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Attentional factors in response time variability after traumatic brain injury: An ERP study

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Abstract

Reaction time (RT) is often used in the assessment of patients with traumatic brain injury (TBI), presumably because it reflects either information processing speed or attentional capacity. To clarify this distinction, we examined behavioral RT and the within-subject variability of RT as they relate to electrophysiological measures of attention and information processing. These include the P300 latency, which reflects stimulus evaluation time, P300 amplitude, which reflects attentional allocation, and the preresponse component of the contingent negative variation (CNV), which reflects sustained attention. We found that the latency and variability in behavioral RT were not correlated with the latency or variability of the P300, suggesting that stimulus evaluation time is not a major contributor to RT and its variability in this paradigm. However, among normal controls, RT was related to P300 amplitude, and therefore to attentional allocation. For the TBI subjects, it was the variability, not the speed, of RT that was related to P300 amplitude and to the preresponse component of the CNV. These data suggest that, while in normal controls RT reflects attentional allocation, among TBI subjects it is the variability in RT that is sensitive to the ability to allocate and sustain attention. (*JINS*, 1997, *3*, 95–107.)

Keywords: TBI, Variability, RT, Attention

INTRODUCTION

Attentional difficulty has long been recognized as a major outcome of traumatic brain injury (TBI; Gronwall & Sampson, 1974; Gronwall, 1989), whether attention is considered a unitary process or is partitioned into several subtypes, such as focused, sustained, divided, and alternating (Sohlberg & Mateer, 1989). Response time is often though not always slowed in TBI (Van Zomeren, Brouwer, & Deelman, 1984; Stuss et al., 1989; but see Shum et al., 1990; Cremona-Meteyard et al., 1992). Presumably this is partly due to information processing difficulties that may be associated with attentional lapses, and due to response selection and motor initiation difficulty (Miller, 1970; Van Zomeren et al., 1984; Shum et al., 1990). Stuss et al. (1989) make a further observation that response time variability is considerably increased in TBI. They stress that "inconsistency and impaired focused attention are important deficits following head injury" (p. 747), and suggest that the inconsistency, which is clinically apparent (Stuss, 1995), is due to the reduced attentional capacity of TBI subjects. While this is a plausible hypothesis, there are alternatives. It could be that response time variability is primarily due to inconsistency in the time taken to process information, or to the impaired ability to select or recruit a motor response.

We examined these hypotheses in an ERP study of information processing, response time, and response time variability with moderate and severely traumatically brain injured subjects. Behavioral response times (RTs) were collected using a standard "oddball" paradigm in which subjects were required to make a simple stimulus discrimination (high *vs.* low tones) by pressing a button only when they heard the high tone, which occurred on 20% of the trials. We used ERPs (1) as a correlate of the time it takes subjects to evaluate whether the tone is a target (*P300 latency*), (2) to determine the amount of attention subjects allocate to the target tone (*P300 amplitude*), and (3) to determine the within-subject trial-to-trial variability in stimulus evaluation time (*within-subject variability of P300 latency*). We also examined the degree to which subjects maintained prestimulus attentional control using a standard contingent negative vari-

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ation (CNV) paradigm, where subjects were presented with a visual cue followed 2.3 s later by a visual target. We hoped that these electrophysiological measures would help us determine the relationship between perceptual processing speed, attentional control, and behavioral RT and its variability.

The Measures

Response time

Our measure of response time is similar to that used by Stuss et al. (1989): Participants are asked to press a key when a relatively rare target stimulus appears, and to withhold a response when the nontarget appears. We used a tone pitch discrimination since this is not vulnerable to being missed due to eye movements, and because ERP differences related to hearing are negligible in the loudness range used (Unsal & Segalowitz, 1995).

Response time variability

Since TBI subjects are expected to have slower response times, we also expect the within-subject standard deviation of the RTs to be greater as a result. For example, if RTs are slowed (increased) by 50%, the standard deviation is increased by 50%, since all the contributing RTs are 50% larger. If this increased variability is simply a function of increased RT, then there is little to be gained from a separate analysis of within-subject variability. One approach would be to control for RT differences by using each participant's coefficient of variation (*CV*)—the standard deviation divided by the mean—instead of the standard deviation (*SD*) as the index of within-subject variability (Segalowitz & Segalowitz, 1993). Another way to obtain variability uncontaminated by RT is to use multiple regression to partial out the absolute reaction time from the within-subject standard deviation as a first step in all analyses allowing us to examine the variance in standard deviation unconfounded by the variance due to RT (Cohen & Cohen, 1983). A third way is to perform the multiple regression partialling RT out of the standard deviation, saving the residual (SD_{RESID}), and using the saved residual as the dependent variable. We ran all analyses with each method and found the results to be virtually identical. Although it need not always be the case, in this sample the measures derived from the three methods were highly correlated, with *r*s ranging from .970 to 1.000. We present only the SD_{RESID} analyses because these are conceptually clearer: We are looking at response variability while controlling for overall response speed.

Event-related potentials (ERPs)

P300 ERP: P300 refers to an electrophysiological response that occurs usually between 300 and 500 ms after stimulus presentation. It is reflected in a positive-going potential that serves as an index of certain cognitive events. The P300 has two dimensions: amplitude and latency. Amplitude refers to the degree of deflection from baseline and is considered to represent the degree to which the nervous system orients to target or novel information (e.g., Ritter et al., 1968). Donchin and Coles (1988) prefer to describe the P300 amplitude as reflecting "context updating," or the revision of one's model of the environment. In either case, evidence is consistent with the view that the P300 amplitude is a manifestation of attentional allocation (e.g., Duncan-Johnson & Donchin, 1977; Isreal, Wickens, & Donchin, 1980; Kramer & Spinks, 1991) and the extent to which the stimulus is salient to the subject (Polich, 1986). ERP amplitudes to target and nontarget stimuli are very similar for the early components, but differ dramatically for later components such as the P300. The P300 peak is considerably greater in amplitude for rare target stimuli in an oddball paradigm (Picton & Hillyard, 1988).

P300 latency refers to the time between stimulus onset and the P300 peak. This interval reflects stimulus evaluation and categorization time (McCarthy & Donchin, 1981; Magliero et al., 1984) and has been associated with the time required for a subject to detect and evaluate the stimulus in a simple choice reaction time task (Kutas, McCarthy, & Donchin, 1977; Parasuraman, Richer, & Beatty, 1982). It is important to note that the P300 latency is independent of response selection and execution factors. This is easily demonstrated by making it more difficult for the subject to select the response by introducing stimulus-response incompatibility: RT increases but P300 latency does not change (McCarthy & Donchin, 1981; Magliero et al., 1984). For this reason, the P300 latency is especially useful in populations where response selection and execution may add variance to RT independently of variance in perceptual speed. Thus, the P300 latency reflects the speed of processing and/or evaluating perceptual information, independent of motor movement and response selection factors.

P300 has proven to be as reliable as other cognitive measures in test–retest paradigms (Segalowitz & Barnes, 1993; Fabiani et al., 1987).

Contingent negative variation (CNV): The CNV is also associated with attending and responding to simple stimuli. This is a slow negative potential that arises after a warning stimulus in anticipation of a second stimulus. For it to occur, the participant must be aware of a contingency between the first stimulus (S1) and the second stimulus (S2). S1 simply acts as a warning that the target will appear after a fixed period of time. The CNV will appear as long as the S1–S2 interval is of a duration that allows the participant to sustain focused attention and is not distracted from the task. Normally periods of 1 to 4 s are used, with an optimal period of about 2 s. The initial portion of the CNV, starting at about 600 ms after S1, has been associated with an orienting response to S1, and is referred to as the O-wave, while the latter portion is associated with the participant's expectancy to respond and is referred to as the E-wave (Gaillard, 1977; Tecce & Cattanach, 1982). When there is no expectancy to respond, as in no-go trials in a go–no-go paradigm,

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the E-wave is dramatically reduced (Campbell, Suffield, & Deacon, 1990). TBI subjects often show smaller E-waves in go trials (Rugg et al., 1989), though not always (Campbell et al., 1990). Although it has been shown that the development of the CNV is not necessarily dependent on the subject performing a motor response (Münte et al., 1984; Ruchkin, et al., 1986), the E-wave has been associated with response initiation in humans (Wei & Ding, 1990) and in monkeys (DiPellegrino & Wise, 1991) in some paradigms.

Neurogenesis of cognitive ERPs: Work over the past 20 years has established that the CNV and P300 are neuroelectric components with different cerebral origins that arise in response to different cognitive components (e.g., McCallum & Knott, 1976). While there is some controversy as to the precise electrogenesis of the P300 ERP, the majority of evidence points to bilateral generators in temporal and inferior parietal cortex (Okada, Kaufman, & Williamson, 1983; Lewine et al., 1989). Some researchers have demonstrated that excisions in the temporal–parietal cortex did not affect scalp distribution of the P300 in humans (Wood et al., 1982; Pineda, Foote, & Neville, 1987; Paller et al., 1988). However, recent evidence (Knight et al., 1989) has also indicated that the area of cortex in the temporal–parietal junction is crucial for the scalp recorded P300. Similarly, Smith et al. (1990), using depth recordings, have shown that activity in the lateral neocortex of the inferior parietal lobule was the largest contributor to the scalp-related P300, although smaller contributions were made from other areas including medial to lateral frontal and temporal areas.

There is more consensus on the probable origins of the CNV. The CNV paradigm is highly similar to the delayed response paradigms used to study functions of the prefrontal and frontal cortex in primates. Both lesion and electrophysiological studies using this paradigm have linked the maintenance of attention during the S1–S2 delay period to activity in the dorsolateral prefrontal lobe area (Fuster, 1987; Goldman-Rakic, 1987). While Fuster (1987) argues that the initial orientation and the maintenance of attention during the interval leading up to the second stimulus are based on activities of different cells in the dorsolateral prefrontal region, DiPellegrino and Wise (1991) report single cell responses to the initial orienting to S1 in the dorsolateral prefrontal area while the expectancy to respond motorically is associated with cell activity in the adjacent premotor region. Thus, we have physiological evidence to associate the O-wave with initial orientation to the attentional task and the E-wave with the continued anticipation of an expected target, and to link these to generators somewhere in the prefrontal region.

To review, in this study we gathered behavioral response times in an oddball choice response time task using simple tone discrimination. We investigated the relationships between average reaction time, trial-to-trial reaction time variability, and electrophysiological indices of attention and speed of information processing. These latter included (1) the latency of the endogenous neural response to target stimuli (P300 latency), (2) the amplitude of neural response that occurs with target recognition (P300 amplitude), and (3) a general trait measure of pretarget attentional control (CNV). If TBI leads to longer RT because of increased stimulus evaluation time, then we should find a correlation between RT and P300 latency. If TBI leads to increased *variability* of RT because of increases in the variability in the speed of stimulus evaluation, then the variability of RT should correlate with the variability of P300 latencies. If, on the other hand, increases in RT and RT variability are primarily due to decreases in attentional control, then these should be accounted for by the indices of attentional allocation (P300 amplitude) and of attentional maintenance (CNV). Examining these parameters within a single experimental paradigm would allow us to assess the relationships among attentional control, speed of information processing, and response time and its variability.

METHOD

Research Participants

Twenty moderate-to-severe TBI patients and 22 controls participated in a standard auditory oddball detection task and a visual CNV task. Their ages ranged from 18 to 49 years ($M = 31.9$), with the control group matched for average age (20–46 years, $M = 32.5$) and sex except that the 2 extra participants in the control group were women. All TBI participants were at least 1-year posttrauma (range 1–12, median 7 years with $SD = 3.6$) and 4 had a history of previous head injury.

Documentation of brain damage when available was obtained from hospital records. Coma duration was greater than 1 month for 7 of the patients, 1 week to 1 month for 8 patients, 1 hr to 1 week for 2 patients, and less than 1 hr for 3 patients. CT scans and angiogram documentation, available for only 15 of the patients, showed an even left–right split in localized damage (4 unilateral left, 4 unilateral right, 7 bilateral), anterior-only damage in 6 cases, posterior-only in 4 cases, and both anterior and posterior in 5 cases. The other 5 cases had no records of brain imaging data. All TBI participants were recruited through a local head injury association. The controls were recruited through the union of a local large manufacturing plant.

Procedure

P300s

An auditory oddball task presented 40 target tones of 1000 Hz and 181 nontarget tones of 1500 Hz in a randomly mixed series 1.3 s apart (tones were square wave with 110 ms duration and 60 db as produced by an MS-DOS computer). ERPs were collected with gold electrodes referenced to linked ears from Fpz, Fz, Cz, and Pz using the international 10–20 system (Jasper, 1958) with a mastoid ground and a supraorbital ridge eye monitor. Subjects were

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asked to press the space bar on a microcomputer as quickly as possible when the target tone was presented. Impedances were kept below 5 kΩ and the signal had a bandpass of .5 to 30 Hz.

CNVs

The CNV task was a cued response paradigm: The warning stimulus was a square appearing on a computer screen for 500 ms followed by a cross 2300 ms after the onset of the warning stimulus. The participants were asked to press the space bar on the microcomputer as quickly as possible when the second stimulus appeared, and were told that the first stimulus should be considered a cue for the second. They were explicitly asked to anticipate the appearance of the second stimulus but not to initiate pressing the space bar until it actually appeared. Bandpass was .01 to 30 Hz.

Choice reaction time

The behavioral reaction times were taken to the target tones of the auditory oddball task.

Scoring the ERPs

As is standard practice, the P300 analyses involved only the Cz and Pz sites and the CNV results involved the Cz site only (Tecce, 1972). Digitizing was performed by a TecMar LabMaster 12-bit A–D converter at 2.5 ms/point for 750 ms for the P300s, and at 10 ms/point for the CNVs. The auditory ERPs were then low-pass filtered for scoring with a double-pass moving window filter, −3 db at 5.6 Hz. P300 latencies were determined by a computer-assisted peak-picking routine selecting the point between 250 and 500 ms with the highest positive amplitude. The 50 ms preceding the stimulus served as baseline. Trials were rejected for movement artifact on the basis of amplitude saturation on any channel or greater amplitude change on anterior (Fpz and Fz) and eye channels than on the posterior channels. P300s were scored on a trial-by-trial basis, with the mean representing the average of the single trial scores. The CNVs were calculated by computer from the raw digitized data, based on the interval from 600 ms post-S1 to the onset of S2. This interval was divided into 5 340-ms epochs. Average amplitude was calculated for these 5 epochs with the 200 ms preceding S1 serving as baseline. Note that for ease of discussion, voltages are inverted so that positive values indicate the presence of a CNV. That is, a negative deflection is indicted by positive numbers reflecting greater amplitude of the CNV.

RESULTS

Speed and Variability of Response

Means and standard deviations of the behavioral and electrophysiological indices of response speed and attention for each group are presented in Table 1.

Behavioral response

The first question addressed was whether TBI patients could be distinguished from controls on the basis of behavioral reaction time or its variability. Reaction times greater than 1000 ms were not recorded, but were treated as missed trials, and the number of missed trials was analyzed separately. The RT analyses were done on both mean and median reaction times, with the same results. In the interests of brevity, only means will be presented. The TBI group had longer behavioral reaction times to the target tones compared to the controls (460 ms *vs*. 335 ms, $t(40) = 6.02$, $p < .001$) and greater standard deviations for the reaction times (101 *vs*. 75, $t(40) = 2.54$, $p < .02$). Reaction time means correlated significantly with standard deviations for the aggregate of TBI and controls ($r = .59$, $p < .001$), and within each group ($r = .61$, $p < .005$ and $r = .45$, $p < .05$ for the control and TBI groups respectively). Consequently, in all subsequent analyses we consider only the residual variance in standard deviation after variance due to RT is partialled out. Thus, SD_{RESID} refers to the residual variability in participants' response time unconfounded by their average speed of response.[1]

One individual in the TBI group had a SD_{RESID} more than 3 standard deviations above the mean. When his score was removed, his SD_{RESID} was almost 4 standard deviations from the recalculated mean, while the others fell within 2.1 standard deviations in a near-normal distribution. His mean RT was well within range for the group. Therefore, we have excluded his scores only from analyses involving SD_{RESID}.

Missed trials

Of the 42 participants, 19 missed no targets, 11 missed only one, and 12 (10 of whom were TBI subjects) missed more than one. One subject in the control group successfully completed 20 trials, after which the RT equipment malfunctioned and this subject was excluded for the analyses on missed trials. Group comparisons indicated that the TBI subjects ($M = 3.05$) missed more targets than the controls [$M = .476$; $t(39) = 2.40$, $p < .025$]. Two thirds of the 20 TBI subjects were responsible for these errors, compared to one third of the 21 control subjects ($\chi^2 = 7.2$, $p < .01$). Only 2 of the control subjects missed more than one target compared to 10 of the TBI subjects ($\chi^2 = 6.6$, $p < .01$). Within the TBI group, the number of misses did not corre-

[1]Once we controlled for overall latency of response by using SD_{RESID} (or through the use of the coefficient of variation as in Segalowitz et al., 1992c), we found that intrasubject variability in behavioral response no longer discriminated the groups. Even so, it was the variability rather than the latency of behavioral response that correlated with electrophysiological measures of attentional control. We cannot assume, however, that the lack of group differences in partialled variability implies that variability in behavioral RT is not important in understanding changes in information processing abilities induced by TBI. It appears that variability has different implications when it occurs in the context of fast versus slow or impaired responses.

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Table 1. Means, standard deviations, minima and maxima of the behavioral and electrophysiological indices of response speed and attention for each group

	TBI group			
Variable	*M*	*SD*	Minimum	Maximum
RT Mean	460.21	85.88	351.05	652.13
RT *SD*	100.99	40.46	56.83	201.24
$P300_{CZ}$ amplitude	6.00	3.65	−.96	11.24
$P300_{PZ}$ amplitude	6.09	3.45	.79	15.17
$P300_{CZ}$ latency	333.20	31.31	273.00	384.00
$P300_{PZ}$ latency	333.76	30.62	278.13	389.24
CNV Epoch 1	−.41	5.66	−12.08	8.92
CNV Epoch 2	1.98	5.22	−6.60	11.05
CNV Epoch 3	3.87	5.64	−4.01	17.24
CNV Epoch 4	4.29	5.75	−3.41	16.51
CNV Epoch 5	4.21	5.75	−3.84	16.27
E-wave	4.13	5.46	−3.75	15.67
	Control group			
Variable	*M*	*SD*	Minimum	Maximum
RT Mean	335.05	44.32	270.12	454.60
RT *SD*	75.32	23.50	43.41	143.66
$P300_{CZ}$ amplitude	15.03	7.06	1.33	28.86
$P300_{PZ}$ amplitude	13.58	5.02	2.92	22.79
$P300_{CZ}$ latency	303.69	16.65	268.00	332.00
$P300_{PZ}$ latency	305.30	13.82	283.41	330.92
CNV Epoch 1	2.98	3.21	−3.10	9.15
CNV Epoch 2	4.85	3.84	−1.63	16.54
CNV Epoch 3	5.56	6.19	−1.77	27.29
CNV Epoch 4	6.00	6.70	−.92	30.72
CNV Epoch 5	6.38	7.76	−.84	35.05
E-wave	5.98	6.77	−1.18	31.02

E-wave is the average of the latter 3 epochs of the CNV.

late significantly with the reaction time ($r = .38$, n.s.), but they did correlate with the SD_{RESID} ($r = .55$, $p < .02$).

P300 latencies

P300 latency was longer for the TBI group for both Pz and Cz sites [Pz: 334 *vs.* 305 ms, $t(40) = 3.94$, $p < .001$; Cz: 333 *vs.* 304, $t(40) = 3.86$, $p < .001$]. Similarly, the standard deviations of the P300 latencies were greater for the TBI group [Pz: $t(40) = 3.44$, $p < .001$; Cz: $t(40) = 3.93$, $p < .001$]. For the P300s, the standard deviations did not correlate with latencies, and so the use of residualized standard deviations was not appropriate.

The relation between behavioral response and P300 latency.

Since the groups differed on both P300 latency measures and on reaction time, separate correlations were computed for these two variables in each group in order to avoid the correlation simply reflecting the group differences. For neither group was P300 latency or its standard deviation related to the behavioral response time, RT, or to its variability, SD_{RESID}, (*r*s ranged from −.35 to .09, where .44 is needed for significance at $\alpha = .05$). It would appear that the P300 latency and its variability account for a relatively small portion of variance in the speed and variability of behavioral RT.

Indices of Attention

Our next question was whether measures of attentional allocation (as measured by the amplitude of the target-related P300 ERPs) or attentional control (as measured by the anticipatory negativity of the CNV) would distinguish TBI patients from controls.

P300 amplitude effects

The control group (CzMean = 15.0 μV; PzMean = 13.6 μV) had a significantly greater P300 amplitude than the TBI group [CzMean = 6.0 μV; PzMean = 6.1 μV; for Cz, $t(39) = 4.92$, $p < .001$ and for Pz, $t(39) = 5.31$, $p < .001$]. Examples of the auditory ERPs of 3 TBI and 3 con-

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trol participants chosen at random are illustrated in Figure 1. Each tracing represents a single scorable, correct target trial.

CNVs

As indicated earlier, the CNV was measured from 600 ms after the onset of the warning stimulus up until the onset of the response stimulus. This 1700-ms period was divided into 5 equal epochs of 340 ms. The groups differed significantly on Epochs 1 and 2 only [$t(40) = 2.42$ and 2.04, $p < .02$ and $p < .05$] and not on the later portion of the CNV. Averaged examples of CNVs are depicted in Figure 2. These represent CNVs from the same participants whose P300s are depicted in Figure 1. Unlike those of Rugg et al. (1989), our TBI patients did not differ from controls in the later portion of the CNV. This may be due to Rugg et al. having elicited CNV with a go–no-go task, which would have increased attentional load, while we used the standard CNV paradigm in which every cue was followed by a target.

The relationship between indices of attention

There was a spurious correlation between the initial portion of the CNV and the P300 amplitude due to the main effects of group ($r = .32$ and $r = .34$ for Cz and Pz sites, respectively, $p < .05$). This correlation was not present in either group (control group: $r = .16$ and .03; TBI group: $r = .12$ and .27, n.s., for the Cz and Pz sites, respectively). On the other hand, the later portion of the CNV, the E-wave, taken as the average of the last three portions of the CNV (the last 1020 ms before the imperative stimulus), correlated significantly with the P300 amplitude for the sample as a whole ($r = .46$, $p = .025$, and $r = .74$, $p < .001$ for Cz and Pz sites, respectively) and within each group (control group: $r = .49$, $p < .05$, and $r = .40$, $p < .07$ for Cz and Pz sites; TBI group: $r = .46$, $p < .05$ and $r = .74$, $p < .001$ for Cz and Pz sites, respectively) indicating considerable overlap between these two electrophysiological indices of attention.

Relationship Between RT and Indices of Attention

RT and P300 amplitude

As was the case for RT and P300 latency, the groups differed on both P300 amplitude and on RT; therefore the relationship between these two variables was examined separately for each group. For the control group, P300 amplitude correlated with RT ($r = -.61$, $p < .005$; $r = -.55$, $p < .01$, for Cz and Pz respectively). Higher amplitude was associated with shorter behavioral response times. In the TBI group there was no such correlation ($r = -.05$ and $r = -.04$ for Cz and Pz respectively). The difference between these correlations (control group *vs*. TBI group) did not quite reach a level of statistical significance ($Z = 1.9$, $p < .07$ and $Z = 1.7$, $p < .10$). Similar correlational analyses were done using SD_{RESID}. In the control group, the relationship between SD_{RESID} and P300 amplitude was not significant ($r = .11$, n.s.; $r = .06$, n.s., for Cz and Pz respectively), while for the TBI group the relationship between SD_{RESID} and P300 am-

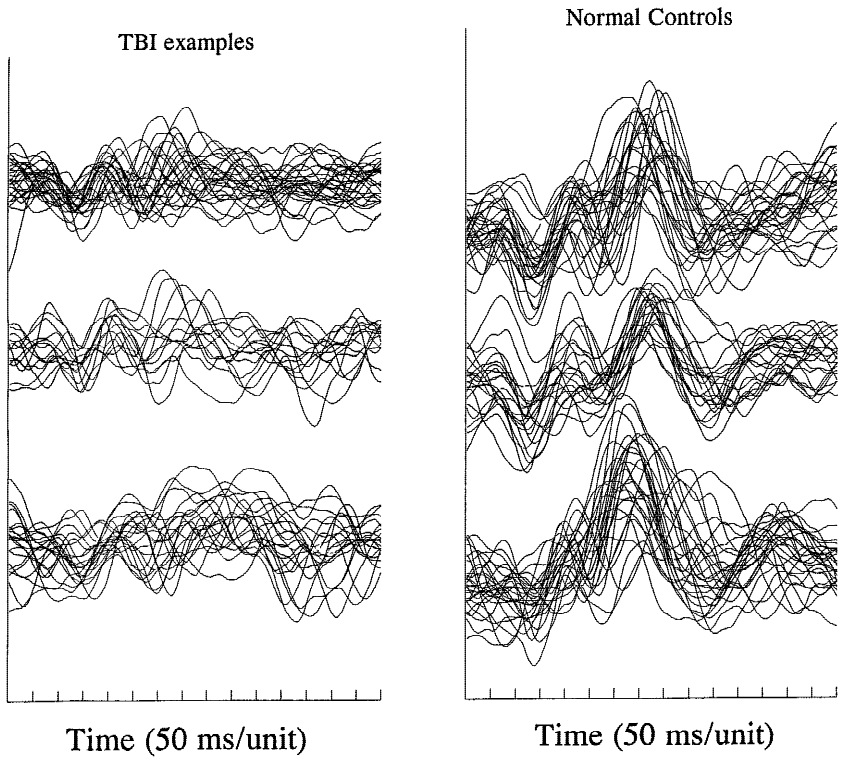

Fig. 1. Examples of target trial tracings for 3 control and 3 TBI participants taken at random. These trials were responded to correctly and were considered scorable. Waveforms are adjusted to a 50-ms prestimulus baseline period. Positive is up.

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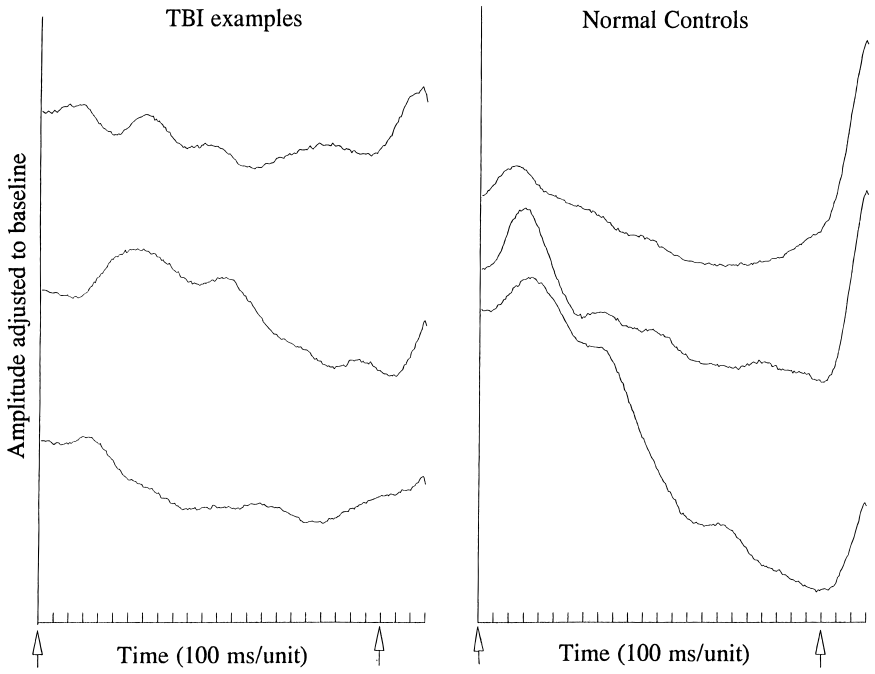

Fig. 2. The averaged CNVs for the same subjects as in Fig. 1. The amplitudes have been vertically adjusted to the prestimulus baseline of 200 ms. Note the P300 response to the first and second stimuli, the timing of which are indicated by the arrows on the horizontal axis. Positive is up.

plitude was very strong ($r = -.85$, $p < .0001$; $r = -.73$, $p < .001$, for Cz and Pz respectively). Higher P300 amplitude was associated with less variability (see Figure 3). The difference between these correlations obtained in each group was significant ($Z = 3.3$, $p < .005$; $Z = 2.5$, $p < .02$, for Cz and Pz respectively).

RT and CNV

Zero-order correlations indicated that CNV was not predictive of behavioral RT in either group. Correlations of RT with CNV Epochs 1 to 5 ranged from $-.09$ to $-.28$, all nonsignificant in the control group and from $-.09$ to .25, all nonsignificant in the TBI group. For the control group the same pattern emerged when examining the relationship between SD_{RESID} and CNV, ranging from $-.14$ to .23, all nonsignificant. However, for the TBI group, CNV was predictive of the SD_{RESID} with the relationship being most striking for the later epochs, that is, those epochs that immediately precede the target in the CNV attentional task ($r = -.14$, $-.48$, $-.50$, $-.57$, $-.60$ for Epochs 1 through 5 respectively). The correlation with Epoch 1 did not reach significance, but the other correlations were significant, all *p*s less than .02.

In order to test the interaction between these correlations and group, we tested the data for a Group × E-Wave interaction using a regression model. With SD_{RESID} as the criterion variable, we entered group on the first step [$F(1,39) = .8$, n.s.], then E-wave [$F(1,38) = 4.7$, $p < .05$], followed by the interaction of Group × E-Wave [$F(1,37) = 5.9$, $p < .025$]. This analysis confirms that the relationship between the E-wave and the SD_{RESID} differed significantly between the groups (Cohen & Cohen, 1983). The relationship between SD_{RESID} and the E-wave among the TBI participants is plotted in Figure 4.

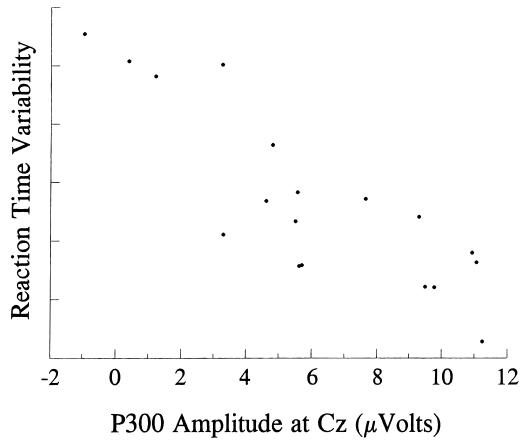

Fig. 3. The relationship between the reaction time variability, as indexed by the coefficient of variation, and the P300 amplitude taken at Cz within the TBI group of participants.

RT variability as predicted jointly by P300 amplitude and CNV

As indicated above, the SD_{RESID} could be predicted in the TBI group by both P300 amplitude and CNV. The next step was to determine the relative contribution of these two indices of attention in predicting TBI participants' response variability. Partial correlations from regression analyses indicated that P300 amplitude at Cz and E-wave shared 30% of the variance in SD_{RESID}, $P300_{CZ}$ accounted for an additional 42% unique variance in SD_{RESID} [$F(1,15) = 26.5$,

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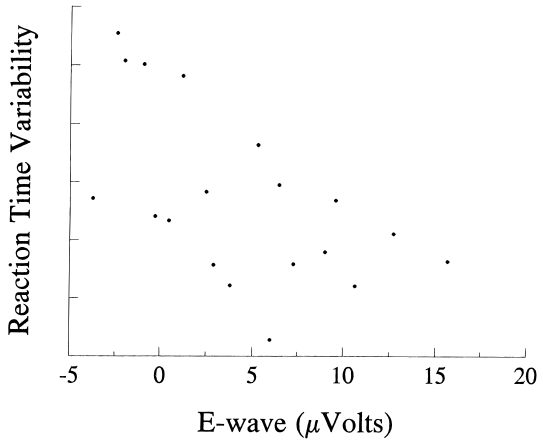

Fig. 4. The relationship between the reaction time variability, as indexed by the residualized standard deviation of the reaction time with the mean reaction time for the participant partialled out (SD_{RESID}), and the E-wave of the CNV event-related potential within the TBI group of participants. Note that the polarity of the E-wave has been reversed, so that the more positive values indicate the larger E-wave amplitude. Units represent the number of microvolts per sampling point.

Table 2. Unique and shared variance in RT variability accounted for by E-wave and by P300 amplitude at Cz and Pz in the TBI group

Predictor	Percent of variance in SD_{RESID} accounted for	F	$d.f.$	p
E-wave (unique)	4.8	3.1	1,15	=.10
$P300_{CZ}$ amplitude (unique)	41.8	26.5	1,15	<.0001
Interaction	7.0	5.9	1,14	<.03
Shared	29.8			
Total	83.4	23.5	3,14	<.0001
E-wave (unique)	0.5	0.2	1,15	n.s.
$P300_{PZ}$ amplitude (unique)	18.4	5.9	1,15	<.03
Interaction	22.2	12.6	1,14	<.005
Shared	34.1			
Total	75.3	14.2	3,14	<.0005

These figures reflect analyses omitting 1 subject whose RT was near the mean of the TBI group but whose SD_{RESID} was outlying more than 2.5 standard deviations.

$p < .0001$], E-wave accounted for 5% unique variance [$F(1,15) = 3.1, p = .10$], and their interaction an additional 7% [$F(1,14) = 5.9, p < .03$]. Thus, these ERP measures together account for 83% of the variance in the variability of RT (see Table 2). Similar analyses for P300 at Pz indicate that 75% of the variance in SD_{RESID} is accounted for (see Table 2). For both Cz and Pz analyses, the variance that the interaction captured was due to quadratic components in the prediction of SD_{RESID}. A purely linear model overestimates the change in SD_{RESID} at the extreme ends of the P300 amplitude and E-wave distribution.

Relations with missed trials on the auditory oddball task

Since the auditory oddball task requires the participant to decide to respond, we can consider the number of errors to be a measure of inattention or extreme slowness (past the allotted time for valid responses). In order to examine this latter possibility, we calculated from each participant's reaction times a confidence interval that would include 99% of responses. For only 1 participant did that range include the 1000-ms time limit. This individual (in the TBI group) had missed 21 of the 40 possible trials. We excluded this subject from all parametric analyses of missed trials.

The number of misses correlated significantly in the TBI group with the later epochs of the CNV [correlations with Epochs 1–5 are −.12 (n.s.), −.12 (n.s.), −.40 ($p < .10$), −.54 ($p < .02$), and −.55 ($p < .02$)], but not in the control group (all rs < .25, n.s.). If we inspect a scatterplot of the number of misses by the CNV E-wave, we see that the significant linear relationship is masked somewhat by a triangular distribution. It appears that while those who miss more targets are likely to have a smaller E-wave, those missing few targets may or may not have a larger E-wave (see Figure 5). Nevertheless, the E-wave accounted for 27% of the variance in missed trials [$r = .52, F(1,17) = 6.3, p < .025$]. The triangular distribution implies that at least one other factor is interacting with the relationship between the E-wave

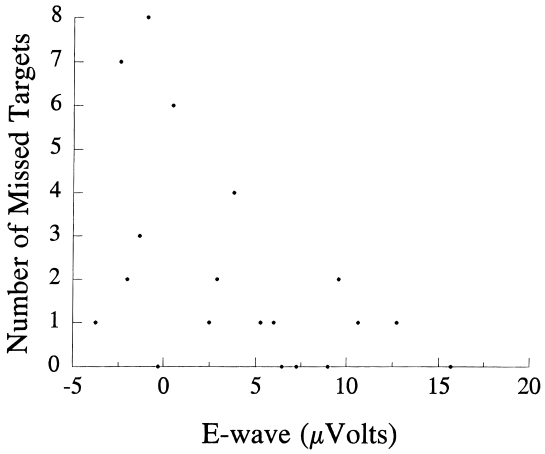

Fig. 5. The relationship between the number of targets missed by the participant and the E-wave of the CNV event-related potential (polarity reversed) within the TBI group of participants. One individual was considered an outlier because of having missed 21 of 40 trials and has been omitted from all analyses involving the number of missed targets. Units represent the number of microvolts per sampling point.

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and the number of missed targets (Woody & Balthazard, 1985).

The P300 amplitude was also related to the number of missed targets ($r = -.54$, $p < .03$, and $r = -.58$, $p < .02$, for Cz and Pz respectively). Since the least number of targets that can be missed is zero, at some point additional P300 amplitude cannot bring down the number of misses, an effect clearly present at the Pz site. In order to capture this factor statistically, we included the quadratic component ($P300_{Pz}$ amplitude squared), with which we are able to account for an additional 23% of the variance [$F(1,15) = 8.27$, $p < .02$], bringing the total to 57% of the variance in missed targets ($R = .76$, $p < .002$; see Figure 6).

We also used regression analyses to separate unique from shared variance in the number of missed trials as predicted by the P300 amplitude and E-wave (see Table 3), and we included the quadratic for the $P300_{Pz}$ amplitude since it accounted for significant variance at the Pz site. $P300_{Cz}$ amplitude and E-wave accounted for 12% and 10% unique variance in the number of misses, and they shared another 17% of the variance in number of misses. The P300 at the Pz site was a more powerful predictor of misses: The $P300_{Pz}$ amplitude accounted uniquely for 31% of the variance in misses, and it shared 26% additional variance in misses with E-wave; the E-wave itself contributed no unique variance when P300 amplitude was measured at the Pz site.

Table 3. Unique and shared variance in number of missed targets accounted for by E-wave and by P300 amplitude at Cz and Pz in the TBI group

Predictor	Percent of variance in number of missed targets accounted for	*F*	*d.f.*	*p*
E-wave (unique)	9.5	2.3	1,15	n.s.
$P300_{CZ}$ amplitude (unique)	11.8	2.8	1,15	n.s.
Shared	17.0			
Total	38.3	4.7	2,15	<.05
E-wave (unique)	0.1	1.57	1,15	n.s.
$P300_{PZ}$ amplitude[1] (unique)	31.0	1.93	2,14	<.025
Shared	16.5			
Total	57.6	6.68	3,14	<.01

[1]Includes the quadratic component ($P300_{PZ}$ squared).

DISCUSSION

RT is widely used as an index of information processing speed, yet we found no relationship between RT or its variability and the latency or the standard deviation of information processing speed as indexed by the P300 ERP for either the TBI or control participants. It may be that a considerable portion of the variance in behavioral RT includes variance due to individual differences in response selection, motor planning and response execution, that is, in knowing what to do with information after it has been registered. Consistent with this, behavioral RT was a reliable index of the allocation of attention as measured by P300 amplitude, but only among controls. This relationship was not observed in the TBI group, suggesting that RT serves as an index of attentional allocation only within the normal range of response times.

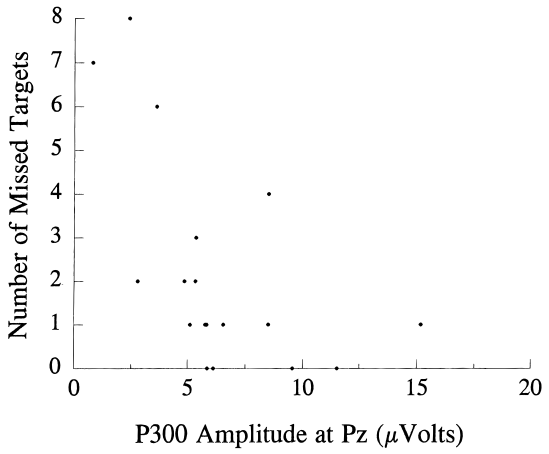

Fig. 6. The relationship between the number of targets missed by the participant and the P300 amplitude at Pz. One individual was considered an outlier because of having missed 21 of 40 trials and has been omitted from all analyses involving the number of missed targets.

Among the TBI subjects, we found that the variability of behavioral response time (not the speed) was associated with electrophysiological indicators of attentional allocation and attentional control. Combining CNV E-wave and P300 amplitude, we were able to predict up to 83% of the variance in RT variability that remained after partialling out variance due to response speed. These data suggest that both pretarget attentional control (E-wave) as well as attentional allocation (P300 amplitude) are related to the variability but not to the latency of behavioral RT in a TBI population. There is considerable overlap between these electrophysiological measures among our participants in their ability to predict RT variability. Unfortunately, this does not permit us to determine which aspect of attentional control is primarily responsible for predicting behavioral response variability, that is, the prestimulus anticipation of response as indicated by E-wave or the attention allocated to the stimulus once it has occurred as indicated by the amplitude of the P300 ERP.

We examined missed targets using separate analyses and found that among the TBI subjects, we could predict from 38 to 58% of the variance on the basis of the CNV E-wave and P300 amplitude. Thus, whether we examine the variability of response or the number of missed targets on a simple tone discrimination task, we are able to account for

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sizable proportions of variance on the basis of electrophysiological measures of attention. These data suggest that differences in response variability result primarily from the processes that are reflected in P300 amplitude and CNV E-wave.

Theoretical Perspectives on RT, Processing Speed, and Attention

The measurement of electrical brain activity serves as a noninvasive index of neural function. Event-related potentials are of particular interest because they occur in response to specific stimuli or events. The P300 component that we use is thought to be generated posteriorly, both because of lesion studies (Knight, 1990) and scalp topography (Johnson, 1993). However, attention is a complex process requiring the integration of many brain areas. It may be that some prefrontal regions are important in regulating the amount of attention allocated, and therefore the amplitude of the P300, while not influencing its latency (Dywan, Segalowitz, & Unsal, 1992; Segalowitz, Unsal, & Dywan, 1992a), just as the latency of the P300 can be manipulated without altering its amplitude (Segalowitz, Velikonja, & Storrie-Baker, 1994).

CNV

While not closely tied to specific stimulus characteristics, CNV is influenced by the contingent relationship between two stimuli. CNV amplitude may be manipulated by changing the significance of the second stimulus through degrading the contingent relationship between S1 and S2 (Walter et al., 1964) or through distracting the attention of the subjects by requiring concurrent performance on an additional task (e.g., Tecce, Savignano-Bowman, & Meinbresse, 1976).

This negativity in CNV is thought to be generated by activity in the prefrontal region and the prefrontal–thalamic network (Tsubokawa & Moriyasu, 1978; Groll-Knapp et al., 1980; Fuster, 1987; DiPellegrino & Wise, 1991). Topographic scalp recordings have also demonstrated that, in humans, the CNV first appears at the frontal pole, reaching maximum amplitude at central and parietal regions (Yamamoto, Saito, & Endo, 1986) and source localization places the generator of the entire CNV in the prefrontal tissue (Basile et al., 1994).

According to Stamm (1987), the role of this frontal activation involves programming for the self-regulation of future actions and the inhibition of interfering responses. Sandrew, Stamm, & Rosen, (1977) found when they introduced delayed response training trials to monkeys during spontaneous, endogenous periods of frontal negativity (as indicated by electrodes implanted in prefrontal cortex), they learned the task at 5.8 times the normal rate. Similarly with human subjects, Bauer and Nirnberger (1981) used slow potential shifts automatically detected by a computer to trigger the presentation of stimuli in a concept formation task. They found that young adults were able to learn a concept significantly faster when the stimulus was preceded by a negative potential shift than when it was preceded by a positive shift. This is consistent with our findings that the early component of the CNV is correlated specifically with performance on psychometric measures that require active monitoring of rule-governed relationships during task performance (Segalowitz et al., 1992a, 1992b).

We readily acknowledge that there is much to be learned about the CNV. Nonetheless, the relationship between pretarget negativity and SD_{RESID} in TBI subjects would suggest that frontally based processes are influential in controlling the variability of behavioral RT, and accords with the view that the ability to maintain attention in anticipation of a target is significant to our understanding of TBI-related decrements in the efficient processing of information (Rugg et al., 1989; Cremona-Meteyard et al., 1992).

P300

The amplitude of the P300 is generally considered to be a manifestation of attentional allocation (Isreal et al., 1980; Kramer & Spinks, 1991), and reflects the updating of one's model of the environment (e.g., Donchin & Coles, 1988) and the salience of the stimulus to the subject (Polich, 1986; Segalowitz et al., 1994). We have demonstrated that the P300 amplitude correlated significantly with the latter portion of the CNV as a subject trait and these together predicted a substantial proportion of variance in RT variability among our TBI subjects. However, from these data we cannot determine whether the pretarget anticipatory attention is causally related to the amplitude of the P300, that is, whether being able to maintain vigilance in the anticipation of a target increases the amount of attention one is able to allocate to a target. It could be that both the CNV E-wave and the P300 amplitude reflect some general attentional resource.

The role of RT in the processing of information

In the study of TBI, increased latency in behavioral RT has often been used as an index of reduced information processing speed. Reduced information processing speed has, in turn, been considered to be causally related to reduced information processing capacity (e.g., Gentilini, Nichelli, & Schoenhuber, 1989; Gronwall, 1989). This view is pervasive in a much broader context than the study of TBI. For example, Eysenck (1987) has linked response speed to human intelligence. Kail (1992) has argued that children's memory growth is reflected in their response speed. Salthouse (1985) has also argued that a decrement in information processing speed, typically indexed by speed in timed perceptual–motor tasks, underlies the reduction in information processing capacity in aging. In all these cases, response speed is taken to be an index of the speed of basic information processing in the brain.

While we found no relationship between behavioral RT and stimulus evaluation time (P300 latency) for either control or TBI subjects, we did find that the behavioral response speed of controls correlated with P300 amplitude, an ERP measure typically linked to attentional allocation.

Thus, aspects of attentional allocation that are not speed-related may account for the major source of variance in RT, for example, the decision to respond. However, RT may not serve this function in populations where it can be influenced by greater variability in response selection, motor planning, and execution independent of conceptual ability. For example, Dywan and Jacoby (1988), using a partial masking paradigm, found that whereas elderly participants were indeed slower than younger individuals to name objects, they were not slower to identify them. Those results suggest that the output aspects of language production adds variance that is not related to the speed of item recognition. Similarly, Dywan et al. (1992) found that P300 latency is predictive of only the simplest of cognitive functions among older adults, and not various complex thinking skills. We have found, however, that complex thinking skills are consistently related to CNV in adolescents (Segalowitz et al., 1992a), adults with TBI (Segalowitz et al., 1992b), and older adults (Dywan, Segalowitz, & Williamson, 1994).

Summary and Conclusions

Examining TBI and control participants on a simple tone discrimination task, we found that the TBI subjects were significantly slower in their behavioral response times, and that the standard deviation of those responses also was greater in the TBI group. Thus, we replicated the results reported by Stuss et al. (1989, 1994) who proposed that inconsistency of response is a distinguishing characteristic of TBI. Our goal, however, was to determine the relationship between this response slowness and inconsistency and concomitant attentional difficulties in the TBI group. We demonstrated that the inconsistency of behavioral response time is highly related to various attentional processes, but not to a general slowing of stimulus evaluation time (P300 latency), or to inconsistency in the speed of stimulus evaluation (as addressed by the standard deviation of P300 latency).

We conclude that TBI results simultaneously in a decrease in signal processing efficiency, in decreased attentional capacities, and in an increase in the latency and variability of motor response. The variability, but not the speed, of a simple motor response is to a large degree related to the allocation and control of attention as reflected in ERPs. While the ability to control attention may often be a trait correlate of information-processing speed, we have shown that information-processing speed and attentional control may be dissociable processes in cases of traumatic brain injury.

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