Cortical and Autonomic Modulation of Attentional Control

By

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Abstract

Whereas the role of the anterior cingulate cortex (ACC) in cognitive control has received considerable attention, much less work has been done on the role of the ACC in autonomic regulation. Its connections through the vagus nerve to the sinoatrial node of the heart are thought to exert modulatory control over cardiovascular arousal. Therefore, ACC is not only responsible for the implementation of cognitive control, but also for the dynamic regulation of cardiovascular activity that characterizes healthy heart rate and adaptive behaviour. However, cognitive control and autonomic regulation are rarely examined together. Moreover, those studies that have examined the role of phasic vagal cardiac control in conjunction with cognitive performance have produced mixed results, finding relations for specific age groups and types of tasks but not consistently. So, while autonomic regulatory control appears to support effective cognitive performance under some conditions, it is not presently clear just what factors contribute to these relations.

The goal of the present study was, therefore, to examine the relations between autonomic arousal, neural responsivity, and cognitive performance in the context of a task that required ACC support. Participants completed a primary inhibitory control task with a working memory load embedded. Pre-test cardiovascular measures were obtained, and on-task ERPs associated with response control (N2/P3) and error-related processes (ERN/Pe) were analyzed.

Results indicated that response inhibition was unrelated to phasic vagal cardiac control, as indexed by respiratory sinus arrhythmia (RSA). However, higher resting RSA was associated with larger ERN amplitude for the highest working memory load condition. This finding suggests that those individuals with greater autonomic regulatory control exhibited

more robust ACC error-related responses on the most challenging task condition. On the other hand, exploratory analyses with rate pressure product (RPP), a measure of sympathetic arousal, indicated that higher pre-test RPP (i.e., more sympathetic influence) was associated with more errors on "catch" NoGo trials, i.e., NoGo trials that simultaneously followed other NoGo trials, and consequently, required enhanced response control. Higher pre-test RPP was also associated with smaller amplitude ERNs for all three working memory loads and smaller amplitude P3s for the low and medium working memory load conditions. Thus, higher pre-test sympathetic arousal was associated with poorer performance on more demanding "catch" NoGo trials and less robust ACC-related electrocortical responses.

The findings from the present study highlight the interdependence of electrocortical and cardiovascular processes. While higher pre-test parasympathetic control seemed to relate to more robust ACC error-related responses, higher pre-test sympathetic arousal resulted in poorer inhibitory control performance and smaller ACC-generated electrocortical responses. Furthermore, these results provide a base from which to explore the relation between ACC and neuro/cardiac responses in older adults who may display greater variance due to the vulnerability of these systems to the normal aging process.

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Dedication

This thesis is dedicated to my Grandma,
who inspired me more than she ever knew.

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Abbreviations

ACC	anterior cingulate cortex
ANS	autonomic nervous system
BESA	brain electrical source analysis
BOLD	blood oxygenation level dependant
BP	blood pressure
CRN	correct-related negativity
CV	cardiovascular
dACC	dorsal anterior cingulate cortex
DLPFC	dorsolateral prefrontal cortex
ECG	electrocardiogram
EEG	electroencephalography
ERN	error-related negativity
ERP	event-related potential
fMRI	functional magnetic resonance imaging
HR	heart rate
HRV	heart rate variability
IBI	interbeat interval
IC	inhibitory control
LORETA	low-resolution electromagnetic tomography
OFC	orbitofrontal cortex
Pe	error positivity
PFC	prefrontal cortex

RPP	rate pressure product
RSA	respiratory sinus arrhythmia
RT	response time
SBP	systolic blood pressure
SCOLP	speed and capacity of language processing test
vACC	ventral anterior cingulate cortex
WM	working memory
WMIC	working memory load inhibitory control task

Introduction

Every day, human beings are bombarded by countless sensory stimuli. To function adaptively in such a complex, often confusing environment, individuals must be able to select relevant information from surrounding distracters and maintain this information in the pursuit of goal-directed behaviours (Miller & Cohen, 2001). This type of cognitive control is essential for efficient processing of information, appropriate allocation of attentional resources, and effective working memory performance (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Ullsperger & von Cramon, 2004)

Numerous studies highlight the importance of the medial frontal brain regions, especially the anterior cingulate cortex (ACC), in the implementation of cognitive control (Ullsperger & von Cramon, 2004; Devinsky, 1995). For instance, empirical evidence suggests that the ACC is involved in attention allocation (Fallgatter, Bartsch, & Herrmann, 2002), executive functions (Carter, Botvinick, & Cohen, 1999), and reward-guided decision making (Amiez, Joseph, & Procyk, 2005; Kennerley, Walton, Behrens, Buckley, & Rushworth, 2006). In addition, increased ACC activity has been observed during attentionally demanding conditions that require participants to selectively attend to relevant information and inhibit responding to irrelevant distracters (Hester, Murphy, & Garavan, 2004). So, it is clear that the ACC plays a critical role in the cognitive control necessary for successful performance on tasks that require effortful attention.

In addition to its role in cognitive control, the ACC also plays a major role in autonomic nervous system (ANS) activity (Baird et al., 2006; Critchley et al., 2003; Luu & Posner, 2003; Matthews, Paulus, Simmons, Nelesen, & Dimsdale, 2004). Through its connections with the vagus nerve to the sinoatrial node of the heart, the ACC exerts

modulatory control over cardiovascular activity (Critchley et al., 2003). Although many studies have investigated the role of autonomic arousal in emotion regulation (Movius & Allen, 2005; Porges, Doussard-Roosevelt, Portales, & Suess, 1994; Thayer, Friedman, & Borkovec, 1996; Thayer & Lane, 2000) and cardiovascular health (Thayer & Lane, 2007; Thayer & Ruiz-Padial, 2006), relatively few have focused on the relationship between autonomic regulation and cognitive functioning. This relationship warrants further investigation as recent studies have demonstrated the importance of the ACC in generating autonomic states of arousal that are adaptive for successful cognitive control (Critchley et al., 2003; Luu & Posner, 2003; Matthews et al., 2004). However, the relationship between autonomic arousal and cognitive performance has not always reliably occurred across all age groups and all tasks. For instance, whereas some researchers have observed a relationship between autonomic regulation and cognitive performance in younger adults (Hansen, Johnson, & Thayer, 2003), others have only observed this relationship among older adults (Dywan, Mathewson, & Segalowitz, submitted). Furthermore, while autonomic regulation has been shown to affect performance on executive function (Hansen et al., 2003) and source memory tasks (Dywan et al., submitted), it does not appear to be associated with performance on perceptually-based speeded decision tasks.

Thus, autonomic regulatory control seems to support the successful performance of specific types of tasks for certain age groups, but it is not presently clear just what factors contribute to these associations. Therefore, the goal of the present thesis is to explore the relationships between autonomic arousal, neural responsivity, and cognitive performance in the context of a task that requires ACC support. Participants were asked to complete a primary inhibitory control task that requires the maintenance of items in working memory

(Hester & Garavan, 2005). This particular task is suitable for exploring the degree to which autonomic regulation is associated with behavioural and/or electrocortical response because it requires effortful attention and inhibitory control, functions linked to the ACC. Furthermore, fMRI work with a similar version of this task has confirmed the presence of ACC activity during task performance (Hester et al., 2004). More specifically, it was of interest to determine whether trait-like resting measures of autonomic arousal could reliably predict behavioural performance and/or electrocortical responsivity on this ACC-based task. Documenting such associations would support a developing view that ACC function must be considered within a broader context that integrates both the cognitive and autonomic regulatory functions of this structure. Furthermore, it would provide a base from which to examine these relationships in older adults who, due to the normal aging process (Raz, 2000), may display more variable patterns of association between these physiological factors.

Attention and Memory

A hallmark of adaptive human behaviour is the ability to select relevant information from surrounding distracters in the pursuit of goal-directed objectives. This type of behaviour is reliant upon two basic cognitive processes, memory and attention (de Fockert, Rees, Frith, & Lavie, 2001; Gazzaley, Cooney, Rissman, & D'Esposito, 2005). To achieve basic behavioural goals, one must be able to maintain a representation of these goals in mind (Paxton, Barch, Racine, & Braver, 2008). This ability is sometimes referred to as working memory (WM) (Baddeley, 2001). The ability to focus attention also plays a key role in goal-directed behaviour as it allows an individual to sort through and focus on goal-relevant information in spite of a myriad of distracting influences (Kane, Bleckley, Conway, & Engle, 2001; Kane & Engle, 2003).

Although WM and attention each provide the support necessary for pursuing goal-directed objectives, it is important to recognize that these two cognitive processes are dependent on one another (de Fockert et al., 2001; Milham et al., 2001). For instance, research suggests that an increased WM load can lead to a decline in attentional control and an increase in the processing of distracters (de Fockert et al., 2001). This finding demonstrates that maintaining stimulus-priorities in WM is essential for effective control over attention (Awh, Vogel, & Oh, 2006). Likewise, attention itself may play a critical role in WM capacity by limiting the contents of WM to task-relevant information (e.g., Awh & Jonides, 1998). In fact, research shows that when attention is strained during a dual task paradigm, overall memory performance weakens (Dywan, Segalowitz, & Webster, 1998). Thus, WM and attention both play key roles in cognitive performance, but the effectiveness of each process is directly dependent upon the other process (Awh & Jonides, 1998).

Neural Structures Involved in Attention Allocation and Response Control

Research suggests that several structures within the brain including the anterior cingulate cortex (ACC), prefrontal cortex (PFC), parietal cortex, and basal ganglia form a network highly involved in attention (Milham et al., 2001). During attentionally demanding conditions, top-down control of this attentional network seems to be essential to complete task goals. For instance, neural activity associated with task-relevant information needs to be enhanced, whereas neural activity associated with task-irrelevant information needs to be suppressed (Bar, 2003; Bar et al., 2006). Research indicates that the dorsolateral prefrontal cortex (DLPFC) is the key structure called upon to implement top-down modulation of this structural network when attentional demands increase (MacDonald, Cohen, Stenger, & Carter, 2000). Thus, it appears that top-down modulation by the DLPFC is crucial for

maintaining attentional control and biasing the selection of task-relevant information within working memory. As a result, distraction and interference effects are reduced, and overall memory performance improves.

Evidence suggests that age-related changes in the DLPFC may be responsible for the aging brain's increased susceptibility to interference and distraction (Milham et al., 2001). For instance, studies indicate that the prefrontal cortices are the structures most vulnerable to the aging process (Raz & Rodrigue, 2006). In addition, fMRI research indicates decreased responsiveness of the DLPFC among older adults during conditions of increased attentional control (Milham et al., 2001). Such findings suggest that aging may compromise the ability of the DLPFC to implement effective attentional control when necessary.

Although the DLPFC plays a central role in the allocation of attentional resources, the ACC also plays a critical role as part of an attentional network (Ridderinkhof et al., 2004). The ACC has traditionally been viewed as a structure involved in emotion regulation (Mayberg, 1997; Mayberg et al., 1999); however, recent research has increasingly recognized the role of the ACC in cognition (Bush, Luu, & Posner, 2000; Carter et al., 1998; Posner & DiGirolamo, 1998). In particular, many researchers have noted the importance of the ACC in various aspects of cognitive control including attention allocation (Fallgatter et al., 2002), performance monitoring (Gehring & Knight, 2000; Magno, Foxe, Molholm, Robertson, & Garavan, 2006; Ridderinkhof et al., 2004; Ullsperger & von Cramon, 2004), and error-related processing (Garavan, Ross, Kaufman, & Stein, 2003; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Taylor, Stern, & Gehring, 2007). These terms have variously been used to describe the role of ACC in the allocation and control of attention and are often used interchangeably. As well, early studies tended to describe the role of the ACC as primarily

one involved in the monitoring of response conflict (Fallgatter et al., 2002). However, more recent work has failed to support the exclusivity of this response monitoring perspective (Burle, Roger, Allain, Vidal, & Hasbroucq, in press). For example, there is growing consensus that ACC activation is more associated with monitoring the need for control and acting like an alarm signal when increased levels of control are necessary (Bartholow et al., 2005; Burle et al., in press). Nonetheless, in the following literature review, the association between ACC and conflict monitoring will be retained because that is the terminology that defined the early work in this field.

Fallgatter and colleagues (2002) examined the role of the ACC in conflict monitoring and attention allocation using a Continuous Performance Task (CPT) that required the execution (Go) and inhibition (NoGo) of response tendencies. Because NoGo conditions require inhibition of prepotent response tendencies, they are considered more cognitively demanding than Go conditions, and therefore, are considered to be associated with increased response conflict and the control and allocation of attention. Fallgatter et al. used LORETA analysis, a method for localizing brain electrical sources (Pascual-Marqui et al., 1994), and confirmed the presence of increased ACC activity on NoGo compared to Go trials. Based on these data, Fallgatter and colleagues concluded that the ACC activity observed on NoGo trials reflected the increase in attention allocation that was necessary for successful performance on higher conflict NoGo trials.

In addition to its purported roles in conflict detection and attention allocation (Fallgatter et al., 2002), the ACC has also been discussed with respect to error-related processing (Garavan et al., 2003; Gehring et al., 1993; Taylor et al., 2007). The majority of this research comes from electrophysiological studies that have shown that there are specific

waveforms that occur when errors are made (Dehaene, Posner, & Tucker, 1994; Gehring et al., 1993). One of these waveforms, the error-related negativity (ERN), occurs approximately 50 – 100 ms after the execution of an erroneous response. Although there is continuing debate surrounding the exact functional significance of this ERP component (Gehring & Willoughby, 2004), it is generally agreed that the ERN reflects critical aspects of error detection and/or correction (Taylor et al., 2007). Moreover, several studies point to a generator in the ACC (Dehaene et al., 1994; Gehring et al., 1993). Thus, a great deal of evidence seems to indicate that the ACC plays a major role in the detection of errors and the monitoring of performance outcomes (Taylor et al., 2007). For this reason, descriptions of ACC function have been extended beyond simple error detection to a more general process known as performance monitoring (Gehring & Knight, 2000; Magno et al., 2006; Ridderinkhof et al., 2004; Ullsperger & von Cramon, 2004).

Performance monitoring refers to the ability to monitor and adjust behavioural performance according to changing environmental circumstances in the pursuit of task goals (Ridderinkhof et al., 2004). Studies with primates indicate that the ACC is sensitive to the expected delivery and unexpected omission of rewards, highlighting this structure's role in evaluating performance outcomes (Magno et al., 2006; Ullsperger & von Cramon, 2004). Kennerley and colleagues (2006) investigated the role of the ACC in reinforcement-guided decision making in a group of monkeys with lesions to the ACC. The ACC-lesioned monkeys, along with a control group, were trained to turn or press a joystick for a food reward. The target response had to be maintained for 25 consecutive trials in order to obtain food at the optimal rate, after which time the action-outcome contingency reversed. Kennerley and colleagues found that both groups were equally likely to switch to a new

response immediately following an error trial; however, unlike the control group, the ACC-lesioned group randomly switched response strategies in the middle of consecutively rewarded responses. Thus, whereas ACC-lesioned monkeys did not have trouble detecting or correcting errors, it seemed as though they were unable to maintain reward-guided behaviour over time. From their findings, Kennerley et al. concluded that the ACC goes beyond just detecting and correcting errors; it plays a critical role in the learning and maintenance of action values that can be used to guide future behavioural performance.

Whether discussed with respect to conflict monitoring, attention allocation, error detection, or performance monitoring, it is clear that the ACC is essential for adaptive cognitive control under attentionally demanding circumstances. A great deal of this cognitive control comes from the ACC itself, as well as its connections with other structures including the DLPFC, parietal cortex, and limbic structures (Bush et al., 2000). For example, some researchers suggest that although the ACC is responsible for detecting the need for increased cognitive control, this control is actually implemented through the DLPFC (Milham et al., 2001; Ridderinkhof et al., 2004). However, in addition to its connections with higher cortical regions, the ACC has important links with subcortical regions, including the hypothalamus, medulla, and pons (Devinsky, Morrell, & Vogt, 1995). It is through these brainstem links that associations with the heart are maintained (Porges et al., 1994). These diverse connections make the ACC a unique structure involved not only in attentional control, but also in autonomic activity (Critchley et al., 2003).

The Autonomic Nervous System and Modulation of Cardiovascular Arousal

The autonomic nervous system is comprised of two major divisions, the sympathetic and parasympathetic branches (Beauchaine, 2001). These two branches innervate most of the

body's major organs, and their actions on any given organ are often antagonistic. This antagonistic control allows for the dynamic regulation of major organ systems throughout the body (Bernston, Cacioppo, & Quigley, 1993). For instance, sympathetic innervation of the heart increases heart rate, whereas it is decreased through parasympathetic innervation. These influences are mediated through the tenth cranial nerve, i.e., the vagus (Beauchaine, 2001).

The vagus nerve originates in the medulla and projects, independently of the spinal cord, to numerous organs throughout the body, including the heart (Porges, 1995b). The vagus involves a bidirectional system containing both afferent and efferent fibers through which neural information flows. According to Porges and colleagues (1994), this bidirectional system permits dynamic, continuous communication between brain control centres and other key organs involved in homeostatic regulation.

Vagal afferent fibers are responsible for providing feedback to the brain through their connection from the heart to the nucleus tractus solaritus (Porges, 1995a). This continuous feedback allows the brain to effectively regulate cardiac activity. Vagal efferent fibres, on the other hand, originate in the brainstem and innervate the upper portion of the right atrium of the heart known as the sinoatrial (SA) node. The SA node is known as the pacemaker of the heart because it fires regularly and allows the heart to beat steadily. Electrical impulses from the SA node trigger a sequence of electrical events in the heart that cause muscle contractions which pump blood out of the heart for circulation throughout the body. Vagal efferent fibres are inhibitory in nature because they effectively decrease SA node firing, and consequently, slow heart rate.

Heart rate (HR) itself is the net outcome of competing sympathetic and parasympathetic influences on the heart (Porges et al., 1994). When external demands are

placed on the system, sympathetic innervation increases the force and frequency of heart beats and simultaneously decreases the variability between heart beats. Sympathetic innervation is countered by parasympathetic modulation of the heart through the vagus nerve, which effectively slows heart rate and increases interbeat variability. Thus, it is the dynamic interplay of sympathetic and parasympathetic inputs on the SA node that produces the complex variability that characterizes healthy heart rate (Thayer & Lane, 2000).

The variability in beat-to-beat intervals of the heart is referred to as heart rate variability (HRV). HRV represents parasympathetic modulation of the heart through the vagus nerve which is in turn modulated through the anterior cingulate cortex (ACC) (Critchley et al., 2003). One of the most common ways to assess HRV, or cardiac vagal tone, is by estimating respiratory sinus arrhythmia (RSA) (Porges et al., 1994). RSA refers to the rhythmic acceleration and deceleration of heart rate that accompanies normal respiration. During inspiration, respiratory mechanisms in the brainstem attenuate vagal efference to the heart and cause heart rate to increase. During expiration, vagal efference to the heart is restored and heart rate decreases. RSA can be assessed through the detection of individual heart beats (R-waves) and the measurement of intervals between successive heart beats (R-R intervals) from electrocardiogram (ECG) analysis (Porges, 1986).

Over the past forty years, cardiac vagal control, measured via RSA, has emerged as a useful psychophysiological marker of various aspects of behavioural and physiological functioning (Beauchaine, 2001; Thayer & Lane, 2000). Research indicates that higher RSA is associated with better cardiovascular health (Thayer & Lane, 2007; Thayer & Ruiz-Padial, 2006), effective emotion regulation (Thayer & Lane, 2000), and adaptive behavioural flexibility (Movius & Allen, 2005; Porges et al., 1994; Thayer et al., 1996; Thayer & Lane,

2000). Recent research has also increasingly focused upon the role of HRV in cognitive performance (Critchley et al., 2003; Matthews et al., 2004). Such research makes sense in light of the fact that the delicate balance between sympathetic and parasympathetic activity that characterizes healthy HR is regulated not only by brainstem structures, but also by higher cortical regions, including the ACC (Ter Horst & Postema, 1997).

Modulation of Cardiovascular Arousal and Cognition

In addition to its established role in cognitive control (Fallgatter et al., 2002; Gehring & Knight, 2000; Magno et al., 2006; Ridderinkhof et al., 2004; Ullsperger & von Cramon, 2004), recent research has increasingly noted the importance of the ACC in generating autonomic states of arousal that are adaptive for successful performance on cognitive tasks (Critchley et al., 2003; Matthews et al., 2004). Critchley and colleagues (2003) used fMRI and ECG to examine areas of activation associated with cardiac arousal during motor and cognitive tasks. They reported that increased activity in the dorsal ACC (dACC) was associated with increased sympathetic modulation of HR. Furthermore, they noted that patients with lesions to the dACC exhibited reduced cardiovascular (CV) responses to cognitive effort tasks. For instance, these patients failed to exhibit the increases in HR and systolic blood pressure that normally accompany effortful cognitive performance. From their findings, Critchley and colleagues concluded that the ACC, particularly the dACC, plays a key role in generating appropriate autonomic states of arousal that are necessary to meet current cognitive demands.

Matthews and colleagues (2004) also noted the importance of the ACC, particularly the ventral ACC (vACC), in modulating autonomic arousal. They recorded HR and brain activity (via fMRI) while participants completed a counting Stroop task that included

congruent and incongruent conditions. Results indicated increased activity in the dACC during incongruent conditions, supporting the role of this structure in cognitive control. Moreover, they noted that increased vACC activity correlated with high frequency HRV (considered an index of parasympathetic modulation) in both congruent and incongruent Stroop conditions. Matthews and colleagues concluded that ANS modulation by the ACC is closely tied with the cognitive processes that this structure supports. Such findings again highlight the importance of examining the role of CV arousal in cognitive performance, especially during tasks that tap executive control processes of the ACC.

Many researchers who have investigated the relationship between cognitive performance and HRV have used HRV as a dependent measure. For instance, Backs and Seljos (1994) noted that increased mental effort on a continuous memory task was associated with greater CV expenditure, such as increased respiration rate, among participants. In addition, Vincent and colleagues (1996) noted that faster stimulus presentation rate on a word recognition task was associated with increased CV arousal, including increased BP and HR. Although these studies reveal important information about the effect of cognitive effort on CV arousal, they only examine CV arousal from a reactive perspective. However, researchers have recently started to examine the relationship between cognitive performance and CV arousal from the reverse perspective, by utilizing CV variables, like HRV, as predictor variables, instead of dependent measures (Hansen et al., 2003).

Evidence indicates that higher HRV, as indexed by higher RSA, is associated with better performance on cognitive tasks. Some of the earliest research in this area came from work with infants. Richards (1985) noted that infants with higher RSA stared at novel stimuli for less time than infants with lower RSA. In addition, Linnemeyer and Porges (1986)

reported that infants with higher RSA looked at unfamiliar stimuli for longer periods and familiar stimuli for shorter periods, than did infants with lower RSA. Findings from these studies were interpreted as evidence for a link between higher RSA and better attentional capacity and processing speed. Later research with adults complemented these earlier findings of a link between higher RSA and enhanced cognitive performance. For instance, Hansen and colleagues (2003) examined the relationship between HRV and performance on various cognitive tasks in a group of sailors. Hansen et al. found that participants with higher baseline RSA made fewer errors and faster responses on executive function tasks compared to those with lower baseline RSA. These findings not only provide additional support for a link between CV arousal and cognitive performance, but they highlight the importance of this relationship for optimal performance on tasks that specifically tap executive functions.

Most of the research that has been focused on examining the role of CV arousal in cognitive performance has done so with infants and younger adults (Hansen et al., 2003; Richards, 1985). However, these relationships have often been overlooked in/among older adults despite clear evidence that warrants such research. For instance, age-related declines in medial regions of the PFC are well documented (Raz & Rodrigue, 2006). In addition, phasic vagal cardiac control, or RSA, is known to decline with age (Antelmi et al., 2004). Age-related declines in the mPFC and RSA may leave older adults vulnerable to cognitive control problems as their CV systems may be less able to modulate arousal appropriately to meet the behavioural demands of a complex, dynamic environment. In fact, recent research from our lab indicates that this is precisely the case.

Dywan, Mathewson, and Segalowitz (submitted) examined autonomic modulation of cardiac function and its relation to behavioural performance and electrophysiological

responses in a group of older and younger adults while they completed a source memory task. Results revealed a strong relationship between source memory performance and CV arousal for the older adults, such that those older adults with higher levels of baseline RSA tended to make fewer source memory errors than those with lower levels of baseline RSA. In fact, RSA did not relate to the ability to correctly identify study words or correctly reject foils. RSA only predicted the probability of making source memory errors, errors that required strategic control over response tendencies. Thus, for older adults, the attentional capacity of the ACC seemed to be more reliant upon the regulatory functions of the CV system for cognitive support and successful completion of this attention-based task.

In another study, Tays and colleagues (in press) had older and younger adults complete a letter Sternberg task, a working memory task with varying levels of interference imbedded. They investigated the N450, a phasic frontal negative waveform associated with response conflict and other conditions known to influence the magnitude of ACC activation (West, Bowry, & McConville, 2004). Tays et al. (in press) used the N450 to examine the electrocortical indices of the attentional control processes involved in this interference-related Sternberg task. Using Tays and colleagues' data, I examined the relationship between the graduated measures of attentional control and CV arousal. I was specifically interested in whether a relationship would emerge between RSA and the N450. I expected that as the level of interference increased, the relationship between RSA and the N450 would also increase. Such a relationship would reflect the fact that additional CV support was necessary to aid the ACC in combating increased interference in these conditions. Contrary to expectations, results indicated that there was no relationship between RSA and N450 amplitude across any of the interference conditions.

In a follow-up study, Tays and colleagues (unpublished data) further modified the Sternberg task. Instead of the letter stimuli, which by necessity come from a limited size set and, therefore, repeat even on trials where repetition is not part of the task structure, Tays et al. utilized words as stimuli. Because words obviously come from a less restricted set than letters, there was no unintended repetition. In this situation, we thought that individuals would be more likely to be aware and more certain of their erroneous responses. Under these conditions, it was expected that the N450 effects would be better defined, and these expectations were confirmed. However, despite the more distinct N450, results again confirmed that there was no relationship between RSA and the N450 in any of the interference conditions.

Thus, the interdependence of attentional control and cardiac function seems to be present only in the context of certain tasks and for certain age groups, and it is not clear just what factors account for the discrepancies in these results. One issue that may be important to consider is the level of difficulty that the task imposes on the participant. For instance, although there seems to be a relationship between higher RSA and better source memory performance for older adults, the same does not hold true for younger adults (Dywan et al., submitted). This may be because the source memory task does not challenge younger adults to the degree that it does older adults. In fact, Dywan et al. noted that a number of participants in the younger group did not generate enough source memory errors to allow for the calculation of stable ERNs, thus reducing the size of the group as well as the variance in error rate that would allow for correlations to occur. As well, in the modified Sternberg task described above, error rates were fairly low for both younger and older adults (Tays et al., in press). Thus, although this task was reliant on ACC function, as evidenced by clear dipole

localization in the Tays et al. (in press) study as well as in imaging studies (Jonides et al., 2000; Nelson, Reuter-Lorenz, Sylvester, Jonides, & Smith, 2003), the letter Sternberg does not appear to activate the ACC in ways that link with CV function.

In addition to task difficulty, another issue to consider is task type. For instance, although speeded perceptual decision tasks, like the Eriksen letter flanker task (Eriksen & Eriksen, 1974), involve attentional control and generate high error rates, this task does not seem to relate to CV arousal (Dywan et al., submitted). Because the information necessary for response decisions is available on screen, it does not engage executive control processes to the degree that other executive tasks do, and therefore, successful performance may not be as reliant on the modulation of arousal. On the other hand, phasic vagal cardiac control seems to be more essential for tasks that involve sustained attention (Middleton, Sharma, Agouzoul, Sahakian, & Robbins, 1999; Suess, Porges, & Plude, 1994) and working memory (Hansen et al., 2003).

In addition to difficulty level and task type, it might also be important to consider the degree to which the ACC is involved in task performance. Recent research suggests that there are particular types of tasks that are more likely to maximally engage the ACC. For instance, Kennerly and colleagues' (2006) work with ACC-lesioned monkeys on a reinforcement-guided decision making task (described earlier) highlighted the role of the ACC in tasks requiring the use of an action outcome history for the guidance of future action selections. Kennerly et al. reported that whereas control monkeys based their response decisions on a number of previous occurring trials, the ACC-lesioned monkeys only based their decisions on the immediately preceding trial. Such results demonstrate the centrality of

the ACC in tasks that require the use of an action-reinforcement history to guide the next action choice.

Thus, to examine potential relationships between autonomic function and cognitive performance, we would need a task that provides a sufficient challenge to both younger and older adults and that maximally engages the ACC. It would be important that error rates for both age groups be sufficiently high and show adequate within-group range to capture any relations with autonomic and electrocortical measures. It would also be advantageous for performance on each task trial to have implications for later trials through some sort of working memory component, because such conditions are known to influence the magnitude of ACC activation (Amiez et al., 2005). The task should also allow for performance at various levels of difficulty, so that if difficulty level is central to these effects, they can be tested for appropriately. In addition, this type of task would allow for the examination of relationships between RSA and behavioural and electrophysiological responses across various levels of task complexity.

One task that offers many of these qualities is Hester and Garavan's (2004) Working Memory Load Inhibitory Control (WMIC) task. The WMIC task (Hester & Garavan, 2005) is a continuous performance task that requires inhibition of prepotent responses, or the inhibitory control of response tendencies. In Hester and Garavan's original design, participants were shown a string of 1, 3, or 5 uppercase letters for 6 seconds, followed by an 8 second rehearsal period. A series of 60 decision trials followed, each lasting 1500 ms. Each trial consisted of a single letter presented on a computer screen for 1000 ms followed by a blank screen for 500 ms. Participants were instructed to respond as quickly as possible and decide whether or not the letter was part of the memory string. The participant was trained to

press a button for trials featuring letters that *were not* part of the memory set (Go trials) and to withhold responding to trials featuring letters that *were* part of the memory set (NoGo trials). Approximately 91% of the trials consisted of Go trials, while the remaining 9% consisted of NoGo trials. The uneven distribution was expected to create a prepotent response tendency for Go trials, and as a result, participants would be accustomed to pressing the button for each trial and would need to suppress a strong prepotent response tendency in order to successfully withhold on NoGo trials.

The WMIC task (Hester & Garavan, 2005) is ideal for the present study for a number of reasons. For instance, unlike other tasks, such as the Stroop and Sternberg (previously described), which emphasize the selective attention element of behavioural inhibition, the WMIC task places greater emphasis directly on the suppression of prepotent motor responses, a function tied to the ACC (Falkenstein, Hoormann, & Hohnsbein, 1999). The WMIC task is also advantageous for the present study because it uses various levels of WM to constrain attentional capacity. This graduated difficulty will allow us to examine the effect of WM load on attentional control and response inhibition in more detail, as it includes 3 levels of task complexity. Finally, successful inhibitory control performance requires that individuals keep the WM letters in mind throughout an entire run, so every trial serves as a kind of rehearsal for ongoing success in this task. Such properties make the WMIC task a unique paradigm for tapping into cognitive control processes of the ACC and examining whether a relationship does indeed exist between cognitive performance and CV arousal.

While the WMIC task offers many attractive properties for our current interests, Hester and Garavan (2004) originally designed this task for work with fMRI. Because we were interested in examining RSA as it relates not only to behavioural performance, but also

to electrophysiological responses, some of the task parameters had to be modified in order to translate the WMIC task into an effective ERP paradigm. For instance, Hester and Garavan's original design included decision trials that were 1500 ms in length, a fairly long time by ERP standards. Event-related fMRI requires that different trials be spaced far enough apart so that accurate blood-oxygenation level-dependent (BOLD) hemodynamic responses can be observed. Consequently, stimulus presentation rates in fMRI studies cannot be as fast they are in ERP paradigms.

We decided to speed up the WMIC task for our ERP study. We assumed that by increasing the speed of stimulus presentation, it would become increasingly difficult for participants to inhibit the prepotent Go response on salient, but inappropriate NoGo trials. These expectations were confirmed when we piloted participants using this faster presentation rate and found that they did indeed commit more commission errors on NoGo trials. Increasing stimulus presentation rate also allowed us to include more overall Go and NoGo decision trials. Because one of our goals was to examine ERP components on both correct and incorrect NoGo trials, including more NoGo trials across conditions increased the number of opportunities available for participants to generate correct and incorrect NoGo responses. This in turn increased the number of trials available for forming stable ERP averages.

In addition to speeding up stimulus presentation rate, we also decided to present strings with 2, 4, and 6 letters instead of 1, 3, and 5 letters. We assumed that by increasing each memory load by one letter, we would enhance the overall challenge of the task within a reasonable range. Pilot work again confirmed that longer letter strings led to more errors of

commission on NoGo trials; however, participants still maintained above-chance accuracy rates, indicative of deliberate, not random, response decisions.

Another adjustment made to the WMIC task was the addition of "catch" NoGo trials. Originally, we designed the task so that NoGo stimuli were always followed by Go stimuli. However, after piloting participants, a few reported that the organization of Go and NoGo stimuli throughout runs was too predictable. For instance, after a few runs participants realized that, regardless of whether or not they correctly withheld responding to a NoGo trial, they could automatically hit on the next trial because NoGo stimuli were always followed by Go stimuli. In order to increase task difficulty and keep participants engaged throughout entire runs, we added "catch" NoGo trials. These were NoGo trials that immediately followed a regular NoGo stimulus. At some point during every run, a regular NoGo stimulus was followed immediately by another NoGo stimulus, and then at another point, by 2 additional NoGo stimuli. So, in addition to the regular Go and NoGo trials, each run contained 3 additional "catch" NoGo stimuli to decrease the predictability of the task.

One final adjustment was made to the WMIC task. In Hester and Garavan's original design, presentation of a letter string and the 8 second rehearsal period were immediately followed by 60 decision trials. At the end of the decision trials, participants were instructed to type in the letters from the memory set. Hester and Garavan claimed that if participants typed in the letter string correctly at the end of the run, then NoGo errors could not be attributed to trouble remembering items, but only to problems inhibiting prepotent response tendencies. Because our main focus was this inhibitory control function, we followed these steps but also included an additional step at the beginning of each new run. After letter string presentation and the 8 second rehearsal, participants were required to type in the correct letter

string before the run even began. If the participant typed the letter string correctly, the run started. If he or she did not type the letter string in correctly, the letters re-appeared, followed by another 8 second rehearsal, and again, the participant was instructed to type in the letter string. This step repeated until the correct letters were entered. By including this step, we ensured that participants really did learn the letter string, so that when they started the run, they were sure which letters were targets for the current trial. This, of course, would be even more relevant when using this task with older adults.

The Electrophysiological Approach to ACC Function

By making the adjustments listed above, we converted the WMIC task into an ERP paradigm. With the high spatial resolution of fMRI, Hester and Garavan (2004) observed inhibition and memory-related activity in the DLPFC and the ACC while participants completed the WMIC task. Albeit with lower spatial resolution, ERP technology allows for the examination of cognitive processes unfolding at a much higher temporal resolution. For instance, ERP research demonstrates that two distinct waveforms, the N2 and P3, occur when a prepotent response is successfully withheld (Bekker, Kenemans, & Verbaten, 2004). This is extremely important because these two components are thought to index distinct aspects of inhibitory control processing (Bokura, Yamaguchi, & Kobayashi, 2001). Whereas fMRI may highlight activity involved in the general process of inhibition, because of its lower temporal resolution, it is impossible to determine the extent to which this inhibition-related activity reflects different stages of inhibitory control processing (Jonkman, Sniedt, & Kemner, 2007). With the enhanced temporal resolution of ERPs, we are able to examine neural activities associated with distinct aspects of inhibition and error-related cognitive processes.

We examined four ERP components associated with attention allocation and error processing. One ERP component of interest in the present study is the NoGo N2. The N2, considered an index of cognitive control, is a negative component that occurs over fronto-central scalp areas approximately 200 to 350 ms after stimulus-onset (Kok, 1986; Tekok-Kilic, Shucard, & Shucard, 2001). Source analyses of this component are fairly consistent in indicating ACC involvement (Bekker et al., 2004), along with possible involvement of the orbitofrontal cortex (OFC) . The N2 is an important component for the present study as it is typically elicited in the context of Go/NoGo tasks (e.g., Bekker et al., 2004; Bokura et al., 2001; Johnstone et al., 2007). Many who have examined the N2 in this context have suggested that the N2 is an index of neural inhibition (Bokura et al., 2001; Falkenstein et al., 1999; Fallgatter & Strik, 1999). For instance, although this negativity follows both Go and NoGo stimuli, the N2 is significantly larger following NoGo trials on which the response has been correctly withheld, presumably because these less frequent trials require increased response inhibition to override the prepotent Go response tendencies. Other researchers (Bekker et al., 2004; Donkers & Van Boxtel, 2004) claim that the N2 is not an index of inhibition, but rather, a reflection of response conflict. Much of this research comes from studies that have localized the N2 to the ACC, a region highly implicated in response conflict monitoring. More recently, it has been suggested that the N2 may represent both neural processes: inhibition and response conflict (Falkenstein, 2006). Falkenstein suggests that perhaps the NoGo N2 that occurs at the level of the ACC represents conflict detection, whereas the NoGo N2 that is localized to the OFC represents an actual inhibitory process.

Another component of interest for the present study is the NoGo P3, a positive component that follows the NoGo N2 and peaks between 300 and 600 ms after stimulus

onset (Kopp, Mattler, Goertz, & Rist, 1996). This P3 is maximal at centroparietal sites following Go targets and frontocentral sites following NoGo trials (Fallgatter & Strik, 1999). Like the NoGo N2, the functional significance of the NoGo P3 is still widely debated. Some suggest the P3 represents the evaluation of inhibitory performance (Dimoska, Johnstone, Barry, & Clarke, 2003), and others claim that the P3 indexes the process of inhibition itself (Bekker, Kenemans, & Verbaten, 2005; Kok, Ramautar, De Ruitera, Band, & Ridderinkhof, 2004). In fact, recent work by Smith and colleagues (2007) suggests that it is the later occurring P3 that actually represents the inhibitory and/or response conflict processes that have traditionally been considered indices of the N2. Thus, although the exact functional significance of the N2 and P3 remain uncertain, these components are clearly present in higher level tasks that require the suppression or inhibition of prepotent response tendencies, and are therefore important components to examine during the WMIC task.

The stimulus-locked N2 and P3 will allow us to examine electrocortical activity related to response control; however, we are also interested in activity related to error processing. Therefore, in addition to the NoGo N2 and P3, we will examine two error-related response-locked components; the ERN and Pe. The ERN occurs approximately 50 to 100 ms after the execution of an error, followed by the Pe around 200 or 300 ms after the erroneous response (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990; Gehring et al., 1993). Although the functional significance of the ERN and Pe is still the subject of much debate (Taylor et al., 2007; van Boxtel, van der Molen, & Jennings, 2005), recent studies have begun to differentiate these two components more clearly.

Originally, the ERN was thought to occur only following error responses, hence its name, the *error-related negativity* (Falkenstein et al., 1990; Gehring et al., 1993). However,

further research revealed that the ERN can occur on both correct and error trials. When this negativity occurs on correct trials it is referred to as a *correct-related negativity*, or CRN. Although a negativity follows both correct and error responses, this negativity is usually much larger when an error has occurred (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). This finding led many investigators (Falkenstein et al., 2000; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000) to extend the role of the ERN beyond simple error detection to include a more general response evaluation process. They argue that during cognitive decision tasks, an individual must compare the appropriate response (that *should have been* executed) with his or her actual response (that *was* executed). This comparison can occur on both correct and incorrect trials, thus producing at least a modest ERN in both cases. They argue, further, that the larger ERN occurring on error trials likely reflects the comparison process along with an additional error detection signal.

The Pe differs from the ERN in that it occurs later and only on error trials. This has led some (Falkenstein et al., 2000) to suggest that the Pe reflects more evaluative aspects of error processing, whereas others (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005) consider it to be a standard P3-like component that is sensitive to the commission of errors. Some even consider the P3 to be an index of error awareness or recognition (O'Connell et al., 2007). For example, O'Connell and colleagues examined the ERN and Pe in an error awareness Go/NoGo Stroop task. Participants were required to press a button for congruent word-color stimuli (Go trials) and to withhold hitting (NoGo) for incongruent word-color stimuli or for stimuli that had repeated. If participants committed an error on a NoGo trial, they were instructed to press an "awareness" button on the following trial, in lieu of the normal response. This allowed the researchers to examine the relationship between the ERN/Pe

components and error awareness. O'Connell et al. found that, whereas there was no difference in ERN amplitude between "aware" and "unaware" NoGo errors, the Pe was significantly larger on "aware" NoGo error trials compared to correct Go and "unaware" NoGo error trials. Thus, the Pe may very well represent error awareness, which makes this an extremely important ERP component to consider when assessing cognitive performance.

Thus, it is clear that the N2/P3 and the ERN/Pe reflect important aspects of inhibitory control and error-related processing, respectively. Furthermore, many studies have acknowledged source generators for these components in or around the ACC (Bekker, Kenemans, Koeksma, Talsma, & Verbaten, 2005; Bokura et al., 2001; Falkenstein, 2006; Gajewski, Stoerig, & Flakenstein, 2008). For this reason, we plan to examine inhibition-related components (N2/P3) and error-related (ERN/Pe) components while participants complete the WMIC task. Although fMRI research has demonstrated inhibition-related ACC activity in the context of the WMIC task (Hester et al., 2004), with the high temporal resolution of ERPs we are able to investigate distinct levels of inhibitory control involved in the WMIC task via the N2 and P3 components. We are also able to examine separate aspects of error-related activity via the ERN and Pe components. Finally, we are able to investigate how the strength of these electrocortical responses change with increasing WM load demands.

The Present Study

The present study has been designed to investigate autonomic modulation of cardiac function and its relation to behavioural and electrophysiological responses in younger adults while they engage in a continuous performance task that requires inhibitory control. Participants completed a modified version of the Working Memory Load Inhibitory Control

(WMIC) task, as described above (Hester & Garavan, 2005). Hester, Murphy, and Garavan (2004) used fMRI to examine performance on the WMIC task. The researchers noted that, although increasing WM demands made inhibition more difficult, individuals who were able to maintain successful inhibitory control produced increased activation in both the DLPFC and the ACC. In fact, Hester et al. suggested that as the number of items that required a "withhold" response increased across levels, it seemed that more top-down control was required to inhibit responding in the presence of the stronger, prepotent response.

Because the ability to withhold a prepotent response appears to be somewhat reliant upon modulatory control of arousal through the parasympathetic nervous system (Porges, 1995a), it is reasonable to assume that as WM demands increase in the WMIC task and inhibition becomes more difficult, the ability to modulate CV arousal should become more relevant to task performance. The DLPFC and ACC may become more engaged when WM loads increase, and this may produce levels of autonomic arousal more optimal for completion of this cognitively demanding task. We expect that the control of CV activity, as indexed by RSA, will relate to the ability to withhold a prepotent response. We also expect that RSA will relate to the amplitude of various scalp-recorded indices of cognitive control. These would include the stimulus-locked N2 (Roberts, Rau, Lutzenberger, Birbaumer, 1994) and NoGo P3 (Picton, 1992) associated with inhibitory control and the response-locked ERN and Pe components associated with error making (Holroyd & Coles, 2002).

Specifically, it is hypothesized that a relationship between RSA and behavioural and electrophysiological indices of inhibitory control will be increasingly associated across the three levels of the WMIC task (Hester et al., 2004), with the strongest relationships being

found in the most demanding task condition. Behaviourally, it is expected that those individuals with higher RSA will make fewer commission errors.

Method

Participants

Participants were recruited from Brock University. Results from two individuals were excluded from analyses due to technical difficulties, leaving a sample of 21 younger adults (5 male, 16 female) between the ages of 18 and 27 ($M = 21$, $SD = 2.2$). All participants had normal or corrected vision, were fluent English speakers, and 19 of the 21 individuals were right-handed. Exclusionary criteria for the present study included neurological or psychiatric conditions, as well as medications expected to affect neural function. Students either received research participation credit or a small honorarium for their time. The study received clearance from Brock University's Research Ethics Board, and all participants gave informed consent.

Experimental Design

Participants completed a modified version of a computer-generated task designed by Hester and Garavan (2004) known as the Working Memory Load Inhibitory Control (WMIC) task. As the name suggests, the WMIC is a primary inhibitory control task with a working memory load embedded. The task included 18 runs in total. At the start of each run, participants were shown a memory string of 2, 4, or 6 uppercase letters for 6 seconds. Memory set letter presentation was followed by a 6-second blank screen that constituted a rehearsal period. After this rehearsal period, a window appeared that instructed participants to type in the memory string letters. No time limit was imposed for this recall period, and participants had to enter the correct letter string before the decision trials began. If the

participant did not enter the memory string correctly the first time, the computer alerted the participant and presented the letters again for 6 seconds, followed by another 6-second rehearsal period. This phase repeated until the participant entered the correct letters. This step was taken to ensure that participants knew which letters were members of the memory string, since the decision trials that followed relied directly upon this information.

Once the participant typed in the correct memory string, a series of 101 decision trials ensued. Each decision trial consisted of a single uppercase letter which was presented for 700 ms followed by a blank screen for 600 ms. Participants were instructed to indicate, as quickly and accurately as possible, whether the letter was part of the memory string or not. Participants were trained to press the space bar of a keyboard whenever the letter on the screen did not match one of the memory string letters (Go trials), and to withhold responding to trials featuring letters that were part of the memory string (NoGo trials). Figure 1 displays a visual illustration of the task.

Insert Figure 1 about here

The response trials included 76 Go (75%), 22 NoGo trials (22%), and 3 "catch" NoGo trials (3%). Previous work suggests that setting the probability in favour of Go stimuli primes prepotent response execution, which in turn, makes it increasingly difficult to withhold on salient, yet inappropriate NoGo trials (Bokura et al., 2001). When the 101 decision trials concluded, an identical window from the start of the run appeared, and again, the participant was instructed to type in the memory string. The only difference was that this time the participant was given only one opportunity to type in the letter string. Again, there

was no time limit imposed for this recall period, and once the participant typed in the memory string, the run was completed.

Participants completed 18 runs in total, 6 of each memory load size (2, 4, or 6 letters). Each run consisted of a new memory string of letters, followed by 101 decision trials which included 76 Go, 22 NoGo, and 3 "catch" NoGo trials. Therefore, in total, the present task included 456 Go, 132 NoGo, and 54 "catch" NoGo trials for each memory load size (2, 4, and 6). Because the response-locked ERP indices of having made an error (ERN and Pe) must be averaged across at least 7 trials to be considered stable, we included a large number of Go and NoGo trials to ensure that participants generated a sufficient number of errors on each level of the task (low, medium, high). We assumed that by including 132 NoGo trials for each level, even if participants only made a few errors on a run, they would make at least 7 erroneous responses across a particular load that could be averaged together to produce a reliable ERN and Pe.

Experimental runs were presented to participants in an identical sequence, with the order of presentation counterbalanced for memory load requirements (2, 4, 6, 4, 6, 2, 6, 4, 2, 4, 6, 2, 2, 6, 4, 6, 4, 2). A 30 second break was provided between each run, with a longer 10 minute break at the half-way point (between runs 9 and 10). The task, including practice and breaks, took approximately 1 hour to complete.

Procedure

Upon arrival to the lab, participants were given a detailed description of all testing procedures and were shown the EEG and ECG equipment. The researcher then went over the consent form and answered any remaining questions the participant had. Once consent was obtained, participants filled out two questionnaires. The first, a health history questionnaire,

required that they answer questions about their general physical and mental health, their use of stimulants and suppressants, as well as diet and exercise patterns. They also completed the Spot-the-Word Vocabulary Test, a measure of basic verbal capacity (Baddeley, Emslie, & Nimmo-Smith, 1992).

Once all questionnaires were completed, electrophysiological equipment was prepared and affixed. EEG was collected using a 128 channel Active Two BioSemi system (BioSemi, Amsterdam). A soft mesh cap with 128 electrode holders was placed over the participant's head and secured. A small amount of electrode gel was then applied to each holder, and 128 wired electrodes were plugged into each of the cap sites. Exogenous electrodes with gel were then affixed to various areas of the skin using 2-sided adhesive disks. Electrodes were placed beside each eye and below the right eye to monitor eye blink activity during the recording. Two other electrodes were then attached to the chest for cardiac recording, one 2 inches below the right clavicle and the other between the 2 bottom ribs on the participant's left-hand side. Finally, to monitor breathing activity, a respiration belt was wrapped comfortably around the participant's upper chest and secured.

Once all electrophysiological equipment was in place, participants sat quietly for 5 minutes and stared at a moving star-pattern screensaver. During this time, baseline electrophysiological activity and cardiac and respiratory rates were recorded. Upon completion of this resting period, each participant's blood pressure and heart rate were taken twice using an Omron IntelliSense Automatic Blood Pressure Monitor. HR and BP were recorded so that pre-test Rate Pressure Product (RPP), a measure of myocardial oxygen consumption, or CV workload, could be computed (RPP = HR x Systolic BP).

Upon completion of the resting period and blood pressure measurements, participants were prepared for the WMIC task. The researcher explained the task instructions and answered any questions the participants had. Individuals then completed a practice session to familiarize themselves with the task demands and speed, and also to give them experience with responding. Participants practiced until it was clear that they were comfortable with the task. At that point, the researcher left the testing room to begin the EEG recording, and the participant started the WMIC task.

Upon completion of the WMIC task, participants completed 2 shorter tasks. The first task was a Sternberg-like word recognition task, and the second was an incidental learning task that involved identifying words from the previous task. Together, both tasks took approximately 20 minutes to complete. However, these tasks were part of another study and will not be discussed further here.

After completion of the 3 cognitive tasks, participants again sat quietly and relaxed for 5 minutes while staring at a moving star-pattern screensaver. Post-task electrophysiological activity and cardiac and respiratory rates were recorded, and another pair of blood pressure and heart rate measures were taken. This final resting period and blood pressure reading constituted the completion of the testing session. At that time, all electrophysiological equipment was removed and individuals were given the opportunity to wash and dry their hair. Participants were then debriefed and awarded research participation credit or a $30.00 honorarium for their time.

Electroencephalography (EEG)

EEG was collected using a 128-channel Active Two Biosemi system (BioSemi, Amsterdam). Signals were sampled at the rate of 512 points per second and digitized with a

24 bit ADC. Because the BioSemi system does A-D conversion at the electrode site, the amplifier gain was 1. A bandpass filter from 1 Hz (time constant 0.1592 s) to 30 Hz was used. All electrodes were referenced offline to averaged mastoids for analysis. Task stimuli were created using E-Prime software (Psychological Software Tools, Inc., 2004) and were presented on a computer screen located 60 cm from the participant.

Due to the nature of the WMIC task, special procedures had to be taken to determine which correct Go trials and NoGo error trials could be included for stimulus-locked (N2, P3) and response-locked (ERN, Pe) ERP analyses. As long as a participant typed in the correct letter string at the end of a run, all correct Go trials and NoGo errors that were made during that run were automatically included for later artifact detection and ERP analyses. However, if a participant typed in one or more letters incorrectly at the end of a run, special steps had to be taken to remove these trials before the data were entered into the artifact detection program. For instance, if a given run's memory string was MXRD, but a participant typed in MXR*C* at the end of the run, all responses made to the letters D and C had to be removed prior to processing the data. This was because somewhere in the middle of the run, the participant may have begun responding to the letter D as if it were a Go trial and the letter C as if it were a NoGo trial. Consequently, if the participant made an inappropriate hit response to the letter D, this trial(s) could not be included as a reliable NoGo error because it would be unclear whether the error was due to a problem inhibiting the prepotent response or a problem remembering the proper letter string. Thus, every participant's responses were entered into an excel spreadsheet that flagged these problem trials. These trials were then manually removed from the participant's data set, and a special filter list was created which included all of the reliable Go and NoGo responses that could be included for that individual

during subsequent artifact detection and correction procedures. By following these steps, we occasionally lost NoGo trials, but we also ensured that responses included for later ERP analysis were made up of reliable Go correct and NoGo error trials.

Each participant's filtered list was entered into MatLab studio (MathWorks, 2004) and reliably remembered Go and NoGo segments were extracted for artifact detection and rejection. Trials that included moderate eye movements or blinks were corrected using a regression procedure developed by Gratton and Coles (1983). For correct Go trials, all epochs with midline amplitudes exceeding 50μV were automatically rejected. Because there were fewer NoGo error trials, each epoch was individually inspected and artifact rejection was performed manually.

First, stimulus-locked, artifact-free epochs associated with successful Go and NoGo trials were averaged relative to a 200 ms pre-stimulus-baseline. N2 amplitude was defined as the most negative peak at fronto-central midline sites between 150 – 350 ms following stimulus onset and P3 amplitude was defined as the most positive peak at fronto-central sites in a 300 – 600 ms window. Response-locked waveforms associated with correct responses to Go trials and commission errors on NoGo trials were formed using artifact-free error epochs averaged relative to a -600 to -400 ms pre-stimulus baseline. This earlier baseline was used to avoid potential contamination of the post-response averages by including the stimulus-related P300 that typically occurs at about the same time as the response. ERN amplitude was defined as the most negative value at fronto-central midline sites in the 100 ms following the erroneous response. The Pe was defined as the most positive peak at centro-parietal sites in a 150 – 400 ms post-response window.

Electrocardiogram (ECG)

ECG activity was monitored throughout the entire WMIC task duration and during 5-minute pre-test and post-test resting periods. Individual RSA values were calculated offline using a moving polynomial algorithm (Porges, 1986), which filters the cardiac signal and statistically removes sources of HRV that are not based on RSA. Two RSA values for each participant were computed; one from the 5 minute baseline HR recording (pre-test RSA) and another from the 5 minute post-test resting period (post-task RSA). On-task ECG activity was also recorded but had not been processed in time for these analyses.

Supplementary Autonomic Variables

RPP was calculated (HR x systolic BP, divided by 100) and used as an approximation of myocardial oxygen demand (Fredericks, Choi, Hart, Butt, & Mital, 2005) and hence an indicator of sympathetic activity. Heart rate (HR), interbeat interval (IBI) and measures of respiration were also calculated and entered into our database.

Results

Behavioural Performance

Proportions of omission and commission errors for the three levels of task difficulty are presented in Table 1. Accuracy rates for all three WM loads were entered into a repeated measures ANOVA. As expected, there was a main effect for load, $F(2,40) = 55.35, p < .001$, $\eta^2 = .74$, and pairwise comparisons showed a linear increase such that higher load increased error rates for NoGo trials across all three levels of the WMIC task (all $ps < .01$). Omission rates for Go trials were analyzed in the same manner and produced the same linear effect, $F(2,40) = 23.62, p < .001$, $\eta^2 = .54$, such that increased WM load led to a greater number of

omitted responses on Go trials with each level being different from the one before (all *p*s < .01).

Insert Table 1 about here

Response time (RT) data for correct Go and error NoGo trials for all three WM loads are presented in Table 2. RTs, like accuracy, also suffered as WM loads increased across conditions. A repeated measures ANOVA indicated a main effect for load on Go RTs, $F(2,40) = 200.70$, $p < .001$, $\eta^2 = .91$. Post hoc analysis again indicated a linear effect such that higher WM loads were associated with slower RTs (all *p*s < .01). Similar analysis indicated that RTs on NoGo error trials increased in the same manner, $F(2,40) = 114.65$, $p < .001$, $\eta^2 = .85$, and follow-up analysis again confirmed a linear increase in RTs (all *p*s < .01).

Insert Table 2 about here

The increase in error rates and RTs associated with increases in WM load indicates that the task manipulation worked as intended. Increasing WM load led to declines in the ability to withhold the prepotent Go response to salient, yet inappropriate NoGo stimuli. Omission errors on Go trials also increased as attentional resources were increasingly engaged. In addition, RTs increased markedly for NoGo error responses as WM load increased, but also for correct Go trials, highlighting the sensitivity of RTs to the task manipulation.

Electrophysiological Response

Stimulus-locked Responses to Correct Decisions.

Figures 2 to 4 represent the stimulus-locked waveforms associated with the accurate withholding of a response to NoGo trials (when inhibitory control is assumed to have occurred) relative to the accurate commission of a response on Go trials (when a prepotent response would be appropriate). The overlayed waveforms are displayed across the 3 WM loads. The ERPs to NoGo responses seem to differ from those to Go responses in the way one would expect in a Go/NoGo paradigm. When a response has been correctly withheld on a NoGo trial, the N2 appears more negative and the NoGo P3 appears more positive. What we see, however, is that the amplitude of the differential NoGo response is diminished relative to the Go response as WM load increases.

Insert Figures 2 to 4 about here

The N2 component. The N2, thought to index neural inhibition or response conflict (Bekker et al., 2004; Bokura et al., 2001; Fallgatter & Strik, 1999) was the first ERP component examined. Peak negativities associated with correct Go trials and correctly inhibited NoGo trials were submitted to a repeated measures ANOVA and analyzed as a function of condition, which represented the three levels of WM load (low vs. medium vs. high), trial-type (Go vs. NoGo), and electrode site (Fz' vs. FCz' vs. Cz' – these sites represent approximate scalp locations on the standard 10-20 system). Although there were main effects for condition, trial-type, and site, they were superseded by interactions between condition and trial-type, $F(2, 40) = 3.33$, $p < .05$, $\eta^2 = .14$, condition and site, $F(4, 80) = 9.00$, $p < .001$, $\eta^2 = .31$, and trial-type by site, $F(2, 40) = 6.65$, $p < .01$, $\eta^2 = .25$.

Simple effects analyses of trial-type by site for each condition were performed to further investigate these interactions. A main effect of trial-type was evident at the low WM load, $F(1, 20) = 6.14, p < .02, \eta^2 = .24$, such that the N2 was generally larger on NoGo relative to Go trials, irrespective of site. At the medium WM load there were no main effects for trial-type or site (all *p*s > .09); however, there was a robust type by site interaction, $F(2, 40) = 9.36, p < .002, \eta^2 = .32$. Follow-up comparisons indicated that the difference in N2 amplitude between Go and NoGo trials was most reliable at sites FCz' and Cz'. Results were similar at the highest WM load condition, but the difference in N2 amplitude between Go and NoGo trials failed to reach significance at the two posterior sites. Because site FCz' showed the most consistent distinction between Go and NoGo trials across all three WM load conditions, this site is considered most representative of the inhibitory N2 effect and will therefore be used in later correlational analyses.

The above analyses were performed using midline scalp sites because ERP studies have reported N2 source models near the frontal midline (Bekker, Kenemans, & Verbaten, 2005). However, further work with topographical maps from Brain Electrical Source Analysis (BESA) indicated a more lateralized N2 effect for the present task (see Figure 5). In future research with the NoGo N2 component we will, therefore, include these left parietal sites where the N2 effect appears to be most robust.

Insert Figure 5 about here

The P3. The frontal, NoGo P3, also thought to be associated with response inhibition (Bekker et al., 2004), was examined next. Peak positivities were submitted to a 3 (condition) by 2 (trial-type) by 3 (site) ANOVA. Although there were main effects for condition, type,

and site, these effects were qualified by interactions between condition and type, $F(2, 40) = 24.10, p < .001, \eta^2 = .55$, condition and site, $F(4, 80) = 6.96, p < .001, \eta^2 = .26$, and type by site, $F(2, 40) = 4.21, p < .03, \eta^2 = .17$.

Simple effects analyses of type by site were conducted for each level of WM load to investigate the interactions more closely. The low WM load condition showed main effects for type and site; however, these effects were qualified by an interaction between type and site, $F(2, 40) = 4.83, p < .01, \eta^2 = .20$. Although the difference in P3 amplitude between Go and NoGo trials was reliable for all 3 sites, the P3 effect was most robust at site FCz'. At the medium WM load, there were no main effects for type or site (all *p*s > .09), but there was a site by type interaction, $F(2, 40) = 9.36, p < .01, \eta^2 = .32$. Again, follow-up comparisons indicated P3 differences at all 3 sites, but FCz' showed the strongest effect. Again, at the highest WM load condition, there were no main effects for type or site, but a site by type interaction, $F(2, 40) = 9.36, p < .01, \eta^2 = .32$. Follow-up comparisons indicated that the difference in P3 amplitude between correct Go and NoGo trials occurred at all sites except Cz', the most posterior site. The scalp topography associated with the NoGo P3 is presented in Figure 6. Analyses indicated that the NoGo P3 effect was most robust at site Fz'.

Insert Figure 6 about here

ERP Responses to NoGo Errors and Correct Go Decisions

Figures 7 to 9 represent the response-locked waveforms associated with the incorrect commission of a response on a NoGo trial (when inhibitory control failed) relative to the accurate commission of a response on a Go trial (when a prepotent response was appropriate). The overlayed waveforms are displayed across all 3 WM loads. The ERPs to

incorrect NoGo responses seem to differ from correct Go responses in the way one would expect in a Go/NoGo paradigm. When an error of commission has occurred on a NoGo trial, the ERN appears more negative and the Pe appears more positive. However, as was the case with the stimulus-locked N2/P3, the amplitude of the differential NoGo response is diminished relative to the Go response as WM loads increase.

Insert Figures 7 to 9 about here

The ERN component. Peak negativities associated with the ERN were examined on the basis of WM load (low vs. medium vs. high), trial-type (correct vs. error), and site (Fz' vs. FCz' vs. Cz' vs. Pz') ANOVA. There was a main effect of condition, $F(2, 38) = 12.36$, $p < .001$, $\eta^2 = .39$, such that the highest WM load condition resulted in the smallest ERNs. Follow-up Bonferroni comparisons indicated that, whereas there was no difference in ERN amplitude between the low ($M = -2.44$, $\pm$ 0.38 μV) and the medium WM load conditions ($M = -1.95$, $\pm$ 0.24 μV), both displayed significantly greater ERN amplitudes when compared with the high memory load condition ($M = -0.95$, $\pm$ 0.30 μV). As expected, there was also a main effect of accuracy, $F(1, 19) = 86.64$, $p < .001$, $\eta^2 = .82$, such that the ERN amplitude was larger for NoGo error trials ($M = -3.66$, $\pm$ 0.42 μV) compared to Go corrects ($M = 0.10$, $\pm$ 0.20 μV). There was also a condition by accuracy interaction, $F(2, 38) = 4.41$, $p < .02$, $\eta^2 = .19$, indicating that, although the differences in ERN amplitude between correct Go and NoGo error trials was reliable at each level of task difficulty, the difference was largest at the low WM load (-4.39 μV) relative to the medium load (-3.90 μV), and relative to the high load (-1.90 μV). And finally, there was an accuracy by site interaction, $F(3, 57) = 11.88$, $p < .001$, $\eta^2 = .39$. Follow-up comparisons indicated that differences occurred at all sites with the

greatest difference in ERN amplitude between correct Go and NoGo error trials occurring at site FCz' (see also the topographical map of the ERN response, Figure 10). On the basis of this finding, we used ERNs from site FCz' in later correlational analyses, as this site best represents the effect in question.

Insert Figure 10 about here

The Pe component. The Pe, another response-locked component, thought to index error awareness or subjective error appraisal (O'Connell et al., 2007; Taylor et al., 2007), was examined in the same way as for the ERN. There was a main effect of condition, $F(2, 38) = 14.45, p < .001, \eta^2 = .43$, with pairwise comparisons indicating a linear decrease in Pe amplitude across all three WM loads ($2 > 4; 2 > 6; ps < .01$) but the difference between medium and high loads did not reach statistical significance ($p = .19$). As expected there was also a main effect of accuracy, $F(1, 19) = 68.37, p < .001, \eta^2 = .78$, such that Pe amplitude was larger for NoGo error trials ($M = 4.54, \pm 0.36$ μV) compared to Go corrects ($M = 1.23, \pm 0.18$ μV). A final main effect occurred for site, $F(3, 57) = 10.08, p < .001, \eta^2 = .35$, with follow-up comparisons indicating that differences occurred at all sites. There was also a condition by accuracy interaction, $F(2, 38) = 19.18, p < .001, \eta^2 = .50$. Although the differences in Pe amplitude between correct Go and NoGo error trials was reliable at each level of task difficulty, it followed the same pattern as the ERN, with the largest difference occurring at the low WM load (5.36 μv), relative to the medium load (3.06 μV), and relative to the high load (2.22 μV). Finally, as with most of the previously examined components, the difference in Pe amplitude between correct Go and NoGo error trials was largest at site

FCz' (for topographical map see Figure 11). Thus, FCz' will serve as the site to be used for all ERP components for subsequent correlational analyses.

Insert Figure 11 about here

Relationship between ERPs and Behavioural Performance

We have confirmed that ERPs elicited by the WMIC are sensitive to the commission of errors and that this sensitivity changes as a function of memory load. The next question to be addressed is whether individual differences in these ACC-generated ERPs would be at all predictive of performance on this newly modified performance monitoring task. We would assume that a more vigorous electrocortical response would be associated with the speed or accuracy of decisions. In each case, ERP components were selected from sites that best represented the effect of interest. As well, because the effect of interest in each case involved the degree to which an error response deviated from the response on correct trials, we formed residual scores by regressing the ERP associated with the correct trial from the ERP associated with the error. These residuals were then entered into a correlational analysis to test their association with the errors and response times across the 3 levels of task difficulty.

Stimulus-locked ERPs (NoGo N2/P3). Correlations between the residualized stimulus-locked ERPs were not generally related to error rates (*ps* > .09). There were also no relations between the stimulus-locked ERPs and the speed with which decisions were made. The only exception was that higher amplitude of the NoGo P3 was associated with faster response times. This occurred whether the NoGo P3 was elicited on low or medium WM load trials and whether the response was to a correct trial or an error (all *ps* < .02). See Figure 12 for a representative scatterplot of these relations. This specific association between the

NoGo P3 and response speed has not been reported before in the context of a Go/NoGo task. Thus, it would seem that a larger NoGo P3 might suggest greater reactivity that might lead to a generally faster response style, or conversely, that those who are very slow produced a weak P3 response.

Insert Figure 12 about here

Response-locked ERPs (ERN/Pe). There were, however, more associations between the response-locked ERP components and behavioural response. First, the residualized ERN amplitude was generally associated with Go omission rates. More specifically, smaller ERNs were associated with an increased likelihood of omitting responses on Go trials. This relationship was most evident for ERNs associated with errors on medium load trials where smaller ERNs were associated with increased omission rates for Go trials during the medium ($r = .530$, $p = .016$) WM load condition. This relation has not been reported before, but could suggest that the smaller ERNs involved a more cautious approach to the task, whereby participants held back making a response whenever there was any uncertainty about the match between the cue and the letters currently held in their WM load, rather than taking a chance of making an error. There is previous evidence that smaller ERNs are associated with uncertainty (Falkenstein et al., 2000; Gehring et al., 1993).

When it came to the Pe, results indicated that higher amplitudes were associated with faster response times to correct and error responses across all working memory load conditions with the exception of error trials on the low load. Relations between Pe amplitude and RTs were all fairly substantial (all *p*s < .01). See Figure 13 for a representative scatterplot of these relations.

Insert Figure 13 about here

What is of interest here is that the pattern of relations associated with the NoGo P3 and the Pe were similar. In both cases, the increased amplitude of these components was associated with faster responses, but not necessarily more accurate ones. It is as though they reflect a tendency toward impulsivity. This is in contrast to the reduced ERN that seemed to relate to caution, i.e., with respect to the tendency of those with smaller ERNs being more cautious in their response tendencies.

Cardiovascular Measures

Means and standard deviations for pre and post-test RSA, RPP, IBI, HR, and systolic BP (SBP) are presented in Table 3. The only difference that occurred between pre and post-test measures was for SBP which was higher for post relative to pre-test phases of the experiment, $t(21) = -5.533, p < .001$.

Insert Table 3 about here

Correlations between CV measures are presented in Table 4. Analyses indicated that indices of CV function correlated in the expected manner.

Insert Table 4 about here

Relationships between Psychophysiological Measures and Behavioural Performance

RSA

It was originally hypothesized that RSA would relate to better response control performance in a graduated way across the three memory loads of the WMIC task, such that

the association would be greatest at the highest WM load condition. To test this hypothesis, pre-test RSA was entered into a correlational analysis that included the probability of making a NoGo error or omitting a Go response at each level of WM load. Results indicated that RSA did not relate to the probability of either making an error of commission on NoGo trials or an error of omission on Go trials, irrespective of difficulty level (all *ps* > .43). As seen in previous analyses, because RTs were also sensitive to task difficulty, we examined correlations between pre-test RSA and RTs on correct Go and NoGo error trials. Again, RSA did not relate to the time participants took to execute correct Go or incorrect NoGo responses, irrespective of difficulty level (all *ps* > .25). These relations did not improve when we partialed out respiratory rate from RSA.

Other Psychophysiological Measures

Although RSA did not relate to inhibitory control performance, exploratory analyses were undertaken to determine whether other physiological variables of interest (Bekker et al., 2004) were related to the behavioural performance measures. Another physiological measure used in the present study was Rate Pressure Product (RPP). Whereas RSA reflects parasympathetic nervous system activity, RPP is a measure of myocardial oxygen consumption and thus, CV workload or stress (Fredericks et al., 2005). Correlational analyses were performed to determine whether pre-test RPP related to behavioural performance on the WMIC task. However, as was the case with pre-test RSA, pre-test RPP did not relate to the probability of making a NoGo error or Go omission (all *ps* > .13), and it also did not relate to the time it took participants to execute correct Go or incorrect NoGo responses (all *ps* > .24). However, one interesting relationship did emerge between RPP and "catch" NoGo error accuracy. Catch NoGo trials were those NoGo trials that followed immediately after a

preceding NoGo trial. Results indicated that higher pre-test RPP was associated with increased error rates for "catch" NoGo trials $r(21) = .445, p = .043$. Thus, individuals with higher pre-test CV stress were less able to inhibit prepotent responses on challenging "catch" NoGo trials that occurred one after the other (see Figure 14).

Insert Figure 14 about here

Whereas neither pre-test RSA nor pre-test RPP related to behavioural performance on the WMIC task, exploratory analysis with pre-test IBI revealed some associations with behavioural response (see Table 5). Although longer IBIs (slower HR) were marginally associated with faster correct Go responses for the low WM load condition ($p = .07$), these relations were even stronger for correct trials at the medium and high loads (all $ps < .05$). Longer IBIs were also related to fast response errors on NoGo trials, but this relationship only occurred at the lowest WM load condition ($p = .045$). These findings suggest that those individuals with less pre-test sympathetic arousal were at an advantage in terms of performance speed on more challenging Go trials.

Insert Table 5 about here

Relations between Physiological Variables and Performance Monitoring ERPs

The final question to be addressed was whether there were relations between electrocortical and psychophysiological measures, both of which are considered to be associated with aspects of ACC function.

Stimulus-locked ERPs (NoGo N2/P3). First examined were relations between indices of autonomic function and the stimulus-locked ERP responses (the NoGo N2 and P3). These

ERP components were found, as expected, to be larger on those trials that resulted in the successful withholding of a prepotent response. Pre-test RSA was examined first, but there were no relationships found with either the N2 or the P3 across any of the WM load levels. Next examined was whether pre-test RPP, our estimate of sympathetic influence, related to either of these response-locked components. Although there was no relationship between pre-test RPP and N2 amplitude (all *p*s > .16), there was evidence of a relationship with P3 amplitude. Results indicated that higher pre-test RPP, i.e., more sympathetic arousal, was associated with reduced NoGo P3 amplitudes at both low, $r(21) = -.494, p = .023$, and medium load conditions, $r(21) = -.526, p = .014$, but not the high load ($p > .36$). One could interpret this as evidence for a relation between the level of stress an individual brings to the situation affecting the amplitude of the NoGo P3. It is also of interest that NoGo P3s, usually associated with attention allocation (Kok, 2001), were related to faster response times across all conditions. Perhaps higher levels of stress reduced attentional allocation and indirectly, response times. In fact, this ability to adjust response times may have resulted in the lack of relations between the psychophysiological measures and error rates per se.

We also examined the relations between IBI and the stimulus-locked ERPs. Whereas there were no relations between pre-test IBI and N2 amplitude (all *p*s > .22), there was a relationship between pre-test IBI and P3 amplitude across two of the three WM load levels. Results indicated that longer pre-test IBI was associated with greater amplitude P3 at the low $r(21) = .473, p = .030$, and medium loads $r(21) = .476, p = .029$, but not the high load ($p > .19$). These relations actually supported the previous interpretation that initial levels of sympathetic arousal, or the inverse, longer IBIs (slower HR), did affect the amplitude of the

P3 response on correct trials and the amplitude of that response was then associated with faster RTs, i.e. perhaps more efficient levels of attentional processing.

To determine whether or not pre-test RPP and IBI were uniquely associated with the NoGo P3, both were entered into a regression. Although both significantly related to P3 amplitude, when examined in the same model, results showed that neither RPP nor IBI uniquely predicted P3 amplitude (*ps* > .25), indicating shared variance between these two physiological variables.

Response-locked ERPs (ERN/Pe). We next examined whether RSA, RPP, or IBI were related to response-locked electrocortical responses on NoGo error trials, i.e., responses associated with a failed attempt to inhibit a prepotent response tendency. The first physiological component examined was RSA, and only one relationship emerged for one condition. Analyses indicated that pre-test RSA correlated with ERN amplitude on the highest WM load condition, such that higher RSA was associated with a more negative ERN amplitude, $r(20) = -.453$, $p = .045$. This is the first evidence of support for our initial hypotheses. If one assumes that both a robust ERN and high levels of RSA are signs of a well functioning ACC, then it would not be surprising that these would relate when task demands are greatest (see Figure 15). Although the significance of this relationship is dependent on the individual with the highest RSA and largest ERN, there was nothing in the records of this individual to suggest that they should be dropped from analyses.

Insert Figure 15 about here

Next, pre-test RPP was entered into a correlational analysis with the ERN and the Pe. Although there was no relationship between pre-test RPP and Pe amplitude across any of the

WM load conditions (all *ps* > .83), there was a relationship between pre-test RPP and ERN amplitude. Results indicated that higher pre-test RPP was associated with shallower ERN amplitudes at the low, $r(20) = .460, p = .041$, medium, $r(20) = .470, p = .036$, and high load, $r(21) = .551, p = .012$. This is basically the inverse of the RSA relations with the ERN and supports our model, i.e., that the ERN, an ACC-based index of performance monitoring (Taylor et al., 2007) is associated with other indices of ACC function in a reliable manner. Higher RSA, indicating higher levels of parasympathetic modulation, is associated with larger ERNs in response to error especially under high load situations, whereas higher RPP, indicating less parasympathetic modulation, is associated with reduced ERNs irrespective of load. Thus, although there is considerable interest in which task parameters affect the size of the ERN (Hajcak, McDonald, & Simons, 2003a, 2003b), these results indicate that the size of the ERN is also influenced by the balance achieved between the two branches of the autonomic nervous system.

Finally, the relations between IBI and ERNs confirmed the above interpretation in that longer pre-test IBI did not relate to Pe amplitude across any of the WM load conditions (all *ps* > .89) but was consistently associated with the size of the ERN at all loads: low $r(20) = -.439, p = .053$, medium $r(20) = -.452, p = .045$, and high WM loads $r(20) = -.490, p = .028$. Thus, again, a slower HR, which is indicative of greater parasympathetic influence, was associated with a more robust error signal from the ACC. Because pre-test RPP and IBI related to ERN amplitude across all three WM load levels, it was of interest to determine whether these variables accounted for unique variance in the ERN. When entered into the same model, neither RPP nor IBI added uniquely to the prediction of the ERN (all *ps* > .31), indicating shared variance between these two physiological variables.

Discussion

The primary issue addressed in the present study was whether autonomic modulation of cardiac function relates to behavioural and electrophysiological responses of younger adults while they complete a continuing performance task that requires the execution and inhibition of prepotent motor response tendencies. Exploring how the modulation of cardiac function affects cognition is a relatively new area of research and results of recent studies have not been consistent. RSA, an index of phasic vagal cardiac control, has been linked to attentional capacity in children (Suess et al., 1994) and found to predict highly specific aspects of neurocognitive function specifically in older adults (Dywan et al., submitted) and maze learning performance in younger adults (Mathewson, Snyder, Tays, & Segalowitz, in preparation). However, RSA appeared to be unrelated to behavioural or electrophysiological function in either older or younger adults in a Sternberg attentional task (Capuana et al., unpublished report). Thus, it is not clear, just what sort of task might best elicit measurable behavioural, neural, and cardiac responses to allow us to examine the relations among them in a reliable fashion and hopefully then extend this research to an examination of age-related change.

The task used in the present study was modeled after a paradigm used by Hester and Garavan (2004) to investigate the role of WM load on attentional control. In Hester and Garavan's original design, high performers, i.e., those with greater inhibitory success, displayed greater activity in the dACC and DLPFC when WM loads increased. Because inhibitory control in the WMIC task is dependent upon the active maintenance of items in WM, Hester and Garavan explained that as the number of items requiring a NoGo response increased, so too did the need for effective top-down control over attention. Hester and

Garavan concluded that high performers seemed to be able to exert more effective top-down attentional control when task demands increased, as evidenced by the increased DLPFC and ACC activation observed in this group.

Research suggests that the ACC is not only involved in cognitive control, but also autonomic activity (Devinsky et al., 1995). Through its connections with the vagus nerve to the SA node of the heart, the ACC exerts modulatory control over CV arousal (Porges, 1995b). Increasingly, research is showing that autonomic states of arousal play an important role in cognitive performance, especially when that performance requires effortful attention (Critchley et al., 2003; Matthews et al., 2004). Thus, the ACC is not only responsible for implementing effective cognitive control, but also for generating autonomic states of arousal that are adaptive for achieving task goals. For this reason, we were interested in exploring whether behavioural and/or electrocortical responses would relate to the regulation of autonomic arousal in a task that required ACC support. Moreover, we were interested in determining whether trait-like resting measures of autonomic control would reliably predict behavioural performance and/or electrocortical responsivity. The WMIC task was chosen for the present study because it requires effortful attention and inhibitory control, both of which are linked to ACC function. Furthermore, Hester and Garavan's (2004) work with fMRI has confirmed the presence of ACC activity on the WMIC task.

We had expected that the ability to modulate cardiovascular activity, as indexed by RSA, would relate to behavioural performance on the WMIC task (i.e., error rates and responses times associated with variance in inhibitory control) as well as electrocortical measures thought to reflect response control. ERP components included the stimulus-locked NoGo N2/P3, which typically increase in size when a prepotent response error has been

successfully avoided (Eimer, 1993; Falkenstein et al., 1999). The other components of interest were the ERN and Pe, response-locked components that emerge in the context of having made an error (Gehring et al., 1993), in this case, an error that reflected the inability to refrain from making a prepotent response. Whereas many researchers have examined these components in a variety of tasks, it is relatively rare that they are elicited within the same task so that their association with autonomic response tendencies could be simultaneously assessed. We expected associations between autonomic modulation and the behavioural and electrocortical measures to strengthen as WM load increased, such that the strongest relations would occur in the most demanding WM load condition when the need for effortful attention and response control would be greatest. Documenting such associations would add to the growing consensus that ACC function must be considered within a broader context that integrates both the cognitive and autonomic regulatory functions of this structure. It would also provide information that would guide our subsequent exploration of the relation between ACC and neuro/cardiac responses in older adults where we would expect to see greater variance due to the vulnerability of these systems to the normal aging process (Raz & Rodrigue, 2006).

Electrocortical Responsivity and Psychophysiological Measures

Of particular interest for the present study was whether a relationship would emerge between pre-test RSA and any of the ERP components of interest. Results indicated that higher pre-test RSA was associated with greater ERN amplitude on the high WM load condition. Thus, those individuals with increased phasic vagal cardiac control (i.e., higher RSA) also displayed greater amplitude ERNs to errors they committed on the most

challenging WM load condition. What this relationship means depends upon what we understand the ERN to be and what variation in the amplitude of the ERN represents.

Traditionally, the ERN has been associated with response monitoring and error-detection processes (Taylor et al., 2007). For instance, many consider the ERN a manifestation of a response evaluation process whereby ongoing behaviour is compared against a mental representation of the appropriate, goal-directed response (Coles, Scheffers, & Holroyd, 2001; Falkenstein et al., 2000). When there is a discrepancy in this comparison process, an enhanced negativity, or ERN, occurs. Holroyd and Coles (2002) have linked the occurrence of the ERN to activity in the mesencephalic dopamine system. For instance, research demonstrates that fluctuations in dopaminergic activity accompany the unexpected occurrence of rewards and punishments. According to Holroyd and Coles, the commission of an error leads to a drop in dopamine that acts as a negative reinforcement signal to the frontal cortex. This negative reinforcement signal disinhibits pyramidal neurons of the ACC, which in turn, generate the ERN response. Thus, the ERN may act as an alarm that sounds when outcomes are worse than expected, and this alarm may, in turn, signal the need for increased cognitive control from the PFC.

In our data, individuals with smaller ERNs tended to commit more errors of omission on Go trials, especially when WM demands were higher. This finding is interesting in light of evidence linking motivational factors with ERN amplitude (Falkenstein et al., 2000). For instance, the magnitude of the ERN response is larger when participants are more certain that they have committed an error (Gehring et al., 1993). Thus, it could be that participants with smaller ERNs in the present task were less certain when they did indeed commit an erroneous response. This uncertainty, in turn, may have led to a more conservative response

strategy for Go trials when WM demands increased and the task became more challenging. Other researchers have shown that larger ERNs occur when participants are more concerned about their performance on a given task (Gehring, Himle, & Nisenson, 2000; Hajcak et al., 2003a). Tops and colleagues (2006) not only observed larger ERNs in individuals high in behavioural shame, but also in individuals high in agreeableness, a personality trait that is related to higher positive affectivity. Although behavioural shame proneness and agreeableness are both at opposite ends of the affectivity spectrum, they both share a common concern over social evaluation. From their findings, Tops et al. concluded that greater ERN responses may not reflect negative affectivity, per se, but rather, a concern over social evaluation that leads to increased task engagement in certain individuals. Thus, it may be that participants with smaller ERNs were less concerned about their performance in general. This lack of concern may have led to lapses in attention, and consequently, more omitted responses on the higher, more challenging WM load conditions.

With respect to the relationship between ERN and RSA, in the present task, those individuals with higher phasic vagal cardiac control may have been more engaged on the most challenging condition of the task, as indexed by larger ERN amplitudes, compared to individuals who exhibited lower pre-test RSA and smaller ERNs. Moreover, because phasic vagal cardiac control and the ERN response are both tied to the ACC (Critchley et al., 2003; Dehaene et al., 1994), the relationship between higher RSA and larger ERN amplitude in the present task may indicate enhanced ACC function for some individuals on the most challenging task condition. As task performance of younger adults does not always seem to be reliant upon autonomic regulation (Dywan et al., submitted), it may be that, if such a relationship were to occur, it would surface in the most cognitively demanding condition.

This was, of course, demonstrated in the relationship we found between RSA and the ERN in the highest WM load condition. However, given that the relationship seemed overly dependant on an individual who had the highest level of RSA and the deepest ERN, it will be prudent to replicate this effect with a larger sample and a fuller range on these variables. I hope to do this by examining these variables in an older population.

Although less central to the main hypotheses, results also indicated that both RPP and IBI were associated with the magnitude of the P3 response. Specifically, we found that higher pre-test RPP and shorter IBI (i.e., faster HR) were both associated with smaller P3 amplitude for correct NoGo trials on the low and medium WM loads. Higher pre-test RPP and shorter IBI were also associated with smaller ERNs at all three WM loads. These results suggest that individuals who experienced more cardiac workload or stress (i.e., higher RPP) and those who showed a faster, less relaxed HR (i.e., shorter IBI) at the start of the task displayed less robust electrophysiological responses to correct (P3) and error responses (ERN) during NoGo trials. It would appear then, that the ability to modulate arousal through the cardiovascular system has implications for the size of electrocortical indices of response.

General Behavioural Outcomes. Behavioural results on the revised version of the WMIC task were well in line with Hester and Garavan's (2004) original findings as well as other studies that have used similar paradigms (Engle, Conway, Tuholski, & Shisler, 1995; Hester & Garavan, 2005; Kane & Engle, 2003). Inhibitory control performance decreased as the number of items in WM increased, such that errors of commission and omission increased systematically from the low to the medium to the high WM load conditions. Response times (RTs) also followed the expected pattern, with RTs for correct hits and errors increasing steadily as WM requirements increased across conditions.

Our results complement other studies that have demonstrated the negative effect of increased WM load on attention allocation (Engle et al., 1995; Hester & Garavan, 2005; Hester et al., 2004). Attentional control purportedly requires both the enhanced processing of task-relevant information and the inhibition or suppression of task-irrelevant information (Gazzaley et al., 2005). In addition, effective attentional control seems to be reliant upon the active maintenance of stimulus priorities in WM (Kane et al., 2001; Kane & Engle, 2003). So, as WM demands increased across conditions in the present study, attentional control began to break down, resulting in less successful inhibitory control over response tendencies.

General Electrophysiological Responses. A major goal for the present study was to translate the WMIC task into an effective ERP paradigm, and this was met with considerable success. This was evident in the robust presence of N2 and P3 responses on correct NoGo withhold trials, as well as the ERN and Pe elicited by the commission of NoGo errors. As described earlier, there is evidence that a fronto-central negativity occurs around 200 – 400 ms of correct NoGo trials, when a prepotent response is correctly withheld (Kok, 1986; Tekok-Kilic et al., 2001). The N2, considered an index of neural inhibition or response conflict (Bekker et al., 2004; Falkenstein, 2006), reliably occurred in the present task on correct NoGo trials, when participants correctly withheld executing the prepotent Go response. Visual inspection of the N2 waveform for the three conditions suggested a decrease in the N2 effect as WM demands increased, although statistical analyses indicated that the N2 effect was most reliably diminished in the highest WM load condition.

Another component of interest in Go/NoGo tasks is the P3, a positive waveform that follows the NoGo N2 and peaks between 300 and 600 ms of stimulus onset (Kopp et al., 1996). As was the case with the N2, the P3 component reliably occurred on correctly-

withheld NoGo trials for all three conditions of the WMIC task. Visual inspection of the P3 component suggested a robust positivity differential between NoGo and Go trials for the low WM load condition, with a gradual flattening of the component as WM demands increased to medium and high loads. Statistical analyses indicated that the strongest P3 effects occurred for the low WM load condition, followed by both the medium and high load conditions, which did not statistically differ from one another in terms of the NoGo P3 effect.

Whereas the N2 and P3 reliably occurred on correct NoGo trials, we were also interested in whether those components sensitive to the commission of errors would occur when participants failed to withhold the prepotent Go response. One of these is the ERN, a negative waveform that peaks between 50 and 100 ms of an erroneous response (Falkenstein et al., 1990; Gehring et al., 1993). Our results confirmed the presence of an ERN whenever participants failed to withhold the prepotent Go response. It appeared that a reliable ERN occurred for all three WM load conditions and further analyses indicated that, although there was no difference in ERN amplitude between low and medium WM loads, both elicited greater amplitude ERNs when compared with the high WM load condition.

Our results also confirmed the presence of an error-related Pe, another response-locked component that follows the ERN on error trials (Falkenstein et al., 1990). Visual inspection of the waveforms seemed to indicate that the Pe was largest for the low WM load condition, followed by the medium, and finally, the high load. Analyses confirmed that the Pe effect was strongest for the low WM load condition, followed by both the medium and high WM load conditions, which did not differ from one another.

Thus, the WMIC task was successfully translated into an ERP paradigm as evidenced by the presence of the N2/P3 and ERN/Pe components on correct NoGo and error NoGo

trials, respectively. Furthermore, these electrocortical responses occurred for all three WM load conditions, with the general pattern indicating diminished amplitude with increased WM load demands. One could, of course, expect that if the size of these components reflects the attentional demands of the task, they should increase with load. There are, however, some possible explanations for the pattern that emerged in the present study.

Smaller ERN amplitudes have been thought to reflect reduced certainty in responses (Gehring et al., 1993). Under our high WM load demands, participants may have became less certain of their responses in general, correct or incorrect resulting in the smaller waveforms. It has also been suggested that the ERN reflects a negative evaluation process, whereby it signals that an expected reward has not been obtained or that an outcome is "worse than expected" (Holroyd & Coles, 2002). The strength of the ERN signal may, therefore, reflect the affective experience of error-making (Hajcak, McDonald, & Simons, 2004). In the present task, the low WM load condition was fairly simple; therefore, an erroneous response may have elicited a strong affective reaction, so producing an enhanced ERN signal in this condition. However, because error rates increased substantially during the more challenging WM loads, erroneous responses may have become less salient to the individual, and therefore, resulted in a less robust electrocortical response in the medium and especially high WM load conditions.

One final explanation for the observed decline in ERP amplitudes across WM load conditions involves a phenomenon known as latency jitter. A grand average ERP is created by averaging together several scalp signals from different trials. One of the assumptions of this averaging process is that the component has a latency, or time to peak, that is consistent across trials. However, as a task becomes more difficult, the time it takes to process a given

stimulus may increase, and as a result, the time it takes for a given component to peak may increase as well. Along with increases in latency, come increases in the standard deviation of this latency. Thus, this introduces variability into ERP latencies, also known as, *latency jitter*. When these variable ERP components are averaged together across trials, the overall amplitude of the component may be reduced in size. Thus, as WM demands increased in the present task, ERP latencies may have become more variable, and as a result, the averaging process may have produced an ERP component reduced in its overall amplitude. Further analyses of these data using a Woody Filter (Woody, 1967) could address this issue by aligning the trials based on the signals themselves, rather than the onset of eliciting stimuli. This way, components of interest are averaged right on top of one another, and not across jitter.

Electrocortical Responsivity and Behavioural Performance. Because evidence indicates that the ERP components in question are generated by or around the ACC (Bekker, Kenemans, Koeksma et al., 2005; Bokura et al., 2001; Falkenstein, 2006; Gajewski et al., 2008), and because the WMIC task is reliant upon ACC function, as evidenced by imaging studies (Hester et al., 2004), it is reasonable to assume that the strength of electrocortical responsivity might relate to the speed or accuracy of response decisions in the present task. Many consider the ability to execute response decisions quickly and accurately indicative of enhanced cognitive processing (Segalowitz, Dywan, & Unsal, 1997).

Both the stimulus-locked P3 and error-related Pe were associated with RTs for correct Go and error NoGo trials. Specifically, those who made faster responses, irrespective of accuracy or WM load tended to produce more positive response-locked P3s, reaching significance for low and medium load conditions, and more positive response-locked Pe's,

reaching significance on the medium and high WM load conditions. Thus, although the Pe and P3 relationships with RTs were not exactly identical across WM load conditions, in general, individuals with greater P3 and Pe responsivity executed correct Go and incorrect NoGo responses more quickly.

There are precedents for these relations. For instance, Segalowitz and colleagues (1997) found that higher P3 amplitude was associated with faster RTs in an auditory oddball task, which is not unexpected as P3 amplitude is considered an index of attention allocation. However, in our data, this increased attentional allocation, although associated with speed, was not necessarily associated with increased accuracy in that individuals who exhibited this increased attention seemed to also execute all responses, correct or incorrect, more quickly.

The Pe followed a similar but not identical pattern with the P3 in terms of its relations with RTs. These findings are interesting in light of recent work linking reward seeking and punishment avoidance with error-related ERPs (Boksem, Tops, Wester, Meijman, & Lorist, 2006). Boksem and colleagues found that individuals high in reward-seeking behaviours exhibited larger Pe responses to errors, whereas those high on punishment avoidance exhibited greater ERNs. The authors noted that individuals sensitive to punishment may be more prone to use a reactive form of control that is a more passive in nature and is invoked only after an important event has occurred and needs to be acted upon. Conversely, individuals sensitive to rewards may invoke a more proactive form of control, whereby they actively bias the processing of information in accordance with task goals. This type of proactive control can lead to more efficient cognitive performance. In fact, Boksem et al. found that individuals higher in reward sensitivity not only displayed greater Pe amplitudes, but also responded more quickly to task stimuli.

The fact that both the P3 and Pe related to RTs in a similar fashion is interesting given the recent suggestion that these two components reflect similar neural and functional processes (Overbeek et al., 2005). For instance, studies indicate that the P3 is evoked by stimuli with some sort of motivational significance, including highly novel, unexpected, or task-relevant stimuli (Nieuwenhuis, Aston-Jones, & Cohen, 2005). Due to their similar timing and midline scalp distribution, Overbeek, and colleagues (2005) have suggested that the Pe may constitute a P3-like component sensitive to the motivational significance of an error (Davies, Segalowitz, Dywan, & Pailing, 2001). Evidence in support of this view comes from studies which have demonstrated that increased Pe amplitude is associated with error salience and recognition (Falkenstein et al., 2000; O'Connell et al., 2007). In addition, just as the P3 has been suggested to reflect the update or revision of a mental model (Donchin & Coles, 1988), Overbeek and colleagues (2005) have suggested that the Pe may reflect the update of a mental model that specifically occurs following an error response.

Despite the similarities between both the P3 and Pe in terms of their relationships with correct and error RTs, it is important to recognize that, as described above, these relationships were not completely identical. Boksem and colleagues (2006) reported similar patterns between both the P3 and Pe components and behavioural measures on an Erikson Flanker Task (Eriksen & Eriksen, 1974). Although Boksem et al. (2006) found a strong relationship between the P3 and Pe, the components did not follow the same pattern in terms of their specific relationships with other performance measures such as post-error slowing. Thus, although the P3 and Pe may be related, the fact they share similar, but not identical patterns of association with other performance measures may indicate that the components themselves index similar, but not identical cognitive processes.

Behavioral Performance and Psychophysiological Measures. We expected that higher baseline RSA would relate to better inhibitory control performance across all three WM load conditions. Furthermore, we expected that the strongest RSA-behaviour relationship would occur in the most cognitively demanding circumstance, the high WM load condition. There has been some evidence of such relationships in the general literature (Hansen et al., 2003) and from previous studies in our own lab (Dywan et al., submitted; Mathewson et al., in preparation). However, contrary to expectations, correlational analyses indicated no relation between baseline RSA and level of inhibitory control across any of the WM load conditions.

Understanding the context in which these relations occur, and in which they do not, is central to understanding the meaning of these autonomic indices of ACC function, and as a consequence, the nature of ACC function itself. Although, as mentioned, some researchers have demonstrated a relationship between higher resting RSA and better executive control performance (Hansen et al., 2003), this relationship has not always consistently occurred (Dywan et al., submitted). For instance, whereas Dywan and colleagues observed a relationship between higher baseline RSA and better source memory performance among older adults, the same relationship did not hold for younger adults. The authors reasoned that because source memory tasks are demonstrably more difficult for older adults (Dywan, Segalowitz, & Arsenault, 2002), they may require increased CV support for successful performance. We thought that a similar relationship might emerge for younger adults at the highest level of the WMIC, i.e., when even young participants were faced with an executive control task that challenged them to a sufficient degree. However, even when faced with the more cognitively demanding WMIC task, younger adults' performance remained

independent of their baseline RSA. It may be that the highly functional ACC of younger adults provides sufficient support for cognitive demands, such that phasic vagal cardiac control contributes little to cognitive performance outcomes. Perhaps as an individual ages and the functional integrity of the ACC declines, it is then that the requirement for phasic vagal cardiac control becomes more essential for successful executive control and cognitive performance. In the future, I will be able to test this hypothesis by examining the performance of older relative to younger adults on this paradigm.

There were, however, some relations between behavioural performance and other psychophysiological measures. For instance, higher baseline RPP, an index of sympathetic dominance (Fredericks et al., 2005), was associated with increased errors on "catch" NoGo trials, or those NoGo trials that occurred consecutively, one after the other. These trials were originally implemented to increase task difficulty and keep participants actively involved throughout an entire run. One or even two additional "catch" NoGo trials could appear directly after the standard NoGo trial. These rare "catch" NoGo trials presumably presented an additional challenge to response control. In this context, the fact that higher RPP was associated with increased errors of commission on "catch" NoGo trials would suggest that individuals with greater pre-test cardiac workload or stress were less able to inhibit the prepotent Go response on the more challenging "catch" trials.

A relationship also emerged between pre-test IBI and response times (RTs) on correct Go trials. Larger pre-test IBI (i.e., slower HR) was associated with faster correct RTs for Go trials at all three WM load levels. Thus, those individuals with a slower, more relaxed HR at the start of the task executed correct Go responses more quickly than those individuals who started the task with a faster HR. Perhaps those individuals with higher pre-test HR (i.e.,

shorter IBIs) were more anxious about the task itself or their performance and, as a result, employed a more cautious response style. Importantly, those individuals with a slower HR responded more quickly to correct trials *only*, and they did not differ from the high HR group in terms of accuracy. Therefore, those individuals with a slower, more relaxed pre-test HR performed more efficiently than those who displayed higher HR, and possibly, more pre-test anxiety.

Limitations and Future Directions

A few important limitations that arose in the present study must be acknowledged. The first issue concerns the relationships that occurred with some of the physiological measures. For instance, higher pre-test IBI was associated with faster correct Go RTs. Although it might be tempting to interpret this finding as evidence that greater parasympathetic control led to faster correct RTs, such an assertion cannot be made. Because HR is antagonistically influenced by both sympathetic and parasympathetic activity, a particular HR can be the result of various combinations of activity between these two autonomic branches (Berntson, Cacioppo, Quigley, & Fabro, 1994). For instance, lower HR can be the result of decreased sympathetic activity, increased parasympathetic influence, or some combination of the two. Thus, although it is clear that slower HR was advantageous for participants in the present study in terms of executing correct Go responses more quickly, the exact contributions of each branch to HR in the present task cannot be deduced. Although we can speculate that this would have been due to increased cardiac vagal control, unfortunately, the evidence for that level of specificity is not present in the current data.

Although the present study showed no evidence of a relationship between RSA and behavioural performance on any level of the WMIC task, a relationship did emerge between

RSA and the ERN. In the future, it might be beneficial to explore this relationship in the context of an emotion-related task that taps the vACC. If the link between RSA and error-related electrocortical responsivity is due to their mutual associations with the vACC (Bush et al., 2000), an attention paradigm that is more directly related to emotional processing may provide the optimal context in which to explore this relationship further. Another option would be to use some type of dual task paradigm. Because the WMIC task requires the maintenance of items in WM for effective inhibitory control over response tendencies, it acts as sort of dual task paradigm. Because higher pre-test RSA was associated with larger ERNs on the high WM load condition, this finding seems to suggest that autonomic regulatory control was only relevant for younger adults when they were faced with the most cognitively demanding dual task condition. In the future, it might be beneficial to examine whether pre-test RSA relates to electrocortical responsivity in younger adults using another type of dual task paradigm.

Dywan and colleagues (1998) showed that when younger adults were instructed to complete a source memory task under divided attention in the form of a dual task, their performance mirrored that of older adults. They not only made more source memory errors, but they also produced electrophysiological responses similar to those of older adults. If RSA was found to relate to the electrocortical responsivity of younger adults under a divided attention source memory task, it would replicate the present finding and provide further evidence that modulatory control of CV arousal is most predictive of younger adults' ERP responses when they are faced with situations that severely strain attentional capacity.

Although higher pre-test RSA was associated with greater amplitude ERNs on the most demanding WM load condition, there was no evidence of a relationship between pre-

test RSA and behavioural performance in the present study. This may have been due to the fact that the present sample included only younger adults who may have found the attentional requirements such that they could manage the task irrespective of the amount of support obtained through the autonomic system. The next step is to examine how CV arousal relates to behavioural and electrophysiological responses in older adults using the WMIC task. It is important to investigate these relationships as our own lab has demonstrated evidence of such associations in older adults in the context of a source memory task (Dywan et al., submitted). Furthermore, because older adults are vulnerable to age-related structural declines in regions like the ACC (Raz & Rodrigue, 2006), the ability to implement effective control over CV arousal may become more critical to cognitive task performance with increased age.

By employing a sample of older adults, I will not only be able to determine whether RSA is associated with the behavioural and electrocortical responses of older adults in the context of the WMIC task, but I will also be able to examine the links between various autonomic, electrocortical, and behavioural measures within the older group to determine if these relations are different from those in younger adults. For instance, although there was no relationship between RSA and behavioural performance for younger adults in the present study, it will be interesting to find out whether such a relationship emerges in older adults who may be more reliant upon effective CV support for successful IC performance. By including a sample of older adults, it will also be possible to determine whether the strength or directionality of the relationships change with age. For instance, although higher pre-test RSA was associated with greater amplitude ERNs in the present group of younger adults, Dywan and colleagues (submitted) reported the inverse relationship for older adults who completed a source memory task. For this reason, it will be especially interesting to examine

if and how RSA relates to the amplitude of the ERN response in older adults completing the WMIC task.

Understanding the role of autonomic arousal in cognitive control will be especially important for future work with older adults. Although it is clear that aging is associated with cognitive decline, research suggests that a great deal of variability exists within the aging population in terms of cognitive abilities (Lupien et al., 2005). Furthermore, the majority of research on aging and cognition has focused solely on the relationship between behaviour and various measures of brain function. Although the exploration of these relationships has undoubtedly advanced our understanding of age-related declines in cognition, these models have not taken into account the role of autonomic regulatory control, a physiological component that has been shown to be highly relevant to task performance in some circumstances (Dywan et al., submitted; Hansen et al., 2003; Mathewson et al., in preparation). Gaining a better understanding of the relationship between autonomic arousal and cognitive control in older adults might not only advance our understanding of age-related cognitive decline, but also highlight key factors involved in healthy aging and options for successful intervention.

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Tables and Figures

Table 1.

Mean Proportion of Error Responses on Go and NoGo Trials for Each Level of Working Memory Load

	Errors of Commission on NoGo Trials			Errors of Omission on Go Trials	
WM Load	Mean	SD		Mean	SD
Low	.18	.09		.01	.01
Medium	.23	.09		.02	.03
High	.36	.13		.05	.04

Table 2.

Mean Response Times for Correct Go and NoGo Error Trials for Each Level of Working Memory Load

	Correct Go response times			NoGo error response times	
WM Load	Mean	SD		Mean	SD
Low	403	50.7		373	59.1
Medium	473	56.7		461	72.9
High	504	61.3		493	61.8

Table 3.

Mean Pre and Post-Test Cardiovascular Measures

	Pre-test			Post-test	
Measures	Mean	SD		Mean	SD
RSA	6.49	0.74		6.48	1.00
RPP	72.97	11.38		75.69	10.54
IBI	819.40	107.90		843.28	118.08
HR	74.38	9.02		72.39	8.91
SBP	98.30	11.80		105.02	13.11

Table 4.

Intercorrelations between Pre-Test Cardiovascular Measures

Subscale	RSA	RPP	IBI	HR	SBP
RSA	--	-.55**	.43*	-.41	-.30
RPP		--	-.67**	.64**	.66**
IBI			--	-.99**	.09
HR				--	-.14
SBP					--

*p < .05. **p < .01

Table 5.

Correlations between Pre-Test IBI and Correct Go Response Times Displayed Separately for Each Level of WM Load

	Low	Medium	High
Pre-Test IBI	-.401*	-.445**	-.469**

*p < .07. **p < .05

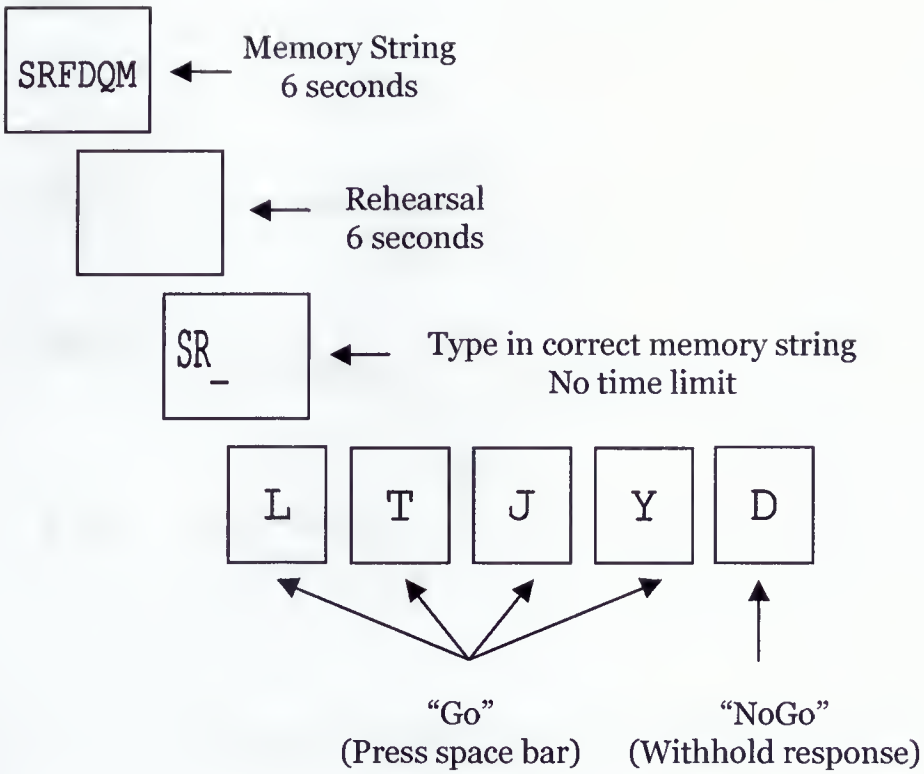

Figure 1.

Visual Illustration of the WMIC Task

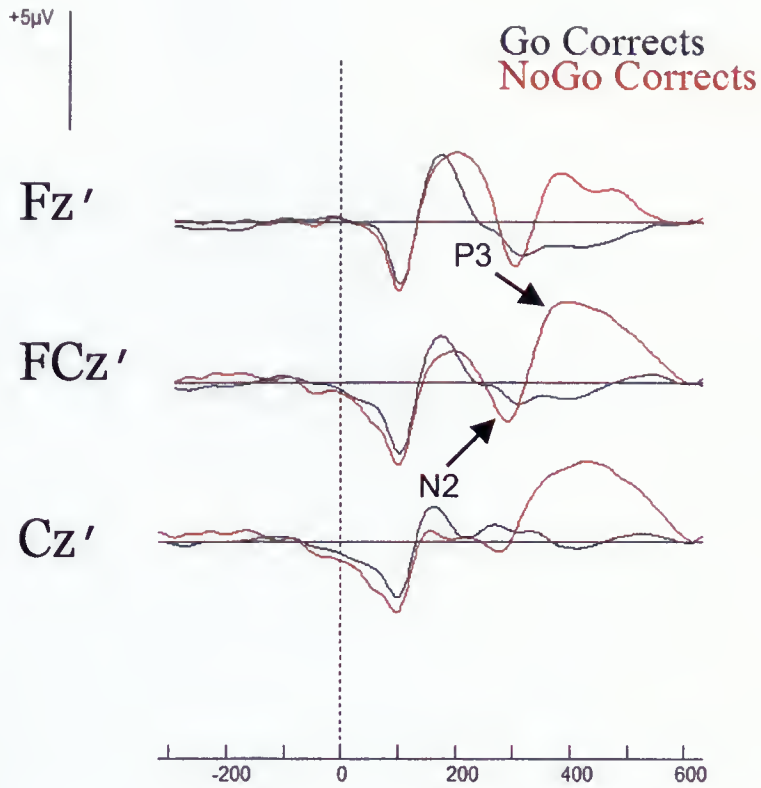

Figure 2.

Stimulus-Locked N2/P3 Components Elicited during Correct Withholding on NoGo Trials for the Low WM Load

′ Represents approximate scalp location

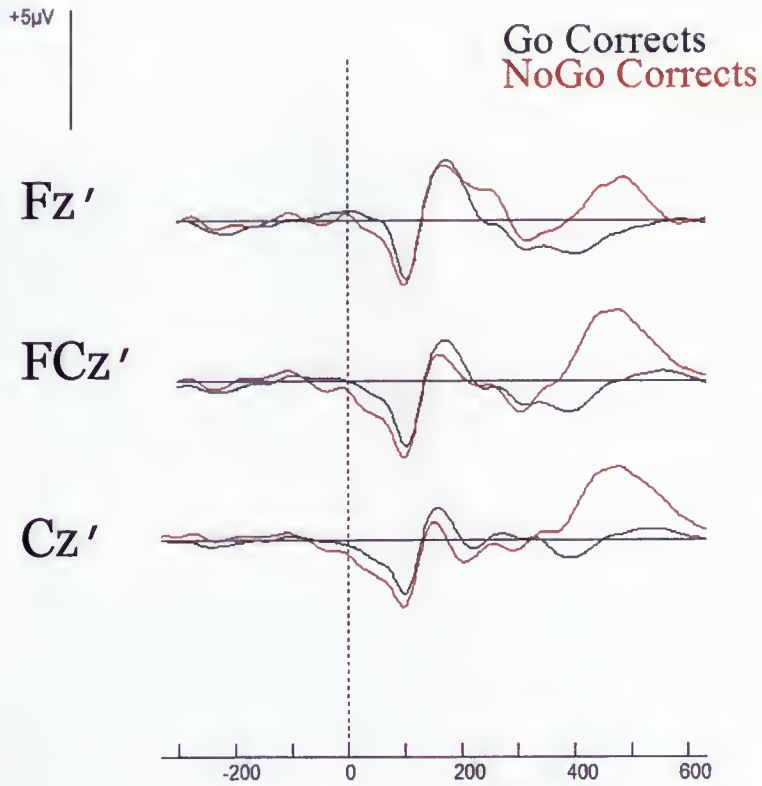

Figure 3.

Stimulus-Locked N2/P3 Components Elicited during Correct Withholding on NoGo Trials for the Medium WM Load

′ Represents approximate scalp location

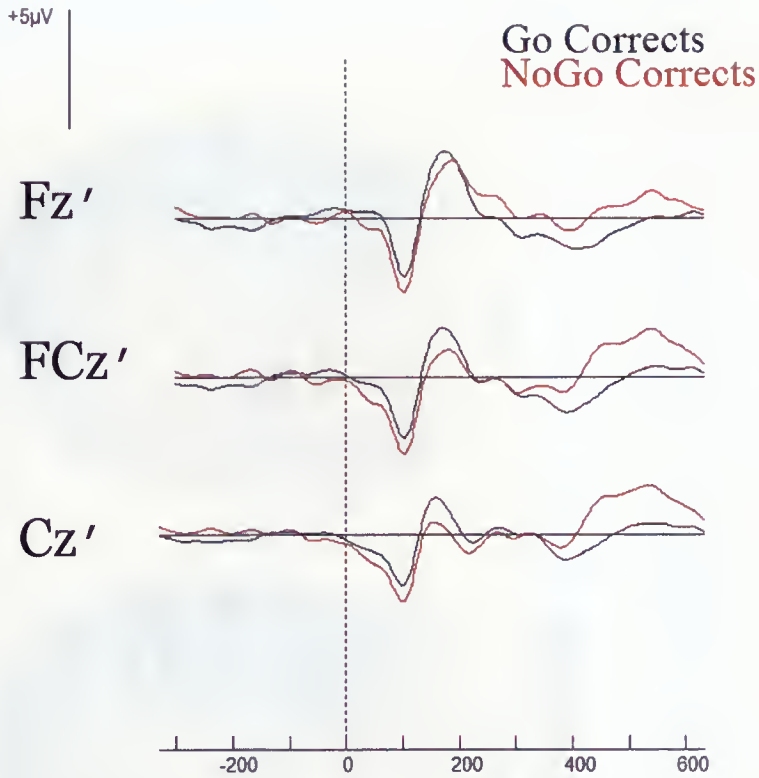

Figure 4.

Stimulus-Locked N2/P3 Components Elicited during Correct Withholding on NoGo Trials for the High WM Load

′ Represents approximate scalp location

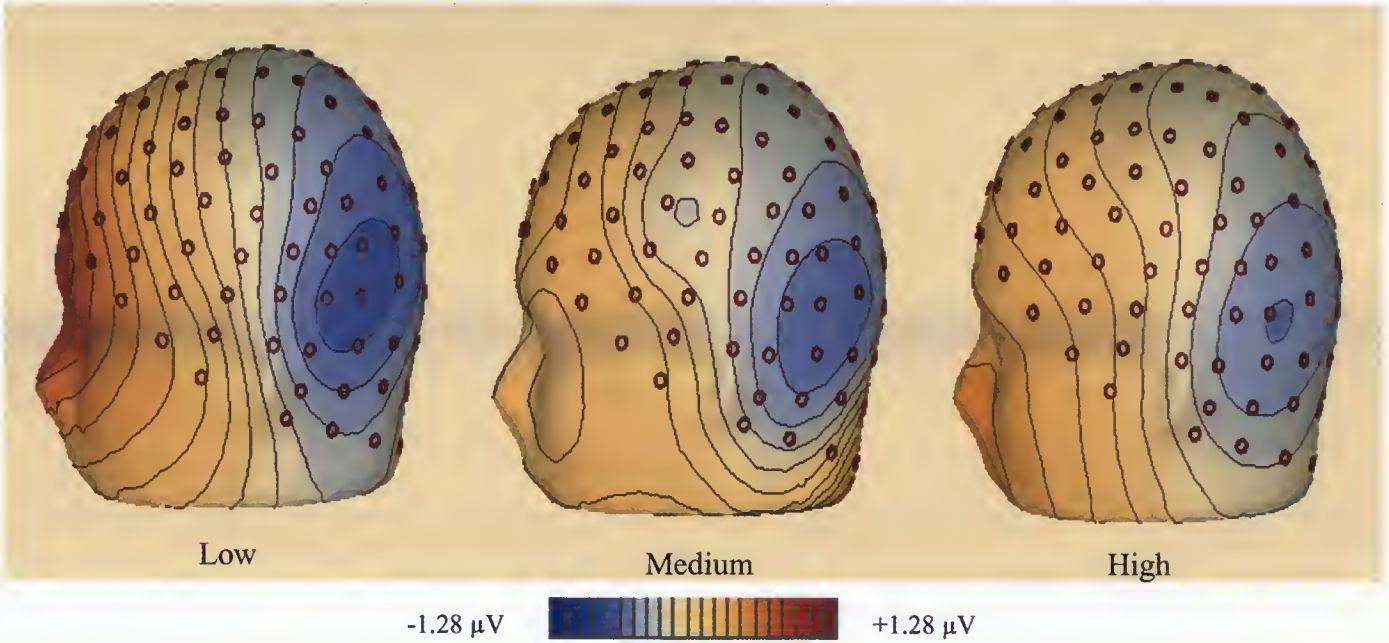

Figure 5
Topographical maps indicating greater N2 activity for the NoGo Correct Trials relative to the Correct Go Trials for all three WM Loads

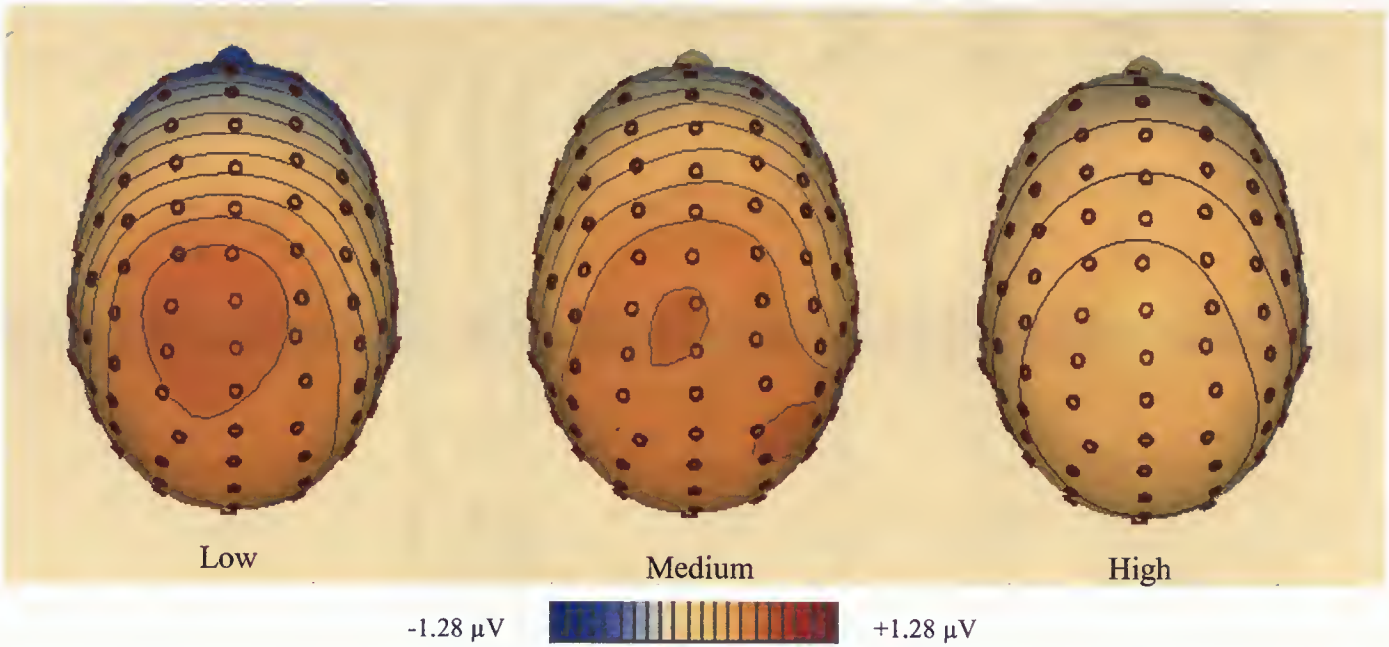

Figure 6
Topographical maps indicating greater P3 activity for the NoGo Correct Trials relative to the Correct Go Trials for all three WM Loads

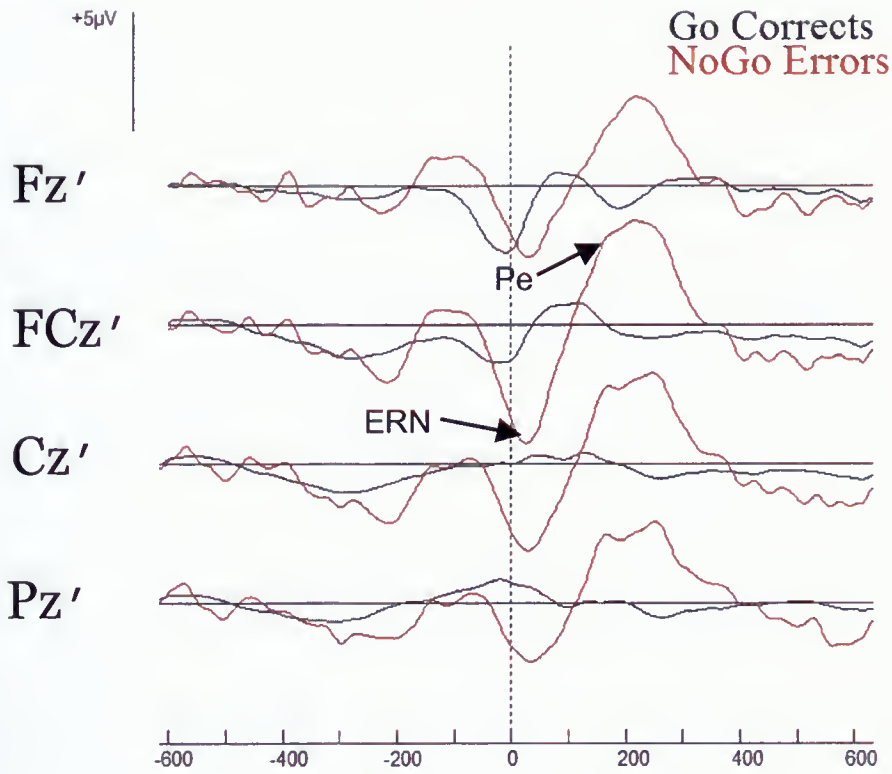

Figure 7.

Response-Locked ERN/Pe Components Elicited during Error Responses on NoGo Trials for the Low WM Load

′ Represents approximate scalp location

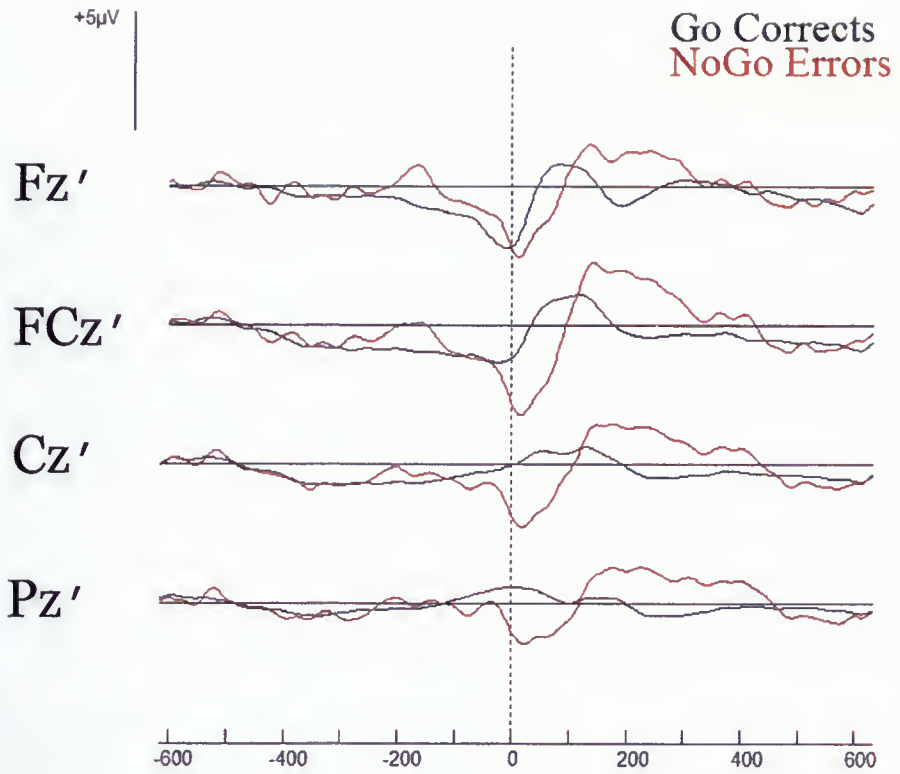

Figure 8.

Response-Locked ERN/Pe Components Elicited during Error Responses on NoGo Trials for the Medium WM Load

′ Represents approximate scalp location

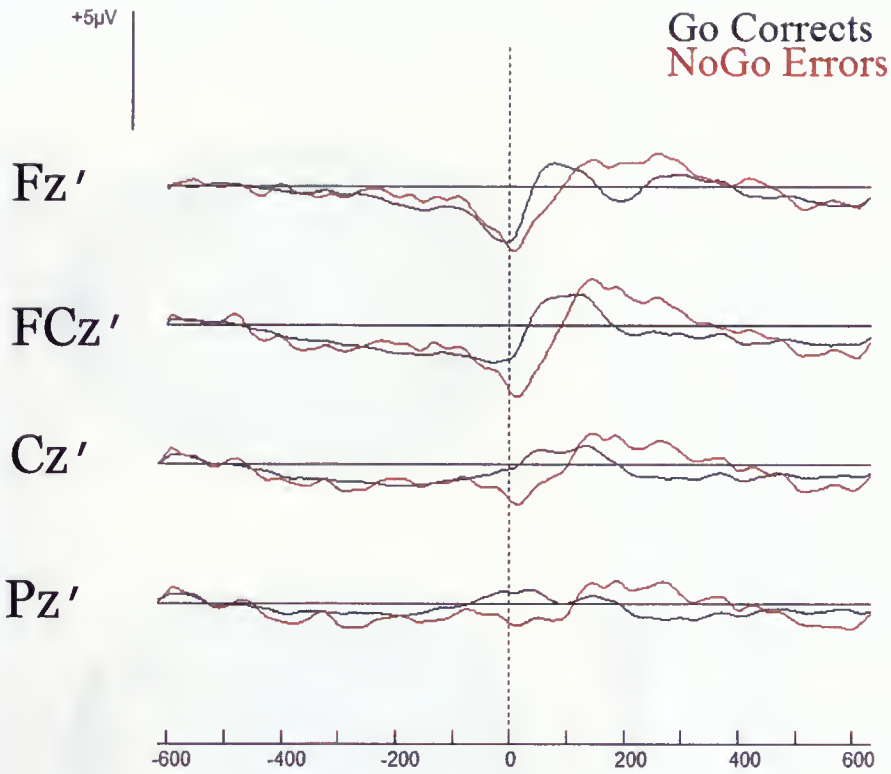

Figure 9.

Response-Locked ERN/Pe Components Elicited during Error Responses on NoGo Trials for the High WM Load

' Represents approximate scalp location

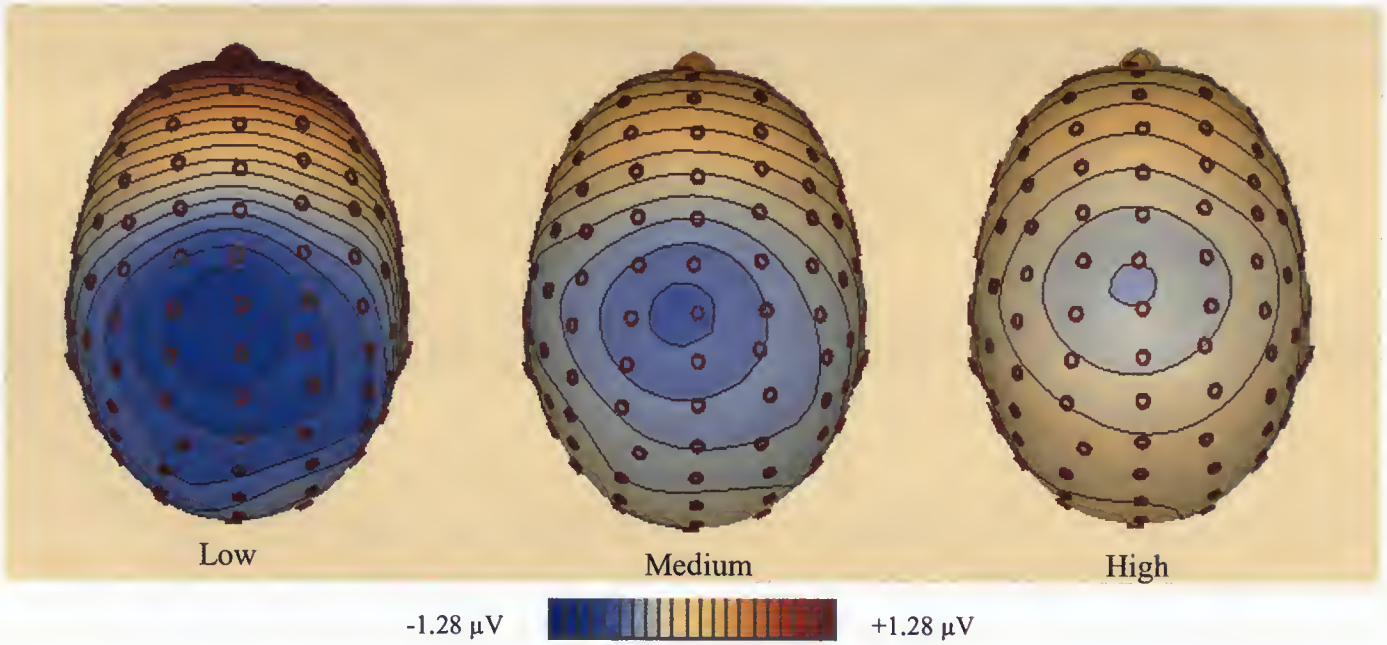

Figure 10
Topographical maps indicating greater ERN activity for the NoGo Error Trials relative to the Correct Go Trials for all three WM Loads

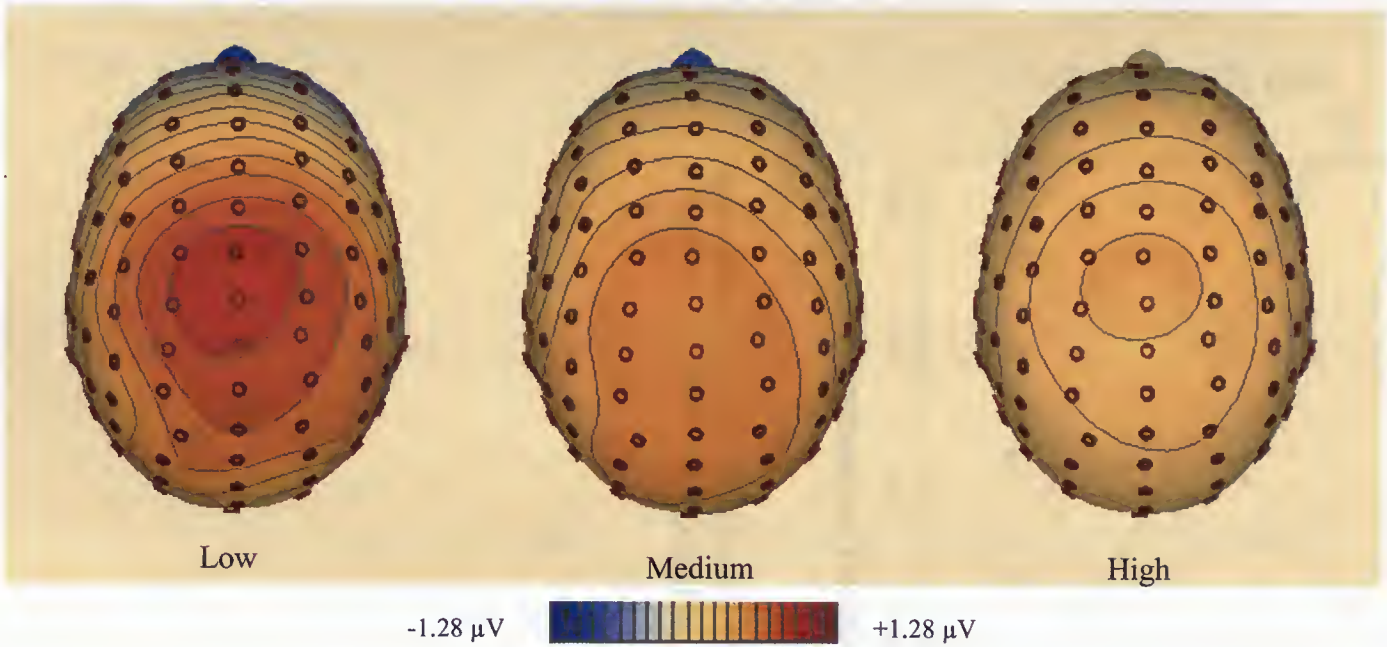

Figure 11
Topographical maps indicating greater Pe activity for the NoGo Error Trials relative to the Correct Go Trials for all three WM Loads

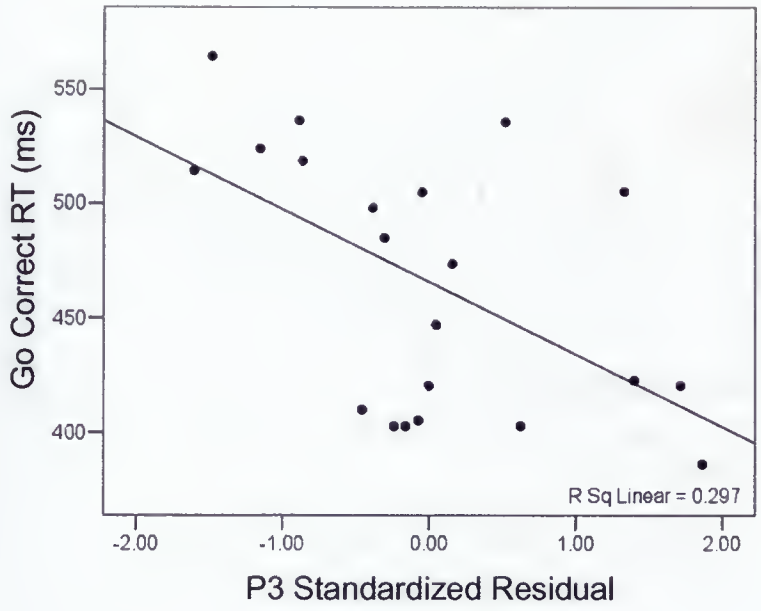

Figure 12.

Relationship Between the NoGo P3 and Correct Go RT for the Medium WM Load Condition

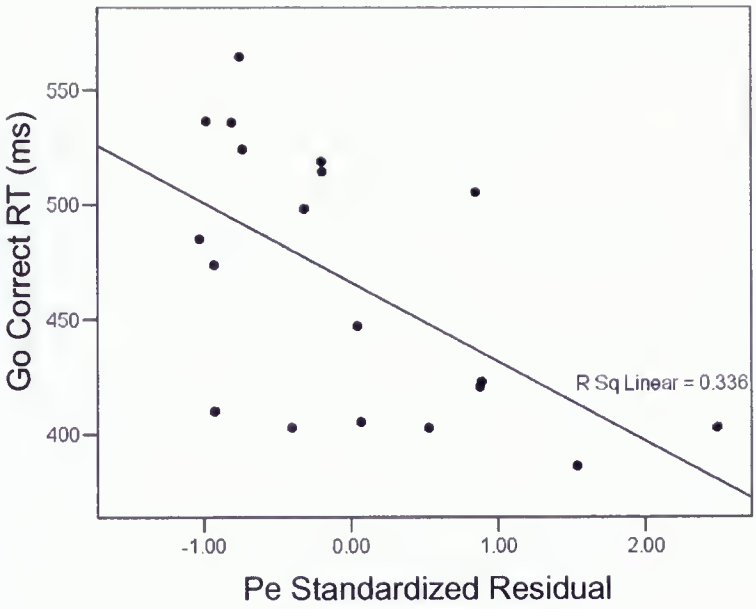

Figure 13.

Relationship Between the NoGo Pe and Correct Go RT for the Medium WM Load Condition

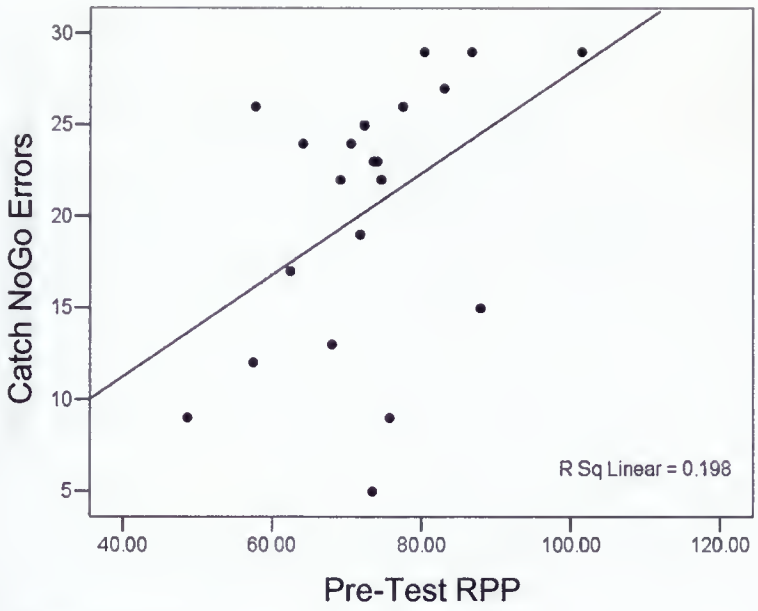

Figure 14.

Relationship Between Pre-Test RPP and Catch NoGo Errors (out of a possible 54 catch trials)

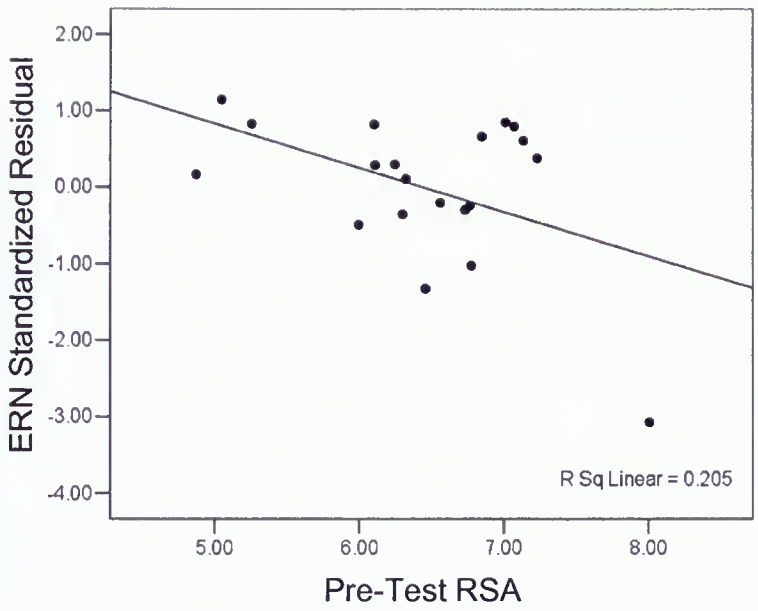

Figure 15.

Relationship Between Pre-Test RSA and ERN Amplitude at the High WM Load Condition

Appendix A: ANOVA Summary Tables

Table A.1.

Within Subjects Analysis of Variance for NoGo Errors across WM Load Conditions

Source	*df*	*F*	η^2	*p*
Condition	2	55.35*	.74	.00
C Within Group Error	40	(.003)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .001$

Table A.2.

Within Subjects Analysis of Variance for Go Omissions across WM Load Conditions

Source	*df*	*F*	η^2	*p*
Condition	2	23.62*	.54	.00
C Within Group Error	40	(.000)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .001$

Table A.3.

Within Subjects Analysis of Variance for Correct Go Response Times across WM Load Conditions

Source	*df*	*F*	η^2	*p*
Condition	2	200.70*	.91	.00
C Within Group Error	40	(270.19)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .001$

Table A.4.

Within Subjects Analysis of Variance for NoGo Error Response Times across WM Load Conditions

Source	*df*	*F*	η^2	*p*
Condition	2	114.65*	.85	.00
C Within Group Error	40	(706.63)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .001$

Table A.5.

Within Subjects Analysis of Variance for the NoGo N2

Source	*df*	*F*	η^2	*p*
Condition	2	4.55**	.19	.02
Trial-type	1	4.27*	.18	.05
Site	2	3.66*	.16	.05
Condition x Trial-type	2	3.33*	.14	.05
Condition x Site	4	9.00****	.31	.00
Trial-type x Site	2	6.65***	.25	.01
Condition x Trial-type x Site	4	.96	.05	.39
C x T x S Within Group Error	80	(.592)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .05$. **$p < .02$. ***$p < .01$. ****$p < .002$.

Table A.6.

Within Subjects Analysis of Variance for the N2 at the Low WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	6.14*	.24	.02
Site	2	9.55**	.32	.00
Trial-type x Site	2	1.04	.05	.34
T x S Within group error	40	(1.02)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .03$. **$p < .001$

Table A.7.

Within Subjects Analysis of Variance for the N2 at the Medium WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	3.12	.14	.09
Site	2	2.17	.10	.15
Trial-type x Site	2	9.36*	.32	.00
T x S Within group error	40	(.44)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .003$.

Table A.8.

Within Subjects Analysis of Variance for the N2 at the High WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	.25	.01	.63
Site	2	.92	.04	.38
Trial-type x Site	2	4.84*	.20	.02
T x S Within group error	40	(.60)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .03$.

Table A.9.

Within Subjects Analysis of Variance for the NoGo P3

Source	*df*	*F*	η^2	*p*
Condition	2	33.34**	.63	.00
Trial-type	1	49.38**	.71	.00
Site	2	15.25**	.43	.00
Condition x Trial-type	2	24.10**	.55	.00
Condition x Site	4	6.96**	.26	.00
Trial-type x Site	2	4.21*	.17	.03
Condition x Trial-type x Site	4	2.26	.10	.09
C x T x S Within Group Error	80	(.376)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .03$. **$p < .001$.

Table A.10.

Within Subjects Analysis of Variance for the P3 at the Low WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	62.41**	.76	.00
Site	2	12.34**	.38	.00
Trial-type x Site	2	4.83*	.20	.01
T x S Within group error	40	(.60)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .02$. **$p < .001$.

Table A.11.

Within Subjects Analysis of Variance for the P3 at the Medium WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	3.12	.14	.09
Site	2	2.17	.10	.15
Trial-type x Site	2	9.36*	.32	.00
T x S Within group error	40	(.44)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .003$.

Table A.12.

Within Subjects Analysis of Variance for the P3 at the High WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	.25	.01	.63
Site	2	.92	.04	.38
Trial-type x Site	2	4.84*	.20	.02
T x S Within group error	40	(.60)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .03$.

Table A.13.
Within Subjects Analysis of Variance for the ERN

Source	*df*	*F*	η^2	*p*
Condition	2	12.34***	.39	.00
Trial-type	1	86.64***	.82	.00
Site	3	4.26*	.18	.03
Condition x Trial-type	2	4.41**	.19	.02
Condition x Site	6	1.70	.08	.16
Trial-type x Site	3	11.88***	.39	.00
Condition x Trial-type x Site	6	1.01	.05	.40
C x T x S Within Group Error	114	(.976)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .03$. **$p < .02$. ***$p < .001$.

Table A.14.

Within Subjects Analysis of Variance for the ERN at the Low WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	50.56***	.72	.00
Site	3	4.54*	.19	.01
Trial-type x Site	3	7.81**	.29	.00
T x S Within group error	57	(2.59)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .02$. **$p < .003$. ***$p < .001$

Table A.15.

Within Subjects Analysis of Variance for the ERN at the Medium WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	90.27**	.83	.00
Site	3	2.76	.13	.09
Trial-type x Site	3	5.84*	.24	.01
T x S Within group error	57	(1.67)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .01$. **$p < .001$.

Table A.16.

Within Subjects Analysis of Variance for the ERN at the High WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	46.37*	.71	.00
Site	3	1.46	.07	.25
Trial-type x Site	3	14.21*	.43	.00
T x S Within group error	57	(1.11)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .001$.

Table A.17.
Within Subjects Analysis of Variance for the Pe

Source	*df*	*F*	η^2	*p*
Condition	2	14.45*	.43	.00
Trial-type	1	68.37*	.78	.00
Site	3	10.08*	.35	.00
Condition x Trial-type	2	19.18*	.50	.00
Condition x Site	6	1.35	.07	.26
Trial-type x Site	3	.51	.03	.55
Condition x Trial-type x Site	6	.85	.04	.49
C x T x S Within Group Error	114	(.816)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .001$.

Table A.18.

Within Subjects Analysis of Variance for the Pe at the Low WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	54.82**	.74	.00
Site	3	6.82*	.26	.00
Trial-type x Site	3	.94	.05	.39
T x S Within group error	57	(2.93)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .003$. **$p < .001$.

Table A.19.

Within Subjects Analysis of Variance for the Pe at the Medium WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	38.08**	.67	.00
Site	3	10.72**	.36	.00
Trial-type x Site	3	6.23*	.25	.01
T x S Within group error	57	(2.76)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .02$. **$p < .001$.

Table A.20.

Within Subjects Analysis of Variance for the Pe at the High WM Load Condition

Source	*df*	*F*	η^2	*p*
Trial-type	1	62.05**	.77	.00
Site	3	6.62*	.26	.00
Trial-type x Site	3	2.77	.13	.07
T x S Within group error	57	(.98)	-	-

Note: Values reported in parenthesis represent mean square errors.
*$p < .005$. **$p < .001$.

Appendix B: Research Ethics Board Acceptance

Date: Mon, 10 Dec 2007 15:18:39 -0500 [10/12/07 03:18:39 PM EDT]
From: Research Ethics Board <reb@brocku.ca>
To: jdywan@brocku.ca, lc06ti@badger.ac.brocku.ca
Cc: ibrindle@brocku.ca, Michelle McGinn <rebchair@brocku.ca>
Subject: REB 07-104 CAPUANA - Accepted as clarified

DATE: December 6, 2007

FROM: Michelle McGinn, Chair
Research Ethics Board (REB)

TO: Jane Dywan, Psychology
Lesley CAPUANA

FILE: 07-104 CAPUANA

TITLE: Cortical and Autonomic Modulation of Attentional Control in Aging

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The Brock University Research Ethics Board has reviewed the above research proposal.

DECISION: Accepted as clarified.

However, please note:

Data are not anonymous if code numbers can be traced back to consent forms.
You specify that participants cannot have visual impairments, yet you do not ask about this during the recruitment stage. If visual acuity is important, please add it to the screen in the telephone script.

This project has received ethics clearance for the period of December 6, 2007 to August 31, 2008 subject to full REB ratification at the Research Ethics Board's next scheduled meeting. The clearance period may be extended upon request. ***The study may now proceed.***

Please note that the Research Ethics Board (REB) requires that you adhere to the protocol as last reviewed and cleared by the REB. During the course of research no deviations from, or changes to, the protocol, recruitment, or consent form may be initiated without prior written clearance from the REB. The Board must provide clearance for any modifications before they can be implemented. If you wish to modify your research project, please refer to http://www.brocku.ca/researchservices/forms to complete the appropriate form Revision or Modification to an Ongoing Application.

Adverse or unexpected events must be reported to the REB as soon as possible with an indication of how these events affect, in the view of the Principal Investigator, the safety of the participants and the continuation of the protocol.

If research participants are in the care of a health facility, at a school, or other institution or community organization, it is the responsibility of the Principal Investigator to ensure that the ethical guidelines and clearance of those facilities or institutions are obtained and filed with the REB prior to the initiation of any research protocols.

The Tri-Council Policy Statement requires that ongoing research be monitored. A Final Report is required for all projects upon completion of the project. Researchers with projects lasting more than one year are required to submit a Continuing Review Report annually. The Office of Research Services will contact you when this form *Continuing Review/Final Report* is required.

Please quote your REB file number on all future correspondence.

MM/law

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