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Selective effect of closed-head injury on central resource allocation: evidence from dual-task performance

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Abstract Two dual-task experiments are reported bearing on the issue of slower processing time for severe chronic closed-head injury (CHI) patients compared to matched controls. In the first experiment, a classical psychological refractory period (PRP) paradigm was employed, in which two sequential stimuli, a pure tone and a colored dot, were presented at variable stimulus onset asynchronies (SOAs), each associated with a distinct task. The task on the tone required a speeded vocal response based on pitch, and the task on the colored dot required a speeded manual response based on color. In the second experiment, either one or three masked letters was presented, followed by a pure tone at variable SOAs. The task on the letters required a delayed report of the letters at the end of each trial. The task on the tone required an immediate manual response based on pitch. In both experiments, both CHI patients and matched controls reported an SOA-locked slowing of the speeded response to the second stimulus, a PRP effect. The PRP effect was more substantial for CHI patients than for matched controls, suggesting that a component of the slower processing time for CHI patients was related to a selective increase in temporal demands for central processing of the stimuli.

Keywords Closed-head injury · PRP paradigm · Speed of processing

Introduction

One of the most pervasive aspects of the influence of a closed-head injury (CHI) on human performance is related to the response slowing that is commonly reported when CHI patients are required to emit overt responses in conditions of speed pressure (Ponsford and Kinsella 1992; see also Ferraro 1996 for a detailed review).

The reason CHI patients are generally slower than controls in simple speeded (i.e., simple and choice reaction time) tasks is far from clear, and current models offer only limited help on this issue. The theoretical development in this field has ranged from simply assuming the slowing phenomenon as always present following a CHI, to benignly ignoring it in order to focus on higher-level cognitive skills. Generally speaking, however, two broad classes of explanation of the slowing deficit following CHI have been advanced in the neuropsychological literature.¹

The first class of explanation has mainly revolved around the suggestion that the slowing phenomenon is an unspecific phenomenon caused by a generalized impairment affecting all automatic subroutines involved in a speeded task. In this view, the slowing phenomenon would emerge as the by-product of a generalized impairment of the human information-processing system that extends over perceptual, cognitive, and motor stages of processing (Gronwall 1987; Ponsford and Kinsella 1992; Van Zomeren et al. 1984).

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¹ The models discussed hereafter are functional models of CHI effects on behavior. Different explanations, however, have been proposed for the anatomical bases of the CHI slowing. Some, for instance, have argued that processing speed is directly modulated by the characteristics of the synaptic transmission, with uniformly slower synaptic transmissions in CHI patients being associated with either slower information processing or increased rate of information loss at each transmission. Others have raised the impaired functioning of the norepinephrine system as the causal factor for the slowing phenomenon, based on the evidence for massive projections from the norepinephrine system to brain areas (i.e., in the prefrontal cortex) which are hypothesized to play a crucial role in cognitive performance (Foote and Morrison 1987)

The second class of explanation has called on slowed central processing speed as a selective component of the slowing phenomenon. Several studies have shown selective difficulties of CHI patients when performing choice RT tasks compared to simple RT tasks, as well as enhanced CHI sensitivity to manipulations involving the number of response alternatives in speeded tasks, on the assumption that the number of response alternatives directly influences the time taken to carry out central mental operations (e.g., response selection: see Miller 1970; Norman and Swahn 1961; Van Zomeren and Deelman 1976, 1978).

The debate on the general vs specific functional locus of the CHI deficit continues to rage to this day, mainly because of the inconclusiveness of several empirical studies in which CHI performance has been investigated under conditions in which attentional factors were selectively manipulated. Although commonly slower or less accurate in most tasks, it is not well established to date whether CHI patients suffer more than controls when two tasks have to be performed close-to-concurrently. Evidence from these studies varies from close-to-nil dual-task performance differences between CHI patients and controls (Brouwer et al. 1989; Gentilini et al. 1989; Hartman et al. 1992; Riese et al. 1999; Spikman et al. 1996; Veltman et al. 1996), to significant dual-task performance differences observed when CHI patients perform in specific combinations of cognitive tasks (Park et al. 1999; Stablum et al. 1994).

The fact that such differences are evident when CHI patients perform in particular a combination of tasks, but not others, has recently been argued to provide, per se, a hallmark of the localizability, within the cognitive architecture, of the functional impairment caused by CHI, and raised against the generalized impairment hypothesis (Park et al. 1999). Park and colleagues have proposed that dual-task deficits arise in conditions in which dual-task performance requires central executive control over information processed in working memory (see also Azouvi et al. 1996; McDowell et al. 1997; Vilkki et al. 1996). In an elegant demonstration, severe and chronic CHI patients performed a test requiring working memory (i.e., the PASAT test; Spreen and Strauss 1998), either as a single test, or in a dual-task combination with a concurrent test also hypothesized to require working memory (i.e., letter recall). Several aspects of the results of the PASAT test were important. The first result was that there was no difference between CHI patients and controls in the single-task condition. Furthermore, dual-task costs were greater for CHI patients than for controls, in a condition in which the letter recall test was delayed to the trial following the one in which the to-be-recalled letter was presented.

One point of interest in this latter study is that these findings, though robust and of undoubted impact, are ambiguous as to the source of the dual-task interference. As the authors explicitly report in their study (Park et al. 1999, p. 1126), the inherent complexity of the tasks used in their paradigms makes it extremely difficult to in-

dividuate the source of the reported effects. The increased dual-task interference observed in CHI patients may have selectively resulted from difficulties in the encoding and/or retrieval of information from a short-term memory store (e.g., Van der Linden et al. 1992), or difficulties engendered by a cumulative, interfering, effect of prior responses on ongoing working memory activity required for the PASAT test, an interpretation Park and colleagues ultimately favored.

Similar objections to those raised for the Park et al. (1999) work may extend to the majority of dual-task studies that belong to the literature on CHI patients. It is fair to say that researchers studying dual-task performance in controlled environments have investigated a variety of task combinations, whose differentiation into the involved mental processes have been quite complex. At first glance, this is surprising, since the experimental literature on normals (and, in two paradigmatic cases, on split-brain patients; see Ivry et al. 1998; Pashler et al. 1994) is rich with examples in which simpler tasks have been combined in dual-task conditions, and useful insights as to the locus of dual-task interference have been provided. One of these examples, likely the most extensively studied, is the combination of choice reaction times to sequential stimuli that characterize the psychological refractory period (PRP) paradigm.

The PRP paradigm

To date, manipulations of the attentional load in the experimental literature have been implemented in a variety of experimental paradigms, primarily involving multiple-task situations in which several stimuli are presented in succession, and some (at least two) of the stimuli require distinct responses. Generally, in all these situations, the logic has been to manipulate the load by varying the rate at which stimuli are presented on each trial.

As mentioned above, one of the best-known dual-task conditions entails the sequential presentation of two bidimensional stimuli, T_1 and T_2 (usually separated by a stimulus onset asynchrony, or SOA, ranging from 0 to 1 s), each requiring a distinct two-alternative choice reaction time, RT_1 and RT_2 , respectively. What is often reported in this condition is the progressive slowing of the second response, RT_2 , as the SOA between the stimuli is decreased (see Pashler 1994 for a detailed review). This phenomenon, labeled the PRP effect, is consensually taken as a ubiquitous finding when two speeded tasks must be performed close to concurrently, and has been shown to be independent of the sensory modalities in which T_1 and T_2 are presented, and of the combination of motor effectors used to produce the speeded responses (e.g., Pashler 1998). Several accounts have been proposed to explain the PRP effect. With some important exceptions (see Meyer and Kieras 1997), most models call upon the notion of postponement of part of the processing required for the task on T_2 while T_1 is being processed. According to such models, RT_2 in overlapping

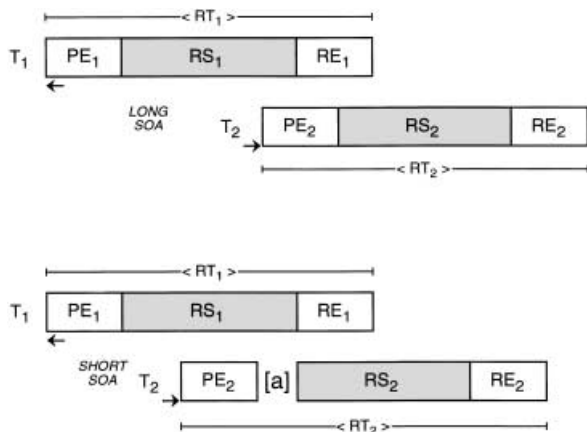


Fig. 1 Stage diagrams showing the postponement-based interaction between speeded tasks in a PRP paradigm (in all diagrams, *PE* perceptual encoding, *RS* response selection, *RE* response execution). RT_1 and RT_2 are estimates of the time taken to respond to T_1 and T_2 , respectively. *Upper diagram* long SOA condition. The two tasks are hypothesized to be carried out in conditions of low central processing overlap for the two speeded tasks. *Lower diagram* short SOA condition. In this condition, the postponement of RS_2 is caused by the impossibility of central mechanisms carrying out RS_1 and RS_2 in parallel. RS_2 can resume only after central mechanisms are no longer occupied with RS_1 . This period of processing suspension for RS_2 is reflected in the prolongation ($[a]$) of RT_2 at short SOA, the PRP effect

task conditions (i.e., at short SOA) would be inflated by a period of waiting in which central, i.e., postperceptual and premotor (Osman and Moore 1993) mechanisms, which cannot be shared across tasks, are busy with T_1 processing. Ancillary to this assumption is that T_2 processing can resume only when these mechanisms are no longer occupied with T_1 processing. Based on recent work on dual-task slowing, one of the most likely candidates for the production of such a functional bottleneck has been hypothesized to be the stage of response selection (McCann and Johnston 1992; Pashler and Johnston 1989; Schubert 1999; Van Selst and Jolicoeur 1997; Van Selst et al. 1999).

A schematic representation of postponement models of the PRP effect is reported in Fig. 1.

Upon T_1 presentation, perceptual encoding of T_1 (PE_1) is assumed to be necessary for the generation of a T_1 identity code. Following perceptual processing, a stage of response selection (RS_1) is required to map the T_1 identity code to the motor code for the appropriate overt response, which is in turn executed as the output of the response execution stage (RE_1). The interval of time elapsing between T_1 presentation and the end of RE_1 is meant to represent the reaction time to T_1 (RT_1). At a certain (relatively long; see upper diagrams) SOA following T_1 , T_2 is presented and T_2 processing, which is assumed to require the same set of stages required for T_1 processing, begins. RT_2 is the estimate of the time elapsed between the presentation of T_2 and the end of RE_2 . Slowing of RT_2 , i.e., the PRP effect, is usually observed at short T_1 - T_2 SOAs. This effect is hypothesized to reflect a specific functional property of the stage of re-

sponse selection (RS_1 and RS_2), i.e., response selection requires processing by central mechanisms, which are sharply capacity limited and operate serially on the incoming signals, T_1 and T_2 . As is clear in Fig. 1, RS_2 is postponed until central mechanisms are freed with RS_1 , and the net result of this functional interaction is that a period of processing suspension simply adds to RT_2 compared to conditions in which central processing overlap does not occur (i.e., at long SOAs). As is indicated in Fig. 1, the temporal length of the period of processing suspension, i.e., an estimate of the amount of PRP effect, may be calculated as the difference between RT_2 at short SOAs and RT_2 at long SOAs ($[a]$ in Fig. 1).

Response selection bottleneck models make a set of very detailed predictions concerning the interaction between SOA and factors selectively affecting the stages of processing involved in the production of a speeded response. Central to the present argument is the prediction related to the effects of perceptual factors affecting T_2 processing at short SOAs. As can be easily inferred from Fig. 1, the effect of factors affecting T_2 perceptual processing (i.e., PE_2) may be absorbed into the period of processing suspension generated by the response selection bottleneck. While the effect of a perceptual difficulty insisting on T_2 would be totally manifested in RT_2 at long SOAs, such an effect will be completely eliminated at short SOAs (i.e., an underadditive interaction between SOA and perceptual difficulty is expected). Underadditive interactions of this kind have been demonstrated in a variety of dual-task situations involving the manipulation of SOA and the perceptual degradation of T_2 (De Jong 1993; Pashler and Johnston 1989, 1998; Van Selst and Johnston 1997).

The present study

The aim of the present study is to provide converging evidence for the notion of a selective involvement of central mechanisms in the cause of the CHI-contingent slowing. To this end, the study follows the tradition of investigating CHI performance in dual-task conditions. Two dual-task experiments were devised in order to provide a direct comparison of the amount of dual-task interference reported by CHI patients and the amount of dual-task interference reported by matched controls, if any. The paradigms in both experiments were designed to minimize, on the one hand, potential sources of perceptual and motor interference and maximize, on the other hand, the involvement of central processing in both tasks. In both experiments, two stimuli were presented in succession through different sensory modalities, and different motor effectors were used to respond to the stimuli, when both stimuli required a speeded response. When a speeded response was required, it always involved a two-alternative discrimination based on an easily detectable physical feature of the stimuli.

In experiment 1, a classic PRP paradigm was employed in which two stimuli, a tone and a color dot, were

presented at variable SOAs, requiring a speeded vocal and a speeded manual response, respectively. The predictions concerning the potential differences between CHI patients' and controls' performance in experiment 1 are derived from the model sketched in Fig. 1.

The first, basic, prediction is based on the widespread evidence mentioned in the "Introduction" for the slower processing speed of CHI patients. Given that two speeded responses must be executed in each trial of experiment 1, we expect CHI patients to be slower than controls in both speeded tasks, i.e., both RT_1 and RT_2 produced by CHI patients should be, on average, longer when compared with the RT_1 and RT_2 produced by controls.

The second prediction refers to the effects of the processing interaction between tasks that can be observed in Fig. 2, where the upper diagrams provide a model of unimpaired performance, and the lower diagrams provide a model of CHI performance according to the slower central processing speed hypothesis. The hypothesis of the slower central processing speed as the causal factor of the slowing phenomenon has been implemented in the lower diagrams of Fig. 2 by prolonging both RS_1 and RS_2 (i.e., the central stages of processing) by a quantity (see areas filled with oblique lines) intended to provide a visualization of the additional time required to carry out central stages of processing on the part of the CHI patients.² According to this hypothesis, a magnified PRP effect is expected for CHI patients compared to controls, because the prolongation of RS_1 for the CHI patients should be totally reflected in an increased processing suspension time before RS_2 resumption. In Fig. 2, this prediction emerges as straightforward if one compares the length of RS_2 processing suspension for controls ([a], in the upper diagrams) with the length of RS_2 processing suspension for CHI patients ([b], in the lower diagrams). As the literature suggests, and the models sketched in Figs. 1 and 2 explicitly predict, no SOA effects are expected on T_1 performance.

The slower central processing speed hypothesis makes a further prediction concerning a comparison between CHI patients and controls yielded by the PRP model in Fig. 2. The rationale underlying the comparison is the following. Suppose we treat the absolute difference in RT_1 between CHI patients and matched controls as the estimate of the effect of a CHI deficit on an intact human processing system. As is clear by comparing the upper and lower diagrams in Fig. 2, such an estimate should equal the length of the additional RS_1 processing

² The areas filled with oblique lines have the same extension across the stage diagrams that refer to CHI patients, meaning that a CHI deficit has equivalent effects on mental operations (i.e., RS_1 and RS_2) that are assumed to be functionally similar. Specifically, both RS_1 and RS_2 are mapping operations performed on abstract perceptual codes (derived from T_1 and T_2) to abstract motor programs through the application of context-based stimulus-response translation rules (e.g., McCann and Johnston 1992). Note, however, that the argument proposed in the present section of the article applies also to a scenario in which different amounts of CHI deficits are assumed for the task on T_1 and the task on T_2

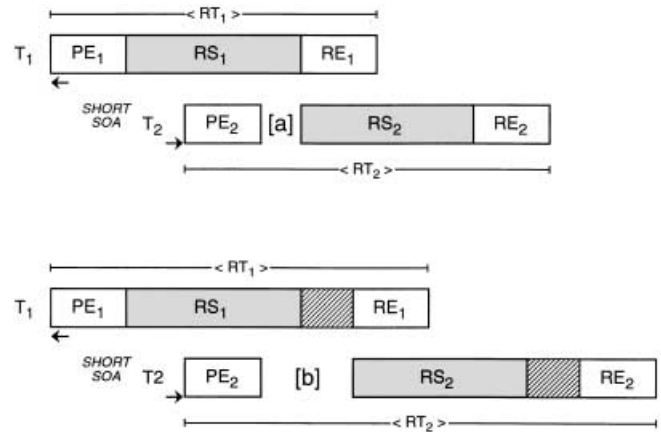


Fig. 2 Stage diagrams showing the hypothesized effects of CHI on dual-task performance. All diagrams show the critical condition of high temporal overlap (i.e., short SOA) between the tasks. *Upper diagram* predicted functional interaction for controls; *lower diagram* predicted functional interaction for CHI patients. As emerges from the comparison between the diagrams, a longer period of processing suspension (hence, a more pronounced PRP effect) is expected for CHI patients than for controls

time represented by the area filled with oblique lines. It is important to note that the estimate based on RT_1 constitutes one of the two possible estimates of the CHI deficit, since a second estimate can be algebraically derived, according to the model reported in Fig. 2, from RT_2 . The estimate based on RT_2 can be derived by subtracting the quantity [a] in Fig. 2 from the quantity [b], that is, by subtracting the amount of PRP effect for controls from the amount of PRP effect for CHI patients. On the assumption that slower processing speed in CHI patients is totally accounted for by a selective prolongation of central processing time required for a speeded task, these two independent estimates should not differ, because a temporal increase in central processing for RT_1 is assumed to carry over, millisecond for millisecond, to RT_2 at short SOAs. Formally, the predictions can be stated as:

$$RT_{1(\text{CHI})} - RT_{1(\text{Controls})} = PRP_{(\text{CHI})} - PRP_{(\text{Controls})}, \quad (1)$$

where $RT_{1(\text{CHI})}$ and $RT_{1(\text{Controls})}$ are the expected speed of response to T_1 for CHI patients and controls, respectively, and $PRP_{(\text{CHI})}$ and $PRP_{(\text{Controls})}$ are the expected amounts of PRP effect for CHI patients and controls, respectively.

Although the temporary exclusion of perceptual stages of processing (i.e., PE_1 and PE_2) as candidate loci for the slowing phenomenon may seem arbitrary, we give hints of the potency of the above prediction by presenting here a formal argument on the effects of a CHI-contingent (selective) perceptual impairment in a PRP paradigm. This prediction is sketched in Fig. 3, where both PE_1 and PE_2 in the stage diagrams that refer to CHI patients (lower diagrams) have been prolonged by an amount intended to represent increased perceptual processing demands for CHI patients. It is evident that, under the short SOAs conditions reported in Fig. 3, the effects of the prolongation of PE_2 are subject to absorption into the period of

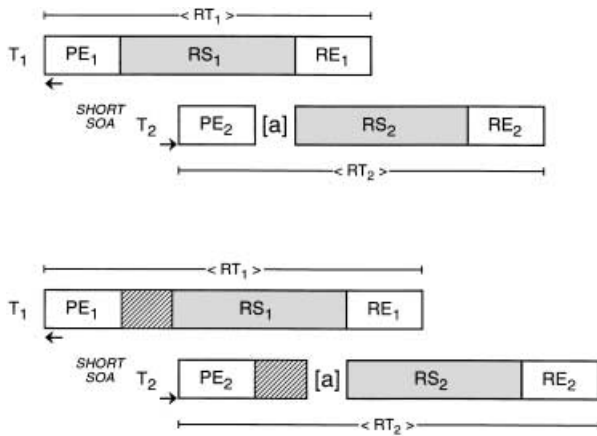


Fig. 3 Stage diagrams showing an alternative view on the effects of CHI on dual-task performance. Contrary to the hypothesized central source of the slowing phenomenon, perceptual stages of processing are assumed to be disrupted following CHI in the present model. All diagrams show the critical condition of high temporal overlap (i.e., short SOA) between the tasks. *Upper diagram* predicted functional interaction for controls; *lower diagram* predicted functional interaction for CHI patients. As emerges from the comparison between the diagrams, equivalent periods of processing suspensions (hence, same amount of PRP effect) are expected for CHI patients and controls

processing suspension created by RS_2 postponement. By comparing the two [a] quantities in Fig. 3, one can immediately see that, in this view, comparable amounts of PRP effects are expected between CHI patients and controls when a perceptual source of dual-task interference is postulated. Of note, comparable amounts of PRP effects in the two tested groups of subjects would render ineffective the prediction expressed in Eq. 1, in case a difference in RT_1 between CHI patients and controls was found.

The final prediction is that, under conditions of high temporal overlap between the speeded tasks (i.e., at short SOAs), RT_1 and RT_2 for both patients and controls should be positively correlated, that is, RT_2 should be shorter when a short RT_1 is observed than when a long RT_1 is observed on the same trial. This prediction is based on the notion that a great part of the variability associated with a choice reaction time is generated at a

central stage of processing (e.g., Pashler and Johnston 1989, 1998). It may reasonably be assumed that a short RT_1 is associated with a short duration of the stage of response selection for T_1 , whereas a long RT_1 is associated with a long duration of the stage of response selection for T_1 . In this view, at short SOAs, short RT_2 s are expected with short RT_1 s because response selection for T_2 can resume earlier in these conditions with respect to conditions in which long RT_1 s are observed.

Experiment 1

Method

Subjects

A group of nine CHI patients (two men and seven women) and a group of nine uninjured controls were the subjects of the experiments. Demographic and clinical data for the patients are reported in Table 1.

The CHI group was selected from referrals at the Seregno Hospital using the following criteria: definite evidence of an acceleration-deceleration CHI (all patients had CHI as a result of road accidents), no use of drugs or medicines, no residual visual or motor deficit, no obvious reason for non-return to work, not seeking financial compensation for the injury, not pursuing litigation, and severe CHI with Glasgow Coma Scale (GCS; Jennett and Bond 1975; Jennett et al. 1981) scores between 3 and 8 on admission in the rehabilitation unit (between 2 and 6 months after trauma). The post-traumatic amnesia (PTA) was estimated using the GOAT scale (Levin and Grossman 1979). PTA duration was assessed by interviewing patients and relatives, and patients with a PTA duration of not less than 7 days were considered as candidates during the selection process. Patients with a history of previous head injury were excluded because studies of patients sustaining successive head injury have demonstrated cumulative effects of the head trauma following successive insults (e.g., Gronwall and Whrightson 1975). CHI and control subjects with a history of alcoholism, psychiatric disorder, mental retardation, or neurological disease were also excluded. Magnetic reso-

Table 1 Demographic and clinical features in the CHI group [N arbitrary numbers assigned to CHI patients, GCS Glasgow Coma Scale scores (on admission in the rehabilitation unit), PTA post-traumatic amnesia (GOAT scores), $Time$ time-postinjury interval]

N	Sex	Age (years)	Education (years)	GCS	Coma duration (days)	Lesions	PTA (days)	Time (months)
1	F	21	13	3	7	Left frontal and right temporal	28	6
2	F	36	8	6	7	Bilateral frontal	7	28
3	M	26	17	8	15	Left temporal	26	12
4	F	20	10	4	13	Bilateral frontal, right temporal and left parietal	30	41
5	M	25	16	5	40	Bilateral frontotemporal and right parietal	50	33
6	F	34	13	5	15	Frontal and diffuse axonal injury	25	5
7	F	25	13	4	5	Left temporal	34	9
8	F	50	5	6	6	Diffuse axonal injury	8	5
9	F	34	13	8	5	Left frontotemporal	15	12

nance imaging (MRI) was carried out for three patients only (nos. 6, 8, and 9), with two of the patients (i.e., nos. 6 and 8) reporting diffuse axonal injury. No further MRI investigations were carried out in the rehabilitation phase because of the lack of clinical or therapeutic indications to perform them.

The selected patients underwent a traditional neuropsychological assessment. Memory was assessed by Corsi Block Tapping and Story Recall. Attention was assessed by Forward and Backward Digit Span and Attentive Matrices. Executive functions were assessed by Phonemic and Categorical Verbal Fluency. All patients showed Equivalent Points (Spinnler and Tognoni 1987) in the normal range as assessed by the administration of Raven's Progressive Matrices (A, B form). No abnormalities were found in the WAIS subtests assessing reasoning and concept formation skills. All tests were performed according to standard published protocols or established procedures. The only indication of an impairment was found for patient 4 on Phonemic Verbal Fluency, and for patient 6 on Story Recall. Despite the apparently good recovery, all the patients continued to have broadly defined complaints, such as difficulty of concentration, fatigue, irritability, and difficulty in performing tasks at the same level as they did before trauma. Furthermore, five of them were still not able to resume work or study.

The mean age and education of CHI patients were 30.11 years ($SD=9.45$, range 20–50 years) and 12 years ($SD=3.77$, range 5–17 years), respectively. The mean Glasgow Coma Scale score on hospital admission was 5.44 ($SD=1.74$, range 3–8), and the mean coma duration was 12.55 days ($SD=11.09$, range 5–40 days). All CHI patients were tested between 5 and 41 months after injury ($M=16.77$, $SD=13.58$). The control group was matched for sex, age ($M=29.44$, $SD=8.71$), and years of education ($M=12$, $SD=3.77$). Controls and CHI patients did not show significant differences in any of these variables (for age: $t(16)=0.156$, $P=0.88$; for education: $t(16)=0.001$, $P=1.0$).

All the subjects (CHI patients and controls) were right-handed, naive to the specific purpose of the experiments, had normal or corrected-to-normal vision, and gave informed consent.

Visual stimuli

The visual stimuli were circular patterns, with a diameter of 1.7 degrees of visual angle, filled with blue color (CIE coordinates: $Y=14.6$, $x=0.266$, $y=0.269$) or red color (CIE coordinates: $Y=18.4$, $x=0.357$, $y=0.339$). The visual stimuli were displayed on an SVGA computer screen (cathode ray tube) controlled by a 586 CPU.

Auditory stimuli

The auditory stimuli were pure tones, presented for 100 ms, with a frequency of 400 or 1200 Hz. The auditory stimuli were presented through the speakers of the com-

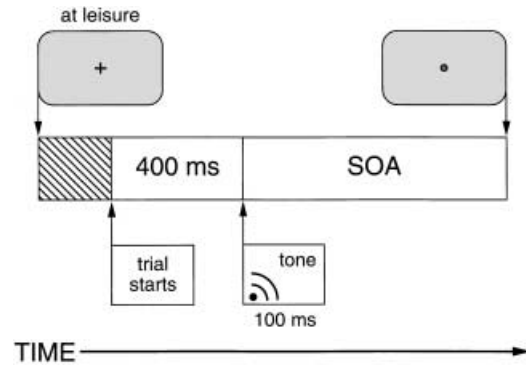


Fig. 4 Schematic representation of the sequence of events in each trial of experiment 1

puter, with the volume set to be always clearly audible (50 dB).

Procedure

Experiment 1 was carried out in a dimly lit, sound-attenuated room, in the constant presence of a research assistant who paced the trial presentation, and controlled the length of the practice phase (see below) before data recording. On each trial of experiment 1, an auditory stimulus (T_1) and a visual stimulus (T_2) were presented in succession, with each stimulus requiring a distinct speeded response, RT_1 and RT_2 , respectively. T_1 was always presented as the first stimulus, followed by T_2 . From written instructions presented on the monitor at the beginning of the experiment, the order of the speeded responses must conform to the order of stimuli arrival, that is, the response to T_1 always had to be emitted before the response to T_2 . The sequence of events in each trial of the present experiment is schematized in Fig. 4.

Each trial began with the presentation of a fixation cross at the center of the monitor. An experimenter initiated the trial by pressing the “+” key on the numeric keypad. After the key press, the fixation cross disappeared, and a fixed blank interval of 400 ms elapsed before the presentation of T_1 . Participants were required to make a first immediate two-alternative forced choice response based on the tone pitch. Using a microphone placed in front of their mouth, at a distance of about 5 cm, participants had to say “high” if the pitch of the tone was high, or “low” if the pitch of the tone was low, while avoiding noise (e.g., coughing) or hesitations (e.g., “hum...”). The task with the tone was defined as the primary task, and both speed and accuracy were emphasized in the instructions. At one of three possible SOAs (either 350, 900, or 1550 ms) following T_1 , T_2 was displayed on the monitor. With a 0.5 probability for each trial, T_2 could be either red or blue. Participants were instructed to make a second two-alternative forced response based on the color of T_2 . The hand-color mapping was varied every two participants, with ten participants (five CHI patients and five controls) pressing the

“Z” key when T_2 was blue, and the “M” key when T_2 was red, and eight (four CHI patients and four controls) participants responding with the opposite mapping. Participants were instructed to keep the index fingers of both their hands on the appropriate response-keys, and were encouraged to also perform the task on T_2 as fast and accurately as they could.

Two distinct sessions preceded the data recording session. In a first session, the microphone sensitivity was set according to each participant’s vocal characteristics. A sequence of ten tones with frequencies of either 400 Hz or 1200 Hz was presented to each participant. The participants were instructed to say, as fast and accurately as possible, “high” if the tone was high pitched, or “low” if the tone was low pitched. The sequence of tones was repeated in case one or more failures to detect the vocal response occurred. On each repetition, the sensitivity threshold of the microphone was lowered by a factor scale of 2/30. The second session was dedicated to practice for the actual experiment. Participants performed a varying number of blocks (from 4 to 6) of 12 trials each. At the end of the practice session, the instructions were repeated, and participants performed 6 blocks of 36 trials each. In each block of trials, levels of SOA, T_1 pitch, and T_2 color were fully randomized, within the constraints of having no more than three equal pairs of responses (i.e., same combination of a T_1 response and a T_2 response) on consecutive trials.

Results

The analyses concentrated on correct RT_1 s and correct RT_2 s, and on the error rates in each speeded task. Correct RTs in each task were screened for outliers using the procedure described by Van Selst and Jolicoeur (1994³). When one outlier (or an error) was found in one or both tasks, the entire trial was excluded from further analysis. The application of the outlier elimination procedure on the present data set resulted in a total loss of 2.6% of correct RTs.⁴ The results from both tasks were analyzed using ANOVA, in which group (CHI patients vs controls) was treated as a between-subject variable, and SOA (350 vs 900 vs 1550 ms) as a within-subject vari-

³ For each task, the data in each cell of the present design were sorted, and the most extreme observation was temporarily excluded from consideration. The mean (M) and standard deviation (SD) of the remaining data points were then computed. Cutoff values were established using the following equations:

$$V_{low} = M - C \times SD \quad V_{high} = M + C \times SD,$$

where C was a parameter that depends on sample size (see Van Selst and Jolicoeur 1994), such that the final estimate of sample means was not influenced by sample size. The smallest and largest observations were then checked against the cutoff values, and treated as outliers if one or both of these data points were outside the bounds. If an outlier was found, then the algorithm was applied anew to the remaining data points

⁴ All the analyses reported in the present article were also carried out with the inclusion of the outlier RTs, and the pattern of results did not change

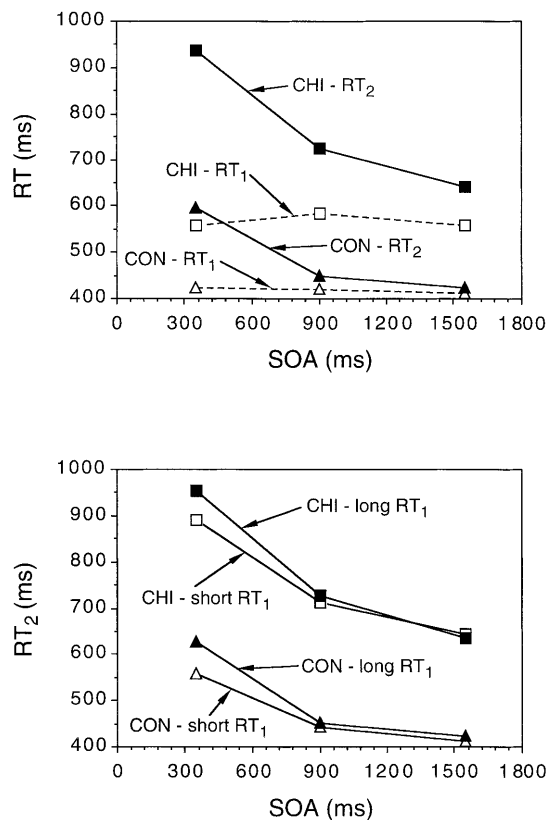


Fig. 5 Summary of the results in experiment 1. *Upper panel* mean RTs in the task on T_1 (dashed lines and empty symbols: RT_1) and mean RTs in the task on T_2 (solid lines and filled symbols: RT_2), as a function of SOA, and as a function of group (CHI CHI patients, CON controls). *Lower panel* mean RTs in the task on T_2 conditionalized on the speed of performing the task on T_1 (short RT_1 vs long RT_1), plotted as a function of group

able. The probability threshold selected for factor significance was $P=0.05$.

Responses to T_1

A summary of the results from the task on T_1 is reported in Fig. 5 (upper panel, dashed lines). Mean RT_1 for patients and mean RT_1 for controls were 560 ms and 418 ms, respectively. This 142-ms difference was statistically significant ($F_{(1,16)}=9.8$, $MSe=30617$, $P<0.001$). No other factor reached the significance level in the analysis carried out on RT_1 ($F_s < 1.3$, $P_s > 0.25$).

The mean error rate in the task on T_1 was 2% for patients and 2% for controls. No factor was significant in the analysis carried out on error rate ($F_s < 1$, $P_s > 0.30$).

Responses to T_2

Mean RTs in the task on T_2 , as a function of group, and as a function of SOA, are reported in Fig. 5 (upper panel, solid lines). CHI patients were, on average, 279 ms slower than controls in the task on T_2 . This produced a

significant main effect of group ($F_{(1,16)}=9.2$, $MSe=114083$, $P<0.008$). There was a sizable SOA effect, with a progressive RT_2 increase from 533 ms to 767 ms as SOA was decreased ($F_{(2,32)}=172.0$, $MSe=1565$, $P<0.001$), a PRP effect. The effects of the interaction between SOA and group were significant ($F_{(2,32)}=11.4$, $MSe=1565$, $P<0.0015$). As Fig. 5 suggests, SOA effects on patients' performance were more pronounced than SOA effects on controls' performance. The amount of PRP effect, that is, the RT_2 difference from the longest SOA to the shortest SOA, was 298 ms for patients, and 162 ms for controls.

In order to provide a test of the prediction concerning the comparison between the amount of PRP effect for CHI patients and the amount of PRP effect for controls (see Eq. 1), the difference in speed in carrying out the task on T_1 , for each CHI patient and associate control, was calculated. The mean RT_1 for each control participant was subtracted from the mean RT_1 of the associate CHI patient. Next, we calculated the difference in the amount of PRP between CHI patients and controls. The amount of PRP effect reported by each control participant was subtracted from the amount of PRP effect reported by the associate CHI patient. An ANOVA was carried out on this newly generated data set, by treating the independent two-level factor "RT₁ slowing vs PRP effect" as a within-subject factor. A null difference between the two estimates considered in the present test was found ($F<1$, $P>0.8$).

The RT_{2s} were further analyzed as a function of the speed in carrying out the task on T_1 . To this end, the data from the task on T_2 , in each cell of the present experimental design and for each participant, were subdivided into trials associated with above-median RT_{1s} and trials associated with below-median RT_{1s} , and submitted to the outlier elimination procedure. In Fig. 5 (lower panel), the RT_{2s} for each group are shown conditionalized on the speed in carrying out the task on T_1 . RT_2 was generally shorter with a shorter RT_1 than with a longer RT_1 ($F_{(1,16)}=7.0$, $MSe=2866$, $P<0.02$). Furthermore, this effect, for both patients and controls, was almost entirely confined to the shortest SOA ($F_{(2,32)}=7.2$, $MSe=1371$, $P<0.003$).

A separate analysis was carried out on the data from the group of CHI patients in order to assess possible effects of the significant variability in the time-postinjury (see Table 1) on RT_2 across the different SOAs. To do so, a subgroup of CHI patients was created by including the four patients who were associated with a time-postinjury shorter than 12 months (nos. 1, 6, 7, 8). The other subgroup included the remaining patients associated with a time-postinjury of 12 months or longer (nos. 2, 3, 4, 5, 9). Both the global speed of performance in Task₂ and

SOA effects were comparable across these two subgroups. The effect of the time-postinjury was significant neither as a main effect ($F_{(1,7)}=1.3$, $P>0.26$), nor in the interaction with SOA ($F<1$).

The mean error rate in the task on T_2 was 6% for patients and 9% for controls. Neither the group nor the SOA factors produced significant main effects. The effects of their interaction, however, were significant ($F_{(2,32)}=3.7$, $MSe=0.0007$, $P<0.04$). The interaction was produced by the fact that patients' performance was stable across SOAs, while a modest decrease in accuracy was observed in controls' performance as SOA was increased. When the results from the different groups were separately analyzed, SOA effects on patients' performance were in fact null (from the shortest to the longest SOA: 6%, 7%, 6%; $F<1$, $P>0.4$), while significant SOA effects were observed in controls' performance (from the shortest to the longest SOA: 7%, 9%, 11%; $F_{(2,16)}=4.2$, $MSe=0.0010$, $P<0.05$).

Discussion

The results of experiment 1 were clear cut, and adhered nicely to the set of predictions put forward on the basis of the hypothesized central source of the CHI-contingent slowing phenomenon. Both RT_1 and RT_2 produced by CHI patients in the present PRP paradigm were, on average, longer than RT_1 and RT_2 produced by controls. A more marked PRP effect was found for CHI patients than for controls. The two independent estimates of the prolongation of time devoted to carry out response selection on the part of the CHI patients did not differ significantly. RT_1 and RT_2 for both CHI patients and controls were positively correlated at the shortest SOA, with shorter RT_{2s} observed following shorter RT_{1s} and longer RT_{2s} observed following longer RT_{1s} .

We interpret the present pattern of results as support for the hypothesis of a selective effect of CHI on human performance. In our view, the present findings fit the proposal that central stages of processing are selectively affected by CHI, in terms of increased time demands for response selection operations in speeded tasks. According to the postponement logic proposed in the present context, the more substantial PRP effect reported by CHI patients, together with evidence of millisecond for millisecond carryover effects of increased central processing time in RT_1 on RT_2 , make it extremely difficult to attribute even part of the CHI deficit to increased perceptual difficulties for the CHI patients compared to matched controls.

One particular aspect related to the paradigm employed in experiment 1 deserves further comment. De Jong (1993) has recently hypothesized that a bottleneck at a motor stage of response initiation, in conjunction with a bottleneck at response selection, may be effective in reducing the second response speed at short SOAs in dual-task paradigms. The hypothesized presence of a bottleneck at a motor level of processing in dual-task

⁵ In a covariate analysis (ANCOVA) carried out on RT_{2s} , mean RT_{1s} for CHI patients and controls were used as a covariate to evaluate the effects of the interaction between group and SOA once absolute differences in RT_1 performance were partialled out. Although statistically attenuated, the interaction was still significant ($F_{(2,30)}=4.8$, $MSe=1458$, $P<0.02$)

conditions would have obvious consequences on the interpretation of the functional impairment following CHI. In our view, the CHI impairment is “pure,” to the extent that we hypothesize an exclusive implication of central mechanisms in the cause of the slowing phenomenon. The central processing postponement account advocated hitherto, however, allows further tests to be provided of the central (as opposed to motor) processing involvement in the generation of the slowing phenomenon. From this perspective, suppose we devise a combination of tasks in the context of a dual-task paradigm, in which the first task requires central processing, but does not involve a speeded response, and the second task requires both central processing and a speeded response. If any motor-related form of interference (i.e., related to the specific requirement to initiate two speeded responses) was responsible for the difference in performance between CHI patients and controls observed in experiment 1, then an overall similarity in performance between CHI patients and controls should be observed in these conditions. This line of argument will be developed more fully in the introduction to experiment 2.

Experiment 2

Jolicoeur et al. (2000) and Dell’Acqua and Jolicoeur (2000) have argued that response selection may be conceived of as one member of the class of mental operations characterized by central processing demands. Processing by central mechanisms has also been shown to be required for the short-term consolidation of visual information (Jolicoeur and Dell’Acqua 1998). Short-term consolidation is a central operation hypothesized to be involved when briefly presented information is to be encoded in short-term memory for later, unspeeded, recall. Evidence for this hypothesis has been produced by presenting, on each of several trials, to-be-encoded visual information as the first stimulus (i.e., in T_1), and auditory information, a pure tone with two possible frequencies, as the second stimulus (i.e., in T_2). The task on T_1 was to report, with no speed pressure, the visual information presented in T_1 (e.g., letter recall) at the end of each trial, and the task on T_2 required an immediate, speeded, two-alternative discrimination based on T_2 pitch. Two factors were independently manipulated in this experiment. A variable temporal interval (or SOA) separated the presentation of T_1 and T_2 . With a 0.5 probability on each trial, either one or three to-be-recalled letters were displayed. The results showed a PRP effect in the task on T_2 , that is, the reaction time to the tone increased monotonically as the T_1 – T_2 SOA was decreased. Furthermore, PRP effects were more pronounced when three letters were displayed in T_1 than when one letter was displayed in T_1 . Although letter recall was less accurate when three letters were displayed than when one letter was displayed, the SOA manipulation had no effect on memory performance in the delayed recall of T_1 . Given that ample time was provided for perceptual pro-

cessing of the letters (e.g., Coltheart 1980), and motor-related interference was ruled out by the requirement to emit a single speeded response in this paradigm, Jolicoeur and Dell’Acqua (1998) concluded that short-term consolidation of visual information required central processing. On the assumption that, while central processing required to carry out short-term consolidation is under way, other operations with equivalent functional demands must wait, Jolicoeur and Dell’Acqua (1998) proposed that, at short SOAs, the short-term consolidation of T_1 postponed response selection for the speeded task on T_2 . That is, a PRP effect was generated by a functional interaction similar to the one illustrated in Fig. 1. The time taken to consolidate T_1 was hypothesized to be proportional to the amount of to-be-recalled information in T_1 . In this view, a longer period of processing suspension for T_2 response selection (i.e., a greater PRP effect) occurred when three letters had to be consolidated than when only one letter had to be consolidated.

The same paradigm as that just described was employed in the present context of investigation, because Jolicoeur and Dell’Acqua’s (1998) model yields straightforward predictions about the potential differences between CHI patients and controls in these particular dual-task conditions, while avoiding the response initiation confound pointed out in experiment 1. As for T_1 , memory for to-be-recalled information presented in T_1 should be minimally, if at all, affected by the tasks’ temporal overlap, although, as in Jolicoeur and Dell’Acqua’s (1998) experiments, T_1 recall performance should be less accurate the more letters are presented in T_1 . Performance on T_1 may also be critical for supporting the assumption made in experiment 1 that perceptual processing was preserved in the present group of CHI patients. If both perceptual processing of the letters presented (for 250 ms and masked) in T_1 and maintenance of the letters in short-term memory are preserved mental operations in CHI patients, then letter recall performance should be comparable between CHI patients and controls. Since the preservation of short-term storage capacity is suggested by the results of the neuropsychological assessment reported in a previous section of this work, any deviations of CHI performance from controls’ performance may be taken as evidence for a CHI-contingent perceptual deficit.

As for T_2 , and as pointed out in the “Introduction,” we expect generally longer reaction time to T_2 (i.e., RT_2) for CHI patients than for controls. Furthermore, we expect the manipulation of the SOA to have a stronger impact on CHI patients’ performance than on controls’ performance. This is because, as hypothesized on the basis on the results of experiment 1, mental operations requiring central processing are subject to increased time demands in CHI patients with comparison to controls. For the same reason, we also expect, at short SOAs (i.e., in conditions of high central processing overlap between the tasks), CHI patients to be more sensitive to the manipulation of the number of to-be-recalled letters. That is, since the number of letters is hypothesized to primari-

ly modulate the time taken for central processing (i.e., with the time to consolidate three letters hypothesized to be longer than the time to consolidate one letter), we expect CHI patients to show, at short SOAs, a difference in RT_2 between the one-letter condition and the three-letter condition to be more pronounced than the difference in RT_2 shown by controls.

Method

Subjects

The subjects were the same subjects who performed in experiment 1. One CHI patient (no. 8) and the matched control subject did not participate in experiment 2. Half of the subjects (CHI patients and matched controls) performed in the present experiment 2 before performing in experiment 1. For the other half of the subjects, the order of the experiments was reversed.

Visual stimuli

The visual stimuli were black letters (all except A and Z) presented on the light gray background (28 cd/m^2) of an SVGA monitor (cathode ray tube), controlled by a 586 CPU. The letters were presented at the center of the computer screen, and each letter subtended 0.85 degrees of visual angle (height) \times 0.8 degrees of visual angle (width), at a distance of about 60 cm set by a headrest. When more than one letter was shown, the letters were arrayed horizontally and the space between adjacent letters was 0.1 degree of visual angle. At display offset, each letter was masked by superimposed "O" and "\$" characters. The letters were randomly selected, without replacement, from the set of available letters on each trial.

Auditory stimuli

The same auditory stimuli as those employed in experiment 1 were used in experiment 2.

Procedure

Experiment 2 was carried out in a dimly lit, sound-attenuated room, in the constant presence of a research assistant who paced the trial presentation, and controlled the length of the practice phase before data recording. A visual representation of the sequence of events on each trial of experiment 2 is reported in Fig. 6. In each trial, two stimuli, T_1 and T_2 , were presented in succession, and each stimulus was associated with a distinct task.

The first stimulus, T_1 , was a visual stimulus, and the second stimulus was an auditory stimulus. Each trial began with the presentation of a message at the center of

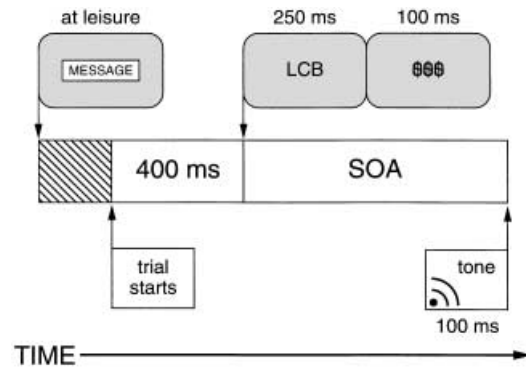


Fig. 6 Schematic representation of the sequence of events in each trial of experiment 2

the screen indicating the number of letters to-be-displayed on that trial ("one letter" or "three letters"). Each trial was initiated by the experimenter by pressing the "+" key of the numeric keypad of the computer keyboard. The message at that point disappeared and, after a fixed delay of 400 ms, one or three letters (always corresponding to the number reported in the initial message) were displayed for 250 ms, followed by a 100-ms mask. The task associated with the letters was to remember the letters and recall them, with no speed pressure and with no regard of the order in which letters were arrayed on the screen, at the end of each trial.

At varying SOAs (350, 900, or 1550 ms) following T_1 , T_2 was presented and participants were instructed to make an immediate two-alternative forced choice response based on T_2 pitch, by pressing the "M" key of the keyboard with the index finger of their right hand if the pitch of T_2 was high (1200 Hz), or the "Z" key with the index finger of their left hand if the pitch of T_2 was low (400 Hz). Participants were instructed to constantly keep their index fingers on the "M" and "Z" keys during the entire experiment, and to respond to the tone as fast as possible, while keeping errors to a minimum. After the execution of the speeded tone response, participants had to type on the keyboard the remembered letters, guessing when uncertain. Participants always had to type in as many letters as those they were presented with. Participants performed a varying number of practice trials before the data-recording phase. The number of practice trials ranged from a minimum of 32 to a maximum of 64. Each participant performed 216 experimental trials, divided into 9 blocks of 24 trials each. After each block of experimental trials, participants were invited to take a short rest before continuing with the next block of experimental trials. Levels of SOA, number of letters in T_1 , and T_2 pitch were fully randomized within each block of trials.

Results

On each trial, one or more responses to T_1 , and one response to T_2 , were produced. Responses to T_1 were

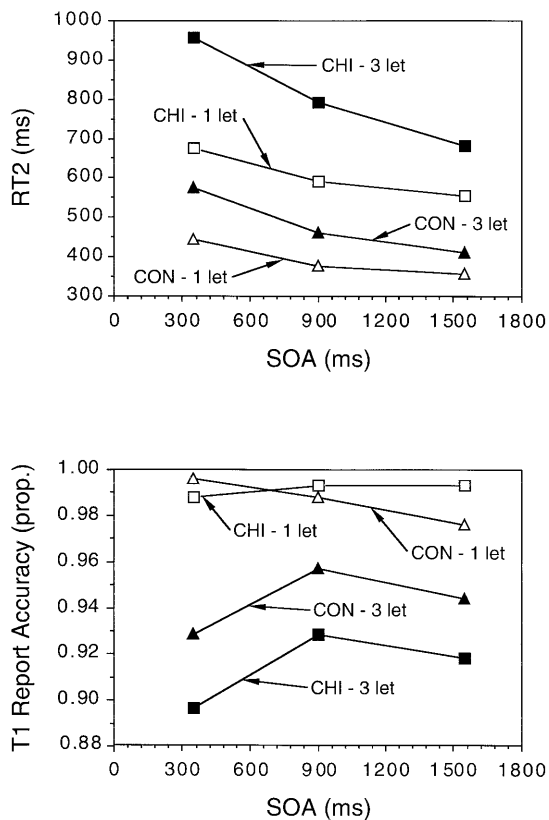


Fig. 7 Summary of the results in experiment 2. *Upper panel* mean RTs in the task on T₂, as a function of SOA, as a function of the number of to-be-remembered letters (1 let one letter, 3 let three letters), and as a function of group (CHI CHI patients, CON controls). *Lower panel* mean proportion of letters correctly reported at the end of each trial, as a function of the number of letters, and as a function of group

scored in terms of proportion of letters correctly reported at the end of each trial, with no regard for order of report when three letters were displayed on a given trial. Responses to T₂ were scored in terms of speed (RT₂) and accuracy (proportion of correct T₂ responses). Only correct RT₂s were included in the analyses, after exclusion of the outliers using the same procedure as that described in experiment 1 (see footnote 3). The application of the outlier elimination procedure resulted in a total loss of 2.7% of correct RT₂s. The results from both tasks were analyzed using ANOVA, in which SOA and number of letters were considered within-subject variables, and group (i.e., CHI patients vs controls) was considered a between-subject variable.

Responses to T1

The proportion of letters correctly reported in the task on T₁ for each group of participants, as a function of SOA and number of letters, is shown in Fig. 7 (lower panel). Both the SOA and number of letter manipulations produced significant main effects in the analysis of the proportion of letters correctly reported at the end of each

trial ($F_{(2,28)}=3.6$, $MSe=0.0005$, $P<0.05$; and $F_{(1,14)}=23.2$, $MSe=0.0037$, $P<0.001$, respectively).

There was a general trend to report fewer letters at the shortest SOA (0.94) than at the medium and longest SOAs (0.96 vs 0.95, respectively); recall performance was lower when three letters had to be reported with respect to the condition in which only one letter had to be reported (0.99 vs 0.93, respectively). Significant effects were also produced by the interaction between SOA and number of letters ($F_{(2,28)}=4.6$, $MSe=0.0005$, $P<0.02$), indicating that the drop in recall performance that can be observed in Fig. 7 at the shortest SOA, for both patients and controls, was restricted to the three-letter condition. In the one-letter condition, SOA effects were not significant ($F<1$, $P>0.3$). Neither the main effect of the type of population nor any interactions involving this factor were significant (all $F_s<1$).

Responses to T2

A summary of the results from the speeded task on T₂ is shown in Fig. 7 (upper panel). Mean RT₂s in Fig. 7 are plotted as a function of the three factors manipulated in the present experimental design, i.e., group, SOA, and number of to-be-remembered characters.

All three factors resulted in significant main effects in the ANOVA carried out on correct RT₂s. Patients were generally slower than controls in performing the task on T₂ (709 vs 437 ms, respectively; $F_{(1,14)}=15.3$, $MSe=115486$, $P<0.002$). The SOA manipulation had a sizable impact on participants' performance ($F_{(2,28)}=73.9$, $MSe=2932$, $P<0.001$), generating a monotonic RT₂ increase as SOA was decreased (from 501 to 663 ms, from the shortest to the longest SOA), the PRP effect. Slower RT₂s were observed when three letters had to be remembered than when a single letter had to be remembered (646 vs 500 ms, respectively; $F_{(1,14)}=32.5$, $MSe=15742$, $P<0.001$).

CHI patients and controls differed in several aspects of their performance in the task on T₂. The PRP effect reported by CHI patients was more pronounced than the PRP effect reported by controls ($F_{(2,28)}=3.7$, $MSe=2932$, $P<0.05$). Differential effects on performance were also generated by the manipulation of the number of to-be-remembered letters ($F_{(1,14)}=5.0$, $MSe=15742$, $P<0.05$), indicating a more marked increase in RT₂s across number-of-letter levels for CHI patients than for controls.

The three-way interaction among group, SOA, and number of letters was also significant ($F_{(2,28)}=4.2$, $MSe=789$, $P<0.03$). As SOA was shortened, the progressive divergence of the SOA functions across the number-of-letter levels was more pronounced for CHI patients than for controls. Further analyses were carried out in which the data from the different groups were separately considered. The two-way interaction between SOA and number of letters was significant for both CHI patients and controls ($F_{(2,14)}=23.9$, $MSe=1053$, $P<0.001$; and $F_{(2,14)}=11.7$, $MSe=526$, $P<0.002$, respectively). An anal-

ysis performed on the results from the one-letter condition indicated a significant main effect of group ($F_{(1,14)}=15.2$, $MSe=36319$, $P<0.002$), a significant main effect of SOA ($F_{(2,28)}=32.1$, $MSe=1403$, $P<0.001$), and a not significant interaction between these two factors ($F<1$, $P>0.4$).

A separate analysis was carried out on the data from the group of CHI patients in order to assess possible effects of the significant variability in the time-postinjury (see Table 1 and experiment 1) on RT_2 across the different SOAs as a function of the number of letters to-be-recalled. The same subgroups of CHI patients considered for an equivalent analysis performed on the data of experiment 1 (CHI patients nos. 1, 6, 7, 8 vs nos. 2, 3, 4, 5, 9) were considered for the present separate analysis. There was no sign of a difference between the two subgroups of CHI patients associated with different length of time-postinjury. The effect of the time-postinjury was significant neither as a main effect ($F_{(1,6)}=1.3$, $P>0.3$), nor in the interaction with SOA or the number of letters (all $F_s<1$).

The mean error rate in the task on T_2 was 3% for CHI patients and 5% for controls. Error rates were analyzed as a function of the same factors considered in the analysis on RT_2 . No factor produced significant effects (all $F_s<2$ and $P_s>0.11$).

Discussion

The results of experiment 2 fit all the predictions. Memory for the letters presented in T_1 was generally stable across SOAs, and comparable across CHI patients and controls. However, CHI patients were generally slower than controls in performing the speeded task on T_2 , a result that bears a close resemblance to the results of experiment 1. The manipulation of the number of to-be-recalled letters presented in T_1 had a stronger impact on CHI patients' performance than on controls' performance in the speeded task on T_2 , that is, the SOA-locked difference between the one-letter and three-letter conditions was more pronounced for CHI patients than for controls. A slight inconsistency, however, emerged between the observed and predicted results of experiment 2. While the progressive divergence of the two RT_2 functions for CHI patients and controls gave rise, as predicted, to a statistically significant interaction in the analysis of the three-letter condition, parallel SOA functions across CHI patients and controls were observed in the one-letter condition (see Fig. 7, upper panel). This finding represents a small discrepancy with respect to the predicted results, because we had predicted a more marked PRP effect for CHI patients than for controls even in this latter condition. Although we do not have a clear explanation of this specific aspect of the results of experiment 2, recent work in our laboratory (Stablum and Dell'Acqua 2000) suggests that a certain degree of fluctuation in the slope of the SOA functions across CHI patients and controls is associated with conditions in

which the to-be-recalled information presented in T_1 is minimal (e.g., one letter) in this type of paradigm. We hypothesize that, had we probed T_1 -related mental activity by presenting T_2 earlier than the shortest SOA adopted in the present design (350 ms), a significant difference between the one-letter RT_2 functions for CHI patients and controls would have been observed, inasmuch as the probability to detect ongoing central processing activity required for T_1 consolidation would have increased.

A further, unpredicted, result is related to the speed in carrying out the task on T_2 at the longest SOA. Note that, while the two SOA functions in the one-letter and three-letter conditions for controls converged at the longest SOA, the equivalent functions for CHI patients did not. At the longest SOA, the difference in RT_2 between the one-letter condition and three-letter condition was not significant for controls ($F<1$, $P>0.2$), whereas this difference was significant for CHI patients ($F_{(1,7)}=7.0$; $MSe=8923$, $P<0.04$). There are two possible interpretations of this result. The first explanation rests on the specific choice of SOAs in our paradigm, which was made to keep the SOA range constant across experiment 1 and experiment 2. What the present results suggest is that the proportion of trials in which controls were done with T_1 consolidation when T_1 was composed of three letters approximated the value of 1.0 at the longest SOA (i.e., at 1550 ms after T_1 presentation), because the RT_2 functions for the one-letter and the three-letter conditions converged at the longest SOA. This might not be so for CHI patients. On the assumption that central processing time is prolonged following a CHI, it is reasonable to hypothesize that, on a subset of trials, central mechanisms were still busy with consolidating three letters at the time when T_2 was presented at the longest SOA.

The second explanation calls into play the capacity demands of rehearsing information in short-term memory. Using a paradigm similar to the present experiment 2, Naveh-Benjamin and Jonides (1984; see also Ogden et al. 1980) have shown dual-task costs on second task performance that persisted, in their paradigm, up to 12 s. These authors attributed the dual-task costs found in their study to the central capacity limitations of maintenance rehearsal, which interfered with the concurrent mental activity required for the secondary speeded task. In this view, one suggestion may be that the residual one-letter to three-letter cost found for CHI patients at the longest SOA may reflect interference from central processing required for rehearsing the encoded information presented in T_1 when T_1 is composed of more than a single chunk of information, that is, three random letters.

General discussion

The present scenario

The present work was undertaken as part of an effort to provide an explanation of one of the core behavioral

manifestations of a CHI deficit in human performance, that is, the slower processing speed commonly reported by CHI patients when performing in speeded tasks. Two opposite hypotheses concerning the cause of slowing phenomenon have been considered, the generalized deficit hypothesis and the central source hypothesis. According to the first hypothesis the slowing phenomenon is caused by a generalized functional deficit involving perceptual, cognitive, and motor processing components required for the execution of a speeded response. According to the second hypothesis, the slowing phenomenon is caused by a processing deficit selectively involving a central stage of processing required for the execution of a speeded response.

By considering in detail the central source hypothesis, several predictions have been derived concerning the hypothetical scenario generated by having CHI patients and matched controls perform in two dual-task experiments devised to minimize perceptual and/or motor interference. In experiment 1, CHI patients and controls performed in a classical PRP paradigm in which distinct, speeded, vocal and manual responses had to be executed to auditory and visual stimuli that were presented at variable SOAs. As postponement models of the PRP effect would predict, an exacerbated PRP effect was reported for CHI patients compared to controls. Furthermore, a statistical comparison involving amount of PRP effect on secondary task performance and absolute speed in primary task performance converged to support the hypothesis of a "pure" central locus of the slowing phenomenon, by showing an equivalence between these two estimates in the results of experiment 1.

Experiment 2 provided further empirical support for the central source hypothesis, and helped rule out the potential of an additional response initiation bottleneck as a determinant of the pattern of results in experiment 1. In experiment 2, CHI patients and controls performed in a dual-task condition in which masked, later recalled, visual information was briefly presented in the first stimulus, followed at different SOAs by a second, auditory, stimulus requiring a speeded response. As predicted based on the notion that central processing was also required for the recall task (i.e., for short-term consolidation of to-be-recalled information), and on the hypothesis of the central source of the CHI-contingent slowing phenomenon, an exacerbated PRP effect was reported for CHI patients compared to controls in experiment 2. Furthermore, recall performance between CHI patients and controls did not differ in any significant respect, suggesting that both perceptual and short-term storage capacity, as tested under these particular experimental conditions, were preserved in this group of CHI patients.

Relations to previous CHI studies

The present results (i.e., the greater PRP effects for CHI patients) strongly support the notion that a crucial component of the CHI slower processing time is related to a selective increase in temporal demands for central pro-

cessing of the stimuli. In this view, the finding of a selective impairment of CHI on central resources allocation suggests important requalifications of previous empirical findings.

Obviously, the present results are in striking contrast to those of studies that did not report increased dual-task deficits for CHI patients (for reviews see Park et al. 1999; Van Zomeren and Brouwer 1994). Although, as all null results, the absence of a deficit in dual-task performance should be taken with serious caution, we would like to pinpoint some of the reasons for the failure to detect a CHI deficit in these studies. One reason may have to do with the nature of the tasks used so far to investigate dual-task performance. Many CHI studies used tasks with prevalent perceptual and motor components (e.g., Brouwer et al. 1989; Veltman et al. 1996), for which central components of processing have hardly even been tested. The absence of dual-task deficits may be due to the fact that the crucial cognitive components were not taken into consideration, and no psychological mechanisms or structures specific to multiple-task conditions were hypothesized. Often implicit in several of these studies was the assumption that each task combination insisted on the same general processing capacities, whose depletion should produce a dual-task deficit. As argued in the "Introduction," however, the experimental literature does not support this assumption. Different task combinations may load on different processing resources, and dual-task interference may be caused by different sources depending on the particular task combinations. Isolating one of these components was a major effort in the present investigation. Therefore, one avenue the present study indicates is that, on the one hand, more empirical work should be carried out on this issue and, on the other hand, a crucial feature of future studies should be to selectively test the different components implicated in specific multitasking conditions.

Moreover, when hypotheses concerning the functional architecture of dual-task process dynamics were explicitly reported in dual-task studies in which CHI-contingent deficits were found (Azouvi et al. 1996; McDowell et al. 1997; Park et al. 1999; Vilkki et al. 1996), very often the tasks were too complex to allow a clear componential analysis. Based on a meta-analysis of results from several dual-task studies, Park and colleagues (1999) suggested that a deficit occurred restricted to conditions in which the tasks depended heavily upon remembering and using stored information. Our results suggest that working memory is not the crucial factor underlying dual-task deficits in CHI patients. In experiment 1, working memory requirements were rather limited. Nonetheless, the patients showed greater PRP effects. One major advantage of the paradigms employed in the present context is that they rely on an extensive theoretical and experimental literature, allowing relatively detailed modeling of the underlying processing components generating PRP effects.

Some recent studies suggested that severe long-term CHI patients showed a response selection deficit

(Schmitter Edgecombe et al. 1992; Shum et al. 1990, 1994). Our results help generalize these findings by relating response selection difficulties to a selective slowness in central processing. As response selection, other operations with equivalent functional demands were expected to be compromised in CHI patients. This prediction has been confirmed in experiment 2, where a CHI-contingent deficit in the short-term consolidation of to-be-recalled information was observed.

The finding of a selective impairment of CHI on central resource allocation has many important implications also for CHI rehabilitation. Limitations in central processing capacity can influence performance on a variety of task and daily living situations. Specific rehabilitation settings may be developed based on recent findings showing drastically reduced PRP effects through extended practice (e.g., Van Selst et al. 1999).

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References

- Azouvi P, Jokic C, Van der Linden M, Marlier N, Bussel B (1996) Working memory and supervisory control after severe closed-head injury: a study of dual task performance and random generation. *J Clin Exp Neuropsychol* 18:317–337
- Brouwer WH, Ponds RWHM, Van Wolfelaar PC, Van Zomeren AH (1989) Divided attention 5 to 10 years after severe closed head injury. *Cortex* 25:219–230
- Coltheart M (1980) Iconic memory and visible persistence. *Percept Psychophys* 27:183–228
- De Jong R (1993) Multiple bottlenecks in overlapping task performance. *J Exp Psychol: Hum Percept Perform* 19:965–980
- Dell'Acqua R, Jolicoeur P (2000) Visual encoding of patterns is subject to dual-task interference. *Memory Cogn* 28:184–191
- Ferraro FR (1996) Cognitive slowing in closed-head injury. *Brain Cogn* 32:429–440
- Foote SL, Morrison JH (1987) Extrathalamic modulation of neocortical functions. *Annu Rev Neurosci* 10:67–95
- Gentilini M, Nichelli P, Schoenhuber R (1989) Assessment of attention in mild head injury. In: Levin HS, Eisenberg HM, Benton AL (eds) *Mild head injury*. Oxford University Press, New York, pp 163–175
- Gronwall D (1987) Advances in the assessment of attention and information processing after head injury. In: Levin HS, Grafman J, Eisenberg M (eds) *Neurobehavioral recovery from head injury*. Oxford University Press, New York, pp 355–371
- Gronwall DMA, Whrightson P (1975) Cumulative effect of concussion. *Lancet* 2:995–997
- Hartman A, Pickering RM, Wilson BA (1992) Is there a central executive deficit after severe head injury? *Clin Rehabil* 6:133–140
- Ivry RB, Franz EA, Kingstone A, Johnston JC (1998) The psychological refractory period effect following callosotomy: uncoupling of lateralized response codes. *J Exp Psychol Hum Percept Perform* 24:463–480
- Jennett B, Bond M (1975) Assessment of outcome after severe brain damage. *Lancet* 1:480–484
- Jennett B, Snoek J, Bond MR, Brooks N (1981) Disability after severe head injury: observations on the use of the Glasgow Outcome Scale. *J Neurol Neurosurg Psychiatr* 44:285–293
- Jolicoeur P, Dell'Acqua R (1998) The demonstration of short-term consolidation. *Cogn Psychol* 36:138–202
- Jolicoeur P, Dell'Acqua R, Crebolder J (2000) Multitasking performance deficits: forging some links between the attentional blink and the psychological refractory period. In: Monsell S, Driver J (eds) *Attention and performance XVIII*. The MIT Press, Cambridge, pp 309–330
- Levin HS, Grossman RG (1979) The Galveston Orientation and Amnesia Test. A practical scale to assess cognition after head injury. *J Nerv Mental Dis* 167:675–684
- McCann RS, Johnston JC (1992) Locus of the single-channel bottleneck in dual-task interference. *J Exp Psychol Hum Percept Perform* 18:471–484
- McDowell S, Whyte J, D'Esposito M (1997) Working memory impairments in traumatic brain injury: evidence from a dual-task paradigm. *Neuropsychologia* 35:1341–1353
- Meyer DE, Kieras DE (1997) A computational theory of executive cognitive processes and human multiple-task performance: part 2. Accounts of psychological refractory-period phenomena. *Psychol Rev* 104:749–791
- Miller E (1970) Simple and choice reaction time following severe head injury. *Cortex* 6:121–127
- Naveh-Benjamin M, Jonides J (1984) Cognitive load and maintenance rehearsal. *J Verbal Learning Verbal Behav* 23:494–507
- Norman B, Swahn K (1961) A follow-up study of severe brain injuries. *Acta Psychiatr Scand* 37:236–264
- Ogden WC, Martin DW, Paap KR (1980) Processing demands of encoding: what does secondary task performance reflect? *J Exp Psychol Hum Percept Perform* 6:355–367
- Osman A, Moore CM (1993) The locus of dual-task interference: psychological refractory effects on movement related brain potentials. *J Exp Psychol Hum Percept Perform* 19:1292–1312
- Park NW, Moscovitch M, Robertson IH (1999) Divided attention impairments after traumatic brain injury. *Neuropsychologia* 37:1119–1133
- Pashler H (1994) Dual-task interference in simple tasks: data and theory. *Psychol Bull* 116:220–244
- Pashler H (1998) *The psychology of attention*. MIT Press, Cambridge, MA
- Pashler H, Johnston JC (1989) Chronometric evidence for central postponement in temporally overlapping tasks. *Q J Exp Psychol* 41A:19–45
- Pashler H, Johnston JC (1998) Attentional limitations in dual-task performance. In: Pashler H (ed) *Attention*. Psychology Press, Hove, UK, pp 155–189
- Pashler H, Luck SJ, Hillyard SA, Mangun GR, O'Brien S, Gazzaniga MS (1994) Sequential operation of disconnected cerebral hemispheres in split-brain patients. *Neuroreport* 5:2381–2384
- Ponsford J, Kinsella G (1992) Attentional deficits following closed head injury. *J Clin Exp Neuropsychol* 14:822–838
- Riese H, Hoedemaeker M, Brouwer WH, Mulder LJM (1999) Mental fatigue after very severe closed head injury: sustained performance, mental effort, and distress at two levels of workload in a driving simulator. *Neuropsychol Rehabil* 9:189–205
- Schmitter-Edgecombe ME, Marks W, Fahy JF, Long CJ (1992) Effects of severe closed-head injury on three stages of information processing. *J Clin Exp Neuropsychol* 14:717–737
- Schubert T (1999) Processing differences between simple and choice reactions affect bottleneck localization in overlapping tasks. *J Exp Psychol Hum Percept Perform* 25:408–425
- Shum DHK, McFarland K, Bain JD, Humphreys MS (1990) Effects of closed-head injury on attentional processes: an information-processing stage analysis. *J Clin Exp Neuropsychol* 12:247–264
- Shum DHK, McFarland K, Bain JD (1994) Effects of closed-head injury on attentional processes: generality of Sternberg's additive factor method. *J Clin Exp Neuropsychol* 16:547–555
- Spikman JM, Van Zomeren AH, Deelman BG (1996) Deficits of attention after closed-head injury: slowness only? *J Clin Exp Neuropsychol* 18:755–767

- Spinnler H, Tognoni G (1987) Standardizzazione e taratura italiana di test neuropsicologici [Standardization for Italian of neuropsychological tests]. *J Neurol Sci Suppl* 8
- Spreen O, Strauss E (1998) A compendium of neuropsychological tests: administration, norms, and commentary (2nd edn). Oxford University Press, New York
- Stablum F, Dell'Acqua R (2000) A neuropsychological investigation of dual-task performance following closed-head injury. Department of General Psychology, University of Padova (manuscript in preparation)
- Stablum F, Leonardi G, Mazzoldi M, Umiltà C, Morra S (1994) Attention and control deficits following closed head injury. *Cortex* 30:603–618
- Van der Linden M, Coyette F, Seron X (1992) Selective impairment of the central executive component of working memory: a single case study. *Cogn Neuropsychol* 9:301–326
- Van Selst M, Johnston JC (1997) Dual-task interference when a response is not required. In: Shafto MG, Langley P (eds) Proceedings of the nineteenth annual conference of the Cognitive Science Society. Erlbaum, Mahwah, NJ, pp 787–792
- Van Selst M, Jolicoeur P (1994) A solution to the effect of sample size on outlier elimination. *Q J Exp Psychol* 47A:631–650
- Van Selst M, Jolicoeur P (1997) Decision and response in dual-task interference. *Cogn Psychol* 33:266–307
- Van Selst M, Ruthruff E, Johnston JC (1999) Can practice eliminate the psychological refractory period effect? *J Exp Psychol Hum Percept Perform* 25:1268–1283
- Van Zomeran AH, Brouwer WH (1994) Clinical neuropsychology of attention. Oxford University Press, New York
- Van Zomeran AH, Deelman BG (1976) Differential effects of simple and choice reaction after closed head injury. *Clin Neurol Neurosurg* 79:81–90
- Van Zomeran AH, Deelman BG (1978) Long-term recovery of visual reaction time after closed head injury. *J Neurol Neurosurg Psychiatry* 41:452–457
- Van Zomeran AH, Brouwer WH, Deelman BG (1984) Attentional deficits: the riddles of selectivity, speed, and alertness. In: Brooks N (ed) Closed head injury: psychological, social, and family consequences. Oxford University Press, New York, pp 74–107
- Veltman JC, Brouwer WH, Van Zomeran AH, Van Wolffelaar PC (1996) Central executive aspects of attention in subacute severe and very severe closed head injury patients: planning, inhibition, flexibility, and divided attention. *Neuropsychology* 10:357–367
- Vilkki J, Virtanen S, Surma-Aho O, Servo A (1996) Dual task performance after focal cerebral lesions and closed head injuries. *Neuropsychologia* 34:1051–1056