38$, $p < .05$), whereas post-error slowing following punishments of predictable intensity did not ($r_{anxiety, \, slowing \, predictable}(33) = -.15$, $p = .39$).

3.3. ERP results: flanker task

Mean (*SD*) ERP amplitudes per condition (for ERN and Pe) or per *Predictability* x *Intensity* combination (for N1, P2/P3 and the LPC) can be found in Table 1.

3.3.1. Error ERPs

Erroneous responses were consistently followed by a negative going deflection with frontocentral topography peaking approximately 36 ms ($SD = 24$ ms) following the response (ERN) (Fig. 4), and a positive going deflection with centroparietal topography (Pe) (Fig. 5). Given that punishments were given 200 ms following the erroneous response, we

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[2] Eliminating these 7 participants rendered the effect of *Condition* on anxiety ratings significant ($p < .001$ $\eta_p^2 = .29$). In that case, anxiety ratings were significantly smaller in the nonpainful condition compared to both painful ($p <.001$) and unpredictable ($p <.05$) conditions, which did not differ significantly ($p = .32$). Eliminating the 5 participants that reported that the nonpainful condition was the most aversive one, an effect of condition was found $F(2, 58) = 8.38$, $p < 0.001$, $\eta_p^2 = .22$, suggesting that participants rated the nonpainful condition as significantly less uncomfortable than the painful and unpredictable conditions (p's < .001 for painful and .05 for unpredictable), and the painful and unpredictable condition did not significantly differ ($_p = .69$).
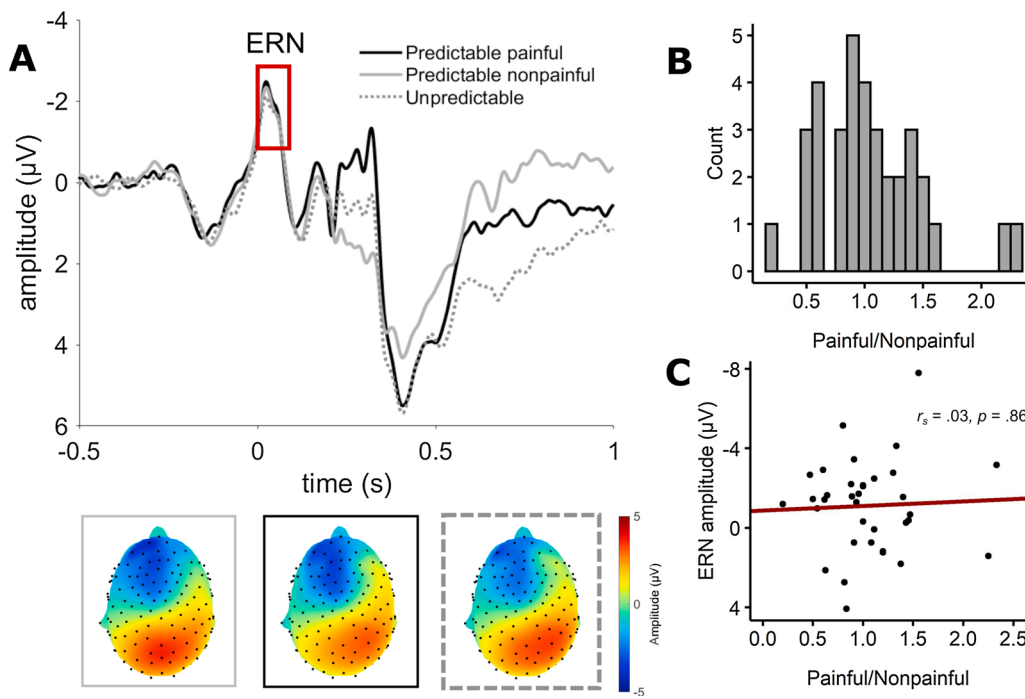
Fig. 4. ERN results. **A**. Average ERP waveforms for frontocentral electrodes (E5, E6, E7, E11, E12, E106) and scalp topography plots for the predictable painful condition, the predictable nonpainful condition, and the unpredictable condition at average peak latency. Time 0 represents the moment of response. ERN amplitudes do not differ over conditions. **B**. Distribution of painful stimulations over nonpainful stimulations in the unpredictable condition. **C**. Correlation between Painful/Nonpainful ratio and ERN amplitudes during the unpredictable condition. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

Fig. 5. Pe results. **A**. Average ERP waveforms for electrodes (E55, E61, E62, E72, E78) and scalp topography plots for the predictable painful condition, the predictable nonpainful condition, and the unpredictable condition at average peak latency. Time 0 represents the moment of response. **Panel B**. Pe amplitudes were smaller in the predictable painful condition as compared to the other two conditions. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).
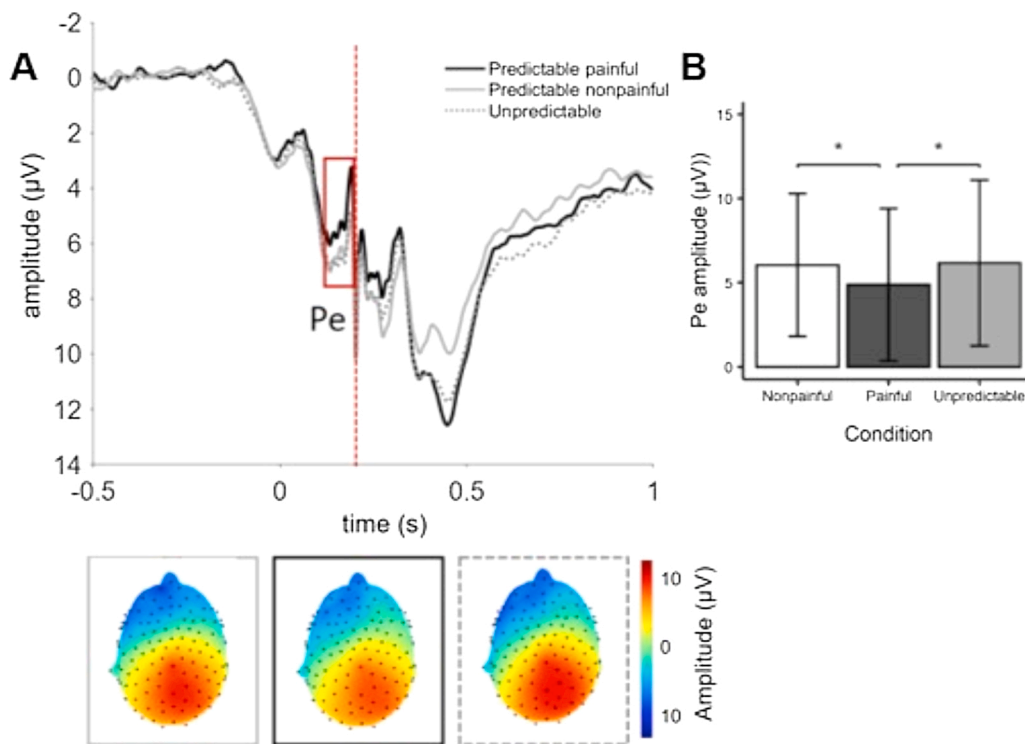
could only measure the Pe over an interval of 120–200 ms post-response, which captures only the early part of the ERP. Therefore, Pe results should be treated with caution.

3.3.1.1. ERN. A 3 (*Condition*) RM-ANOVA on the ERN amplitudes revealed that the amplitudes did not significantly differ across conditions, $F(2,68) = 0.12$, $p = .89$. As the frequentist statistical approach does not allow drawing conclusions about the null hypothesis (H0), we performed an additional Bayesian analysis to compare the evidence in

favor of the null (H0) and alternative (H1) hypotheses. Specifically, we ran a Bayesian one-way RM-ANOVA in SPSS using the default estimation method (Bayesian information criteria), which returned a Bayes Factor B_{10} of 0.027 (comparing the alternative model with the null model), indicating that there was very strong evidence *in favor* of the null hypothesis, therefore supporting that ERN amplitudes were comparable across conditions (Jeffreys, 1961; Lee & Wagenmakers, 2013).

The *Anxiety* x 3 (*Condition*) RM-ANCOVA furthermore did not reveal any effect of *Anxiety* on ERN amplitudes, as evidenced by the absence of

a main effect of *Anxiety*, $F(1,33) = 2.34$, $p = .14$, $\eta_p^2 = .07$, and *Anxiety* x *Condition* interaction, $F_{G-G}(1.71, 56.37) = 0.24$, $p = .75$, $\eta_p^2 < .01$.

A potential post-hoc explanation for why ERN amplitudes did not differ across conditions, might have been related to the counterbalancing of the conditions. Previous studies have suggested that the punishment of errors may have a lasting effect on error processing and the ERN (Riesel, Kathmann, Wüllhorst, Banica, & Weinberg, 2019, 2012). Hence, potentiation of the ERN caused by the painful punishments in either the predictable painful and unpredictable condition could have had lasting effects on the amplitude of the ERN in the subsequent nonpainful condition. To test this hypothesis, we reran our analysis for the subsample of participants who received predictable nonpainful punishments in the first condition ($N = 10$). As these participants only received painful punishments in later conditions, the presumed carryover effects should not affect the predictable nonpainful condition. Again, this analysis yielded no significant difference between any of the conditions, $F(2,18) = 0.17$, $p = .84$, $\eta_p^2 = .02$. An obvious caveat is that such subsample might have been too small to render the differences significant.

Furthermore, there were no gender-related differences in the ERN amplitudes (p's > .21), nor did ERN amplitudes in any of the conditions correlate significantly with the associated stimulation intensities (both objective and subjective), error rates, reaction times, post-error slowing or reported anxiety/discomfort ratings (all p's > .15, Bonferroni-corrected for 6 correlations). This suggests that behavioral performance and intensity-related differences were unrelated to the measured ERN amplitudes. Finally, to exclude the possibility that the relative over- or under-representation of painful compared to nonpainful stimulations in the unpredictable condition could have affected the ERN amplitude in the that condition, we computed for every participant a painful over nonpainful error-ratio (Painful/Nonpainful) (Fig. 4B). As expected, participants varied greatly with respect to the proportion of painful to nonpainful stimulations they received in the unpredictable condition. In fact, only three participants were equally punished by painful and

nonpainful stimulations (Painful/Nonpainful = 1). Nevertheless, relative over- or underexposure to painful compared to nonpainful stimulation appeared to be equally likely within our sample. Furthermore, the ERN amplitude in the unpredictable condition did not correlate with the Painful/Nonpainful ratio, $r_s(33) = .03$, $p = .86$ suggesting that the experienced inequality did not influence the ERN amplitude (Fig. 4C).

3.3.1.2. Pe. A 3 (Condition) RM-ANOVA on the Pe amplitudes revealed that the Pe was significantly lower within the predictable painful condition compared to the predictable nonpainful and the unpredictable conditions (p's < .05 in post-hoc tests), which did not differ significantly ($p = 1$), $F(2,68) = 7.6$, $p < .01$, $\eta_p^2 = .18$ (Fig. 5B).

Adding Anxiety as a covariate revealed a marginally significant main effect of Anxiety, $F(1,33) = 3.39$, $p = .075$, $\eta_p^2 = .093$, but no *Anxiety* x *Condition* interaction ($p = .95$). A post-hoc correlation between the average Pe across conditions and STAI scores showed that more anxious participants had overall lower Pe amplitudes $r(33) = -.31$, $p = .075$.

3.3.2. Somatosensory evoked potentials (SEPs)

The electrical stimulation that followed erroneous responses consistently elicited several positive and negative deflections in the EEG, such as the N1 (Fig. 6), peaking approximately 110 ms ($SD = 22$ ms) post-stimulation, the P2/P3 (Fig. 6), peaking approximately 240 ms ($SD = 35$ ms) post-stimulation, and a slow positive deflection (LPC) we measured over the 400–600 ms post-stimulation interval (Fig. 7). Because of our design, the number of painful and nonpainful stimulations in the unpredictable condition ($M_{painful} = 11$, $SD_{painful} = 8$; $M_{nonpainful} = 11$, $SD_{nonpainful} = 6$) was significantly smaller than the number of painful and nonpainful stimulations in the predictable conditions ($M_{painful} = 19$, $SD_{painful} = 10$; $M_{nonpainful} = 23$, $SD_{nonpainful} = 17$), $F(1,34) = 103.27$, $p < .001$, $\eta_p^2 = .75$. In order to assess if this difference influenced the results, SEP-results were additionally checked by RM-ANOVAs with mean amplitudes (as opposed to peak amplitudes, see Luck, 2014). Given that these control analyses rendered similar results, we report the
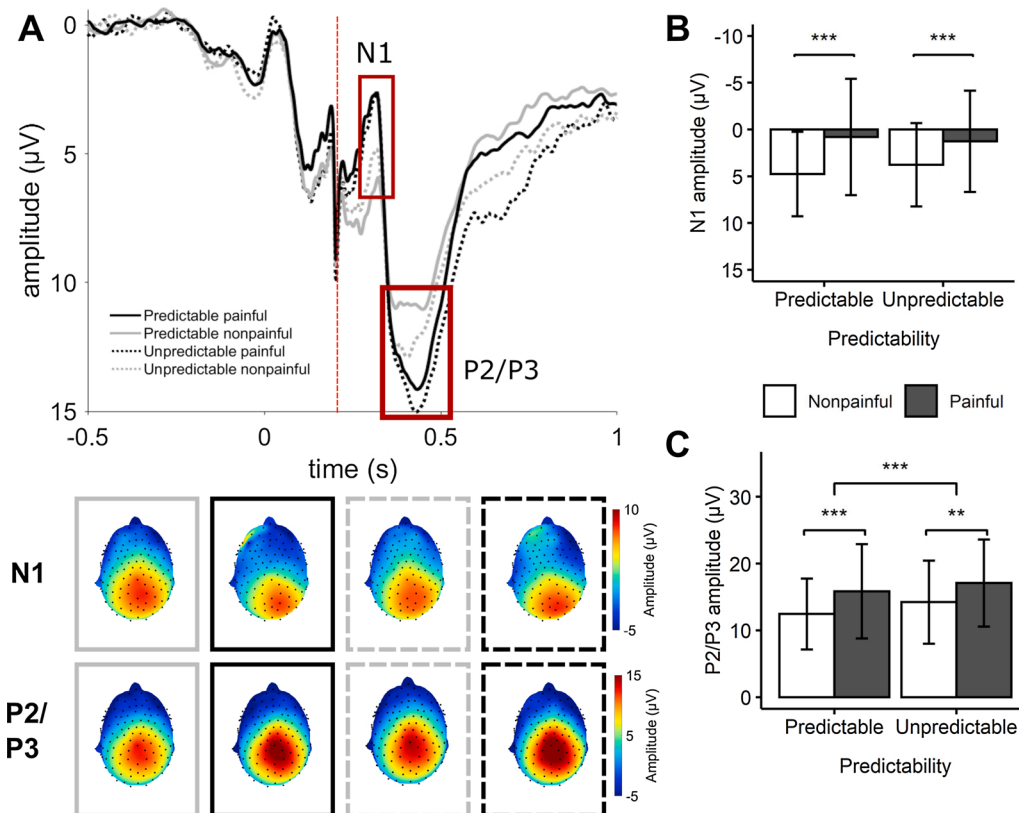

Fig. 6. SEP N1 and P2/P3 results. A. Average ERP waveforms recorded at electrodes (E55, E129) and scalp topography plots at average peak latency (post-stimulation) for predictable nonpainful stimulation (N1: 123 ms, P2/P3: 250 ms), predictable painful stimulation (N1: 118 ms, P2/P3: 233 ms), unpredictable nonpainful stimulation (N1: 119 ms, P2/P3:204 ms), and unpredictable painful stimulation (N1: 118 ms, P2/P3: 231 ms). Time 0 reflects the moment of response. Stimulation onset is represented by the dotted red line. B. Mean N1 amplitudes per stimulation predictability and intensity. C. Mean P2/P3 amplitudes per stimulation predictability and intensity. The component is labeled P2/P3 due to the likely overlap between the two (see Miltner et al., 1989). Error bars represent the standard deviations. ** $p < .01$ and *** $p < .001$, Bonferroni-corrected. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

results with peak amplitudes.

3.3.2.1. N1. A 2 (*Predictability*) x 2 (*Intensity*) RM-ANOVA on N1 amplitudes revealed a significant main effect of *Intensity*, $F(1, 34) = 35.52, p < .001, \eta_p^2 = .51$, in the absence of a main effect of *Predictability*, $F(1, 34) = 0.37, p = .55, \eta_p^2 = .01$ or a *Predictability* x *Intensity* interaction, $F(1, 34) = 2.87, p = .099, \eta_p^2 = .08$, suggesting that the N1 component was more pronounced following painful compared to nonpainful stimulation (Fig. 6B) . An *Anxiety* x 2 (*Predictability*) x 2 (*Intensity*) RM-ANCOVA furthermore did not reveal any effect of *Anxiety* (*p*'s > .37) on N1 amplitudes.

3.3.2.2. P2/P3. A 2 (*Predictability*) x 2 (*Intensity*) RM-ANOVA on P2/P3 amplitudes revealed significant main effects of *Predictability*, $F(1,34) = 20.50, p < .001, \eta_p^2 = .38$, and *Intensity*, $F(1,34) = 25.56, p < .001, \eta_p^2 = .43$, suggesting that P2/P3 amplitudes were generally higher for unpredictable compared to predictable and painful compared to nonpainful stimulations (Fig. 6C). There was no *Predictability* x *Intensity* interaction, $F(1,34) = 0.25, p = .62, \eta_p^2 = .01$. The *Anxiety* x 2 (*Predictability*) x 2 (*Intensity*) RM-ANCOVA furthermore revealed no effects of *Anxiety* (*p*'s > .57) on P2/P3 amplitudes.

3.3.2.3. LPC. A 2 (*Predictability*) x 2 (*Intensity*) RM-ANOVA on the mean LPC amplitudes revealed a significant main effect of *Predictability*, $F(1,34) = 6.96, p < .05, \eta_p^2 = .17$, but no effect of *Intensity*, $F(1,34) = 2.55, p = .12, \eta_p^2 = .07$, nor a *Predictability* x *Intensity* interaction, $F(1,34) = 0.02, p = .89, \eta_p^2 < .001$ suggesting that amplitudes were higher following unpredictable compared to predictable stimulation (Fig. 7B) . Again, the *Anxiety* x 2 (*Predictability*) x 2 (*Intensity*) RM-ANCOVA revealed no effects of *Anxiety* on the LPC amplitudes (*p*'s > .82).
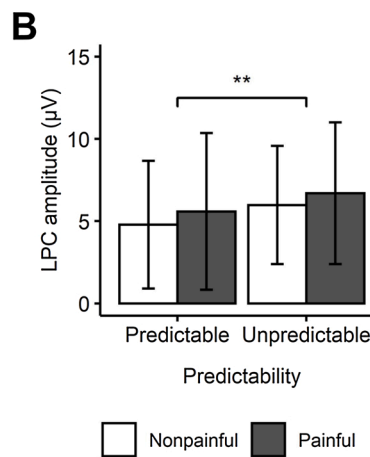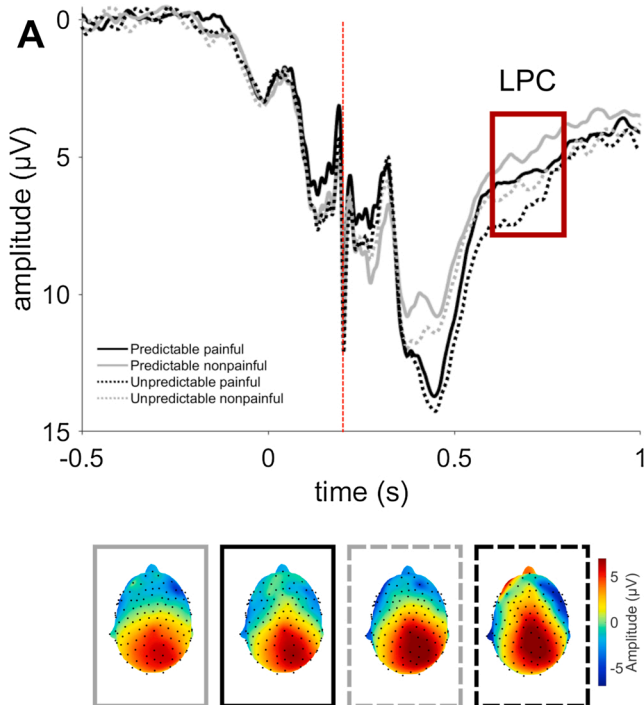
4. Discussion

Errors have long been conceptualized as unpredictable, endogenous threats that have the potential to put the organism in grave danger (Hajcak & Foti, 2008; Hajcak, 2012; Proudfit et al., 2013). Accordingly, increasing evidence has demonstrated that both unpredictable contexts and state-manipulations that affect the consequences of errors enhance the error-related negativity (ERN) (Jackson et al., 2015; Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel, Kathmann, Wüllhorst et al., 2019; Speed et al., 2017; Tan et al., 2019; Tullett et al., 2015). Interestingly, however, both effects have predominantly been studied in isolation. Therefore, the current study attempted to combine both research lines and examined the effects of unpredictable and predictable punishment intensity on ERN amplitudes. Additionally, in line with evidence suggesting that trait anxiety might impact people's sensitivity to such state-manipulations (Meyer & Gawlowska, 2017; Riesel et al., 2012), we investigated whether and how trait anxiety might have modulated these effects.

To this end, 35 healthy volunteers completed an arrowhead-version of the Erikson flanker task under three conditions: Errors were either consistently followed by (1) predictable painful, (2) predictable nonpainful, or (3) unpredictable painful/nonpainful electrical stimulation. Contrary to our predictions, we found no differences in ERN amplitudes and reaction times nor did we find any effect of anxiety. Nevertheless, we found that Pe amplitudes were significantly lower within the predictable painful condition compared to the predictable non-painful and unpredictable conditions. More anxious participants, furthermore, trended to have overall lower Pe amplitudes. Additionally, we found that error rates differed modestly across conditions (with lower error rates within the painful condition), yet these differences did not survive Bonferroni-correction. Furthermore, we found that although participants generally slowed down more following errors with a predictable

Fig. 7. LPC results. **A.** Average ERP waveforms for electrodes (E55, E62) and scalp topography plots at 500 ms post-stimulation for predictable nonpainful stimulation, predictable painful stimulation, unpredictable nonpainful stimulation, and unpredictable painful stimulation. Time 0 reflects the moment of response. Stimulation onset is represented by the dotted red line. **B.** Mean LPC amplitudes per stimulation predictability and intensity. Error bars represent standard deviations. ** $p < .01$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

punishment intensity, the amount of slowing following errors of unpredictable punishment intensity increased with increasing anxiety. Likewise, the effects of *Predictability* and punishment *Intensity* were present in the Somatosensory Evoked Potentials (SEPs) elicited by the sensory stimulation used for the punishments. N1 amplitudes were increased for painful compared to nonpainful stimulation. P2/P3 amplitudes were increased for painful compared to nonpainful, and for unpredictable compared to predictable stimulation, and the LPC were increased for unpredictable compared to predictable stimulations. These results suggest that while unpredictability and increased painfulness of punishments enhance the motivational significance of the errors, they did not potentiate ERN amplitudes beyond the ones elicited by errors punished with predictable nonpainful stimulation.

The Pe findings were in line with previous studies showing that task-irrelevant exteroceptive (e.g. presence of a spider; Moser, Hajcak, & Simons, 2005) or interoceptive threats (e.g. threat of dyspnea; Sucec et al., 2019) reduced Pe amplitudes. In these studies, the authors argued that the anticipation of imminent threat drew the attention toward the source of threat and away from the error itself, hence lowering the Pe amplitudes. Furthermore, we found a trend for lower Pe amplitudes in more anxious individuals. Previous studies have found similar, opposite or no association (Aarts & Portois, 2010; Moser, Moran, Schroder, Donnellan, & Yeung, 2013, see Koban & Pourtois, 2014 for a neurobiological model about the ERN and Pe). As a caveat, this study was designed primarily to investigate the ERN, and the results exploratorily obtained for the Pe should be confirmed in a design more specifically suited to investigate the Pe component as well.

While the stimulation-related results were in line with our predictions, ERN-related results were not. Contrary to our predictions, we found no support for the modulation of the ERN by unpredictable punishment intensity. Although surprising, this finding does not exclude the possibility that unpredictability of other punishment-related aspects, such as the (non-) occurrence of punishment following a given error, might still modulate ERN amplitudes. In fact, previous studies by Riesel et al. (2012; Riesel, Kathmann, Wüllhorst et al., 2019), and Meyer and Gawlowska (2017) that showed potentiation of ERN amplitudes within punishing conditions did *not* systematically punish erroneous responses. Instead, they adopted 50 % reinforcement schedules, rendering the consequence of errors (punishment or not) completely unpredictable. As a result, the potentiation of ERN amplitudes they attributed to the punishment might have been confounded with the unpredictability related to the reinforcement schedule. Accordingly, manipulations of the reinforcement rate might be a more suitable method for examining the effect of consequence-related unpredictability on the ERN. Furthermore, although 50 % reinforcement schedules may yield maximal unpredictability, Lindström, Mattsson-Mårn, Golkar, and Olsson (2013) found that only high compared to low risk of electrical stimulation contingent on error commission amplified the electromyographic activity in the corrugator supercilii (cEMG) muscle of the upper face within the first 100 ms following error commission, a measure that is believed to be highly related to the ERN.

Somewhat more surprising than the absent effect of unpredictability, was the absent effect of punishment intensity across the predictable conditions. Past studies have repeatedly reported a potentiation of ERN amplitudes as a consequence to the enhancement of the motivational significance of errors, for instance, by making correct responses more attractive and/or by making incorrect responses more aversive (e.g., by using secondary reinforcers such as monetary gains/losses: Boksem, Tops, Kostermans, & De Cremer, 2008; Endrass et al., 2010; Hajcak, Moser, Yeung, & Simons, 2005; Maruo, Schacht, Sommer, & Masaki, 2016; Maruo, Sommer, & Masaki, 2017; Pailing & Segalowitz, 2004; Potts, 2011; Stürmer, Nigbur, Schacht, & Sommer, 2011; or by using primary reinforcers such as loud noises: de Bruijn, Jansen, & Overgaauw, 2020; Pasion, Paiva, Fernandes, Almeida, & Barbosa, 2018; Riesel et al., 2012; Riesel, Kathmann, Wüllhorst et al., 2019; or electrical stimulation: Meyer & Gawlowska, 2017).

Our finding that ERN amplitudes were comparable across the predictable painful and nonpainful conditions therefore seems at odds with the motivational hypothesis of the ERN. Interestingly, however, only a relatively small subset of the aforementioned studies has directly compared different levels of reward (Hajcak et al., 2005; Pailing & Segalowitz, 2004), whereas only two recent studies have, to our knowledge, compared different levels of punishment (de Bruijn et al., 2020; Maruo et al., 2017). Notably, one of these studies also did not find any significant differences between the punishment conditions and a non-punishing control condition (Maruo et al., 2017). Still, neither study used primary punishers, thereby complicating a direct comparison with our findings. At the same time, our study cannot be easily compared with previous studies that used primary punishers, as there are several design-related differences that might have contributed to the conflicting results. Specifically, besides the differences in reinforcement schedules (100 % versus 50 % in Meyer & Gawlowska, 2017; Pasion et al., 2018; Riesel et al., 2012; Riesel, Kathmann, Wüllhorst et al., 2019), we used a different punishment modality (single electrical pulse versus 500 ms of AC stimulation in Meyer & Gawlowska, 2017, or aversive loud sounds, Pasion et al., 2018; Riesel et al., 2012; Riesel, Kathmann, Wüllhorst et al., 2019). Finally, punishments were delivered 200 ms post-error in our study, whereas they were only delivered 600–1000 ms post-error in other studies (600 ms in Meyer & Gawlowska, 2017; and 1000 ms in; Pasion et al., 2018; Riesel et al., 2012; Riesel, Kathmann, Wüllhorst et al., 2019).

Regardless of these differences with previous studies, a number of potential explanations for the absent *Intensity* effect can be postulated. Importantly, because of the absence of a no-punishment control condition, all post-hoc interpretations of the null findings remain speculative. We acknowledge that this limits the scope of the conclusions we can draw from our findings.

The first possibility is that our experimental manipulation might simply not have been successful at increasing the motivational significance of errors, and consequently, at inducing differences between the three punishing conditions. While the small differences in the level of anxiety generated by the different conditions would support this explanation, it is also worth noting that small behavioral differences were also observed in studies wherein the ERN amplitude was successfully modulated (e.g. Endrass et al., 2010; Hajcak et al., 2005). Besides, the analyses of the SEPs, perceived intensity and post-error slowing suggest that predictability and intensity manipulations did affect the processing of punishments. This supports the fact that our manipulation was effective in modulating the responses to punishments, but it cannot rule out that the same design was not perfectly tailored to investigate the modulation of the ERN.

Moreover, the order of the conditions could have additionally confounded any potential modulation of the ERN. Riesel et al. (2012; Riesel, Kathmann, Wüllhorst et al., 2019) previously showed that the punishment of errors has a lasting effect on the ERN, maintaining the elevated amplitudes even after the error-punishment pairing had ended. Hence, any potential modulation of the ERN could have been countered by carryover effects between the different conditions. Although our control analysis did not provide evidence in favor of this hypothesis, we cannot exclude this possibility, given the relatively small sample in which the control analysis was run. Still, we want to note that the lasting effects of punishment described by Riesel et al. (2012; Riesel, Kathmann, Wüllhorst et al., 2019) were only found in contexts that were identical to the initial punishing context, and not in the established non-punishing contexts. Given that we clearly indicated at the start of each condition that the punishment intensities had changed, we argue that carryover effects should have been minimized in our study. This is furthermore in line with another study that did not find support for these long-lasting effects of punishment (Pasion et al., 2018).

Another explanation for the absent modulation of the ERN would be that the manipulation was successful, but that it affected all punishment conditions in a similar way. Again, a non-punishing baseline condition is

needed to reveal the direction of this effect. In the absence of such a condition, we could speculate that the threat of electrical stimulation could have affected the ERN in an all-or-nothing manner, thereby upregulating behavioral responses and ERN amplitudes equally for all conditions. Indeed, the risk of receiving any electrical stimulation seems to be more aversive than the risk of losing money (Maruo et al., 2017) or of being responsible for the punishment of a social partner (de Bruijn et al., 2020). By contrast, it could also be that the ERN was potentiated by neither condition, or that it was equally attenuated by all conditions. Notably, such effects have also been rarely described in the past (e.g., Maruo et al., 2017; Pasion et al., 2018).

In further support of the post-hoc interpretation that all punishment conditions affected the ERN in a similar way, we found no anxiety-related differences in ERN modulation. Previous studies demonstrated that the moderating effects of anxiety on the ERN were specific to certain contexts (e.g. Endrass et al., 2010; Olvet & Hajcak, 2009b; Riesel, Kathmann, & Klawohn, 2019). For instance, ERN amplitudes of OCD patients were only increased compared to those of healthy controls in standard and speed-focused conditions, but not in conditions that emphasized accuracy (through punishments: Endrass et al., 2010; or instructions: Riesel, Kathmann, Klawohn et al., 2019). In the latter contexts, all participants (OCD and controls) showed elevated ERN amplitudes, whereas amplitudes of healthy controls declined more in non-punishing and speed-focused contexts. This suggests that higher levels of anxiety might lead to overactive error-monitoring, but only in situations with lower or uncertain error significance (e.g., when punishment occurred intermittently; Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel, Kathmann, Wüllhorst et al., 2019). In that sense, we speculate that the consistent punishment of errors in our study might have motivated participants to always maximally avoid errors, regardless of punishment intensity. Indeed, under such circumstances, the upregulation of error-monitoring, and consequently the ERN, might have been the most adaptive response. Anxiety-related differences in ERN amplitudes that could have been present at baseline, or that could have been more pronounced under highly unpredictable circumstances (e.g., when punishment occurred intermittently; Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel, Kathmann, Wüllhorst et al., 2019), might therefore have been attenuated in our design.

Finally, the difference in results between the ERN and the Pe adds on the current literature suggesting that the two components have a different functional significance (Steinhauser & Yeung, 2010), and seem to rely on different neural networks (Di Gregorio, Maier, & Steinhauser, 2018; Pezzetta, Wokke, Aglioti, & Ridderinkhof, 2021, with the Pe that may be associated to attention orienting and context updating (similarly to the P300, see Overbeek et al., 2005; Wessel, 2012, but also Steinhauser & Yeung, 2010 for a different interpretation).

Taken together, our results do not provide support for a modulation of ERN amplitudes between conditions that punished erroneous responses with (1) predictable painful, (2) predictable nonpainful, and (3) unpredictable painful/nonpainful electrical stimulation. Furthermore, there were no differences related to individual differences in trait anxiety. Nevertheless, our results revealed that both predictability and intensity modulated perception and cortical responses to somatosensory stimuli (SEPs), suggesting that our manipulations did in fact increase the motivational significance of errors, at least for post-stimulus responses. Although the lack of a baseline condition does not allow us to draw definite conclusions, based on these findings, we propose that under certain experimental conditions, increasing the motivational significance of errors is not directly accompanied by proportional increases in ERN amplitude.

Declaration of Competing Interest

The authors report no declarations of interest.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.biopsycho.2021.108177.

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