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Visual P300 Effects Beyond Symptoms in Concussed College Athletes

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ABSTRACT

In order to assess whether cerebral anomalies may be observed in the absence of clinical symptoms, the current study compared the effects of concussions on attentional capacities (reaction times, accuracy) and Event-Related Potentials (ERPs) in concussed athletes with ($n = 10$) or without ($n = 10$) symptoms as well as in athletes who never had a concussion ($n = 10$). The P300 response was recorded from 28 electrodes during a modified visual oddball paradigm. Participants were instructed to press a key upon the appearance of the frequent stimuli as well as when a rare nontarget stimulus followed the frequent one. The other key was to be pressed when the subsequent rare stimuli (rare target) appeared until a frequent one reappeared. The symptomatic athletes displayed longer reaction times than the other two groups of athletes. The P300 amplitude to the rare target stimuli was significantly more attenuated in the symptomatic athletes than in the other two groups. Moreover, the P300 amplitude varied inversely with the severity of postconcussion symptoms but was not influenced by time elapsed since injury. Although the clinical significance of the P300 differences shown by the symptomatic athletes is still uncertain, the results do indicate that symptom severity may be a crucial indicator of functional impairments following mild traumatic brain injury.

In North America, about 2 million individuals incur head injuries every year, which represents the main cause of brain damage in adults under 40 years of age (Sullivan, Schefft, Warm, & Dember, 1994). Many mild and moderately head-injured patients report no adverse effects following injury but about half develop postconcussion symptoms (Mandel, 1989). Concussions may be defined as ''a transient disturbance of neuronal function as a result of acceleration'' (Parkinson, 1996) and as ''a trauma-induced alteration in mental status that may or may not involve a loss of consciousness'' (Kelly, Nichols, & Filley, 1991). The observed impacts on behavior are often a state of confusion, memory loss, fatigue and headaches (American Psychiatric Association, 1994). The symptoms appear in different intensities and depend on several factors such as the location of the injury, the intensity of the hit and the number of previous concussions (Boden, Kirkendall, & Garrett, 1998; Cantu, 1992). This problem constitutes the most common type of head injury in sports and young professional athletes represent a large proportion of that population (Harmon, 1999). As many as 250 000 athletes are affected each year by concussions, thereby representing a population at risk

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(Wilberger, 1993). This type of injury accounts for 75% of the total number of reported injuries on or about the head in college football players (Buckley, 1988). Because of the apparent absence of neurological signs, athletes who suffer concussions are rarely investigated in the long term and the residual pathophysiological changes are relatively unknown (Leblanc, 1994).

The present study proposes to examine whether brain electrical activity could be a sensitive tool to identify the effects of concussion. As a result of concussion, brain electrical activity aberrance has been found in a large number of patients and electroencephalographic (EEG) abnormalities are directly related to the severity of the concussion soon after the accident (Geets & de Zegher, 1985). Raw EEG measurements, however, are still inconsistent in identifying long-term impairments after a brain injury (Haglund & Persson, 1990). In an extended review, it was emphasized that standard EEG procedures are not useful for the assessment of mild concussion. On the other hand, the so-called Event-Related Potentials (ERPs), offer more promising results in detecting anomalies following brain injuries (Gaetz & Bernstein, 2001). The ERP measure represents the averaged EEG signal time-locked to the stimulus and consists of different components labeled by their amplitude polarity (e.g., P for positive and N for negative) and temporal range in milliseconds. ERPs provide a high temporal resolution method for studying the timing and integrity of cognitive processing such as attention and memory updating (Coles & Rugg, 1995a). Many investigations have shown that the P300 amplitude is a manifestation of attentional allocation (Duncan-Johnson & Donchin, 1977; Johnson & Donchin, 1978; Kramer & Spinks, 1991), subjective significance (Polich, 1986) and stimulus probability (Duncan-Johnson, 1981; Duncan-Johnson & Donchin, 1977), whereas the P300 peak latency is normally considered to reflect the time necessary for stimulus evaluation (Coles, Gratton, & Fabiani, 1990; Coles, Smid, Scheffers, & Otten, 1995b; Donchin & Coles, 1988). The P300 components may typically be evoked by oddball tasks, among others. The oddball paradigm is a task in which two different categories of stimuli are presented unevenly. Various versions of this paradigm have also been proposed, such as the insertion of a second rare stimulus in the sequence, identified as the target (Katayama & Polich, 1996) or different stimuli within the same category such as words or pictures (Greenham, Stelmack, & Campbell, 2000).

Using the oddball task, some investigators have examined differences in ERP components on groups of concussed patients who showed different degrees of severity and who were tested at varying times after the trauma. In general, the P300, elicited by standard auditory oddball tasks, showed little or no sensitivity in discriminating Mild Head Injury (MHI) athletes (Breton, Pincemaille, Tarriere, & Renault, 1991; Haglund & Persson, 1990) or nonathletes (Gaetz & Weinberg, 2000; Sangal & Sangal, 1996; Werner & Vanderzant, 1991). It may, however, provide useful indices of the cumulative damage that can occur following multiple concussions (Gaetz, Goodman, & Weinberg, 2000). Most of these auditory oddball studies have shown absolute differences in the P300 latency and/or amplitude but they did not demonstrate that MHI patients are selectively impaired in a specific condition. Findings that are more comprehensive were achieved with a dichotic listening task, which showed an attenuated P300 (P3b) amplitude in MHI patients during the unattended condition with no effect on P300 latency (Solbakk, Reinvang, Nielsen, & Sundet, 1999). It is interesting to underline that in the monaural condition, which constitutes an analog of a standard auditory oddball task, there were no P300 differences between MHI and controls. A three-tone auditory oddball task was also used with MHI where the patients had to detect rare target tones and withhold responding to other rare targets with an equal probability of occurrence (Solbakk, Reinvang, & Nielsen, 2000). MHI patients were able to detect both classes of deviants but the P300 amplitude was smaller than that of the controls in both conditions. Another study comparing P300 responses to a passive neutral and an active auditory oddball task found that MHI have smaller N200 and P250 in response to both tasks. A significantly smaller and delayed P300 was also noted in this group for both target and nontarget conditions in the active tasks (Reinvang, Nordby, & Nielsen, 2000). Encouraging results with visual

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oddball tasks have also recently been found with MHI patients. For example, an abnormal delayed P300 latency was observed in 70–75% of MHI patients (Gaetz & Weinberg, 2000; Sangal & Sangal, 1996). About half of the MHI also manifested a significantly smaller visual P300 (Gaetz & Weinberg, 2000).

Recently, the transient aspect of concussion has been challenged. Several investigations underlined that postconcussion symptoms can affect individuals on a long-term basis, because of a permanent diffuse damage to cellular brain systems occurring most often in the outer layer of the brain, with mild forces shock (Garnett et al., 2000; Gennarelli, 1996). In order to address this issue, few studies have been carried out to evaluate whether the P300 follows a rapid return to normal values or whether it remains abnormal on a longer term. In this context, a study conducted on severe brain injury patients (Olbrich, Nau, Lodemann, Zerbin, & Schmit-Neuerburg, 1986) revealed that, while cognitive abnormalities returned to normal after 5–6 months, prolongation of P300 latency persisted, suggesting residual cerebral dysfunction. With regard to MHI, Pratap-Chand, Sinniah, and Salem (1988) have reported that the auditory oddball P300 was delayed and smaller in the patient group four days after brain injury. However, repeated testing revealed a return to normal values after 30 days without particular therapy. A normalization of the auditory P300 has also been observed, 8 weeks posttrauma, after feedback training in concussions (Bierbrauer & Weissenborn, 1998) or closed head injury patients (Deacon & Campbell, 1991). In the same context, we have recently shown (Dupuis, Johnston, Lavoie, Lepore, & Lassonde, 2000) that the visual oddball effect (i.e., rare-frequent differences) was significantly smaller in concussed patients than controls or injured athletes who were no longer symptomatic. In fact, the symptom severity was negatively correlated with the P300 oddball effect and we have shown that at least in a single case, a normal oddball effect reappeared after symptoms had abated.

Thus, various sets of results reported herein would tend to suggest that ERP tasks may be sensitive to concussion if the cognitive demands trigger a certain attentional load and are more challenging for the patients. However, few studies have convincingly shown that symptom severity may be related to P300 abnormalities and that, even in the absence of observable symptoms, P300 anomalies could still be observed. The first aim of the present study was therefore to assess whether concussions may indeed affect electrophysiological and behavioral responses in a modified visual oddball task that has a higher level of attentional and cognitive requirements than a standard oddball task. Our second aim was to assess whether P300 anomalies remain even in the absence of observable symptoms. We hypothesized that the P300 amplitude should be related to the intensity of symptom severity but further predicted that, in a condition requesting a high load of cognitive resources, asymptomatic patients should continue to show some anomalies, reflected by a reduced P300 amplitude.

METHOD

Participants

Controls, symptomatic and asymptomatic concussed athletes were selected in order to maintain sample homogeneity. From the original total sample of 47 male participants, 12 participants were rejected because of age (>26; $n = 2$), academic background (<13 years of education; $n = 2$), etiology of concussion (accidents or falls that did not occur within a sports context; $n = 3$) and time elapsed since the last concussion (>24 months; $n = 5$). Individual demographic and clinical data of the selected participants are presented in Table 1. All were university students, practicing sports (football $n = 16$, hockey $n = 10$, rugby $n = 1$, soccer $n = 2$, wrestling $n = 1$) that were referred by their athletic trainer and/or team physician. The first group was composed of athletes ($n = 10$) who had never sustained a concussion and served as a control. The second group consisted of athletes ($n = 10$) who suffered a concussion between 1 month and 2 years before testing ($M = 9.75$ months; $SD = 7.75$), but who were not experiencing more symptoms than the control group at the time of the recording. Participants of the third group sustained a concussion between 1 week and 6 months before testing ($M = 1.7$ months; $SD = 1.95$) and were symptomatic at the time of recording. The three groups did not differ in terms of gender (male), age ($M = 21.5$, 21.4, and 21.6 years, respectively, $F(2, 28) = .081$; $p = .922$) and education

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Table 1. Demographic and Clinical Profile of the College Athlete Participants.

Groups	Cases	Sports	Age	Education (years)	Years in sports	Previous concussion	Severity AAN[a]	Months since last injury	PCS[b] at baseline	PCS at testing	sMRI[c]	CT-Scan[d]
Control												
	1	Football	22	16	10	0	–	–	15	–	–	–
	2	Football	20	14	15	0	–	–	5	–	–	–
	3	Football	21	15	9	0	–	–	8	–	–	–
	4	Football	24	18	20	0	–	–	6	–	–	–
	5	Football	20	14	14	0	–	–	2	–	–	–
	6	Football	21	15	16	0	–	–	4	–	–	–
	7	Football	22	16	8	0	–	–	0	–	–	–
	8	Football	22	15	14	0	–	–	3	–	–	–
	9	Football	22	16	8	0	–	–	8	–	–	–
	10	Football	22	15	18	0	–	–	3	–	–	–
Asymptomatic												
	11	Football	21	15	9	2	2	2	2	3	–	–
	12	Hockey	23	16	19	3	1	23	13	0	–	–
	13	Football	21	16	7	3	2	15	28	0	–	–
	14	Football	19	14	8	2	2	3	15	3	Negative	–
	15	Hockey	21	15	16	3	1	14	0	0	–	–
	16	Football	22	16	8	1	1	13	10	0	–	–
	17	Football	22	16	4	3	2	5	3	9	–	Negative
	18	Hockey	23	17	19	4	2	4	3	3	–	–
	19	Football	22	16	7	2	2	18	4	0	–	–
	20	Football	20	14	5	3	3	2	15	2	–	–
Symptomatic												
	21	Football	21	15	6	2	2	0	3	24	Positive	–
	22	Soccer	21	16	8	3	2	1	1	14	Negative	–
	23	Hockey	21	15	17	3	2	0	3	17	Negative	–
	24	Hockey	21	15	17	2	2	0	1	34	Negative	–
	25	Football	21	15	8	1	3	2	0	68	Negative	–
	26	Football	20	14	7	1	2	0	7	39	–	–
	27	Wrestling	22	16	7	7	3	1	46	52	Negative	Negative
	28	Soccer	21	15	15	3	2	3	14	49	Negative	–
	29	Football	24	18	17	6	2	4	0	25	–	–
	30	Rugby	23	17	11	4	1	6	–	14	Negative	–

Note. [a]American Association of Neurology. [b]Postconcussion Symptoms. [c]Structural Magnetic Resonance Imaging. [d]Computed Tomography Scanning.

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level ($M = 15.6$, 15.5, and 15.4 years, respectively, $F(2, 28) = .069$; $p = .934$).

A standardized concussion history form was administered to obtain information regarding the number and characteristics (e.g., confusion, retrograde and/or anterograde amnesia, loss of consciousness) of all concussions ever sustained. On average, participants from the asymptomatic concussion group reported having experienced 2.6 concussions (range = 1–4) and those from the symptomatic group, 3.2 concussions (range = 1–7). Severity of the latest concussion was classified by a neurosurgeon (KMJ) using the practice parameters of the American Academy of Neurology (AAN). Hence, a Grade 1 concussion corresponded to a state of transient confusion, with no Loss Of Consciousness (LOC), and mental status abnormalities, which disappeared within 15 min. A Grade 2 referred to a state of transient confusion, with no LOC, and mental status abnormalities, which did not disappear within 15 min. Finally, a Grade 3 concussion was characterized by a brief LOC. Using these criteria, the second group (asymptomatic group) had a concussion score of 1.8 ($SD = 0.63$) whereas the third group (symptomatic group) obtained a score of 2.1 ($SD = 0.57$). Mann–Whitney U tests showed that the asymptomatic and symptomatic groups did not differ either in the number of previously sustained concussions or the severity of the last concussion. Ten athletes underwent brain imaging, either Computed Tomography (CT) or Magnetic Resonance Imaging (MRI), some both, ranging weeks to years after the last concussion. All CT scans were normal. Among the 8 patients who underwent MRI using T1, PdT2 and FLAIR sequences, all were considered normal except for 1 participant who showed two hyperintense foci, one in the frontal subcortical white matter and the other, in the anterior third of the left corona radiata.

A Postconcussion Symptoms (PCSs) scale assessed the severity of 19 symptoms rated on a scale ranging from 0 (none) to 6 (severe), for a maximum score of 114 (see appendix National Hockey League PCS evaluation, Lovell & Collins, 1998). Except for 1 symptomatic athlete, all participants underwent the PCS evaluation at the beginning of the sports season as a baseline measure. No statistical differences were noted between the control ($M = 5.4$, $SD = 4.2$), asymptomatic ($M = 10.3$, $SD = 9.6$) and the future-symptomatic ($M = 8.3$, $SD = 14.8$) groups. As noted in Table 1, evaluations were carried out at baseline and following concussion (at time of testing) in asymptomatic and 9 symptomatic athletes. With regard to symptoms, no significant differences were found between evaluations carried out at baseline and at the time of testing in the asymptomatic group. Following concussion, significantly more symptoms were reported in the symptomatic group ($M = 34$, $SD = 18.2$), $t(8) = 4.52$, $p = .002$ (two-tailed). The most commonly reported symptoms included headaches and 'feeling slowed down.'

All participants were also administered a battery of neuropsychological tests (approximately 30 min in

Table 2. Mean, Standard Deviation and Median of the Neuropsychological Testing Results Before (All Groups) and After Concussion (Symptomatic Group Only).

Tests	Time of testing	Controls			Asymptomatic			Symptomatic[a]		
		M	*SD*	*Mdn*	*M*	*SD*	*Mdn*	*M*	*SD*	*Mdn*
Immediate Hopkins	Baseline	31.4	8.2	29.5	30.2	3.4	30.0	31.7	2.0	33
	Postconcussion	–	–	–	–	–	–	29.4	4.2	29
Delayed Hopkins	Baseline	10.3	1.5	11.0	10.7	1.3	10.5	11.1	0.7	11
	Postconcussion	–	–	–	–	–	–	10.1	2.0	10
Symbol Digit	Baseline	*64.3*	10.6	64.0	66.3	*9.9*	63.0	55.9	10.0	58
	Postconcussion	–	–	–	–	–	–	59.9	11.3	57
COWAT	Baseline	41.2	11.2	41.0	43.2	11.5	40.5	45.3	4.7	45
	Postconcussion	–	–	–	–	–	–	46.4	8.9	45
Symbol Cancellation	Baseline	47.8	11.7	44.5	44.9	5.9	45.0	45.6	5.8	43
	Postconcussion	–	–	–	–	–	–	47.2	7.1	45
Color Trail 1[b]	Baseline	*90*	4	90	*84*	*16*	87	82	18	88
	Postconcussion	–	–	–	–	–	–	80	19	82
Color Trail 2[b]	Baseline	82	13	86	80	11	81	76	24	90
	Postconcussion	–	–	–	–	–	–	70	14	82

Note. COWAT = Controlled Oral Word Association Test.
[a]One participant was not evaluated after concussion.
[b]Data of the color trail test are reported in percentiles.

length) adapted from the one used by the National Hockey League (Lovell & Collins, 1998) at the beginning of the sports season. This battery included the Hopkins Verbal Learning Test (verbal learning, delayed memory), the Color Trails Parts I and II (visual scanning and executive functions), the Controlled Oral Word Association Test (COWAT, word fluency), the Symbol Digit Modalities Test (SDMT, information processing speed), the Ruff Graphic Fluency Test (graphic fluency) and the Penn State University (PSU) Symbol Cancellation Task (visual attention and speed of processing). With respect to normative data provided for most by the test administration manuals and for a few (PSU), through personal communication, performances on those neuropsychological tests fell within the normal range for all participants at baseline (preconcussion). Moreover, no significant differences in performances were noted between the three groups on any of the tests (see Table 2). A second neuropsychological evaluation was completed after concussion with 9 symptomatic participants. Statistical analyses reveal that the pre- and postconcussion performances were comparable. The postconcussion results of the symptomatic athletes also did not differ from the baseline data obtained by the other two groups (see Table 2).

Approval for this research with human participants was granted from the Ethics committees of the McGill Medicine Sports Center and the Université de Montréal. Each participant provided written informed consent for voluntary participation.

Experimental Setting and Procedure

The task involved a visual modified oddball paradigm in which the participants were instructed to press a button response upon the appearance of frequent stimuli that appeared 75% of the time. They also pressed the same button whenever a rare target

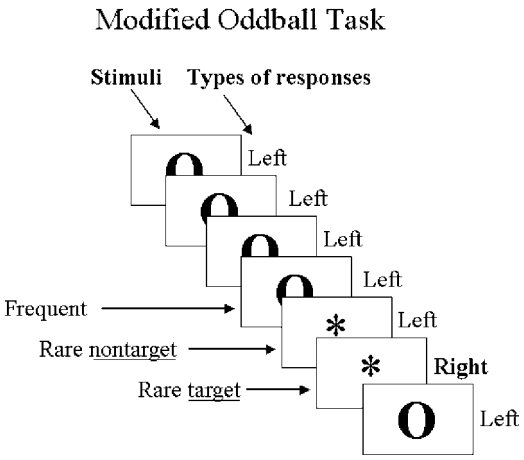

Fig. 1. The response-shift oddball task. The rare stimuli consisted of an asterisk (∗) and the frequent ones, of a circle (O). Participants were told to press one button for both the frequent stimulus and the first rare that appeared following a frequent one (rare nontarget). However, participants had to press the other button whenever another rare stimulus appeared (rare target), until a frequent stimulus reappeared (response shift). No more than three rare stimuli appeared consecutively. The conditions were counterbalanced in terms of hand responses.

Table 3. Mean, Standard Deviation, Median, and Multiple Comparisons of Hit Rates and Reaction Times in Each Condition.

		Control (1)			Asymptomatic (2)			Symptomatic (3)			ANOVA All groups	1–2	1–3	2–3
		M	*SD*	*(Mdn)*	*M*	*SD*	*(Mdn)*	*M*	*SD*	*(Mdn)*				
Rare nontarget	RT (ms)	338	100	(311)	326	76	(315)	458	119	(485)	**	*ns*	*	*ns*
	Hit%	94	7	(96)	92	6	(91)	92	4	(93)	*ns*			
Rare target	RT (ms)	357	46	(340)	337	17	(331)	391	45	(384)	*	*ns*	*ns*	**
	Hit%	87	4	(86)	82	11	(83)	90	3	(90)	*	*ns*	*ns*	*ns*
Frequent	RT (ms)	306	46	(295)	299	19	(303)	359	49	(361)	**	*ns*	*	*ns*
	Hit%	95	6	(97)	95	5	(98)	98	2	(99)	*ns*			

Note. *ns*: nonsignificant.
$^{*}p < .05$.
$^{**}p < .01$.

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immediately followed a frequent one. The latter stimulus, which was labeled 'rare nontarget,' occurred 12.5% of the time. The other button was to be pressed whenever another rare stimulus, labeled 'rare target,' (12.5%) appeared until a frequent stimulus reappeared (see Fig. 1). No more than three rare stimuli appeared consecutively. The total duration of testing, including breaks and practice was approximately 30 min.

These conditions were counterbalanced across participants in terms of hand response. The task thus required careful monitoring not only of the stimulus category but also of the stimulus sequence. The stimuli were black printed characters (100 mm × 100 mm) on a white background using the InStep systems font (cobb_h60.fnt) at the center of the computer screen (coordinates: $x = 0$ and $y = 0$). The stimuli were the letter 'O' (frequent stimuli), or an asterisk '∗' (rare) stimuli. A total of 280 trials (35 rare target, 35 rare nontarget and 210 frequent) were presented in randomized order with a duration of 100 ms and a variable interstimulus interval ranging between 2200 and 2800 ms. Participants were instructed to answer as quickly and as accurately as possible to each stimulus.

The recordings were carried out in a dimly lit faradized room where the participant was seated in an adjustable chair, 90 cm in front of a 19 in. Optiquest Viewsonic SVGA monitor. The recording room

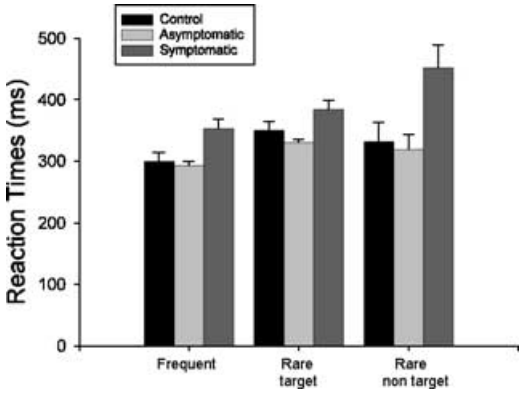

Fig. 2. Mean reaction times (+SE) in response to the frequent, rare target and rare nontarget stimulus presentation. In the control and asymptomatic participants, reaction times were more delayed in response to both rare stimuli than in response to the frequent ones, a result that is in keeping with the standard oddball paradigm. Symptomatic participants generally responded more slowly that the other two groups but they were even slower in response to the rare nontarget stimuli.

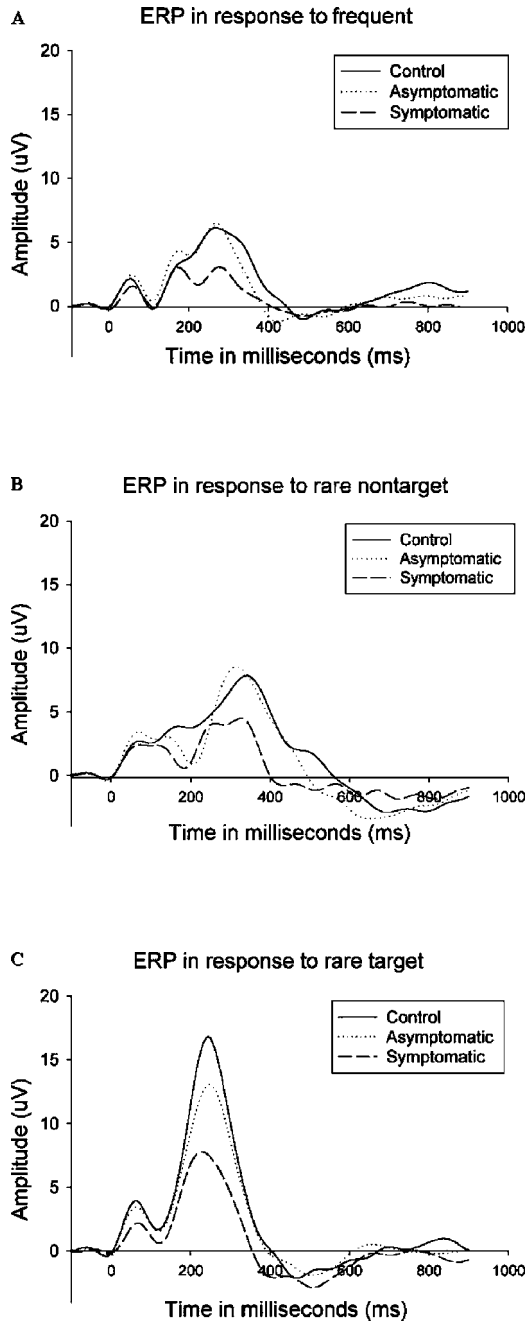

Fig. 3. ERP waveforms comparing the responses of the three groups at Pz to the frequent (Panel A), rare nontarget (Panel B) and rare target (Panel C) stimuli. Although the symptomatic group generally exhibited a reduced P300 amplitude compared to the other two groups, this difference became significant only in response to the rare target stimulus (Panel C).

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constituted a separate corner of a larger room in which the experimenters, amplifiers, and computers were located. Constant visual contact of the participant was achieved by means of a video camera and auditory contact was maintained by a two-way intercom. One of the experimenters was assigned to explain the task to the participant while the other operated the computers. The electrocap (ElectroCap International), electro-oculogram and earlobe references were installed in a session that usually did not exceed 40 min. The height of the monitor was aligned to eye level and the participants were instructed to fix their gaze on a dark cross in the middle of a white background screen waiting for the stimulus to appear.

EEG Recordings and ERP Extraction

EEG was recorded from 28 tin electrodes mounted in an E-Cap (Electro-Cap International Inc.). The electrodes were placed according to the guidelines for standard electrode positions by the American EEG Society (1994) at AF3, AF4, F7, F3, Fz, F4, F8, FC3, FC4, T7, C3, C1, Cz, C2, C4, T8, TP7, CP3, CP4, TP8, P7, P3, Pz, P4, P8, O1, Oz and O2. All electrodes were referenced to linked earlobes and their impedance was kept below 5 KΩ. The Electro-Oculogram (EOG) was recorded using four 9 mm tin external electrodes as well as Fp1 and Fp2 recording sites. The horizontal EOG electrodes were placed at the outer canthus of each eye and the vertical EOG were positioned infra- and

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Table 4. Mean Voltage (Microvolts), Standard Deviation, and Multiple Comparisons of the P300 Amplitude Across Electrode Locations in Response to the Frequent Stimuli.

Electrodes	Controls (1)		Asymptomatic (2)		Symptomatics (3)		ANOVA All groups	Scheffe		
	M	*SD*	*M*	*SD*	*M*	*SD*		1–2	1–3	2–3
AF3	2.48	1.43	2.28	0.92	1.60	0.58	*ns*			
AF4	2.87	1.49	2.70	1.05	1.86	0.97	*ns*			
Fz	5.51	1.97	5.76	1.23	3.01	1.39	**		**	**
F3	4.38	1.70	4.52	0.97	2.84	0.87	**		**	*
F4	5.11	1.72	4.90	1.34	3.06	1.36	**		*	*
F7	1.54	1.23	1.40	0.79	2.00	1.74	*ns*			
F8	2.11	0.70	2.13	1.11	1.88	1.48	*ns*			
FC3	6.02	2.06	6.30	1.96	4.30	1.62	*ns*			
FC4	6.63	2.04	7.27	1.88	4.71	1.89	*		*	
Cz	8.64	2.42	9.71	2.49	5.27	2.16	**		**	*
C1	8.38	2.62	9.11	2.30	5.75	2.35	*		*	
C2	8.53	2.69	9.37	2.04	4.60	4.89	*		*	
C3	6.11	2.19	7.01	1.88	4.47	1.65	*		*	
C4	6.68	1.98	7.67	1.53	3.43	3.91	**		**	*
CP3	6.29	2.21	7.25	1.94	5.15	1.88	*ns*			
CP4	6.98	2.15	7.81	1.61	5.39	1.91	*		**	*
P3	5.64	2.02	6.62	1.50	4.66	1.64	*ns*			
P4	5.96	1.93	6.81	1.38	4.77	1.97	*ns*			
P7	3.35	1.49	4.19	1.89	2.79	1.24	*ns*			
P8	3.28	1.13	4.49	2.41	2.80	1.16	*ns*			
Pz	7.46	1.91	8.30	1.40	5.61	2.04	**		**	
T7	2.56	0.93	2.75	1.16	2.39	1.14	*ns*			
T8	3.33	0.94	3.58	1.18	2.84	1.53	*ns*			
TP7	3.66	1.37	3.64	1.49	3.38	2.24	*ns*			
TP8	3.93	1.32	4.12	1.63	3.01	3.27	*ns*			
Oz	5.39	1.42	7.13	2.10	3.99	1.55	*		**	
O1	5.61	1.77	6.57	2.28	4.61	2.27	*ns*			
O2	5.77	1.93	6.98	2.01	4.94	1.72	*ns*			

Note. *ns*: nonsignificant.
*$p < .05$.
**$p < .05$.

supraorbitally to the left eye, in line with the pupil when looking straight ahead. A bioelectric analog amplifier model ISS3-32BA (SAI – InstEP), amplified the EEG signals (Gain = 3500 for the EOG and 10 000 for the EEG) with a bandpass between 1 and 30 Hz and was digitized continuously at a sampling rate of 250 Hz. The P300 baseline-to-peak amplitudes and latencies were taken in a 250–400 ms time-window.

All EEG epochs related to a voltage exceeding ±100 μV were eliminated from the average ($M = 1\%$). Clippings due to saturation or blocking of the amplifiers were removed from the averaging process ($M = 0.7\%$). Only the epochs related to reaction time trials between 200 and 1000 ms were included in the analyses in order to avoid fast guesses ($M = 0.5\%$) and overly slow responses ($M = 0\%$). No significant group differences were detected on the presence of artefacts.

Statistical Analyses

Group comparisons of demographic and neuropsychological data were computed using a one-way analysis of variance (ANOVA) with group as the between subjects factor (SPSS v7.0 for Windows). Post hoc contrasts were analyzed using Sheffe tests. Mann–Whitney tests were applied to compare the number of previous concussions as well as the severity of the last concussion. Behavioral and P300 data were submitted to an ANOVA with repeated measures. The analysis of the behavioral data (reaction times and accuracy) included one between-group factor (symptomatic/asymptomatic/control) and one within-subject factor (rare nontarget/rare target/frequent). For the psychophysiological data, there was one between-group factor (symptomatic/asymptomatic/control), two within-subject factors (rare nontarget/rare target/frequent and 28

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Table 5. Mean Voltage (Microvolts), Standard Deviation, and Multiple Comparisons of the P300 Amplitude Across Electrode Locations in Response to the Rare Nontarget Stimuli.

Electrodes	Controls (1)		Asymptomatic (2)		Symptomatics (3)		ANOVA All groups	Scheffe		
	M	*SD*	*M*	*SD*	*M*	*SD*		1–2	1–3	2–3
AF3	3.31	1.28	3.52	1.17	2.14	1.86	*ns*			
AF4	3.19	1.38	2.91	0.90	2.87	1.80	*ns*			
Fz	7.04	2.16	6.46	1.34	4.62	3.85	*ns*			
F3	5.76	1.83	5.47	1.60	4.19	3.53	*ns*			
F4	6.16	1.61	5.29	1.24	4.59	2.86	*ns*			
F7	2.07	1.01	2.27	1.61	2.44	2.50	*ns*			
F8	1.92	1.21	2.38	0.80	2.12	1.39	*ns*			
FC3	8.43	2.60	8.23	2.62	5.98	4.25	*ns*			
FC4	8.21	2.33	8.52	2.01	6.36	3.77	*ns*			
Cz	12.16	4.26	11.26	2.99	7.90	5.26	*ns*			
C1	11.68	3.90	10.85	2.45	7.71	5.19	*ns*			
C2	11.24	3.68	11.02	2.65	7.47	4.74	*ns*			
C3	9.81	3.25	9.06	2.48	6.94	4.41	*ns*			
C4	9.10	2.99	9.28	2.59	6.68	3.76	*ns*			
CP3	10.30	3.82	9.96	2.32	7.28	4.38	*ns*			
CP4	9.76	3.30	9.76	2.92	7.01	4.27	*ns*			
P3	10.30	3.86	10.22	2.47	7.20	4.24	*ns*			
P4	9.92	3.14	9.82	3.10	6.72	4.62	*ns*			
P7	8.46	2.99	8.12	1.81	5.91	2.98	*ns*			
P8	7.71	1.89	7.99	1.97	5.13	3.37	*ns*			
Pz	11.49	3.79	10.76	2.72	7.58	4.63	*ns*			
T7	5.77	3.02	5.57	2.56	4.58	3.01	*ns*			
T8	5.40	1.62	5.39	2.24	3.73	2.41	*ns*			
TP7	7.65	2.37	6.93	2.46	5.60	2.80	*ns*			
TP8	6.67	1.80	6.72	2.04	4.65	3.04	*ns*			
Oz	9.49	2.97	10.82	3.26	6.46	4.34	*ns*			
O1	10.42	2.47	10.59	2.86	6.92	4.49	*ns*			
O2	9.75	2.62	10.95	3.78	7.14	4.14	*ns*			

Note. *ns*: nonsignificant.

electrodes). In all analyses, the significance level was set at 5% (two-tailed) with Greenhouse-Geisser corrections for degrees of freedom being used when necessary. In order to examine the influence of time since the last concussion and postconcussive symptom severity on P300 amplitude, two separate linear regression analyses were applied. A first linear regression analysis was used with the PCS scale value as a predictor of the integrity of the P300 amplitude from each electrode. A second multiple linear regression analysis was applied with time since concussion as the predictor of the same factor.

RESULTS

Accuracy and Reaction Times

Mean and median Reaction Times (RTs), as well as percent of hits are reported in Table 3. As statistical analyses carried out on median values as well as on the mean RTs yielded comparable results, only the statistics relevant to mean RT data will be presented. The number of hits was higher than 60% for all participants and was on

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Table 6. Mean Voltage (Microvolts), Standard Deviation, and Multiple Comparisons of the P300 Amplitude Across Electrode Locations in Response to the Rare Target Stimuli.

Electrodes	Controls (1)		Asymptomatic (2)		Symptomatic (3)		ANOVA All groups	Scheffe		
	M	*SD*	*M*	*SD*	*M*	*SD*		1–2	1–3	2–3
AF3	4.49	2.37	5.03	1.48	3.03	1.39	*ns*			
AF4	4.95	1.05	5.74	1.30	3.64	1.62	**			**
Fz	8.90	1.81	9.71	2.42	5.85	1.00	***		**	***
F3	8.26	1.85	8.33	2.23	5.39	1.17	***		**	**
F4	8.14	1.48	9.66	2.76	5.97	1.22	***			***
F7	3.43	1.78	3.95	1.26	3.32	3.01	*ns*			
F8	4.00	1.69	4.35	1.18	2.38	1.05	**		*	*
FC3	11.73	2.55	11.84	2.52	7.49	2.00	***		**	**
FC4	12.71	2.73	13.28	3.76	8.21	1.78	***		**	**
Cz	16.38	3.24	15.61	4.39	10.33	2.76	***		**	**
C1	17.12	3.21	15.71	3.66	9.46	3.13	***		**	**
C2	16.60	3.08	15.94	4.34	9.61	3.01	***		**	**
C3	14.02	3.17	13.41	2.48	8.65	2.54	***		***	**
C4	14.33	2.02	14.05	3.36	9.25	2.05	***		***	**
CP3	15.91	3.17	15.09	2.65	9.35	3.22	***		***	**
CP4	16.45	2.87	15.54	3.73	8.79	6.04	***		**	**
P3	16.15	3.33	14.78	2.72	9.54	3.54	***		**	**
P4	16.11	3.54	14.49	3.27	10.04	3.61	**		***	*
P7	10.46	3.09	10.28	2.43	6.60	2.84	**		*	*
P8	10.90	2.78	9.66	2.59	6.74	2.56	**		**	
Pz	18.47	3.18	16.65	3.50	11.18	3.94	***		***	**
T7	6.54	2.23	7.65	1.41	5.04	2.40	*			*
T8	7.51	1.46	8.31	1.89	5.30	1.57	**		*	**
TP7	8.68	2.13	10.01	3.02	5.18	3.70	**		*	**
TP8	9.99	1.96	9.74	3.07	6.01	2.33	**		**	**
Oz	15.79	4.19	12.26	3.29	8.46	3.75	***		***	
O1	15.47	3.90	12.86	3.89	8.05	3.84	***		**	*
O2	15.38	4.79	13.01	4.03	8.21	4.20	**		**	

Note. *ns*: nonsignificant.
*$p < .05$.
**$p < .05$.
***$p < .005$.

average very high (91%). A significant group effect with regard to hit rate, $F(2, 27) = 3.34$, $p < .05$, was noted during the rare target condition. Post hoc tests, however, failed to reveal any significant differences between paired group comparisons (see Table 3). For all conditions averaged together (frequent, rare nontarget and rare target), symptomatic participants responded significantly more slowly than both asymptomatic and control participants leading to a main group effect, $F(2, 27) = 6.29$, $p < .01$. The symptomatic participants also exhibited a different reaction time profile from the other two groups (Fig. 2). In the control and asymptomatic participants, RTs were more delayed in response to both rare stimuli (target and nontarget) than in response to the frequent one, a result that is in keeping with the standard oddball paradigm. In the symptomatic group, however, RTs were even more delayed in response to the rare nontarget, leading to a significant group × condition interaction, $F(2.48, 33.49) = 3.63$, $p < .05$.

P300 Latency

A significant condition effect was found regarding differences in the P300 latency, $F(1.46, 39.48) = 42.06$, $p < .001$, $\varepsilon = 0.7310$. For all groups, the P300 latency following the rare target stimulus

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Table 7. Summary of the Linear Regression Between Postconcussion Symptoms and the P300 Amplitude at Each Electrode Position.

Electrodes	Frequent			Rare nontarget			Rare target		
	R^2	Slope	Intercept	R^2	Slope	Intercept	R^2	Slope	Intercept
AF3	0.10	2.30	−0.02	**0.23***	3.39	−0.04	0.09	4.50	−0.03
AF4	0.07	2.65	−0.02	0.05	3.15	−0.01	**0.16****	5.12	−0.03
Fz	**0.27****	5.32	−0.06	**0.21***	6.73	−0.07	**0.28****	8.85	−0.07
F3	**0.24****	4.29	−0.04	**0.15***	5.66	−0.05	**0.23****	7.91	−0.06
F4	**0.23****	4.80	−0.05	0.13	5.74	−0.04	**0.24****	8.58	−0.07
F7	0.07	1.46	0.02	0.03	2.10	0.01	0.03	3.38	0.02
F8	0	2.03	0	0.01	2.18	−0.00	**0.22****	3.98	−0.04
FC3	0.1	5.89	−0.04	**0.17***	8.28	−0.07	**0.28****	11.23	−0.09
FC4	0.12	6.62	−0.04	0.11	8.20	−0.05	**0.33****	12.53	−0.12
C1	0.13	8.28	−0.05	**0.18***	11.05	−0.09	**0.31****	15.53	−0.15
C2	0.06	8.03	−0.05	**0.16***	10.80	−0.09	**0.30****	15.45	−0.14
C3	**0.18****	6.35	−0.05	0.12	9.26	−0.06	**0.24****	12.99	−0.1
C4	0.12	6.51	−0.06	**0.13***	8.99	−0.06	**0.36****	13.66	−0.11
CP3	0.04	6.47	−0.02	0.11	9.86	−0.07	**0.28****	14.66	−0.12
CP4	0.08	7.05	−0.03	0.11	9.51	−0.06	**0.20****	14.94	−0.14
Cz	**0.26****	8.7	−0.08	**0.18***	11.47	−0.10	**0.25****	15.28	−0.12
P3	0.11	5.97	−0.03	0.1	9.90	−0.06	**0.26****	14.67	−0.12
P4	**0.16****	6.26	−0.04	0.09	9.43	−0.06	**0.22****	14.63	−0.11
P7	0.05	3.64	−0.02	0.07	7.89	−0.04	**0.13****	9.75	−0.07
P8	0.07	3.78	−0.03	0.06	7.31	−0.03	**0.18****	9.81	−0.07
Pz	**0.25****	7.69	−0.06	0.12	10.69	−0.07	**0.28****	16.77	−0.14
T7	0.01	2.63	−0.01	0.03	5.55	−0.02	0.03	6.63	−0.02
T8	0.05	3.39	−0.02	0.05	5.10	−0.02	**0.33****	7.68	−0.07
TP7	0.05	3.35	0.02	0.04	6.99	−0.02	0.11	8.60	−0.07
TP8	0.02	3.54	0.02	0.02	6.19	−0.01	**0.26****	9.43	−0.09
Oz	**0.25****	6.08	−0.06	0.11	9.62	−0.07	**0.15****	13.17	−0.1
O1	0.01	5.71	−0.01	0.09	9.91	−0.06	**0.17****	13.22	−0.11
O2	0.02	6.04	−0.01	0.07	9.81	−0.05	**0.16****	13.31	−0.11

* $p < .05$.
** $p < .005$.

presentation ($M = 256$ ms) was faster than that observed after appearance of the rare nontarget stimulus ($M = 314$ ms) or the frequent one ($M = 288$ ms). The magnitude of this effect was attenuated in the symptomatic participants but no significant group differences were detected across conditions and regions.

P300 Amplitude

A significant condition effect $F(1.96,\ 52.80) = 100.97$, $p < .001$ was found with regard to differences in the P300 amplitude. The P300 amplitude was greater for the rare target stimulus than for the rare nontarget and the frequent stimuli, respectively, in all groups. Figure 3 shows that the group differences were not significant during presentation of the frequent (Fig. 3A) and rare nontarget (Fig. 3B) stimuli, even if a trend toward depleted amplitude was shown in the symptomatic patients. A significant group difference, however, was observed upon presentation of the rare target stimuli (Fig. 3C). The P300 amplitude was most prominent in controls, but was attenuated in the asymptomatic participants and more so in the symptomatic group, leading to a significant group × condition interaction, $F(3.91,\ 52.80) = 3.39$, $p < .05$, $\varepsilon = 0.9777$.

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Table 8. Summary of the Linear Regression Between Time Since Last Concussion and the P300 Amplitude at Each Electrode Position.

Electrodes	Frequent			Rare nontarget			Rare target		
	R^2	Slope	Intercept	R^2	Slope	Intercept	R^2	Slope	Intercept
AF3	0.01	2.17	−0.01	0	2.99	0	0.03	3.97	0.06
AF4	0.02	2.57	−0.02	0.01	3.06	−0.02	**0.09**	4.5	0.07
Fz	0	4.84	−0.02	0.02	5.81	0.06	0.06	7.79	0.1
F3	0.01	3.99	−0.02	0.01	5.02	0.03	0.01	7.23	0.03
F4	0	4.38	−0.01	0.01	5.22	0.03	**0.15****	**7.35**	**0.15**
F7	0.02	1.76	−0.03	0	2.28	−0.01	0.01	3.41	0.04
F8	0.01	2.09	−0.01	0.1	2.36	−0.06	0.1	3.27	0.08
FC3	0.01	5.65	−0.03	0.01	7.3	0.06	0.03	10.01	0.09
FC4	0.02	6.37	−0.04	0	7.64	0.02	**0.15****	**10.54**	**0.23**
Cz	0.01	8.04	−0.04	0.03	9.93	0.13	0	13.93	0.05
C1	0.02	7.98	−0.06	0.02	9.69	0.1	0.02	13.72	0.1
C2	0.01	7.66	−0.04	0.01	9.67	0.06	0.06	13.35	0.18
C3	0.02	6.05	−0.05	0.05	8.13	0.13	0.01	11.85	0.05
C4	0	6	−0.02	0.01	8.2	0.04	0.03	12.17	0.1
CP3	0.03	6.46	−0.06	0.04	8.74	0.11	0.04	12.96	0.13
CP4	0.05	7	−0.07	0.01	8.64	0.05	0.04	12.97	0.16
P3	0.02	5.79	−0.04	0.04	8.77	0.12	0.01	13.21	0.07
P4	0.03	6.05	−0.05	0.02	8.53	0.08	0	13.47	0.03
P7	0	3.5	−0.01	**0.19****	**6.74**	**0.2**	0.07	8.59	0.02
P8	0.01	3.64	−0.03	0.01	6.74	0.05	0	8.99	0.14
Pz	0.01	7.23	−0.03	0.04	9.43	0.13	0.01	15.21	0.06
T7	0.01	2.62	−0.01	0.07	4.84	0.12	0.08	6.01	0.1
T8	0.01	3.3	−0.01	0.01	4.69	0.04	0.05	6.77	0.07
TP7	0.01	3.66	−0.03	**0.15****	**6.11**	**0.16**	**0.20****	**6.97**	**0.26**
TP8	0	3.72	−0.01	0.02	5.78	0.06	0.09	8.03	0.14
Oz	0	5.51	0	0.02	8.6	0.09	0	12.24	−0.02
O1	0	5.62	−0.01	0.04	8.85	0.12	0.04	11.56	0.15
O2	0.03	6.12	−0.06	0	9.16	0.03	0.02	11.77	0.11

*$p < .05$.
**$p < .005$.

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The stimulus conditions also produced a distinct ERP topographical representation in the three groups of participants depending on the condition used. In the frequent condition, the post hoc contrasts demonstrated that there were some significant differences between control and symptomatic, and also between asymptomatic and symptomatic participants, mainly localized in frontal and central areas (see Table 4). Presentation of the rare nontarget stimuli yielded no significant P300 amplitude differences across groups (see Table 5). By contrast, a significant group × condition × electrodes was found, $F(25.08,\ 338.56) = 1.56$, $p < .05$, $\varepsilon = 0.2322$ regarding P300 amplitude in response to the rare target. The P300 amplitude was strongly attenuated over 28 of the 30 electrode sites during the presentation of the rare target stimuli in the symptomatic participants. A Scheffe's post hoc test applied on the amplitude variables related to the rare target stimuli revealed significant differences between control and symptomatic and also between asymptomatic and symptomatic participants. The comparison between the control and asymptomatic participants did not reveal any significant differences (see Table 6).

Relationships Between Symptoms, Time Elapsed Since Injury and P300 Amplitude

The linear regression analysis between the score obtained on the PCS scale and P300 amplitudes yielded highly significant results at almost all electrode sites (see Table 7). The P300 amplitudes that appeared following rare and frequent stimulus presentations were significantly smaller when the PCS scale indicated a higher level of impairment. In contrast, the linear regression analysis between time since last concussion and P300 amplitudes yielded significant results on only four electrode sites and only during the rare stimulus presentations (Table 8).

DISCUSSION

The present study examined the behavioral and psychophysiological consequences of concussions and assessed whether this event may affect electrophysiological responses in a modified oddball task that requires a higher level of attention than a standard oddball task. Both behavioral and electrophysiological factors were examined.

At the behavioral level, the symptomatic patients performed significantly worse than the asymptomatic and control groups with regard to RT across all conditions but especially in response to the rare nontarget stimuli (or distractors). During that condition, the symptomatic patients RTs were delayed by about 120 ms relative to the other two groups but their hit rates were comparable to those of the other two groups. Indeed, their performance remained at an average level of 82% indicating that they were able to sustain the task and did not respond at random. These data suggest that the symptomatic participants are slower in performing this attentional task suggesting a lack of flexibility in the strategy to categorize a distractor and a target in the oddball sequence. This interpretation is in agreement with neuropsychological studies that have reported deficits in mental flexibility tasks following concussions (Ferland, Ramsay, Engeland, & O'Hara, 1998; McAllister, 1992). These behavioral results are also in accord with other investigations that used a modified auditory oddball task and showed that the behavior of patients with head injury was marked with uncertainty about the correctness of their responses (Campbell & De Lugt, 1995; Solbakk et al., 2000).

The psychophysiological data showed interesting results in relation with the P300 latency and amplitude. The P300 latency refers to the time between stimulus onset and the peak amplitude. It is thought to reflect stimulus evaluation and categorization independently from movement and response selection factors (McCarthy & Donchin, 1981). Our data indicate that with regard to P300 latency, symptomatic patients do not differ from asymptomatic and control participants. This finding is in contrast with other investigations that have shown a delayed P300 in severe (Olbrich et al., 1986) as well as in mild closed head injury (Gaetz & Weinberg, 2000; Reinvang, Nordby, & Nielsen, 2000). However, this result has not been consistently found in other studies, which have only showed group

differences in P300 amplitude (Dupuis et al., 2000; Sangal & Sangal, 1996; Solbakk et al., 2000). Moreover, the P300 latency differences that have occasionally been found between patients with head injury and controls are often smaller than the differences in RT, denoting that mild injury could affect processes related to response selection rather than stimulus categorization (Campbell & De Lugt, 1995).

The oddball task typically elicits a P300 component effect in which the rare stimuli evoke a larger amplitude response than the frequent ones (Picton & Hillyard, 1988). In addition, the P300 generally becomes larger when more effort is being invested and when more attentional resources are recruited (Begleiter, Porgesz, Chou, & Aunon, 1983; Johnson, 1986). In fact, our findings consistently showed that all athletes, including the symptomatic ones, display a classical oddball effect with larger P300 amplitudes to rare than to frequent stimuli and with maximal peak intensity at centro-parietal regions. In addition, the results showed that the rare target stimuli elicited larger P300 amplitude than the rare nontarget, indicating that more attentional resources were recruited to detect the target stimuli. Similar results were found in an analogous study with healthy participants that used a modified oddball task in which the task complexity was enhanced by adding a rare distracting target to the rare and frequent stimuli (Katayama & Polich, 1996). Taken together, the latter findings and ours concur with the interpretation that the P300 amplitude is a measure of limited-capacity attentional resources devoted to stimulus evaluation (Naatanen, 1991; Picton, 1992).

It is noteworthy that it is precisely in the rare target condition that the P300 amplitude was found to distinguish the symptomatic athletes from the other two groups. Whereas no group differences in P300 amplitudes were observed during the rare nontarget presentations, the symptomatic athletes displayed a smaller P300 than the other two groups over almost all electrode sites following the rare stimulus appearance. These findings suggest that concussion impacts on the ability to gather attentional resources, especially when more efforts are needed (i.e., during the rare target condition). These results are somewhat surprising in view of the fact that our patients sustained a very mild brain injury and that most of them did not suffer from a LOC. Nevertheless, our results partially reproduced recent findings, which showed that patients with mild injury have smaller P300 amplitudes during modified (Ford & Khalil, 1996; Reinvang et al., 2000; Solbakk et al., 1999, 2000; Solbakk, Reinvang, & Anderson, 2002) and standard oddball tasks (Gaetz & Weinberg, 2000; Sangal & Sangal, 1996). These studies, however, have indicated a general reduction in P300 amplitude over all conditions. In contrast, although a strong trend toward reduced P300 amplitudes was observed for the symptomatic versus the other two groups regardless of stimulus type, our results demonstrated that the symptomatic group was especially impaired during the processing of the rare target.

One factor that could explain the partial differences between our findings and these earlier investigations might be related to the fact that our symptomatic patients were tested in a more acute phase. Whereas all our symptomatic concussed athletes were examined in an interval varying between 1 week and 6 months postinjury, the selected patients in previous ERP studies were evaluated between 2 and 5 years (Gaetz & Weinberg, 2000), at 3 years (Reinvang et al., 2000), between 4 and 6 years (Solbakk et al., 1999, 2000) or between 6 months and 9 years (Sangal & Sangal, 1996) following their injury. The latter interpretation gains support from the differential results obtained by our symptomatic and asymptomatic participants, the latter having been tested on average 10 months after concussion. Indeed, asymptomatic patients do not appear to carry any significant residual effects of concussion measurable by brain electrical activity. Despite a clear trend toward lower P300 amplitude, the asymptomatic group failed to show any significant differences in P300 amplitude when compared with control participants. Thus, it could be argued that time since injury could be the determining factor in producing electrophysiological anomalies following concussion. However, the regression analysis carried out to determine whether there was a relationship between the reduction in P300 amplitude and time after

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concussion revealed that this relationship was only marginally significant on a few electrode sites. By contrast, significant linear associations between P300 amplitude and symptom severity were found, mainly in the 'rare target' condition. In fact, the P300 amplitude showed a linear negative relationship with the amount of symptoms indicating that the P300 amplitude reductions are not an all-or-none phenomenon, but arise along a continuum with the magnitude of symptoms. Symptom severity therefore appears to be the crucial factor distinguishing our two concussed groups and it may as well account for the partially different pattern of results that was found between our study and previous ones.

One could also argue that our findings could be explained, at least in part, by the presence of depressive symptoms among concussed athletes. Undeniably, a postconcussion syndrome may initially be related to acute cerebral dysfunction but it may also arise as a psychological consequence of head trauma (Mittenberg & Strauman, 2000). P300 amplitude reductions have often been reported in depressed patients (Blackwood et al., 1996; Levit, Sutton, & Zubin, 1973; Pfefferbaum, Wenegrat, Ford, Roth, & Kopell, 1984; Shagass & Roemer, 1992). These results, however, have been challenged by other studies (Kaustio, Partanen, Valkonen-Korhonen, Viinamaki, & Lehtonen, 2002; Kraiuhin et al., 1990; Patterson, Michalewski, & Starr, 1988; Sara et al., 1994; Weir, Fiaschi, & Machin, 1998) that failed to show such an effect. Moreover, the relative independence between distress observed in mild head injury and P300 amplitude has recently been demonstrated (Solbakk et al., 2000), making unlikely an interpretation of our results within a context of depression.

In conclusion, the present study showed, based on an equivalent sample regarding age, gender, educational background, number and severity of concussions, that the P300 component may be more sensitive than standard measurements (neuropsychological, radiological, etc.) to detect the neural impact of a concussion. Earlier studies have often included injured patients from different ages, backgrounds, and types of injuries (i.e., car or work accident) adding variances to the observed results and these methodological differences could explain why certain findings turned out to be nonsignificant. We do, however, have to underline that the clinical significance of the P300 differences shown by the symptomatic athletes is not completely certain: there is as yet no evidence that these differences are associated with neurocognitive changes, even in the acute phase. Moreover, the ultimate duration of the symptoms as well as that of the P300 changes is still unknown but may be relatively short lived. To address this question, a longitudinal study of a symptomatic sample should be carried out in order to assess the time needed to show a complete recovery, optimally with a P300 amplitude matching that observed in athletes who never suffered a concussion.

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APPENDIX

National Hockey League
Neuropsychological Testing Program
Postconcussion Symptoms scale (see also Lovell & Collins, 1998).

Rating

None			Moderate			Severe
0	1	2	3	4	5	6

Name: Code: Date of concussion:

Symptoms	Baseline	Time 2	Time 3	Time 4	Time 5
Dizziness					
Headache					
Nausea					
Vomiting					
Balance problems					
Trouble falling asleep					
Sleeping more than usual					
Drowsiness					
Sensitivity to light					
Sensitivity to noise					
More emotional than usual					
Irritability					
Sadness					
Nervousness					
Numbness or tingling					
Feeling slowed down					
Feeling like in a fog					
Difficulty concentrating					
Difficulty remembering					
Other					
Total score					

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