REVIEW

Do sensorimotor perturbations to standing balance elicit an error-related negativity?

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Abstract

Detecting and correcting errors is essential to successful action. Studies on response monitoring have examined scalp ERPs following the commission of motor slips in speeded-response tasks, focusing on a frontocentral negativity (i.e., error-related negativity or ERN). Sensorimotor neurophysiologists investigating cortical monitoring of reactive balance recovery behavior observe a strikingly similar pattern of scalp ERPs following externally imposed postural errors, including a brief frontocentral negativity that has been referred to as the balance N1. We integrate and review relevant literature from these discrepant fields to suggest shared underlying mechanisms and potential benefits of collaboration across fields. Unlike the cognitive tasks leveraged to study the ERN, balance perturbations afford precise experimental control of postural errors to elicit balance N1s that are an order of magnitude larger than the ERN and drive robust and well-characterized adaptation of behavior within an experimental session. Many factors that modulate the ERN, including motivation, perceived consequences, perceptual salience, expectation, development, and aging, are likewise known to modulate the balance N1. We propose that the ERN and balance N1 reflect common neural activity for detecting errors. Collaboration across fields could help clarify the functional significance of the ERN and poorly understood interactions between motor and cognitive impairments.

KEYWORDS
balance, ERN, error-related negativity, N1, perturbation, sensorimotor

1 | ERROR-RELATED NEGATIVITY

For nearly three decades, psychophysiologists have studied a specific neural response to error commission, referred to as the error-related negativity (ERN or Ne; Falkenstein, Hohnsbein, & Hoormann, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993, 2018). The ERN is elicited when participants make errors (i.e., motor slips) in forced-choice speeded-response tasks. The most common tasks that have been used to elicit and study the ERN are variations on the flanker task, Stroop task, and go/no-go tasks (Meyer, Riesel, & Proudfit, 2013), which involve basic stimulus-response pairs that are verbally explained at the beginning of the task, for example, "When you see Stimulus A, press response Button 1." Although the tasks are relatively simple, participants make mistakes on a small percentage of trials. By having participants perform hundreds of trials while recording EEG activity, it is possible to evaluate ERPs time-locked to errors compared to correct responses. The ERN is observed as a sharp negative peak within the first 100 ms of the ERP following incorrect response commission at frontocentral EEG electrodes (Fz, FCz, or Cz; Gehring et al., 1993). Figure 1a shows the ERN evoked by errors in a flanker task from Marlin et al. (Marlin, Mochizuki, Staines, & McIlroy, 2014).
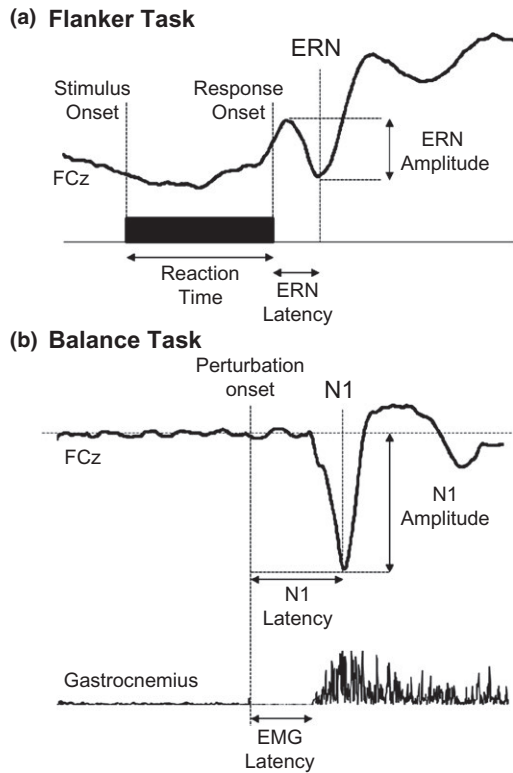
(a) Flanker Task

(b) Balance Task

FIGURE 1 Comparison of the error-related negativity (ERN) and balance N1, collected in the same recording session in $N = 11$ healthy young adults (8 female) from Marlin et al. (2014). (a) ERN evoked by errors in an arrow flanker task. (b) N1 evoked by sudden release of a cable supporting a portion of body weight from an upright leaning posture. Republished from Marlin et al. (2014), with permission from the *Journal of Neurophysiology*

The ERN appears to reflect activation of the anterior cingulate cortex and/or the supplementary motor area based on extracranial EEG (Dehaene, Posner, & Tucker, 1994; Gentsch, Ullsperger, & Ullsperger, 2009; Marlin et al., 2014; Miltner, Braun, & Coles, 1997), intracranial EEG (Bonini et al., 2014), fMRI (Badgaiyan & Posner, 1998; Carter et al., 1998; Hauser et al., 2014), and magnetoencephalography studies (Miltner et al., 2003). The ERN is thought to represent the activation of a generic neural system for error detection because it is relatively consistent across different tasks (Meyer et al., 2013; Riesel, Weinberg, Endrass, Meyer, & Hajcak, 2013) and responding limbs (Holroyd, Dien, & Coles, 1998). Theoretical and computational models suggest that the ERN reflects detection of errors, situations conducive to errors (Carter et al., 1998; Kerns et al., 2004; Ullsperger & von Cramon, 2001; van Veen, Cohen, Botvinick, Stenger, & Carter, 2001; Yeung, Botvinick, & Cohen, 2004), or unexpected events (Alexander & Brown, 2011) to recruit cognitive control to improve behavior (Holroyd & Coles, 2002; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Shackman et al., 2011; Ullsperger, Danielmeier, & Jocham, 2014). That is, theories of the ERN generally suggest it is functionally linked

to post-error adaptation. However, observable changes in behavior accompanying errors in the tasks most commonly used to study ERN are limited to less forceful entry of errors compared to correct responses, subsequent entry of the correct response on the same trial, slower reaction time on subsequent trials, and increased probability of responding correctly on the next trial (Dutilh et al., 2012; Gehring et al., 1993). Thus, behavioral changes include differences in error-related responses or post-error performance measures.

Although the amplitude of the ERN has been correlated with changes in behavior after errors (Gehring et al., 1993; Ullsperger et al., 2014), a number of experimental factors limit the ability to rigorously test adaptive hypotheses of the ERN. Primarily, reliance on subjects to sporadically commit errors limits experimental control over error frequency, timing, and sequencing. Additionally, discrete classification of responses as either overtly correct or erroneous limits the ability to observe continuous behavioral adaptation within subjects. Although some groups have begun to assess partial errors in the form of muscle activation in the nonresponding limb in trials with an overtly correct response (Spieser, van den Wildenberg, Hasbroucq, Ridderinkhof, & Burle, 2015), adaptation in these tasks is typically estimated as an increasing probability of an overtly correct response on subsequent trials (Gehring et al., 1993), rather than being directly measured in terms of incremental progress (e.g., skill acquisition toward the development of expertise within individuals; Shadmehr, Smith, & Krakauer, 2010). Given that accuracy often exceeds 90% in these simple tasks, the odds of a correct response following an error would be quite high even in the absence of behavioral adaptation. While it is possible to observe incremental changes in response latency across trials within individuals, it is unclear if behavioral changes such as post-error slowing after errors in speeded-response tasks actually reflect control-related processes or, rather, orienting responses to infrequent events (Dutilh et al., 2012; Notebaert et al., 2009; Wessel, 2018; Wessel & Aron, 2017). Further, whether such orienting responses increase (Houtman & Notebaert, 2013) or decrease (Botvinick, Braver, Barch, Carter, & Cohen, 2001) the likelihood of errors on subsequent trials depends on, and is thus confounded by, the duration of intertrial intervals (Jentzsch & Dudschig, 2009; Wessel, 2018). A more complex behavioral task providing better experimental control and the presence of robust behavioral adaptation could overcome these limitations to facilitate a more mechanistic investigation of the ERN in relation to post-error changes in behavior.
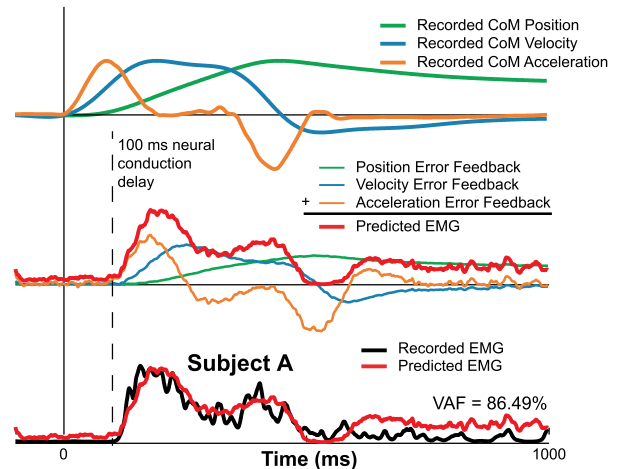
2 | BALANCE PERTURBATIONS AND THE BALANCE N1

Reactive balance recovery following externally imposed balance errors provides greater experimental control over

errors than tasks typically used to elicit the ERN. Reactive balance recovery can be evoked by a variety of physical perturbations imposing errors on whole body posture, including shove (Adkin, Quant, Maki, & McIlroy, 2006) or release (Mochizuki, Boe, Marlin, & McIlroy, 2010) perturbations of the upper torso, as well as tilts (Ackermann, Diener, & Dichgans, 1986) or translations of the floor during standing (Welch & Ting, 2009), walking (Dietz, Quintern, & Berger, 1985), or sitting (Mochizuki, Sibley, Cheung, Camilleri, & McIlroy, 2009; Staines, McIlroy, & Brooke, 2001). In contrast to cognitive paradigms that rely on subjects to sporadically commit errors, perturbation devices can be used to precisely control the type, frequency, extent, and sequencing of balance perturbations, which can be repeated across subjects (Adkin et al., 2006; Welch & Ting, 2008, 2009, 2014). In each case, a rapid and highly motivated motor reaction is necessary to prevent a fall or possible bodily harm. The earliest balance-correcting muscle activity, called the automatic postural response, is an involuntary behavior mediated by brainstem sensorimotor circuits (Carpenter, Allum, & Honegger, 1999; Jacobs & Horak, 2007), which can be predicted in fine detail from movement-based error trajectories, that is, the deviation of the body from the upright, standing position (Welch & Ting, 2008, 2009). In this way, balance recovery is an ecologically relevant error-correcting behavior that evokes error-related and error-correcting muscle activity. And, most importantly, balance perturbations evoke an error-related scalp ERP resembling the ERN, which has been referred to as the balance N1. Figure 1b shows the balance N1 evoked by sudden release of a cable supporting a portion of body weight from an upright leading posture from Marlin et al. (2014).

Motor reactions to balance perturbations have been well characterized as rapidly adapting, error-driven responses. Balance performance error can be measured by the position, velocity, and acceleration of the body's center of mass relative to the feet, which serve as three error signals that simultaneously evoke balance-correcting muscle activations (Figure 2a; Lockhart & Ting, 2007; Welch & Ting, 2008, 2009). Sensitivities to each of these error signals can vary independently and substantially within a range of solutions that are sufficient to generate forces to correct balance errors, and differences in these sensitivities can parsimoniously explain apparently complex differences in balance-correcting motor responses between individuals (Figure 2a,b,c; Welch & Ting, 2008, 2009). These error sensitivities can adapt on a trial-by-trial basis within an experimental session toward optimal solutions that can be predicted through physics (Welch & Ting, 2014). Such adaptation can also occur over motor rehabilitation, as demonstrated by an increase in sensitivity to velocity and position errors in cats when the sensory afferents encoding acceleration

(a) Kinematic errors define balance-correcting EMG

(b) Different error sensitivities explain subject differences
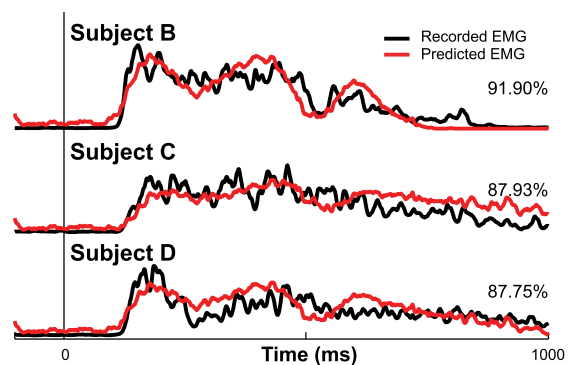
(c) Each error signal differentially biases corrective EMG
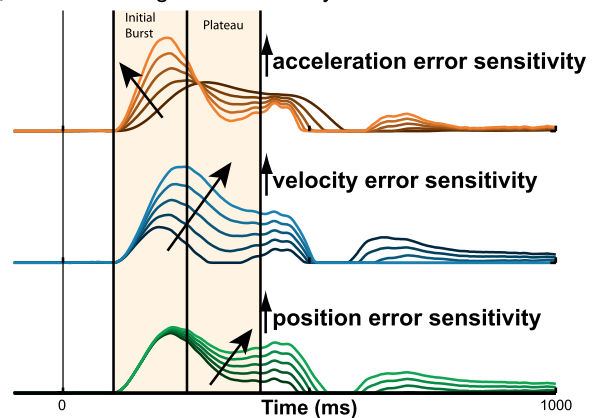
FIGURE 2 Kinematic error signals define the balance-correcting motor response to perturbation. (a) Recorded center of mass kinematics (top) are multiplied by subject- and muscle-specific error sensitivities, added together, clipped below zero, and delayed 100 ms (middle) to reconstruct recorded balance-correcting electromyogram (EMG) response (bottom). (b) Different kinematic error sensitivities can explain differences in balance-correcting EMG between subjects responding to the same perturbation. (c) Changes in sensitivity to acceleration error (top) primarily influence the initial burst of the balance-correcting response, whereas changes in sensitivity to velocity (middle) or position errors (bottom) influence later portions of the response due to the relative peak timings of the error signals. Republished from Welch and Ting (2008), with permission from the *Journal of Neurophysiology*

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error were damaged by pyridoxine overdose (Lockhart & Ting, 2007). The mechanisms underlying such changes in error sensitivities remain unclear, but a better understanding could facilitate rehabilitation of balance disorders.

In addition to error-related and error-correcting muscle activity, balance perturbations elicit error-related cortical activity resembling the ERN (Figure 1). Specifically, a frontocentral negativity called the balance N1 is evoked simultaneous to the balance-correcting muscle activity (Payne, Hajcak, & Ting, 2018). The balance N1 is a negative peak of cortical activity occurring between 100–200 ms after balance perturbation at frontal and central midline EEG electrodes (Fz, FCz, Cz; Marlin et al., 2014; Mierau, Hulsdunker, & Struder, 2015), with amplitudes large enough to observe on single trials (Mierau et al., 2015; Payne et al., 2018). The balance N1 has been localized to the supplementary motor area (Marlin et al., 2014; Mierau et al., 2015), but theories of its function are extremely limited. The balance N1 was initially thought to reflect sensory activity from balance perturbations (Dietz, Quintern, & Berger, 1984, 1985; Dietz, Quintern, Berger, & Schenck, 1985). However, the absence of the balance N1 when perturbations are predictable (Adkin et al., 2006) suggests that the balance N1 may represent an error signal similar to the ERN. In fact, many factors known to modulate the ERN, including motivation, perceived consequence, perceptual salience, expectation, development, and aging, are likewise known to modulate the balance N1 (see Section 3). If the balance N1 and ERN are manifestations of a common neural system for error detection, sensorimotor perturbations may present a more controllable experimental paradigm to study the relationship between errors, the action monitoring system, and subsequent changes in behavior (see Section 4).

3 | PARALLELS BETWEEN BALANCE N1 AND ERN

The previous sections defined the ERN and balance N1 as frontocentral negativities time-locked to an error event, which have largely overlapping scalp distributions (Figure 3) and sources localized to the medial frontal cortex (Marlin et al., 2014). In this section, we describe parallel outcomes of investigations of the balance N1 and the ERN to support the argument that these brain responses reflect similar functions of the action monitoring system, and in the following section, we conclude with the suggestion that collaboration across fields could overcome barriers to progress in both fields. One apparent contrast is that the ERN is typically quantified within the first 100 ms of the response-locked ERP waveform, whereas the balance N1 is typically quantified in the second 100 ms of the stimulus-locked ERP waveform. However, in tasks that elicit the ERN, the onset of muscle activity
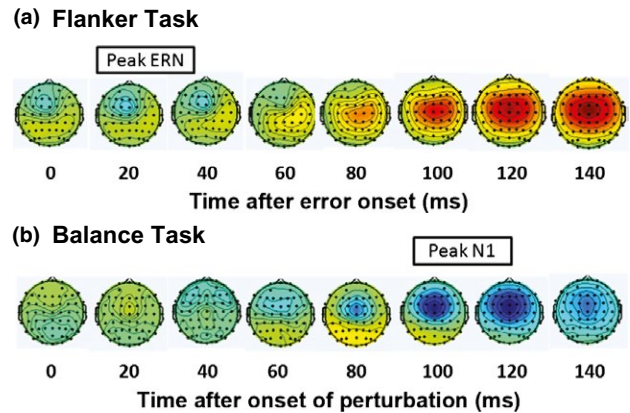

FIGURE 3 Scalp distributions of the error-related negativity (ERN) and balance N1, collected in the same recording session in $N = 11$ healthy young adults (8 female) from Marlin et al., 2014. (a) Scalp distribution of the ERN evoked by errors in an arrow flanker task. (b) Scalp distribution of the N1 evoked by sudden release of a cable supporting a portion of body weight from an upright leaning posture. Republished from Marlin et al. (2014), with permission from the *Journal of Neurophysiology*

associated with the erroneous response entry can be observed 100 ms before the response button is pressed (Spieser et al., 2015). Thus, if the ERN were quantified relative to the onset of the error event rather than completion of the error event, its timing would be more aligned with the timing of the balance N1 relative to the onset of perturbation acceleration. In other words, both the balance N1 and ERN could be equivalently quantified within the second 100 ms relative to the onset of an error, whether the error is internally generated, as in the case of the ERN, or externally applied, as in the case of the balance N1. Much like the ERN, which displays a similar scalp distribution whether responses are entered by the hand or foot (Holroyd et al., 1998), or even by the eyes (Endrass, Reuter, & Kathmann, 2007; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; Van't Ent & Apkarian, 1999), the balance N1 displays a similar scalp distribution regardless of whether perturbations are delivered during standing or sitting (Mochizuki, Sibley, Cheung, Camilleri, & McIlroy 2009), consistent with a generic system for error detection.

Larger and more intense balance perturbations elicit a larger balance N1 response (Mochizuki et al., 2010; Payne et al., 2018; Staines et al., 2001). Likewise, the ERN increases in amplitude with the extent of an error, such as when an error is committed with both the wrong finger and the wrong hand compared to errors committed with either the wrong finger or the wrong hand (Bernstein, Scheffers, & Coles, 1995). The balance N1 also decreases in amplitude when sensory input is partially blocked by tendon vibration (Staines et al., 2001) or when sensory input is naturally suppressed during walking (Dietz, Quintern, & Berger, 1985). These data are similar to observed reductions in the amplitude of the ERN in a visual two-choice reaction task under degraded stimulus conditions

(Scheffers & Coles, 2000). In contrast to the balance N1 studies that directly altered the sensory experience of the error, the degraded visual stimulus condition in Scheffers and Coles altered the representation of the error by making subjects more uncertain about the identity of the appropriate response. In either case, the error-related cortical response was influenced by altering the perceptual intensity of the information necessary to compare the actual and desired events, thereby implicating some dependency on the sensory manifestation of the error-related stimulus.

The balance N1 and the ERN both scale in amplitude with the perceived consequence of an error. The balance N1 is increased in amplitude when a balance failure would be more significant, such as when the participant is perturbed at an elevated height (Adkin, Campbell, Chua, & Carpenter, 2008; Sibley, Mochizuki, Frank, & McIlroy, 2010). In this case, the externally applied force of the perturbation is the same, but the change in context from standing at ground level to standing at the edge of an elevated platform increases the perceived consequences of a possible balance failure and increases the amplitude of the balance N1. The ERN likewise increases when an error is perceived as more significant, for example, when an error incurs a higher monetary loss (Hajcak, Moser, Yeung, & Simons, 2005; Pailing & Segalowitz, 2004a) or when the participant is informed that their performance is being evaluated and judged (Hajcak et al., 2005; Kim, Iwaki, Uno, & Fujita, 2005). Interestingly, the elevated height perturbation context also increased self-reported level of anxiety and worry about falling (Adkin et al., 2008), which aligns with findings of higher ERN amplitudes in individuals with greater general anxiety and worry (Hajcak, McDonald, & Simons, 2003), which may modulate the perceived consequences of errors. These studies suggest that the balance N1 and ERN depend not only on the sensory phenomena of errors, but also on the cognitive valuation of the perceived consequences of balance perturbations and errors, respectively.

The balance N1 is reduced in amplitude when a participant's attention is diverted from a balance task by performing a simultaneous visuomotor tracking task (Quant, Adkin, Staines, Maki, & McIlroy, 2004) or visual working memory task (Little & Woollacott, 2015). Likewise, ERN amplitude is reduced when a participant's attention is diverted from the primary speeded-response task by simultaneous performance of an interleaved visual working memory task (Klawohn, Endrass, Preuss, Riesel, & Kathmann, 2016; Maier & Steinhauser, 2017) or while simultaneously listening to a series of numbers for a particular sequence (Pailing & Segalowitz, 2004b). These studies suggest that the amplitude of both the balance N1 and the ERN are modulated by the availability of cognitive resources.

When a balance perturbation is predictable in timing, magnitude, and direction, the balance N1 amplitude is substantially reduced (Mochizuki et al., 2010) or even absent (Adkin et al., 2008, 2006). Along similar lines, an unexpected balance perturbation in a different direction following a series of predictable perturbations elicits a large balance N1 (Adkin et al., 2008, 2006). Additionally, self-initiated balance perturbations elicit smaller balance N1s than those initiated by an experimenter (Dietz, Quintern, Berger, & Schenck, 1985; Mochizuki, Sibley, Cheung, & McIlroy, 2009; Mochizuki, Sibley, Esposito, Camilleri, & McIlroy, 2008). These data suggest that the balance N1 scales with predictability and are consistent with observations that ERN amplitude is larger when errors are less frequent and therefore more unexpected (Holroyd & Coles, 2002; Santesso, Segalowitz, & Schmidt, 2005). Collectively, these studies suggest a parallel potentiation of ERN when errors are more infrequent and unexpected and balance N1 when perturbations are unexpected.

The balance N1 evoked by whole body perturbations increases in amplitude from early childhood (Berger, Quintern, & Dietz, 1987) and declines in amplitude with old age (Duckrow, Abu-Hasaballah, Whipple, & Wolfson, 1999). This developmental trajectory is similar to that of the ERN, which has also been observed to increase in amplitude from childhood to adolescence, plateauing in adulthood (Ladouceur, Dahl, & Carter, 2007; Santesso & Segalowitz, 2008; Tamnes, Walhovd, Torstveit, Sells, & Fjell, 2013; Wiersema, van der Meere, & Roeyers, 2007), and declining with old age (Beste, Willemssen, Saft, & Falkenstein, 2009; Nieuwenhuis et al., 2002). However, increases in ERN amplitude from childhood to adulthood are often confounded by a reduction in error frequency (Ladouceur et al., 2007; Santesso & Segalowitz, 2008; Wiersema et al., 2007), making it unclear whether the increase in ERN amplitude from childhood to adulthood is due to age or increased unexpectedness of errors due to performance improvement. However, when comparing younger adults to older adults, errors continue to become less frequent while the ERN amplitude declines (Beste et al., 2009; Nieuwenhuis et al., 2002), suggesting there are indeed changes in ERN amplitude with age that do not depend on changes in error rate.

Time-frequency analyses of the ERN (Luu, Tucker, & Makeig, 2004) and balance N1 (Peterson & Ferris, 2018; Varghese et al., 2014) have been used to suggest that both of these ERPs may reflect a transient synchronization of theta frequency (4–7 Hz) brain activity. However, in simulated data sets, such analyses are unable to distinguish synchronization of oscillatory components from discrete component peaks (Yeung, Bogacz, Holroyd, Nieuwenhuis, & Cohen, 2007). The extent to which these ERPs relate to theta frequency brain activity observed in other contexts remains unclear. Although theta power and ERN amplitudes are similarly modulated by novelty, conflict, and error within subjects (Cavanagh, Zambrano-Vazquez, & Allen, 2012), theta power and ERN amplitude can be relatively independent individual difference

measures (Cavanagh, Meyer, & Hajcak, 2017). Whether the balance N1 evoked by postural perturbations is mechanistically related to continuous theta frequency brain activity observed in continuous balance tasks (Hulsdunker, Mierau, Neeb, Kleinoder, & Struder, 2015; Sipp, Gwin, Makeig, & Ferris, 2013) remains unclear but presents another interesting area of future investigation and integration.

On the basis of these parallels, we propose that the balance N1 and the ERN reflect similar functions of the action monitoring system and suggest that balance and other sensorimotor perturbation paradigms could be leveraged to probe neural mechanisms of error detection and behavioral adaptation. While anatomical studies have suggested that the ERN reflects activity within a cortical node of cortico-basal ganglia-thalamocortical circuits (Ullsperger et al., 2014), the balance N1 may likewise reflect activity within a cortical node of parallel or overlapping cortico-basal ganglia-thalamocortical circuits, which are known to be highly parallel in nature and are suspected to perform similar functions based on detailed anatomical studies in animals (Alexander, DeLong, & Strick, 1986). In particular, the possible differences in localization of the ERN to the anterior cingulate cortex and the balance N1 to the supplementary motor area (Marlin et al., 2014) provides further support for the possibility that these ERPs may arise from the aforementioned parallel circuits, as the anterior cingulate cortex represents the cortical node of the so-called cognitive loop of the cortico-basal ganglia-thalamocortical circuit, and the supplementary motor area represents the cortical node of the so-called motor loop of the cortico-basal ganglia-thalamocortical circuit (Alexander et al., 1986). However, these differences in localization may also be influenced by differences in overlap between the ERN or balance N1 and associated stimulus-locked visual or proprioceptive and vestibular ERPs, respectively (Hajcak, Vidal, & Simons, 2004). If the balance N1 is a perturbation-elicited ERN, the experimental control and robust adaptation within balance paradigms could be leveraged to test adaptive hypotheses of the ERN, and theoretical and computational models of the ERN could be leveraged to design mechanistic investigations of the balance N1. Further, collaboration across fields could reveal interactions between motor and cognitive impairments as well as cross-modal and synergistic benefits seen in combined motor and cognitive rehabilitation interventions.

4 | SUMMARY AND FUTURE DIRECTIONS

Detecting and correcting errors is essential to successful behavior. By error, we refer to any deviation from a desired or expected goal or bodily state, which can be recognized by the nervous system as the impetus to modify behavior to achieve the desired state or goal. Although a perturbation does not reflect commission of a motor error, it produces a deviation from the desired upright posture that must be rapidly detected and corrected to prevent bodily harm. In this way, we believe that balance perturbations recruit many of the same control processes that are recruited by commission of motor errors, which is supported by a range of parallel influences on scalp ERPs described in the preceding section. Because of the similarities between error-correcting motor responses to balance perturbations and error-correcting motor responses in perturbations to the arms during voluntary movement (Crevecoeur & Kurtzer, 2018), it is possible that these cortical responses would generalize more broadly across sensorimotor control paradigms.

In contrast to cognitive paradigms that rely on subjects to sporadically commit errors, perturbation devices can be used to precisely control the type, frequency, extent, and sequencing of errors, which can be repeated both within and across subjects (Adkin et al., 2006; Welch & Ting, 2008, 2009, 2014) across a wide range of ages (Berger et al., 1987; Duckrow et al., 1999). Prediction or expectation errors can also be controlled in sensorimotor paradigms by altering verbal instructions or sequencing of perturbations, for example, by providing a series of perturbations that are predictable in timing, direction, and magnitude and manipulating any of these dimensions on selected "catch" trials (Adkin et al., 2006; Welch & Ting, 2014). In addition, it is also possible to examine outcome errors (e.g., by instructing a subject to recover balance without stepping in perturbations large enough to guarantee stepping reactions; Chvatal & Ting, 2012; McIlroy & Maki, 1993). It is therefore possible to leverage precise control over sensorimotor errors to experimentally isolate factors for a more detailed understanding of how each aspect of errors influences cortical activity. In turn, leveraging knowledge of the ERN to design sensorimotor perturbation experiments could be a major step toward identifying the functional role of cortical action monitoring on adaptation of sensorimotor behaviors.

Given the parallel outcomes of investigations of the ERN and the balance N1, several questions arise from the ERN literature that have yet to be tested of the balance N1. First, if the balance N1 and ERN share neural circuitry, then drugs that influence the ERN should also influence the balance N1. In particular, do dopamine agonists and antagonists, which increase (de Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004) and decrease (de Bruijn, Sabbe, Hulstijn, Ruigt, & Verkes, 2006; Zirnheld et al., 2004) the amplitude of the ERN in healthy young adults, likewise influence the amplitude of the balance N1? Second, if the balance N1 and ERN share neural circuitry, then disorders that influence the ERN should also influence the balance N1. In particular, do individuals with Parkinson's disease who present with reduced ERN amplitudes (Seer, Lange, Georgiev, Jahanshahi, & Kopp, 2016) likewise show reduced balance N1s? And could this relate to

balance impairment in Parkinson's disease? Do individuals with obsessive-compulsive disorder who present with larger ERN amplitudes (Endrass et al., 2010; Klawohn, Riesel, Grutzmann, Kathmann, & Endrass, 2014) also display larger balance N1s? And could this relate to reduced postural sway in obsessive-compulsive disorder (Kemoun, Carette, Watelain, & Floirat, 2008)? Further, can reward and punishment, which can cause a lasting increase in ERN amplitude (Riesel, Weinberg, Endrass, Kathmann, & Hajcak, 2012), likewise cause lasting changes in the balance N1 amplitude? Or, if the balance N1 and ERN share underlying circuitry, can the effect of punishment on ERN amplitude cross domains to increase the balance N1 in the absence of punishment in balance tasks? While such correlational investigations are interesting and may provide insight into the neural underpinnings of performance monitoring, a much greater challenge will be leveraging such insight to benefit people with altered performance monitoring.

ERN amplitude is characteristically altered in multiple patient populations that seek rehabilitation from persistent pathological behaviors and thought processes, including those with obsessive-compulsive disorder (Gehring, Himle, & Nisenson, 2000; Klawohn et al., 2014), generalized anxiety disorder (Weinberg, Klein, & Hajcak, 2012; Weinberg, Olvet, & Hajcak, 2010), substance abuse (Franken, van Strien, Franzek, & van de Wetering, 2007; Schellekens et al., 2010), old age, Parkinson's disease (Seer et al., 2016), and Huntington's disease (Beste et al., 2009). While such between-population differences can be leveraged as a risk factor to predict the development of certain neuropsychiatric disorders (Meyer, Hajcak, Torpey-Newman, Kujawa, & Klein, 2015), attempts to leverage knowledge about altered cortical action monitoring and error processing to devise treatment strategies are just beginning. Potential rehabilitation strategies targeting the action monitoring system include, but are not limited to, strategic reorienting of attention from overvalued errors in individuals with obsessive-compulsive disorder (Klawohn et al., 2016) or anxiety (Waters et al., 2018) or toward undervalued or unrecognized errors in individuals with Parkinson's disease, habituation of maladaptive error responses or training of alternative behavioral responses to compete with pathological behaviors (Jacoby & Abramowitz, 2016), or targeted noninvasive electrical stimulation of the action monitoring system (Bellaiche, Asthana, Ehlis, Polak, & Herrmann, 2013; Reinhart & Woodman, 2014). A better understanding of the action monitoring system could therefore be advantageous for treating a range of neuropsychiatric disorders and could even extend into balance rehabilitation if action monitoring spans behavior more broadly. Although the brainstem-mediated involuntary balance-correcting motor responses can adapt rapidly within a single experimental session in healthy young adults (Horak & Nashner, 1986; Welch & Ting, 2014), understanding brain involvement is

critical for rehabilitation of balance recovery behavior in individuals with balance impairments due to Parkinson's disease (Grimbergen, Munneke, & Bloem, 2004), cerebellar dysfunction (Horak & Diener, 1994), or cognitive impairment (Herman, Mirelman, Giladi, Schweiger, & Hausdorff, 2010). Further, the rapid behavioral adaptation observable in balance paradigms could provide an experimental model in which to test hypotheses about error-driven changes in behavior more broadly and may prove particularly helpful in the context of comorbidities between motor and psychiatric disorders.

Finally, we suggest that collaboration across fields could clarify poorly understood interactions between motor, cognitive, and psychiatric disorders, leading to more integrated models of the ERN and balance N1, as well as potential treatment strategies. Many populations with altered error responses also display differences in balance behavior, including frequent comorbidities between anxiety disorders and balance disorders (Balaban, 2002; Balaban & Thayer, 2001; Bolmont, Gangloff, Vouriot, & Perrin, 2002; Yardley & Redfern, 2001), and substantially reduced postural sway in individuals with obsessive-compulsive disorder (Kemoun et al., 2008). Further, balance impairment is strongly associated with cognitive impairment in older adults with (Allcock et al., 2009; Mak, Wong, & Pang, 2014; McKay, Lang, Ting, & Hackney, 2018) and without (Camicioli & Majumdar, 2010; Gleason, Gangnon, Fischer, & Mahoney, 2009; Herman et al., 2010; Mirelman et al., 2012) Parkinson's disease, and rehabilitation interventions that simultaneously target cognitive engagement show greater improvement in motor function in healthy aging (Kraft, 2012; Wu, Chan, & Yan, 2016) and in Parkinson's disease (McKay, Ting, & Hackney, 2016; Petzinger et al., 2013) than interventions that target motor function alone. Collaboration across fields could provide new insight into these synergistic benefits of combined interventions and may help explain counterintuitive findings that balance training can ameliorate anxiety disorders (Bart et al., 2009) or that cognitive training can improve balance and gait (Smith-Ray et al., 2015), leading to the development of more integrated treatment strategies for comorbid motor, cognitive, and psychiatric disorders.

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