Pathologies of attentional networks following traumatic brain injury

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Abstract—High-density electrical recordings were used to study the time course of processing in tasks that mark the operation of different attention networks. We examined 16 normal subjects and 11 individuals who had suffered traumatic brain injury (TBI) and complained of reduced ability to concentrate. We used three tasks—Continuous Performance Task (CPT), covert orienting task, and Stroop task—to examine vigilance, sensory orienting, and executive control, respectively. Patients showed an overall slowing in response latencies and reduced amplitude of late Event Related Potentials (ERPs). They also showed slower sensory orienting and reduced hit rate in the CPT task, but no performance decrement over time. In both CPT and covert orienting, TBI patients had similar ERP abnormalities in processing cue information relevant for target selection. In the Stroop task, patients showed larger interference from the irrelevant color-words and abnormal electrical activity in midline electrodes, which in normals has been associated with cingulate gyrus activation. These data suggest specific deficits in the executive control network mediated by frontal areas after TBI.

Key Words: Event related potentials, attention deficits, orienting, vigilance, executive control.

Introduction

Recent work in cognitive neuroscience has suggested the existence of separate anatomical systems associated with three distinct attentional functions: vigilance, sensory orienting, and executive control (Posner & Petersen, 1990). Vigilance refers to the ability to develop and maintain an alert state through time and is measured in tasks in which participants are asked to detect rare targets for extended periods of time without interruptions (Parasuraman, 1984). Sensory orienting refers to the ability to allocate attention in space and is measured by asking participants to detect the onset of simple visual stimuli that can appear in different spatial locations (Posner, 1980). Executive control refers to the ability to resolve conflict between competing information (Norman & Shallice, 1986; Posner & Digirolamo, 1998). Executive control can be measured by presenting participants with multiple sources of conflicting information and asking to respond to one of these sources and to ignore the others.

The functional anatomy of these three attentional systems has been investigated in neuroimaging studies using positron emission tomography (PET) and functional magnetic resonance imaging (fMRI). In particular, three tasks requiring different kinds of attentional control have been used in neuroimaging studies. The vigilance system has been investigated by using the continuous performance task (CPT), a test of sustained attention widely used in neuropsychology (Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). This task requires participants to respond to targets specified by a conjunction of conditions ("press a key if you see a three only if it is preceded by an odd digit") and to withhold response in

This work was supported by a grant from the James S. McDonnell Foundation 96-47 and by the Office of Naval Research Grant N00014-96-0273. A preliminary version was presented at a conference on Temporal Attention, Philadelphia, PA, September 1997 and at the Meeting of the Cognitive Neuroscience Society, Washington DC, April 1999.

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all the other cases. PET investigations (Coull, Frith, Frackowiak, & Grasby, 1996; Pardo, Fox, & Raichle, 1991) have revealed that the CPT activates a right lateralized network that includes frontal and superior parietal regions.

The sensory orienting system has been investigated using the covert orienting task, a test involving the ability to shift to a location in the visual field eccentric to the fovea following a central cue (Corbetta, 1998; Mangun, Jha, Hopfinger, & Handy, in press). One version of this task requires detecting a target stimulus that is presented after a cue indicating the probable location of the target. In this task, performance is influenced by cue validity: participants are faster when the target appears in the cued location than when it appears in a non-cued location (Posner, 1980). This function has been traced to a network of posterior areas including the parietal lobe, superior colliculus and pulvinar (Posner & Petersen, 1990), but has also access to control from frontal areas (Corbetta, 1998).

The executive control system has been investigated using the Stroop task, a test of conflict between a word and color cue. In the Stroop task, color-words printed in color are presented an individuals are asked to name the color and to ignore the word. Typically, compared to a neutral condition in which the word is not associated to the concept of color, people are slower and less accurate when the color and the word are conflicting (incongruent condition) and faster and more accurate when color and word match (congruent condition, MacLeod, 1991). Functional imaging studies have shown the involvement of the frontal midline in this task, and in particular of the anterior cingulate cortex (Digirolamo, Heidrich, & Posner, 1998; Pardo, Pardo, Janer, & Raichle, 1990; Posner & Digirolamo, 1998). It is thought that the cingulate cortex carries out important functions related to monitoring and resolution of conflict, which are aspects of executive control.

The present study investigates how brain networks associated with vigilance, sensory orienting, and executive control may be altered after brain injury and how complaints of attentional deficits in people who have suffered brain injury are reflected in impairment of specific cognitive operations performed by these networks. Event related potentials (ERPs)—a technique long familiar from chronometric studies in psychology (Rugg & Coles, 1995)—measure scalp electrical activity time-locked to stimulus events and has high temporal resolution, in the order of milliseconds. Thus, ERPs are a good tool for the study of time course of brain activation during specific task performance, especially when the underlying functional anatomy of the task is already known (Heinze, Münte, Gobiet, Nieman, & Ruff, 1992; Posner & Raichle, 1994).

In this study, we collected both behavioral and EEG data while participants were performing tasks requiring different types of attentional control. We choose three tasks that have been already investigated in neuroimaging studies and for which we have a good understanding of the neural substrate: the CPT (vigilance), the covert orienting task (sensory orienting), and the Stroop task (executive control).

Traumatic brain injury is the most common cause of acquired diffuse brain damage and is the consequence of violent head impacts, often at high speed (e.g., motor vehicle accidents), which result in both focal and diffuse cerebral damage. The shaking of the brain within the skull is responsible for the diffuse damage, which typically consists of axonal shearing in the white matter of the cerebral hemisphere and brain stem (Katz, 1992). The focal damage is usually concentrated in the inferior surface of the frontal lobes and inferior/anterior surfaces of the temporal lobes.

Table 1.
Injury and Demographic Information for Each Patient

*S*ubject	Age at injury	Months post injury	Etiology	site(s) of lesion	length of coma
GC	18	26	MVA[a]	Fronto-parietal	11 days
MS	49	15	MVA	Frontal	1 day
CM	21	16	MVA (motorcycle)	R temporo-parietal with skull fracture	2 weeks
SO	29	38	Struck by falling object	Primary site unclear; later scan negative	< 1 hour
JD	52	30	Fall	Primary site unclear; scan negative	< 1 hour
SF	45	19	MVA	Frontal; scan negative	< 1 hour
TB	16	141	MVA	L temporo-parietal, with R temporo-parietal involvement	5 days
GG	49	12	MVA	Frontal	< 1 hour
JS	17	26	Fall	Occipital with frontal involvement	< 1 hour
RW	42	13	MVA (motorcycle)	Occipital	< 1 hour
GrG	44	26	Fall	Parietal-occipital	2 weeks

[a] *MVA = motor vehicle accident; L = left; R = right.*

Complaints of attention and memory deficits are common after moderate to severe traumatic brain injury (TBI). These impairments are associated with long-term cognitive deficits and have a strong impact on the everyday activities and performance at work (Kinsella, 1998). TBI patients report problems with concentration, distractibility, forgetfulness and difficulty doing more than one thing at a time (Hinkeldey & Corrigan, 1990; Mateer, Sohlberg, & Crinean, 1987). Experimental investigations have found evidence of deficits in the allocation of attentional resources, switching between tasks with different cognitive requirements, time-sharing processing resources and overcoming automatic responses when faced with non-routine situations (Mateer & Mapou, 1996; Stablum, Leonardi, Mazzoldi, Umiltà, & Morra, 1994).

A better knowledge of specific cognitive impairment after brain injury is important not only for understanding attention and memory deficits in TBI patients, but also to address rehabilitation and treatment issues.

General Methods

Subjects

Controls. The control group included 16 right-handed undergraduate students (6 females) at the University of Oregon. Their average age was 21.75 (range 18-36 years).

Patients. We selected 11 patients with traumatic brain damage (5 females, 10 right-handed). Table 1 summarizes injury and demographic information of the patient group. Those patients were selected for the presence of deficits in attention and for being at least one full year after the accident. Their average age was 37.7 (range 20-55 years).

Because the two groups of subjects differed in age, we also analyzed data from a sub-group of six patients and six controls who were age-matched. In this subgroup, the average age was 24.8 years for the control group and 27.3 years for the patient group ($t[6] = 0.552$, $p = .59$). We found no evidence of differences in the pattern of behavioral and EEG results in the age-matched groups and in the complete groups.

Session, procedure, apparatus

During the experimental session, participants were presented with the three attentional tasks mentioned above. The methodological details for each task are described in the following sections. The experiments were programmed in EGIS (Osgood, 1990) and presented on a Radius 20e computer monitor controlled by a Macintosh PowerPC 8500. The order of tasks was counterbalanced between subjects. The experimental session lasted about three hours.

EEG Methods

EEG was recorded from the scalp using the 128-channel Geodesic Sensor Net (Tucker, 1993). Figure 1 shows the 128-electrode layout compared with the 10-20 system. The recorded EEG was amplified with a 0.1-50 Hz bandpass, 3dB attenuation, and 60-Hz notch filter, digitized at 250 Hz with a 12 bit A/D converter, and stored on magnetic disk. The EEG epochs lasted 1 second and began with a 200 ms pre-stimulus baseline. All recordings were referenced to Cz. ERPs were re-referenced against the average of all the 128 channels, and averaged for each condition and for each subject after automatic exclusion of trials containing eye blinks and movement artifacts. Subject averages were baseline corrected unless otherwise specified. Grand averages across subjects were computed for each task and each group. All statistical comparisons between ERP waveforms used a non-parametric Wilcoxon signed-rank test for each 4 ms sample. Differences were accepted as significant only if two conditions differed for at least four samples (16 ms) at the $p < .01$ level at each test in at least two contiguous channels.

ERP Amplitude differences between normals and patient

It is possible that particular brain processes are more vulnerable than others to brain damage after TBI. To investigate this issue, we compared the amplitudes of three ERP components, P1, N1, and P300, in patients and controls. Physical features of the stimuli and attentional manipulations should have differential influence on early and late components. For example, the P1 is sensitive to the physical characteristics of the stimulus and to some attentional factors (i.e., allocation of attention in space), whereas later components, such as the P300, reflect primarily attentional and strategic factors (Coles, Gratton, & Fabiani, 1990). Therefore, abnormality in early or

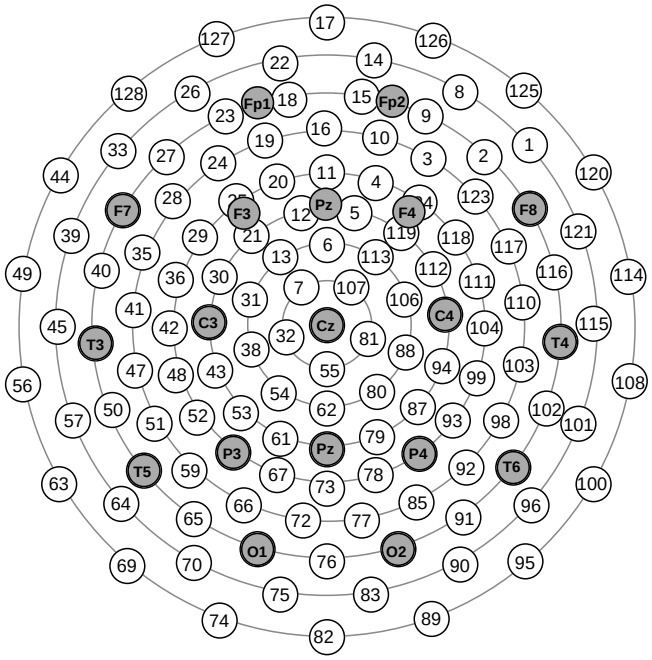


Figure 1. Layout of the 129 channels in the Geodesic Sensor Net® used in this study. The location of the electrodes according to the 10-20 system is superimposed for comparison.

late components may reveal something about the level of processing that is more impaired in TBI patients. Several results in the literature suggest that TBI patients have reduced amplitude of specific ERP components. For example, some studies have shown a reduction of P300 amplitude in patients with brain injury (Campbell, Houle, Lorrain, Deacon-Elliott, & Proulx, 1986; Curry, 1980; Unsal & Segalowtiz, 1995; Wirsén, Stenberg, Rosen, & Ingvar, 1992), whereas other studies indicated a selective reduction of the N1 (Heinze et al., 1992).

To investigate amplitude differences in early and late ERP components we computed, for each subject and each task, the peaks of P1, N1, and P300. Table 2 shows the average values in microvolts of these ERP components in patients and controls. P1 and N1 peaks were computed at electrodes 71 and 84, corresponding to the O1 and O2 locations in the 10-20 system. The P1 peak was computed as the maximum amplitude in the interval 80-120 ms and the N1 as the minimum amplitude in the interval 100-200 ms. The peak of the P300 was computed as maximum amplitude in the interval 300-500 ms. The P300 was measured in the central channel that showed that largest positive deflection around 300 ms after stimulus onset (channel 55, just below Cz, for the covert orienting task, and channel 68, corresponding to Pz, for CPT and Stroop task). The condition that showed the largest modulation of the components under investigation was chosen for each task. We used the four-target condition for the CPT task, the invalid condition for the covert orienting task[1], and the congruent condition for the Stroop task (see description of each task for further details). Average peak values for each subject were analyzed in a one-tailed t-test.

As shown in Table 2, our data replicates the reduction in P300 amplitude in TBI patients found in other studies, especially in the Stroop task and in the CPT. Amplitude of earlier ERP components such as P1 and N1 did not reveal any significant difference between the two groups although trends were generally in the direction of smaller ERPs in patients.

Data in the literature suggest that the attenuation of the P300 amplitude in the averaged ERPs is likely to reflect a true reduction in single trial amplitude rather than increased latency jitter or decreased absolute EEG power. For example, Unsal and Segalowitz (1995) compared ERP from normal controls and traumatic brain injury patients in an auditory oddball paradigm and found that approximately 90% of all the variance in the amplitude of the averaged P300 was accounted for by single trial P300 amplitude.

These results suggest that the ERP signature of early and intermediate perceptual processing is not greatly different in patients and normal controls, at least when simple visual stimuli are used. This finding is in contrast with the marked reduction of the N1 in closed head injury patients reported by Heinze et al. (1992) in a visual search task. We believe that the perceptual nature of the visual search task used by Heinze and colleagues may have been responsible for the greater sensitivity of the N1 to brain damage. In our tasks, the perceptual demands of the tasks were minimal. Each trial required either identification of a single stimulus at fixation or detection of a peripheral stimulus. Thus, it is possible that only tasks requiring greater perceptual processing are able to reveal abnormality in early ERP components after brain damage. This hypothesis is supported by the results of other studies that did not find any N1 difference between normal controls and brain injury subjects using tasks that were not perceptually demanding (Rugg et al., 1989; Rugg, Pickles, Potter, Doyle, & et al., 1993). A second factor that may have contributed to the discrepancy in results is the location of the stimuli in the visual field. In our study, the stimulus that elicited P1 and N1 was always foveal. Some unpublished data from our laboratory indicate that TBI patients show an abnormal reduction of the P1 when the stimulus is presented in the periphery rather than at fixation. This suggests that peripheral stimulus presentation may reveal ERP abnormalities in the first 100 ms that are not apparent with foveal presentation.

Vigilance network

Patients with lesions of the right posterior parietal lobe (Robertson et al., 1997) and right frontal lobe (Wilkins, Shallice, & McCarthy, 1987) show deficits in maintaining the alert state during sustained attention tasks. Converging evidence of a right lateralized brain network for vigilance is provided by neuroimaging studies. Pardo, Fox, and Raichle (1991) showed that tasks that require maintaining an alert state over

Table 2

Average peak amplitude and standard deviation (in microvolts) of the components P1 , N1, and P300 in normal controls and patients as a function of task (covert orienting, Stroop, and CPT).

	P1					*N1*					*P300*				
	Controls		*Patients*			*Controls*		*Patients*			*Controls*		*Patients*		
TASK	M	SD	M	SD		M	SD	M	SD		M	SD	M	SD	
CPT	3.88	2.05	3.33	2.10	ns	-3.62	3.64	-2.94	2.19	ns	8.10	2.50	4.85	3.00	***
Covert orienting	2.77	1.98	2.96	2.24	ns	-3.48	3.04	-3.03	2.73	ns	5.22	2.85	3.25	1.77	*
Stroop	4.95	3.77	3.92	2.49	ns	-4.74	3.74	-3.24	2.54	ns	8.47	2.63	5.47	3.60	**

*Note: the asterisks indicate the probability values of the comparison between controls and patients; * = $p < .06$; ** = $p < .02$; *** = $p < .005$; ns = non significant*

time produce a strong activation of right frontal and right superior parietal brain areas, regardless of stimulus modality and side of presentation. More recently, Coull et al. (1996) found similar results using a version of the continuous performance task.

Clinicians often report difficulties in maintaining arousal and alert state in TBI patients (Whyte, Polansky, Fleming, Coslett, & Cavallucci, 1995) and a deficit in sustained attention may be expected following the type of brain damage associated with TBI. Diffuse axonal injury may disrupt the reticular activating system involved in arousal regulation (Jane, Steward, & Gennarelli, 1985; Parasuraman, Mutter, & Molloy, 1991; van Woerkom, Teelken, & Minderhous, 1977; Whyte et al., 1995). Furthermore, frontal cortex dysfunction, often reported in this type of injury, has been associated with vigilance and goal-oriented behavior impairments (Pardo et al., 1991; Wilkins et al., 1987).

Most of the empirical studies investigating vigilance and sustained attention after TBI found a reduction in hit rates and slower reaction times in patients as compared to normal controls (Burg, Burright, & Donovick, 1995; Parasuraman et al., 1991; Ponsford & Kinsella, 1992). Surprisingly however, data on decrement in performance over time in TBI patients are inconsistent (Loken, Thornton, Otto, & Long, 1995; Parasuraman et al., 1991; Whyte et al., 1995) and several studies show no deterioration of performance, even after long experimental sessions (Parasuraman et al., 1991; Ponsford & Kinsella, 1992).

Methods

The continuous performance task consisted of the presentation of a sequence of digits. Subjects were asked to pay attention to the sequence and to press a key only when they saw a four that was preceded by an odd number.

The digits were presented in the center of the monitor using the font times, print size 24, typeface bold. The trial started with a 200-ms blank followed by a digit that was displayed for 500 ms. The time available for the response was 800 ms after nontarget trials and 1500 after target trials. EEG recording started 200 ms before the onset of the digit and lasted 1 second.

The experiment consisted of 500 trials, divided in two 250-trial blocks. In 80% of the trials (400 trials) the stimulus was not a four (non-four non-target), in 10% of the trials (50 trials) the stimulus was a four that followed an even digit (four not-target), and in the remaining 10% of the trials (50 trials) the stimulus was a four that followed an odd number (four target). The entire session lasted about 15 minutes.

Results

Behavioral data

We were interested in investigating overall hit rate, commission errors and latencies and their change over time (Parasuraman, 1984). The experiment was divided in two blocks of 250 trials, separated by a short rest period. We wanted to examine whether performance differed in the two blocks and whether within in each block there was a decline in performance between the first and the second half. Therefore, performance of patients and controls were analyzed in mixed ANOVAs examining proportion of hits, mean RTs to hits, and proportion of false alarms as a function of block (first and second) and of block part (first half and second half; see Table 3).

Table 4
Averages and Standard Deviations of the Number of hits and Reaction Times to correct trials (in milliseconds) as a function of group and session in the Continuous Performance Task.

	Patients		Controls	
	M	SD	M	SD
Proportion of Hits				
First Session				
First Half	0.91	0.14	0.97	0.05
Second Half	0.92	0.11	0.98	0.03
Second Session				
First Half	0.93	0.07	0.97	0.04
Second Half	0.92	0.09	0.99	0.03
Reaction Times (ms)				
First Session				
First Half	593	119	521	119
Second Half	664	159	588	109
Second Session				
First Half	652	157	609	127
Second Half	681	189	601	97

Patients had significantly less hits than controls, $\underline{F}(25, 1) = 9.33$, $\underline{MSE} = 0.009$, $\underline{p} < .006$. However, none of the effects and interactions including block and block part approached significance. Patients and controls had a uniform level of hit rates across blocks and block parts. Therefore, there was no indication of a decrement in performance in the hit rate, but rather a slight nonsignificant improvement over time.

The analysis of correct RTs indicated that patients were slightly slower than controls (647 and 580 ms, respectively), but this effect did not reach significance, $\underline{F}(25, 1) = 2.074$, $\underline{MSE} = 57608$, $\underline{p} > .15$. None of the interactions involving group was significant. The effect of block was significant, $\underline{F}(25, 1) = 11.920$, $\underline{MSE} = 4168$, $\underline{p} = .002$, indicating that both patients and controls were slower in the second block than in the first block. Also the effect of block part was significant, $\underline{F}(25, 1) = 7.738$, $\underline{MSE} = 5296$, $\underline{p} = .01$. Both groups were slower in the second half of the block than in the first. Finally, the block by block part interaction was significant, $\underline{F}(25, 1) = 5.729$, $\underline{MSE} = 3839$, $\underline{p} < .05$, indicating that the slowing between first and second half of the first block was larger than the slowing between first and second half of the second block.

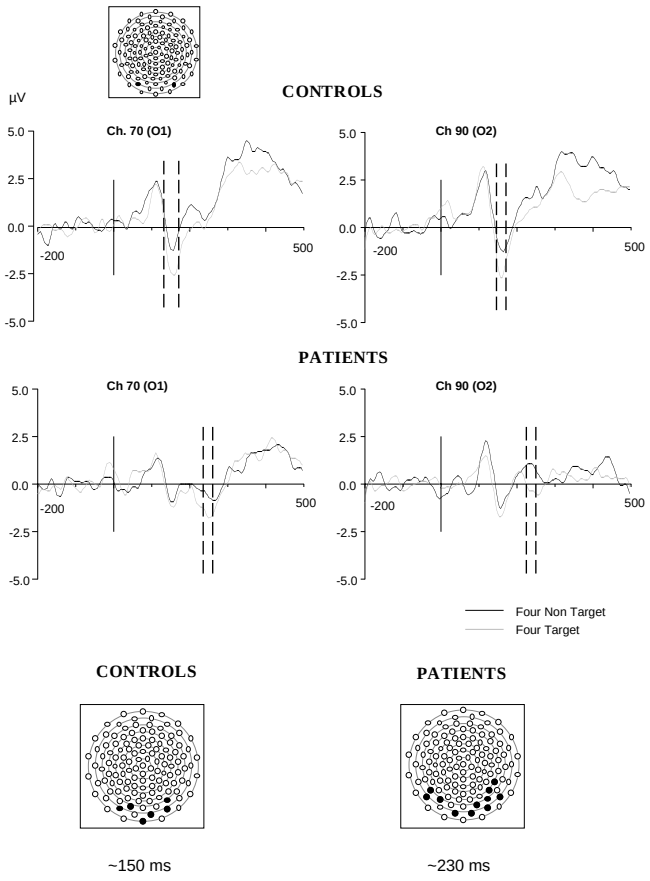
Two types of commission errors (incorrect responses to four-nontargets and incorrect responses to non-fours) were

used as dependent variables in a mixed ANOVA that examined the effect of block, block part, and error type. The only significant effect was that of error type, $F(25, 1) = 14.79$, $MSE = 0.004$, $p < .001$. Both patients and controls had a higher rate of commission errors to four-nontargets than to nonfour-nontargets (4% and .03% in the control group and 3.9% and .05% in the patient group).

EEG data

The present behavioral data, in agreement with the literature, indicate that TBI patients were impaired in measures of tonic alertness (percentage of hit rate), rather than in measures of vigilance maintenance (performance over time, Parasuraman, 1984). Therefore, in analyzing the ERP data we focussed on the comparison that could shed some light on this deficit.

Figure 2. Left channel 70 and right channel 90 in controls and patients for four-target and four non-target trials in the continuous performance task. These two electrodes are slightly more external and more posterior than the O1 and O2 locations in the 10-20 system (the electrode layout on top indicates the position of the two electrodes). At about 150 ms, controls show a remarkable enhancement of N1 amplitude in the four target condition that is absent in patients. Patients show a difference between the two conditions only later, at about 230 ms. The dashed lines indicate the interval in which the difference between the two conditions is significant. The two electrode layouts on the bottom indicate channels that show significant differences in this time window in patients and controls.



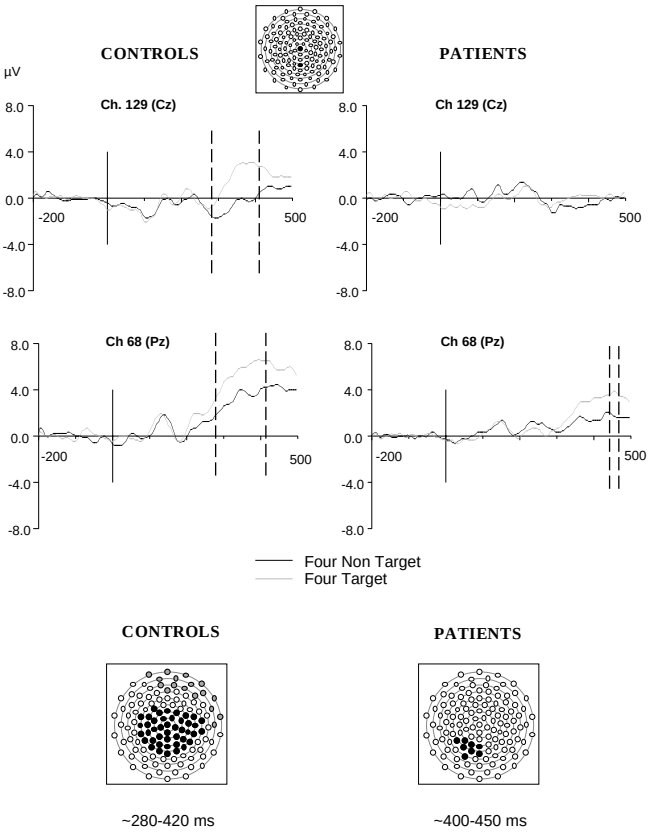


Figure 3. Channels 129 (Cz) and 68 (Pz) in controls and patients for four-target and four-nontarget trials in the continuous performance task. The electrode layout on top indicates the position of these two channels. In patients, the P300 is much smaller than in controls and virtually absent at Cz. Controls show a broad enhancement of the P300 for four-target trials in both electrodes at about 300 ms, whereas patients show a reduced and later (450 ms) enhancement of the P300 in the four-target condition at Pz but not at Cz. The dashed lines indicate the interval in which the difference between the two conditions is significant. The two electrode layouts on the bottom indicate channels that show a significant difference in these intervals. The gray dots indicate a significant waveform inversion in the same interval.

In the CPT, two logical conditions must be satisfied for a stimulus to be a target: The stimulus must follow an odd number and must be a four. The three experimental conditions in the continuous performance task—four-target, four-nontarget, and nonfour-nontarget—allowed us to dissociate the two mental operations that are required to select the target. The first is a context evaluation operation (whether the previous stimulus was an odd or an even number) that can be examined by looking at the difference between four-targets and four-nontargets. These two conditions only differ in the category of the previous stimulus, an odd or even digit, and in whether the current stimulus is a target. The second operation is the evaluation of the physical difference between fours and other digits, which can be examined by looking at the difference between the nonfour-nontarget and four-nontarget conditions.

Comparison between four-targets and four-nontargets. In normals, early difference between four-targets and four-nontargets appeared at about 150 ms after stimulus presentation as a symmetric enhancement of the N1 component in posterior channels for target trials. In patients, a similar but somewhat more extended group of channels showed increased negativity in the four-target condition at a later time, at about 230 ms (Figure 2).

The second effect in normals is a central and broadly distributed difference in correspondence of the P300 component, starting at about 280 ms and lasting about 150 ms (see Figure 3). During this interval, the four-target condition is more positive than the four-nontarget condition. Patients also show a later positivity associated with the four-target condition. However, in this group the difference between the two conditions is much smaller, occurs later (at about 450 ms), and is significant in fewer channels. In particular, in patients the distribution of the P300 enhancement in the target condition is more posterior than in controls.

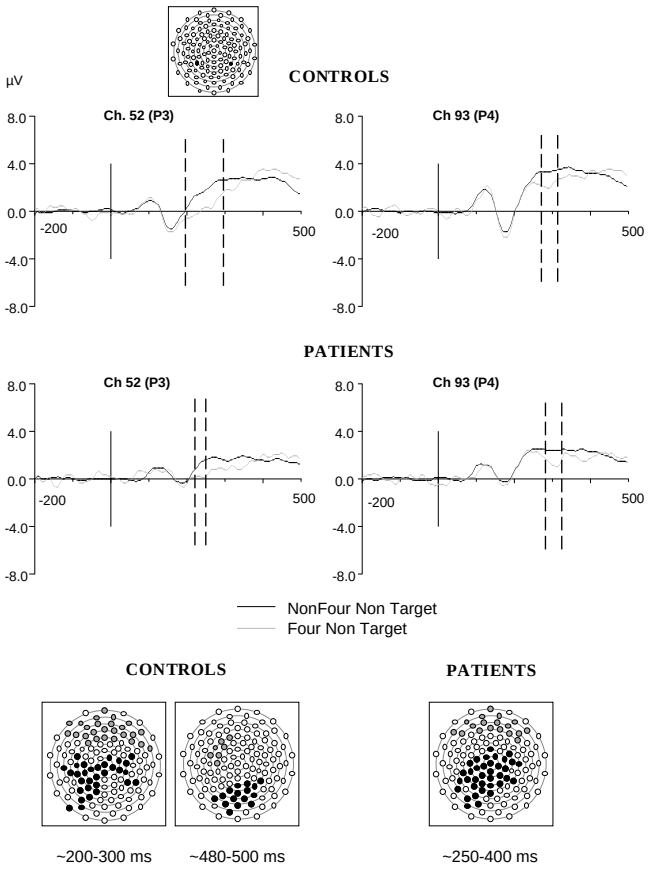
Comparison between nonfour-nontargets and four-nontargets. Nonfour-nontargets and four-nontargets are physically dissimilar non-target digits. In normals, this comparison revealed a broad difference starting at about 200 ms. In both conditions, parietal and posterior channels showed a positive deflection, which tended to peak later and had a larger amplitude in the four-nontarget condition than in the non-four condition. Temporal and topographic characteristics of this effect were similar in patients and in controls (see Figure 4). In particular, it is worth noting that patients did not show any delay in discriminating between fours and non-fours, suggesting that the cognitive slowing that has been reported in TBI patients may not be general, but rather dependent on the type of cognitive operation performed. Controls, but not patients, showed a second difference in posterior channels between non-four non-targets and four-non targets at about 500 ms.

Discussion

In the behavioral data, patients showed a significantly worse performance than controls in the hit rates, but no differences in commission errors and response latencies. The only effect of session was an increase in RTs over time, similar in both patients and controls, with no increase in errors. This slowing in RTs was particularly pronounced between the first and second half of the first block and may be the result of a shift in response criterion over time. Therefore, this type of task does not seem able to reveal phasic decreases in alertness during the task. Other studies have reported a similar impairment in tonic vigilance without a concomitant vigilance decrement. However, some studies showed decrement in performance in severe TBI (Loken et al., 1995). Whyte et al. (1995) suggested that longer sessions (about 15 minutes) and longer interstimulus intervals (about 6 sec) may be a crucial factor in increasing the sensitivity of the task to decrements in vigilance.

The ERP data suggest a specific deficit in target detection in TBI patients. Our results indicate that patients did not differ from controls in processing the physical aspect of the target. The difference between four-nontarget and non-four stimuli appeared as a larger positivity for non-four stimuli at about 200 ms after stimulus onset. In both groups this difference had similar time course and was larger and appeared earlier on the left side. However, a striking difference between the two groups was clear in the comparison between four-target and four-nontarget conditions. These two conditions did not differ in the physical appearance of the stimulus, but merely in the category of the previous stimulus (i.e., odd numbers versus even numbers). In the control group, the differ-

Figure 4. Channels 52 and 93 in controls and patients for non-four non-target and four non-target trials in the continuous performance task. These electrodes lie just above P3 and P4 locations in the 10-20 system (the electrode layout on top indicates the position of these two electrodes). The waveforms in these two conditions appear strikingly similar in patients and in controls. Both groups show a larger positivity in the non-four non-target condition starting at about 200 ms in left channels and at about 250 ms in right channels. The dashed lines indicate the interval in which the difference between the two conditions is significant. The two electrode layouts at the bottom indicate channels that show significant differences in these conditions. The gray dots indicate electrodes in which there is a significant inversion of the effect in the same interval. Note that controls, but not patients, have a second significant difference between non-four non-targets and four non-targets at about 500 ms, which is more posterior and left lateralized.



ence between four-targets and four-nontargets appeared early, as an enhancement of the N1 in four-target trials, and was apparent again later as enhancement of the P300 for targets. In patients, the early N1 enhancement in the four-target condition was absent and only 80 ms later posterior channels showed a negative deflection in the four-target condition when compared to the four-nontarget condition. The enhancement of the P300 appeared 150 ms later than in controls, was smaller and limited to fewer and more posterior electrodes.

These results suggest that patients have difficulties in selecting a target when they have to rely on previously presented information rather than on the physical features of the current stimulus. This conclusion is also supported by an analysis of the interval preceding the stimulus in these two conditions. We found that the baseline of four-target trials was significantly more negative than the baseline of four-nontarget trials in the control group but not in the patient group. This finding indicates that controls had a larger contingent negative variation (CNV) after odd numbers than after even numbers, whereas patients did not. Similar abnormalities in the CNV of brain injury patients in a go/no go task have been previously reported (Curry, 1980; Rugg et al., 1989).

These ERP results suggest that overall hit rate and changes of performance over time are independent measures of vigilance and are associated to different brain processes. Decrement in performance over time can be maximized by using non-engaging tasks that last long periods of time without interruption. It has been suggested that a slow stimulus presentation rate is a crucial factor to increase the likelihood to detect deficits in sustained attention in TBI patients (Whyte et al., 1995). Overall hit rate seems to be sensitive to other types of manipulations. In tasks such the CPT, which require to use previously presented information for target selection, the decreased hit rate in TBI patients is at least in part the result of specific deficits in the ability to use information that is not physically present. It is likely that tasks that use fast presentation rates and difficult target selection (Parasuraman et al., 1991) measure deficits in this mechanism of target selection. In the present experiment, we found that TBI patients have a specific deficit in this aspect of vigilance, in the absence of a performance decrement.

The sensory orienting network

The covert orienting task (Posner, 1978) has been widely used to study the mechanism involved in orienting toward visual stimuli. During this task, individuals are asked to detect the onset of simple visual stimuli that can appear in different spatial locations. At various intervals before the target, a cue stimulus is presented that sometimes indicates the probable location of the target. Participants are required to keep their eyes at fixation, so that changes in performance as a function of the cue cannot be the result of eye movements or changes in visual acuity. The typical finding in this paradigm is a benefit in performance when the target appears in the cued location (valid condition) and a cost when the target appears in a non-cued location (invalid condition), as compared to a condition in which the cue does not provide spatial information about the target (neutral condition) (Posner, 1980).

It is important to note that cues provide two types of information: *when* the target will occur and *where* it will appear. The where information allows attention to be moved to the probable target location (sensory orienting); the when information allows one to prepare for the appearance of the target (phasic alertness). There is some evidence that these two mechanisms are independent. Behaviorally, the effects of orienting and alerting are different. Orienting is a location-specific effect associated with more efficient processing (shorter latency and higher accuracy), whereas alerting is a global (non location-specific) effect associated with a speed-accuracy tradeoff (faster RTs and higher error rate) (Posner, 1993). Furthermore, the effects of orienting and alerting appear to be additive and independent (Fernandez-Duque & Posner, 1997). At the neurophysiological level, there is evidence that different neurotransmitters are involved in alerting and orienting. A warning signal that indicates when the target will occur operates by changing the alert state through the norepinephrine pathway (Marrocco & Davidson, 1998), while the effect of an orienting cue seems to involve the cholinergic system (Davidson, 1998; Marrocco & Davidson, 1998).

Changes in performance as a function of the cue in the covert orienting task provide information on both orienting and alerting mechanisms. The efficiency of the orienting mechanism can be measured by examining the pattern of benefits and costs in the valid and invalid conditions. Alerting can be measured as global effect of a warning signal (such as the cue) regardless of validity (Fernandez-Duque & Posner, 1997).

There is considerable information on the mechanisms involved in orienting toward visual signals. Lesion, alert monkey and imaging studies have suggested a network of brain areas that include the posterior parietal lobe, pulvinar and superior colliculus as well as frontal areas (Posner & Petersen, 1990). Neuroimaging studies have shown that a network of superior parietal and superior frontal lobe is activated during spatial orienting of attention. In particular, Corbetta and colleagues (1993) have shown that parietal regions are active any time spatial locations are selected, whereas frontal regions are active only when overt response is required.

Visuo-spatial deficits in TBI patients have been reported. However, the tests used in these studies, for example the letter cancellation task (Geldmacher & Hills, 1997; Hills & Geldmacher, 1998) or the trail making test, require not only to move attention to locations in space but also to perform a visual search. Deterioration in performance in these tasks may be caused by problems in planning and monitoring the search rather than in spatial orienting deficits (Sohlberg, McLaughlin, Pavese, Heidrich, & Posner, 1998).

Methods

In the covert orienting task, subjects detected the onset of an asterisk presented in the left or in the right visual field. The display consisted of two squares, one on the left and one

on the right, which subtended 4.1° of visual angle at the viewing distance of 60 cm. The edge-to-edge distance between the two boxes was 7.3° of visual angle. A fixation dot was displayed in the center of the monitor, between the two boxes. Participants were instructed to fixate the dot during the entire duration of the trial. The cue appeared in the same position as the fixation dot and consisted in either a cross, an arrow pointing to the left box, or an arrow pointing to the right box. The cue always subtended a visual angle of .9° in height and in width.

Each trial began with a 700 ms interval in which only the two squares and the fixation dot were shown, followed by the presentation of the cue. After a stimulus onset asynchrony (SOA) of 150 ms (short) or 800 ms (long), an asterisk appeared in the left or in the right box. Participants were asked to press a key with the left hand as soon as they detected the target stimulus. Cue and target were displayed until response, or for 5 seconds. EEG recording started 200 ms before the cue onset and lasted 1 second.

The experimental design included three conditions, each defined by the type of cue and the relationship between cue and target position: (a) in the neutral condition the cue consisted of a cross that did not provide any information about the target location; (b) in the valid condition the cue consisted of an arrow and the target was displayed in the box indicated by the arrow; (c) in the invalid condition the cue consisted of an arrow and the target was displayed in the noncued box. Laterality effects were examined looking at left and right orienting cues.

Subjects contributed 156 trials to each of the two SOA conditions. The neutral cue appeared in 48 trials (30.8%). Of the remaining 108 trials, 84 (77.8%) were valid and 24 (22.2%) invalid.

Results

Behavioral data

The data were analyzed in three-way mixed analyses of variance (ANOVAs) that examined the effect of group (patients and controls), validity (valid, invalid and neutral), and SOA (short and long). The dependent variables were RTs (mean of response latencies longer than 100 ms) and percentage of anticipation (responses shorter than 100 ms). Mean RTs and percentage of anticipations for patients and controls are reported in Table 4.

Table 4
Mean RT (in milliseconds) and anticipations (in percentage) as a function of validity, SOA, and group in the covert orienting task.

	Patients			Controls		
	Valid	Neutral	Invalid	Valid	Neutral	Invalid
Short SOA						
RT (ms)	422	451	449	331	348	356
Anticipations (%)	.19	.19	1.53	.45	.78	.52
Long SOA						
RT (ms)	368	400	422	275	294	303
Aticipations (%)	4.54	3.22	4.17	4.24	3.25	6.25

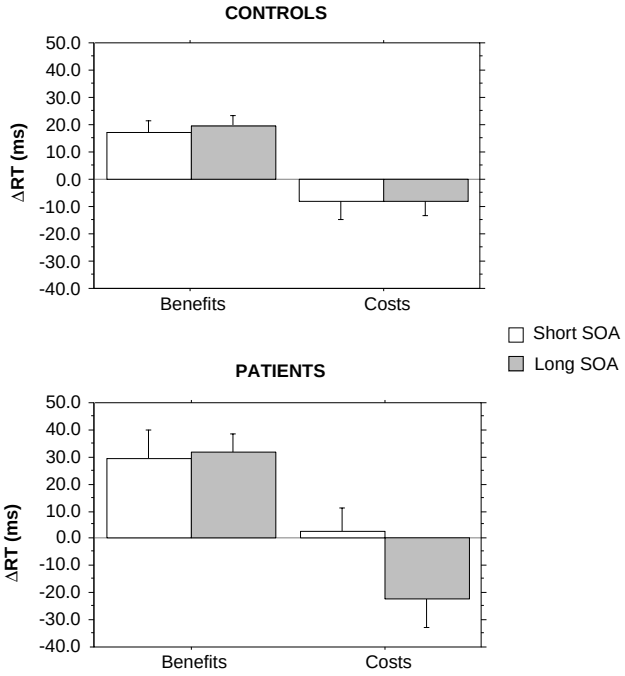


Figure 5. Benefits (neutral condition minus valid condition) and costs (neutral condition minus invalid condition) in milliseconds as a function of group (Controls and Patients) and SOA in the covert orienting task.

The RT analysis showed a significant effect of group, $\underline{F}(1, 25) = 5.57$, $\underline{MSE} = 71788$, $\underline{p} < .03$. Patients were 101 ms slower than controls. The effect of validity was significant, $\underline{F}(2, 50) = 36.55$, $\underline{MSE} = 429$, $\underline{p} < .0001$. As expected, valid trials were faster than neutral trials, and invalid trials were slower than neutral trials (340, 364, and 373 ms for valid, neutral, and invalid, respectively). The effect of SOA was significant, $\underline{F}(1, 25) = 61.27$, $\underline{MSE} = 1537$, $\underline{p} < .0001$, indicating that participants were slower to respond to short SOA trials than to long SOA trials (384 and 334 ms, respectively).

To better understand the pattern of costs (invalid condition minus neutral condition) and benefits (neutral conditions minus valid condition) in the two groups, we ran two separate repeated measures two-way ANOVAs on patient and control groups. The two factors were cue effect (costs and benefits) and SOA. In the control group, neither the effect of SOA, $\underline{F}(1, 15) < 1$, nor the SOA by cue effect interaction, $\underline{F}(1, 25) < 1$, approached significance. In the patient group, however, the interaction SOA by effect was significant, $\underline{F}(1, 10) = 5.80$, $\underline{MSE} = 341$, $\underline{p} < .05$. As shown in Figure 5, SOA did not influence costs and benefits in the control group. In the patient group, however, costs were present only at the longer SOA. Finally, a comparison of benefits between the two groups showed a trend toward larger benefits in patients (31 ms) than in controls (18 ms; $p = .065$).

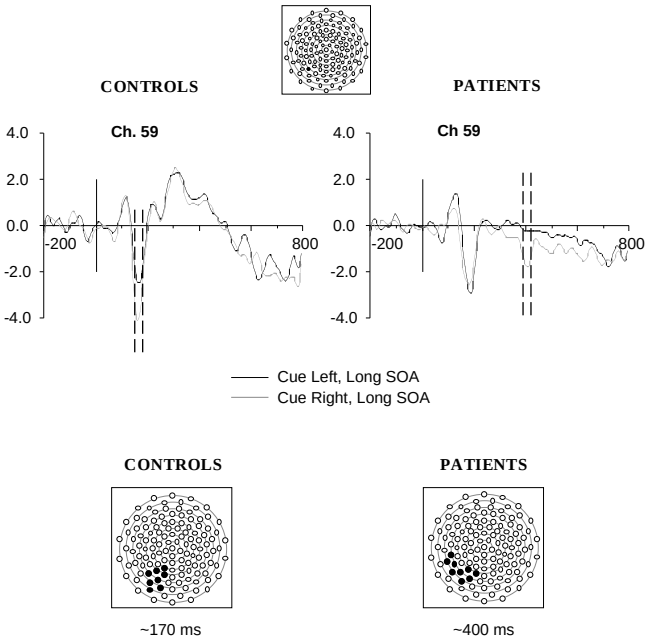


Figure 6. Left channel 59 (between T5 and P3) in patients and controls for left and right cued trials at the long SOA interval in the covert orienting task. The black dot in the electrode layout on top indicates the position of electrode 59. ERP waveforms indicate the interval after cue onset and before target onset. In controls, right cued trials produce a larger N1 in a group of left posterior channels at about 170 ms. The electrode layout on the bottom left indicates channels that show a significant difference in this interval in the control group. In patients, a negative enhancement for right cued trials was apparent in the same region at about 400 ms. The electrode layout on the bottom right indicates channels that show a significant difference in this interval in the patient group. The dashed lines indicate the intervals in which the difference between the two conditions is significant.

The analysis of anticipations did not reveal any effect of group. Patients and controls had about the same anticipation rate (2.5% and 2.6%, respectively). No interactions involving group approached significance. The effect of SOA was significant, $F(1, 25) = 26.86$, $MSE = 17.89$, $p < .0001$, indicating that the percentage of anticipations was higher at the long than for at short SOA (4.3 and 0.7, respectively).

Analyses carried out on the two age-matched subgroups replicated these results. In particular, in these subgroups patients were 134 ms slower than controls and showed costs at the longer, but not at the shorter, SOA.

EEG data

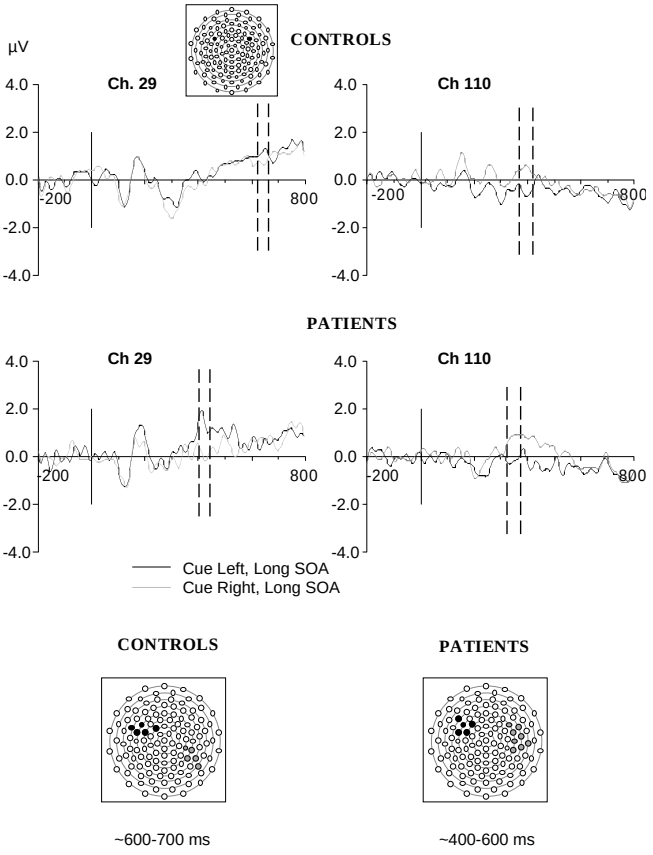
Brain correlates of the cognitive preparation to target response that occurs after cue presentation were investigated in this task[2]. To examine this issue, we examined the cue-target interval in the long SOA condition, and looked at the differences between ERP associated with left cued, right cued, and neutral trials. We expected a CNV in the three conditions, lateralized in the cued trials, and differences between neutral and cued trials, likely located in parietal regions.

ERP in the Cue-Target interval (Long SOA trials).

In the control group, the comparison between left and right cued trials revealed two main differences. A group of left posterior channels showed an enhanced N1 in right-cued trials as compared to left-cued trials at about 170 ms (see Figure 6). An opposite effect (N1 larger for left cues) was visible on the right side, but it did not reach significance. A lateralized anterior difference was apparent at about 400 ms. In this interval, right anterior channels showed greater negativity for left-cued trials and left parieto-temporal channels showed greater negativity for right-cued trials. This difference may correspond to the lateralized CNV that is associated to the preparation for the target (see Figure 7).

In patients, the early effect of cue presentation was ab-

Figure 7. Left channel 29 (between F3 and F7) and right channel 110 (above T4) in patients and controls for left and right cued trials at the long SOA interval in the covert orienting task. The two black dots in the electrode layout on top indicate the position of electrodes 29 and 110. ERP waveforms were recorded in the interval after the cue onset and before target onset. Right cued trials are more negative on the left at about 600 ms in controls and 400 ms in patients. Left cued trials are more negative on the right at about 400 ms in controls and 350 ms in patients. The dashed lines indicate the intervals in which the difference between the two conditions is significant. The two electrode layouts on the bottom indicate channels that show significant differences in controls and patients. Black dots indicates channels in which right cued trials are more negative than left cued trials whereas gray dots indicate the opposite trend.



sent. A group of right posterior channels—partially overlapping with the group of channels that showed the N1 enhancement in controls—showed increased negativity in right cued trials much later, at about 400 ms. Patients showed a lateralized fronto-parietal CNV. This effect had a similar topography in patients and in controls but appeared earlier in patient (at about 400 ms rather than 600 ms).

Discussion

Behavioral data indicate that patient RTs were about 100 ms slower than control RTs. For both patients and controls valid RTs were fastest and invalid were slowest with neutral trials in between. Patients showed slightly larger benefits than controls and a modulation of costs as a function of SOA that was absent in controls. In particular, patients did not show any costs at the short SOA but they showed a large cost at the long SOA (22 ms, as compared to the 9 ms showed by controls).

Both the greater benefits and the dissociation between costs and benefits at short SOAs may be accounted for by the general cognitive slowing of the patient group. If attention moves more slowly in TBI patients, we would expect that any reorienting of attention would take longer than in control. Thus, the latency difference between valid (no reorienting) and neutral trials (orienting from a central or diffuse allocation of attention to the target location) would be larger, as well as the difference between neutral trials and invalid trials (reorienting from the invalidly cued position to the target position).

Anticipation rate was higher at the long SOA and slightly higher for cued trials than non-cued trials, but no difference between the two groups was apparent from the data. Patient's performance as a function of SOA is identical to normal controls: both groups were 50 ms faster and showed more anticipation responses at long SOAs than at long SOA. This suggests that the alerting effect of the cue is similar in patients and controls. This result in agreement with previous results in the literature indicating no difference in alerting between normal controls and brain injury patients (Ponsford & Kinsella, 1992; Whyte, Fleming, Polansky, Cavallucci, & Coslett, 1997).

The onset of the cue generated a lateralized enhancement of the N1 in controls. At about 170 ms, right-cued trials showed a greater negativity on left posterior channels than left-cued trials. Patients also showed a lateralized increased negativity in a similar group of right posterior channels, but this effect appeared much later, at about 400 ms.

Normal subjects showed a CNV over frontal and parietal electrodes sites following the cue. This negative deflection was particularly evident in long SOA trials. The CNV showed a lateralized effect of cue direction. Right-cued trials showed greater negativity on left frontal channels and left-cued trials showed a greater negativity on right parieto-temporal channels. This pattern fits well with the CNV usually found to follow the cue in this task (Harter, Aine, & Schroeder, 1984). The topography of patients' CNV was similar to that observed in controls, but this difference appeared about 200 ms earlier in patients than in controls.

These results suggest the some abnormalities in the effect of central cues on spatial orienting. In particular, patients have delayed lateralized enhancement of contralateral components after the onset of the cue. However, other effects of spatial cueing appeared normal in patients. Phasic alerting following a warning signal was normal in TBI patients, in agreement with previous results. For example, White and colleagues (Whyte et al., 1997) found that accuracy and RT changes after an auditory warning signal were identical in patients and controls. Also the frontal CNV in preparation for target presentation appeared similar in normals and patients, although patients showed an earlier onset of the effect.

The executive attention network

The most widely held view of executive function identifies the importance of resolution of conflict between competing information (Norman & Shallice, 1986; Posner & Digirolamo, 1998). The Stroop test (Stroop, 1935) is one of the most studied task exploring conflict resolution. In a version of this task, color-words printed in color are presented and individuals are asked to name the color and to ignore the word. Typically, compared to a neutral condition in which the word is not associated to the concept of color, people are slower and less accurate when the color and the word are conflicting (incongruent condition) and faster and more accurate when color and word match (congruent condition) (for a review see MacLeod, MacLeod, 1991)

Tasks that involve conflict resolution, such as the Stroop test, show consistent activation of areas within the frontal midline, especially in the anterior cingulate cortex (Bush et al., 1998; Pardo et al., 1990; Posner & Petersen, 1990; Posner & Rothbart, 1998). Posner and Peterson (Posner & Petersen, 1990) have proposed that anterior cingulate cortex, together with other frontal structures, is part of a frontal network that is involved in executive attention.

Because frontal lobes are especially vulnerable to TBI, one should expect executive function deficits in these patients. There is some evidence that Stroop test scores are a good index of recovery outcome in TBI patients. For example, performance in the Stroop test discriminates between mild TBI patients that have a good recovery 3 months after the injury and patients that have persistent postconcussive symptoms (Bohnen, Twijnstra, & Jolles, 1992). Similarly, Stroop scores differentiated individuals who required no assistance with activities of daily living from those requiring some level of assistance (Leahy & Lam, 1998). However, some researchers have suggested that deficits in the Stroop task are only a measure of cognitive slowing and not a specific deficit in conflict resolution (Ponsford & Kinsella, 1992).

In this experiment, we used the Stroop task to investigate the frontal executive attention network in normal controls and patients. High-density ERP recording, together with behavioral measures, allowed us to compare not only the pattern of facilitation and interference in RTs but also time course and topography of activation in the two groups.

Methods

We used a variation of the Stroop test (Stroop, 1935), in which subjects were asked to name the color in which color-words were written and to ignore the identity of the word. The words were printed in four colors—yellow, green, blue, and red—and appeared against a gray background. The color of the background was chosen to maximize color visibility. At the viewing distance of 60 cm, each letter subtended a visual angle of 0.6° in height and 0.5° in width, and word length varied between 1.4 and 3.3 degrees (three to six letters).

Subjects were required to name aloud the color in which the word was written. There were three experimental conditions, defined by the identity of the word: (a) congruent condition, when the word corresponded to the ink color; (b) incongruent condition, when the word was the name of a color different from the ink color; and (c) neutral condition, when the word was not a color name. The stimulus set consisted, therefore, of four color-words corresponding to the four print colors (RED, YELLOW, BLUE, and GREEN) used in the congruent and incongruent conditions, and in 72 high frequency non-color words used in the neutral condition[5].

Each trial started with a fixation dot in the center of the monitor that was displayed for 1200 ms. A color word was then presented until response or for 10 seconds. Subjects were asked to fixate the center of the monitor and to respond vocally to the color in which the word was printed. The experimenter entered the identity of the response through the computer keyboard. Each participant performed 288 trials, 96 in each condition.

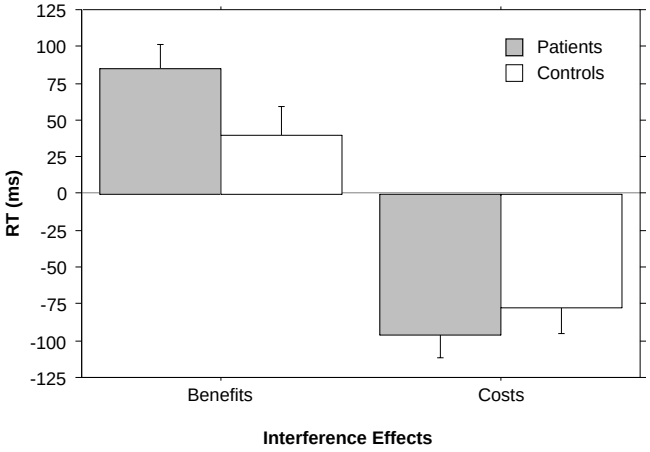
Results

Behavioral data

In this experiment, RTs and accuracy were measured from the manual response entered by the experimenter and not from the subject vocal response. As a consequence, recorded RTs are longer than the actual response latencies and the data are in general more noisy. However, the experimenter could not see the stimulus display and was not aware of the experimental condition at the time the stimulus was presented; therefore, no bias should be present in the data.

Mean reaction times and percentage of error were analyzed in two-way mixed ANOVAs that examined the effect of group (patients and controls) and condition (congruent, neutral, and incongruent). Average RTs and error percentages for patients and controls are reported in Table 5.

Figure 8. Benefits (neutral condition minus congruent condition) and costs (neutral condition minus incongruent condition) in the RT data of the Stroop task (in milliseconds).



The RT analysis showed a significant effect of group, $F(1, 25) = 14.1$, $MSE = 141640$, $p < .001$. Patients were 319 ms slower than controls. Condition was significant, $F(2, 50) = 57.8$, $MSE = 7043$, $p < .0001$. As expected, neutral trials were slower than congruent trials and faster than incongruent trials ($ps < .0001$; 1924, 1982, and 2068 ms for congruent, neutral, and incongruent, respectively). Finally, the condition by group interaction approached significance, $F(2, 50) = 2.8$, $MSE = 2538$, $p < .075$, indicating a trend toward a larger interference in the patient group than in the control group. In particular, patients showed a greater facilitation effect in the congruent condition than controls (Figure 8). This result suggests that patients were slightly more influenced by the irrelevant color word than controls.

The error analysis did not show a significant effect of group, $F(1, 25) = 1.9$, $MSE = 5.1$, $p > .15$, although patients had a slightly larger error rate than controls (2.3% and 1.6%, respectively). The effect of condition was significant, $F(2, 50)$

Table 5
Mean and Standard Deviation of RT (in milliseconds) and errors (in percentage) as a function of condition and group in the Stroop task

	Patients						Controls					
	Congruent		Neutral		Incongruent		Congruent		Neutral		Incongruent	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
RT (ms)	2091	320	2177	323	2273	345	1809	76	1849	85	1926	118
Errors (%)	1.2	1.7	1.1	1.5	4.5	3.0	.9	1.0	1.3	1.9	2.6	3.4

= 10.0, MSE = 8.4, $p < .0005$. Planned comparisons indicated that the incongruent condition was significantly less accurate than the congruent and neutral conditions ($p < .001$; see Table 5). The group by condition interaction did not reach significance, $F(2, 50) = 1.6$, $p > .20$, even though the patient group showed a higher cost in the incongruent condition than the control group (differences in accuracy between incongruent and neutral conditions were 3.4% and 1.3% for patients and controls, respectively).

EEG data

The most salient feature of the Stroop effect, which we replicated in our behavioral data, is the difference in latencies and errors between congruent and incongruent conditions. This result supports the traditional hypothesis that the Stroop effect measures the conflict between the response activated by the color and the response activated by the word in incongruent trials (MacLeod, 1991). Previous evidence from this laboratory (Digirolamo et al., 1998), however, suggests that in normals the earliest and most prominent ERP effect in the Stroop task is a significant difference between trials in which the word represents a color (congruent and incongruent conditions) and trials in which the word is not related to color (neutral condition).

Our control data replicate this finding. In normals, congruent and incongruent waveforms start to diverge from neutral trials at about 300 ms. This difference occurs in correspondence with the beginning of the P300 and results in a larger positivity for congruent and incongruent trials than for neutral trials in midline electrodes posterior to Cz (see Figure 9). Congruent and incongruent conditions start to diverge at about 400 ms, but this difference reaches significance only later, at about 430 ms, and only in few left parietal and left frontal channels.

In patients, the ERP pattern is quite different. In this group, neutral and incongruent trials have similar waveforms. Only at about 300 ms these two conditions show significant difference in few central channels. This is very different from the broadly distributed difference found in controls. In patients, the congruent condition significantly diverges from both incongruent and neutral condition at about 200 ms, especially in left posterior and central anterior channels. Later, at about, 350 ms, a second difference between congruent and neutral conditions is apparent, concentrated in central electrodes. Figure 9 also clearly show the great reduction of the P300 in patients that we discussed in the amplitude analysis.

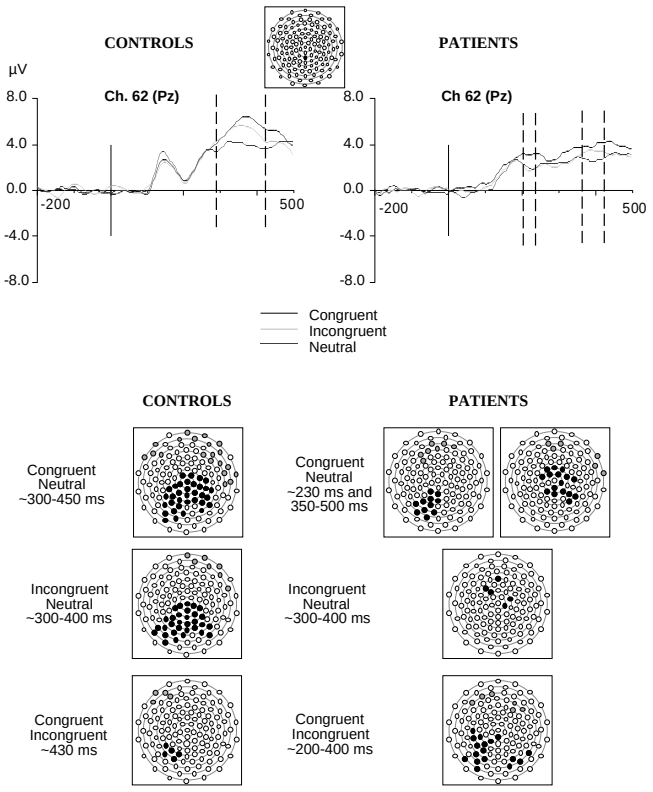


Figure 9. Channel 62 (just above Pz) in controls and patients for congruent, incongruent, and neutral trials in the Stroop task. The black dot in the electrode layout on top indicates the position of electrode 62. In normals, the neutral condition significantly differs from the congruent and incongruent conditions starting at about 300 ms, in correspondence of the P300 peak. Congruent and incongruent trials are significantly different only much later and in few right posterior and right anterior channels. In patients, the congruent condition is significantly more positive than the neutral and incongruent conditions at about 200 ms. At about 350 ms the incongruent condition is significantly more positive than the neutral condition in a group of central channels. The dashed lines indicate the interval in which the difference between the two conditions is significant. The electrode layouts on the bottom indicate channels that show significant differences between pairs of conditions in patients and controls. The gray dots indicate an inversion of the effect in the same interval.

Discussion

The behavioral data show some evidence that TBI patients are impaired in the Stroop task. Patients were much slower than controls in this task (more than 300 ms) and showed somewhat larger interference effects. In particular, patients showed a larger facilitation in the congruent condition in the RT data and a larger cost in the incongruent condition in the error data. However, behavioral evidence for a specific deficit in dealing with response conflict is not very strong. Impairment in conflict resolution should appear as disproportionate increase in RTs and errors in the incongruent condition as compared to the neutral condition. Differences in interference effects, however, only approached significance and were not very large in absolute value.

The ERP data offer more compelling evidence of impairment of the mechanism of conflict resolution in TBI patients. In normals, brain electrical activity first discriminates between color-word stimuli (congruent and incongruent conditions) and noncolor-word stimuli (neutral trials) and only later discriminates between congruent and incongruent trials. Therefore, it seems that a first process is specifically associated to

the suppression of word reading, especially when the word does not represent a color (Digirolamo et al., 1998). The distinction between congruent and incongruent conditions that is so evident in the behavioral data arises only later, and is likely associated with response selection processes.

Digirolamo and colleagues (Digirolamo et al., 1998) have suggested that this pattern of results is consistent with a process of selection in two stages. In the first stage, the relevant dimension (in this case, color) is selected against the non-relevant dimension (word name) by suppressing word reading. Stronger semantic association between relevant and non-relevant dimensions causes greater interference (Klopfer, 1996; Pavese & Umiltà, 1998) and therefore suppression of word reading should be greater in congruent and incongruent conditions (strong semantic association between color and word) than in the neutral condition (no semantic association between color and word). The second stage corresponds to response selection and is responsible for the cost associated to incongruent trials and the facilitation associated to congruent trials in RT and error data. Digirolamo and colleagues have also shown that the larger positive deflection observed in midline electrodes around 300 ms for congruent and incongruent trials is compatible with the activation of the anterior cingulate cortex. Activation of the anterior cingulate during the Stroop task has been shown in several PET studies (Carter, Mintun, & Cohen, 1995; George et al., 1994; Pardo et al., 1990) and is consistent with the involvement of a frontal executive attentional system in the Stroop task (Posner & Digirolamo, 1998). Interestingly, in their PET study of the Stroop task Bench et al. (Bench et al., 1993) found no difference in cingulate activation between congruent and incongruent conditions. Similarly, Carter et al. (Carter et al., 1995) found activation in the right anterior cingulate in both congruent and incongruent blocks of trials.

In patients, the congruent condition showed early differentiation from incongruent and neutral waveforms at about 200 ms after stimulus onset and was expressed in larger positivity for the congruent condition. Only later, at about 300 ms, congruent and incongruent trials diverged from the neutral trials in few midline electrodes. This pattern of activation, taken together with the strong reduction in the P300, suggests abnormal target selection in the Stroop task. In particular, these results are consistent with a greater influence of automatic processing in the patient group. If the automatic activation of the word dimension is not efficiently controlled by a mechanism that suppress non-relevant information, we would expect larger effects of word activation in early stages of processing. The congruent condition is different from the other two conditions in that both word and color are associated to the same color representation. We propose that the larger early positivity in the congruent condition is a manifestation of greater activation of the representation associated with both color and word, due to a reduction in word suppression. This hypothesis is also consistent with the behavioral data showing increased facilitation in the congruent condition and increased errors in the incongruent condition.

General Discussion

This study examined behavioral and ERP measures of performance in a group of TBI patients and a group of normal controls. We used three tasks that are known to involve three different attentional networks: vigilance, sensory orienting, and executive attention.

Our results suggest four groups of abnormalities in TBI patients. The first two abnormalities, which generalize to all tasks, are RT slowing and reduction of amplitude of the P300 in the ERP. The third abnormality is apparent in the results of the cover orienting task and CPT as specific impairment in the use of cue information for strategic changes. This abnormality is associated to slower spatial orienting in the cue task and reduced hit rate in the CPT. Finally, the Stroop data show a slightly larger effect of distractor information in the behavioral data and a marked ERP abnormality in midline parietal electrodes.

Other cognitive operations did not seem impaired in the patient group. For example, patients showed normal perceptual processing and normal alerting response after a warning signal. Finally, we found increased response latencies over time in the vigilance task—similar in patients and in controls—but no difference in hit rates.

General effects of TBI: cognitive slowing and amplitude reduction of the P300

Cognitive slowing is the most common outcome in people that have suffered brain injury (Ponsford & Kinsella, 1992; Van Zomeren, 1981). We replicated this effect in all three tasks. However, the slowing was not equivalent in the three tasks. An analysis of variance on the RT data that examined the effects of task (covert orienting, CPT, and Stroop) and group (controls and patients), revealed a significant effect of group, $\underline{F}(1, 25) = 13.22$, $\underline{MSe} = 39136$, $\underline{p} < .002$, indicating that patients were overall slower than controls, and an effect of task, $\underline{F}(1, 25) = 1204.99$, $\underline{MSe} = 121416$, $\underline{p} < .0001$. Interestingly, the task by group interaction was also significant, $\underline{F}(1, 25) = 7.06$, $\underline{MSe} = 17207$, $\underline{p} = .002$, indicating that the difference in RT between patients and controls was not equivalent in the three tasks (101, 67, and 319 ms for covert orienting task, CPT, and Stroop task, respectively). This finding is consistent with other results in the literature showing differential slowing in TBI patients as a function of the type of processing (Schmitter-Edgecombe, Marks, Fahy, & Long, 1992; Shum, McFarland, Bain, & Humphreys, 1990).

Another indication that the cognitive slowing in patients is not homogeneous across levels of processing comes from the ERP data of the CPT. In this task, the discrimination between a four preceded by an even number (non-targets) and a four preceded by an odd number (targets) occurred 80 ms later in patients than in controls. However, the discrimination between the digit four (potential target) and the other digits was very similar in the patient and control groups, showing no ERP differences in latency or topography. This result suggests that some cognitive operations are not impaired or de-

layed in TBI patients.

Our ERP results indicated that in all three tasks there is some reduction in the P300 component in the patient group, whereas earlier components such as the P1 and the N1 have similar amplitudes in the two groups. Several studies have reported reduction in the amplitude of later components in the ERP of brain injury patients (Campbell et al., 1986; Curry, 1980; Unsal & Segalowtiz, 1995; Wirsén et al., 1992) and there is evidence suggesting that this effect is not the result of latency jitters or decreased absolute EEG power (Unsal & Segalowtiz, 1995). We believe that this result reflects problems in transmission of data between brain areas arising from axonal shearing typical of TBI. Decrease in P300 amplitude probably reflects difficulty in conveying information to mechanisms such as midline frontal structures related to cognitive control.

Deficit in using previous information to select the target: covert orienting and CPT and covert orienting results

One striking difference between normals and patients is apparent in the comparison between brain electrical activity during the CPT and the covert orienting task. A lower hit rate in brain injury patients than in controls in vigilance tasks is a common result in the literature (Brouwer & Van Wolffelaar, 1985; Parasuraman et al., 1991; Ponsford & Kinsella, 1992), even in the absence of a greater decrement in performance over time. Our results suggest that, at least in the CPT, decrement in hit rate is associated with impairments in the capacity to use previously presented information to select the current target. Comparisons between patients' and controls' waveforms revealed that controls, but not patients, showed a negative deflection before the onset of digits that followed an odd number (potential targets) as compared to digits that followed an even numbers (non-targets). Furthermore, after the onset of the stimulus, controls showed an amplification of the N1 for four-targets, whereas patients showed a later and reduced negative deflection. The same abnormality was found in the covert orienting task in which cue presentation elicited a lateralized enhancement of the N1 in controls but a much later increased negativity in patients. This suggests that TBI patients have a specific deficit either in keeping track of the flow of information in continuous tasks or in getting that information to the posterior brain areas appropriate for target detection, or both. Difficulties in keeping track of continuous information flow can also explain, at least in part, the impairment that TBI patients show in other tasks that have similar demands, such as the paced auditory serial addition task (PASAT, Cicerone, 1997).

Deficit in Conflict resolution: the Stroop task results

The results of the Stroop task revealed slower latencies and a slight increase in interference effects in patients as compared to controls (greater facilitation in the congruent condition and a larger error rate in the incongruent condition). However, these results are not a strong index of impairments of the mechanism that supervises conflict resolution. The absence of a substantial increase in Stroop interference in brain injury patients is common in the literature. Some authors have proposed that performance in the Stroop test can be explained just in terms of cognitive slowing, without assuming any specific deficit in conflict resolution (i.e., Ponsford and Kinsella, Ponsford & Kinsella, 1992).

Our ERP data, however, suggest that patients and controls use different strategies in dealing with the Stroop task. Controls show a clear distinction between the congruent and incongruent conditions and the neutral condition, starting at about 280 ms. Electrical activity in this interval is consistent with activation of the anterior cingulate cortex (Digirolamo et al., 1998). Patients show a very different ERP pattern, with an earlier discrimination between the congruent condition and the incongruent and neutral conditions, starting at about 200 ms. This result suggests a deficit in the executive control networks involving the anterior cingulate in TBI patients. Although deficits in executive attention in brain injury populations have been previously reported, we believe that our study has provided more precision on the time course and localization of this effect.

Functions that are preserved after TBI

We think that our strategy of combining behavioral and neuroimaging data to study the effect of brain injury is promising. Some of the differences between patients and controls that were evident in the ERP data were not apparent from the behavioral data alone. For example, ERP data allowed identification of the characteristics and the time course of deficit associated with lower hit rate in the CPT. In the Stroop task, a small behavioral effect was associated with remarkable midline abnormalities in the ERPs. Neuroimaging techniques such as ERPs can help us understanding when some differences between patients and normals appear and where in the brain the abnormality may originate. Further research using these methods can give important information on the brain mechanisms underlying cognitive impairment in brain injury and possibly give us important suggestions for the rehabilitation of attentional deficits after TBI.

Notes

[1] In the covert orienting task, maximum component amplitude was computed on trials in which the stimulus onset asynchrony (SOA) between cue and target was 150 ms. Therefore, in this task P1 and N1 components primarily reflect processing of the cue stimulus, whereas the P300, which appears at about 500 ms after the cue and 350 ms after the target, primarily reflects target processing.

[2] Comparisons between validly- and invalidly-cued targets presented on left and right visual field are more traditional in the ERP literature on spatial orienting. However, the design of the experiment, combined with technical limitations,

made this comparison impossible in the current study. When the cue-target interval was short (150 ms), components associated to target processing overlap with components associated with cue processing. Furthermore, invalid trials for each visual hemifield were only 12, too few to produce clean ERP waveforms, especially in the patient group. In the long cue-target interval (800 ms), only waveforms following to cue were available, because EEG recording lasted 800 ms after cue onset.

[3] Identical results were replicated in our laboratory on a group of normal controls using only four unrelated words in the neutral condition (Digirolamo et al., 1998).

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