CONFLICT MONITORING, ERROR DETECTION, AND INHIBITION: BEHAVIOURAL AND ELECTROPHYSIOLOGICAL FEATURES

by

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Abstract

Event-related potentials of performance monitoring, including N2 (conflict monitoring), error-related negativity and error positivity (ERN and Pe; error monitoring), and P3 (inhibition) have been studied. However, conflict monitoring lacks a behavioural measure, and the functional significance of ERN, Pe, and P3 are debated. To address these issues, a behavioural measure of conflict monitoring was tested by subtracting the reaction time (RT) of a simple from a choice RT task to isolate conflict monitoring; the functions of error monitoring and inhibition were examined.

The RT difference correlated with the N2 area (longer conflict monitoring related to a larger N2). ERN and Pe areas were negatively and positively correlated with errors, respectively. P3 magnitude and onset were correlated with an inhibition index.

The new behavioural measure provides an accessible way to study conflict monitoring. Theories of conflict monitoring for ERN, error awareness for Pe, and inhibition for P3 were replicated and extended.

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Introduction

Overview

Performance monitoring is a set of cognitive processes that includes conflict monitoring, error processing, and inhibition. Each of these processes have been studied with an electrophysiological approach, and have been linked with the N2 (conflict monitoring), error-related negativity (ERN; error monitoring), error positivity (Pe; awareness and/or affective processing of errors), and P3 (inhibition). However, the links between the electrophysiological components and the behavioural measures are still not unequivocally established, and some theories continue to be debated. For instance, conflict monitoring (N2 component) does not have an established behavioural measure, and whether the Pe functions as an index of error awareness or affective processing is not entirely clear. The theories and debates surrounding these issues are discussed below.

In an attempt to address these issues, the N2 was correlated to a behavioural index of conflict monitoring; this index was the difference in performance time between a simple (one response) and forced choice (two responses) reaction time tasks, which parsed out the unique component of conflict monitoring. The magnitude of the ERN and Pe were correlated with actual errors, estimated errors, the difference between the two, and frustration with errors in order to elucidate their roles in error detection, error awareness, and affective processing. Lastly, the P3 was related to indices of inhibition (and other variables, including conflict monitoring and subjective performance measures) to confirm its functional role in inhibition.

Performance Monitoring: Behavioural and Cognitive Aspects

Background. Performance monitoring is a multifaceted aspect of cognition that monitors the competition between multiple potential responses to a given stimulus (e.g., pressing the correct button on an elevator), the inhibition of inappropriate responses, and the detection and correction of erroneous responses. Among the components of performance monitoring, error monitoring and inhibition have the richest research history, though the latter is typically studied on its own, rather than in the context of performance monitoring.

In experimental settings, performance monitoring is typically probed with a variety of RT tasks, such as the Erikson flanker task[1], Go/NoGo task[2], and the Stop Signal Task (SST; please refer to the methods for description of study-related tasks). Outcome measures include rates of correct hits and errors, RT, and post-error RT and accuracy. The latter two indices refer to the transient slowing of responses and increase in performance accuracy following the commission of an error. These post-error behaviours are thought to reflect an orienting reaction in response to an unexpected and motivationally salient event, which subsequently increases selective attention to enhance processing of task-relevant stimuli while suppressing irrelevant stimuli (Danielmeier & Ullsperger, 2011; Notebaert et al., 2009).

Error monitoring. Early studies on performance monitoring focused on behavioural measures of error detection and correction in reaction time (RT) tasks, invariably finding that healthy individuals are able to detect and indicate most, though not all errors they committed via a button press (Rabbitt, 1966, 1967, 1968, 2002). However, this is a slow process and accuracy is

[1] The Eriksen flanker task is a test of cognitive inhibition, in which target stimuli are flanked by distractor stimuli. Different targets require unique responses, and the distractors are either congruent with the target (indicating the same type of response) or incongruent (indicating different responses). Incongruent distractors are more difficult to ignore as they are discordant with the target stimuli.

[2] The Go/NoGo task is a test of motor inhibition/sustained attention. The majority of stimuli require a response, and some infrequent stimuli require responses to be inhibited too. Due to the rare presentation of the inhibition stimuli, they are relatively difficult to suppress.

susceptible to distracting stimuli. Reporting on error commissions was found to take an average of 700 ms, and error detection rates dropped from 79% to 56% when subsequent stimuli were presented quickly (150 ms) after erroneous responses, despite being asked to ignore the successive stimuli (Rabbitt, 2002). In contrast, error correction is a fast and apparently automatic response. Participants are quick to provide correct responses following errors, doing so within approximately 250 ms (Rabbitt, 2002). Interestingly, participants provide expedient corrections even when they are not asked to do so (Maylor & Rabbitt, 1987; Rabbitt & Rodgers, 1977). Rabbit and colleagues postulated that errors stem from incomplete perceptual information processing, and the consequent fast error corrections occur due to continued stimulus processing (Rabbitt, Cumming, & Vyas, 1978; Rabbitt & Vyas, 1981). This carries the implication that longer stimulus processing should result in improved error correction; this was confirmed by the finding that longer stimulus durations corresponded with higher rates of error correction (Rabbitt & Vyas, 1981).

Inhibition. In the realm of neuropsychology, inhibition is typically categorized as a standalone executive function (Barkley, 1997; Dempster & Corkill, 1999)[3], and is classically categorized into either motor or cognitive inhibition (Bari & Robbins, 2013; Nigg, 2000). Motor inhibition refers to the withholding of an overt behaviour, while cognitive inhibition typically refers to the stopping or overriding of mental contents and attentional processes (MacLeod, Gorfein, & Colin, 2007). Motor inhibition is easier to study as it involves overt behaviours (or lack thereof). It is studied using tasks such as the Go/NoGo and the SST, for which motor responses have to be withheld when prompted (e.g., a "NoGo" stimulus on the Go/NoGo, or the stop sound on the SST). The primary difference between these two tasks is the timing of the stop signal. On the

[3]However, there are others who view inhibition as the product of other executive functions, rather than a specific process (Alderson, Rapport, Hudec, Sarver, & Kofler, 2010; Friedman et al., 2008).

Go/NoGo, the stop signal is presented concurrently, or in place of, the go signal, while the stop signal on the SST is presented after the go stimulus. Given the different timing presentations of the stop signals, the Go/NoGo and SST invoke "action restraint" and "action cancellation." The former refers to withholding a planned response and the latter involves the suppression of an ongoing response (Schachar et al., 2007). Both paradigms have also been implemented with several patient populations (e.g., attention-deficit/hyperactivity disorder [ADHD], obsessive-compulsive disorder [OCD]) to demonstrate differences in inhibition abilities (Lipszyc & Schachar, 2010; Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014).

Cognitive inhibition, on the other hand, is more difficult to study as it does not involve manifest behaviours. It can be conceptualized as the suppression of cognitive processes to prevent or limit performance (MacLeod et al., 2007). It is frequently probed using the Stroop task (please refer to the methods for a description of this task) or, alternately, mental counting of some stimuli, but not others (e.g., counting Go, but not NoGo stimuli) or mentally imagining performing or withholding responses to stimuli (Nigg, 2000). As with motor inhibition, cognitive inhibition tasks (e.g., Stroop) have been used to identify cognitive impairment in psychopathologies (e.g., Westerhausen, Kompus, & Hugdahl, 2011).

Performance Monitoring: Event-Related Potentials

Many electrophysiological measures of performance monitoring have been established in research. Each of the four aspects of performance monitoring have been paired with event-related potentials (ERPs) thought to represent these aspects. An ERP reflects the voltage changes (either negative or positive depending on the ERP) generated by multiple action potentials in response to a stimulus or cognitive process (Blackwood & Muir, 1990). Error-related negativity,

N2, error positivity, and P3 are thought to represent error detection, conflict monitoring, error awareness, and inhibition, respectively.

N2. The N2 ERP is a negative voltage deflection occurring 250-350 ms following stimulus presentation, and occurring primarily at FCz (Folstein & Van Petten, 2008; Van Veen & Carter, 2002; Yeung, Botvinick, & Cohen, 2004), a frontal midline electrode according to the International 10-20 system (Klem, Lüders, Jasper, & Elger, 1999; please refer to the methods section for a description of the 10-20 system). Studies using electroencephalography (EEG) source localization (Ladouceur, Dahl, & Carter, 2007; Van Veen & Carter, 2002; Yeung et al., 2004), functional magnetic resonance imaging (fMRI; Carter et al., 1998), and intracranial stimulation (Wang, Ulbert, Schomer, Marinkovic, & Halgren, 2005) have identified the caudal anterior cingulate cortex (ACC) as the likely locus of the N2. Until the early 2000s, this ERP was considered an index of inhibition (Kok, 1986; Kopp, Rist, & Mattler, 1996). However, in recent years it has become more accepted as an index of conflict monitoring. Yeung and colleagues (2004) proposed that conflict between competing responses prior to the actual response produces the N2. This theory was tested through a computer simulation paradigm that produced a negative ERP component, identified as the N2, on correct trials prior to the response. This finding was replicated with human participants; a negative deflection was observed 88 ms prior to responses at the FCz electrode. Further, N2 amplitude and latency increased with slower RT, and was not apparent for trials with the fastest RTs, suggesting that amplitude and latency are related to the time required to resolve conflicting responses. This pattern also clarified previous findings that the N2 was larger on incongruent versus congruent trials on Flanker tasks, because completion of incongruent trials tended to be slower than congruent ones (Heil, Osman, Wiegelmann, Rolke, & Henninghausen, 2000; Kopp et al., 1996; Liotti, Woldorff, Perez, & Mayberg, 2000). Smith,

Smith, Provost, and Heathcote (2010) provided further evidence that the N2 reflects conflict monitoring rather than inhibition processes. This study scrutinized the N2 and P3 ERPs on expected and unexpected trials on the Go/NoGo (i.e., trial n is expected when it is the same type as n-1, and unexpected when they are different). An inhibition account would predict that inhibition is more difficult on unexpected inhibition trials, and would be reflected by larger N2 amplitudes on unexpected NoGo versus Go trials. Conversely, a larger N2 magnitude on all unexpected trials (i.e., both Go and NoGo) would be congruent with the conflict monitoring theory, as this would be indicative of greater pre-response conflict regardless of the subsequent successful/erroneous response. The conflict monitoring model was found to be supported, as N2 amplitudes on unexpected trials did not differ between Go and NoGo trials.

Additional research has indicated that the magnitude of N2 is related to the degree of conflict incited by the stimuli. Closer flanker proximity to the target stimulus is associated with greater N2 (Danielmeier, Wessel, Steinhauser, & Ullsperger, 2009), as is greater incongruity between stimuli (Forster, Carter, Cohen, & Cho, 2011; however, see Yeung et al. (2004) for an alternate explanation relating to RT), though the brightness of the target stimulus does not influence N2 magnitude (Yeung, Ralph, & Nieuwenhuis, 2007). Interestingly, opposing findings on N2 amplitude as a function of conflict frequency have been observed. Whereas Grützmann, Riesel, Klawohn, Kathmann, and Endrass (2014) found that higher rates of conflict corresponded with greater N2 amplitudes, Clayson and Larson (2011) found the opposite association. However, there were key differences in their task designs and analyses. Grutzmann and colleagues manipulated conflict frequency by changing the rate of incompatible flanker trials between blocks, ranging from 25%-75%. In contrast, Clayson and Larson maintained a constant incompatible trial rate of 55%, but implemented two versions of the flanker task with either

always-matching or never-matching adjacent trials (i.e., successive trials were either incompatible-incompatible or incompatible-compatible). Repetition habituation effects on N2 amplitudes were observed for the matching trials version, likely reflecting a priming effect. Lastly, N2 amplitude appears to positively correlate with RT (Beste, Saft, Andrich, Gold, & Falkenstein, 2008; Yeung et al., 2004; Yeung & Nieuwenhuis, 2009), though others have only observed this relation within incongruent flanker trials (Larson, South, & Clayson, 2011).

ERN. Error-related negativity (ERN) is among the most studied ERPs of performance monitoring. This ERP is typically observed 50-100 ms following an erroneous response at the frontal midline electrode, FCz. Akin to the N2, the neural generator of ERN is hypothesized to be the caudal ACC as evidenced by studies using EEG source localization (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Holroyd, Dien, & Coles, 1998), magnetoencephalography (MEG; Miltner et al., 2003), intracranial stimulation (Brazdil, Roman, Daniel, & Rektor, 2005), multimodal imaging (Debener et al., 2005; Dehaene, Posner, & Tucker, 1994; Van Veen & Carter, 2002), and a neuropsychological study of patients with ACC lesions who exhibited diminished ERN amplitudes compared with healthy individuals (Stemmer, Segalowitz, Witzke, & Schönle, 2004). Task parameters and performance can influence the amplitude of the ERN, such as greater amplitude when fewer errors are committed (Amodio, Jost, Master, & Yee, 2007; Amodio, Master, Yee, & Taylor, 2008; Gehring et al., 1993; Hajcak, McDonald, & Simons, 2003), and when errors have greater emotional salience due to negative feedback (Endrass et al., 2010).

Despite being one of the most ubiquitously studied conflict ERPs, multiple hypotheses regarding its mechanism and functional significance exist, none of which provide a fully comprehensive model. As of yet, no consensus has been reached.

The mismatch theory was an early model of ERN, theorizing that the ERN indicates a mismatch between intended and actual outcomes (Bernstein, Scheffers, & Coles, 1995; Coles, Scheffers, & Holroyd, 2001; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991). This model predicts that the magnitude of ERN should correspond to the degree of discordance between the correct and incorrect responses, which has found some support in the literature (Bernstein et al., 1995; Falkenstein, Hohnsbein, & Hoormann, 1995; M. Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996).

An extension of the theory is the reinforcement learning model of ERN, stemming primarily from non-human animal and pharmacological research. This model posits that the basal ganglia monitors both external (environmental stimuli) and internal (self-generated actions) information and compares them to expected outcomes (Holroyd & Coles, 2002). The primary neurotransmitter implicated in this process is dopamine (DA), which is transmitted along pathways originating in subcortical structures. Of relevance are the mesolimbic and mesocortical pathways that originate from the ventral tegmental area (VTA) and project to limbic (e.g., nucleus accumbens) and frontal (e.g., dorsolateral prefrontal cortex) regions. The mesolimbic pathway plays an important role in reinforcement learning and motivation, and the mesocortical pathway is thought to be involved in cognitive control and affective processes (Meyer & Quenzer, 2013).

In support of the reinforcement learning model, the basal ganglia has been demonstrated to induce an increase and decrease in phasic mesencephalic DA activity following better or worse than expected outcomes, respectively (Barto, 1995; Houk, Adams, & Barto, 1995; Schultz, 2002). Decreased midbrain DA activity following unexpected poor outcomes is proposed to disinhibit ACC activity, manifested by the ERN. This process of detecting errors can influence

learning by evaluating rewarding and non-rewarding outcomes, and consequently modifying behaviour (Barto, 1995; Montague, Dayan, & Sejnowski, 1996; Schultz, 2002). Evidence for the link between ERN and dopaminergic activity comes from pharmacological studies demonstrating that the DA agonist D-amphetamine increases the magnitude of ERN (de Bruijn, Sabbe, Hulstijn, Ruigt, & Verkes, 2006), while haloperidol, a DA antagonist decreases ERN magnitude (de Bruijn et al., 2006; Zirnheld et al., 2004). The finding that antidepressant and anxiolytic medication with no DA receptor affinity do not influence ERN amplitudes obviates the counterargument that general psychoactive medication can influence ERN (de Bruijn et al., 2006; Stern et al., 2010). These medications included selective serotonin/norepinephrine reuptake inhibitors (primarily increase serotonin or norepinephrine levels), benzodiazepines (primarily increases gamma-aminobutyric [GABA] activity) and tricyclic antidepressants (primarily increase serotonergic and noradrenergic activity). Of note, Stern and colleagues included a few patients taking buproprion and methylphenidate, both of which have varying actions on DA receptors (particularly methylphenidate; buproprion only has a weak DA action). Nevertheless, these medications did not appear to influence ERN, though the small number of patients taking them (four taking buproprion, one taking methylphenidate) precluded strong conclusions. A limitation of the mismatch and the subsequent reinforcement learning theories is that they cannot explain why errors occur even though the mental representation of the correct stimulus is present when the error is committed.

The conflict monitoring theory accounts for some of the shortcomings of the above theories by emphasizing response conflict rather than error detection. Specifically, this model predicts that conflict between potential responses is monitored by the ACC, and that conflict between erroneous and correcting responses is just one instance of response conflict monitoring,

manifesting as ERN (Yeung et al., 2004). In these instances, both correct and incorrect response schemas are coactivated during stimulus processing, but the incorrect response ultimately crosses the response threshold and is executed. However, continued stimulus processing maintains activation of the latent correct response and the consequent conflict with the executed incorrect response (Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988; Rabbitt & Vyas, 1981; Yeung et al., 2004). Thus, the ERN is apparent following erroneous responses, up until the error is corrected (Carter et al., 1998; Van Veen & Carter, 2002; Van Veen, Cohen, Botvinick, Stenger, & Carter, 2001; Van Veen, Holroyd, Cohen, Stenger, & Carter, 2004). As with the N2, this theory was thoroughly examined by Yeung and colleagues (2004) in their seminal study using five computer simulation ERP paradigms, and replicated with human behavioural data. Through these simulations, Yeung and colleagues demonstrated that: the N2 (pre-response ERP) and ERN (post-error response ERP) are separate components of response conflict resolution (simulation 1); erroneous responses on congruent versus incongruent trials are associated with larger ERNs due to stronger activation of the unambiguously correct response schema and the subsequent competition it provides (simulation 2); emphasizing accuracy over speed results in larger ERNs (versus emphasizing speed over accuracy) due to an augmented activation of correcting responses, presumably due to greater attention devoted to avoiding errors (simulation 3); increasing the frequency of error-inducing (i.e., incongruent) trials is associated with larger ERNs due to a proclivity to quickly and robustly activate the correcting responses through its frequent activation (simulation 4); and error force is negatively correlated with ERN magnitude, meaning that stronger activation of the erroneous response decreases the likelihood of the correcting response to reach threshold activation and produce conflict. Other studies have supported this model by demonstrating that greater competition between responses corresponded

with larger (Botvinick, Braver, Barch, Carter, & Cohen, 2001) and, conversely, that ERN was smaller when the flanker stimuli in a Flanker task were spaced farther apart (Danielmeier et al., 2009) and were larger (Maier, Di Pellegrino, & Steinhauser, 2012). Presumably, the flanker stimuli in these instances were less viable options to respond to since they were clearly disparate from the primary stimulus, thus precluding the incorrect response activation and competition with the correct response. Hughes and Yeung (2011) demonstrated that the ERN was larger following a traditional Flanker task response conflict (i.e., on incongruent trials) compared to conditions in which congruent stimuli were visually masked. In the masked conditions, errors occurred due to diminished stimulus processing, which suppressed the activation of a response and any competition it may have encountered with another possible response. Lastly, the overlap between neural generators of N2 and ERN, namely the caudal ACC, further consolidates the view that both ERPs are aspects of the same conflict monitoring system.

An alternative to the above models is the motivational significance theory, which describes the ERN as an index of error significance. An early study demonstrated that an emphasis on accuracy over speed of performance resulted in larger ERNs following errors (Gehring et al., 1993), which may be interpreted to indicate a larger emphasis on errors, thus increasing their salience. In support of this, subsequent studies have found that errors produce measurable physiological effects, including changes in skin conductance and decreased heart rate (Hajcak et al., 2003; Hajcak, McDonald, & Simons, 2004), and that the defensive startle reflex is enhanced following error, compared to correct responses (Hajcak & Foti, 2008). Exaggerating the salience of errors by punishing incorrect responses (losing 100 “points” for erroneous responses on a modified Flanker task) was also found to enhance ERN (Hajcak, Moser, Yeung, & Simons, 2005), a finding replicated by other studies (Chiu & Deldin, 2007; Kim, Iwaki, Uno,

& Fujita, 2005). However, the motivation feature is somewhat equivocal. While it is implied that increasing the salience of errors concurrently increases the motivation to avoid them, the behavioural manifestation of this is not specifically addressed. Additionally, if larger ERNs following errors indicate increased salience of incorrect responses, it might follow that on tasks emphasizing accurate performance, other task-related ERPs would also be enhanced due to a greater motivation to perform well. In particular, correct responses should also correspond with larger correct-related negativities (CRN; see below). However, Hajcak and colleagues (2005) did not observe any differences in CRN between salient error and control groups.

Of note, an ERP similar to ERN can be observed following correct responses, termed the CRN, though its appearance is inconsistent (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Ford, 1999; Gehring & Knight, 2000; Scheffers & Coles, 2000; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). When present, it occurs 50-100 ms following correct responses and, akin to the N2 and ERN, the CRN appears to share the underlying neural source of frontal midline electrodes (Eichele, Juvodden, Ullsperger, & Eichele, 2010; Steinhauser et al., 2012) and the ACC as indicated by fMRI (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). Multiple functions have been suggested for the CRN, including response comparison (Falkenstein et al., 2000; Vidal et al., 2000), recruitment of greater cognitive control (Grützmann et al., 2014; Luu, Collins, & Tucker, 2000), or simply an artifact of response timing evaluations or contamination between response-locked and timing-locked averages (Coles et al., 2001). Ultimately, the exact function of CRN is currently unclear.

Pe. The error positivity is a positive deflection occurring 200-400 ms following an erroneous response, after the ERN (Falkenstein et al., 1991). Unlike the other conflict-related ERPs, Pe activity is more distributed along midline electrodes (typically Fz, Cz and Pz; Overbeek,

Nieuwenhuis, & Ridderinkhof, 2005) and has been linked with the rostral ACC (Vincent Van Veen & Carter, 2002). With regard to error rate and amplitude, some have found more errors to be related to smaller ERN, but unaltered Pe (Hajcak et al., 2003; Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004), while others have found reduced Pe with higher error rates (Dywan, Mathewson, & Segalowitz, 2004; Falkenstein et al., 2000). A within-subject analysis of ERPs and error rate found slightly larger Pe magnitude when speed was favored over accuracy (resulting in higher error commission; Ullsperger & Szymanowski, 2004). Lastly, genuine errors were associated with greater Pe compared to intentional errors (Stemmer, Witzke, & Schönle, 2001) and correct responses misinterpreted as errors (Ehlis, Herrmann, Bernhardm, & Fallgatter, 2005).

The functional significance of Pe is debated. It has been suggested to reflect an affective response to errors (Falkenstein et al., 2000; Van Veen & Carter, 2002), adjustment of post-error behavioural strategies (Hajcak et al., 2003; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001), or conscious awareness of errors (Nieuwenhuis et al., 2001). The first two theories have mixed evidence. In support of the notion that Pe reflects an affective response to errors, the neural locus of Pe, the rostral ACC, has been linked with affective processing (Bush, Luu, & Posner, 2000). Moreover, Pe amplitude correlates with skin conductance, a measure of the physiological expression of affective responses (Hajcak et al., 2003), and participants with more stolid attitudes towards errors have smaller Pes (Falkenstein et al., 2000). However, Pe amplitude has also been inversely correlated with negative affect (Hajcak et al., 2004). As for the theory of post-error behaviour adjustments, though some studies have related Pe magnitude to post-error behaviours, including post-error slowing (Hajcak et al., 2003; Nieuwenhuis et al., 2001), others have instead found this relation with ERN (Debener et al., 2005).

Conscious error awareness appears to be the strongest explanation, though this theory is not unequivocally supported either. Nieuwenhuis and colleagues (2001) used an anti-saccade task to examine performance errors, a task that has the property of inducing unrecognized errors due to a proclivity for automatic saccadic eye movements. The ERP data showed that while ERN was present for both recognized and unrecognized errors, Pe was only apparent on consciously perceived errors. Interestingly, post-error slowing was only observed following errors which were recognized, which lends credence to the theory regarding the recruitment of post-error rectifying behaviours (alternately, post-error behavioural adjustments could be the consequence of conscious error perception, and the relation with Pe could simply occur through its association with error awareness). Other studies have demonstrated that hypnotized individuals show reduced Pe, but unaltered ERN in response to errors (Kaiser, Barker, Haenschel, Baldeweg, & Gruzelier, 1997), and that Pe was also reduced when incongruent, error-inducing stimuli were decreased in visual salience (Leuthold & Sommer, 1999). However, neither Pe nor ERN was altered between easy- and hard-to-distinguish stimuli in a perceptual error task (Elton, Spaan, & Ridderinkhof, 2004).

P3. The P3 component is thought to reflect the process of inhibition – both motor and cognitive – and can typically be observed just prior to inhibiting a response or a cognitive process at both frontal midline (FCz, Cz) and posterior midline locations (Cz, CPz, Pz). Multiple studies have found increased P3 amplitudes related to successfully inhibited responses (Etchell, Sowman, & Johnson, 2012; Greenhouse & Wessel, 2013; Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004; Senderecka, Grabowska, Szewczyk, Gerc, & Chmylak, 2012; Wessel & Aron, 2015). Additionally, P3 magnitude appears to be contingent on prioritizing successful response withholding, as demonstrated in a study that linked larger P3 amplitudes when monetary

incentives were provided for prioritizing inhibition (i.e., accuracy) over speed (Greenhouse & Wessel, 2013). Both motor and cognitive inhibition appear to be reflected by ERP components (Bruin & Wijers, 2002; Burle, Vidal, & Bonnet, 2004; Pfefferbaum, Ford, Weller, & Kopell, 1985; Smith, Johnstone, & Barry, 2008; Wang, Tian, Wang, Cui, & Zhang, 2002).

Some researchers have argued that the P3 occurs too late in relation to the stop signal reaction time (SSRT), an index of inhibition on the SST (Dimoska, Johnstone, Barry, & Clarke, 2003; Huster, Enriquez-Geppert, Lavallee, Falkenstein, & Herrmann, 2013), instead suggesting that it reflects an evaluative process of inhibition (e.g., positive affect following a good outcome, such as successful inhibition; Huster et al., 2013). However, Wessel and Aron (2015) provided contrasting evidence in a large sample of 62 participants pooled from several studies. Here, the P3 component was identified using independent component analysis to increase the signal-to-noise ratio, and the onset of the P3 was quantified in relation to the SSRT. P3 onset was found to correlate with SSRT, meaning that the sooner the P3 occurred, the faster participants withheld their responses. Furthermore, the P3 following successful inhibition occurred before P3s on failed inhibitions, indicating that successful inhibitions require the P3 to occur prior to responding.

With respect to neuroanatomical substrates, the prefrontal cortex (PFC) is thought to inhibit behaviour via the basal ganglia, which subsequently prevents action execution by suppressing the primary motor cortex (Brooks, 1986; Fuster, 1989; Greenhouse, Swann, & Aron, 2011; Norman & Shallice, 1986; Robbins, 1996). A key structure in the network is the subthalamic nucleus (STN), which receives input from the inferior frontal gyrus (IFG) and pre-supplementary motor cortex (pre-SMA; Aron, Behrens, Smith, Frank, & Poldrack, 2007; Inase, Tokuno, Nambu, Akazawa, & Takada, 1999). Through input from the cortical regions, the STN

is thought to convey the stop commands by intensifying inhibitory signals to the globus pallidus, ultimately suppressing output from the basal ganglia and consequently stopping movement (Alexander & Crutcher, 1990; Parent & Hazrati, 1995). Multiple other regions have been linked to inhibition, including the supplementary motor area (SMA) and pre-SMA (Mostofsky et al., 2003; Simmonds, Pekar, & Mostofsky, 2008), premotor cortex (Picton et al., 2007; Watanabe et al., 2002), parietal cortex (Menon, Adleman, White, Glover, & Reiss, 2001; Rubia et al., 2001), ventrolateral PFC (Boehler, Appelbaum, Krebs, Hopf, & Woldorff, 2010; Swick, Ashley, & Turken, 2008), dorsolateral PFC (Fassbender et al., 2004; Garavan, Hester, Murphy, Fassbender, & Kelly, 2006; Hester et al., 2004; Menon et al., 2001), the right inferior frontal cortex (IFC) and insula (Aron, Robbins, & Poldrack, 2004; Garavan et al., 2006; Garavan, Ross, & Stein, 1999; Kelly et al., 2004; Konishi, Nakajima, Uchida, Sekihara, & Miyashita, 1998). However, it is important to note that many of these structures may not be directly related to inhibition, and activity in some regions may serve auxiliary functions or concomitant processes, including interference resolution in the insula (Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli, 2002; Bunge, Hazeltine, Scanlon, Rosen, & Gabrieli, 2002), and maintenance of task rules and working memory in the dorsolateral PFC (Levy & Goldman-Rakic, 2000; Mostofsky et al., 2003; Petrides, 2000; Simmonds et al., 2008).

Li, Huang, Constable, and Sinha (2006) attempted to parse out cortical regions involved directly in inhibition, as opposed to stimulus and response monitoring and post-response processes, and found that activity in the SMA, specifically the superior and precentral gyri correlated with the speed of response inhibition on the SST. This is supported by findings that stimulation of the pre-SMA via direct cortical stimulation in monkeys (Isoda & Hikosaka, 2007) and humans (Fried et al., 1991; Lüders et al., 1988) induces inhibition, while interference with

the pre-SMA via transcranial magnetic stimulation (Chen, Muggleton, Tzeng, Hung, & Juan, 2009) and lesions to the medial PFC (including the pre-SMA; Floden & Stuss, 2006; Nachev, Wydell, O'Neill, Husain, & Kennard, 2007) impairs inhibition.

Summary of performance monitoring ERPs. The electrophysiological signature of performance monitoring is composed of multiple ERPs. Occurring first, the N2 is a negative deflection in frontal midline regions following stimulus presentation; the most common interpretation is the conflict produced by competing potential responses to a stimulus. The ERN is another negative frontal midline waveform occurring almost immediately after an error is committed. Though multiple theories of its functional significance exist, the most prominent one describes ERN as the result of conflict between the incorrectly executed response and the latent correct one. The Pe is a positive deflection in midline to posterior regions following the commission of an error (and occurring after the ERN). As with the other ERPs, multiple theories have been posited, but it might reflect conscious error awareness. Lastly, the P3 component, observed at frontal and posterior midline regions, is thought to reflect inhibition. It can occur on both successful and failed inhibitions, but faster onsets are required for responses to be accurately withheld.

As shown by the somewhat mixed literature, the theories associated with many performance monitoring-related ERPs are not undisputed. As a result, comparisons between studies can be obfuscated if ERPs are not conceptualized similarly (e.g., taking the N2 as an index of inhibition rather than conflict monitoring). This study has the potential to strengthen theories of performance monitoring ERPs by replicating findings regarding key features, and by elucidating some conflicting points. This would also support the study of performance monitoring in patient populations. For instance, though deficient inhibition has been proposed as

a model of OCD (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005), there is a surprising paucity, and lack of cohesiveness, of research on the inhibition P3. Part of the reason for this is that the P3 has been defined in different ways (e.g., inhibition, semantic processing, allocation of attention; Fan et al., 2014; Herrmann, Jacob, Unterecker, & Andreas, 2003; Keskin-Ergen et al., 2014), and not all studies found differences between patients with OCD and healthy controls. The null findings could potentially be attributed to the variable conceptualizations of the P3, an issue that could be rectified by a clarification of its functional relevance to inhibition (and lead to future studies adopting this conceptualization).

Establishing a Behavioural Measure of Conflict Monitoring

As described above, conflict monitoring has been linked with the N2 component, but is lacking a behavioural measure. A potential solution to this issue is to use the subtraction method to estimate a performance index of conflict monitoring.

Subtraction method. The rationale underlying this method is that taking the difference of a performance measure (e.g., RT) between two behavioural tasks that share all cognitive processes except for one provides an index of the unique cognitive process (Donders, 1969). For instance, subtracting RT of one task from a second task with a unique cognitive feature yields the processing time for that unique feature. Though research on this method is sparse, its validity has been demonstrated in select areas such as the Trail Making Test (TMT) B-A difference index. This is obtained by subtracting the mean RT of the TMT-A from the mean RT of TMT-B. The rationale of this computation is to parse out the processing speed element common to both A and B versions in order to provide a purer measure of set shifting (Fujiki et al., 2013; Jacobson,

Blanchard, Connolly, Cannon, & Garavan, 2011; Sánchez-Cubillo et al., 2009; Zakzanis, Mraz, & Graham, 2005).

For instance, Gottsdanker and Shragg (1985) found support for Donders' idea that simple and choice RT tasks are identical except for stimulus discrimination and response selection components of choice RT tasks, and that these processes are inserted along a series of other cognitive processes common to both (stimulus encoding, premotor and motor engagement). This was established by varying the latency of the precue (i.e., indicators of the type of response needed). Longer precues were associated with shorter RTs on the choice RT task, but not the simple RT task; further, at the longest precue periods (150 ms), choice RT was equivalent to simple RT. The purported explanation for these findings is that longer precues provided more time to identify the correct stimuli and activate the correct response schema; these processes were completed in a graded fashion until the longest precue interval, at which point the cognitive processes required for both simple and choice RT tasks were approximately equivalent.

Van De Laar, Van Den Wildenberg, Van Boxtel, and Van Der Molen (2010) used the subtraction method to identify different aspects of behavioural inhibition in the SST by using different stop signal combinations (standard stop signal, responses withheld on one, but not another type of stop signal, and responses withheld only when the go and stop stimuli matched). The standard stop signal task required only the Go stimuli to be identified, while the other two conditions required additional stimulus discrimination. In the second version, participants were required to discriminate the Go stimuli not only for the appropriate responses, but also for the stop signals (when they occurred). In the third version, the Go stimuli were the standard ones, but responses only needed to be withheld for select stop signals. Because all cognitive processes (including stimulus identification, motor execution, and inhibition) were shared across task

conditions, subtracting the reaction times from each task was thought to provide indices of their unique stimulus discrimination processes. Using this approach, two different aspects of stop signal processing – stop signal discrimination and inhibition mapping – were parsed out.

Process invariance. One of the key aspects of the subtraction method is the assumption of process invariance between tasks (Sternberg, 1969); that is, all cognitive processes (except the unique feature) are identical between the tasks which are compared. This is an important assumption, since differences in addition to the unique feature could bias the subtraction process. For instance, if two tasks to be compared differed not only on process A but also on process B, then a subtraction method to estimate the duration of process A would be invalid because differences in process B could also influence processing time. One way to examine process invariance is to use an ERP approach to compare component latencies between tasks. If the components, related to various cognitive processes, are similar between tasks, then process invariance could be supported. One such popular ERP is the lateralized readiness potential (LRP). The LRP is a slow negative deflection appearing contralateral to the responding hand, and increases in magnitude until the response is executed (Kutas & Donchin, 1980). It appears after the stimulus and is indicative of motor processing separate from pre-motor preparation (Leuthold, Sommer, & Ulrich, 1996). Depending on whether the component is stimulus- or response-locked, different aspects of motor processing may be isolated. The time between stimulus onset and the LRP (LRP-s) indicates stimulus processing prior to response activation, whereas the time between the LRP and response (LRP-r) is considered the duration of response activation (Smulders & Miller, 2012).

Miller and Low (2001) examined motor invariance between simple RT, choice RT, and Go/NoGo tasks, using the LRP. Importantly, the LRP-r latency was equal across the three tasks,

suggesting that the assumption of motor invariance was met. However, Danek and Mordkoff (2011) argued that the precue used in the choice RT task provided information on the type of response required, thus eliminating a unique feature of the choice RT task. In order to decouple precue from the stimulus discrimination and response selection processes, responses were alternated between hands. After controlling for response side (left versus right hand response), the LRPs were found not to be equivalent across the tasks. However, the additional requirement to switch responses in addition to stimulus identification may have increased task difficulty, and potentially influenced the LRP.

Other ERP components may also be used to examine process invariance between tasks. When comparing simple and choice RT tasks, such as the simple RT (SRT) and SST tasks used herein, the unique feature is response/conflict monitoring in the choice RT task. Processes that are shared between tasks are stimulus encoding and discrimination, and motor preparation (the LRP, described above).

The P1 is a well-studied component that reflects early sensory encoding (Hillyard, Vogel, & Luck, 1998; Myers, Walther, Wallis, Stokes, & Nobre, 2015; Zalar, Martin, & Kavcic, 2015). It is a positive deflection that occurs 50 to 100 ms after the stimulus at occipito-parietal electrodes (PO7 and PO8 on the 10-20 system). Using this component as a comparison between tasks can elucidate whether the initial stimulus encoding phase is equivalent. Following the P1 is the N1, a negative deflection that also occurs at PO7 and PO8 approximately 100 to 200 ms post-stimulus. This component is thought to relate to stimulus discrimination stemming from the modulation of attention (Fedota, McDonald, Roberts, & Parasuraman, 2012; Luck, Woodman, & Vogel, 2000; Warbrick, Arrubla, Boers, Neuner, & Shah, 2014). Examining the N1 component could establish whether stimulus discrimination differs between tasks. This step is important

because the response monitoring process in choice RT tasks presumably relies on the successful discrimination and identification of the stimulus. Though the SRT in the current study also employed two types of stimuli (please refer to the methods section for details), it may not have as much emphasis on this process due to the requirement for only one type of response.

Purpose and Hypotheses

Despite the relatively dominant view of N2 as an index of conflict monitoring, research has nearly exclusively focused on electrophysiological features at the expense of its behavioural expression. Establishing a behavioural measure of conflict monitoring – particularly if its precision is supported by process invariance – could provide an alternate method of studying conflict monitoring. This would eliminate the need to use neuroimaging technologies, which can be expensive and somewhat complicated to use. Additionally, though there is a rich history on some (but not all) ERP components of performance monitoring, debates around the function of ERN, Pe, and the P3 still remain.

In an attempt to contribute to these unresolved areas, the subtraction method was applied to the SST and SRT to provide a "purer" index of conflict monitoring (while parsing out cognitive processes common to both tasks). A larger difference in RT between the tasks would indicate greater response conflict monitoring. To relate it to the more-established index of conflict monitoring, the N2 component, the correlation between this index and the magnitude of the N2 was examined. Multiple hypotheses were proposed:1a) the SST-SRT difference measure would correlate positively with the N2 component area, such that larger RT-difference values would correspond with larger N2 areas; 1b) stimulus encoding and discrimination, and motor activation processes (corresponding to P1, N1, and LRP-r) were hypothesized to be equivalent

between the SRT and SST; 2) both ERN and Pe areas were hypothesized to negatively correlate with the number of actual and estimated error commissions; 3a) if Pe is a function of error awareness, then this component would correlate with both the estimated frequency and number errors committed, and the absolute difference between actual and estimated errors (i.e., the degree of accurate error commission awareness); 3b) alternately, if Pe is related to affective processing of errors, then its magnitude should correlate with the degrees of performance and error frustration; 4a) the P3 area was hypothesized to correlate with SSRT and percent successful inhibition (PSI); and, lastly, 4b) that P3 onset latency would also negatively correlate with the SSRT.

Methods

Participants

41 healthy participants were recruited, 35 of whom completed the study in full. Two were unable to complete the EEG portion due to scheduling conflicts, and four were excluded for endorsing psychiatric symptoms (please refer to the procedures section for details on psychiatric symptom assessment). The demographics of the sample are presented in Table 1.

Eligibility criteria were: over 18 years of age; normal or corrected-to-normal vision; fluent English; no colour-blindness; no history of psychiatric, neurological, neurodevelopmental, or medical conditions; and no current prescription medication use, or psychological treatment such as cognitive behavioural therapy.

The study received ethical approval from the Research Ethics Board at Ryerson University. Participants were recruited from Ryerson University through posters. All participants were paid $20.

Table 1. Demographics

	Mean (SD)
Age	32.27 (14.14)
Gender	24 F, 11 M
Years of Education	17.06 (2.76)
Handedness	32 right, 3 left
Caucasian	45.71%
Asian	28.57%
South Asian	17.14%
African-American	5.71%
Middle-Eastern	2.86%
Predicted WAIS-III	112.37 (8.49)
BDI-II	3.23 (4.22)
BAI	3.66 (5.37)
STAI-State	26.97 (7.94)
STAI-Trait	31.66 (8.40)

Measures

The neuropsychological test battery included the Stroop Color-Word Interference Test, Symbol Digits Modalities Test (SDMT), Wechsler Test of Adult Reading (WTAR), Stop Signal Task (forced-choice RT task), and a simple RT task (simple RT task). The SRT and SST were completed with the EEG. A short questionnaire on subjective evaluations of task performance was administered following the SST.

Stop Signal Task. The SST (Logan, Cowan, & Davis, 1984) is a test of motor inhibition and conflict monitoring. This task has demonstrated good reliability (intra-class correlations from .42 - .86, fair to excellent range; Congdon et al., 2012) and convergent validity with the Go/NoGo task (r = .46; Rana & Rao, 2013). Each trial of the SST began with a fixation cross, followed by either an 'X' or 'O' stimulus (Go stimuli) on each trial with equal probability. The inter-stimulus intervals (ISI) was jittered between 500-1000 ms. Participants responded to the Go stimuli with keyboard presses: the "x" key for "X" stimuli, and "." key for "O" stimuli. On 25% of the trials, an auditory tone was presented after a Go stimulus, indicating that the response should be withheld (stop signal). Though the initial ISI between go and stop signals was 750 ms, the stop signal was delayed by 50 ms following each successful response inhibition, and decreased by 50 ms following failed inhibitions. This online algorithm attempts to ensure a failure rate of 50%, (though some variation will exist with each participant; Logan et al., 1984). A minimum of 70% accuracy on go trials was the cut-off (all participants had >91% accuracy). Each block contained 24 stimuli (six followed by stop signals), and the task included five trials, the first of which was practice and not counted in the outcome measures. The task was given four times consecutively in order to ensure a sufficient number of correct responses (342 Go trials), and successful and failed inhibitions (approximately 57 each) for reliable ERPs. The primary outcome measures

included accuracy, percent successful inhibition (PSI), RT on correct Go (CRT) and failed Stop (SRT) trials, the mean delay of the stop signal (SSD), and the stop signal RT (SSRT; calculated by subtracting the mean stop signal delay from the mean correct trial RT). The SSRT is an index of inhibition, with smaller values indicating faster inhibition.

Simple Reaction Time Task. The simple RT task was similar to the SST, except the response was identical to both Go stimuli (i.e., same button press for both 'X' and 'O'), and no stop signals were presented (i.e., no response inhibition). The purpose for this task was to calculate the SST-SRT difference index. As with the SST, the SRT contained 24 trials per block and five blocks per task (the first of which was practice and not counted). The SRT was administered three times, for a total of Go 336 trials, to provide a sufficient number of trials for reliable ERPs.

Stroop Colour-Word Interference Task. The Stroop task (Stroop, 1935) is a measure of cognitive inhibition that has been shown to be reliable (r ranged from .50 - .85; Graf, Uttl, & Tuokko, 1995) and valid for frontal lobe-related inhibition (Stuss, Floden, Alexander, Levine, & Katz, 2001). It includes three trials, the first with a list of colour-related words (e.g., "red" or "blue"), the second with a series of "X"s printed in different colours, and the third with a list of colour words printed in different colours (e.g., the word "red" printed in green). On all three trials, participants had 45 s to read as many words as they could. On the last trial, participants were instructed to read the ink colour, not the word. Raw and t-scores were calculated for the number of words read on each trial. Additionally, the interference score, an index of cognitive inhibition ability, was calculated by subtracting the predicted colour-word score ([word trial x colour trial]/[word trial + colour trial]) from the actual colour-word trial score. A more positive score indicates better inhibition, and more negative scores indicate poorer inhibition.

Symbol Digits Modalities Test. The SDMT (Smith, 2002) is a test of processing speed that is reliable (r = .88) and valid (receiver opertaing curve = .81; López-Góngora, Querol, & Escartín, 2015; Sonder, Burggraaff, Knol, Polman, & Uitdehaag, 2014). This measure was included to control for processing speed in the relationship between N2 area and the SST-SRT index. It involves matching numbers to unique symbols with a 90 s time limit. Raw scores for the total number of correct numbers are provided for the test.

Wechsler Test of Adult Reading. The WTAR provides an estimate of premorbid intelligence, and can be used to predict the Wechsler Adult Intelligence-III full scale intelligence quotient (WAIS-III FSIQ; Psychological Corporation, 2001). It has strong construct validity with measures of verbal intelligence (r = .75; Psychological Corporation, 2001; Whitney, Shepard, Mariner, Mossbarger, & Herman, 2010) and is reliable (r ranged from .90 - .94; Dykiert & Deary, 2013). This measure was administered to rule out intellectual deficits (below an FSIQ of 70; American Psychiatric Association, 2013). The test is a list of 50 words of increasing difficulty that must be pronounced by participants. Raw scores are converted to standard scores, which can then be used to estimate WTAR- and demographics-predicted WAIS-III FSIQ.

Beck Depression Inventory-II. The BDI-II is a self-report questionnaire of depressive symptoms based on DSM-IV criteria (Beck, Steer, & Brown, 1996). It is sensitive to depressive symptoms, reliable, and valid as a screening tool (Subica et al., 2014; Wang & Gorenstein, 2013). This questionnaire was included to provide a quantitative supplement to the psychiatric interview to ensure eligibility criteria. It contains 21 questions related to Major Depressive Disorder criteria, and depressive symptom severity is rated as minimal (scores of 0-13), minor (14-19), moderate (20-28), or severe (29-63). Symptom severity greater than minimal was used as the cut-off.

Beck Anxiety Inventory. The BAI is a self-report questionnaire of general anxiety symptoms not specific to any particular anxiety disorder (Beck & Steer, 1993). Strong reliability and construct validity (relative to the STAI and anxiety diaries) have been demonstrated (Fydrich, Dowdall, & Chambless, 1992; Osman et al., 2002). This questionnaire was included to provide a quantitative supplement to the psychiatric interview to ensure eligibility criteria. The BAI contains 21 questions on a variety of anxiety symptoms (e.g., feelings of numbness, nervousness), and is rated as minimal (scores of 0-9), mild (10-16), moderate (17-29), or severe (30-63) anxiety. Symptom severity greater than minimal was used as the cut-off.

State-Trait Anxiety Inventory. The STAI is a self-report measure of state (current/temporary anxiety) and trait (enduring disposition for anxiety) levels of anxiety (Spielberger, 1983). The Trait form is reliable, but the State form is less so because it was designed to detect transitory states (Spielberger, 1983). Construct validity for anxiety symptoms is high, but is frequently confounded with depressive symptoms (Kabacoff, Segal, Hersen, & Van Hasselt, 1997). This questionnaire was included to provide a quantitative supplement to the psychiatric interview to ensure eligibility criteria, and also to provide additional information about anxiety symptom (state and trait). Both the State and Trait forms contain 20 questions related to anxiety symptoms without a specific diagnostic focus. Raw scores as well as z-scores to indicate symptom severity (relative to non-anxious adults) may be obtained; a z-scores of -2 was used as a cut-off (Knight, Waal-Manning, & Spears, 1983).

Subjective Performance Questionnaire. This questionnaire was used to obtain subjective evaluations of task performance on the SST. The questions were based on the ones administered by Stern and colleagues (2010). The questions were: 1. “Did you make any mistakes?”, 2. “How many mistakes do you think you made?”, 3. “Were you ever frustrated with your performance?”,

and 4. “When you made a mistake, were you flustered? Did you find it hard to get back on track?” Questions 1, 3 and 4 were on a 10-point Likert scale, and participants filled in a numerical response for question 2 (please refer to the Appendix A for the questionnaire).

Psychiatric interview. A structured interview that screened for a range of psychological disorders based on DSM-IV TR criteria (e.g., Major Depressive Disorder, Social Anxiety Disorder, Obsessive-Compulsive Disorder) was included (Ickowicz et al., 2006). This interview was used for its relative brevity, structure, and availability. Please refer to Appendix B for the interview. Please note that although the interview is referred to as a phone screen in the title, it was conducted in person.

Procedure

Following informed consent, participants were administered the psychiatric interview and clinical questionnaires to probe for the presence of possible psychiatric diagnoses. Upon establishing eligibility, the test battery (SDMT, Stroop, and WTAR) was administered in a quiet interview room. The EEG equipment was then applied (e.g., cap placed on head, conducting gel applied to scalp, electrodes attached onto the cap), and participants were led into the sound-attenuated EEG testing room. Participants were instructed to sit in a relaxed position to minimize muscle activity, focusing on the centre of the screen while limiting blinking to reduce artifacts. The order of the SRT and SST were counterbalanced based on participant IDs to avoid potential ordering effects. The subjective performance questionnaire was administered after the SST. Following the completion of the two tasks, the electrodes and cap were removed, and participants were provided a towel and shampoo to clean out any residual gel from their hair.

EEG Recording and Analysis

EEG data were recorded using the BioSemi Active Two system (BioSemi Inc., Amsterdam, Netherlands), with 64 Ag-AgCl electrode sites distributed according the guidelines of the International 10-20 system (Klem et al., 1999). The 10-20 system refers to the ubiquitously used map of electrode locations. The electrodes are distributed on the scalp in a standardized fashion to promote comparability of electrophysiological data across studies. The Common Mode Sense (CMS) active electrode and the Driven Right Leg (DRL) passive electrode were used as the reference and ground, respectively. The sampling rate was 512 Hz with a 24 bit analog to digital conversion.

EEG Preprocessing

The EEG data were preprocessed using MATLAB 2016a (The Mathworks Inc., Natick, MA) and the EEGLab toolbox, version 13.6.5b (Delorme & Makeig, 2004). The data were rerefenced to the mastoids electrodes. Stimulus-locked data were epoched -200 to 800 ms with respect to the stimulus onset, using -200 to 0 ms for baseline correction. Stimulus-locked epochs were used for analyses involving the P1, N1, N2, and P3 ERPs. Response-locked data were epoched -600 to 400 ms around the response, using -600 to -400 as baseline correction, for the SRT, and -700 to 300 ms (-700 to -500 as baseline correction) around the response for the SST. The rationale for using slightly different response-locked epochs between the tasks is that the SRT had shorter mean RTs; using a longer pre-response timespan would have resulted in interference from the stimulus onset and potentially other ERPs. Response-locked epochs were used for ERN, Pe, and LRP-r analyses. Eye blink and shift artifacts were removed using independent component analysis (ICA) using the ICA EEGLab toolbox (Makeig et al., 2002).

Briefly, ICA separates and adds weights to individual ERP components, including eye blink/shift movement artifacts. These components can be removed, and the waveform is reconstructed using redistributed weights. Note that the ICA process reduces the sampling rate to 256 Hz. However, this rate was deemed suitable for the ERP analyses performed, and is commonly used in EEG studies. A minimum of 15 error trials for ERN and Pe (Larson, Baldwin, Good, & Fair, 2010), and 30 go and no-go trials for N2 and P3, respectively (Brunner et al., 2013; Clayson & Larson, 2013),

EEG Analysis

Primarily two types of ERP measurements were utilized: area and fractional area latency. Area calculations can be made by either taking the total areas above and below baseline, or by taking the area of only positive or negative regions (Luck, 2014). The former is useful for ERPs that have both positive and negative regions, but is sensitive to the time-window; the latter reduces the impact of the time-window as it will include any region that is entirely positive or negative, though it can underestimate the area of an ERP if it includes both positive and negative regions. Of note, negative area calculation counts positive regions as zeros (and vice versa, positive area calculations will zero negative regions), thus including only positive integers; the total area calculation counts both positive and negative regions, resulting in integers ranging from negative to positive. This can affect the direction (but not interpretation) of some analyses. For the most part, both types of area measurements were used. Another subtype of area measurements are difference waves. The waveforms of two ERPs are subtracted from one another (SST minus SRT, in this case), which highlights the unique aspects of the component of interest; this method has been used previously for the N2 (Clayson & Larson, 2011a, 2011b;

Ladouceur et al., 2007) and was used for this component (because the difference wave has not typically been used for the other ERPs, it was not implemented for them). As stimulus-locked epochs were used for the N2, the same timespan was used from both SRT and SST.

Fractional area latency is the time-point that divides the ERP into a specified fraction. For instance, 50% fractional area latency is the point at which an ERP reaches half of its area. This is a more accurate method of identifying the midpoint of a waveform than peak measurements; identifying the peak can be problematic because waveforms, especially from individual participants, typically include multiple peaks and may not actually represent the true "peak" of the ERP (as well, peaks are not so much informative features of ERP components as they are artifacts of the EEG process). 50% fractional area latency, on the other hand, is not biased by multiple peaks and considers the entire waveform; this approach was used to obtain the midpoint of the P3 component, as was performed by Wessel and Aron (2015). This method can also be used to identify the onset and offset of waveforms by specifying, say, 15% and 85% fractional areas for onset and offset, respectively (these values are typically used for onsets and offsets [Luck, 2014], though the percent will vary somewhat with the component and its literature).

Based on previous research on the scalp distribution and timing of the ERP components, the N2 difference wave area was measured from 250-350 ms post-stimulus at Fz and FCz between the SRT and SST tasks; ERN area 0-100 ms post-error at Fz and FCz; Pe area 100-450 ms post-error at Fz, Cz, CPz, and Pz; P3 area and 50% fractional area latency (midpoint) 250-450 ms post-stop signal at Cz, CPz, and Pz. P1 15% and 85% fractional area latency (onset and offset, respectively) 75-150 ms post-stimulus at PO7 and PO8; N1 15% and 85% fractional area latency 100-200 ms post-stimulus at PO7 and PO8; and the LRP-r 90% fractional area latency

(cutoff based on Miller, Patterson, & Ulrich, 1998) -500 until the response on the SRT, and -200 until the response on the SST at C3 and C4.

The LRP-r was calculated using the double subtraction method (de Jong, Wierda, Mulder, & Mulder, 1988). The potentials related to each responding hand are larger in the contralateral hemisphere; in other words, the LRP-r for right hand responses is larger in the left hemisphere (C3), and that of the left hand is larger in the right hemisphere (C4). Difference waves were first calculated for both response sides using the formulas $(C3 - C4)_{\text{right hand}}$ and $(C3 - C4)_{\text{left hand}}$. Because the potential for a right sided response is larger in C3, the potential following the subtraction will become more negative; conversely, left sided responses are more negative at C4, and the subtraction results in a more positive potential. To obtain the combined LRP-r, the formula $(C3 - C4)_{\text{right hand}} - (C3 - C4)_{\text{left hand}}$ is used, which combines the two effects.

Statistical Analyses

The SSRT was calculated by subtracting the mean stop delay from the mean RT (Logan et al., 1984), and the SST-SRT difference index was calculated by subtracting the SRT RT from that of the SST. N1, P1, and LRP-r latencies were compared between the SRT and SST using paired t-tests. The relationships between performance variables (SSRT, SST-SRT, CRT, SST errors, PSI, performance questionnaire scores, the absolute difference between actual and estimated errors committed on the SST, Stroop color-word and interferences scores, and the SDMT) and ERP components were examined using Pearson's correlation coefficient *r* or hierarchical regressions, bootstrapped with 1000 iterations. Following Cohen's (1988) suggestion, Pearson's *r*s of $\geq .1$, $\geq .3$ and $\geq .5$ were considered small, medium and large effects, respectively. The variables from these measures were included in the analyses as they related to

the hypotheses; other analyses of the existing data that did not directly relate to the hypotheses were not performed to reduce the family-wise error rate. Since the sample was larger than 30, normality was assumed; this was based on the central limit theorem that posits that a large sample (e.g., ≥30) tends to follow a normal distribution (Field, 2013).

Outliers were identified and removed using the median absolute deviation (MAD) method (Leys, Ley, Klein, Bernard, & Licata, 2013). Due to the strong influence of outliers on the mean, detecting outliers using this method can be biased and result in the improper inclusion of extreme values (Cousineau & Chartier, 2010). In contrast, the median is insensitive to outliers and can be considered a better estimate of central tendency when used to detect outliers. In this calculation, the absolute deviation of each data point from the median is calculated (MAD). The MAD is then multiplied by 1.4826 (a constant related to the assumption of normality); this value is multiplied by 3 (the rejection criterion value), and any data point that is greater or less than this value around the median was considered an outlier and removed. The formula is as follows: $MAD = b\ M_i|x_j-M_j|$, where $b = 1.4826$, M_i is the median of the absolute deviations, x_j are the original data points, and M_j is the median of the original dataset. Thus, outliers can be identified with $M_j \pm (MAD*3)$. A total 51 outliers (2.79% of the database) were removed from the database using this method. A sample of 30 or more remained for each analysis after removing outliers, allowing for assumed normality of the data.

The jackknife procedure was used to estimate onset and offset latencies of the P1, N1, and LRP-r components. This procedure involves calculating a series of grand averages minus one dataset at each iteration; each grand average – 1 is then treated as a unique data point (Miller et al., 1998). The rationale for this approach is that while individual datasets tend to be noisy, the grand average mitigates much of the variability. Using grand averages as data points thus

reduces the error variance, and decreases the risk of Type II error without raising that of a Type I error (Luck, 2014). When comparing two groups using a t-test (in this case, the latencies of ERP components between the SRT and SST), the t value will be inflated due to the artificially reduced error variance. To rectify this, the t value is divided by N – 1.

Results

Behavioural Results

The mean (SD) of the tests administered are presented in Table 2. A visual examination of the histograms of the data from questions 1, 3 and 4 of the Subjective Performance Questionnaire did not indicate obvious skewing for 1 and 3, though the responses for 4 were biased towards the lower half (data not shown). The correlation between actual and estimated SST errors was $r = .49$ ($p < .05$), indicating that participants were fairly accurate at quantifying the mistakes they made. The correlation between actual errors and the absolute actual-estimated error difference was $r = .69$ ($p < .05$), indicating that participants with the most inaccurate estimates had the highest error rates. The correlation between actual SST errors and the actual-estimated error difference was $r = -.52$ ($p < .05$), meaning that those who most underestimated their error rates had the highest error commissions (these correlations are explored further in relation to ERN and Pe, and hypotheses 2, 3a and 3b).

The correlation between SDMT scores and SST-SRT difference was not significant ($r = -.11$, $p > .05$), suggesting that processing speed did not directly relate to this variable (this relationship was explored further in the context of N2, vis-à-vis hypothesis 1a). The SSRT was negatively correlated with the Stroop color-word score ($r = -.38$, $p < .05$), indicating that motor inhibition scores were related to similar cognitive inhibition performance.

ERP Results

N2. The N2 component was apparent from approximately 200 to 350 ms after stimulus onset, with difference areas (the subtraction of the SRT from SST waveform to isolate the N2 component) of 0.27 and 0.20 on the grand average waveform at Fz and FCz, respectively

Table 2. Behavioural Results

Test	Mean (SD)
SDMT	83.14 (19.68)
Stroop Word Trial	97.91 (17.19)
Stroop Color Trial	71.35 (13.65)
Stroop Color-Word Trial	45.43 (11.64)
Stroop Interference Score	4.65 (6.44)
Subjective Performance Question 1 ("Did you make any mistakes?")	5.86[a] (1.48)
Subjective Performance Question 2 ("…how many mistakes…?)	25.97 (15.14)
Subjective Performance Question 3 ("…frustrated with your performance?")	5.29[b] (1.92)
Subjective Performance Question 4 ("…made a mistake, were you flustered…?")	3.82[c] (1.96)
SRT PCR (%)	94.87 (12.56)
SRT CRT (ms)	360.12 (95.79)
SST PCR (%)	98.67 (1.74)
SST CRT (ms)	688.78 (153.54)
SST PSI (%)	64.63 (10.25)
SST Tracker SR (ms)	615.27 (162.85)
SST Stop Delay (ms)	417.87 (161.02)
SSRT (ms)	282.01 (52.78)
SST Errors	42.03 (15.11)
SST Absolute Error Difference	22.31 (16.44)
SST-SRT (ms)	310.00 (124.84)

Abbreviations: correct response time (CRT), percent correct response (PCR), percent successful inhibition (PSI), simple reaction time task (SRT), stop signal reaction time (SSRT), stop signal task (SST), symbol digits modalities test (SDMT).
[a]5.86 was around the "sometimes" mark on the Likert scale.
[b]5.29 was around the "sometimes" mark on the Likert scale.
[c]3.82 was between "not at all" and "sometimes" on the Likert scale.

(representative FCz component in Figure 1). The N2 area at Fz correlated positively with the SST-SRT difference ($r = .38$, $p < .05$), indicating that a greater difference between SST and SRT performance time was associated with a larger N2 area. N2 area did not correlate significantly with SST RT, indicating that the magnitude of N2 was not related to general performance speed on Go trials. Correlations are presented in Table 3.

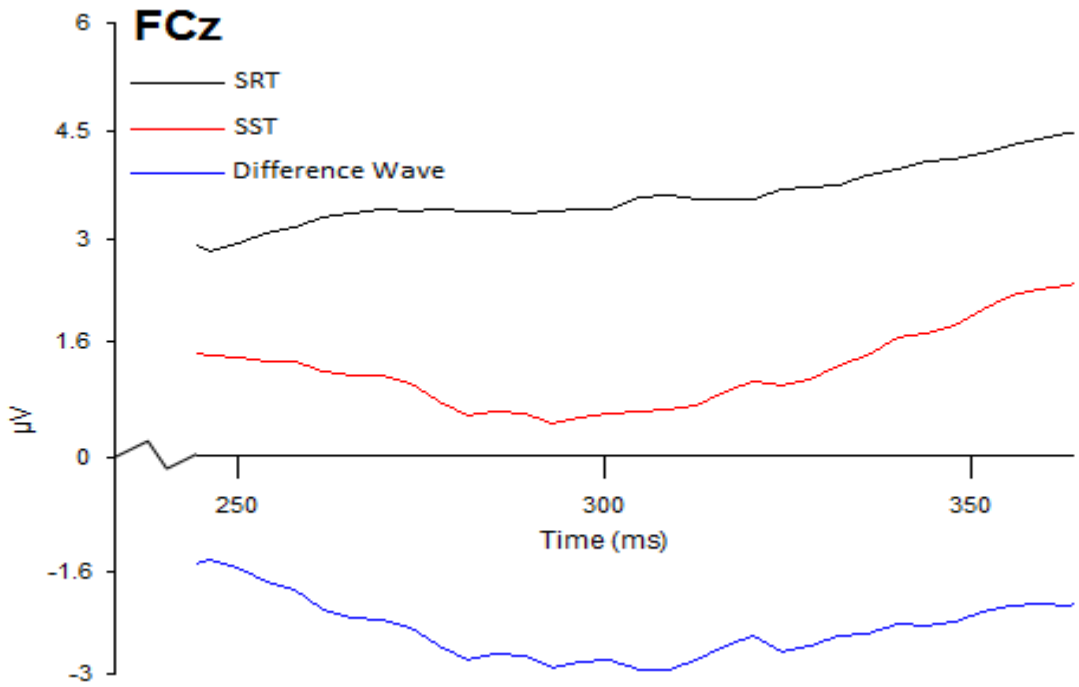

Figure 1. Stimulus-locked N2 component at FCz. Black and red lines are Go stimuli in the SRT and SST, respectively. The blue line is the difference wave (SRT-SST).

To evaluate whether the relationship between N2 area and the SST-SRT index was accounted by the SDMT score (processing speed), these variables were entered into a hierarchical regression, with the SST-SRT index entered first and SDMT score entered second. The SDMT scores explained some of the variance of the N2 area, approaching significance: $F(1,29) = 3.50$, $p = .072$, $R^2 = .092$, suggesting that processing speed had some influence on the relationship between the difference index and N2.

Lastly, to confirm that the N2 is related to conflict monitoring and not inhibition, as suggested previously (Bruin, Wijers, & van Staveren, 2001; Smith, Johnstone, & Barry, 2007), the relation between N2 area and the SSRT was examined. The correlations were not significant, supporting the function of the N2 as conflict monitoring.

ERN. The ERN component occurred approximately from 0 to 150ms following SST errors, with areas of -0.31 and -0.27 on the grand average waveform at Fz and FCz, respectively

Table 3. Correlation Matrix Between Test Scores and ERPs.

Variable	SST CRT	SSRT	SST PSI	SST-SRT	SST errors	SST [error diff.]	SPQ 1	SPQ 2	SPQ 3	SPQ 4	SDMT
N2 int. Fz	.14			.38*							.25
N2 int. FCz	.23			.31							.34
ERN int. Fz					-.45*	-.04	-.33	-.55*	-.23	-.03	
ERN int. FCz					-.53*	-.11	-.02	-.46*	-.39	-.12	
ERN neg. Fz					.38	-.01	-.12	.55*	.18	.11	
ERN neg. FCz					.39	-.01	-.09	.40	.37	.26	
Pe int. Cz					-.37	-.33	-.03	-.20	.16	.15	
Pe int. CPz					-.33	-.39	-.08	-.10	.17	.11	
Pe int. Pz					-.28	-.28	.03	-.07	.16	.07	
Pe int. Fz					-.43*	-.15	-.06	-.50*	.05	.06	
Pe int. FCz					-.45*	-.28	-.03	-.34	.05	.05	
Pe pos. Cz					-.36	-.33	-.03	-.17	.19	.18	
Pe pos. CPz					-.30	-.40	.10	-.07	.24	.17	
Pe pos. Pz					-.21	-.26	.09	-.04	.28	.14	
Pe pos. Fz					-.43*	-.16	-.08	-.43	.03	.04	
Pe pos. FCz					-.45*	-.29	-.4	-.30	.05	.06	
P3 int. Cz		-.42*	.04							.20	
P3 int. CPz		-.43*	-.03							.022	
P3 int. Pz		-.27	-.06							.020	
P3 pos. Cz		-.42*	.04							.24	
P3 pos. CPz		-.43*	-.02							.26	
P3 pos. Pz		-.26	-.05							.24	
P3 lat. Cz	.13	.51*									
P3 lat. CPz	.04	.45*									
P3 lat. Pz	.09	.44*									

Abbreviations: integral (int.), latency (lat.), positive (pos.), subjective performance questionnaire (SPQ).
*$p < .05$; all values indicate Pearson's correlation r.

(representative FCz component in Figure 2). The total area at both FCz and Fz negatively correlated with error commissions (r = -.53 and -.45, p < .05, respectively), indicating that higher error rates were associated with larger ERN areas. The estimated number of error commissions (question 2 on the subjective performance questionnaire) was also negatively correlated with ERN total area at both FCz (r = -.46, p < .05) and Fz (r = -.55, p < .05) electrodes, and positively correlated with negative ERN area (r = .55, p < .05) at Fz. These results indicate that, similar to actual errors committed, larger ERN components were associated with higher error commission estimates (the discrepancy between positive and negative correlations is due to differences arising from total and negative area calculations; please refer to the EEG analysis section for a discussion of this phenomenon). ERN area was not significantly correlated with the estimated frequency of error commission (question 1), frustration with performance and errors (questions 3 and 4), or the difference between actual and estimated error commissions.

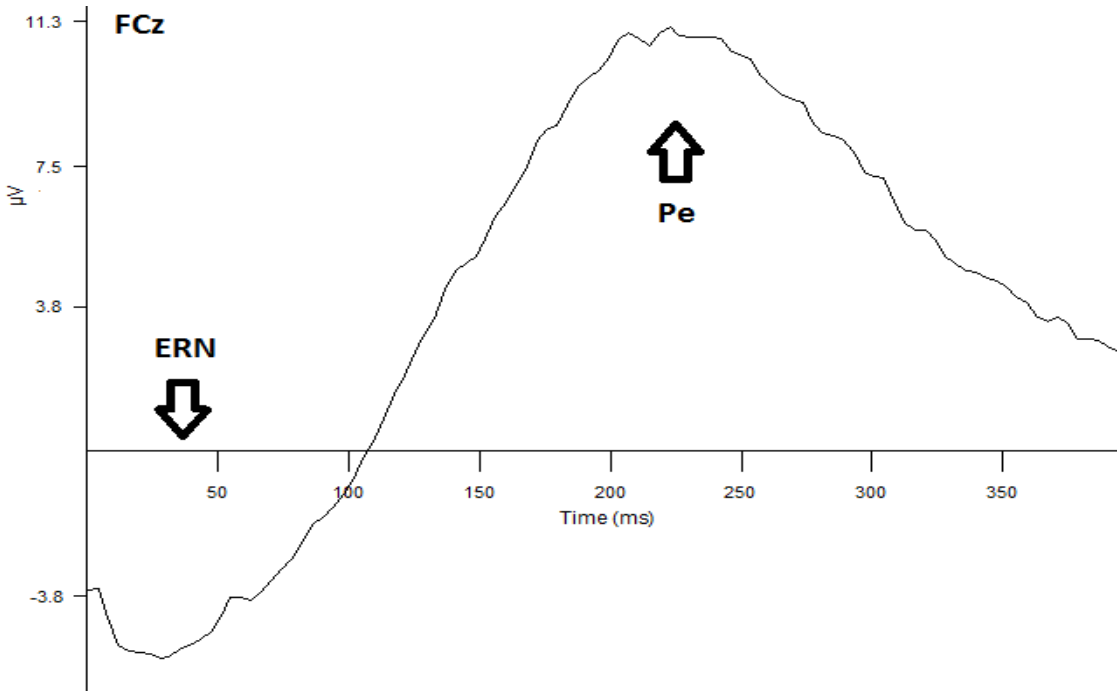

Figure 2. Response-locked ERN and Pe components at FCz.

Pe. The Pe component occurred approximately 100 to 450 ms following SST errors. The areas ranged from 1.44 at Fz, 2.07 at FCz, 2.29 at Cz, 2.01 at CPz, and 1.76 at Pz on the grand average waveform (representative FCz component in Figure 2). Both positive and total area correlated negatively with error commissions at FCz (both r = -.45, p < .05) and Fz (r = -.43, p = .05 and r = -.43, p = .05, respectively). Pe total area was also negatively correlated with estimated error commission at Fz (r = -.50, p = .05). Put simply, smaller Pe components were associated with higher actual and estimated error rates, opposite of the pattern observed with ERN.

Correlations between positive and total Pe areas and the absolute difference between actual and estimated error commissions (i.e., the discrepancy between subjective and objective error rates; smaller values indicate more accurate estimates) approached significance (r = .40, p = .075 and r = .39, p = .079). Regressions were used to assess if conscious awareness of errors (estimated errors, and the difference between actual and estimated errors) explained any variance in Pe area beyond error commissions. Neither estimated errors nor the difference between estimated and actual errors accounted for additional variance at any electrode (R^2 ranged from .001 to .061, all p > .05); since actual and estimated errors were significantly correlated, there is little unique variance that could be accounted for by adding both variables to the regression.

P3. The P3 component emerged approximately 250 to 450 ms after the stop signal. The areas of the grand average ranged from 2.44 at Cz, 2.25 at CPz, and 1.94 and Pz (representative Cz component in Figure 3). The P3 grand average onset latencies were 253.91 at Cz, 265.63 at CPz, and 281.25 at Pz. SSRT was negatively correlated with both positive and total areas at Cz (both r = -.42, p < .05) and CPz (both r = -.43, p < .05). In other words, smaller SSRT values, indicating superior inhibition, were associated with larger P3 components. P3 area was not significantly correlated with PSI.

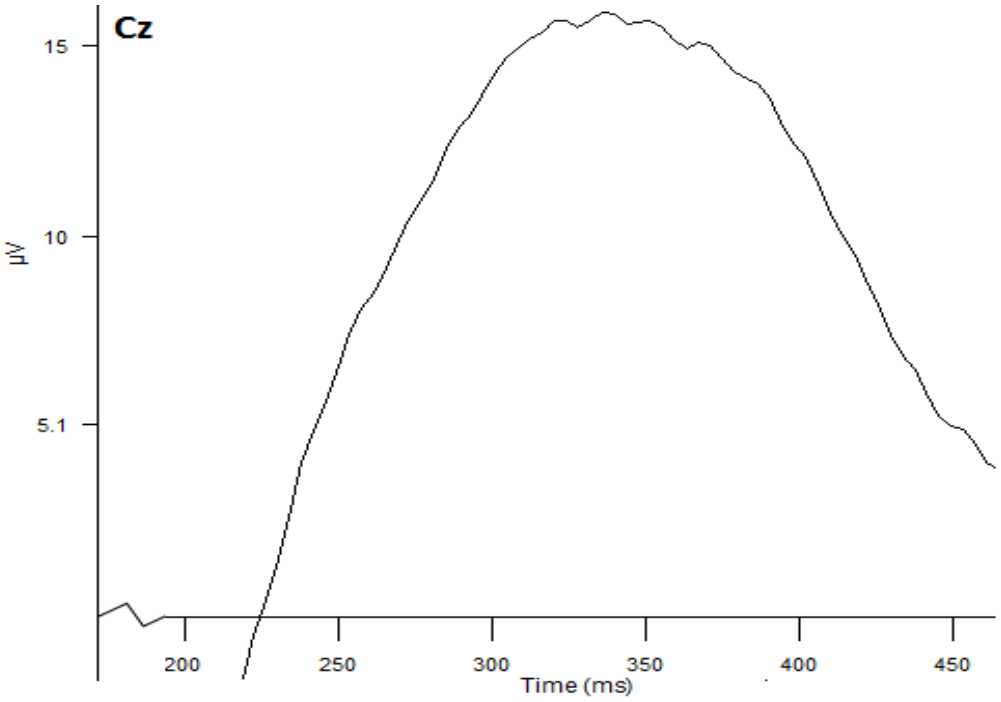

Figure 3. Stimulus-locked P3 component at Cz.

The P3 onset latencies positively correlated with SSRT at all three electrode locations: r = .51 at Cz, r = .45 at CPz, and r = .44 at Pz (all $p < .05$). This indicates that P3 components occurred earlier with smaller SSRTs (i.e., faster inhibition). Of note, the onset latencies highly and negatively correlated with the areas at each electrode (indicating larger magnitudes were associated with earlier onsets; r ranged between -.40 and -.64, all $p < .05$). As well, hierarchical regression models demonstrated that P3 area did not predict any additional variance of SSRT when it was entered after onset latency (at Cz: $F[1,26] = 0.35$, $p = .56$, $R^2 = .01$; at CPz: $F[1,26] = 0.062$, $p = .81$, $R^2 = .002$; at Pz: $F[1,26] = 0.33$, $p = .57$, $R^2 = .009$). As the two forms of measurement were so highly correlated, the area was likely influenced by the onset time. Further, because the combination of area and onset latency did not explain any additional SSRT variance, it likely does not matter which component is correlated to the SSRT, as they are inter-correlated.

To check whether this effect was simply an artifact related to general performance time, the P3 onset latencies were correlated with SST CRT. None of the correlations at any electrode emerged as significant, suggesting that P3 onsets were related to inhibition efficiency on Stop trials only, and not faster due to quicker responses on Go trials.

Lastly, to explore the alternate theories that P3 reflects the evaluation of the inhibitory process or outcome (Huster et al., 2013) or conflict monitoring (Bruin et al., 2001; Smith et al., 2007) rather than inhibition, P3 areas were correlated with question 4 of the subjective performance questionnaire (frustration level with errors) to test the former, and the SST-SRT difference and N2 areas for the latter. The area measure did not correlate significantly with scores on question 4, suggesting that the P3 was unrelated to inhibitory outcome evaluations. P3 also did not correlate with the SST-SRT difference score or N2 areas, implying that it is indeed an index of inhibition rather than conflict monitoring.

P1, N1, and LRP-r. To examine process invariance, the latencies of the P1, N1, and LRP-r; corresponding to early sensory encoding, stimulus discrimination, and preparation for motor execution, respectively; were compared between the tasks. The jackknife mean P1 offset was 105.78 ms post-stimulus on the SRT and 99.28 ms on the SST ($t[35] = 0.04$, $p > .05$), mean N1 offset was 113.28 ms on both SRT and SST ($t[35] = 0$, $p > .05$), and the latency between LRP-r and the response was 3.56 ms on the SRT and 0.22 ms on the SST ($t[35] = 0.1$, $p > .05$). None of the latencies were statistically different between the tasks. The jackknife mean P1 and N1 onsets were 95.78 ms and 105.78 ms post-stimulus, respectively, on both tasks. The latencies did not differ between tasks either ($t[35] = 0$, $p > .05$ for both). This suggest that the processes shared between the tasks did not differ significantly, demonstrating process invariance.

Discussion

Performance monitoring is a cognitive function that encompasses multiple components, including the monitoring and resolution of competing responses, the detection of correct and erroneous responses, and the inhibition of inappropriate responses. The purpose of this study was to validate a behavioural measure of conflict monitoring using the N2 ERP component, clarify the functions of the ERN and Pe components in relation to error awareness and affective processing of errors, and explore the relations of the P3 with neuropsychological measures of inhibition. With respect to hypotheses, 1a) the SST-SRT performance time difference was hypothesized to correlate with the area of the N2 component, and 1b) the P1, N1, and LRP-r latencies would be equivalent between the SRT and SST, implying process invariance and supporting the validity of the SST-SRT difference index; 2) both ERN and Pe areas were hypothesized to negatively correlate with the number of actual and estimated error commissions; 3a) Pe area was hypothesized to correlate either with the estimated frequency (question 1 of the Subjective Performance Questionnaire) and number (question 2) of errors committed and the absolute difference between actual and estimated errors (i.e., the degree of accurate error commission awareness) if the component is a function of error awareness, or 3b) with the degrees of performance and error frustration if the component is related to affective processing of errors; 4a) the P3 area was hypothesized to correlate with SSRT and PSI (motor inhibition measures) and, lastly, 4b) the P3 onset latency was hypothesized to correlate with SSRT. Overall, Hypotheses 1a, 1b, and 4b were supported by the data, and 2, 3a, and 4a were partially supported.

N2 and Conflict Monitoring

N2 and the SST-SRT difference. The premise for the subtraction method is that the difference of a performance measure between two similar tasks – say, the RT difference between simple and choice reaction time tasks – will parse out the common cognitive factors, and that the difference score will reflect the unique component. Consider the comparison between simple and choice reaction time tasks. The former involves stimulus encoding and discrimination, motor preparation, and lastly motor execution. The latter also starts with stimulus encoding and discrimination and ends with motor preparation and execution, but additionally requires conflict monitoring of responses, which is thought to occur after stimulus discrimination but before motor preparation (Gottsdanker & Shragg, 1985). Thus, subtracting the RT of the simple from the choice reaction time task should yield the processing time for conflict monitoring.

This procedure was applied to the SST and SRT to yield the SST-SRT RT difference score. This index is purported to represent response monitoring process unique to the SST, while zeroing out the stimulus encoding and discrimination, and motor preparation and execution processes. Consistent with Hypothesis 1a, the SST-SRT index positively correlated with the area of the N2 component. Participants with larger difference indices took disproportionately longer on the SST relative to the SRT, indicating more time spent resolving the conflict of competing potential responses. As the index correlated significantly with N2 area, it appears to have accurately captured the behavioural expression of conflict monitoring. Additionally, the correlation with the SST CRT was not significant. Evidently, using total trial RTs introduces excessive variability from other cognitive processes (e.g., stimulus encoding, motor preparation) that are inherently included in the overall RT, and the conflict monitoring duration is lost among

noise. This null finding indicates that the difference index is necessary to accurately represent conflict monitoring, as the overall RT confounds it by the inclusion of other processes.

With respect to the relation to processing speed, SDMT scores accounted for some of the variance of N2 area beyond the SST-SRT index. It is possible that a fast processing speed also translates to quicker response conflict resolution. As described above, the conflict monitoring theory posits that the concomitant activation of potential response schemas creates conflict between them, giving rise to the N2 component (Yeung et al., 2004). The greater the conflict between potential responses (e.g., responses that are highly similar), the more pronounced the conflict, and hence the N2. Faster processing speed and, consequently, faster conflict resolution may decrease the time that potential responses compete with one another, thus affecting the N2 component. Of note, SDMT scores were not correlated with the SST-SRT difference. It is possible that the processing speed index is too broad to directly relate to the highly specific cognitive process of conflict monitoring, but, taken together, they account for a larger portion of the N2 component.

Process invariance. A key point to establish validity for the subtraction method is process invariance; that is, subtracting a performance index between two tasks can only provide an accurate index of the unique process if the other, shared processes are equivalent (Sternberg, 1969). If they vary in some fashion (e.g., if a process takes longer for one task), then this discrepancy would contaminate the subtracted index. To consolidate the validity of the SST-SRT index, three shared process components were compared between the two tasks: the P1, N1, and LRP-r. Consistent with Hypothesis 1b, analyses of these component latencies indicated that they occurred at approximately equivalent (and, importantly, not statistically different) times in the SRT and SST. Participants encoded and discriminated the visual stimuli, and prepared to execute

their motor responses at effectively the same time in both tasks. Thus, the longer RTs in the SST appears attributable to conflict monitoring of responses, which was captured by the difference index.

ERN, Pe, Error Awareness, and Affective Processing

Both ERN and Pe areas were negatively correlated with actual errors rates, demonstrating opposite patterns: ERN area increased with higher error rates whereas Pe area decreased. The same pattern emerged with error commission estimates: higher error estimates were associated with larger ERN and smaller Pe areas. To clarify, the opposite interpretations stemming from the same negative correlations relates to the polarity of the ERPs: ERN is negative and larger components are more negative (higher error rates were related to more negative areas, and thus larger ERN components), whereas Pe is positive and larger components are more positive (higher error rates were related to smaller positive areas, and thus smaller Pe components). Furthermore, there was a statistically trending negative correlation between Pe area and error estimation accuracy (the absolute difference between actual and estimated errors). Neither ERN nor Pe were related to frustration with errors or task performance. The results for Pe and ERN are discussed separately within the context of Hypotheses 2, 3a and 3b below.

The Pe results supported Hypothesis 2 that the area would be negatively correlated with actual and estimated error rates. One possible explanation for this is that the smaller Pe components related to higher error rates reflect habituation, in that the component is mitigated after repeated error commissions. Alternately, the reverse could be the case, in that individuals who are less aware of errors (reflected by smaller Pe components) may be more prone to make errors. The latter argument is supported by the finding that actual error rates were highly

correlated with the absolute difference between actual and estimated errors; put differently, participants who were less accurate at estimating errors actually committed more errors. In particular, those who underestimated their error commissions had the highest error rates. This is also partially supported by the trending correlation between absolute actual-estimated error difference and Pe area, which showed that more inaccurate estimates were associated with smaller Pe components. These findings also support Hypothesis 3a, and are congruent with some of the literature demonstrating decreased magnitude with higher rates of errors (Dywan et al., 2004; Falkenstein et al., 2000; but see Hajcak et al., 2004; Herrmann et al., 2004). Some inconsistencies notwithstanding, the significant correlations between Pe magnitude and error estimates, in conjunction with the supporting literature, suggests that Pe may be related to error awareness, as per the error-awareness theory (Nieuwenhuis et al., 2001). Conversely, the finding that Pe was unrelated to the affective responses to performance does not support the affective processing theory (Hypothesis 3b). This is congruent with some literature showing no relation between affect and Pe (Clayson, Clawson, & Larson, 2012; Wiswede, Münte, & Rüsseler, 2009). Taken together, these findings suggest that a decreased awareness of error commission is associated with greater error commission.

In contrast to both the Pe results and much of the extant literature (e.g., Amodio et al., 2007; Hajcak et al., 2003), larger ERN magnitudes were related to higher actual and estimated error rates. A few speculative explanations may be provided for these results. One, for the most part, errors are quite salient and may result in strong error processing. Though participants sometime made mistakes on the Go trials, the vast majority of errors occurred on Stop trials. The stop signals are difficult to ignore, and it is reasonable to expect that participants would notice whether they respond erroneously during or after a signal. Due to the relative significance of

these errors, error monitoring and processing (and the related ERN) may subsequently be enhanced. In support of this, other studies have demonstrated that inflating error significance by pairing them with punishments enhanced ERN components (Endrass et al., 2010), and individuals who benefitted more from negative feedback had larger ERNs (Frank, Woroch, & Curran, 2005). However, if error significance is related to ERN magnitude, then this begs the question of why the same correlation did not occur with Pe, given its purported function of error awareness. Though error processing/correction and awareness are not necessarily linked (i.e., individuals often correct mistakes before they become aware of them; Rabbit, 2002), it is unclear whether the salient stop signal would not have the same orienting effect on Pe as it did for ERN. Two, the SST maintains a relatively high level of difficulty by dynamically adjusting the delay of the stop signal (i.e., it increases the length of the delay after a successful inhibition, making the task harder). Task difficulty, particularly in response conflict-type tasks, has been shown to enhance ERN (Mathews, Perez, Delucchi, & Mathalon, 2012), and it is possible that the consistent difficulty of the task ensured large neural responses to errors. This factor differentiates this study from much of the literature that tends to use other tasks (e.g., flanker, Go/No-Go). Third, although previous reports have found smaller ERNs with higher errors rates, the relatively low number of errors produced in the SST may have mitigated this effect. To provide an approximate quantification, each block of the SST contains 24 stimuli. Stop signals occur on 25% of trials (six of 24), and the stop delay algorithm attempts to ensure a 50% failure rate, resulting in, on average, three errors per block. Of course, errors will vary with each block and participant, and many more errors are accrued over the multiple blocks; nevertheless, error rates are fairly low. It is possible that previous studies that found an inverse relationship between ERN magnitude and error rates did so because those tasks produced higher error rates in general

compared to the SST (e.g., Nieuwenhuis et al., 2003 and Amodio et al., 2007 reported error rates of approximately 30%). Thus, the relatively low number of errors produced on the SST may not have been sufficient to dampen the ERN.

P3 and Inhibition

As expected (Hypothesis 4a), the P3 component area was correlated with shorter SSRTs, indicating that participants with more efficient inhibition (shorter SSRTs) had larger P3s. This result is congruent with the findings of a host of other studies (e.g., Etchell, Sowman, & Johnson, 2012; Greenhouse & Wessel, 2013; Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004) and suggests that more efficient and/or successful inhibition is associated with larger P3 components. Surprisingly, and not consistent with Hypothesis 4a, P3 areas were unrelated to PSI. Given its association with superior inhibition, a similar relation might have been expected with higher PSI indices. However, given that the SST algorithm attempts to maintain a 50% failure rate on Stop trials (the actual PSI was 64.63 [10.25]; the algorithm is not perfect, and behavioural adaptation may have occurred throughout the task), the variation in the data may have been insufficient for a statistical correlation with P3 magnitude.

Congruent with Hypothesis 4b and the results of Wessel and Aron (2015), P3 onset latencies were correlated with SSRT. This demonstrates that participants who were faster, and more effective, at inhibiting their responses had earlier P3 onsets. The implication is that successful motor inhibition is indeed linked with the magnitude and onset of P3 (as area and onset were highly correlated, and the combination of the two did not explain additional SSRT variance, the relationship between SSRT and area and onset likely have the same interpretations). If the P3 component was a broader inhibitory process that encompassed more than just the suppression of a behavioural output (or a different function altogether), then the

onset would not be expected to correlate so closely with the SSRT. A potential counterargument is that the relation between P3 onset and SSRT is an artifact of general performance time, and that P3 onsets occur earlier simply because of shorter trials; indeed, RT is half of the SSRT equation (CRT minus mean stop delay) and the correlation could be related to the variance in RT, not the stop delay. A small SSRT can be obtained in two ways: if the CRT is small, or the stop delay is large. To illustrate, consider two hypothetical participants, one with a mean CRT of 1000 ms and mean stop delay of 500 ms, and the other with a mean CRT of 800 ms and mean stop delay of 300 ms. Calculating the SSRT for both yields equal numbers: 500 ms (1000 – 500, and 800 – 300). Ostensibly, both participants have equally efficient inhibition, mathematically speaking; however, the participant with the longer stop delay has superior inhibition due to the ability to withhold responses at longer delays. To test this empirically, P3 onset was correlated with mean CRT, but the effect was not significant. This dissociation demonstrates that the P3 onset is in fact related to inhibition efficiency, not general response speed.

Despite a significant history linking the P3 component to inhibition, unequivocal consensus on its functional significance has not been reached. For instance, P3 has been proposed to indicate evaluation of the inhibitory process and/or outcome rather than the inhibitory process itself (Huster et al., 2013), and also an index of conflict monitoring (Bruin et al., 2001; Smith et al., 2007). To test the former hypothesis, the relation between P3 area and question 4 of the subjective performance questionnaire (frustration with errors) was examined. If larger P3 components indicated positive affect following good outcomes; in this case, successful inhibitions; then the P3 area would be expected to correlate negatively with higher scores on this question. However, this was not the case as the results were not significant, suggesting that the P3 was not linked with outcome evaluation. It should be noted that this analysis only

approximated the relationship between affective/outcome evaluation and P3 magnitude. To examine this more precisely, questions related to how satisfied participants felt after they successfully inhibited a response should be recorded. Nevertheless, this finding provides preliminary evidence against the outcome evaluation theory. As for the theory that P3 reflects conflict monitoring, this was not supported either as P3 area was not correlated with either the SST-SRT difference or N2 areas and, conversely, N2 areas were not correlated with SSRT. These findings support the functional distinction between the P3 and N2 components as indices of inhibition and conflict monitoring, respectively.

Limitations

Though process invariance was purportedly established between the SRT and SST, there were important task differences (e.g., a single response, the lack of inhibition) that could have affected their comparability. As demonstrated by Danek and Mordkoff (2011), increasing uniformity in task requirements may provide more accurate comparison of motor invariance, after which motor invariance between the tasks no longer remained. The bearing of those findings on the SST-SRT comparison is unclear, as modified versions of the SST and other RT tasks were used by Danek and Mordkoff. Differences in task design notwithstanding, the task differences in the present study may have influenced the conflict monitoring index in untested ways. This issue may be explored by using tasks that only differ on the response type (single versus forced choice).

The questions in the subjective performance questionnaire may also have insufficiently captured the constructs they were meant to measure. For consistency, the questionnaire was based on the one used by Stern and colleagues (2010). However, some of the questions may have

been too vague; questions 1, 3 and 4, in particular. Question 1 was meant to provide supplemental information to question 2, which asked participants to estimate the number of errors they committed. However, question 1 did not correlate with question 2, nor did it correlate with the number of actual errors, suggesting that it failed to capture the accuracy of error awareness. Questions 3 and 4 were meant to capture the affective responses to general performance and error commission. Though question 3 was fairly straightforward ("Were you ever frustrated with your performance?"), it may have been more precise to ask the degree of frustration (e.g., "How frustrated were you with your performance?"). Furthermore, participants may have experienced emotions other than frustration, which were not represented by this question. Question 4, on the other hand, may have been too broad. Though it was meant to elicit feedback on affective responses to errors, this may have been obfuscated by the additional question related to attentional control ("…Did you find it hard to get back on track?"). A more focused question relating to affective responses may have captured this construct more effectively (e.g., "How frustrated did you feel when you made a mistake?").

With respect to sample demographics, there was a bias towards female participants (approximately two-thirds of the sample), which may have influenced the results. For instance, the magnitude of the ERN component has been shown to be sex-dependent, with somewhat larger amplitudes in males than females (possibly an artifact of RT differences between sexes, or differential engagement of cognitive control to achieve similar performance; Larson et al., 2011). However, given the focus on within-subject analyses, this bias likely did not significantly influence the results. Furthermore, independent t-tests comparing ERP components did not indicate any differences between males and females (t[33] ranged from 0.021 to 1.98, all $p >$ .05).

Lastly, although EEG has excellent temporal precision (256 Hz in this case, though it can vary according to EEG processing parameters), its spatial precision is lacking compared to other neuroimaging methods. EEG detects activity from large groups of neurons, rather than small clusters. Moreover, because multiple brain regions can contribute to ERP components, detecting activity from subcortical structures is substantially less accurate than that of surface activity, as more and more regions could contribute to a component as the signal travels to the surface. Cortical folding patterns can also influence the ability for potentials to travel to surface detectors, which accounts for some of the differences observed between participants (Luck, 2014). ICA or principal component analysis procedures can assist with more precise source localization, though they too are unable to accurately link activity to deeper structures. Techniques such as fMRI are better suited for spatial precision, though they lack the temporal precision of EEG.

Future Directions

Although the conflict monitoring index was supported in this study, it will require replication to establish its validity. Though the SST was a convenient task as it required both conflict monitoring of responses and inhibition, the involvement of multiple processes may have influenced the results. As such, alternate choice RT tasks should be used to confirm validity (e.g., no inhibition requirements in either task).

Though the Stroop task is a test of cognitive inhibition, it too may rely on conflict monitoring. Specifically, the words on the colour-word trial invoke two potential responses: processing and reading either the word or the ink colour. Indeed, some research points to the N450, a negative ERP that occurs at fronto-central scalp locations, likely stemming from ACC activity (similar to the N2), following stimulus presentation (Liotti et al., 2000; West, Jakubek,

Wymbs, Perry, & Moore, 2005). Further, the N450 is larger on incongruent than congruent trials (e.g., colour-word versus word trials). Thus, it is possible that conflict monitoring between the two competing responses occurs on the Stroop task as well, and may be amenable to the subtraction method. Paradigms that focus on establishing process invariance, a subtraction method applied to the Stroop variables, and a link with the N450 (or potentially N2) could help establish the applicability of a behavioural conflict monitoring index.

As with the literature on normally functioning individuals, links between ERP and behavioural data is limited in patient populations. For instance, patients with OCD are sometimes slower (Kaczkurkin, 2013; Ursu, Stenger, Shear, Jones, & Carter, 2003) and commit fewer errors (Riesel, Endrass, Kaufmann, & Kathmann, 2011; Riesel, Kathmann, & Endrass, 2014) than healthy controls on SST, Go/NoGo, and flanker tasks, though performance differences are often not observed (e.g., Carrasco et al., 2013; Endrass et al., 2014; Klawohn, Riesel, Grützmann, Kathmann, & Endrass, 2014). However, it is unclear whether the increased RT found by Kaczkurkin et al. and Ursu et al. reflects an overall performance slowing or a specifically increased latency of conflict monitoring (i.e., SST-SRT difference). Further, many studies base group comparisons on peak amplitude, which is not the ideal method of quantifying ERPs as it loses timespan information (please refer to the EEG analysis section for a discussion of this issue). Component latency or area (the latter takes into account both the size and timespan of the component) may be a better method for both group comparisons of N2 and relating it to behavioural measures. This area of the literature could be expanded in (at least) two ways: using more accurate measurement methods (e.g., using area rather than peak amplitude) could improve the quantification of the components, and thus more accurate group comparisons. Additionally, clarifying the reason for slowed RT in OCD populations (and potentially other patient

populations as well) could better define their deficits; whether their slower performance is due to generally slowed processing, or specifically slower conflict monitoring. This could, potentially, inform treatment approaches utilizing cognitive rehabilitation methods by focusing on either processing speed or response conflict monitoring (e.g., training for expedient and accurate response selection).

Previous studies have observed altered performance monitoring ERPs in different psychopathologies. For instance, compared to healthy controls, the ERN is enlarged in patients with OCD (Endrass, Klawohn, Schuster, & Kathmann, 2008; Endrass et al., 2014; Riesel et al., 2011; Stern et al., 2010), and there is mixed evidence for N2 with a few studies reporting smaller N2 components (Keskin-Ergen et al., 2014; Kim, Kim, Yoo, & Kwon, 2007), with another finding a larger N2 (Ruchsow et al., 2007). Michelini and colleauges (2016) found that the N2 component was enhanced in patients with bipolar disorder compared to controls. A meta-analysis of ERN and CRN in a variety of anxiety disorders (e.g., OCD, generalized anxiety disorder and subclinical anxiety) had larger components relative to healthy controls (Moser, Moran, Schroder, Donnellan, & Yeung, 2013). Data on the P3 are mixed regardless of the disorder. Some studies found either increased (Johnstone, Barry, & Clarke, 2007; Michelini et al., 2016) or decreased (Tye et al., 2014) P3 in children with ADHD compared to controls; either smaller (Hughes, Fulham, Johnston, & Michie, 2012) or equivalent (Neuhaus et al., 2010) P3 in patients with schizophrenia versus controls; and reduced (Herrmann et al., 2003; Thomas, Gonsalvez, & Johnstone, 2014), larger (Fan et al., 2014), or equivalent (Keskin-Ergen et al., 2014; Kim et al., 2007; Ruchsow et al., 2007) in patients with OCD versus controls. The latter finding is somewhat surprising given the deficient inhibition model of OCD (Chamberlain et al., 2005).

There is a great potential to study performance monitoring ERPs in patient populations, but the different interpretations of the components hinders the ability to detect group differences and compare findings between studies (please refer to the performance monitoring ERP summaries section for a discussion of this issue). Consolidation of the theories could help resolve this issue and support the study of, for example, presentation of inhibition deficits and the significance of enhanced ERNs in OCD. There is also a potential to use ERPs as proxies of treatment efficacy. For instance, pharmacological treatment of ADHD appears to increase the inhibition P3 (Groom et al., 2010; Janssen et al., 2016; Logemann, Böcker, Deschamps, Kemner, & Kenemans, 2013). However, ERPs are not always influenced by treatment, as ERN was not altered after cognitive behavioural therapy for OCD (Hajcak, Franklin, Foa, & Simons, 2008). Future studies could examine the link between ERPs and treatment efficacy in different disorders to potentially expand the repertoire of ERP markers.

Conclusions

Using the subtraction method to obtain the performance time difference between the simple RT and a choice RT (SST) tasks, support was found for a behavioural index of conflict monitoring by relating it to the N2. This link was bolstered by determining process invariance between the tasks (i.e., all common cognitive processes occurred at the same latencies), and doing so helped establish its validity. This measure offers the potential to be used as a simple and accessible index of conflict monitoring, without the need for neuroimaging methods. Replication and the application in patient populations can further support its validity. Additionally, important findings regarding error processing and awareness, and inhibition – all aspects of performance monitoring – were replicated and extended. Specifically, the function of Pe in error awareness,

rather than affective processing, was partially supported by its association with subjective error estimates but not degree of frustration with errors. Unexpectedly, the ERN was positively correlated with error rates, possibly due to low error rates, error salience, or task difficulty. Lastly, the role of P3 in inhibition was supported by its link with the SSRT but not performance evaluation or conflict monitoring. These findings helped strengthen theories by elucidating conflicting points in the literature.

Appendix A

Ryerson University Consent Agreement

Study Participants/Consent/2015

TITLE OF THE STUDY:
Neural Correlates of Performance Monitoring

You are being asked to participate in a research study being carried out at Ryerson University. Before you give your consent to participant in the study, it is important that you read the following information and ask as many questions as necessary to be sure you understand what you will be asked to do.

INVESTIGATORS:

Tisha Ornstein, Ph.D., C. Psych., (Principal Investigator), Department of Psychology, Ryerson University
Peter Egeto, HBSc., (Project Administrator), Department of Psychology, Ryerson University
Ceilagh MacDonald, BSc., (Research Assistant), Department of Psychology, Ryerson University
Eleenor Abraham, HBA., (Research Assistant), Department of Psychology, Ryerson University

PURPOSE OF THE STUDY

The aim of this study is to help researchers evaluate the unique links between cognition and brain activity in healthy individuals and patients with obsessive-compulsive disorder (OCD). OCD is a mental disorder characterized by intrusive thoughts, impulses, or repetitive behaviours; it is also associated with unique cognitive impairments. We hope to recruit at least 25 healthy participants and 25 OCD patients.

DESCRIPTION OF THE STUDY

If you agree to participate, you will come to the Psychology Research & Training Center at 105 Bond Street on the day of your appointment. You will be working with one of the members of the research team in a quiet test room. We will ask you if you consent (agree) to participate in the research, and a brief questionnaire assessing eligibility for the study will be administered (age 18+, no current psychiatric, medical or neurological disorder, not currently taking prescription medication). You will then be asked to complete a series of paper-and-pencil and computerized tests of neurocognitive functions, such as memory, attention, and thinking abilities. For example, in one task, the experimenter will ask you to match a card with a particular symbol on it to one of four key cards. This task tells us about your ability to think flexibly. You will also be asked to complete some questionnaires that ask about your emotional status. For example, you may be

asked to indicate how much you have been bothered by a particular anxiety symptom during the past month. You will be expected to come to Ryerson University for one visit that will take 2 hours. During the study, brain activity while performing a cognitive task will be monitored using electroencephalography (EEG). This will involve wearing a cap connected to multiple wires. After putting the cap on the head, the wires will be attached onto the cap, and a gel will be applied to each wire to enhance the detection of brain electrical activity. After the cognitive task is completed, the wires and the cap will be removed, and a sink and a towel will be provided to help clean out any remaining gel from the hair. Please note that you may refuse any portion of the study, including the EEG component.

RISKS OR DISCOMFORTS

There are no known harms associated with participation in this study; risk is minimal. The risk, if any, will be no greater than that encountered in day-to-day activities. You will be exposed to paper-and-pencil and computerized tasks, as well as electroencephalographic equipment, which involves a cap with wires connected to it. You may experience discomfort from having to sit relatively still for an hour during the EEG testing.

BENEFITS OF THE STUDY

You will not receive any direct benefits from participating in this study. However, upon request, you can receive a summary of your results. We expect to gain a deeper understanding of neurocognitive and brain functioning in healthy individuals and OCD.

CONFIDENTIALITY AND DATA STORAGE

To ensure confidentiality, all documents containing participant names and/or contact information will be stored separately from the data. All physical copies of the data will be stored in a locked cabinet in the research lab, and access will be restricted to research investigators. Computerized data will also be deidentified and encrypted, accessible only by the research investigators. The data will be kept in secure storage for a two-year period, after which it will be destroyed by the principle investigator. The results of the experiment will be used solely for research purposes.

INCENTIVES TO PARTICIPATE

You will be compensated $20 for participating in this study, once eligibility is confirmed.

COSTS AND/OR COMPENSATION FOR PARTICIPATION

There are no costs associated with participation.

VOLUNTARY NATURE OF PARTICIPATION

Participation in this study is voluntary. Your choice of whether or not to participate will not influence your future relations with Ryerson University. If you decide to participate, you are free to withdraw your consent and to stop your participation at any time without penalty or loss of benefits to which you are allowed. Further, at any particular point in the study, you may skip any question for whatever reason.

QUESTIONS ABOUT THE STUDY

If you have any questions about the research now, please ask. If you have questions later about the research, you may contact:

Peter Egeto, MA student
Project Administrator
416-979-5000 x 4988

If you have questions regarding your rights as a human subject and participant in this study, you may contact the Ryerson University Research Ethics Board for information.

Research Ethics Board
c/o Office of the Vice President, Research and Innovation, Ryerson University
350 Victoria St., Toronto, ON, M5B 2K3
416-979-5042
rebchair@ryerson.ca

AGREEMENT

Your signature below indicates that you have read the information in this agreement and have had a chance to ask any questions you have about the study. Your signature also indicates that you agree to be in the study and have been told that you can change your mind and withdraw your consent to participate at any time. You have been given a copy of this agreement.

You have been told that by signing this consent agreement you are not giving up any of your legal rights.

_____
Name of Participant (please print)

_____ _____
Signature of Participant Date

_____ _____
Signature of Investigator Date

Appendix B

ID #: _____ Date: _____

DOB: _____

Age: _____

Gender: _____

Ethnicity: Caucasian Black Hispanic Asian South Asian Other:_____

Highest education level attained: _____

Years of education completed: _____

First language: _____

English fluency: YES NO

Handedness: RIGHT LEFT

Colour blind: YES NO

History of neurological disorders (e.g., stroke, traumatic brain injury): _____

General medical conditions (e.g., diabetes, cardiovascular disease): _____

Prescription medication (e.g., insulin, thryroid medication): _____

Appendix C

Subjective Performance Questionnaire

1. Did you make any mistakes?

1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
None				Sometimes					All the time

2. How many mistakes do you think you made?

_____

3. Were you ever frustrated with your performance?

1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Not at all				Sometimes					All the time

4. When you made a mistake, were you flustered? Did you find it hard to get back on track?

1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Not at all				Sometimes					All the time

Appendix D

Telephone Screen for Normal Controls[4]

The purpose of this telephone interview is to screen individuals for the major DSM-IV disorders to ensure that they have no history of these problems. Eligible participants will be offered the opportunity to participate in our research as control subjects.

Date of Interview: _____ Interviewer: _____

Participant's Name: _____

Address:_____

_____

_____

Home Telephone: _____ Work Telephone: _____

Questionnaires Sent: _____ Questionnaires Completed:_____

General Questions

1. *Have you ever had any emotional difficulties such as severe anxiety, depression, excessive alcohol or drug use, or an eating disorder*?

_____ No

_____ Yes (please explain _____

_____)

[4] Note: The name, address, and telephone numbers were not obtained to maintain confidentiality. They were left in to preserve the original, published format of the interview.

2. *Have you ever seen a psychiatrist, psychologist, or other professional for psychotherapy, counseling, or medication to deal with stress or emotional difficulties*?

_____ No

_____ Yes (please explain _____

_____)

3. *Have you ever been hospitalized for emotional difficulties*?

_____ No

_____ Yes (please explain _____

_____)

Comments

_____

_____

_____

_____

Revised October 17, 1996

Panic Disorder

1. *Have you ever had a panic attack, when you suddenly felt frightened, or anxious or suddenly developed a lot of physical symptoms?*

   _____ No (go to "Social Phobia")

   _____ Yes (please explain _____)

2. *Have these attacks ever come out of the blue - in situations where you didn't expect to feel uncomfortable*?

   _____ No (go to "Social Phobia")

   _____ Yes (please explain _____)

3. *How many of these kinds of attacks have you had*? _____

4. How frequent were these attacks when they were worst? _____

5. *Did you ever avoid situations because you might have one of these attacks?*

   _____ No

   _____ Yes (please explain _____)

6. *Did it ever bother you that you had these attacks?*

   _____ No

   _____ Yes (please explain _____)

7. *Did these attacks ever interfere with your life (e.g., at work, school, leisure, or with friends and family)?*

_______ No

_______ Yes (please explain ____)

◆◆

Presence of recurrent unexpected panic attacks: _____ No

_____ Yes

Presence of distress or functional impairment: _____ No

_____ Yes (rule out of study)

Social Phobia

1. *Have you ever been afraid or nervous about being in situations where you might be noticed, evaluated, or judged by others, such as parties, meetings, public speaking, talking to strangers, eating in public, or other social situations?*

_______ No (go to "Specific Phobia")

_______ Yes (please explain ____)

2. *Did it bother you that you felt uncomfortable in these situations?*

_______ No

_______ Yes (please explain ____)

3. *Did your social anxiety ever interfere with your life (e.g., at work, school, leisure, or with friends and family)?*

_______ No

_______ Yes (please explain ____)

◆◆◆

Presence of excessive social anxiety: _____ No

_____ Yes

Presence of distress or functional impairment: _____ No

_____ Yes (rule out of study)

Specific Phobia

1. *Have you ever been afraid or nervous when exposed to other specific objects or situations, such as animals (e.g., snakes, bugs, mice, dogs, cats, etc.), seeing blood, getting injections, dentists, heights, storms, water, flying, driving, or enclosed places?*

_______ No (go to "Obsessive Compulsive Disorder")

_______ Yes (please explain ____)

2. *Did it bother you that you felt uncomfortable in this situation?*

_______ No

_______ Yes (please explain ____)

3. *Did your fear of _______________ ever interfere with your life (e.g., at work, school, leisure, or with friends and family)?*

_______ No

_______ Yes (please explain ____)

◆◆◆

Presence of an excessive specific fear: _____ No

_____ Yes

Presence of distress or functional impairment: _____ No

_____ Yes (rule out of study)

Obsessive Compulsive Disorder

1. *Have you ever been bothered by thoughts that didn't make sense and kept coming back to you even when you tried not to have them, such as thoughts of hurting someone even though you didn't want to or thoughts of being contaminated by germs or dirt?*

_______ No (go to OCD question no. 4)

_______ Yes (please explain ____)

2. *How often do these thoughts occur?* _________________

3. *How long do they last?* _________________

4. *Was there ever anything that you had to do over and over again and couldn't resist doing, like washing your hands again and again, counting up to a certain number, or checking something several times to make sure that you'd done it right?*

_______ No (go to "Posttraumatic Stress Disorder" if response to OCD question no. 1 was "No")

_______ Yes (please explain ____)

5. *How often did it happen?* ________________

6. *Did it bother you that you had these thoughts or behaviours?*

_______ No

_______ Yes (please explain ____)

7. *Did these repetitive thoughts or behaviours ever interfere with your life (e.g., at work, school, leisure, or with friends and family)?*

_______ No

_______ Yes (please explain ____)

◆◆◆

Presence of obsessions or compulsions: _____ No

_____ Yes

Presence of distress or functional impairment: _____ No

_____ Yes (rule out of study)

Posttraumatic Stress Disorder

1. *Sometimes things happen to people that are extremely upsetting -- things like being in a life-threatening situation like a major disaster, very serious accident or fire; being physically assaulted or raped; seeing another person killed or dead, or badly hurt, or hearing of something horrible that has happened to someone you are close to. At any time in your life, have any of these kinds of things ever happened to you?*

_____ No (go to "Generalized Anxiety Disorder")

_____ Yes (please explain _____)

2. *Did this event lead to any difficulties with severe anxiety or fear?*

_____ No (go to "Generalized Anxiety Disorder")

_____ Yes (please explain _____)

3. *How long did the problems last?* _____

4. *Did the anxiety or fear that followed this event ever interfere with your life (e.g., at work, school, leisure, or with friends and family)? Or did it bother you that you developed this fear?*

_____ No

_____ Yes (please explain _____)

If "Yes" to items 1, 2, and 4

Rule out of Study: _____ No

_____ Yes

Generalized Anxiety Disorder

1. *Have you ever had a period of six months or longer in which you were bothered by excessive worry about a variety of things, such as work, finances, family, health, or day to day matters?*

_____ No (go to "Major Depressive Episode")

_____ Yes (please explain _____)

2. How long did this period last? _____

3. *Did you tend to worry even when everything was okay?*

_____ No

_____ Yes (please explain _____)

4. *Was your worry out of proportion to the situation?*

_____ No

_____ Yes (please explain _____)

5. *Did you worry more days than not?*

_____ No

_____ Yes (please explain _____)

6. *Did it bother you that you worried so much?*

_______ No

_______ Yes (please explain ____)

7. *Did the anxiety and worry interfere with your life (e.g., at work, school, leisure, or with friends and family)?*

_______ No

_______ Yes (please explain ____)

◆◆◆

Presence of excessive worry for required period: _____ No

_____ Yes

Presence of distress or functional impairment: _____ No

_____ Yes (rule out of study)

Major Depressive Episode

1. *Was there ever a time in your life, when you felt depressed, down, or uninterested in almost all of your usual activities for most of the day, nearly every day, for at least two weeks?*

_______ No (go to "Manic Episode")

_______ Yes (please explain ____)

2. *Which of the following symptoms did you experience during the depressed period (check)?*

_____ Depressed mood

_____ Lack of interest in almost all of your usual activities

_____ Significant appetite changes

_____ Sleep difficulties

_____ Being extremely restless or slowed down

_____ Fatigue

_____ Feeling worthless or guilty

_____ Impaired concentration

_____ Suicidal ideation

<u>Presence of 5 symptoms?</u> _____ No (go to "Manic Episode")

_____ Yes

3. *Did the depression happen after someone close to you died?*

_______ No

_______ Yes (go to "Manic Episode")

4. *Did it bother you that you were feeling depressed?*

5. *Did the depression interfere with your life (e.g., at work, school, leisure, or with friends and family)?*

◆◆◆

Presence of depression (5 symptoms) for two weeks: _____ No

_____ Yes

Presence of distress or functional impairment: _____ No

_____ Yes (rule out of study)

Manic Episode

1. *Have you ever had a period of time when you were feeling so good, high, excited or hyper that other people thought you were not your normal self or so hyper that it got you into trouble?*

_______ No (go to question no. 2)

_______ Yes (please explain ____)

2. *Did you ever have a period when you were so irritable that you found yourself shouting at people you didn't know well or starting fights or arguments?*

_______ No (go to "Psychotic Symptoms" if response to question no. 1 was "No")

_______ Yes (please explain ____)

3. *How long did this period last?* ________________

◆◆

Manic Episode Likely: _____ No

_____ Yes (rule out of study)

Psychotic Symptoms

1. *Have you ever had any strange or unusual experiences such as hearing voices or seeing visions that other people could not hear or see?*

_______ No

_______ Yes (please explain ____)

2. *Did you ever have any unusual beliefs such as believing that others were plotting against you, that you had special powers that others did not have, or that you were receiving special messages from the TV, radio, newspaper, or the way things were arranged around you?*

_______ No

_______ Yes (please explain ____)

◆◆

Psychotic Symptoms Likely: _____ No

_____ Yes (rule out of study)

Substance Abuse

1. *How much alcohol do you drink?* _______________

2. *Have you ever used illegal or recreational drugs on a regular basis?*

_______ No

_______ Yes (please explain ____)

3. *Have other people expressed concern about your use of alcohol or drugs?*

________ No

________ Yes (please explain ____)

4. *Do you get annoyed if others comment on your alcohol or drug use?*

________ No

________ Yes (please explain ____)

5. *Have you ever felt guilty about your use of alcohol or drugs?*

________ No

________ Yes (please explain ____)

6. *Have you ever used alcohol or drugs first thing in the morning to feel comfortable?*

________ No

________ Yes (please explain ____)

7. *Has alcohol or drug use ever led to problems for you, such as conflicts with other people, problems with the law, or difficulties at work or school?*

________ No

________ Yes (please explain ____)

◆◆

Alcohol or Drug Problems Likely: ______ No

______ Yes (rule out of study)

Eating Disorders

Have you ever had significant difficulties with your eating, such as:

(1) weighing much less than others thought you ought to weigh, due to a fear of gaining weight

(2) recurrent binge eating (i.e., eating very large amounts of food within two hours)

_______ No

_______ Yes (please explain ____)

◆◆

Eating Disorder Likely: _____ No

_____ Yes (rule out of study)

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