Doing it Wrong: A Systematic Review on Electrocortical and Behavioral Correlates of Error Monitoring in Patients with Neurological Disorders

Pezzetta, R.; Wokke, M.E.; Aglioti, S.M.; Ridderinkhof, K.R.

DOI
10.1016/j.neuroscience.2021.01.027

Publication date
2022

Document Version
Final published version

Published in
Neuroscience

NEUROSCIENCE

REVIEW

R. Pezzetta et al. / Neuroscience 486 (2022) 103–125

Doing it Wrong: A Systematic Review on Electrocortical and Behavioral Correlates of Error Monitoring in Patients with Neurological Disorders

R. Pezzetta,[a]* M. E. Wokke,[b,c] S. M. Aglioti[d,e] and K. R. Ridderinkhof[f,g]

[a] *IRCCS San Camillo Hospital, Venice, Italy*

[b] *Programs in Psychology and Biology, The Graduate Center of the City University of New York, New York, NY, USA*

[c] *Department of Psychology, The University of Cambridge, Cambridge, UK*

[d] *Sapienza University of Rome and CNLS@Sapienza at Istituto Italiano di Tecnologia, Via Regina Elena 295, 00161 Rome, Italy*

[e] *Fondazione Santa Lucia, IRCCS, Rome, Italy*

[f] *Department of Psychology, University of Amsterdam, Nieuwe Achtergracht 129B, 1018, WS, Amsterdam, The Netherlands*

[g] *Amsterdam Brain & Cognition (ABC), University of Amsterdam, Amsterdam, The Netherlands*

Abstract—Detecting errors in one's own and other's actions is a crucial ability for learning and adapting behavior to everchanging, highly volatile environments. Studies in healthy people demonstrate that monitoring errors in one's own and others' actions are underpinned by specific neural systems that are dysfunctional in a variety of neurological disorders. In this review, we first briefly discuss the main findings concerning error detection and error awareness in healthy subjects, the current theoretical models, and the tasks usually applied to investigate these processes. Then, we report a systematic search for evidence of dysfunctional error monitoring among neurological populations (basal ganglia, neurodegenerative, white-matter diseases and acquired brain injury). In particular, we examine electrophysiological and behavioral evidence for specific alterations of error processing in neurological disorders. Error-related negativity (ERN) amplitude were reduced in most (although not all) neurological patient groups, whereas Positivity Error (Pe) amplitude appeared not to be affected in most patient groups. Also theta activity was reduced in some neurological groups, but consistent evidence on the oscillatory activity has not been provided thus far. Behaviorally, we did not observe relevant patterns of pronounced dysfunctional (post-) error processing. Finally, we discuss limitations of the existing literature, conclusive points, open questions and new possible methodological approaches for clinical studies.

This article is part of a Special Issue entitled: Error Processing

Key words: neurological disorders, error processing, EEG, behavior, systematic review.

INTRODUCTION

The ability to detect errors in one's own as well as in others' actions is central for flexible behavioral interactions with objects and people (Maier et al., 2011; Cavanagh and Frank, 2014; Ullsperger et al., 2014a). Mounting evidence suggests specific ways in which the brain signals the occurrence of an error and the need to monitor performance and adjust it to the environment. However, error monitoring and cognitive control may be dysfunctional in neurological disorders, in which deviant brain reaction to errors, and great difficulty to inhibit intrusive and conflicting responses may occur (Ullsperger and von Cramon, 2006). Exploring the functioning of the error monitoring system in patients with neurological disorders may complement studies in healthy populations and provide fundamental contributions to elucidate the complexity of the system itself.

In this vein, in the present review we discuss error processing in patients with neurological diseases, focusing mainly on EEG and behavioral performance alterations. For studies based on techniques other than EEG, which fall beyond the scope and space of the present review, the reader is referred to discussions elsewhere (for reviews see Koban and Pourtois, 2014; Gratton et al., 2018a, 2018b).

We performed a systematic search of the existing literature by following the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA; Moher et al., 2009). In the introductory part, we will briefly overview the current state of psychophysiology of error

*Corresponding author.
E-mail address: rachele.pezzetta@ospedalesancamillo.net (R. Pezzetta).

https://doi.org/10.1016/j.neuroscience.2021.01.027
0306-4522/

monitoring in healthy participants and animals, and of the main tasks employed in this very active research domain. In the core part of the paper, we will describe the main results of the systematic search of data from neurological populations, by focusing on dysfunctional performance monitoring depending on a given pathological state. Finally, conclusive points, open questions and new possible methodological approaches of relevance for clinical studies will be discussed.

WHAT IS AN 'ERROR'?

In the context of this review, the term error refers to an event in which action performance fails, and the outcome is worse than expected. Based on this definition, the interpretation of an event can change according to task instructions or fluctuations in expectations (Shadmehr, 2017). The capacity to quickly detect a wrong output in an action and to plan future strategies (i.e. by selecting contextually relevant information for preventing further errors) is a fundamental ability in everyday life (Clark, 2013; Kilner et al., 2007). This type of skill is particularly relevant in specific domains that require online detection of relevant information, such as in sport (Aglioti et al., 2008; Proverbio et al., 2012; Ridderinkhof and Brass, 2015; Özkan et al., 2019), music (Panasiti et al., 2016) or rehabilitation trainings (Bettcher et al., 2008; Klein et al., 2013).

Over the years, the concept of error assumed slightly different connotations. An early hypothesis suggested that the error signal reflects a process that compares the output of the motor system with the original planning of an action (efference copy) (Falkenstein et al., 1991). Later studies suggested that the error monitoring system serves to detect conflicts in information processing when two or more options emerge, a perspective that has been called *conflict processing* (Botvinick et al., 2001; Yeung et al., 2004). The *reinforcement learning hypothesis* (RFL, Holroyd and Coles, 2002) suggested instead that when performance is worse than expected, errors in reward prediction are coded by phasic decreases in the activity of mesencephalic dopamine-generating neurons, serving as signals that help optimize task performance in accordance with the principles of reinforcement learning. The RFL model can be also integrated in turn with the conflict processing perspective, supporting the notion that error-related EEG signatures predict reinforcement learning and conflict biases (Frank et al., 2005). A more recent model of error monitoring is the *predicted-response outcome* (PRO, Alexander and Brown, 2011) that assumes that the frontal regions of the brain react to prediction-errors and generate standard learning rules of probability. This model builds on Brown and Braver's (2005) suggestion that the brain predicts error likelihood rather than response conflict. More generally, the accounts that consider error as a prediction deviation share some similarities with the *predictive coding framework*, according to which prediction error depends and is modulated by the incongruence between predicted and actual outcomes (Friston, 2010; Clark, 2013; Ridderinkhof, 2014).

ELECTROPHYSIOLOGICAL SIGNATURES OF ERROR-MONITORING

Studies in humans (Luu et al., 2004; Hajcak et al., 2005), nonhuman primates (Tsujimoto et al. 2006) and rodents (Narayanan et al., 2013) have shown that the monitoring for erroneous outcomes is associated with specific cortical responses. As widely investigated with fMRI studies, the processes of error monitoring trigger the activity in a network centered on the middle-frontal regions of the brain (Luu et al., 2004; Ridderinkhof et al., 2004; Cohen, 2011; Hoffman & Beste, 2015), with a fundamental role of the anterior cingulate cortex (ACC), the medial and lateral prefrontal areas, encompassing also limbic and motor regions (Carter et al., 1998; Wang et al., 2005; Ullsperger et al., 2014a; Ullsperger et al., 2004b). However, electrophysiological studies provide an important methodological approach to the investigation of action monitoring. First, event-related potentials (ERPs) and changes in oscillatory activity provide a more direct measure of neural activity compared to MRI methods; second, error-related EEG signatures have revealed temporal and topographical properties which have been associated with specific aspects of performance monitoring; third, techniques such as EEG, magnetoencephalography (MEG) and intracranial electrophysiological recordings (iEEG or ECoG) allow inferences about the time course and the temporal sequence of the neurophysiological processes, with millisecond temporal resolution (Cohen, 2014).

Psychophysiological studies of error processing have highlighted a negative potential that appears when an error is committed, originally called 'Error Negativity' (Ne; Falkenstein et al., 1991), and later identified also with the term 'Error-related negativity' (ERN). Early studies explored the brain mechanisms related to a correct or incorrect choice (Rabbitt et al., 1977; Renault et al., 1980), but interest in this topic was boosted in the early 1990′s by the studies of Falkenstein et al. (1991) and Gehring et al. (1993). The ERN develops at the time of the first incorrect muscle activity and peaks about 100 ms later. It is triggered by errors elicited under speeded response conditions independently from the response effector (such as hands, feet, eyes, or voice), and increases in amplitude with the size or degree of error (Falkenstein et al., 2000). The mainstream interpretation of the functional significance of the ERN appears to be that is signifies the activity in posterior ACC in translating conflict signals and/or reward prediction errors into signaling the need for behavioral adaptation.

Another ERP component that has been extensively linked to error monitoring is the Error Positivity (Pe). The Pe is a sustained positivity, with a diffuse topography and maximal amplitude in correspondence of the posterior electrodes. The Pe is considered an ERP component that shares many similarities with the P300 (Polich, 2007; Ridderinkhof et al., 2009). In addition to a shared scalp distribution and ERP morphology, the P300 and Pe are elicited by task-salient stimuli (e.g. low-probability events). The Pe may consist of at least two distinct components with dissociable features; an early frontocentral component associated with the orientation of attention (Ruchsow et al., 2005) and a later

centroparietal component linked with the conscious recognition and the motivational significance of the erroneous event (Overbeek et al., 2005; Ridderinkhof et al., 2009). Other scholars suggested that the Pe might reflect the evaluation of evidence in working memory, thus representing a gradual constant updating and accumulation of evidence (Donchin and Coles, 1988; Dehaene and Changeux, 2003; Steinhauser and Yeung, 2012; Wessel, 2012; Di Gregorio et al., 2016). More recently, post-decisional evidence accumulation (thought to continue after an incorrect decision and response have been made) has been proposed to provide a fruitful, mechanistically explicit framework for understanding the functional significance of the Pe, accounting not only for its association with error detection, but also for specific aspects of its morphology (Murphy et al., 2015) and more recent links with graded confidence judgments (Boldt and Yeung, 2015) and future behavioural adjustments (e.g. Desender et al., 2019).

The literature also documents a series of additional conflict-related ERPs. First, the N2 is a component elicited by a mismatch condition, that has been often studied in combination with the following P3 component, generating the N2-P3 complex; the N2 has been taken to stem from ACC and to reflect conflict arising from competition between response alternatives (Nieuwenhuis et al., 2003). Second, a cluster of Stroop-related later potentials (referred to as the N450, the conflict slow potential (SP) also called negative slow wave (NSW)) have been linked to slower aspects of conflict processing. The N450 is an index of conflict that is elicited during the Stroop task and likely represents activity of neural generators localized in the ACC after incongruent trials (Perlstein et al., 2006); the SP or NSW is a slow wave which appears to covary with processes important for adaptive task performance and has been associated to conflict resolution (for a detailed review on conflict-related ERPs see Larson et al., 2014). The feedback-related negativity (FRN) is a potential sensitive to negative feedbacks as well as negative prediction errors and has been also related to corrective actions or performance adjustments (Nieuwenhuis et al., 2004). As responses to feedbacks might or might not rely on processes different than the ones implicated in response to errors, in this review we only included studies that had also direct implications for error-rather than only feedback-responses. In the time–frequency domain, error monitoring has been primarily found in relation to mid-frontal theta oscillations (4–8 Hz) that increase when an error is committed (Cohen, 2011; Cavanagh and Frank, 2014; Wokke et al., 2017). Theta oscillations and ERN generation are functionally linked, and studies suggest that the latter may originate – at least partially – from the phase-locking of the former (Luu et al., 2004; Pezzetta et al., 2018). Theta enhancement has been observed not only in mid-frontal regions, but also in fronto-occipital regions when processing of bodily stimuli is involved (Arrighi et al., 2016; Moreau et al., 2020a; Fusco et al., 2020). Beta oscillations (12–30 Hz) appear also to be enhanced when an error is committed or observed (Koelewijn et al., 2008; Torrecillos et al., 2015). However, while mid-frontal theta oscillations have been observed frequently in response to errors, consistent evidence on the beta activity has not been provided thus far.

Interestingly, studies on healthy populations have found dissociations between the occurrence of an ERN (or increased theta activity) and the occurrence of the Pe (Di Gregorio et al., 2016, 2018; Hughes & Yeung, 2011). These data might indicate that the error monitoring system is characterized by at least two processes that are independent of one another and rely on different neural networks (Nieuwenhuis et al., 2001; Overbeek et al., 2005; Endrass et al., 2007). This view is supported by source analyses and dipole fitting indicating that the origin of the ERN/theta activity and the Pe might rely on different neural regions. While the ERN seems to be generated by posterior areas of the medial prefrontal cortex (Herrmann et al., 2004; Ullsperger and von Cramon, 2006; Ullsperger et al., 2014a), the generator of the Pe seems to be situated in the rostral (Bush et al., 2000) or caudal ACC (Herrmann et al., 2004). Interesting findings are also obtained by combined EEG/fMRI studies (Debener et al., 2005; Iannaccone et al., 2015). However, the debate on the generators of the ERN/theta and Pe is still ongoing, and studies on patients might help clarify the dispute.

HOW DO WE INVESTIGATE ERROR PROCESSES IN COGNITIVE NEUROSCIENCE?

In experimental psychology, in order to investigate the error processes, researchers have been using tasks in which they could control confounding variables and isolate those of interest. In most of these tasks, errors are defined as incorrect responses in relation to specific task instruction (i.e. press a certain button when a stimulus appears on the screen), stressing the speed as well as accuracy of the response. In this sense, most of the times, the committed erroneous action is a wrong action compared to an external request. In some conditions, also external feedback is provided so that participants learn whether the performance generated a positive or negative feedback (Cohen et al., 2007; Holroyd and Coles, 2002). The most common computerized tasks used to investigate conflict and error monitoring using EEG are: (i) the Eriksen Flanker task (in the original or modified versions), in which responses to irrelevant (spatial) stimuli have to be inhibited in favor of responses to a target stimulus (Eriksen and Eriksen, 1974). This task usually elicits the ERN/mid-frontal theta and the Pe component in response to errors. The N2 and the N2P3 complex are also seen on incompatible trials; (ii) the Stroop task in which participants have to name the colour of the ink of coloured words, inhibiting to pronounce the name of the colour instead (Stroop, 1935). The Stroop (as well as modified versions of it) has been associated with the observation of the N450, the SP (or NSW) in association to slower aspects of conflict processing, as well as the ERN and the Pe in response to errors; (iii) the go/no-go task in which individuals have to press a button when a certain stimulus appear

(go) and withhold a response to a proportion of other stimuli (no-go; Simson et al., 1977). EEG studies with the go-/no-go have generally found the ERN and Pe in response to errors. The N2 is often seen on the appearance of no-go trials; (iv) the antisaccade task, in which the participants is asked to make a saccade in the opposite direction of the stimulus (e.g. if the stimulus appear on the right, the saccade has to be made towards the left; Hallett, 1978). This task has shown to elicit the ERN and Pe; (v) the stop-signal task in which the participants have to respond as quicky as possible to a predetermined stimulus (go) and abort any response when a stop stimulus is displayed (Logan and Cowan, 1984). At variance with the go/no-go task, the go stimulus in the stop-signal task (SST) is always shown first, and is followed by a stop stimulus after a short delay. The SST is associated with the generation of the ERN, the Pe and the N2/P3 complex associated to conflict and inhibition processes. Although not frequently used in neurological populations, the SST is widely applied to investigate error processing and response-inhibition efficiency (Verbruggen et al., 2019). Interestingly, employing tasks in which participants observe someone else committing an error elicits analogous error-related signatures (Miltner et al., 2003; van Schie et al., 2004). This parallel neural response indicates that observational learning relies on neural processes that are similar to those involved in action execution and may denote an adaptive mechanism that allows to learn from other's behavior and establish effective social interactions (i.e. synchronous grasping; Candidi et al., 2017; Era et al., 2020; Moreau et al., 2020b). In this more naturalistic direction, also tasks that engage participants during complex motor tasks (i.e. throwing a ball, moving a lever) require natural behavioral adaptations after error commission (Maurer et al., 2015; Joch et al., 2017). In recent years, the advent of immersive virtual reality has also stimulated studies of error-related processes in more ecological settings and everyday scenarios (Padrao et al., 2016; Pavone et al., 2016; Pezzetta et al., 2018; Spinelli et al., 2018).

Tasks that require speeded responding are typically used to examine cognitive efficiency by calculating the speed of responding (reaction time, RT) and the level of accuracy (choice errors in choice-RT tasks such as the Eriksen and Stroop tasks; commission errors in go/no-go tasks and stop signal tasks). In such tasks, when participants make a response error, they tend to shift their speed-accuracy balance to prevent further errors (referred to as post-error slowing, PES; Notebaert et al., 2009; Ullsperger et al., 2014a; Ullsperger and Danielmeier, 2016). In conflict tasks such as the Stroop and Eriksen tasks, a similar performance adaptation is seen in the form of post-error reduction of interference, showing that response conflict has less detrimental effect on response speed immediately after a response error (Ridderinkhof et al., 2003).

THE INTEGRITY (OR DISRUPTION) OF ERROR PROCESSES IN NEUROLOGICAL POPULATIONS

Dysfunctional performance monitoring may be tested by examining the behavioral consequences of errors. Deficits in the monitoring system can result in decreased and delayed error correction, absence of post-error slowing or post-error reduction of interference (Fusco et al., 2018; Rabbitt et al., 1977). Performance adjustments in speed-response tasks usually imply an increase of top-down control and the implementation of strategies, when needed.

Dysfunctional performance monitoring may be observed not only at the behavioral level (RT, accuracy, PES), but also in terms of modulation of error-related brain responses, such as ERN/Pe or oscillatory activity. Previous work pointed at impaired performance monitoring in neurological patients (Ullsperger, 2006). However, to the best of our knowledge, no recent and systematic review has been conducted with the aim to examine psychophysiological evidence of alterations of the error monitoring system in a wide spectrum of neurological disorders. Investigating the integrity (or disruption) of the performance monitoring functions in patients can help better elucidate the processes implied in the monitoring system, as well as the cognitive symptoms that might be directly linked with a given disease. Additionally, understanding which monitoring processes and which networks are altered by a given pathology, could help create better strategies in clinical practice.

EXPERIMENTAL PROCEDURES

Search

We performed a literature search on two databases, PubMed and PsycInfo, using the search terms ‘‘prediction error”, ‘‘error detection”, ‘‘error processing”, ”cognitive control”, ‘‘error negativity”, ‘‘error positivity”, ‘‘error-correction”, ‘‘error-related negativity”, ‘‘ERN”, ‘‘feedback negativity”, ‘‘FRN”, in combination with ‘‘electroencephalography (MeSh Terms)”, ‘‘magnetoencephalography (MeSh Terms)”, ‘‘event-related potential/s”, ‘‘EEG”, ‘‘ERP”, ‘‘MEG”, ‘‘electrical”, or ‘‘oscillations”. Those terms had to co-occur in combination with one of the following search terms for the main neurological disorders: ‘‘brain damage”, ‘‘lesion/s”, ‘‘neurological”, ‘‘Parkinson disease”, ‘‘multiple sclerosis”, ‘‘spinal cord”, ‘‘stroke” (all MeSh terms), ‘‘mild cognitive impairment”, ‘‘dementia”, ‘‘Alzheimer”, ‘‘Parkinson”, ‘‘Huntington”, or ‘‘multiple sclerosis” (all keywords). All included articles were required to be published in peer-reviewed journals and indexed in PubMed or PsycInfo from 1st 1991 to 8th April 2020. The search was limited to articles written in English. Criteria of exclusion were established a priori and were the following: (i) studies focusing on animals, children, single-cases or healthy adults only; (ii) studies on sleep, treatments or invasive interventions; (iii) studies with techniques which did not include also EEG/MEG; (iv) studies without empirical data (i.e., reviews or theoretical models); (v) studies that consider electrocortical signatures that do not have also direct implications for error processes (i.e. FRN); (vi) studies on psychiatric disorders that do not primarily entail some neurological disorder.

Risk of bias

To reduce the risk of bias, two reviewers (R.P. and M.W.) independently screened the articles and decided for appropriateness. Discrepancies in the evaluation of an article, were resolved through consensus.

Results of the systematic search

The systematic literature search revealed 249 articles. The search included also papers that matched the inclusion criteria and have been found through external sources (i.e. 5 articles, obtained from hand-search). Out of these, 98 were rejected because of duplicates. A total of 73 studies fulfilled our inclusion criteria (details can be seen in the Fig. 1, Prisma Flow Diagram). After the screening of each full-text article, a total of 55 articles were included. Identified studies were categorized into the following subgroups: basal ganglia diseases (including Parkinson's Disease, Huntington Disease), neurodegenerative disease (including Alzheimer, Mild-Cognitive Impairment), acquired brain injury (which included stroke, cerebellar lesion, prefrontal lesion, thalamic damage, traumatic brain injury, basal ganglia lesion), and white-matter lesions. A summary with the main results of the systematic search for each pathological section can be found in the corresponding Tables (1–5).

Basal ganglia disease

The basal ganglia are a group of subcortical nuclei involved in the error monitoring system that comprise the striatum (caudate nucleus and putamen), the external and internal pallidal segments, the subthalamic nucleus, and the substantia nigra. The basal ganglia are involved in complex non-motor functions (Calabresi et al., 2006; Klaus et al., 2019; Ponsi and Panasiti, 2020), and can be considered also as a hub largely involved in information processing, reward and cognition. These regions are indeed vital nodes in complex cognitive networks, densely interconnected with parietal and prefrontal cortices as well as with the ACC (Alexander et al., 1986, Ridderinkhof et al., 2003). Numerous studies showed how dopaminergic neurons in the ventral tegmental area respond to unexpected reward (Schultz, 1997) and prediction error (Bayer and Glimcher, 2005), two key elements in developing appropriate goal-directed actions (Schultz, 2002). The main pathologies in which the basal ganglia functions are greatly affected are Parkinson's and Huntington's Disease (see also Table 1 for a summary of results).

Parkinson's Disease. Parkinson's Disease (PD; Parkinson, 1817) is a progressive pathology characterized by the degeneration of dopaminergic neurons in the substantia nigra pars compacta, which results in a depletion of dopamine that gradually extends to the limbic and neocortical regions (Chaudhuri et al., 2005; Kendi et al., 2008; Halliday, and McCann, 2010). The alteration of the functionality of those circuits impairs motor abilities and leads to the typical motor alterations associated with PD (i.e. extreme slowness of movements and reflexes). However, clinical and neuropsychological studies demonstrated that as the disease progresses, motor deficits are frequently associated also to cognitive impairment (i.e. executive dysfunctions; Cools, 2006; Farooqui et al., 2011; Angelucci et al., 2015) and altered brain activity (Seer et al., 2016). **Studies: ERN/mid-frontal theta.** Several studies found reduced ERN/Ne amplitude and lower theta power in PD (13 studies). In an early, influential study, Falkenstein and colleagues (2001) tested nondemented PD patients in three different tasks (a modified Eriksen flanker task, Simon-type task and complex go/no-go) and found smaller ERNs. Similarly, later studies found reduced ERN amplitude in PD patients, using the Flanker task (Stemmer et al., 2007; Willemssen et al., 2008, 2009; Seer et al., 2017) or a lexical decision task (Ito & Kitagawa, 2005). In another study they did not find an ERN in PD, compared to HCs (Rustamov et al., 2014). Only two studies, namely, Holroyd et al. (2002) and Verleger et al. (2013), found no difference in Ne/ERN amplitude when PD and HCs were compared. Three studies used time–frequency analyses to investigate the brain reaction to errors and found reduced theta oscillatory activity in PD (Carriere et al., 2016; Beste et al., 2017; Singh et al., 2018). Notably, from the studies found by the systematic search, only a few tested PD patients both on and off medication (i.e., during dopaminergic medication and during a period of withdrawal) and found no difference in the ERN/theta amplitude (Stemmer et al., 2007; Willemssen et al., 2008; Volpato et al., 2016; Singh et al., 2018) or a larger ERN when the PD were in the off, compared to the on, state (Seer et al., 2017). **Pe.** The few studies that investigated the Pe found mixed results: reduced amplitude when PD are compared to healthy adults (Ito & Kitagawa, 2005) or no difference between groups (Falkenstein et al., 2005). Tellingly, no study with manipulation of the dopaminergic medication (i.e. by testing medicated versus not medicated patients or PD during dopaminergic medication versus withdrawal) performed analysis on the Pe response. **Other error signatures.** One study used two versions of the go-nogo task (compatible/incompatible) and found greater amplitude and prolonged latency of the N2, compared to HCs (Beste et al., 2009a). This was interpreted as an intensification of premotor inhibition in PD (Beste et al., 2009b). In another study with a Flanker-switching task, PD did not show any N2, as compared to HCs (Rustamov et al., 2014). Studies on dopaminergic medication (patients under medication or in withdrawal) found no difference in N2 amplitude (Willemssen et al., 2011). **Behavioral performance:** PD patients generally show prolonged RT during erroneous responses compared to matched controls; no difference was seen in RTs in PD patients when measured on and off medication. Results concerning accuracy are inconsistent. One study reported reduced PES effect compared to controls (Stemmer et al., 2007), another study reported no pes effect in the PDs (Rustamov et al., 2014) and one other study no difference compared to HCs (Singh et al., 2018). **Summary.** Error monitoring is supported by a complex system characterized by multiple high-level processes,

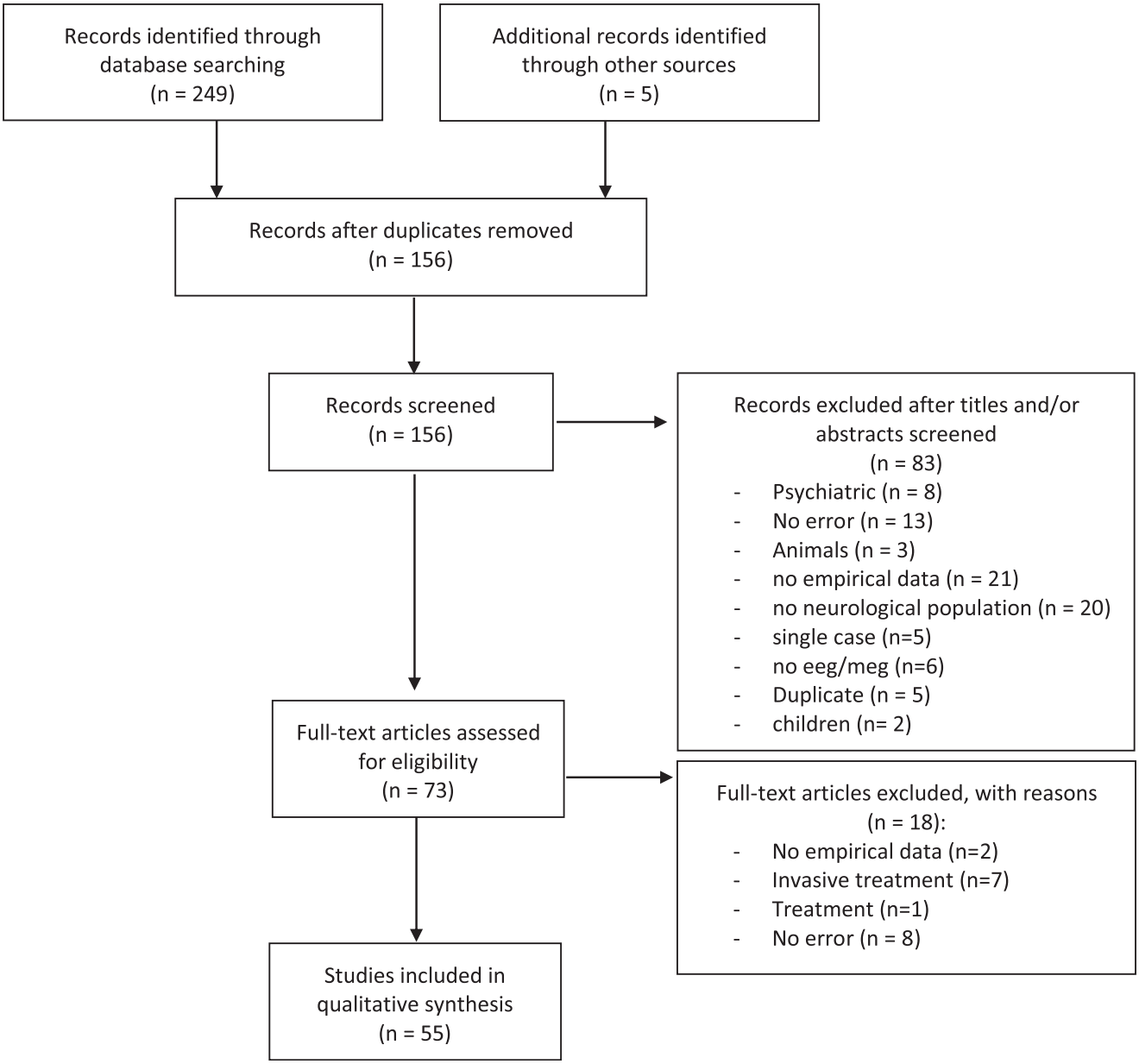

Fig. 1. Prisma Flow Diagram of the retrieved articles evaluated according to the inclusion/exclusion criteria and included in the systematic review.

and Parkinson's disease appears to affect one or more of those processes, while sparing others. From the studies included in the systematic search, it seems reasonable to conclude that mesencephalic dopamine functioning, as expressed in ERN/theta, is altered in PD. Also, the N2 showed an altered pattern compared to healthy controls. Relevant from a clinical point of view, while some studies investigate the effects of dopamine on the ERN/-theta (by manipulating the balance of dopamine), no evidence has been collected on the role of dopaminergic medication on the Pe component. It is worth noting that there is no consistent evidence for altered behavioral expressions of error-related processing in PD patients compared to HCs, or how this might be linked with the altered brain activity.

Huntington's disease. Huntington's Disease (HD; Huntington, 1872) is an autosomal dominant neurological disorder characterized by a reduction in D1 and D2 receptor density (Beste et al., 2006; Walker, 2007). HD patients are mostly affected by rapid, arrhythmic and complex involuntary movements, which are supposed to be an effect of dysfunctional basal ganglia processing (Thompson et al., 1988). The neuroanatomical pathology of the HD is characterized by the accumulation of the protein huntingtin in the striatum, as well as in other cortico-subcortical regions (Walker, 2007). HD is accompanied by cognitive impairment, which comprises also deficit in error-feedback control (Smith et al., 2000).
Studies: ERN/mid-frontal theta. Most of the studies on HD found reduced amplitude of the ERN or reduced

Table 1. Basal ganglia disorders

Parkinson's Disease (PD)

Studies: ERN/mid-frontal theta

Results (Amplitude)	Participants	Task	Behavior	Reference	Extra information
ERN: PD < HC, ERN: PD off = PD premed	17 PD off, 15 PD premed, 17 HC	Eriksen Flanker	RT: PD = HC	Beste et al., 2009b	UPDRS: PD OFF: 15.9 (±5.3)PD premed: 12.6 (±5.4)
ERN: PD < HC	21 PD XDP, 21 HC	Eriksen Flanker	RT: PD > HC	Beste et al., 2017	The PD are patients with XDP UPDRS: PD: 15.17 (±10.6)
ERN: PD < HC	13 PD, 13 HC	Eriksen Flanker	RT: PD > HC	Falkenstein et al., 2001	UPDRS: PD: 25 (range 13-58)
ERN: PD < HC	13 PD, 13 HC	Simon-type	RT: PD > HC	Falkenstein et al., 2001	UPDRS: PD: 25 (range 13-58)
ERN: PD < HC	14 PD, 14 HC	Go/Nogo	RT: PD > HC	Falkenstein et al., 2001	UPDRS: PD: 25 (range 13-58)
ERN: PD < HC, ERN: PD med = PD nonmed	9 PD nonmed, 9 PD med, 14 HC	Eriksen Flanker	RT: PD < HC; acc: PD < HC; pes: PD < HC	Stemmer et al., 2007	UPDRS: nonmed: 22.7 med: 21.3
ERN: PD < HC	17 PD, 15 HC	Lexical decision	RT: PD > HC; acc: PD < HC	Ito & Kitagawa, 2006	
ERN: PD < HC, ERN: PD on = PD off	18 PD, 18 HC	Eriksen Flanker	RT: PD = HC; acc: PD = HC;	Willemssen et al., 2008	UPDRS: ON: 10.8 OFF: 14.8
ERN: PD < HC	14 PD pre-med, 14 HC	Eriksen Flanker	RT: PD > HC; acc: PD = HC;	Willemssen et al., 2009	UPDRS: PD: 12.5± 5.6
ERN: PD < HC, ERN: PD off < HC, ERN: PD on = PD off	10 PD (on and off), 10 HC	Probabilistic learning	acc negative learning: PD off < HC	Volpato et al., 2016	UPDRS: PD: 26.8 ± 15.81
ERN: PD < HC	20 PD on, 20 HC	Eriksen Flanker with switching	RT: PD > HC, PD < HC (shift trials); acc = PD < HC; Pes: PD no effect	Rustamov et al., 2014	UPDRS: PD on: 15.85 (±6.71)
ERN: PD < HC	13 PD on, 13 PD off, 13 HC	Eriksen Flanker	RT: PD = HC; acc: PD > HC, PD on = PD off	Seer et al., 2017	Greater ERP attenuation when PD under medication
ERN: PD = HC	9 PD, 9 HC	Eriksen Flanker	RT: PD = HC; acc: PD = HC	Holroyd et al., 2002	UPDRS: 26.9 ± 3.8
ERN: PD = HC	12 PD, 12 HC	Eriksen Flanker	RT: PD = HC	Verleger et al., 2013	UPDRS: PD: 19.3 (±88.4)
Theta: PD < HC. THETA: PD on = PD off	28 PD, 28 HC	Simon	RT: PD = HC; acc: PD = HC; pes: PD = HC	Singh et al., 2018	UPDRS: ON: 22.1 OFF: 23.8

Studies: Pe

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
Pe: PD = HC	13 PD, 13 HC	Erikesen Flanker	RT: PD > HC	Falkenstein et al., 2005	UPDRS: 25 (range 13-58)
Pe: PD = HC	13 PD, 13 HC	Simon-type	RT: PD > HC	Falkenstein et al., 2005	UPDRS: 25 (range 13-58)
Pe: PD = HC	14 PD, 14 HC	Go/Nogo	RT: PD > HC	Falkenstein et al., 2005	UPDRS: 25 (range 13-58)
Pe: PD = HC	17 PD, 15 HC	Lexical decision	RT: PD > HC; acc: PD < HC	Ito & Kitagawa, 2006	UPDRS: 15.85 (±6.71)

(*continued on next page*)

Table 1 (*continued*)

Studies: other error signatures					
Results (amplitude)	**Participants**	**Task**	**Behavior**	**Reference**	**Extra information**
N2: PD > HC	15 PD on, 15 HC	Go/Nogo (compatible; incompatible)	RT: PD > HC	Beste et al., 2009a	PD: RT (incompatible) > RT (compatible)
N2: PD < HC	20 PD on, 20 HC	Eriksen Flanker with switching	RT: PD > HC, PD < HC (shift trials); acc = PD < HC; Pes: PD no effect	Rustamov et al., 2014	UPDRS: PD on: 15.85 (±6.71)
N2: PD = HC N2: PD on = PD off, N2: PD premed = HC	20 PD on, 32 HC, 15 PD premed	Erikesen Flanker	RT: PD off = PD on	Willemssen et al., 2011	PD were tested on and off. PD premed were tested before and after 8 weeks after medication onset. UPDRS: PD on:10.8 ± 5.6 PD off: 14.8 ± 5.3
Huntington's Disease (HD)					
Studies: ERN/mid-frontal theta					
Results (amplitude)	**Participants**	**Task**	**Behavior**	**Reference**	**Extra information**
ERN: HD < HC	11 HD, 12 HC	Eriksen Flanker	RT: HD > HC; acc: HD = HC	Beste et al., 2006	positive correlation between ERN and CAG-index UHDRS: HD: 24.27 ± 12.47
ERN: HD < HC, ERN: preHD = HC	15 HD, 15 preHD, 15 HC	Eriksen Flanker	RT: HD = HC	Beste et al., 2009b	UPHDRS: HD: 17.1(±7.9) preHD: /
ERN: HD < preHD	21 HD, 12 preHD	Eriksen Flanker	acc: HD = preHD; pes: HD = preHD	Beste et al., 2008	No controls but only HD and preHD are present UHDRS: HD:25.44 (± 9.03) preHD: 0.81(± 1.2)
ERN: HD = HC; Theta: HD = HC	11 HD, 9 HC	Eriksen Flanker	RT: HD = HC; pes: HD = HC	Beste et al., 2007	UHDRS: HD: 0.55 ± 0.52
Studies: Pe					
Results (Amplitude)	**Participants**	**Task**	**Behavior**	**Reference**	**Extra information**
Pe: HD < preHD	21 HD, 12 preHD	Eriksen Flanker	acc: HD = preHD; pes: HD = preHD	Beste et al., 2008	No controls but only HD and preHD are present

Abbreviation: PD: Parkinson's disease; HD: Huntington's disease; preHD: presymptomatic Huntington Disease; med = medicated with dopaminergic medication; non medicated or drug-naïve patients; XDP = X-linked dystonia-parkinsonism; Ne: negativity error; ERN: error-related negativity; Pe: positivity error; RT: response time; acc: accuracy; HC: healthy controls; UPDRS: Unified Parkinson's Disease Rating Scale (only motor scale is reported); UHDRS: Unified Huntington's Disease Rating Scale (only motor scale is reported).

theta power when patients are compared with HCs (Beste et al., 2006, 2008, 2009b). One study found no difference between HD patients and HCs on the ERN amplitude (Beste et al., 2007). **Pe**. Concerning the Pe, only one study has been conducted and no significant difference was found between HD and patients with presymptomatic HD (preHD; Beste et al., 2008). **Behavioral performance:** HD patients show a preserved ability to correctly execute the task and equal RT compared to matched controls. One study found no PES difference between HD and HCs (Beste et al., 2007). **Summary.** Similar to the studies on PD patients, most studies on HD found reduced amplitude of the ERN, which might be related to the dopaminergic dysfunction. In one study on HD no difference was found on Pe amplitude when HD and HCs were compared. Studies of error processing in HD are still scarce, and thus no clear conclusions can be drawn.

Neurodegenerative disease

Neurodegenerative disease is an umbrella term for a range of debilitating conditions, which are characterized by the progressive degeneration of nerve cells that primarily occurs in the later stages of life. The origin of the neurodegeneration can entail a variety of factors, including cell atrophy or shrinkage in subcortical and/or cortical regions. The neurodegenerative diseases are characterized by cognitive impairment, which can affect different domains, including performance monitoring and implementation of cognitive control (Mars et al., 2011). As the disease progresses, cognitive symptoms are worsened and could be associated with lack of awareness of the deficits caused by their pathology (Rosen, 2011). One of the most common neurodegenerative disease is dementia, the most prominent form of which is the Alzheimer's disease (AD), which likely develops after a stage of Mild Cognitive Impairment (MCI). Other important examples of dementia are the fronto-temporal dementia, vascular dementia or Lewy body dementia (Karantzoulis & Galvin, 2011). Note that Parkinson's and Huntington's diseases can also be categorized as neurodegenerative disorders, but in this review they have been included in a separate section on "basal ganglia".

Mild Cognitive Impairment. MCI is an intermediate stage in between normal aging and dementia, in which a gradual impairment of cognitive functions can be observed (Petersen et al., 2014). There are different forms of MCI with the amnestic subtype (aMCI) being the most predictive of progression to AD, in which memory deficits can occur alone or in combination with deficits in other cognitive functions, such as executive deficits and cognitive control. Three studies have been included on MCI patients. **Studies: ERN/mid-frontal theta.** One study analysed the ERN and observed no difference in amplitude compared to matched controls (Thurm et al., 2013). Another study used time–frequency analyses and found reduced theta activity in MCI patients in the Nogo trials during a Go/NoGo task (Nguyen et al., 2017). **Pe.** Concerning the Pe, no study has been collected. **Other error signatures:** One study with a semantic go/nogo found equal amplitude of the N2 when MCI and HCs are compared (Chiang et al., 2018). **Behavioral performance:** From a behavioral point of view, heterogeneous findings have been collected as MCI patients showed reduced or equal performance compared to matched controls. **Summary.** Individuals with aMCI differed from cognitively normal aging controls on behavior (accuracy rates and response time; Table 2). Less pronounced results have been obtained from a neurophysiological point of view. Discrepancies between studies can be explained by differences in the operationalization of the MCI criteria. However, too few studies are available to draw a clear overview on the EEG response of MCI patients on performance and error monitoring tasks.

Alzheimer's disease. AD is a progressive pathology in which not only memory deficits, but also impaired executive function has been described (Perry and Hodges, 1999). In AD patients, pathological changes are in fact prominent in the temporal lobes, but also the frontal and prefrontal areas are affected. Two studies have been included from the systematic search. **Studies: ERN/mid-frontal theta.** In two studies on AD and response-monitoring dysfunction, AD patients showed reduced ERN amplitudes (Mathalon et al., 2003; Ito and Kitagawa, 2005). **Pe.** The Pe was found to be reduced in amplitude among AD patients compared to HCs in one study (Ito & Kitagawa, 2005), but not in another (Mathalon et al., 2003). **Behavioral performance:** AD patients generally show slower RT during erroneous responses and worse or equal accuracy. **Summary.** Results showed that AD patients presented altered error detection response (ERN or Ne), whereas inconsistent results have been observed for the Pe response. Mathalon and colleague (2003) suggested that the ERN and Pe might rely on different and independent systems, which justify the dissociated response to errors. Only two studies have been done, preventing the possibility to draw firm conclusions on error monitoring in AD (see Table 2).

Acquired brain injury

With acquired brain injury, we refer to brain damage that occurs after birth and that can provoke motor or cognitive deficits, with different degree of impairment. In this section, we included those studies that were related to traumatic brain injury (TBI) and brain lesions (prefrontal lesion, thalamic damage and cerebellar lesion). A brain injury or lesion may entail many symptoms that can vary depending on the nature/timing of the injury, the extension and location of the lesion and the treatment received (Chen et al., 2010). Interestingly, patients with neurological disorders are often partially (if not completely) unaware of their deficits (*anosognosia*; Moro, 2013; Canzano et al., 2016; Scandola et al., 2020). With the advent of functional neuroimaging, it was clearer how the complex system of executive abilities actually relies on distributed neural networks that engage the prefrontal cortex, but also the parietal cortex, the cerebellum and subcortical areas (i.e. basal ganglia and thalamus).

Traumatic brain injury. The most frequent causes of traumatic brain injuries (TBI) are falls, motor vehicle crashes, and sport concussions (Langlois et al., 2006). Traumatic impacts on the cranium can lead to neuropathological changes in the brain tissue underlying the skull at the side of the impact or on the opposite side (due to bouncing), with functional neuronal alterations manifesting in a myriad of different patterns (Giza and Hovda, 2001; Aubry et al., 2002). The most common cognitive consequences of TBI (at all levels of severity) are disturbances of attention, memory, and executive dysfunctions. Indeed, individuals with moderate-to-severe TBI show impaired cognitive control processes relative to neurologically healthy controls (HCs; Larson et al., 2012). **Studies: ERN/mid-frontal theta.** Six studies investigated the ERN, observing a reduced amplitude of the ERN (Larson et al., 2007, 2009a; Pontifex et al., 2009; De Beaumont et al., 2013), an increased ERN amplitude (Olson et al., 2018), or no difference (Larson et al., 2012). Three of those mentioned studies were conducted on athletes with multiple concussions. One study (Larson & Perlstein, 2009c) investigate the ERN, showing no difference between correct and incorrect trials in a

Table 2. Neurodegenerative diseases

Mild cognitive impairment (MCI)					
Studies: ERN/mid-frontal theta					
Results (amplitude)	**Participants**	**Task**	**Behavior**	**Reference**	**Extra information**
ERN: MCI = HC	15 MCI, 14 HC	Erikesen Flanker	RT: MCI > HC (incongr.), RT: MCI = HC (congr.)	Thurm et al., 2013	
Theta: MCI < HC (nogo)	22 aMCI, 22 HC	Semantic Go/NoGo	acc: aMCI < HC	Nguyen et al., 2017	
Studies: other error signatures					
Results (amplitude)	**Participants**	**Task**	**Behavior**	**Reference**	**Extra information**
N2: aMCI = HC	25 aMCI, 25 HC	Semantic Go/NoGo	RT: PD > HC	Chiang et al., 2018	
Alzheimer's Disease (AD)					
Studies: ERN/mid-frontal theta					
Results (amplitude)	**Participants**	**Task**	**Behavior**	**Reference**	**Extra information**
ERN: AD < HC	16 AD, 14 HC	Picture-Name verification	RT: AD > HC; acc: AD > HC	Mathalon et al., 2003	
ERN: AD < HC	16 AD, 15 HC	Lexical decision paradigm	RT:AD > HC; acc: AD < HC	Ito & Kitagawa, 2005	
Studies: Pe					
Results (amplitude)	**Participants**	**Task**	**Behavior**	**Reference**	**Extra information**
Pe: AD = HC	16 AD, 14 HC	Picture-Name verification	RT: AD > HC; Acc: AD > HC	Mathalon et al., 2003	
Pe: AD < HC	16 AD, 15 HC	Lexical decision paradigm	RT:AD > HC; acc: AD < HC	Ito & Kitagawa, 2005	

Abbrevation: MCI: Mild Cognitive Impairment; aMCI: amnestic Mild Cognitive Impairment; AD: Alzheimer Disease; HC: healthy controls; ERN: error-related negativity; Pe: error positivity; acc: accuracy; RT: response time.

Stroop task, however no control group was included. **Pe.** The same six studies also performed analyses on the Pe response. All studies reported equal amplitude in comparison to healthy matched adults. Another study (Larson et al., 2009c) also investigate the Pe, showing a difference between correct and incorrect trials in a Stroop task, with incorrect trials eliciting a stronger positivity; however, no control group was included in this report. **Other error signatures.** Two studies investigated the N450 response showing mixed results, as they found no amplitude difference (Larson et al., 2011) or reduced amplitude (Perlstein et al., 2006). Two studies showed reduced amplitude of the SP (Larson et al., 2009b, 2011) and reduced NSW (Perlstein et al., 2006) in TBI, respectively. One study showed greater N2-P3 amplitude in response to threat-related stimuli in a NoGo situation in the TBI group (Maki-Marttunen et al., 2015). **Behavioral performance:** Generally, no significant behavioral differences were reported in comparison to healthy adults, particularly as concern the accuracy rate. Few studies reported altered RTs during error responses on speeded tasks (Larson et al., 2007, 2009a), while others found no RT differences (Pontifex et al., 2009). When emotional threat related stimuli were present, TBI showed an altered behavior with faster RT and worse accuracy (Maki-Marttunen et al., 2015). No difference was found in the PES between TBI patients and HCs (Pontifex et al., 2009). **Summary.** Behaviorally, TBI patients and healthy controls showed similar accuracy rates. From a neurophysiological point of view, EEG studies on error monitoring in TBI focused on the ERP domain, investigating processes which elicited the most common error-related components as ERN/Pe but also other conflict-related ERPs as the N450, NSW and the SP. The included studies consistently reported no differences in the Pe response, but did in most cases observe reduced ERN response. Decreased amplitudes were also found in other conflict-related ERPs (N450, NSW, SP), whereas increased amplitude has been found in the N2-P3 when related to emotional stimuli. However too few studies have been conducted on these potentials (see Table 3).

Prefrontal lesions. Some of the most common effects of damage to the frontal lobe include decision making deficits, impairment of insight and judgement, and executive dysfunction, which can have a profound effect on many aspects of everyday life. A growing literature indicates that the activation of the ACC, insula and surrounding medial prefrontal regions (mPFC) are

Table 3. Acquired brain injury

Traumatic Brain Injury (TBI)

Studies: ERN/mid-frontal theta

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
ERN: TBI < HC	14 TBI, 14 HC	N2pc	Acc: TBI = HC	De Beaumont et al. (2013)	The TBI group comprises athletes (sport multi-concussion)
ERN: TBI < HC	18 TBI, 21 HC	SPCN	Acc: TBI = HC	De Beaumont et al. (2013)	The TBI group comprises athletes (sport multi-concussion)
ERN: TBI < HC	19 TBI, 21 HC	Modified Stroop	RT: TBI > HC; acc: TBI= = ACC	Larson et al., 2007	GCS < 9
ERN: TBI < HC	20 TBI, 20 HC	Stroop	RT: TBI < HC; acc: TBI = HC	Larson et al., 2009a	GCS < 9
ERN: TBI < HC	30 TBI, 36 HC	Eriksen Flanker	RT: TBI = HC, TBI > HC (interference effect); acc: TBI < HC; pes: TBI = HC	Pontifex et al., 2009	
ERN: TBI = HC	36 TBI, 46 HC	Modified Stroop	RT: TBI = HC; acc: TBI = HC	Larson et al., 2012	GCS range 13-15
ERN: TBI > HC	25 TBI, 22 HC	Eriksen Flanker	Pes: TBI = HC	Olson et al., 2018	The TBI group comprises athletes (sport concussion)
ERN: TBI: no ern	16 TBI	Stroop	RT: incongr > congr; acc: incongr < congr	Larson et al., 2009c	GCS range 3-8. There is no control group

Studies: Pe

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
Pe: TBI = HC	14 TBI, 14 HC	N2pc	Acc: TBI = HC	De Beaumont et al. (2013)	The TBI group comprises athletes (sport multi-concussion)
Pe: TBI = HC	18 TBI, 21 HC	SPCN	Acc: TBI = HC	De Beaumont et al. (2013)	The TBI group comprises athletes (sport multi-concussion)
Pe: TBI = HC	19 TBI, 21 HC	Modified Stroop	RT: TBI > HC; acc: TBI = ACC	Larson et al., 2007	GCS < 9
Pe: TBI = HC	20 TBI, 20 HC	Stroop	RT: TBI < HC; acc: TBI = HC	Larson et al., 2009a	GCS < 9
Pe: TBI = HC	30 TBI, 36 HC	Eriksen Flanker	RT: TBI = HC, TBI > HC (interference effect); acc: TBI < HC; pes: TBI = HC	Pontifex et al., 2009	
Pe: TBI = HC	36 TBI, 46 HC	Modified Stroop	RT: TBI = HC; acc: TBI = HC	Larson et al., 2012	GCS range 13-15
Pe: TBI = HC	25 TBI, 22 HC	Eriksen Flanker	Pes: TBI = HC	Olson et al., 2018	The TBI group comprises athletes (sport concussion)
Pe: TBI: Pe effect (inc > cor)	16 TBI	Stroop	RT: incongr > congr; acc: incongr < congr	Larson et al., 2009c	GCS range 3-8. There is no control group

Studies: other error signatures

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
N450: TBI = HC	29 TBI, 36 HC	Modified Stroop	RT: TBI = HC; acc: TBI = HC	Larson et al., 2011	GCS range 13-15
N450: TBI = HC	18 TBI, 21 HC	Modified Stroop	RT: TBI = HC; acc: TBI = HC	Larson et al., 2009b	GCS < 9
N450: TBI < HC	11 TBI, 11 HC	Cued Stroop and Reading Span	RT: TBI = HC (Stroop interference); acc: TBI < HC (Read Span)	Perlstein et al., 2006	GCS < 9
SP: TBI < HC	18 TBI, 21 HC	Modified Stroop	RT: TBI = HC; acc: TBI = HC	Larson et al., 2009b	GCS < 9
SP: TBI < HC	29 TBI, 36 HC	Modified Stroop	RT: TBI = HC; acc: TBI = HC	Larson et al., 2011	GCS range 13-15
NSW: TBI < HC	11 TBI, 11 HC	Cued Stroop and Reading Span	RT: TBI = HC (Stroop interference); acc: TBI < HC (Read Span)	Perlstein et al., 2006	GCS < 9

(*continued on next page*)

Table 3 (*continued*)

Studies: other error signatures

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
N2-P3: TBI > HC	27 TBI, 17 HC	Go/Nogo	RT: TBI > HC (emotional relevant); acc: TBI = HC;	Maki-Marttunen et al., 2015	GCS range 13-15. The control group consisted of patients with previous ankle injury

Prefrontal lesions

Studies: ERN/mid-frontal theta

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
ERN: PFC = HC	6 PFC, 10 HC	Letter discrimination	RT: PFC > HC; acc: PFC = HC; pes: PFC as HC	Ghering et al., 2000	The PFC group showed no difference between correct and incorrect trials
ERN: PFC inc > Cor (Naming; no comparison to HC)	6 PFC, 12 HC	Naming (linguistic) and Simon (non-linguistic)	RT: PFC = HC; acc: PFC < HC	Riès et al., 2013	HC did not have enough errors to elicit ERPs, so there is no comparison between PFC and HC
ERN: MPFC = HC	2 MPFC, 21 HC	Visual Stop-signal	Acc: MPFC = HC	Lovstad et al., 2012	
ERN: LFC < HC; ERN: bOFC = HC; ERN: uTC = HC	7 LFC, 6 bOFC, 6 uTC, 9 HC	Eriksen Flanker	RT: LFC > HC, bOFC = HC; acc: LFC = HC, bOFC = HC	Ullsperger et al., 2002	
ERN: BDC = HC; ERN: rACC < HC; ERN: rACC < BDC	7 rACC, 7 BDC, 7 HC	Eriksen Flanker	RT: HC < rACC (congr/incongr); HC = BDC (congr/incongr); pes: Error awareness (behavioral): HC < rACC/BDC; No pes in rACC	Maier et al., 2015	rACC: lesions centred on the rACC and LFC; BDC: brain damaged control group of patients with lesions outside of the rACC and LFC
ERN: 3 ACC: no ERN, 1 ACC: only in one paradigm, 1 ACC: only in one paradigm	5 ACC 11 HC	Eriksen Flanker	RT: ACC > HC; acc: ACC = HC; pes: 4 ACC had pes effect (2 for each paradigm)	Stemmer et al., 2004	The task was characterized by letter and geometric stimuli in two different paradigms
ERN: OFC < HC	4 OFC, 8 HC	Stroop	RT: OFC > HC; acc: OFC = HC	Turken & Swick, 2008	
ERN: LFC < HC,	7 LFC, 7 HC	Eriksen Flanker	RT: LFC > HC, acc: LFC = HC	Ullsperger et al., 2006	
ERN: BG < HC	9 BG, 9 HC	Eriksen Flanker	RT: BG > HC; acc: BG = HC	Ullsperger et al., 2006	
ERN: PCMN < HC	8 PCMN, 8 HC	Eriksen Flanker and novelty	Acc: PCMN < HC; pes: PCMN < HC	Wessel et al., 2014	

Studies: Pe

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
Pe: HC = BDC = rACC	7 rACC, 7 BDC, 7 HC	Eriksen Flanker	RT: HC < rACC (congr/incongr), HC = BDC (congr/incongr); pes: No pes in rACC	Maier et al., 2015	Error awareness: HC < rACC/BDC
Pe: OFC > HC	12 OFC, 14 HC	Visual stop signal	RT: OFC = HC; pes: OFC = HC	Solbakk et al., 2014	
Pe: ACC → only in one paradigm	5 ACC, 11 HC	Eriksen Flanker	RT: ACC > HC; acc: ACC = HC; pes: 4 ACC had pes effect (2 for each paradigm)	Stemmer et al., 2004	The task was characterized by letter and geometric stimuli in two different paradigms
Pe: LFC < HC; Pe: bOFC = HC; Pe: uTC = HC	7 LFC, 6 bOFC, 6 uTC, 9 HC	Eriksen Flanker	RT: LFC > HC, bOFC = HC; acc: LFC = HC, bOFC = HC	Ullsperger et al., 2002	
Pe: BG < HC	9 BG, 9 HC	Eriksen	RT: BG > HC; acc:	Ullsperger et al.,	

Table 3 (*continued*)

Studies: Pe

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
Pe: LFC < HC	7 LFC, 7 HC	Flanker Eriksen Flanker	BG = HC RT: LFC > HC; acc: LFC = HC	2006 Ullsperger et al., 2006	

Studies: other error signatures

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
N2: OFC < HC	12 OFC, 14 HC	Visual stop signal	RT: OFC = HC; pes: OFC = HC	Solbakk et al., 2014	
N2P3: OFC = HC (nogo); N2P3: OFC < HC (threat-related stimuli)	12 OFC, 12 HC	Executive RT	RT: OFC = HC; acc: OFC < HC	Kuusinen et al., 2018	
N2P3: OFC = HC (task irrelevant stimuli); N2P3: OFC > HC (threat-related stimuli)	13 OFC, 11 HC	Eriksen Flanker	RT: OBC = HC; acc: OFC = HC; OFC > HC (emotional irrelevant)	Maki-Marttunen et al., 2017	The control group consisted of patients with previous ankle injury, but no head injury

Cerebellar lesion (CL)

Studies: ERN/mid-frontal theta

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
ERN: CL < HC	8 CL, 11 HC	Antisaccade	RT: CL > HC; acc: CL = HC	Peterburs et al., 2012	
ERN: CL < HC	16 CL, 16 HC	Antisaccade	Acc: CL < HC	Peterburs et al., 2015	

Studies: Pe

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
Pe: CL = HC	8 CL, 11 HC	Antisaccade	RT: CL > HC; acc: CL = HC	Peterburs et al., 2012	
Pe: CL = HC	16 CL, 16 HC	Antisaccade	Acc: CL < HC	Peterburs et al., 2015	

Thalamic lesion (TL)

Studies: ERN/mid-frontal theta

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
ERN: TL < HC	6 TL, 28 HC	Antisaccade	RT: TL > HC; acc: TL < HC; pes: TL no effect	Peterburs et al., 2011	One out of the six patient had a strong pes effect
ERN: TL < HC	15 TL, 16 HC	Eriksen Flanker	RT: TL = HC; acc: TL = HC; pes: TL no effect	Seifert et al., 2011	

Studies: Pe

Results (amplitude)	Participants	Task	Behavior	Reference	Extra information
Pe: TL = HC	15 TL, 16 HC	Eriksen Flanker	RT: TL = HC; acc: TL = HC; pes: TL no pes effect	Seifert et al., 2011	

Abbreviation: OFC: orbitofrontal cortex; rACC: rostral anterior cingulate cortex; ACC: anterior cingulate cortex; LFC: lateral prefrontal cortex; bOFC: bilateral orbitofrontal cortex; BDC: uTC: unilateral temporal cortex, HC: healthy controls; BDC: brain damage control group; BG: basal ganglia; PCMN: prefrontal-cingulate monitoring network; TBI: traumatic brain injury; ERN: error-related negativity; Pe: error positivity; pes: post-error slowing; GCS: Glasgow Come Scale; CL: cerebellar lesion; TL: thalamic lesion.

Table 4. White matter diseases

White matter lesions					
Studies: ERN/mid-frontal theta					
Results (amplitude)	**Participants**	**Task**	**Behavior**	**Reference**	**Extra information**
ERN: SCD-FL < SCD-C, ERN: SCD-FL < HC	11 SCD-FL, 11 SCD-C, 11 HC	Fast choice	RT: SCD = HC; acc: SCD = HC	Hogan et al., 2006	
Studies: other error-signatures					
Results (amplitude)	**Participants**	**Task**	**Behavior**	**Reference**	**Extra information**
BETA: FSL < HC	12 FSL, 12 HC	Hybrid Eriksen Flanker-novelty	acc: FSL = HC; pes: FSL no effect	Wessel et al., 2016	
Multiple Sclerosis (MS)					
Studies: ERN/mid-frontal theta					
Results (Amplitude)	Participants	**Task**	**Behavior**	**Reference**	**Extra information**
ERN: MS > HC	27 MS, 31 HC	Stop signal	RT: MS = HC; acc: MS = HC	Lopez-Gongora et al., 2015	ERN amplitude correlated positively with scores on the EDSS and the MSSS

Abbreviation: MS: multiple sclerosis; HC: healthy controls; SCD-FL: frontal lobe sickle cell disease; SCD-C: sickle cell disease without infarcts; ERN: error-related negativity; pes: post-error slowing; acc: accuracy; RT: response time; FSL: fronto-striatal lesion; EDSS: Expanded Disability Status Scale; MSSS: Multiple Sclerosis Severity Score.

crucial during performance monitoring and cognitive control, to internally guide behavior in accordance with goals, plans and broader contextual knowledge (Van Noordt et al., 2012; Wessel, 2016). Eleven articles have been included in this section according to the systematic search. **Studies: ERN/mid-frontal theta.** One study with patients with prefrontal cortex (PFC) lesions showed no difference on the ERN amplitude compared to HCs, but they also did not show difference between correct and incorrect responses, differently than HCs (Gehring & Knight, 2000). In another study PFC patients showed preserved ERN in a naming but not in a non-naming task, when comparing correct versus incorrect trials; however, in this case no comparison with controls have been done (Riès et al., 2013). Two studies with orbitofrontal cortex (OFC) patients found an alteration of the ERN response, such that OFC patients had reduced amplitude (Turken & Swick, 2008; Solbakk et al., 2014). Previously, Ullsperger and colleagues (2002) performed a study with three different groups of patients with prefrontal lesions: patients with lesions in bilateral OFC (bOFC), lateral frontal cortex (LFC), and unilateral temporal cortex (uTC). The bOFC and uTC patients had no difference in the ERN response as compared to HCs, whereas LFC patients showed reduced ERN amplitudes. Similar results on LFC patients, namely reduced ERN amplitude had been reported also in a later work (Ullsperger and von Cramon, 2006). In this same study, also patients with basal ganglia (BG) lesion showed reduced ERN amplitude (Ullsperger and von Cramon, 2006). A study with 2 patients presenting mPFC lesions showed preserved ERNs (Lovstad et al., 2012). Patients with rostral ACC (rACC) lesions showed decreased ERN amplitudes when compared to HCs and control patients with brain damage outside the target regions, whereas the HCs and the control patients did not differ in ERN amplitude (Maier et al., 2015). Another study with 5 patients with lesions in the rACC showed that 3 of them did not produce an ERN, while the other 2 patients did (Stemmer et al., 2004, Table 3). Finally, a study on patients with lesions in the prefrontal-cingulate monitoring network (PCMN) showed decreased ERN amplitudes (Wessel et al., 2014). **Pe.** The study with OFC patients found greater Pe amplitudes compared to HCs (Solbakk et al., 2014). Both bOFC and uTC patients showed no difference in Pe amplitude compared to HCs, whereas LFC patients showed reduced Pe amplitude (Ullsperger et al., 2002). The latter finding was confirmed also in a later study (Ullsperger and von Cramon, 2006). In this same study, also patients with basal ganglia (BG) lesion showed reduced Pe amplitude (Ullsperger and von Cramon, 2006). Two out of five patients with lesions in rACC failed to show a Pe response (Stemmer et al., 2004). **Other error signatures.** Two studies with OFC patients used a task that elicited the N2-P3 complex, and found that patients showed no difference in amplitude compared to HCs but showed a modulation when exposed to emotional stimuli (Kuusinen et al., 2018; Maki-Marttunen et al., 2017). **Behavioral performance:** PFC patients showed prolonged RTs during error responses but no difference in performance accuracy (Gehring & Knight, 2000). OFC patients did not show significant differences with HCs, except for increased RTs during a stop-signal task (Solbakk et al., 2014). In the

study by Ullsperger et al. (2002), the bOFC patients had no difference in behavior, as compared to HCs, whereas LFC patients showed increased RT, but comparable accuracy rate. The latter result was later replicated by Ullsperger et al. (2006). Patients reporting lesions in the PCMN showed impaired behavioral performance, and a reduced PES effect compared to HCs (Wessel et al., 2014). **Summary.** Some of the studies that involved patients with lesion damage in certain regions of the frontal cortex reported the disappearance of the ERN/Pe (i.e. LFC, bOFC, ACC, BG, PCMN). Differently, studies who considered patients with brain damage in the mFLC and uTC showed preserved error-related response. Some other studies found a dissociated behavior in the ERN and Pe response (i.e. OFC patients, Solbakk et al., 2014), resembling the patterns typically observed among healthy adults (Nieuwenhuis, et al., 2001).

Cerebellar lesion: Cerebellar activity has been linked to ongoing motor behavior, incoming sensory information as well as to predictive motor control (Ide & Li, 2011; Peterbus et al., 2012). The role of the cerebellum therefore may have implications for the cognitive aspects of performance monitoring, including error processing. Previous studies underlined the close connection of the cerebellum with the thalamus and the supplementary motor area (SMA), in the process of error and post-error processing (Ide et al., 2011). Two studies have been included according to our search algorithm. **Studies: ERN/mid-frontal theta.** Two studies using an antisaccade task found that cerebellar patients showed reduced ERN amplitudes (Peterburs et al., 2012, 2015). **Pe.** These same two studies reported a preserved Pe response (Peterburs et al., 2012, 2015). **Behavioral performance:** cerebellar patients showed either equal or decreased accuracy compared to HCs in the error-monitoring tasks, but no other deviant patterns. **Summary**. Patients with lesions of different extent in the cerebellum showed reduced ERNs but preserved Pe's. However, the available number of studies is too limited to allow clear conclusions.

Thalamic lesion: Using diffusion-based tractography, studies found that among the thalamic nuclei, the ventral anterior and ventral lateral anterior nuclei have a strong connectivity with the ACC. Patients with lesions in that area show impaired error monitoring abilities, suggesting how the thalamic areas are important constituents of the performance monitoring network, anatomically and functionally closely interacting with the ACC (Klein et al., 2013; Ullsperger et al., 2014a). Two studies have been included. **Studies: ERN/mid-frontal theta:** Two studies with thalamic patients found reduced ERN amplitudes in the antisaccade task (Peterburs et al., 2011) and the Eriksen flanker task (Seifert et al., 2011). **Pe.** Seifert et al. (2011) found a preserved Pe among thalamic patients. Interestingly, the analysis by Klein and colleagues (2013) of previously unpublished data (Seifert et al., 2011) found a double dissociation of ERN and Pe responses. In specific, thalamic lesions affecting the ventral anterior and ventrolateral anterior nuclei abolished the ERN but only slightly reduced the Pe, whereas lesions affecting the mediodorsal nucleus led to the opposite pattern (namely reduction of ERN and abolished Pe). **Behavioral performance:** In the study with the antisaccade task, thalamic patients showed impaired behavioral performance and no PES effect, beside one patient who showed strong slowing (Peterburs et al., 2011). In the study using the flanker task, no differences were observed in either RT, accuracy, or PES (Seifert et al., 2011). **Summary:** patients with lesions of in the thalamic nuclei suggest an altered error-monitoring processes.

White matter diseases

Beside the crucial role of grey-matter integrity for the correct functioning of the performance monitoring processes, white matter may also play a fundamental role in the short- as well as long-distance communication between the areas that comprise the error monitoring system (Laughlin & Sejnowaki, 2003). Studies on patients with white-matter disruptions can be helpful in investigating whether structural disconnections within and across networks impairs the neural processes involved in error monitoring (Mierzwa et al., 2015). Here we included studies on white-matter disruption and studies on multiple sclerosis (MS; see table 4).

White-matter lesions: Disconnections or white matter changes can lead to impaired long-range neural communication, with direct consequences also for performance monitoring abilities. **Studies: ERN/mid-frontal theta.** In one study, patients with frontal white-matter lesions due to sickle cell disease (SCD) vasculopathy (SCD-FL) showed reduced ERN amplitudes in comparison to both a group of patients with SCD but without brain lesion and a group of HCs (Hogan et al., 2006). **Pe:** no studies have been performend on the Pe. **Other error signatures.** A study with patients with fronto-striatal white-matter lesions performing a flanker-novelty task revealed decreased beta power following errors compared to HCs (Wessel et al., 2016). **Behavioural performance:** The behavioral performance of the SCD-FL patients showed no difference in RT and accuracy with HCs (Hogan et al., 2006). Patients with fronto-striatal white-matter lesions also showed no difference in performance compared with HCs. However, a PES effect was found in HC but not in PD (Wessel et al., 2016). It is also worth noting that Wessel and colleagues (2016) found a significant group difference for the post-novelty slowing (PNS), thus the slowing of reaction times in subsequent trials after novel stimuli that was found in the HCs in comparison to the patients. **Summary:** studies with white-matter disruption suggest alteration in the error-monitoring signatures but no difference in performance.

Multiple sclerosis: MS is a chronic demyelinating disease of the central nervous system that produces cognitive, motor and neuropsychiatric deficits (Chiaravalloti & De Luca, 2008). Studies suggest that MS patients might develop compensatory mechanisms potentially involving enhanced performance monitoring. **Studies: ERN/mid-frontal theta.** One study with 27 MS patients showed increased ERN amplitudes as compared to HCs during a stop-signal task (Lopez-Gongora et al. 2015). **Pe:** no studies have been done on the Pe. **Beha-**

viioural performance: MS showed similar accuracy and response times than HCs (Lopez-Gongora et al. 2015). **Summary:** patients with MS showed alteration in error monitoring system, but only one study has been done so far on this pathology.

DISCUSSION

The present systematic review aimed at investigating error processing in a number of neurological alterations. Results showed several findings that may expand our current knowledge of error monitoring in neurological disorders (Klein et al., 2013; Ullsperger, 2006).

First, we found results in support of the notion that different and likely independent processes are part of the error monitoring system. In some cases, a decreased ERN/N2 response is not followed by an altered Pe response (and *vice versa*), similar to results on young adults (Nieuwenhuis et al., 2001; Overbeek et al., 2005; di Gregorio et al., 2016; Masina et al., 2019). Also, pharmacological studies in animals and humans (e.g. affected by Parkinson's Disease) suggest that ERN and mid-frontal theta rhythms are influenced by the balance of dopamine in the system, while the results concerning the Pe component are less clear-cut (Falkenstein et al., 2001; Holroyd et al., 2002; Ito & Kitagawa, 2005; Stemmer et al., 2007; Willemssen et al., 2008, 2009; Jocham and Ullsperger, 2009; Beste et al., 2009a, 2009b; Verleger et al., 2013; Cavanagh and Frank, 2014; Seer et al., 2016, 2017).

Second, we observed that the ERN amplitude was reduced in patients with PD, HD, AD, TBI, lesions in LFC, ACC, PCMN, cerebellum, thalamus and in white matter. The ERN did not appear affected in patients with MCI, lesions to mFLC or TC. The ERN was found to be increased in MS, although only a single study was available here. Pe amplitude appeared reduced in HD patients, but in no other patient group (note that no studies on the Pe have been found in MCI or patients with white matter diseases). Theta activity was found reduced in basal ganglia (PD and HD) and neurodegenerative disorders (MCI), but only few studies have been presented. Only one study investigated error-related beta oscillations in patients with white matter lesions, showing reduced power compared to HCs. On the other error-related signatures we found mixed results. Behavioral patterns of dysfunctional (post-)error processing were quite unsystematic, although by and large no massive deficiencies were observed.

Third, in several studies on patients, particularly including deficits in the basal ganglia, LFC, as well as TBI patients, there is an apparent paradox: most of the patients still had a preserved behavioral ability to detect errors or to correct them; however, this did not correspond to the altered brain response found in several studies. So, what does this tell us? Could it be that error-related signatures are an epiphenomenon of the error processing mechanism? The issue of different results at the behavioral and neural levels was raised also by Ullsperger, 2006. It is of course possible that the ERN or mid-frontal activity just are not sensitive enough to pick up on the processing of prediction errors in these cases. However, this is not entirely plausible given the many positive instances of picking up on these signals and revealing disease-related impairments. Instead, there may be various roads to post-error adaptation. It is possible, for instance, that slower and more strategic processes take over if the fast error-monitoring route is compromised. It is worth noting here that the ability of healthy individuals to correct errors in behaviour do correlate with 'error-related' brain responses (Gehring et al., 1993; Ridderinkhof et al., 2003); possibly, this correlation is weakened in some neurological patients. These considerations notwithstanding, the observed patterns of deficiencies seem to suggest that mid-frontal theta activity and especially the ERN may constitute highly sensitive indicators of the integrity of the performance-monitoring network. Also, different brain processes might be recruited in people affected by neurological disorders, in order to perform the task successfully.

A *fourth* point is that studies on neurological patients support the idea that the error-monitoring system goes well beyond the activity of the ACC, and mostly rely on distributed networks, including regions as PFC, LFC, OFC, TC, but also the cerebellum and subcortical regions such as the BG and thalamic regions (Stemmer et al., 2003; Swick and Turken, 2002; Ullsperger et al., 2014a, 2014b). Our systematic review also demonstrates how impairments in short- and long-distance communication across white-matter connections between the nodes in the network may produce deficits in the performance monitoring system (Hogan et al., 2006; Lopez-Gongora et al., 2015; Wessel et al., 2016). Krigolson and Holroyd (2006) proposed a hierarchical vision of the error processing in the human brain, according to which different kinds of errors might activate more frontal or posterior regions of the network. It should also be noted that different types of errors are associated with different EEG dynamics (e.g., Maier et al., 2008), with for instance impulsive errors being associated with rapid midfrontal theta responses whereas attentional lapses are associated with slower parietal alpha responses (van Driel et al., 2012).

Fifth, the lack of consistent findings among patients affected by the same pathology could be due to a number of different reasons, including: (i) the variety of tasks employed across studies and their level of difficulty (go/nogo, flanker, stroop, error awareness, antisaccade task etc.); (ii) the low sample size (Wessel, 2012; Brush et al., 2018); (iii) the heterogeneity of the sample (particularly in studies on brain lesions) and the severity of the illness; (iv) the study design; (v) the task instructions (i.e. asking participants to be more accurate or faster during the task performance). Also, relative to the first point (i), we should be cautious in comparing results between studies not only within the same pathology but also between different neurological populations. In fact, while the EEG marker elicited by the task could be the same (e.g., ERN, N2, Pe), the paradigm behind it could be characterized by relevant differences (in terms of context or task difficulty). For example, while studies on basal ganglia patients mostly used the Flanker tasks to elicit error monitoring signals, studies on neurodegenera-

tive disorders preferably used other kind of paradigms (e.g., go/nogo or lexical decision paradigms).

Sixth, and in relation to the previous point, the fact that most of the neurological disorders included in the systematic search count a limited number of studies, often with only a few patients, limits the conclusions that can be drawn. The points discussed so far thus have to be interpreted with some caution; future studies are needed to characterize in details alterations of error monitoring systems as consequence of neurological disorders.

FUTURE DIRECTIONS

Further studies on the complex neurofunctional architecture of the error monitoring alterations in neurological disorders have to be performed along several directions.

First of all, informative data can be collected not only by traditional tasks on error execution, but also by studies on error observation, eliminating task performance differences. Also new technologies like immersive virtual reality may help to investigate the error-monitoring processes also in patients with motor disabilities. Paradigms in which EEG activity is recorded while patients observe errors performed by their embodied avatar, seem promising. Immersive virtual reality scenarios can indeed generate the feeling of sensory feedback related to an individual's virtual body and thus induce effects on the individual's cognition (Fusaro et al., 2019). However, to the best of our knowledge, immersive virtual reality studies of error monitoring have been done only in healthy adults (Padrao et al., 2016; Pavone et al., 2016; Pezzetta et al., 2018; Spinelli et al., 2018; Tieri et al., 2018), while patients have been tested with speed-response tasks. We suggest that error-related behavioral and brain functions should be tested in ecological contexts (Ozkan and Pezzetta, 2018). Such an approach is in principle safe as studies show that older adults accept well immersive virtual reality, exhibit a positive attitude toward virtual reality and do not experience cybersickness (Huygelier et al., 2019; Appel et al., 2020). We also note that tasks in which patients have to execute semi-virtual and goal-oriented motor tasks in order to probe forward model predictions during motor learning (e.g. throwing a ball, grasping an object) can provide more naturalistic environments that engage different brain regions and require natural behavioral adaptations (Maurer et al., 2015, 2019; Joch et al., 2017; Era et al., 2020; Moreau et al., 2020b; Villa et al., 2020). Indeed, patients with parietal damage show that the ability to understand an observed action is impaired, as well as the ability to engage in more complex object-directed actions (Fontana et al., 2012); thus, it would be interesting to understand whether and how those alterations have an impact on different levels of error processing. Moreover, creating more ecological scenarios could thus help investigate different kinds of error, which might require distinct brain networks (Krigolson and Holroyd, 2006; van Driel et al., 2012).

Another direction that can be relevant for clinical purposes, it is the investigation of the relation between error awareness and interoceptive system in neurological disorders, as a proper understanding of self-monitoring deficits is crucial in rehabilitation circumstances (see review on the role of insula in error monitoring; Klein et al., 2013). In this context, metacognitive abilities allow to modify behavioural performance even in the absence of an external feedback (Wokke et al., 2020); thus, a deeper understanding of the relation of error signatures with interoceptive and metacognitive abilities could highlight deficits in monitoring and decision confidence (Gleichgerrcht et al., 2010; Hoerold et al., 2013; Fleming et al., 2014; Boldt and Yeung, 2015; Tan et al., 2019; Tan et al., 2019; Yeung and Summerfield, 2012; Yeung and Summerfield, 2012). In relation to previous data on thalamic patients have also shown a double dissociation for error responses in thalamic subregions (Klein et al., 2013). It has been suggested that a basal-ganglia-thalamocingulate circuit could be involved in the ERN generation, whereas the mediodorsal nuclei are related to to the Pe generation. Since these findings have been collected on a small sample, future studies on thalamic patients should confirm these results.

Concerning these issues, more data are needed on the assumption that the Pe might or might not rely on the dopaminergic system and significant results can be obtained from pharmacological studies or from patients with basal ganglia diseases (tested during dopaminergic medication (on) and dopaminergic withdrawal (off)). As shown by our systematic review, the evidence collected so far on the role of dopamine in PD is scarce. The few studies that focused on the ERN/mid-frontal theta report no difference between on or off state (Stemmer et al., 2007; Willemssen et al., 2008; Volpato et al., 2016; Singh et al., 2018) or enhanced ERN in off compared to on state (Seer et al., 2017). Also, no evidence about the influence of dopamine on the Pe component is available. To explain the results concerning the ERN/theta findings, we might hypothesize that – in addition to the abovementioned limits that often characterize patient studies (e.g., small sample sizes), residuals dopaminergic effects of medication could be still present when PD are tested during off state, making more difficult to detect differences between medication states. Also, Seer and colleagues suggested that their interesting but unexpected results could have been due to a slightly unbalanced design (Seer et al., 2017). Indeed, various theories and findings point in the direction that ERN and mid-frontal theta depend on dopamine inputs, as dopamine neurons seem to transmit signals at the occurrence of salient events (Schultz, 1997; Jocham and Ullsperger, 2009; Cavanagh and Frank, 2014). In contrast, Pe seems not to depend on dopaminergic inputs. It is also important to consider that other neurotransmitters might play a role in the performance monitoring processes, either through direct modulation of the ACC or through influence on the DA system (Jocham & Ullsperger, 2009; Ullsperger et al., 2014b). Further, a previous study on a single case showed that if the generator of the error-related potential (i.e. ERN) was damaged by unilateral lesion, the ampli-

tude of the ERN was reduced, while the amplitude of the conflict-related potential (i.e. N450) increased (Swick and Turken, 2002). Studies on the hemispheric difference of the error monitoring and on the connectivity between several areas in the healthy and lesioned brain (Ambrosini and Vallesi, 2016; Campanella et al., 2016) will expand the current knowledge on error- and conflict-related signatures.

Finally, from a methodological point of view, most of the EEG analyses on neurological patients have been limited to the ERPs domain. Important, novel information can be obtained through analyses of the oscillatory activity, including that concerning non-phasic alterations in response to erroneous events. In fact, potentially task-relevant dynamics may be lost during ERP averaging. Time-frequency decomposition and network analysis can instead provide insights into neurocognitive processes that go beyond what we learn from the ERPs (Cohen, 2011).

The combination of advanced technological and methodological approaches, including larger sample sizes and thus adequate power, could increase the reliability of the theoretical considerations drawn from the clinical data (Suresh and Chandrashekara, 2012). In this direction, several datasets with similar design can be aggregated applying Bayesian statistics in order to achieve reliable results.

This review represents a systematic appraisal of error-monitoring studies in neurological disorders. In particular, it aims to assess if and how the pathological changes that characterize these conditions negatively affect performance monitoring, at both the behavioural and electrophysiological levels. We observed that the ERN amplitude was reduced in most neurological patient groups, with the exception of patients with MCI or with lesions to mFLC or TC. We also found reduced theta activity in some populations, but less consistent and numerous findings are present on this, as well as on other error-related signatures. Pe amplitude appeared not to be affected in any patient group except HD. Consistent behavioral patterns of dysfunctional (post-) error processing were not observed. Future studies are needed to expand our current theoretical and translational knowledge concerning the human proneness to errors and its adaptive function.

FUNDINGS

R.P. was funded by IBRO PERC InEurope Short Stay grants program. S.M.A. is funded by the PRIN grant (Italian Ministry of University and Research, Progetti di Ricerca di Rilevante Interesse Nazionale, Edit. 2017, Prot. 2017N7WCLP). The authors declare no conflict of interests.

REFERENCES

Aglioti SM, Cesari P, Romani M, Urgesi C (2008) Action anticipation and motor resonance in elite basketball players. Nat Neurosci 11 (9):1109.

Alexander GE, DeLong MR, Strick PL (1986) Parallel organization of functionally segregated circuits linking basal ganglia and cortex. Annu Rev Neurosci 9(1):357–381.

Alexander WH, Brown JW (2011) Medial prefrontal cortex as an action-outcome predictor. Nat Neurosci 14(10):1338–1344. https://doi.org/10.1038/nn.2921.

Ambrosini E, Vallesi A (2016) Asymmetry in prefrontal resting-state EEG spectral power underlies individual differences in phasic and sustained cognitive control. Neuroimage 124:843–857.

Angelucci F, Peppe A, Carlesimo GA, Serafini F, Zabberoni S, Barban F, Costa A (2015) A pilot study on the effect of cognitive training on BDNF serum levels in individuals with Parkinson's disease. Front Hum Neurosci 9:130.

Appel L, Appel E, Bogler O, Wiseman M, Cohen L, Ein N, Campos JL (2020) Older adults with cognitive and/or physical impairments can benefit from immersive virtual reality experiences: a feasibility study. Front Med 6:329.

Arrighi P, Bonfiglio L, Minichilli F, Cantore N, Carboncini MC, Piccotti E, Andre P (2016) EEG theta dynamics within frontal and parietal cortices for error processing during reaching movements in a prism adaptation study altering visuo-motor predictive planning. PLoS ONE 11(3) e0150265.

Aubry, M., Cantu, R., Dvorak, J., Graf-Baumann, T., Johnston, K., Kelly, J., et al. (2002). Summary and agreement statement of the first International Conference on Concussion in Sport, Vienna 2001. Phys Sportsmedicine, 30(2), 57-63.

Bayer HM, Glimcher PW (2005) Midbrain dopamine neurons encode a quantitative reward prediction error signal. Neuron 47 (1):129–141.

Beste C, Dziobek I, Hielscher H, Willemssen R, Falkenstein M (2009a) Effects of stimulus-response compatibility on inhibitory processes in Parkinson's disease. Eur J Neurosci 29(4):855–860. https://doi.org/10.1111/j.1460-9568.2009.06621.x.

Beste C, Muckschel M, Rosales R, Domingo A, Lee L, Ng A, et al. (2017) Striosomal dysfunction affects behavioral adaptation but not impulsivity-Evidence from X-linked dystonia-parkinsonism. Mov Disord 32(4):576–584. https://doi.org/10.1002/mds.26895.

Beste C, Saft C, Andrich J, Gold R, Falkenstein M (2006) Error processing in Huntington's disease. PLoS ONE 1. https://doi.org/10.1371/journal.pone.0000086 e86.

Beste C, Saft C, Konrad C, Andrich J, Habbel A, Schepers I, Falkenstein M (2008) Levels of error processing in Huntington's disease: a combined study using event-related potentials and voxel-based morphometry. Hum Brain Mapp 29(2):121–130. https://doi.org/10.1002/hbm.20374.

Beste C, Saft C, Yordanova J, Andrich J, Gold R, Falkenstein M, Kolev V (2007) Functional compensation or pathology in cortico-subcortical interactions in preclinical Huntington's disease? Neuropsychologia 45(13):2922–2930. https://doi.org/10.1016/j.neuropsychologia.2007.06.004.

Beste C, Willemssen R, Saft C, Falkenstein M (2009b) Error processing in normal aging and in basal ganglia disorders. Neuroscience 159(1):143–149. https://doi.org/10.1016/j.neuroscience.2008.12.030.

Bettcher BM, Giovannetti T, Macmullen L, Libon DJ (2008) Error detection and correction patterns in dementia: a breakdown of error monitoring processes and their neuropsychological correlates. J Int Neuropsychol Soc 14(2):199–208. https://doi.org/10.1017/S1355617708080193.

Botvinick MM, Braver TS, Barch DM, Carter CS, Cohen JD (2001) Conflict monitoring and cognitive control. Psychol Rev 108 (3):624.

Brown JW, Braver TS (2005) Learned predictions of error likelihood in the anterior cingulate cortex. Science 307(5712):1118–1121. https://doi.org/10.1126/science.1105783.

Boldt A, Yeung N (2015) Shared neural markers of decision confidence and error detection. J Neurosci 35(8):3478–3484.

Brush CJ, Ehmann PJ, Olson RL, Bixby WR, Alderman BL (2018) Do sport-related concussions result in long-term cognitive impairment? A review of event-related potential research. Int J Psychophysiol 132:124–134.

Bush G, Luu P, Posner MI (2000) Cognitive and emotional influences in anterior cingulate cortex. Trends Cognitive Sci 4(6):215–222.
Calabresi P, Picconi B, Parnetti L, Di Filippo M (2006) A convergent model for cognitive dysfunctions in Parkinson's disease: the critical dopamine–acetylcholine synaptic balance. Lancet Neurol 5 (11):974–983.
Campanella F, Skrap M, Vallesi A (2016) Speed-accuracy strategy regulations in prefrontal tumor patients. Neuropsychologia 82:1–10.
Candidi M, Sacheli LM, Era V, Canzano L, Tieri G, Aglioti SM (2017) Come together: human–avatar on-line interactions boost joint-action performance in apraxic patients. Social Cognitive Affective Neurosci 12(11):1793–1802.
Canzano L, Scandola M, Gobbetto V, Moretto G, D'Imperio D, Moro V (2016) The representation of objects in apraxia: from action execution to error awareness. Front Hum Neurosci 10:39.
Carter CS, Braver TS, Barch DM, Botvinick MM, Noll D, Cohen JD (1998) Anterior cingulate cortex, error detection, and the online monitoring of performance. Science 280(5364):747–749.
Carriere N, Bourriez JL, Delval A, Derambure P, Defebvre L, Dujardin K (2016) Impulse control disorders in Parkinson's disease are associated with alterations in reward-related cortical oscillations. J Parkinsons Dis 6(3):651–666. https://doi.org/10.3233/JPD-160828.
Cavanagh JF, Frank MJ (2014) Frontal theta as a mechanism for cognitive control. Trends Cognitive Sci 18(8):414–421.
Chaudhuri KR, Yates L, Martinez-Martin P (2005) The non-motor symptom complex of Parkinson's disease: a comprehensive assessment is essential. Curr Neurol Neurosci Rep 5(4):275–283.
Chen H, Epstein J, Stern E (2010) Neural plasticity after acquired brain injury: evidence from functional neuroimaging. PM&R 2: S306–S312.
Chiang HS, Spence JS, Kraut MA, Mudar RA (2018) Age effects on event-related potentials in individuals with amnestic Mild Cognitive Impairment during semantic categorization Go/NoGo tasks. Neurosci Lett 670:19–21. https://doi.org/10.1016/j.neulet.2018.01.034.
Chiaravalloti ND, DeLuca J (2008) Cognitive impairment in multiple sclerosis. Lancet Neurol 7(12):1139–1151.
Clark A (2013) Whatever next? Predictive brains, situated agents, and the future of cognitive science. Behav Brain Sci 36 (3):181–204.
Cohen MX (2014) Analyzing neural time series data: theory and practice. MIT press.
Cohen MX (2011) Error-related medial frontal theta activity predicts cingulate-related structural connectivity. Neuroimage 55 (3):1373–1383.
Cohen MX, Elger CE, Ranganath C (2007) Reward expectation modulates feedback-related negativity and EEG spectra. Neuroimage 35(2):968–978.
Cools R (2006) Dopaminergic modulation of cognitive function-implications for L-DOPA treatment in Parkinson's disease. Neurosci Biobehav Rev 30(1):1–23.
Dehaene S, Changeux JP. (2003). Neural mechanisms for access to consciousness. The cognitive neurosciences III.
De Beaumont L, Beauchemin M, Beaulieu C, Jolicoeur P (2013) Long-term attenuated electrophysiological response to errors following multiple sports concussions. J Clin Exp Neuropsychol 35(6):596–607. https://doi.org/10.1080/13803395.2013.800023.
Debener S, Ullsperger M, Siegel M, Fiehler K, Von Cramon DY, Engel AK (2005) Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. J Neurosci 25(50):11730–11737.
Desender K, Boldt A, Verguts T, Donner TH (2019) Confidence predicts speed-accuracy tradeoff for subsequent decisions. Elife 8 e43499.
Di Gregorio F, Steinhauser M, Maier ME (2016) Error-related brain activity and error awareness in an error classification paradigm. NeuroImage 139:202–210.
Di Gregorio F, Maier ME, Steinhauser M (2018) Errors can elicit an error positivity in the absence of an error negativity: evidence for independent systems of human error monitoring. Neuroimage 172:427–436. https://doi.org/10.1016/j.neuroimage.2018.01.081.
Donchin E, Coles MG (1988) Is the P300 component a manifestation of context updating? Behav Brain Sci 11(3):357–374.
Endrass T, Reuter B, Kathmann N (2007) ERP correlates of conscious error recognition: aware and unaware errors in an antisaccade task. Eur J Neurosci 26(6):1714–1720.
Era V, Aglioti SM, Candidi M (2020) Inhibitory theta burst stimulation highlights the role of left aIPS and Right TPJ during complementary and imitative human-avatar interactions in cooperative and competitive scenarios. Cereb Cortex 30 (3):1677–1687.
Eriksen BA, Eriksen CW (1974) Effects of noise letters upon the identification of a target letter in a nonsearch task. Perception Psychophys 16(1):143–149.
Falkenstein M, Hohnsbein J, Hoormann J, Blanke L (1991) Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. Electroencephalogr Clin Neurophysiol 78(6):447–455.
Falkenstein M, Hoormann J, Christ S, Hohnsbein J (2000) ERP components on reaction errors and their functional significance: a tutorial. Biol Psychol 51(2–3):87–107.
Falkenstein M, Hielscher H, Dziobek I, Schwarzenau P, Hoormann J, Sundermann B, Hohnsbein J (2001) Action monitoring, error detection, and the basal ganglia: an ERP study. NeuroReport 12 (1):157–161.
Falkenstein M, Willemssen R, Hohnsbein J, Hielscher H (2005) Error processing in Parkinson's Disease. J Psychophysiol 19 (4):305–310. https://doi.org/10.1027/0269-8803.19.4.305.
Farooqui AA, Bhutani N, Kulashekhar S, Behari M, Goel V, Murthy A (2011) Impaired conflict monitoring in Parkinson's disease patients during an oculomotor redirect task. Exp Brain Res 208 (1):1–10.
Fleming SM, Ryu J, Golfinos JG, Blackmon KE (2014) Domain-specific impairment in metacognitive accuracy following anterior prefrontal lesions. Brain 137(10):2811–2822.
Fontana AP, Kilner JM, Rodrigues EC, Joffily M, Nighoghossian N, Vargas CD, Sirigu A (2012) Role of the parietal cortex in predicting incoming actions. Neuroimage 59(1):556–564.
Frank MJ, Woroch BS, Curran T (2005) Error-related negativity predicts reinforcement learning and conflict biases. Neuron 47 (4):495–501. https://doi.org/10.1016/j.neuron.2005.06.020.
Friston K (2010) The free-energy principle: a unified brain theory? Nat Rev Neurosci 11(2):127–138. https://doi.org/10.1038/nrn2787.
Fusaro M, Tieri G, Aglioti SM (2019) Influence of cognitive stance and physical perspective on subjective and autonomic reactivity to observed pain and pleasure: an immersive virtual reality study. Conscious Cogn 67:86–97.
Fusco G, Fusaro M, Aglioti SM (2020) Midfrontal-occipital tACS modulates cognitive conflicts related to bodily stimuli. Social Cognitive Affect Neurosci.
Fusco G, Scandola M, Feurra M, Pavone EF, Rossi S, Aglioti SM (2018) Midfrontal theta transcranial alternating current stimulation modulates behavioural adjustment after error execution. Eur J Neurosci 48(10):3159–3170.
Gehring WJ, Knight RT (2000) Prefrontal–cingulate interactions in action monitoring. Nat Neurosci 3(5):516–520.
Gehring WJ, Goss B, Coles MG, Meyer DE, Donchin E (1993) A neural system for error detection and compensation. Psychol Sci 4:385–390.
Giza CC, Hovda DA (2001) The neurometabolic cascade of concussion. J Athletic Train 36(3):228.
Gleichgerrcht E, Ibanez A, Roca M, Torralva T, Manes F (2010) Decision-making cognition in neurodegenerative diseases. Nat Rev Neurol 6(11):611–623. https://doi.org/10.1038/nrneurol.2010.148.
Gratton C, Sun H, Petersen SE (2018a) Control networks and hubs. Psychophysiology 55(3) e13032.

Gratton G, Cooper P, Fabiani M, Carter CS, Karayanidis F (2018b) Dynamics of cognitive control: theoretical bases, paradigms, and a view for the future. Psychophysiology 55(3) e13016.
Hallett, P. E. (1978). Primary and secondary saccades to goals defined by instructions. Vision research, 18(10), 1279-1296.
Halliday GM, McCann H (2010) The progression of pathology in Parkinson's disease. Ann N Y Acad Sci 1184(1):188–195.
Hajcak G, Nieuwenhuis S, Ridderinkhof KR, Simons RF (2005) Error-preceding brain activity: robustness, temporal dynamics, and boundary conditions. Biol Psychol 70(2):67–78.
Herrmann MJ, Römmler J, Ehlis AC, Heidrich A, Fallgatter AJ (2004) Source localization (LORETA) of the error-related-negativity (ERN/Ne) and positivity (Pe). Cognitive Brain Res 20(2):294–299.
Hoerold D, Pender NP, Robertson IH (2013) Metacognitive and online error awareness deficits after prefrontal cortex lesions. Neuropsychologia 51(3):385–391.
Hoffmann S, Beste C (2015) A perspective on neural and cognitive mechanisms of error commission. Front Behav Neurosci 9:50. https://doi.org/10.3389/fnbeh.2015.00050.
Hogan AM, Vargha-Khadem F, Saunders DE, Kirkham FJ, Baldeweg T (2006) Impact of frontal white matter lesions on performance monitoring: ERP evidence for cortical disconnection. Brain 129(Pt 8):2177–2188. https://doi.org/10.1093/brain/awl160.
Holroyd CB, Praamstra P, Plat E, Coles MG (2002) Spared error-related potentials in mild to moderate Parkinson's disease. Neuropsychologia 40(12):2116–2124.
Holroyd CB, Coles MGH (2002) The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. Psychol Rev 109(4):679–709. https://doi.org/10.1037/0033-295x.109.4.679.
Hughes G, Yeung N (2011) Dissociable correlates of response conflict and error awareness in error-related brain activity. Neuropsychologia 49:405–415.
Huntington, G. (1872). On chorea. the medical and surgical reporter of Philadelphia. cited in H. Skirton (2005),'Huntington's Disease: a nursing perspective', MedSurg Nursing, 14(3), 167-174.
Huygelier H, Schraepen B, van Ee R, Abeele VV, Gillebert CR (2019) Acceptance of immersive head-mounted virtual reality in older adults. Sci Rep 9(1):1–12.
Iannaccone R, Hauser TU, Staempfli P, Walitza S, Brandeis D, Brem S (2015) Conflict monitoring and error processing: new insights from simultaneous EEG–fMRI. Neuroimage 105:395–407.
Ide JS, Li CS (2011) A cerebellar thalamic cortical circuit for error-related cognitive control. Neuroimage 54(1):455–464. https://doi.org/10.1016/j.neuroimage.2010.07.042.
Ito J, Kitagawa J (2005) Error processing in patients with Alzheimer's disease. Pathophysiology 12(2):97–101. https://doi.org/10.1016/j.pathophys.2005.02.003.
Ito J, Kitagawa J (2006) Performance monitoring and error processing during a lexical decision task in patients with Parkinson's disease. J Geriatr Psychiatry Neurol 19(1):46–54. https://doi.org/10.1177/0891988705284716.
Karantzoulis S, Galvin JE (2011) Distinguishing Alzheimer's disease from other major forms of dementia. Expert Rev Neurother 11 (11):1579–1591. https://doi.org/10.1586/ern.11.155.
Kendi AK, Lehericy S, Luciana M, Ugurbil K, Tuite P (2008) Altered diffusion in the frontal lobe in Parkinson disease. Am J Neuroradiol 29(3):501–505.
Kilner JM, Friston KJ, Frith CD (2007) Predictive coding: an account of the mirror neuron system. Cogn Process 8(3):159–166.
Klaus A, Alves da Silva J, Costa RM (2019) What, if, and when to move: basal ganglia circuits and self-paced action initiation. Annu Rev Neurosci 42:459–483.
Klein TA, Ullsperger M, Danielmeier C (2013) Error awareness and the insula: links to neurological and psychiatric diseases. Front Hum Neurosci 7:14.
Koban L, Pourtois G (2014) Brain systems underlying the affective and social monitoring of actions: an integrative review. Neurosci Biobehav Rev 46:71–84.
Koelewijn T, van Schie HT, Bekkering H, Oostenveld R, Jensen O (2008) Motor-cortical beta oscillations are modulated by correctness of observed action. Neuroimage 40(2):767–775.
Krigolson OE, Holroyd CB (2006) Evidence for hierarchical error processing in the human brain. Neuroscience 137(1):13–17.
Kuusinen V, Cesnaite E, Perakyla J, Ogawa KH, Hartikainen KM (2018) Orbitofrontal lesion alters brain dynamics of emotion-attention and emotion-cognitive control interaction in humans. Front Hum Neurosci 12:437. https://doi.org/10.3389/fnhum.2018.00437.
Jocham G, Ullsperger M (2009) Neuropharmacology of performance monitoring. Neurosci Biobehav Rev 33(1):48–60.
Joch M, Hegele M, Maurer H, Müller H, Maurer LK (2017) Brain negativity as an indicator of predictive error processing: the contribution of visual action effect monitoring. J Neurophysiol 118 (1):486–495.
Langlois JA, Rutland-Brown W, Wald MM (2006) The epidemiology and impact of traumatic brain injury: a brief overview. J Head Trauma Rehabil 21(5):375–378.
Larson MJ, Clayson PE, Clawson A (2014) Making sense of all the conflict: a theoretical review and critique of conflict-related ERPs. Int J Psychophysiol 93(3):283–297.
Larson MJ, Clayson PE, Farrer TJ (2012) Performance monitoring and cognitive control in individuals with mild traumatic brain injury. J Int Neuropsychol Soc 18(2):323–333. https://doi.org/10.1017/S1355617711001779.
Larson MJ, Farrer TJ, Clayson PE (2011) Cognitive control in mild traumatic brain injury: conflict monitoring and conflict adaptation. Int J Psychophysiol 82(1):69–78. https://doi.org/10.1016/j.ijpsycho.2011.02.018.
Larson MJ, Kaufman DA, Schmalfuss IM, Perlstein WM (2007) Performance monitoring, error processing, and evaluative control following severe TBI. J Int Neuropsychol Soc: JINS 13(6):961.
Larson MJ, Kaufman DA, Kellison IL, Schmalfuss IM, Perlstein WM (2009a) Double jeopardy! The additive consequences of negative affect on performance-monitoring decrements following traumatic brain injury. Neuropsychology 23(4):433.
Larson MJ, Kaufman DA, Perlstein WM (2009b) Conflict adaptation and cognitive control adjustments following traumatic brain injury. J Int Neuropsychol Soc 15(6):927–937. https://doi.org/10.1017/S1355617709990701.
Larson MJ, Perlstein WM (2009c) Awareness of deficits and error processing after traumatic brain injury. NeuroReport 20 (16):1486–1490. https://doi.org/10.1097/WNR.0b013e32833283fe.
Laughlin SB, Sejnowski TJ (2003) Communication in neuronal networks. Science 301(5641):1870–1874.
Logan GD, Cowan WB (1984) On the ability to inhibit thought and action: a theory of an act of control. Psychol Rev 91(3):295.
Lopez-Gongora M, Escartin A, Martinez-Horta S, Fernandez-Bobadilla R, Querol L, Romero S, Riba J (2015) Neurophysiological evidence of compensatory brain mechanisms in early-stage multiple sclerosis. PLoS ONE 10(8). https://doi.org/10.1371/journal.pone.0136786 e0136786.
Lovstad, M., Funderud, I., Meling, T., Kramer, U. M., Voytek, B., Due-Tonnessen, P. et al. (2012). Anterior cingulate cortex and cognitive control: neuropsychological and electrophysiological findings in two patients with lesions to dorsomedial prefrontal cortex. Brain Cogn, 80(2), 237-249. doi:10.1016/j.bandc.2012.07.008.
Luu P, Tucker DM, Makeig S (2004) Frontal midline theta and the error-related negativity: neurophysiological mechanisms of action regulation. Clin Neurophysiol 115(8):1821–1835.
Maier M, Steinhauser M, Hübner R (2008) Is the error-related negativity amplitude related to error detectability? Evidence from effects of different error types. J Cognit Neurosci 20 (12):2263–2273.
Maier ME, Yeung N, Steinhauser M (2011) Error-related brain activity and adjustments of selective attention following errors. Neuroimage 56(4):2339–2347.

Maier ME, Di Gregorio F, Muricchio T, Di Pellegrino G (2015) Impaired rapid error monitoring but intact error signaling following rostral anterior cingulate cortex lesions in humans. Front Hum Neurosci 9:339. https://doi.org/10.3389/fnhum.2015.00339.

Maki-Marttunen V, Kuusinen V, Brause M, Peräkylä J, Polvivaara M, dos Santos Ribeiro R, et al. (2015) Enhanced attention capture by emotional stimuli in mild traumatic brain injury. J Neurotrauma 32 (4):272–279.

Maki-Marttunen V, Kuusinen V, Perakyla J, Ogawa KH, Brause M, Brander A, Hartikainen KM (2017) Greater attention to task-relevant threat due to orbitofrontal lesion. J Neurotrauma 34 (2):400–413. https://doi.org/10.1089/neu.2015.4390.

Mars RB, Sallet J, Rushworth MF, Yeung N. (Eds.). (2011). Neural basis of motivational and cognitive control. MIT Press.

Masina F, Tarantino V, Vallesi A, Mapelli D (2019) Repetitive TMS over the left dorsolateral prefrontal cortex modulates the error positivity: an ERP study. Neuropsychologia 133 107153.

Mathalon DH, Bennett A, Askari N, Gray EM, Rosenbloom MJ, Ford JM (2003) Response-monitoring dysfunction in aging and Alzheimer's disease: an event-related potential study. Neurobiol Aging 24(5):675–685. https://doi.org/10.1016/s0197-4580(02)00154-9.

Maurer LK, Maurer H, Müller H (2015) Neural correlates of error prediction in a complex motor task. Front Behav Neurosci 9:209.

Maurer LK, Joch M, Hegele M, Maurer H, Müller H (2019) Predictive error processing distinguishes between relevant and irrelevant errors after visuomotor learning. J Vision 19(4):18.

Mierzwa AJ, Marion CM, Sullivan GM, McDaniel DP, Armstrong RC (2015) Components of myelin damage and repair in the progression of white matter pathology after mild traumatic brain injury. J Neuropathol Exp Neurol 74(3):218–232.

Miltner WH, Brauer J, Hecht H, Trippe R. (2003). Parallel brain activity for self-generated and observed errors Wolfgang HR Miltner1, Jens Brauer1, Holger Hecht1, Ralf Trippe1, Michael GH Coles2 1Department of Biological and Clinical Psychology, Friedrich-Schiller-University, Jena, Germany.

Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group (2009). Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement.

Moreau Q, Parrotta E, Era V, Martelli ML, Candidi M (2020a) Role of the occipito-temporal Theta rhythm in hand visual identification. J Neurophysiol 123(1):167–177.

Moreau Q, Candidi M, Era V, Tieri G, Aglioti SM (2020b) Midline frontal and occipito-temporal activity during error monitoring in dyadic motor interactions. Cortex.

Moro V (2013) The interaction between implicit and explicit awareness in anosognosia: emergent awareness. Cognitive Neurosci 4(3–4):199–200.

Murphy PR, Robertson IH, Harty S, O'Connell RG (2015) Neural evidence accumulation persists after choice to inform metacognitive judgments. Elife 4 e11946.

Narayanan NS, Cavanagh JF, Frank MJ, Laubach M (2013) Common medial frontal mechanisms of adaptive control in humans and rodents. Nat Neurosci 16(12):1888–1895. https://doi.org/10.1038/nn.3549.

Nguyen LT, Mudar RA, Chiang HS, Schneider JM, Maguire MJ, Kraut MA, Hart Jr J (2017) Theta and alpha alterations in amnestic mild cognitive impairment in semantic Go/NoGo tasks. Front Aging Neurosci 9:160. https://doi.org/10.3389/fnagi.2017.00160.

Nieuwenhuis S, Holroyd CB, Mol N, Coles MGH (2004) Reinforcement-related brain potentials from medial frontal cortex: origins and functional significance. Neurosci Biobehav Rev 28:441–448.

Nieuwenhuis S, Ridderinkhof KR, Blom J, Band GP, Kok A (2001) Error-related brain potentials are differentially related to awareness of response errors: evidence from an antisaccade task. Psychophysiology 38(5):752–760.

Nieuwenhuis S, Yeung N, Van Den Wildenberg W, Ridderinkhof KR (2003) Electrophysiological correlates of anterior cingulate function in a go/no-go task: effects of response conflict and trial type frequency. Cognitive Affect Behav Neurosci 3(1):17–26.

Notebaert W, Houtman F, Van Opstal F, Gevers W, Fias W, Verguts T (2009) Post-error slowing: an orienting account. Cognition 111 (2):275–279.

Olson RL, Brush CJ, Ehmann PJ, Buckman JF, Alderman BL (2018) A history of sport-related concussion is associated with sustained deficits in conflict and error monitoring. Int J Psychophysiol 132(Pt A):145–154. https://doi.org/10.1016/j.ijpsycho.2018.01.006.

Ozkan DG, Pezzetta R (2018) Predictive monitoring of actions, EEG recordings in virtual reality. J Neurophysiol 119(4):1254–1256.

Özkan DG, Pezzetta R, Moreau Q, Abreu AM, Aglioti SM (2019) Predicting the fate of basketball throws: an EEG study on expert action prediction in wheelchair basketball players. Exp Brain Res 237(12):3363–3373.

Overbeek TJ, Nieuwenhuis S, Ridderinkhof KR (2005) Dissociable components of error processing: on the functional significance of the Pe vis-à-vis the ERN/Ne. J Psychophysiol 19(4):319–329.

Padrao G, Gonzalez-Franco M, Sanchez-Vives MV, Slater M, Rodriguez-Fornells A (2016) Violating body movement semantics: neural signatures of self-generated and external-generated errors. Neuroimage 124:147–156.

Panasiti MS, Pavone EF, Aglioti SM (2016) Electrocortical signatures of detecting errors in the actions of others: an EEG study in pianists, non-pianist musicians and musically naïve people. Neuroscience 318:104–113.

Parkinson J (1817) An essay on the shaking palsy. London: Sherwood, Neeley & Jones.

Pavone EF, Tieri G, Rizza G, Tidoni E, Grisoni L, Aglioti SM (2016) Embodying others in immersive virtual reality: electro-cortical signatures of monitoring the errors in the actions of an avatar seen from a first-person perspective. J Neurosci 36(2):268–279.

Perlstein WM, Larson MJ, Dotson VM, Kelly KG (2006) Temporal dissociation of components of cognitive control dysfunction in severe TBI: ERPs and the cued-Stroop task. Neuropsychologia 44:260–274.

Perry RJ, Hodges JR (1999) Attention and executive deficits in Alzheimer's disease: A critical review. Brain 122(3):383–404.

Peterburs J, Gajda K, Koch B, Schwarz M, Hoffmann KP, Daum I, Bellebaum C (2012) Cerebellar lesions alter performance monitoring on the antisaccade task-an event related potentials study. Neuropsychologia 50(3):379–389. https://doi.org/10.1016/j.neuropsychologia.2011.12.009.

Peterburs J, Pergola G, Koch B, Schwarz M, Hoffmann KP, Daum I, Bellebaum C (2011) Altered error processing following vascular thalamic damage: evidence from an antisaccade task. PLoS ONE 6(6). https://doi.org/10.1371/journal.pone.0021517 e21517.

Peterburs J, Thurling M, Rustemeier M, Goricke S, Suchan B, Timmann D, Bellebaum C (2015) A cerebellar role in performance monitoring - evidence from EEG and voxel-based morphometry in patients with cerebellar degenerative disease. Neuropsychologia 68:139–147. https://doi.org/10.1016/j.neuropsychologia.2015.01.017.

Petersen RC, Caracciolo B, Brayne C, Gauthier S, Jelic V, Fratiglioni L (2014) Mild cognitive impairment: a concept in evolution. J Intern Med 275(3):214–228. https://doi.org/10.1111/joim.12190.

Pezzetta R, Nicolardi V, Tidoni E, Aglioti SM (2018) Error, rather than its probability, elicits specific electrocortical signatures: a combined EEG-immersive virtual reality study of action observation. J Neurophysiol 120(3):1107–1118.

Polich J (2007) Updating P300: an integrative theory of P3a and P3b. Clin Neurophysiol 118(10):2128–2148.

Ponsi G, Panasiti MS (2020) Impulsive-compulsive disorders in Parkinson's Disease: influence on individual and social decision-making processes. Rivista di Psichiatria 55(4):213–221.

Pontifex MB, O'Connor PM, Broglio SP, Hillman CH (2009) The association between mild traumatic brain injury history and cognitive control. Neuropsychologia 47(14):3210–3216. https://doi.org/10.1016/j.neuropsychologia.2009.07.021.

Proverbio AM, Crotti N, Manfredi M, Adorni R, Zani A (2012) Who needs a referee? How incorrect basketball actions are automatically detected by basketball players' brain. Sci Rep 2 (1):1–8.

Rabbitt P, Rodgers B (1977) What does a man do after he makes an error? An analysis of response programming. Q J Exp Psychol 29:727–743.

Renault, B., Ragot, R. and LesOvre, N. Correct and incorrect responses in a choice reaction time task and the endogenous components of the evoked potential. In: H.It. Kornhuher and L. I) eecke (Eds.), Motivation, Motor and Sensory Processes of the Brain; Electrical Potentials, Behaviour and ('linical /se. El,∼cvier, Amsterdam, 1980: 647-654.

Ridderinkhof KR (2014) Neurocognitive mechanisms of perception-action coordination: a review and theoretical integration. Neurosci Biobehav Rev 46:3–29. https://doi.org/10.1016/j.neubiorev.2014.05.008.

Riès SK, Xie K, Haaland KY, Dronkers NF, Knight RT (2013) Role of the lateral prefrontal cortex in speech monitoring. Front Hum Neurosci 7:703.

Ridderinkhof KR, Nieuwenhuis S, Bashore TR (2003) Errors are foreshadowed in brain potentials associated with action monitoring in cingulate cortex in humans. Neurosci Lett 348 (1):1–4.

Ridderinkhof KR, Brass M (2015) How kinesthetic motor imagery works: a predictive-processing theory of visualization in sports and motor expertise. J Physiol-Paris 109(1–3):53–63.

Ridderinkhof KR, Ramautar JR, Wijnen JG (2009) To PE or not to PE: A P3-like ERP component reflecting the processing of response errors. Psychophysiology 46(3):531–538.

Ridderinkhof KR, Ullsperger M, Crone EA, Nieuwenhuis S (2004) The role of medial frontal cortex in cognitive control. Science 306:443–447.

Rosen HJ (2011) Anosognosia in neurodegenerative disease. Neurocase 17(3):231–241.

Ruchsow M, Grön G, Reuter K, Spitzer M, Hermle L, Kiefer M (2005) Error-related brain activity in patients with obsessive-compulsive disorder and in healthy controls. J Psychophysiol 19(4):298–304.

Rustamov N, Rodriguez-Raecke R, Timm L, Agrawal D, Dressler D, Schrader C, Kopp B (2014) Attention shifting in Parkinson's disease: an analysis of behavioral and cortical responses. Neuropsychology 28(6):929.

Scandola M, Canzano L, Avesani R, Leder M, Bertagnoli S, Gobbetto V, et al. (2020) Anosognosia for limb and bucco-facial apraxia as inferred from the recognition of gestural errors. J Neuropsychol e12203.

Schultz W (1997) Dopamine neurons and their role in reward mechanisms. Curr Opin Neurobiol 7(2):191–197.

Schultz W (2002) Getting formal with dopamine and reward. Neuron 36(2):241–263.

Seer C, Lange F, Georgiev D, Jahanshahi M, Kopp B (2016) Event-related potentials and cognition in Parkinson's disease: an integrative review. Neurosci Biobehav Rev 71:691–714.

Seer C, Lange F, Loens S, Wegner F, Schrader C, Dressler D, et al. (2017) Dopaminergic modulation of performance monitoring in Parkinson's disease: an event-related potential study. Sci Rep 7:41222.

Seifert S, von Cramon DY, Imperati D, Tittgemeyer M, Ullsperger M (2011) Thalamocingulate interactions in performance monitoring. J Neurosci 31(9):3375–3383. https://doi.org/10.1523/JNEUROSCI.6242-10.2011.

Shadmehr R (2017) Learning to predict and control the physics of our movements. J Neurosci 37(7):1663–1671. https://doi.org/10.1523/JNEUROSCI.1675-16.2016.

Simson R, Vaughan Jr HG, Ritter W (1977) The scalp topography of potentials in auditory and visual Go/NoGo tasks. Electroencephalogr Clin Neurophysiol 43(6):864–875.

Singh A, Richardson SP, Narayanan N, Cavanagh JF (2018) Mid-frontal theta activity is diminished during cognitive control in Parkinson's disease. Neuropsychologia 117:113–122. https://doi.org/10.1016/j.neuropsychologia.2018.05.020.

Smith MA, Brandt J, Shadmehr R (2000) Motor disorder in Huntington's disease begins as a dysfunction in error feedback control. Nature 403(6769):544–549. https://doi.org/10.1038/35000576.

Solbakk, A. K., Funderud, I., Lovstad, M., Endestad, T., Meling, T., Lindgren, M., et al. (2014). Impact of orbitofrontal lesions on electrophysiological signals in a stop signal task. J Cogn Neurosci, 26(7), 1528-1545. doi:10.1162/jocn_a_00561.

Spinelli G, Tieri G, Pavone EF, Aglioti SM (2018) Wronger than wrong: graded mapping of the errors of an avatar in the performance monitoring system of the onlooker. NeuroImage 167:1–10.

Steinhauser M, Yeung N (2012) Error awareness as evidence accumulation: effects of speed-accuracy trade-off on error signaling. Front Hum Neurosci 6:240.

Stemmer B, Segalowitz SJ, Dywan J, Panisset M, Melmed C (2007) The error negativity in nonmedicated and medicated patients with Parkinson's disease. Clin Neurophysiol 118(6):1223–1229. https://doi.org/10.1016/j.clinph.2007.02.019.

Stemmer B, Segalowitz SJ, Witzke W, Schönle PW (2004) Error detection in patients with lesions to the medial prefrontal cortex: an ERP study. Neuropsychologia 42(1):118–130. https://doi.org/10.1016/s0028-3932(03)00121-0.

Stroop JR (1935) Studies of interference in serial verbal reactions. J Exp Psychol 18(6):643.

Suresh KP, Chandrashekara S (2012) Sample size estimation and power analysis for clinical research studies. J Human Reprod Sci 5(1):7.

Swick D, Turken U (2002) Dissociation between conflict detection and error monitoring in the human anterior cingulate cortex. Proc Natl Acad Sci 99(25):16354–16359.

Tan Y, Vandeput J, Qiu J, Van den Bergh O, von Leupoldt A (2019) The error-related negativity for error processing in interoception. Neuroimage 184:386–395.

Thompson PD, Berardelli A, Rothwell JC, Day BL, Dick JPR, Benecke R, Marsden CD (1988) The coexistence of bradykinesia and chorea in Huntington's disease and its implications for theories of basal ganglia control of movement. Brain 111(2):223–244.

Thurm F, Antonenko D, Schlee W, Kolassa S, Elbert T, Kolassa IT (2013) Effects of aging and mild cognitive impairment on electrophysiological correlates of performance monitoring. J Alzheimers Dis 35(3):575–587. https://doi.org/10.3233/JAD-121348.

Tieri G, Morone G, Paolucci S, Iosa M (2018) Virtual reality in cognitive and motor rehabilitation: facts, fiction and fallacies. Expert Rev Med Devices 15(2):107–117.

Torrecillos F, Alayrangues J, Kilavik BE, Malfait N (2015) Distinct modulations in sensorimotor postmovement and foreperiod β-band activities related to error salience processing and sensorimotor adaptation. J Neurosci 35(37):12753–12765.

Tsujimoto T, Shimazu H, Isomura Y (2006) Direct recording of theta oscillations in primate prefrontal and anterior cingulate cortices. J Neurophysiol 95(5):2987–3000.

Turken AU, Swick D (2008) The effect of orbitofrontal lesions on the error-related negativity. Neurosci Lett 441(1):7–10. https://doi.org/10.1016/j.neulet.2008.05.115.

Ullsperger M, von Cramon DY (2006) The role of intact frontostriatal circuits in error processing. J Cognit Neurosci 18(4):651–664.

Ullsperger M, Danielmeier C (2016) Reducing speed and sight: how adaptive is post-error slowing? Neuron 89(3):430–432. https://doi.org/10.1016/j.neuron.2016.01.035.

Ullsperger M, Danielmeier C, Jocham G (2014a) Neurophysiology of performance monitoring and adaptive behavior. Physiol Rev 94 (1):35–79. https://doi.org/10.1152/physrev.00041.2012.

Ullsperger M, Fischer AG, Nigbur R, Endrass T (2014b) Neural mechanisms and temporal dynamics of performance monitoring. Trends Cogn Sci 18(5):259–267. https://doi.org/10.1016/j.tics.2014.02.009.

Ullsperger M, von Cramon DY, Muller NG (2002) Interactions of focal cortical lesions with error processing: evidence from event-related brain potentials. Neuropsychology 16(4):548–561. https://doi.org/10.1037//0894-4105.16.4.548.

van Driel J, Ridderinkhof KR, Cohen MX (2012) Not all errors are alike: theta and alpha EEG dynamics relate to differences in error-processing dynamics. J Neurosci 32(47):16795–16806.

van Schie HT, Mars RB, Coles MG, Bekkering H (2004) Modulation of activity in medial frontal and motor cortices during error observation. Nat Neurosci 7(5):549–554.

Verbruggen F, Aron AR, Band GP, Beste C, Bissett PG, Brockett AT, Colzato LS (2019) A consensus guide to capturing the ability to inhibit actions and impulsive behaviors in the stop-signal task. Elife 8 e46323.

Verleger R, Schroll H, Hamker FH (2013) The unstable bridge from stimulus processing to correct responding in Parkinson's disease. Neuropsychologia 51(13):2512–2525. https://doi.org/10.1016/j.neuropsychologia.2013.09.017.

Villa R, Tidoni E, Porciello G, Aglioti SM (2020) Freedom to act enhances the sense of agency, while movement and goal-related prediction errors reduce it. Psychol Res.

Volpato C, Schiff S, Facchini S, Silvoni S, Cavinato M, Piccione F, Birbaumer N (2016) Dopaminergic medication modulates learning from feedback and error-related negativity in Parkinson's Disease: a pilot study. Front Behav Neurosci 10:205. https://doi.org/10.3389/fnbeh.2016.00205.

Wang C, Ulbert I, Schomer DL, Marinkovic K, Halgren E (2005) Responses of human anterior cingulate cortex microdomains to error detection, conflict monitoring, stimulus-response mapping, familiarity, and orienting. J Neurosci 25(3):604–613.

Walker FO (2007) Huntington's disease. Lancet 369(9557):218–228. https://doi.org/10.1016/s0140-6736(07)60111-1.

Wessel JR (2012) Error awareness and the error-related negativity: evaluating the first decade of evidence. Front Hum Neurosci 6:88.

Wessel JR, Klein TA, Ott DV, Ullsperger M (2014) Lesions to the prefrontal performance-monitoring network disrupt neural processing and adaptive behaviors after both errors and novelty. Cortex 50:45–54. https://doi.org/10.1016/j.cortex.2013.09.002.

Wessel JR, Ullsperger M, Obrig H, Villringer A, Quinque E, Schroeter ML, et al. (2016) Neural synchrony indexes impaired motor slowing after errors and novelty following white matter damage. Neurobiol Aging 38:205–213. https://doi.org/10.1016/j.neurobiolaging.2015.10.014.

Willemssen R, Falkenstein M, Schwarz M, Muller T, Beste C (2011) Effects of aging, Parkinson's disease, and dopaminergic medication on response selection and control. Neurobiol Aging 32(2):327–335. https://doi.org/10.1016/j.neurobiolaging.2009.02.002.

Willemssen R, Muller T, Schwarz M, Falkenstein M, Beste C (2009) Response monitoring in de novo patients with Parkinson's disease. PLoS ONE 4(3). https://doi.org/10.1371/journal.pone.0004898 e4898.

Willemssen R, Muller T, Schwarz M, Hohnsbein J, Falkenstein M (2008) Error processing in patients with Parkinson's disease: the influence of medication state. J Neural Transm (Vienna) 115 (3):461–468. https://doi.org/10.1007/s00702-007-0842-1.

Wokke ME, Cleeremans A, Ridderinkhof KR (2017) Sure I'm sure: prefrontal oscillations support metacognitive monitoring of decision making. J Neurosci 37(4):781–789.

Wokke ME, Achoui D, Cleeremans A (2020) Action information contributes to metacognitive decision-making. Sci Rep 10 (1):1–15.

Yeung N, Botvinick MM, Cohen JD (2004) The neural basis of error detection: conflict monitoring and the error-related negativity. Psychol Rev 111(4):931–959. https://doi.org/10.1037/0033-295X.111.4.931.

Yeung N, Summerfield C (2012) Metacognition in human decision-making: confidence and error monitoring. Philos Trans Royal Soc B: Biol Sci 367(1594):1310–1321.

(Received 30 October 2020, Accepted 19 January 2021)
(Available online 29 January 2021)