Published in final edited form as:
Neuropsychologia. 2013 December ; 51(14): 3033–3040. doi:10.1016/j.neuropsychologia.2013.10.008.

Increased Response Variability as a Marker of Executive Dysfunction in Veterans with Post-Traumatic Stress Disorder

Diane Swick, **Nikki Honzel**, **Jary Larsen**, and **Victoria Ashley**
Research Service, Veterans Affairs Northern California Health Care System, Martinez, CA and Department of Neurology, University of California, Davis

Abstract

The stability of cognitive control processes over time can be indexed by trial-to-trial variability in reaction time (RT). Greater RT variability has been interpreted as an indicator of executive dysfunction, inhibitory inefficiency, and excessive mental noise. Previous studies have demonstrated that combat veterans with post-traumatic stress disorder (PTSD) show substantial impairments in inhibitory control, but no studies have examined response variability in this population. In the current experiment, RT variability in the Go/NoGo response inhibition task was assessed for 45 veterans with PTSD and 34 control veterans using the intra-individual coefficient of variation and ex-Gaussian analysis of RT distributions. Despite having mean RTs that were indistinguishable from controls, the PTSD patients had significantly greater RT variability, which was not solely due to attentional lapses. More variable RTs were in turn associated with a greater number of false alarm errors, suggesting that less consistent performers were less successful at inhibiting inappropriate responses. RT variability was also highly correlated with self-reported symptoms of PTSD, depression, and attentional impulsiveness. Furthermore, response variability predicted diagnosis even when controlling for PTSD symptom severity. In turn, PTSD severity was correlated with self-rated attentional impulsiveness. Deficits in the sustained attention and top-down cognitive control processes that cause greater response variability might contribute to the maintenance of PTSD symptomology. Thus, the distractibility issues that cause more variable reaction times might also result in greater distress related to the trauma.

Keywords

PTSD; TBI; Go/NoGo; cognitive control; inhibitory control; impulsivity

Introduction

Consistency in behavioral responding is required for the efficient performance of many cognitive tasks. Often measured as trial-to-trial variability in reaction time (RT), intra-individual variability reflects the stability of executive control processes over time (West, Murphy, Armilio, Craik & Stuss, 2002). A high level of response variability has been

Corresponding author: Diane Swick, VA Northern California Health Care System, Research Service (151), 150 Muir Rd., Martinez CA 94553 USA, swicklab@gmail.com, phone: (925) 370-4081, fax: (925) 228-5738.

The authors declare that they have no conflicts of interest.

characterized as a marker of executive dysfunction and inhibitory inefficiency (Chuah, Venkatraman, Dinges & Chee, 2006), cognitive instability (Fjell, Westlye, Amlien & Walhovd, 2011), and mental noise (Ode, Robinson & Hanson, 2011). In line with the literature on attentional lapses and mind wandering (Smallwood, O'Connor, Sudbery & Obonsawin, 2007; Smallwood, Fitzgerald, Miles & Phillips, 2009), greater RT variability in cognitive tasks has been linked to a greater propensity towards negative affect and dysphoria (Ode et al., 2011). These observations suggest that individuals with anxiety and mood disorders would show greater RT variability than controls, but little is known about this relationship (but see Kaiser et al., 2008).

Response variability can also increase for a number of reasons not directly related to negative affect, including sleep deprivation (Chuah et al., 2006), aging (West et al., 2002), brain injuries (Segalowitz, Dywan & Unsal, 1997), and developmental disorders (Tamm et al., 2012). Increased RT variability in attention deficit hyperactivity disorder (ADHD) is a highly replicable finding. Several explanations have been proposed, including deficits in sustained attention or top-down control over attention, problems with temporal information processing, and difficulties in regulating behavioral state (Johnson et al., 2007; Tamm et al., 2012).

Patients with traumatic brain injuries (TBI) also show greater response variability as assessed by individual standard deviations (SD) in simple and choice RT tasks (Stuss et al., 1989) and more convincingly, by a metric that controls for differences in raw RT (Stuss, Pogue, Buckle & Bondar, 1994). The intra-individual coefficient of variation (ICV) takes into account the speed of responding, since patient populations can have longer RTs than controls (Stuss, Murphy, Binns & Alexander, 2003).

Many studies have also examined more complex aspects of response variability to quantify the role of attentional lapses (West et al., 2002; Epstein et al., 2011). RT distributions generally consist of two components: the Gaussian (normal) distribution and an exponential component, the rightward skew or tail consisting of long RTs (Heathcote, Popiel & Mewhort, 1991; Whelan, 2008). The ex-Gaussian model uses three parameters to describe the RT distribution: mu (μ = mean of normal distribution), sigma (σ = SD of normal distribution), and tau (τ = mean and SD of the exponential tail). This model has demonstrated that children with ADHD show significant increases in tau relative to controls, which reflects a greater percentage of slow RTs (Hervey et al., 2006; Tarantino, Cutini, Mogentale & Bisiacchi, 2013). The increase in tau can account for their overall RT slowing, since mu values were smaller (or unchanged) in the ADHD group. This pattern of results may reflect more frequent attentional lapses. However, this interpretation is disputed by some, who maintain that the mapping of ex-Gaussian parameters onto cognitive processes is not straightforward (Matzke & Wagenmakers, 2009).

Specific regions of the prefrontal cortex (PFC) have been associated with response variability. A neuropsychological study demonstrated that patients with lesions including the lateral PFC or the dorsomedial PFC showed more variable RTs both within and across testing sessions (Stuss et al., 2003). Patients with injuries to ventromedial PFC alone did not show this impairment. Neuroimaging experiments also support a role for PFC in response

variability. Greater response variability in a Go/NoGo (GNG) task was positively correlated with activity in bilateral middle frontal, right inferior parietal, and thalamic regions (Bellgrove, Hester & Garavan 2004). The authors suggested that the less behaviorally consistent subjects needed to recruit the inhibitory control network to a greater extent. Similar results were obtained in typically developing children: ICV and NoGo activation showed a positive correlation in right PFC and right caudate/anterior thalamus (Simmonds et al., 2007). Furthermore, response variability was highly correlated with false alarm error rates in these studies, suggesting that more variable participants were less successful at inhibiting prepotent responses.

There have been fewer investigations of response variability in psychiatric populations. The most studied population has been patients with schizophrenia, who consistently show greater RT variability than controls (van den Bosch, Rombouts & van Asma, 1996; Vinogradov, Poole, Willis-Shore, Ober & Shenaut, 1998). A GNG study by Kaiser et al. (2008) observed an increase in ICV in groups of inpatients being treated for schizophrenia, depression, or borderline personality disorder. Interestingly, the groups did not differ from each other. This raises the question of whether impairments in a general cognitive mechanism (such as attentional control) can account for the findings, or whether different mechanisms can be implicated. It could also be the case that various causes of attentional control problems (e.g., distractibility, negative ruminations, delusions, etc.) are responsible for the deficits in different groups.

At present, no studies have examined response variability in post-traumatic stress disorder (PTSD), an anxiety disorder that can interfere with cognitive performance and emotion regulation (Vasterling & Verfaellie, 2009). Individuals with PTSD exhibit symptoms of re-experiencing, avoidance/numbing, and hyperarousal. The severity of re-experiencing symptoms in particular (flashbacks, nightmares, and intrusive memories of the traumatic event) has been associated with worse performance on inhibitory control tasks (Vasterling, Brailey, Constans & Sutker, 1998; Swick, Honzel, Larsen, Ashley & Justus, 2012). This suggests that inhibitory control might be critical for disengaging from an overwhelming preoccupation with the traumatic event(s) (Aupperle, Melrose, Stein & Paulus, 2012).

The current study assessed RT variability in combat veterans with PTSD (n=45) and age-matched control veterans (n=34). The PTSD patients were veterans of the wars in Iraq and Afghanistan, most of whom had also reported mild TBI (n=34). The majority of control veterans had been deployed, but none had been diagnosed with PTSD or mTBI. We predicted that PTSD patients would show greater response variability than controls in a GNG task. If lapses of attention are responsible for this effect, then an increase in tau, but not in sigma, would be expected (Lee et al., 2012). The experimental design manipulated the difficulty of inhibition, and we predicted the patients would show relatively greater impairment in the harder condition (Swick et al., 2012). We also predicted that ICV would correlate with the severity of PTSD symptoms. In addition, we expected that ICV would correlate with the severity of self-reported depressive symptoms, since depression and PTSD are highly co-morbid in this population (Pittman, Goldsmith, Lemmer, Kilmer & Baker, 2012). Finally, a strong correlation between ICV and false alarm error rates was expected

(Bellgrove et al., 2004), which would suggest that more variable participants are less successful at inhibiting inappropriate responses.

Methods

Participants

The participants were 45 combat veterans diagnosed with PTSD (44 male, 1 female) and 34 age-matched veteran controls (32 male, 2 female). Among the PTSD patients, 34 had sustained one or more mTBIs (primarily due to blast injury while serving in the military), while 11 had no history of mTBI (see Table 1 for details). Participants with evidence of significant medical disease, severe psychiatric problems (such as schizophrenia or bipolar disorder), active substance abuse, visual deficits, or history of other neurological events were excluded. Another 7 participants (5 patients, 2 controls) were initially enrolled, then excluded when additional information was revealed (childhood TBI; non-military PTSD; moderate TBI; other neurological or psychiatric disorder; not Iraq or Afghanistan). Most of the patients were identified and diagnosed in the TBI clinic of the consulting neurologist. A semi-structured clinical interview was conducted, and mild TBI was diagnosed based on patient self-report of the following criteria from the VA/DoD Clinical Practice Guidelines – loss of consciousness (LOC) 30 min or less or altered mental status (e.g., feeling dazed, disoriented, or confused), with post-traumatic amnesia less than 24 hrs (The Management of Concussion/mTBI Working Group, 2009). In most patients with LOC, the duration was very brief (1–2 min). PTSD diagnosis was based on semi-structured clinical interview using DSM-IV criteria. The diagnoses of mTBI and PTSD were corroborated with available VA medical records, to the fullest extent possible.

Controls were recruited primarily through advertisements. Potential control subjects were screened for history of mTBI, PTSD, and other exclusionary criteria through an initial telephone interview, and further assessed at the first visit. Demographic information is shown in Table 1. The groups were matched for age but not education level. This could be due to the inability of many of the patients to return to school after their military service, and is typical of earlier studies on veterans with PTSD (e.g., McNally Kaspi, Riemann & Zeitlin, 1990; Vrana, Roodman & Beckham, 1995). However, another possibility is that low education serves as a risk factor for developing PTSD (Iversen et al., 2008; Larson, Booth-Kewley, Highfill-McRoy & Young, 2009); thus, those with lower educational attainment were at greater risk for PTSD. Level of education did not influence the outcome, however, as will be discussed in the Results (see also Swick et al., 2012).

Sample sizes were determined by aiming for target enrollment numbers specified in the grant proposal. Initially there was to be a third patient group: a cohort of mTBI patients without PTSD. It was not known in advance that most patients who met the selection criteria for mTBI would also have a formal PTSD diagnosis. Therefore, it was necessary to drop the mTBI-only group (n=3). Other researchers have observed high levels of mTBI/PTSD co-morbidity in U.S. combat veterans who served in Iraq and Afghanistan (Carlson et al., 2011; Taylor et al., 2012). A group of civilian controls (n=31; one excluded) was recruited specifically for comparison to veteran control subjects in an emotional Stroop task that used combat-related words (Ashley, Honzel, Larsen, Justus & Swick 2013). To conform to the

procedures conducted with the other groups, they were tested on the GNG task in the same session. Their data are not reported here.

English was the primary language for all participants. The subjects signed informed consent statements approved by the Institutional Review Board of the VA Northern California Health Care System and were paid for their participation. All procedures were in compliance with the Declaration of Helsinki.

Go-NoGo Task

Stimuli consisted of letters printed in a large black font on a white background (Swick et al., 2012). Single uppercase letters were rapidly and serially presented at the center of a computer screen for 200 ms duration once every 1500 ms. Subjects were instructed to respond as quickly as possible to every letter except for "X" by pressing a button on the keyboard with the index finger of the dominant hand. In four alternate blocks, the proportion of "Go" to "NoGo" trials alternated between 50/50 and 90/10. There were 140 trials per block, with short rest breaks between each block. A short practice set of 30 trials (15 Go and 15 NoGo, randomly intermixed) preceded the experimental trials.

Questionnaires

At the end of the session, the subjects completed three self-report questionnaires: the Barratt Impulsiveness Scale (BIS-11A), the PTSD Checklist, Military Version (PCL-M), and the Beck Depression Inventory (BDI). The BIS-11 is a 30 item self-report instrument designed to measure the personality construct of impulsiveness (Patton, Stanford & Barratt, 1995). The BIS-11 contains three subscales thought to assess attentional, motor, and non-planning impulsiveness. The widely-distributed BIS-11A was used inadvertently in this study instead of the more validated BIS-11 (Stanford et. al., 2009). We used the prorating method developed by Dr. Marijn Lijffijt to score the data (http://impulsivity.org/BIS-11/bis-10r-bis-11a-issue). The PCL-M is a 17-item self-report tool that establishes the presence and degree of PTSD symptoms in military personnel (Weathers, Litz, Huska & Keane, 1994). It has three clusters or subsets: re-experiencing, avoidance/numbing, and hyperarousal. PTSD is indicated in a veteran population with a score of 50 or greater (Forbes, Creamer & Biddle, 2001). The PCL-M scores of two control participants who had not yet sought clinical care placed them in the PTSD group (and vice versa: one veteran recruited from the community initially self-identified as having PTSD but had a low score on the PCL-M). The BDI is one of the most commonly used self-report screens for major depressive disorder and has been validated with well-established psychometric properties (Beck, Steer & Gabin, 1988).

Data Analysis

As recommended by Simmons, Nelson and Simonsohn (2012): "We report how we determined our sample size, all data exclusions (if any), all manipulations, and all measures in the study."

The measure of RT variability on correct Go trials, the intra-individual coefficient of variation (ICV), was obtained from SD/mean RT (Stuss et al., 2003). The ICV ratio is a standard measure designed to correct for differences in group RTs. The ex-Gaussian

parameters mu, sigma, and tau were calculated using the QMPE program (described in Heathcote, Brown, & Cousineau, 2004). The average number of trials used in the estimation for the 50/50 condition was 136 trials (range 70–140) for patients and 139 trials (range 127–140) for controls. There was an equipment malfunction during one block of this condition in one of the patients, but his data from the other block were still analyzed. In the 90/10 condition there was an average of 248 trials for patients (range 223–252) and 251 trials (range 245–252) for controls.

Statistical analyses were carried out using repeated measures analyses of variance (ANOVAs) with factors of group (patients, controls) and probability (50/50, 90/10). The alpha level was set at 0.05. Secondary analyses compared patients with mTBI and PTSD to those with PTSD only. The correlations between self-report measures and ICV in the difficult 90/10 condition were determined using the Spearman rank-order statistic, with a Bonferroni correction for multiple comparisons (alpha level .005). Spearman's correlation was chosen because the questionnaire data are on an ordinal scale.

The error data from most of the present participants were reported in a separate publication on inhibitory control (Swick et al., 2012). The inclusion of six additional participants here (five patients, one control) did not change the outcome. The current paper focuses on RT variability and its relationship to self-reported symptoms of PTSD and depression.

Results

Response Variability

An ANOVA examining ICV revealed that the PTSD patients showed significantly greater response variability than controls [$F(1,77)=12.63$, $p=.0007$, $\eta_p^2=.14$]. RTs were more variable in the difficult 90/10 condition than in the 50/50 condition, and the patients showed this probability-related increase to a greater extent (Fig. 1, top). These findings were confirmed by a main effect of probability [$F(1,77)=17.28$, $p<.0001$, $\eta_p^2=.18$] and a group × probability interaction [$F(1,77)=8.24$, $p=.005$, $\eta_p^2=.10$]. Veterans with PTSD showed greater ICV than controls in both the 50/50 [$F(1,77)=8.78$, $p=.004$, $d=0.67$] and 90/10 conditions [$F(1,77)=13.07$, $p=.0005$, $d=0.81$]. A secondary ANOVA using SD values also observed a main effect of group [$F(1,77)=6.12$, $p=.02$, $\eta_p^2=.07$] and a group × probability interaction [$F(1,77)=4.74$, $p=.03$, $\eta_p^2=.06$].

In contrast to the group differences in response variability, mean RTs did not differ between the PTSD patients and controls [$F(1,77)=.25$, $p=.62$], nor did group interact with probability [$F(1,77)=.22$, $p=.64$] (Fig. 1, bottom). The significant main effect of probability indicated that all participants were faster in the 90/10 condition than in the 50/50 condition [$F(1,77)=203.49$, $p<.0001$, $\eta_p^2=.73$]. Thus, an interesting aspect of the present findings is that patients and controls were very well matched on mean RT, yet they showed significant differences in response variability.

To examine the shape of the RT distribution, the three ex-Gaussian parameters were entered into separate ANOVAs. Mu was numerically smaller in the patients (Table 2), indicating faster RTs for the normal component of the curve [$F(1,77)=2.04$, $p=.16$, $\eta_p^2=.03$], but this

was not significant. Sigma was larger in the patients, reflecting greater SD for the normal component [F(1,77)=3.38, p=.07, η_p^2 =.04], but this was only a trend. Likewise, tau was larger in the patients at the trend level [F(1,77)=3.73, p=.06, η_p^2 =.05]. Taken together, these results suggest that increased response variability in PTSD was not entirely due to a preponderance of long RTs in the rightward tail, but also included more variability around the Gaussian distribution. This interpretation should be made with caution, however, since none of the main effects of group reached significance. The group × probability interactions were not significant, either (p's>.3). This could be due to the fact that unlike ICV, these parameters are not ratios that adjust for RT differences in the two conditions.

Next, the relationship between RT variability and error rate was examined across groups. The Pearson correlations between ICV and false alarm errors were significant for both the 50/50 (r=.56, p<.0001) and the 90/10 conditions (r=.49, p<.0001), suggesting that more variable RTs were associated with a greater propensity to respond inappropriately. As reported previously (Swick et al., 2012), the ANOVA for false alarm errors demonstrated that the patients were significantly less accurate than the controls [F(1,77)=25.68, p<.0001, η_p^2 =.25], and that this impairment was greater in the 90/10 condition [group × probability, F(1,77)=12.07, p=.0008, η_p^2 =.14].

In addition to tau, another possible indicator of attentional lapses is the rate of missed responses on Go trials. However, the rate of misses was very low for all participants in this study (controls: 0.46% and patients: 1.73%). Although the difference between groups was statistically significant [F(1,77)=5.74, p=.02, η_p^2 =.07], the floor effect makes the result difficult to interpret in that fashion.

To determine whether the presence of mild TBI exacerbated the increase in RT variability seen with PTSD, a secondary analysis compared patients with PTSD and mTBI (n=34) to those with PTSD only (n=11) for the difficult condition. The addition of mTBI did not affect performance, as there was no difference between the ICV values of the two patient subgroups (p=.41), nor were there differences in raw RTs (p=.73) or error rates (p=.99). Further analyses within the PTSD + mTBI group determined that neither LOC (p=.70) nor number of events (one vs. more than one, p=.68) affected response variability. However, given the relatively small sample sizes, the power to detect differences between the patient subgroups was low.

Finally, to examine the effects of education, a group of 23 patients matched in education to the controls (14.56 yrs vs. 14.68 yrs, p=.77) still differed significantly for ICV in both the 50/50 (p=.006, *d*=0.73) and 90/10 conditions (p<.0001, *d*=1.12). The ANOVA comparing these groups confirmed that the education-matched patients were more variable than the controls, especially in the 90/10 condition: main effect of group [F(1,55)=16.49, p=.0002, η_p^2 =.23]; group × probability interaction [F(1,55)=12.26, p=.0009, η_p^2 =.18].

Post-Error Slowing

One potential source of greater ICV in the patients could be due to their higher error rates and subsequent slowing down after error commission (Rabbitt, 1966). This would inflate the degree of variability. Therefore, we compared RTs on correct Go trials after a false alarm

error to RTs on correct Go trials that did not follow an error, using a subtraction measure. This ANOVA did not find a significant effect of group [F(1,76)=1.30, p=.26] or a group × probability interaction [F(1,76)=0.44, p=.51]. The amount of post-error slowing in this task was highly variable and did not differ from zero in either group (controls: −4.8 + 35.6 ms; patients: 2.8 + 46 ms). One control was omitted from the analysis because he did not have any errors in the 90/10 condition. Further examination of the data revealed that a number of participants had few errors in one or both of the conditions (out of 140 NoGo trials in 50/50 and 28 NoGo trials in 90/10). Because of possible instability for cells based on very low numbers of trials, we repeated the analysis where only participants with at least five errors in each cell were included (n=38 patients, n=22 controls). The same result was obtained: neither the main effect of group [F(1,58)=0.52, p=.47] nor the interaction [F(1,58)=1.13, p=.29] were significant.

Correlations Between Experimental and Self-Report Measures

As expected, the patients reported substantially higher PCL-M, BDI, and BIS-11 scores than the control group, but there were no differences between PTSD patients with and without mTBI (Table 1). The association between self-report measures and ICV in the difficult 90/10 condition (across all 79 participants) was determined using Spearman Rank Correlations (corrected at p<.005). Scores on the PCL-M and BDI showed a strong correlation with performance: more severe levels of PTSD (rho=.40, p=.0003) and depression symptoms (rho=.33, p=.003) were both associated with more variable RTs. All three PTSD symptom clusters showed individual correlations with ICV: re-experiencing (rho=.43, p=.0001), avoidance/numbing (rho=.33, p=.003), and hyperarousal (rho=.40, p=.0003). Since these PCL-M subscales were highly correlated with each other (r's>.8), we did not include them in one model.

To further examine the relationship between response variability and PTSD, we first note that overall PCL-M score predicted group membership in a logistic regression (O.R. = 1.164, C.I. = 1.095 to 1.238, p<.0001), demonstrating good agreement between clinician diagnosis and self report. In addition, group predicts PCL-M score [t(77)=11.51, p<.0001; see Table 1] and ICV [t(77)=3.51, p=.0005; Controls, mean = 0.199, range = 0.123 to 0.317; Patients, mean = 0.283, range = 0.165 to 0.855], and PCL-M predicts ICV in a simple linear regression, r=.36, p=.001. We conducted two additional logistic regressions that included ICV*100 as predictors of group. In the first, ICV*100 alone predicted group (O.R. = 1.237, C.I. = 1.092 to 1.403, p=.0009). The second regression controlled for PCL-M scores and still found that ICV*100 predicted group membership (O.R. = 1.245, C.I. = 1.004 to 1.545, p=.046). Thus, for every increase of 100 units in ICV (e.g., from 0.247 to 0.347), assuming PCL remains constant, the odds of PTSD increase by 24.5%. PCL-M score also predicted group in this model (O.R. = 1.167, C.I. = 1.090 to 1.250, p<.0001). For every one unit increase in PCL, assuming ICV remains constant, the odds of PTSD increase by 16.7%. Therefore, the effect of ICV on PTSD is not simply mediated by its relationship to PCL. Even after controlling for PCL-M score, ICV predicts whether an individual will have PTSD, in spite of the strong relationship between PCL-M and ICV.

Self-rated impulsivity (total BIS-11 score) was not significantly correlated with response variability (rho=.20, p=.07), nor were scores on the motor (rho=.05) and non-planning subscales (rho=.12). However, scores on the attentional impulsiveness subscale were significantly correlated with ICV (rho=.33, p=.003). We wished to see if there was a further relationship between self-rated attentional impulsiveness and PTSD symptoms. The correlation between attentional impulsiveness and PCL-M scores was very high (rho=.77, p<.0001). Finally, a multiple regression that included the three BIS-11 subscales indicated that attentional impulsiveness was the only significant predictor of PTSD severity (p<.0001; see Table 3). The BIS-11 subscores were less highly intercorrelated (r's<.6) than the PCL-M subscores.

Discussion

Combat Veterans with PTSD had more variable reaction times in a Go/NoGo motor inhibition task than control veterans. The patients showed greater intra-individual variation in response times despite having mean RTs that were indistinguishable from controls. The increase in variability was not only due to a greater proportion of unusually slow responses, but also to greater variability around the normal RT distribution. More variable RTs were in turn associated with a greater number of errors, replicating previous findings in adult controls (Bellgrove et al., 2004) and children with ADHD (Suskauer et al., 2008). RT variability was correlated with the severity of self-rated PTSD and depression symptoms, suggesting a relationship between the amount of personal distress and the lapses of attention that contribute to less efficient cognitive function. Furthermore, response variability predicted group membership even when controlling for PTSD symptoms. These findings are consistent with the "mental noise" hypothesis, in which negative affective states serve as a distraction from stable task performance (Ode et al., 2011). However, as these authors have noted, response variability is a rather general marker of executive dysfunction, because alterations have been observed in a range of conditions. It is not yet known whether impairments in a general cognitive mechanism (e.g., top-down attentional control) can account for similar deficits across groups, or whether different mechanisms are involved.

For example, previous experiments have demonstrated that patients diagnosed with major depression, borderline personality disorder, and schizophrenia all show greater RT variability than controls (van den Bosch et al., 1996; Vinogradov et al., 1998; Kaiser et al., 2008). Under the mental noise rubric, negative affect results in increased response variability, and this might be due to depressive rumination, dysregulated emotional control, and psychotic symptoms in these three groups (respectively). The study of Kaiser et al. (2008) manipulated the probability of responding in a GNG task. All three patient groups showed increased variability on the standard version of GNG (80% Go, 20% NoGo). However, only the schizophrenia group showed an increase in ICV in the easy condition, where the probability of Go and NoGo trials was reversed (20% and 80%, respectively). Thus, the task context of response inhibition vs. target detection was able to distinguish RT variability in schizophrenia from that seen in depression and borderline personality disorder.

In the present study, the PTSD patients showed significantly higher ICV values than controls in both the easy (50/50) and difficult (90/10) conditions, with relatively greater

impairment in the latter. This might reflect the more boring or monotonous nature of responding on 90% of the trials, when the task becomes more routine. In this condition, when cognitive control is low and the mind more prone to wander, all participants speed up, make more false alarm errors, and have more variable RTs. The patients showed exacerbated deficits in the difficult condition, suggesting limitations in executive control of behavioral responses. A question for future research is the degree to which performance on various executive control tasks is impaired or spared in PTSD (Honzel, Justus & Swick., submitted). Working memory capacity has been related to mind wandering in both laboratory tasks and daily activities (Kane & McVay, 2012), so it will be informative to examine working memory in relation to response variability. A recent review of the PTSD literature (Polak, Witteveen, Reitsma & Olff, 2012) generally found impairments in some standardized neuropsychological measures (Trails B, Wisconsin Card Sorting Test and Digit Span), but less consistently so in others (Color-Word Stroop and Digits Backwards). The existence of such dissociations is supported by latent variable analyses in controls, suggesting that response inhibition, set shifting, and working memory updating are separable (Miyake et al., 2000).

A greater contribution from post-error slowing could not account for the ICV increase in patients. Under this view, since PTSD patients made more errors than controls, they could have shown greater compensatory slowing down after each error, or an increase in the number of slower responses in line with the error rate. Either could cause an increase in ICV. However, neither group showed significant post-error slowing at all in the present Go/NoGo task, in line with previous observations (Hester Bell, Foxe & Garavan, 2013). In that study, abstinent cocaine dependent participants and controls did not show a difference in RT on correct trials immediately preceding and following an error (Hester et al., 2013). Post-error speeding has been observed by this group as well (Hester, Foxe, Molholm, Shpaner & Garavan, 2005). These findings may reflect a difference in compensatory strategies seen in GNG compared to two-choice RT tasks, which comprise the bulk of the literature on this topic (Danielmeier & Ullsperger, 2011). Another consideration is that post-error slowing is largest under accuracy conditions (Danielmeier & Ullsperger, 2011), and speed was emphasized here.

Results from the ex-Gaussian analyses suggested that attentional lapses could not uniquely account for overall RT variability. Tau and sigma both showed trend-level differences at the group level, indicating a tendency for patients to have a greater proportion of unusually slow RTs as well as a broader distribution of RTs in the Gaussian component, respectively. A greater number of slow responses might reflect failures of sustained attention, while a greater number of fast responses (along with a substantial increase in false alarm errors) might suggest failures of inhibition and top-down attentional control (see also Swick et al., 2012).

However, strong claims about specific cognitive deficits cannot be made on the basis of the current ex-Gaussian results. Although more limited in explanatory power, overall RT variability as assessed by ICV provides a clearer picture of group differences in the present study. Furthermore, cognitive interpretations of ex-Gaussian parameters have certain limitations of their own. The behavioral processes reflected in the group difference in sigma

in particular are not clear at present (Matzke & Wagenmakers, 2009). Tau has been associated with attentional lapses in some studies (Lee et al., 2012; McVay & Kane, 2012) but not others (Schmiedek, Oberauer, Wilhelm, Süss & Wittmann, 2007). Lee et al. (2012) provided supportive evidence in an experiment that jittered the interstimulus interval (ISI) in a GNG task. This manipulation eliminated the increase in tau shown by children with ADHD, compared to when the ISI was fixed (a more monotonous and boring condition). The use of thought-sampling procedures also suggested that attentional lapses contribute to tau (McVay & Kane, 2012). Mind wandering was measured during a GNG task by periodically asking subjects whether they were experiencing task-unrelated thoughts. These subjective attentional lapses were significantly correlated with tau, but not with sigma (McVay & Kane, 2012). The preponderance of evidence supports a role for tau in assessing attentional lapses, but the present results do not suggest a *specific* role for either sigma or tau in enhancing RT variability in the PTSD patients.

Total scores on the PCL-M correlated with the ICV measure of response variability, as did subscores for the individual symptom clusters and depressive symptoms on the BDI. Furthermore, ICV strongly predicted patient diagnosis even when controlling for PTSD symptom severity. However, increased response variability can be due to many different factors (e.g., attention difficulties and sleep deprivation) and is not restricted to trauma-related disturbances in cognition. In a similar manner, greater RT variability has been conceptualized at different levels, as reflective of cognitive (Tamm et al., 2012), affective (Ode et al., 2011), and neural dysfunction (Sonuga-Barke & Castellanos, 2007).

One neurally-based view of response variability is that spontaneous fluctuations in very low frequency activity recorded by resting state fMRI – i.e., the default mode network (DMN) in medial prefrontal, parietal, and precuneus/posterior cingulate regions – intrude into active task performance to cause unusually slow RTs (Sonuga-Barke & Castellanos, 2007). Under optimal conditions, attentional networks in frontal-parietal regions are activated during task performance, while the task-negative DMN is suppressed (Fox et al, 2005). Activity in the DMN has been related to mind-wandering and introspection (Gusnard, Akbudak, Shulman & Raichle, 2001; Sonuga-Barke & Castellanos, 2007). Therefore, periodic or cyclical interference from the DMN could disrupt performance on an attention task, resulting in more variable RTs (Sonuga-Barke & Castellanos, 2007). In the case of PTSD, alterations in the functional connectivity of the DMN and the salience detection network have been observed in fMRI studies (Daniels et al., 2010; Sripada et al., 2012). Examining ICV in relation to the DMN network in PTSD patients is an important next step for future research.

How can the mood-based and attention-based theories be integrated in the current study? There was a strong correlation between PTSD severity and RT variability, as mentioned previously. In addition, PTSD severity was predicted by self-rated attentional impulsiveness, which assesses “focusing on the task at hand” and “thought insertions and racing thoughts” (Patton et al., 1995). Here, we speculate that deficits in the sustained attention and top-down cognitive control processes that cause greater response variability might contribute to the maintenance of PTSD symptomology. In other words, the mind wandering or distractibility issues that cause more variable reaction times might also result in greater distress related to the trauma. This idea is consistent with prior work that has associated more frequent mind

wandering during cognitive tasks and daily activities with dysphoria and worry (Smallwood et al., 2007; McVay, Kane, & Kwapil, 2009).

The presence of one or more mTBIs (in addition to PTSD) did not affect response variability, a result we also obtained for false alarm errors in this population (Swick et al., 2012). However, other studies have demonstrated that TBI patients do show greater response variability (Stuss et al., 1994; Segalowitz et al., 1997; Dockree et al., 2006). An important difference between those studies and the present one is the severity of TBI, with the former studies primarily testing moderate to severe patients. In contrast, the participants in our study were likely at the mild end of mTBI, given the brief LOC (1–2 min) in most cases. Nonetheless, we cannot completely rule out the possibility that mTBI contributed to the deficits observed in the combined mTBI + PTSD group.

Stuss et al. (2003) have suggested that an alteration in the consistency of task performance could contribute to the difficulties that PFC patients experience in everyday life. Likewise, the combination of inconsistent performance and impaired response inhibition shown by the veterans with PTSD could have deleterious effects on daily activities requiring these cognitive control functions, such as driving (Lew, Amick, Kraft, Stein & Cifu, 2010) and multitasking (Honzel et al., submitted). Difficulties with sustained attention and executive control might also impact the effectiveness of therapies that rely on cognitive reappraisal and disengagement from traumatic stimuli (Vasterling & Verfaellie, 2009; Aupperle et al., 2012). Given the relationship between self-rated attentional impulsivity and PTSD symptoms, considering these problems in tandem may be therapeutically beneficial.

Conclusions

The present study demonstrated that combat veterans with PTSD showed more variable reaction times on Go trials, despite having raw RTs that were well-matched to controls in the GNG task. This increased level of variability was more pronounced in the difficult condition, suggesting that cognitive control processes became less stable over time, especially when responses were prepotent and routine. RT variability was strongly correlated with false alarm errors, replicating previous results in controls (Bellgrove et al., 2004; Simmonds et al., 2007) and other clinical populations (Stuss et al., 2003; Kaiser et al., 2008; Suskauer et al., 2008), and providing another indication of executive control dysfunction. Furthermore, ICV was significantly correlated with self-reported symptoms of PTSD and depression, but not with overall levels of impulsivity. However, the attentional subscale of the BIS-11 was correlated with both ICV and overall PCL-M scores. Attentional impulsiveness was a significant predictor of PTSD symptom severity, while motor and non-planning impulsiveness were not. Finally, the combination of mTBI and PTSD did not result in worse performance than PTSD alone, strengthening the association between clinically significant psychiatric symptoms and increased response variability. The distractibility issues that can lead to more variable RTs might also result in greater distress related to the trauma, because of the difficulties in suppressing these traumatic memories. Further, deficits in the sustained attention and top-down cognitive control processes that result in greater response variability might contribute to the maintenance of PTSD symptomology.

Acknowledgements

We are grateful to Dr. Andrew Kayser for patient referrals and to all participants for taking part in the study. We thank Dr. Catherine Fassbender for her suggestions on data analysis. We thank Dr. Machelle Wilson (University of California, Davis) and Timothy Heron (VA Northern California Health Care System) for statistical advice. This work was supported by the U.S. Army Medical Research and Materiel Command under W81XWH-08-2-0086 and a VA Merit Review grant from Clinical Science Research and Development. Dr. Wilson is supported by the National Center for Advancing Translational Sciences (NCATS), National Institutes of Health (NIH), through grant #UL1 TR000002. The contents reported within do not represent the views of the Department of Veterans Affairs or the United States Government.

References

Ashley V, Honzel N, Larsen J, Justus T, Swick D. Attentional bias for trauma-related words: Exaggerated emotional Stroop effect in Afghanistan and Iraq war veterans with PTSD. BMC: Psychiatry. 2013; 13:86. [PubMed: 23496805]

Aupperle RL, Melrose AJ, Stein MB, Paulus MP. Executive function and PTSD: Disengaging from trauma. Neuropharmacology. 2012; 62:686–594. [PubMed: 21349277]

Beck AT, Steer R, Gabin M. Psychometric properties of the BDI: Twenty-five years of evaluation. Clinical Psychological Review. 1988; 8:77–100.

Bellgrove MA, Hester R, Garavan H. The functional neuroanatomical correlates of response variability: evidence from a response inhibition task. Neuropsychologia. 2004; 42:1910–1916. [PubMed: 15381021]

Carlson KF, Kehle SM, Meis LA, Greer N, Macdonald R, Rutks I, …Wilt TJ. Prevalence, assessment, and treatment of mild traumatic brain injury and posttraumatic stress disorder: a systematic review of the evidence. Journal of Head Trauma Rehabilitation. 2011; 26:103–115. [PubMed: 20631631]

Chuah YM, Venkatraman V, Dinges DF, Chee MW. The neural basis of interindividual variability in inhibitory efficiency after sleep deprivation. Journal of Neuroscience. 2006; 26:7156–7162. [PubMed: 16822972]

Danielmeier C, Ullsperger M. Post-error adjustments. Frontiers in Psychology. 2011; 2:233. [PubMed: 21954390]

Daniels JK, McFarlane AC, Bluhm RL, Moores KA, Clark CR, Shaw ME, … Lanius RA. Switching between executive and default mode networks in posttraumatic stress disorder: alterations in functional connectivity. Journal of Psychiatry and Neuroscience. 2010; 35:258–266. [PubMed: 20569651]

Dockree PM, Bellgrove MA, O'Keeffe FM, Moloney P, Aimola L, Carton S, Robertson IH. Sustained attention in traumatic brain injury (TBI) and healthy controls: enhanced sensitivity with dual-task load. Experimental Brain Research. 2006; 168:218–229. [PubMed: 16044297]

Epstein JN, Langberg JM, Rosen PJ, Graham A, Narad ME, Antonini TN, … Altaye M. Evidence for higher reaction time variability for children with ADHD on a range of cognitive tasks including reward and event rate manipulations. Neuropsychology. 2011; 25:427–441. [PubMed: 21463041]

Fjell AM, Westlye LT, Amlien IK, Walhovd KB. Reduced white matter integrity is related to cognitive instability. Journal of Neuroscience. 2011; 31:18060–18072. [PubMed: 22159119]

Forbes D, Creamer M, Biddle D. The validity of the PTSD checklist as a measure of symptomatic change in combat-related PTSD. Behaviour Research and Therapy. 2001; 39:977–986. [PubMed: 11480838]

Fox MD, Snyder AZ, Vincent JL, Corbetta M, Van Essen DC, Raichle ME. The human brain is intrinsically organized into dynamic, anticorrelated functional networks. Proceedings of the National Academy of Sciences. 2005; 102:9673–9678.

Gusnard DA, Akbudak E, Shulman GL, Raichle ME. Medial prefrontal cortex and self-referential mental activity: relation to a default mode of brain function. Proceedings of the National Academy of Sciences. 2001; 98:4259–4264.

Heathcote A, Brown S, Cousineau D. QMPE: estimating Lognormal, Wald, and Weibull RT distributions with a parameter-dependent lower bound. Behavior Research Methods, Instruments, & Computers. 2004; 36:277–290.

Heathcote A, Popiel SJ, Mewhort DJK. Analysis of response time distributions: An example using the Stroop task. Psychological Bulletin. 1991; 109:340–347.

Hervey AS, Epstein JN, Curry JF, Tonev S, Arnold LE, Conners CK, Hinshaw SP, … Hechtman L. Reaction time distribution analysis of neuropsychological performance in an ADHD sample. Child Neuropsychology. 2006; 12:125–140. [PubMed: 16754533]

Hester R, Bell RP, Foxe JJ, Garavan H. The influence of monetary punishment on cognitive control in abstinent cocaine-users. Drug and Alcohol Dependence. 2013

Hester R, Foxe JJ, Molholm S, Shpaner M, Garavan H. Neural mechanisms involved in error processing: a comparison of errors made with and without awareness. Neuroimage. 2005; 27:602–608. [PubMed: 16024258]

Honzel N, Justus T, Swick D. Post-traumatic stress disorder is associated with limited executive resources in a working memory task. submitted.

Iversen AC, Fear NT, Ehlers A, Hacker Hughes J, Hull L, Earnshaw M, Greenberg N, … Hotopf M. Risk factors for post-traumatic stress disorder among UK Armed Forces personnel. Psychological Medicine. 2008; 38:511–522. [PubMed: 18226287]

Johnson KA, Kelly SP, Bellgrove MA, Barry E, Cox M, Gill M, Robertson IH. Response variability in attention deficit hyperactivity disorder: evidence for neuropsychological heterogeneity. Neuropsychologia. 2007; 45:630–638. [PubMed: 17157885]

Kaiser S, Roth A, Rentrop M, Friederich HC, Bender S, Weisbrod M. Intra-individual reaction time variability in schizophrenia, depression and borderline personality disorder. Brain and Cognition. 2008; 66:73–82. [PubMed: 17604894]

Kane MJ, McVay JC. What mind wandering reveals about executive-control abilities and failures. Current Directions in Psychological Science. 2012; 21:348–354.

Larson GE, Booth-Kewley S, Highfill-McRoy RM, Young SY. Prospective analysis of psychiatric risk factors in marines sent to war. Military Medicine. 2009; 174:737–744. [PubMed: 19685846]

Lee RW, Jacobson LA, Pritchard AE, Ryan MS, Yu Q, Denckla MB, Mahone EM. Jitter reduces response-time variability in ADHD: An ex-Gaussian analysis. Journal of Attention Disorders. 2012

Lew HL, Amick MM, Kraft M, Stein MB, Cifu DX. Potential driving issues in combat returnees. NeuroRehabilitation. 2010; 26:271–278. [PubMed: 20448316]

Matzke D, Wagenmakers EJ. Psychological interpretation of the ex-Gaussian and shifted Wald parameters: a diffusion model analysis. Psychonomic Bulletin & Review. 2009; 16:798–817. [PubMed: 19815782]

McNally RJ, Kaspi SP, Riemann BC, Zeitlin SB. Selective processing of threat cues in posttraumatic stress disorder. Journal of Abnormal Psychology. 1990; 99:398–402. [PubMed: 2266215]

McVay JC, Kane MJ, Kwapil TR. Tracking the train of thought from the laboratory into everyday life: An experience-sampling study of mind wandering across controlled and ecological contexts. Psychonomic Bulletin & Review. 2009; 16:857–863. [PubMed: 19815789]

McVay JC, Kane MJ. Drifting from slow to “d'oh!”: Working memory capacity and mind wandering predict extreme reaction times and executive control errors. Journal of Experimental Psychology: Learning, Memory, and Cognition. 2012; 38:525–549.

Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A, Wager TD. The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: a latent variable analysis. Cognitive Psychology. 2000; 41:49–100. [PubMed: 10945922]

Ode S, Robinson MD, Hanson DM. Cognitive-emotional dysfunction among noisy minds: predictions from individual differences in reaction time variability. Cognition and Emotion. 2011; 25:307–327. [PubMed: 21432673]

Patton JH, Stanford MS, Barratt ES. Factor structure of the Barratt impulsiveness scale. Journal of Clinical Psychology. 1995; 51:768–774. [PubMed: 8778124]

Pittman JO, Goldsmith AA, Lemmer JA, Kilmer MT, Baker DG. Post-traumatic stress disorder, depression, and health-related quality of life in OEF/OIF veterans. Quality of Life Research. 2012; 21:99–103. [PubMed: 21516356]

Polak AR, Witteveen AB, Reitsma JB, Olff M. The role of executive function in posttraumatic stress disorder: a systematic review. Journal of Affective Disorders. 2012; 141:11–21. [PubMed: 22310036]

Rabbitt PM. Errors and error correction in choice-response tasks. Journal of Experimental Psychology. 1966; 71:264–272. [PubMed: 5948188]

Segalowitz SJ, Dywan J, Unsal A. Attentional factors in response time variability after traumatic brain injury: an ERP study. Journal of the International Neuropsychological Society. 1997; 3:95–107. [PubMed: 9126851]

Schmiedek F, Oberauer K, Wilhelm O, Süss HM, Wittmann WW. Individual differences in components of reaction time distributions and their relations to working memory and intelligence. Journal of Experimental Psychology: General. 2007; 136:414–429. [PubMed: 17696691]

Simmonds DJ, Fotedar SG, Suskauer SJ, Pekar JJ, Denckla MB, Mostofsky SH. Functional brain correlates of response time variability in children. Neuropsychologia. 2007; 45:2147–2157. [PubMed: 17350054]

Simmons JP, Nelson LD, Simonsohn U. A 21 word solution. Dialogue: The Official Newsletter of the Society for Personality and Social Psychology. 2012; 26:4–7.

Smallwood J, Fitzgerald A, Miles LK, Phillips LH. Shifting moods, wandering minds: Negative moods lead the mind to wander. Emotion. 2009; 9:271–276. [PubMed: 19348539]

Smallwood J, O'Connor RC, Sudbery MV, Obonsawin M. Mind-wandering and dysphoria. Cognition and Emotion. 2007; 21:816–842.

Sonuga-Barke EJ, Castellanos FX. Spontaneous attentional fluctuations in impaired states and pathological conditions: a neurobiological hypothesis. Neuroscience and Biobehavioral Reviews. 2007; 31:977–986. [PubMed: 17445893]

Sripada RK, King AP, Welsh RC, Garfinkel SN, Wang X, Sripada CS, Liberzon I. Neural dysregulation in posttraumatic stress disorder: evidence for disrupted equilibrium between salience and default mode brain networks. Psychosomatic Medicine. 2012; 74:904–911. [PubMed: 23115342]

Stanford MS, Mathias CW, Dougherty DM, Lake SL, Anderson NE, Patton JH. Fifty years of the Barratt Impulsiveness Scale: An update and review. Personality and Individual Differences. 2009; 47:385–395.

Stuss DT, Murphy KJ, Binns MA, Alexander MP. Staying on the job: the frontal lobes control individual performance variability. Brain. 2003; 126:2363–2380. [PubMed: 12876148]

Stuss D, Pogue D, Buckle L, Bondar J. Characterization of stability of performance in patients with traumatic brain injury: Variability and consistency on reaction time tests. Neuropsychology. 1994; 8:316–324.

Stuss DT, Steinhem LL, Hugenholtz H, Picton T, Pivik J, Richard MT. Reaction time after head injury: Fatigue, divided and focused attention, and consistency of performance. Journal of Neurology, Neurosurgery, and Psychiatry. 1989; 52:742–748.

Suskauer SJ, Simmonds DJ, Caffo BS, Denckla MB, Pekar JJ, Mostofsky SH. fMRI of intrasubject variability in ADHD: anomalous premotor activity with prefrontal compensation. Journal of the American Academy of Child and Adolescent Psychiatry. 2008; 47:1141–1150. [PubMed: 18724253]

Swick D, Honzel N, Larsen J, Ashley V, Justus T. Impaired response inhibition in veterans with post-traumatic stress disorder and mild traumatic brain injury. Journal of the International Neuropsychological Society. 2012; 18:1–10. [PubMed: 22152338]

Tamm L, Narad ME, Antonini TN, O'Brien KM, Hawk LW Jr, Epstein JN. Reaction time variability in ADHD: a review. Neurotherapeutics. 2012; 9:500–508. [PubMed: 22930417]

Tarantino V, Cutini S, Mogentale C, Bisiacchi PS. Time-on-task in children with ADHD: an ex-Gaussian analysis. Journal of the International Neuropsychological Society. 2013; 19:820–828. [PubMed: 23777609]

Taylor BC, Hagel EM, Carlson KF, Cifu DX, Cutting A, Bidelspach DE, Sayer NA. Prevalence and costs of co-occurring traumatic brain injury with and without psychiatric disturbance and pain among Afghanistan and Iraq War Veteran VA users. Medical care. 2012; 50:342–346. [PubMed: 22228249]

The Management of Concussion/mTBI Working Group. VA/DOD clinical practice guideline for management of concussion/mild traumatic brain injury (mTBI). Journal of Rehabilitation Research Development. 2009; 46:CP1–CP68. [PubMed: 20108447]

van den Bosch RJ, Rombouts RP, van Asma MJ. What determines continuous performance task performance? Schizophrenia Bulletin. 1996; 22:643–651. [PubMed: 8938918]

Vasterling JJ, Brailey K, Constans JI, Sutker PB. Attention and memory dysfunction in posttraumatic stress disorder. Neuropsychology. 1998; 12:125–133. [PubMed: 9460740]

Vasterling JJ, Verfaellie M. Introduction-posttraumatic stress disorder: a neurocognitive perspective. Journal of the International Neuropsychological Society. 2009; 15:826–829. [PubMed: 19891816]

Vinogradov S, Poole JH, Willis-Shore J, Ober BA, Shenaut GK. Slower and more variable reaction times in schizophrenia: what do they signify? Schizophrenia Research. 1998; 32:183–910. [PubMed: 9720123]

Vrana SR, Roodman A, Beckham JC. Selective processing of trauma-relevant words in posttraumatic-stress-disorder. Journal of Anxiety Disorders. 1995; 9:515–530.

Weathers FW, Litz BT, Huska JA, Keane TM. PTSD Checklist - Military Version (PCL-M) for DSM-IV. Boston: National Center for PTSD - Behavioral Science Division. 1994

West R, Murphy KJ, Armilio ML, Craik FI, Stuss DT. Lapses of intention and performance variability reveal age-related increases in fluctuations of executive control. Brain and Cognition. 2002; 49:402–419. [PubMed: 12139961]

Whelan R. Effective analysis of reaction time data. The Psychological Record . 2008; 58:475–482.

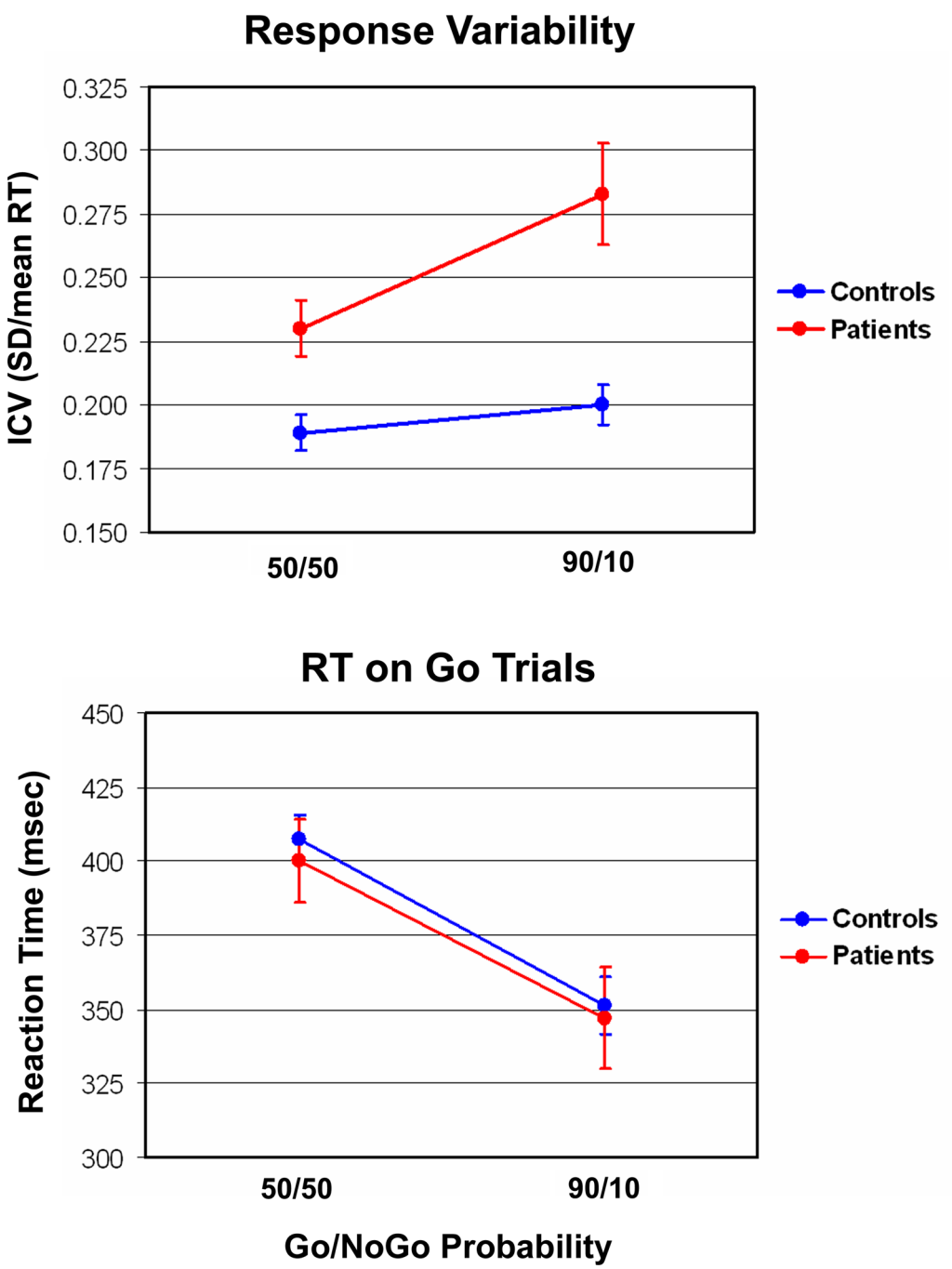

Figure 1.

Top: Response time variability (Standard Deviation of Go RT/mean RT) for the patients (n=45) and controls (n=34) in the easy (50/50) and difficult (90/10) conditions. The error bars depict standard errors. **Bottom:** Reaction times (RTs) on correct Go trials (in milliseconds in the easy (50/50) and difficult (90/10) conditions. The error bars depict standard errors.

Table 1

Demographic information and self-rating scores for controls, PTSD patients, and patient subgroups with and without mTBI.

	Controls (n=34)	Patients (n=45)	PTSD+mTBI (n=34)	PTSD only (n=11)
Age (yrs)	33.3 + 8.0	32.6 + 7.1 (n.s.)	32.5 + 7.1	32.8 + 7.3
Education (yrs)	14.7 + 1.6	13.5 + 1.6 (***)	13.4 + 1.7	13.6 + 1.1
Handedness	31 R, 3 L	38 R, 5 L, 2 ambi	27 R, 5 L, 2 ambi	11 R
Deployed (n)	20	45	34	11
Combat (n)	9	45	34	11
TBI events (n)	--	11 one, 23 > one	11 one, 23 > one	--
LOC	--	25; 5 dazed, 4 uncertain	25; 5 dazed, 4 uncertain	--
Years post	--	4.0 + 1.8	3.9 + 1.6	4.4 + 2.5
PCL-M	27.0 + 11.0	58.0 + 13.1 (***)	57.9 + 12.6	58.2 + 15.2
BDI	5.9 + 7.1	19.8 + 10.1 (***)	19.4 + 9.5	21.0 + 12.2
BIS-11A	56.7 + 9.4	70.5 + 10.9 (***)	71.0 + 10.5	68.9 + 12.4

Note: The mean + standard deviation are given for age, education, estimated years post-event(s), PCL-M, BDI, and BIS-11A. n.s. = not significantly different from controls;

*** significantly different from controls at p<.001;

R = right, L = left, ambi = ambidextrous; LOC = loss of consciousness (of 34 patients with mTBI, 25 had LOC, 5 did not, and 4 were not sure whether they had LOC); PCL-M = PTSD checklist, military version; BDI = Beck Depression Inventory; BIS-11A = Barratt Impulsiveness Scale. The BIS-11A was scored using a prorating method to make it more comparable to the BIS-11. The patient subgroups did not differ from each other for age, education, years post-event, PCL-M, BDI, and BIS-11A.

Table 2

The ex-Gaussian parameters for the easy (50/50) and difficult (90/10) conditions in controls and PTSD patients.

Condition	Controls Mean (SD)	Patients Mean (SD)
mu 50/50	344.9 (41.2)	320.0 (76.3)
mu 90/10	291.0 (47.0)	268.3 (106.2)
sigma 50/50	40.4 (13.9)	47.0 (25.2)
sigma 90/10	33.7 (12.6)	44.2 (28.2)
tau 50/50	62.5 (19.1)	76.9 (34.8)
tau 90/10	59.0 (24.7)	74.6 (52.9)

Note: The means (standard deviations) are in msec. The parameters are mu (μ = mean of normal distribution), sigma (σ = SD of normal distribution), and tau (τ = mean and SD of the exponential tail).

Table 3

Relationship of self-reported PCL-M scores to the three BIS-11 subscales.

Impulsiveness Type	B	Std. Error	β	*t*	*p*
Attentional	2.526	.315	.722	8.030	<.0001
Motor	.620	.451	.126	1.374	.173
Non-Planning	−.071	.354	−.020	−.200	.842

Note: PCL-M = PTSD checklist, military version. BIS = Barratt Impulsiveness Scale. R =.775; Adjusted R^2 = .585; F(3,75)=37.58, p<.0001.