New insights into neural networks of error monitoring and clinical implications: a systematic review of ERP studies in neurological diseases

Sabrina Lenzoni[1,2*], Joshua Baker[2,4], Alexander L. Sumich[2], Daniel C. Mograbi[1,3]

[1] Department of Psychology, Pontifical University of Rio de Janeiro, 22451-900, Rio de Janeiro, Brazil

[2] Department of Psychology, Nottingham Trent University, NG1 4FQ, Nottingham, UK

[3]Institute of Psychiatry, Psychology & Neuroscience, King's College London, SE5 8AF, London, UK

[4]Institute for Systems Neuroscience, University Hospital Hamburg-Eppendorf, 20251, Hamburg, Germany

* Corresponding author

E-mail address: sabrinalenzoni@gmail.com

Phone: +39 3466558877

Department of Psychology, Pontifical University of Rio de Janeiro, 22451-900, Rio de Janeiro, Brazil

Abstract

Error monitoring allows for efficient performance of goal-directed behaviors and successful learning. Furthermore, error monitoring as a metacognitive ability, may play a crucial role for neuropsychological interventions, such as rehabilitation. In the past decades, research has suggested two electrophysiological markers for error monitoring: the error related negativity and the error positivity, thought to reflect, respectively, error detection and error awareness. Studies on several neurological diseases have investigated the alteration of the error related negativity and the error positivity, but these findings have not been summarized. Accordingly, a systematic review was conducted to understand what neurological conditions present alterations of error monitoring event-related potentials and their relation with clinical measures. Overall, ERN tended to be reduced in most neurological conditions while results related to Pe integrity are less clear. ERN and Pe were found to be associated with several measures of clinical severity. Additionally, we explored the contribution of different brain structures to neural networks underlying error monitoring, further elaborating on domain-specificity of error processing and clinical implications of findings. In conclusion, electrophysiological signatures of error monitoring could be reliable measures of neurological dysfunction and a robust tool in neuropsychological rehabilitation.

Keywords

ERPs; Error positivity: Error-related negativity; Neurology; Self-monitoring

1.Introduction

Error monitoring is crucial to successfully perform goal-directed behaviors (Ullsperger et al., 2014) and for adaptive control in daily life (Krönke et al., 2018; Overmeyer et al., 2021). Over the last decades, error processing has captured the attention of clinical research, showing that deficient error monitoring characterizes various mental disorders (for example Clayson et al., 2020; Meyer, 2016; Riesel et al., 2019). Moreover, error monitoring, as a metacognitive ability involved in online cognitive control, is thought to contribute to the emergence of self-awareness (Morris and Mograbi, 2013).

According to the Cognitive Awareness Model (Agnew and Morris, 1998; Mograbi and Morris, 2014), performance monitoring plays a key role in in the integrity of metacognitive awareness. Error monitoring impairments, underlying frontal cortico-subcortical loops can occur at multiple levels and result in anosognosia, that is defined as the lack of awareness of symptoms or deficits in clinical conditions, such as neurological disorders (Mograbi and Morris, 2018). A central dysfunction of monitoring mechanisms would result in executive anosognosia while domain-specific impairments would lead to local, domain-specific unawareness, such as anosognosia for hemiplegia.

Self-awareness has been extensively investigated in neurodegenerative diseases and acquire brain injury (Amanzio et al., 2020; Chavoix and Insausti, 2017; Leung and Liu, 2011; Mazancieux et al., 2019; Prigatano and Sherer, 2020). Crucially, impaired self-awareness in neurological conditions can hinder rehabilitation (Medley and Powell, 2010; Ownsworth and Clare, 2006; Trahan et al., 2006) and community reintegration (Kelley et al., 2014). Self-awareness, and specifically error awareness, is believed to be essential for successful rehabilitation of cognitive functions (Dockree et al., 2015; Leung and Liu, 2011). Common approaches in rehabilitation are errorless and error-based learning. In the first case, the training consists of observing and practicing only correct actions, through the support of the therapist

who prevents the patient from performing errors (Haslam and Kessels, 2018). This approach has been shown to be effective for memory impairments (Clare and Jones, 2008; Dunn and Clare, 2007; Ehlhardt et al., 2008). Instead, error-based learning training focuses on a trial-and-error process, including prompt and feedback provided by the psychotherapist, allowing self-correction and facilitating strategy use (Toglia, 2011). Recently, it has been shown that error-based learning can be more effective than errorless learning approaches in rehabilitation for brain injury patients, improving patient's self-awareness and allowing skill transfer (Ownsworth et al., 2017), thus highlighting the relevance of error detection and correction for clinical conditions. Critically, Overmeyer et al. (2021) showed that ERN can predict self-control in real life behavior. Assessing error-related integrity in brain injury patients may be indicative of self-monitoring abilities within specific cognitive domains and thus guide the choice of rehabilitation techniques. Therefore, functional brain biomarkers of error monitoring could potentially become a robust tool for clinical assessment and rehabilitation.

Electroencephalography (EEG) research has identified two event-related potentials (ERPs) underpinning error processing: the error-related negativity (ERN) and the error positivity (Pe). The ERN is a negative deflection occurring around 50ms over fronto-central sites following error commission (Gehring et al., 1993). According to the *Mismatch Theory*, the ERN reflects the mismatch between action efferent copies and top-down representations of intended (correct) and actual response (Dehaene, 2018; Falkenstein et al., 1991; Gehring et al., 1993; Scheffers and Coles, 2000). Alternatively, it has been proposed that the ERN represents the degree of conflict between competing representations (*Conflict Monitoring theory*; Botvinick et al., 2001; Yeung et al., 2004).

The neural generator of the ERN has been localized in the anterior cingulate cortex (ACC; Brázdil et al., 2005; Debener et al., 2005; Dehaene et al., 1994; Reinhart and Woodman, 2014; Van Veen and Carter, 2002). According to *Reinforcement Learning* theory, the ERN is

mediated by changes in levels of phasic dopaminergic activity in the basal ganglia resulting in inhibitory error signaling from the basal ganglia to the ACC (Holroyd and Coles, 2002). According to the *Predicted Response-Outcome* (PRO) model (Alexander and Brown, 2011), learning processes relying on medial prefrontal cortex function follow standard rules of probability. The authors also proposed that error effects may reflect the comparison between actual and intended outcomes, while conflict derives from the prediction of multiple responses and their outcomes. Evidence from behavioral studies, focusing on post-error slowing, suggested that unexpected events (either correct responses or errors) elicit a maladaptive shift of the attention away from the ongoing task (Notebaert et al., 2009). A more recent account of error processing is the adaptive orienting theory (Wessel, 2018), which posits that errors, as unexpected events, trigger a series of adaptive automatic processes, including rapid motor and cognitive suppression, and subsequent attentional reorienting. This is supported by further research on the association between the ERN and attentional post-error adjustments, showing temporal proximity between ERN and subsequent attentional reallocation, and that the strength of post-error adjustments varies with ERN amplitude (Steinhauser and Andersen, 2019). The consensus among different theoretical accounts is that the ERN indexes a performance monitoring system, that enables learning and behavioral adjustments (Weinberg et al., 2015).

The Pe is a later positive component, peaking at centro-parietal sites between 200-500ms after error commission (Falkenstein et al., 1991; Overbeek et al., 2005). It has been shown that ERN and Pe represent two independent systems of error monitoring (Di Gregorio et al., 2018; Overbeek et al., 2005). However, the functional role of Pe is still debated. It has been proposed that Pe is a P3b-like component, associated with motivational significance of the response (Overbeek et al., 2005; Ridderinkhof et al., 2009), that may reflect working memory updating (Donchin and Coles, 1988; Polich, 2007). In this context, post-error processing has been associated with locus coeruleus-norepinephrine system activity (Nieuwenhuis et al., 2005;

Overbeek et al., 2005; Ridderinkhof et al., 2009), as one possible input into the salience network (Wessel, 2018). Error awareness and processing of salience have been shown to rely on overlapping neural networks, involving the anterior insula, dorsal ACC, thalamus, supplementary motor area, and parietal regions (Harsay et al., 2012). Previous studies have demonstrated a relationship between Pe and conscious perception of errors (Endrass et al., 2007; Murphy et al., 2012; Nieuwenhuis et al., 2001). In line with the *Accumulation Account*, the Pe reflects error awareness, which emerges from a process of evidence accumulation about the erroneous response (Steinhauser and Yeung, 2012, 2010; Ullsperger et al., 2010; Wessel et al., 2011). Error awareness is believed to emerge from the integration of different input signals, such as cognitive, sensory, proprioceptive and interoceptive inputs (Ullsperger et al., 2010; Wessel et al., 2011). Ullsperger et al. (2010) also suggested that the neural network underlying error awareness and the Pe involved structures such as ACC, anterior insula and, somatosensory areas.

The aim of this review is to understand whether ERN and Pe alterations are specific to certain neurological conditions and examine their relation with clinical factors. Furthermore, this evaluation will provide insights to elucidate the role of different brain areas in neural networks underlying error monitoring.

2.Methods

2.1 Search strategy

Article selection was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Moher et al., 2009) guidelines. A flowchart of this selection process is displayed in Figure. 1. PubMed, Scopus and Web of Science databases were systematically searched for eligible studies from inception to January 31st, 2021. The search terms used were: ERP OR “event related” OR “event-related” OR “evoked potential” OR

"evoked-potential" AND "error related negativity" OR "error-related negativity" OR ERN OR Ne OR "error positivity" OR Pe. Reference lists from detected studies were also checked for additional unidentified studies.

2.2 Study selection

Only English-language studies were included. Eligible studies fulfilled the following criteria: 1) the study design was cross-sectional; 2) the study included a clinical group with a neurological condition and a control group, as determined by neurological diagnosis; 3) all participants were adults; 4) each group was composed of at least 5 participants; 5) the amplitude and/or the latency of the ERN and/or the Pe were measured by ERP technique. Studies without group-level statistics were excluded. Studies including neurodevelopmental diseases were excluded. Reviews and conferences papers were also excluded.

2.3 Quality assessment

A quality assessment form was devised which focused on sampling, measurement of outcomes and analysis (Table 1). In accordance with the Cochrane Collaboration recommendations (Higgins and Green, 2008), an overall score was not generated, with a risk of bias judgment of "yes", "no" or "unclear" being given instead for individual domains. Study quality was assessed by two independent reviewers If a study received more than two "no" or three "unclear" judgments, the study was considered as having poor quality and was excluded from the review.

2.4 Data extraction

The following data were extracted by two independent reviewers: authors, publication year, diagnosis, sample size, task (experimental task and stimuli description), results comparing

patient and control behavioral performance and ERPs measures, and correlations between ERPs measures and other measures.

Please insert Table 1 here

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3.Results

A total of 41 studies met inclusion criteria and were selected for review. Of these, 39 measured the ERN and 23 the Pe, with 21 measuring both ERPs. ERN and Pe were typically measured at midline electrodes. The most common recording sites of interest were Fz, FCz, Cz and Pz.

The most common task (n studies=23) to investigate error monitoring was the Flanker Task, which relies on the conflict between task-relevant and task-irrelevant stimuli (Eriksen and Eriksen, 1974). Other tasks based on interference suppression were the Stroop Task (n=3), Letter Discrimination Task (n=1), Simon-type Task (n=1) and the Error Awareness Task (n=1). The Error Awareness Task is a paradigm developed by Hester et al.(2005) and is an adapted version of the Stroop Task, incorporating a Go/NoGo component and a button press response to signal error awareness. Paradigms involving response inhibition included the Go/NoGo (n=3), the Oddball Task (n=1), and the Stop Signal Task (n=3). The remaining paradigms relied on a variety of cognitive tasks. In the Anti-saccade Task (n=3), participants are asked to quickly perform a saccade to the opposite direction of a cue stimulus presentation (Hallett, 1978). In the Lexical Decision paradigm (n=2), participants are asked to decide whether a string of letters is a word or not (Rubenstein et al., 1971). The Picture-Name Verification task (n=1) consists of the presentation of a word followed by a picture. Participants are then asked to decide

whether they semantically matched or not (Wingfield, 1968). Finally, a visual search (n=1) and a visual short-term memory task (n=1) were also employed as experimental paradigms.

Neurological conditions included Alzheimer's disease (n=2), Tourette syndrome (n=3), multiple sclerosis (n=1), amyotrophic lateral sclerosis (n=1), Parkinson's disease (n=11), Huntington's disease (n=4), cerebellar ataxia (n=1), cerebellar degeneration (n=1), focal lesions (n=9+ 1 cerebellar lesion) and traumatic brain injury (n=8). ERPs findings are reviewed by disorder. Cerebellar ataxia, cerebellar degeneration and one study involving cerebellar lesion were grouped together as "Cerebellar Dysfunction". Results of single studies are displayed in Table 2.

3.1 Alzheimer's disease (AD)

Both studies showed lower ERN amplitude in AD patients compared to healthy controls (Ito and Kitagawa, 2005; Mathalon et al., 2002), with the former reporting longer ERN latency in AD. One study reported no difference between AD and controls for Pe amplitude (Mathalon et al., 2002), while Ito and Kitagawa (2005) showed decreased Pe amplitude and prolonged latency in AD. Mathalon et al., (2005) used a Picture-Name Task Verification, while Ito and Kitagawa (2005) a Lexical Decision Paradigm.

3.2 Tourette Syndrome (TS)

All studies reported higher ERN amplitude in TS as compared to controls (Johannes et al., 2002; Schüller et al., 2018; Warren et al., 2020). No differences in ERN latency were reported by Johannes et al., (2002). Pe amplitude was measured in only one study (Schuller et al., 2018), which indicated lower amplitude in TS than healthy controls. No significant correlation was reported between ERPs amplitude and clinical parameters (Schuller et al.,2018; Warren et al.,

2020) or neuroepileptic medication (Schuller et al., 2018). Tasks used were the Oddball task (Johannes et al., 2002), Stop Signal Task (Schuller et al., 2018) and the Flanker Task (Warren et al., 2020).

3.3 Multiple Sclerosis (MS)

Only one study investigated error monitoring in MS (López-Góngora et al.,2015) using a Flanker Task with an inhibition of response variant ("Stop task"). They reported higher ERN amplitude in MS as compared to healthy controls. Additionally, correlational analyses showed a negative association between ERN amplitude and time since last relapse and a positive association between ERN amplitude and Multiple Sclerosis Severity Score (MMSS) and Expanded Disability Status Scale (EDSS).

3.4 Amyotrophic Lateral Sclerosis (ALS)

Only one study using the Flanker task to measure error related ERPs in ALS was found (Seer et al., 2017a). Results showed no differences in ERN amplitude between ALS and controls. Further analyses on subgroups showed that ALS patients with low executive performance had lower ERN amplitude than ALS patients with high executive performance and controls with low executive performance, while ERN did not differ between controls with low executive performance and controls with high executive performance. Correlational analyses revealed that executive performance negatively correlated with ERN amplitude in the ALS group.

3.5 Cerebellar Dysfunction

A study on patients affected by cerebellar ataxia (CA; Tunc et al., 2019) reported no differences in ERN amplitude between CA and controls, while two studies conducted by the Peterburs group revealed lower ERN in amplitude in patients with cerebellar lesion (CL; Peterburs et al.,

2012) and cerebellar degeneration (CD; Peterburs et al., 2015). No alteration of ERN latency was reported in CL (Peterburs et al., 2012). Peterburs et al., 2015 reported that ERN amplitude negatively correlated with grey matter volume in the cerebellum right lobule V and left lobule VIIb/VIIIa. Moreover, Pe amplitude was found to be lower in CA patients (Tunc et al.,2019) and higher in CL patients (Peterburs et al., 2012) as compared to healthy controls. No differences in Pe amplitude between CD and controls were reported (Peterburs et al., 2015). Shorter Pe latency in CL was reported (Peterburs et al., 2012). Peterburs et al. (2015) reported that Pe was positively correlated with grey matter volume in the right posterolateral cerebellum. The task used in the study on CA by Tunc et al. (2019) was a Flanker task while the Anti-saccade Task was the experimental task for CL and CD studies (Peterburs et al., 2012, 2015)

3.6 Parkinson's Disease (PD)

Eight studies reported reduced ERN amplitude in PD (Beste et al., 2009; Falkenstein et al., 2001; Ito and Kitagawa, 2006; Rustamov et al., 2014; Seer et al., 2017b; Stemmer et al., 2007; Willemssen et al., 2008; Willemssen et al., 2009). Two studies reported no ERN amplitude differences between PD and controls (Holroyd et al., 2002; Verleger et al., 2013). ERN latency was shown to be unaltered in PD in five studies while Falkenstein et al. (2001) reported no differences for Flanker task performance and shorter latencies for Go/NoGo and Simon-type tasks.

Further analyses have been conducted on medication state in PD. Stemmer et al. (2007) and Beste et al. (2009) found no differences in ERN amplitude between drug-naïve and medicated PD. Another study on treated PD patients (Willemssen et al., 2008) revealed no difference between on- and off-medication groups. Seer et al., (2017b) reported reduced ERN amplitude in on-medication PD as compared to off-medication PD. Although the on-medication group

presented reduced ERN amplitude as compared to healthy controls, no differences were found between off-medication PD and control groups.

Correlational analyses showed that BDI scores were positively associated with ERN amplitude in PD (Willemssen et al., 2009). Seer et al (2017b) reported that in the PD off-medication group, ERN amplitude was inversely associated with higher scores in apathy, depression, psychiatric status, and schizotypal scales and positively associated with health status. In the on-medication group reduced ERN amplitude was associated with higher apathy scores.

Pe was measured in only two studies. Ito et al. (2006) showed that Pe amplitude is reduced in PD while Pe latency did not differ between PD and controls. The other study reported no differences in Flanker task and Simon-type task performance, while Pe amplitude was found to be lower in Go/NoGo performance (Falkenstein et al., 2005). Ito et al. (2006) used a Lexical Decision Paradigm; Falkenstein et al. (2001, 2005) applied three paradigms: Flanker task, Simon type task, Go/NoGo task. The other ten studies employed the flanker task.

3.7 Huntington's Disease (HD)

ERN amplitude was shown to be lower in HD as compared to healthy controls (Beste et al., 2009) and pre-clinical HD (pHD; Beste et al., 2008, 2009). No differences in ERN amplitude between pHD and healthy controls were found (Beste et al., 2007). Beste et al. (2009) reported that ERN amplitude did not differ between pHD and young controls, and that was higher in pHD as compared to old controls. Correlation analyses revealed that CAG-index was inversely associated with ERN amplitude (Beste et al., 2006). Additionally, medial frontal gyrus grey matter volume was found to be correlated with ERN amplitude (Beste et al., 2008) No group difference for ERN latency was reported when comparing HD and healthy controls (Beste et al, 2006, Beste et al.,2009), HD and pHC, or pHC and controls (Beste et al., 2009). Beste et al.

(2008) did not find Pe amplitude differences between HD and pHD. All studies used a Flanker Task as the experimental paradigm (Beste et al., 2006, 2007, 2008, 2009).

3.8 Focal Lesions

Studies investigating error related ERPs included lesions of the ACC (1), lateral prefrontal cortex (n=4), orbitofrontal cortex (n=1), frontopolar cortex (n=1), temporal cortex (n=1), basal ganglia (n=1), thalamus (n=2), and left hemisphere regions (n=1). Patients with ACC lesions showed reduced ERN amplitude as compared to healthy controls and a participant group (brain damage control) with brain lesions not involving the ACC (Maier et al., 2015). No between group differences in Pe amplitude and latency were found.

One study involving patients presenting basal ganglia lesions showed reduced ERN and Pe amplitude, while no differences in latency were found as compared to healthy controls (Ullsperger and Von Cramon, 2006). Three studies on lateral prefrontal cortex (PFC) lesions showed lower ERN amplitude as compared to controls (Ullsperger et al., 2002; Wessel et al., 2014; Ullsperger and Von Cramon, 2006). In contrast, Gehring and Knight (2000) reported no differences in ERN amplitude between lateral PFC lesions and control groups. No differences in ERN latency were found (Ullsperger and Von Cramon, 2006; Ullsperger et al., 2002). Pe amplitude was found to be reduced in lateral PFC patients (Ullsperger and Van Cramon, 2006; Ullsperger et al., 2002). Solbakk et al. (2014) reported reduced ERN amplitude and higher Pe amplitude in orbitofrontal lesion patients as compared to healthy controls. The study by Ullsperger et al. (2002) involving a frontopolar and temporal lesion group reported no differences in ERN amplitude and latency and Pe amplitude between lesion groups and controls. Studies on thalamic lesions revealed reduced ERN amplitude (Peterburs et al., 2011; Seifert et al., 2011) and Pe amplitude (Seifert et al., 2011).

Finally, Niessen et al. (2020) reported no differences either in ERN or Pe amplitude and latency between patients with a left hemisphere lesion and healthy controls. Moreover, they found a correlation between ERN latency and lesion size. Peterburs et al (2011) used an anti-saccade task as experimental task, Gehring and Knight used a letter discrimination task, Solbakk et al. (2014) used a stop signal task and Niessen et al. (2020) a Go/NoGo Task. All the other studies used a Flanker task.

3.9 Traumatic Brain Injury (TBI)

Five studies reported reduced ERN amplitude in TBI patients (De Beaumont al., 2013; Larson et al., 2009; Larson et al., 2007; Pontifex et al., 2009). Two studies showed no differences in ERN amplitude between TBI patients and healthy controls (Larson et al., 2012; Shen et al., 2020). Olson et al. (2018) reported higher ERN amplitude in TBI as compared to controls. No differences in ERN latency between TBI and controls were found (Larson et al., 2007; Larson et al., 2009; Shen et al.,2020).

Seven studies reported no difference in Pe amplitude between TBI and healthy controls (De Beaumont et al., 2014; Larson et al., 2009; Larson et al., 2012; Logan et al., 2015; Olson et a., 2018; Pontifex et al., 2009; Shen et al., 2020). One study showed reduced Pe amplitude in TBI (Larson et al., 2007).

While Larson et al. (2007) reported no differences in Pe latency between TBI and controls, Shen et al. (2020) found longer Pe latency in TBI.

Correlational analyses showed that the ERN was negatively associated with number of prior incidents (Pontifex et al., 2009) and concussions (De Beaumont et al., 2014). Moreover, negative affect was inversely correlated with ERN amplitude (Larson et al., 2009). ERN latency was found to be inversely associated with prior TBIs (Larson et al.,2012)

Pe amplitude associated with length of post-traumatic amnesia, and negatively associated with time since injury (Larson et al., 2012). Shen et al., (2020) reported that probability of inhibition (likelihood of response inhibition for Stop trials) negatively correlated with Pe latency and positively correlated with Pe amplitude.

Two studies used a Flanker task (Olson et al., 2018; Pontifex et al., 2009), The Stroop task was used in three studies (Larson et al., 2007,2009,2012). The Error Awareness Task was used by Logan et al (2015). Shen et al. (2020) employed a Stop signal task while De Beaumont et al. (2014) employed a visual search task variant and a visual short-term memory task.

Please insert Table 2 here

4.Discussion

This systematic review included 41 articles assessing the ERN and the Pe in neurological disorders including Alzheimer's disease, Tourette's syndrome, multiple sclerosis, amyotrophic lateral sclerosis, Parkinson's disease, Huntington's disease, cerebellar ataxia, cerebellar degeneration, focal lesions and traumatic brain injury, in comparison with controls. Overall, ERN amplitude tended to be reduced in clinical conditions, with the exception of Tourette syndrome and multiple sclerosis, which seemed to be characterized by enhanced ERN amplitude. Pe amplitude was investigated in fewer studies and did not present a consistent pattern of alteration across different neurological disorders. ERN and Pe latency were generally unaltered with the exception of a few individual studies across different clinical groups. The Flanker Task was the most commonly employed experimental task across neurological conditions, but the use of other paradigms relying on different cognitive processes needs to be considered to discuss contradictory results.

One of the aims of this review was to understand whether alterations of error monitoring are specific to certain neurological conditions, examining the contribution of different brain structures to error monitoring. ERN alterations can be consistently observed in neurological disorders affecting core structures involved in error monitoring. In line with the PRO model (Alexander and Brown, 2011), the medial prefrontal cortex plays a crucial role in error processing. Patients with lesion of the ACC, which is thought to be the ERN neural generator (Brázdil et al., 2005; Debener, 2005; Dehaene et al., 1994; Reinhart and Woodman, 2014; Veen and Carter, 2002), were found to present reduced ERN (Maier et al., 2015). Similar findings were found in studies in AD (Ito and Kitagawa, 2005; Mathalon et al., 2002), in which ACC dysfunction is well documented (Rosenberg et al., 2015). Interestingly, previous research associated ACC alterations with self-awareness in AD (for a review, see Lenzoni et al., 2020), thus confirming its crucial role in self-monitoring alterations in these patients. According to the *Reinforcement Learning* theory (Holroyd and Coles, 2002), error signaling relies on mesencephalic dopaminergic activity from the basal ganglia to the ACC. Neurodegenerative disorders affecting basal ganglia and dopamine regulation consistently presented alteration of the ERN. The ERN was shown to be reduced in PD (Beste, et al., 2009; Falkenstein et al., 2001; Ito and Kitagawa, 2006; Rustamov et al., 2014; Seer, et al., 2017b; Stemmer et al., 2007; R. Willemssen et al., 2008; Willemssen et al., 2009) and in HD (Beste et al., 2006; Beste et al., 2009), while higher ERN amplitude was found in TS (Johannes et al., 2002; Schüller et al., 2018; Warren et al., 2020). Moreover, the ERN was found to be reduced in patients with a focal basal ganglia lesion (Ullsperger and Von Cramon, 2006). These findings show that changes in dopamine levels mediate performance monitoring processes, as previously suggested by research reporting the impact of dopamine antagonists (Forster et al., 2017; Zirnheld et al., 2004) and dopamine receptors genotypes (Biehl et al., 2011; Krämer et al., 2007) on the ERN. Focal thalamic lesions were also associated with reduced ERN (Peterburs et al., 2011; Seifert

et al., 2011). Crucially, the thalamus plays a key role in the generation and updating of mental representation (Wolff and Vann, 2019), and is considered a relay of efferent copies (or corollary discharge) of motor commands (Sommer, 2003). Therefore, thalamic alterations may have a disruptive impact on the cognitive conflict between competing representations and their "translation" into the appropriate motor commands to be selected during task performance.

Interestingly, even when the "core" neural network underlying error monitoring is not directly affected, the ERN may be altered. When analyzing the studies involving these neurological conditions, contradictory results can be found and no clear pattern can be defined. However, considering the interaction between the experimental task and the lesion localization, it can be hypothesized that the ERN does not merely rely on the integrity of the structures involved in conflict processing and error detection, but also on the alteration of those cognitive processes mediating the generation of the competing representations. Two studies showed reduced ERN in participants affected by cerebellar dysfunctions (Peterburs et al., 2012, 2015). Peterburs et al. (2012) included patients vascular focal damage to the cerebellum. By comparison, Peterburs et al (2015) patient group, that we labeled cerebellar degeneration, included pathologies that primarily affect the cerebellar cortex, such as spinocerebellar ataxia, sporadic adult onset ataxia, and autosomal dominant ataxia. In both studies the paradigm used was the Antisaccade Task. The third study reported spared ERN amplitude in cerebellar ataxia (Tunc et al, 2019), which includes different types of spinocerebellar ataxia. The task used was the Flanker Task. Therefore, online performance monitoring in patients affected by cerebellar dysfunction results to be impaired for cognitive abilities that rely on cerebellar integrity, such as saccadic eye movement generation, as shown by the Peterburs group. In contrast, error monitoring appears to be spared for functions that are not prominently mediated by cerebellar activation, such as for the Flanker Task. Similarly, the ERN was shown to be reduced in patients with a lateral

PFC lesion during the Flanker Task performance (Ullsperger et al., 2002; Ullsperger and Von Cramon, 2006; Wessel et al., 2014), while it was unaltered as compared to controls when performing a letter discrimination task (Gehring and Knight, 2000). Furthermore, the ERN was found to be larger in patients with orbitofrontal lesions during a Stop Signal Task (Solbakk et al., 2014) but was shown to be unaltered during the Flanker Task performance (Ullsperger et al., 2002). This suggests the presence of domain-specific mechanisms underlying error monitoring, that may selectively affect task performance. Beyond a domain-general "core" network, domain-specific neural signals contribute to the generation of competing representations, and therefore, mediate error processing. This notion would imply the existence of domain-specific alterations of representations (and their correctness), supporting theoretical accounts such as the *Mismatch Theory (*(Dehaene, 2018; Falkenstein et al., 1991; Gehring et al., 1993; Scheffers and Coles, 2000) and PRO model (Alexander and Brown, 2011), that emphasize the pivotal role of multiple competing representations and their response outcome during performance monitoring, rather than a general mechanisms of response conflict, as proposed by the *Conflict Monitoring* hypothesis (Botvinick et al., 2001; Yeung et al., 2004). Although, domain-specificity of metacognitive processes, such as self-monitoring, have been previously discussed (Mograbi and Morris, 2014) and supported by behavioral (Bellon et al., 2020; Chapman et al., 2018; Dentakos et al., 2019) and neuroimaging (Morales et al., 2018) studies, it is yet to be investigated in ERP research. Nonetheless, inconsistent findings within neurological conditions may be also mediated by heterogeneity in methodology, such as task instructions (Morris et al., 2006), number of error trials (Fischer et al., 2017), or task difficulty (Riesel et al., 2015), and individual differences, such as motivation (Boksem et al., 2006), affective state (Wiswede et al., 2009), or stress (Hu et al., 2019) that may modulate the ERN differently across clinical and healthy populations.

A smaller proportion of the studies included analyses of the Pe. Overall, the results are in line with a functional distinction between ERN and Pe (Di Gregorio et al., 2018; Overbeek et al., 2005) as demonstrated by the lack of unidirectional changes across many different neurological conditions. Pe was shown to be unaltered in the presence of ERN reduction (Beste et al., 2008; De Beaumont et al., 2013; Larson et al., 2009; Maier et al., 2015; Olson et al., 2018; Peterburs et al., 2015; Pontifex et al., 2009), thus supporting the idea that ERN and Pe represent independent systems of error monitoring. Critically, in most of the neurological conditions considered in this review, we have evidence on the Pe from one study only. Therefore, it is more difficult to draw major conclusions about Pe integrity within individual conditions.

However, it is important to observe that the ACC may not have a prominent role in Pe generation, as shown by unaltered Pe in patients with ACC lesion (Maier et al., 2015) and AD (Ito et al., 2005). Among basal ganglia disorders, Pe was reduced in focal lesion (Ullsperger and Von Cramon, 2006) and TS (Tunc et al., 2019) patients. It should be noted that the Ullsperger group reported reduced Pe in lateral PFC lesion patients, and in 5 out of 7 patients, the lesion extended to the insula (Ullsperget et al., 2002; Ullsperger and Von Cramon, 2006). Contradictory results were found in PD patients. Ito et al. (2006) reported a reduction in Pe, while another study involving three experiments found no differences in Pe amplitude between PD and controls for Flanker Task and Simon-type task performances, and reduced Pe during a Go/NoGo task (Falkenstein et al., 2005). Findings from cerebellar dysfunction are also contradictory, but an association between Pe amplitude and dystonia severity was found in cerebellar ataxia patients (Tunc et al., 2019), suggesting a relation between motor dysfunction and decrease in Pe.

According to the *Accumulation Account* (Steinhauser and Yeung, 2010; Ullsperger et al., 2010), the Pe emerges when sufficient evidence about error commission has been accumulated. This would involve the integration of conflict/response information, proprioception, interoception, and sensory inputs (about action performance). It has been hypothesized that brain structures involved in the emergence of error awareness includes cingulate structures, somatosensory areas and anterior insula (Ullsperger et al., 2010; Klein et al., 2013; Hester et al., 2005). Importantly, peripheral and visceral signals could contribute to the generation of the Pe and such factors must be considered in central and peripheral nervous system pathologies that could affect sensorimotor processing. It could be hypothesized that motor diseases, including those affecting the peripheral nervous system, may suffer from changes in sensorimotor information processing that could contribute to accumulation processing underlying error awareness. The anterior insula, integrating signals ascending from peripheral pathways, plays a key role in interoceptive awareness (Chen et al., 2021), and the ACC are key nodes of the salience network (Uddin, 2015). In line with *Adaptive Orienting* theory, the salience network, as well as the frontobasal ganglia network, are involved in post-error processing (Wessel, 2018). Recent evidence on cross-network interactions involved in cognitive control suggests that that salience network may play a crucial role in real-life self-control by initiating switching between default mode and executive networks (Krönke et al., 2020), thus underlining the critical involvement of anterior insula and ACC in self-monitoring and self-regulation.

Nevertheless, error awareness and its relation with the Pe has not been explored in neurological conditions, except for Logan et al. (2015). In their study, they used the Error Awareness Task which allowed to investigate ERPs differences for aware and unaware errors in TBI patients. Although they found no differences between patients and controls, a significant effect of awareness on Pe amplitude in both groups was observed. Such experimental manipulations can

be critical in the analyses of the Pe, by potentially revealing differences otherwise undetectable, and exploring the differential association between aware and unaware errors with other measures.

Moreover, we explored whether lesion lateralization was associated with error monitoring system dysfunction; one recent study's sample included only patients with left hemisphere lesion (Niessen et al., 2021), reporting no group differences in either ERN or Pe. In the rest of the studies, the patient group included either both hemispheres lesions or bilateral lesions, and in the first case, no subgroup analyses exploring lateralization effect was conducted. Therefore, considering the heterogeneity of lesion localization and size in the study by Niessen et al. (2021), it is difficult to discuss any potential hemispheric asymmetry of the performance monitoring system.

Importantly, some methodological issues need to be acknowledged when considering the presence of inconsistent findings across and within neurological conditions. The studies reviewed present relevant differences in sample size (ranging from 6 to 36 for the clinical group), experimental manipulations (Fischer et al., 2017; Mathewson et al., 2005; Morris et al., 2006), and quantification of ERP-related metrics (Overbeek et al., 2005). Moreover, a large part of the studies employed the Flanker task (n=23/41), and the number of studies focusing on specific neurological disorders is unbalanced, with, for example, wider research on Parkinson's disease (n=11) and TBI (n=8) and limited investigation of multiple sclerosis (n=1), amyotrophic lateral sclerosis (n=1).

The second aim of this review was to investigate the associations between error-related ERPs and clinical factors. Overall, across different neurodegenerative disorders, we can observe that

ERN and Pe are associated with disease severity measures (n=14/17 including correlational analysis). Pe amplitude was shown to be associated with dystonia severity in CA patients (Tunc et al., 2019) and both ERN and Pe with cerebellar GWM in CD (Peterburs et al., 2015). In MS patients, ERN correlated with time since last relapse and disease severity measures (Lopez-Gongora et al., 2015). In HD, the ERN correlated with size of CAG repetitions (Beste et al, 2009), which is typically used as a severity index (Duyao et al., 1993; Rosenblatt et al., 2006), and with medial frontal grey matter volume (Beste et al., 2008). This suggests that error monitoring ERPs may represent a reliable measure of neurodegeneration processes.

Given the heterogeneity of neurological profiles, clinical outcomes and recovery trajectories in TBI patients (Azouvi et al., 2017; Bigler, 2001; Chastain et al., 2009; Green et al., 2008; Perlbarg et al., 2009; Rabinowitz et al., 2018), it is cautious to say that we cannot establish whether ERPs alterations are specific for this neurological condition. However, this line of research provided relevant knowledge about the association between clinical factors and error monitoring. Several measures of trauma severity were found to be associated with the ERN across many studies. ERN amplitude and latencies were shown to be associated with higher number of TBIs (De Beaumont et al., 2013; Larson et al., 2007; Pontifex et al., 2009) and Pe amplitude was found to be correlated with post-traumatic amnesia length and time since the injury (Larson et al., 2012). Among others, post-traumatic amnesia is considered a strong predictor of clinical outcomes (Ponsford et al., 2016), thus suggesting that error-related ERPs may not only index injury severity but also predict outcomes after TBI. Importantly, self-awareness impairments are very common in TBI (Prigatano, 2005; Sherer et al., 2003, 1998) and multi-dimensional measures of self-awareness, including error monitoring, play a critical role in TBI interventions (Robertson and Schmitter-Edgecombe, 2015; Simmond and Fleming, 2003). In TBI patients, performance monitoring deficits were found to be associated with

activity of the dorsal ACC and anterior insula (Ham et al., 2014), supporting the relevance of these areas for error processing.

Moreover, the relations between the ERN and depression (Willemssen et al., 2008; Seer et al., 2017b), negative affect (Larson et al., 2009) and psychiatric symptoms (Seer et al., 2017b) point out the critical relevance of error monitoring in clinical profiles of neurological disorders. Extensive research on psychiatric conditions identified impairment of error processing, as reflected by ERN alterations. For example, the ERN has been proposed as endophenotype of internalizing disorders (Olvet and Hajcak, 2008; Weinberg et al., 2015), specifically of obsessive-compulsive disorder (OCD; Riesel, 2019), anxiety (Riesel et al., 2019), and as candidate biomarker of depression (Clayson et al., 2020). Reduction of reduction of error-related ERPs have also been reported in psychopathy (Vallet et al., 2021), schizophrenia (e.g., Bates et al., 2002; Foti et al., 2012; Simmonite et al., 2012), and bipolar disorder (Minzenberg et al., 2014; Morsel et al., 2014). Further research is needed to extend the knowledge about overlapping neural networks underlying performance monitoring in neurological and psychiatric disorders. For instance, TS and OCD have been suggested to share pathophysiological mechanisms, possibly reflecting similarities between tics and repetitive behaviors associated with cortico-striato-thalamo-cortical circuitry dysfunction (Hartmann and Millet, 2018). Moreover, OCD is often present of comorbidity of TS (Sheppard, 1999). Although performance monitoring in these two clinical populations has never been directly compared, previous research showed that the ERN is typically enhanced, suggesting hyperactive error signals in both conditions (Johannes et al., 2002; Riesel, 2019; Schüller et al., 2018; Warren et al., 2020). Warren et al. (2020) observed that increased ERN in TS may reflect compensatory mechanisms that allow successful behavioral performance.

As shown by the current review, all the studies (n=3) including TS showed higher ERN amplitude but comparable behavioral performance as compared to healthy controls. A similar phenomenon has been highlighted by Lopez-Góngora et al. (2015) concerning error monitoring in multiple sclerosis. However, this hypothesis is not supported by findings from other neurological disorders, in which no systematic pattern linking ERN to task performance, especially for accuracy and reaction times, can be identified. A limited number of studies within and across neurological disorders included measurea of post-error adjustments, such as post-error slowing (n=16) and post-error accuracy (n=4). Nonetheless, no consistent association between post-error measures and ERPs was found.

In conclusion, these findings highlight the link between error monitoring networks, self-awareness, and neurocognitive rehabilitation outcomes. Error-related ERPs may be employed in assessment protocols to evaluate patient ability to monitor their own functioning and understand the severity of their conditions. Error monitoring ERPs can also be important measures for rehabilitation effectiveness, because error detection and correction can be critical for (re)learning mechanisms (Ownsworth et al., 2017).

Future research should investigate domain-specificity of error monitoring and the role of functional disconnection within performance monitoring networks. This would extend our knowledge on brain processes underlying error monitoring and provide useful information on specific cognitive deficits for neuropsychological assessment and rehabilitation. Furthermore, future studies investigating error monitoring in neurological disorders would benefit from including: 1) both ERN and Pe amplitude and latency analyses; 2) experimental manipulations to distinguish aware and unaware errors in order to explore the relation between Pe, error awareness, and other variables; 3) clinical and neurocognitive measures, and assessment of

psychiatric comorbidities; 4) subgroup analyses exploring differences between left and right hemisphere lesion; 5) behavioral measures of post-error adjustments. Finally, combining EEG with non-invasive brain stimulation techniques could offer new perspectives to elucidate the relative contribution of different brain structures in error monitoring and potential tools for error processing and self-awareness rehabilitation.

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