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Frontal lesions increase post-target interference in rapid stimulus streams

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Abstract—We examined the effects of frontal lesions on the attentional processes surrounding the discrimination of target stimuli by comparing patients with frontal excisions, patients with temporal excisions and controls on target-letter identification in rapid visual streams. Subjects were asked to look at streams of 18–26 letters presented centrally at rates of 6, 8, or 10 letters/sec and to name the two white target letters (T1 and T2) embedded among black letters in each stream. The two target letters were separated by either 0, 2, 4, or 6 black letters. Normals and temporals correctly reported T1 at all rates, they showed the expected T2 identification errors peaking 300 msec after T1 at high rate and little T2 interference at lower rates. However, frontals showed T2 interference at the two lower rates and were unable to identify T1 at high rate. The effects observed suggest that an inertia of target discrimination processes contributes to the frontal attention deficits. Copyright © 1996 Elsevier Science Ltd.

Key Words: attention; frontal lobes; rapid serial visual presentation.

Introduction

Although it is widely accepted that the frontal lobes play an important role in attentional functions [6, 8, 16], the evidence concerning the nature of this role is rather scarce. Clinical descriptions indicate that frontal lobe lesions often produce concentration difficulties especially in complex tasks involving multiple steps [8, 17], but they can also show deficits in simple detection tasks which require sustained attention [20]. Frontal patients can also lead to problems in visual search often characterized as inertia of gaze and haphazard exploration of scenes or stimulus arrays [8, 18]. In addition, their attentional difficulties can also take the form of distractibility or increased susceptibility to interference, although this symptom is more clearly associated with orbitofrontal lesions than with damage elsewhere in the frontal regions [6, 16].

Despite the frequency of these deficits in several neurological and psychiatric populations and their importance for complex behaviour, few studies have analysed the frontal attentional deficits in terms of their underlying operations. There is evidence that frontal lesions cause problems in relatively elementary attentional functions such as those involved in the simple discrimination of target stimuli from distractors. Indeed, in a task requiring the search for occurrences of target symbols among distractors, frontals make more errors and are slowed more than temporals or normals, especially when the target is categorically defined [13]. This suggests that processes involved in making simple categorical responses to stimuli are inefficient after frontal lesions. Because the discrimination of target stimuli from distractors is a basic attentional function, an analysis of this deficit may help understand a number of frontal attentional problems observed at the clinical level.

The present study was designed to explore the nature of the frontal deficit in target discrimination. The inefficient performance of frontals in target discrimination may come from a variety of sources. One possibility is that the processing limits which affect target discrimination in normals are exacerbated in patients with frontal lesions. Visual search involves a rapid sampling of the stimulus array and categorization of successive stimuli as target or non-target. There is evidence that this process of serial categorization is limited in rate because of the interference produced by the discrimination of a target on the processing of stimuli presented in close temporal proximity [2, 4, 5]. For example, the identification of a target character in a stream of distractors can interfere with a

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second identification for several hundred milliseconds and this interference does not occur if the first character is ignored [2, 3, 12, 15]. If the duration or size of the interference produced by target discrimination is larger after a frontal lesion, this could produce decrements in accuracy or speed in visual search as well as contribute to other attentional problems. The time course of the interference between simple categorical responses can be examined using a rapid serial visual presentation (RSVP) task. In one version of the RSVP task, a stream of characters is presented rapidly at fixation, with target stimuli embedded at different positions among distractor stimuli. An identification of the target characters is asked at the end of the stimulus stream, providing an unspeeded accuracy measure. This task reproduces the major aspects of a visual search task in the temporal domain while providing an external control of stimulus sampling rate. This study examined the interference associated with target identification in patients with frontal excisions, patients with temporal excisions and normals. Subjects were tested in an RSVP task in which they had to identify two white target letters embedded in streams of black letters presented in rapid succession and we examined the effects of presentation rate and of the separation between the two target letters.

Methods

Subjects

We compared the performance of eight patients with a unilateral frontal excision (six right, two left; all men) to that of eight patients with a unilateral temporal excision (four right, four left; six men, two women) and of eight controls (six men, two women) with no history of cerebral damage. Groups were matched in age (mean: 38 years; range: 26–55 years) and education level (mean: 11 years; range: 6–18 years). The resections were performed in adulthood to alleviate a drug-resistant epilepsy and patients were tested between 1 and 12 years following the surgical intervention. Informed consent to participate in the study was obtained according to the rules of the institution.

Frontal excisions were variable in extent but always included dorsomedial structures (anterior cingulate gyrus, superior frontal gyrus) and a variable amount of dorsolateral cortex anterior to the precentral sulcus. Figure 1 shows the extent of the frontal resections. Seven of the frontal patients showed a marked reduction in seizure frequency after surgery (80% or more), two were seizure free and all were on anticonvulsant medication. Anterior temporal excisions involved resection of the anterior portion of the temporal lobe (about 5 cm from the anterior tip of the lobe), partial resection of the hippocampus, and sparing of Heschl's gyrus. All temporal patients showed a marked reduction in seizure frequency (four were seizure free) and four were still on anticonvulsant medication.

All patients underwent post-surgery neurological and neuropsychological evaluation. None of the patients exhibited sensory or motor impairment on clinical measures. All patients had a WAIS IQ above 80 and all could demonstrate comprehension and retention of the instructions of the task. Neuropsychological evaluation revealed no significant deficits in language, episodic memory, or praxis. Frontals were more impaired than temporals on verbal fluency, sequential responses, the Stroop task and speeded visual search, but were not significantly more impaired than temporals on digitspan or short-term recall of trigrams under interference [13, 14].

Tasks and procedure

Subjects were tested in a single session divided into four blocks of trials separated by short pauses, including a practice block of 20 trials and three experimental blocks of 60 trials, one for each of the three presentation rates examined. The task was controlled by a 486 computer running Neuroscan software. The subject initiated a trial by pressing a button after fixating the central portion of the screen. On each trial, a stream of 18–26 upper-case letters was presented in the centre of a computer screen. All letters appeared black with the exception of the two target letters (T1 and T2) that were white. The letters were presented 60 cm in front of the subject, subtending a visual angle of 1.5° and with a duration of 15 msec. The streams were constructed from random letter sequences with the condition of no repetitions. In each stream, T1 was preceded by 7–15 letters and followed by 10 letters. The position of T2 was varied randomly so that either 0, 2, 4, or 6 distractors separated the two targets. These four positions of T2 were respectively called T1+1, T1+3, T1+5 and T1+7.

The subject was instructed that the streams contained two white letters and that he or she should name them after the end of the stream. Instructions emphasized the precision of the naming response and not its speed. Pilot work determined that at low presentation rates none of the targets are misidentified, whereas at moderate rates T2 errors are observed while T1 is reported correctly and at high rates all targets are misidentified. We chose to examine three presentation rates that would assure observation of the first two stages in normals: 6, 8 and 10 letters/sec, which produced inter-stimulus delays of 0.167, 0.125 and 0.100 sec, respectively. The sequence of conditions was fixed with the slowest rate tested first and the fastest rate last.

Results

Table 1 summarizes the performance of the three groups in the identification of the first target (T1). At 6 and 8 letters/sec, subjects showed no difficulty identifying T1, performance averaging between 93 and 99% correct. At 10 letters/sec, frontals were unable to do the task, making frequent T1 identification errors and reporting frustration and this block was therefore not completed in this group. T1 identification performance did not differ significantly among the two other groups at the fastest rate [$F(1, 14) = 2.9$, ns]. At the two slower rates, T1 identification accuracy was also similar among the three groups [$F(2, 21) = 2.6$, ns], and although it was affected by presentation rate [$F(1, 21) = 12.5, P < 0.01$], there was no significant group × rate interaction [$F(2, 21) = 2.3$, ns]. We investigated the relationship between errors at T1 and the position of T2 when the number of T1 errors permitted comparisons. T1 errors were most frequent in trials in which T2 was at position T1+1, the proportion of such errors varying between 38 and 80%. Errors consisting of naming the stimulus following T1 only occurred when T2 was at position T1+1 and represented between 0 and 70% of errors.

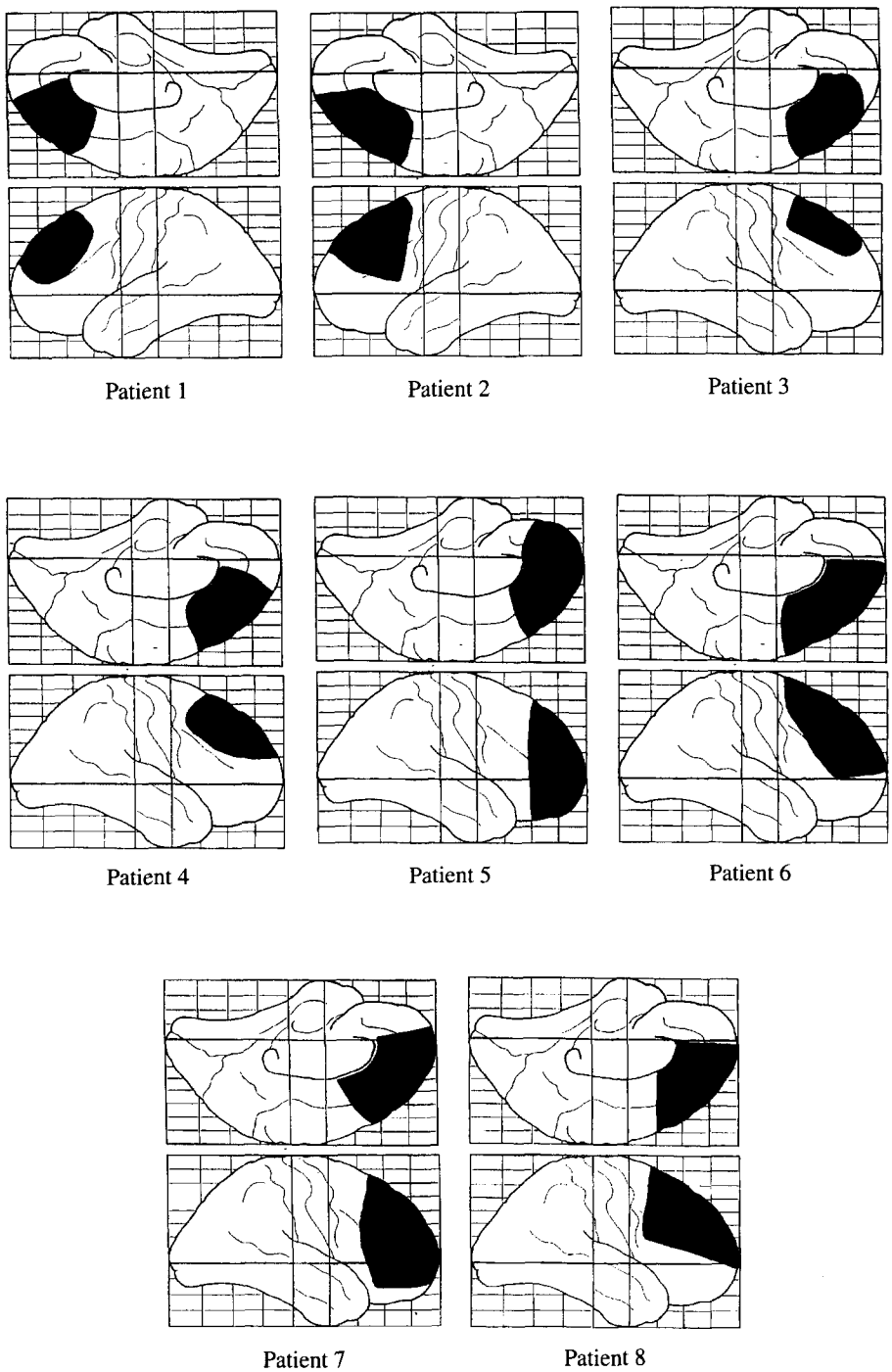

Fig. 1. Extent of medial and lateral resection of the frontal excisions.

Table 1. Mean percentage of correct identifications of the first target in the three groups for the three presentation rates. Standard deviations are in parentheses

Condition	Normals	Temporals	Frontals
6 letters/sec	99.6 (1.2)	99.2 (1.3)	97.7 (3.6)
8 letters/sec	99.0 (1.5)	95.6 (6.6)	92.5 (6.8)
10 letters/sec	92.9 (5.5)	85.8 (10.5)	–

However, comparisons of groups or rates were not possible on the types of errors made.

Figure 2 shows the ability of the three groups to identify the second target letter in trials in which the first target letter was correctly identified. At 10 letters/sec the performance of normals and temporals was very similar showing a large T2 interference with a peak when T2 was at position T1+3. T1 performance was used as a comparison to determine whether significant T2 interference was present at each position, using unidirectional *t*-tests. At the fastest rate both normals and temporals

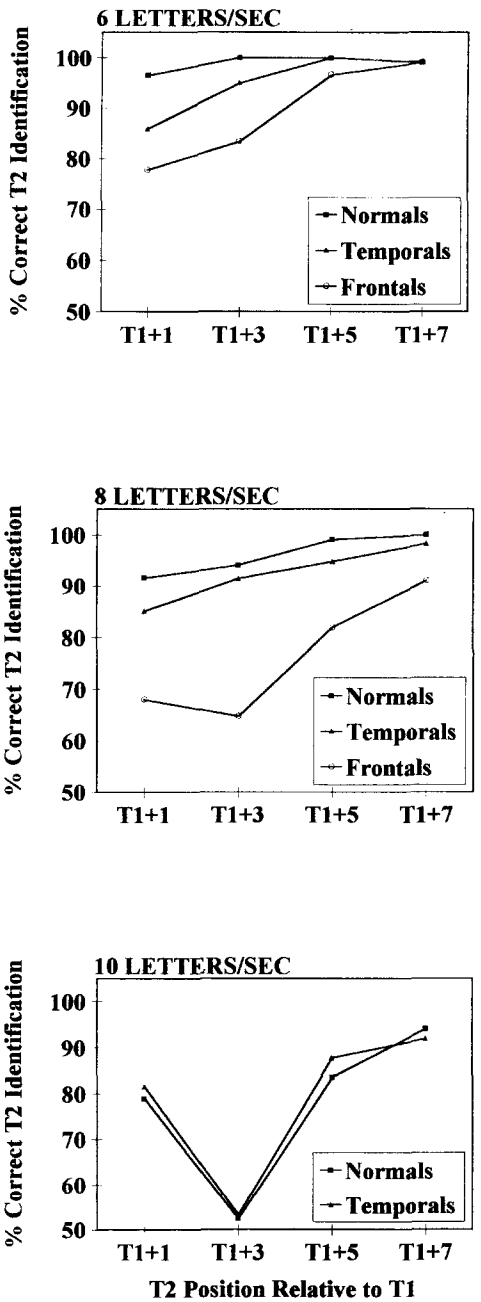

Fig. 2. Percentage of correct identifications of the second target (T2) in relation to its position after the first target (T1) in the three groups and at the three presentation rates.

showed a significant T2 interference at position T1 + 3 [all $t(7) > 3.85$, all $P < 0.005$]. Normals also showed a smaller but significant interference at position T1 + 1 [$t(7) = 2.2, P < 0.05$] and T1 + 5 [$t(7) = 3.3. P < 0.01$]. No significant interference was observed at position T1 + 7 at the fastest rate [all $t(7) < 1.8$, ns].

At 8 letters/sec, normals and temporals showed no significant T2 interference at any position [all $t(7) < 1.8$, ns]. Frontals, on the other hand, showed a significant T2 interference for positions T1 + 1 [$t(7) = 3.5, P < 0.005$] and T1 + 3 [$t(7) = 2.9, P < 0.01$]. At 6 letters/sec, normals still showed no significant T2 interference [all $t(7) < 1.0$, ns], while temporals showed a small interference when T2 was at position T1 + 1 [$t(7) = 3.1, P < 0.01$]. Frontals showed a generally smaller interference at 6 than at 8 letters/sec, but the interference was still significant for positions T1 + 1 and T1 + 3 [all $t(7) > 2.6$, all $P < 0.02$].

At the two lower rates, T2 performance was also examined in terms of group and rate differences using analyses of variance (ANOVAs) at each position. When T2 was at position T1 + 1, groups were significantly different [$F(2, 21) = 7.1, P < 0.004$], due to differences between frontals and the two other groups [$F(1, 21) > 4.5$, $P < 0.05$]. However, there was no significant effect of rate [$F(1, 21) = 2.6$, ns] nor any group × rate interactions [$F(2, 21) = 0.67$, ns]. When T2 was presented at position T1 + 3, groups were also significantly different [$F(2, 21) = 6.3, P < 0.007$] and again because of differences between frontals and other groups [$F(1, 21) > 4.8$, $P < 0.05$]. The rate effect was also significant [$F(1, 21) = 10.5, P < 0.004$], but not the interaction [$F(2, 21) = 2.7$, ns]. Ceiling effects prevented meaningful analyses at the other two positions. We also examined the type of errors made in the identification of T2 when the number of errors permitted such an analysis. The proportion of errors on which the letter following T2 was reported instead of T2 varied between 16 and 80%.

The effects obtained were examined in relation to the side and size of the frontal lesion. Although the sample did not permit quantitative correlations, neither the side nor the extent of excision was systematically associated with the size of the interference effects.

Discussion

The results indicate that the performance of patients with frontal lesions was more subject to interference in this target discrimination task than that of temporals and normals. More specifically, frontals showed a significant interference by a first identification on a second identification at lower presentation rates than temporals and normals. Also, unlike normals and temporals, frontals showed problems identifying the first target at the highest rate.

In normals and temporals, we observed a prolonged interference on T2 identification at the highest rate. This effect replicates previous reports using a similar task with two targets embedded among distractors [3, 12]. In our task, this interference on T2 disappears when the presentation rate is lowered from 10 to 8 letters/sec. This suggests that distractors are involved in the appearance of the effect, since the only difference between the two conditions is the proximity of successive stimuli. Recent evidence suggests that distractors in general and the distractor immediately following T1 in particular play an important role in the appearance of the post-target interference by affecting the discriminability of T1 [3, 12].

In frontals, the large T2 interference at 8 letters/sec indicates that target discrimination processes can generate interference at a lower presentation rate in this group. This suggests that the proximity of distractors has a more deleterious effect on frontals than on other groups, a form of susceptibility to interference. The susceptibility could be due to a number of underlying mechanisms. For example, target discrimination processes may have a slower onset in frontals, making them more susceptible to the interference of the distractor that immediately follows the target. Alternatively, the general pace of stimuli in a stream may have a slowing effect on discrimination processes, and this slowing could be exacerbated in frontals. Future studies will need to clarify the mechanism involved in this deficit.

In neuropsychology, frontal lesions have already been associated with susceptibility to interference. This concept has often been used to explain a wide variety of problems associated with frontal lesions whether in everyday behaviour, in complex problem solving, or in memory tasks [6, 8, 16]. However, there are multiple differences between the identification failures observed here and other situations in which patients either orient to irrelevant stimuli or confuse multiple presentations of stimuli. More work will be needed to characterize the different types of susceptibility to interference better. The problem shown by frontals in T2 identification in the RSVP task appears to be a special kind of susceptibility to interference which affects the efficiency of target discrimination and exacerbates the normal limits in the capacity to shift from one discrimination to another. The net effect is an increased attentional inertia, i.e. attentional engagement on one target interferes with engagement on a following target for a significant interval at rates which do not affect temporals and normals.

The increased susceptibility to post-target interference in frontals can give rise to a number of problems in visual attention. Many tasks require the exploration of a visual scene either to search for a specific stimulus or to memorize some aspect of the scene. In this situation, the stimulus stream is self-generated through saccades or covert sampling of portions of the scene. At certain rates, this search activity could produce sequential interference effects which would lead to categorization errors. Another possible consequence is that subjects could adaptively slow down the rate at which they sample the scene to reduce the frequency of misidentifications. These problems could underlie some of the reported effects of frontal lesions on visual search such as inertia of gaze or haphazard exploration [8, 17, 18]. Increased susceptibility to post-target interference can also account for the frequent errors produced by frontals in speeded target search [13].

In addition to being relevant to frontal deficits in attention tasks, the attentional inertia of frontals in rapid stimulus streams may be linked to a number of shifting problems associated with frontal lesions. For example, frontal lesions can produce problems in shifting response set in categorization or choice tasks, although such deficits are not always specific to frontal lesions [1, 9, 11, 19]. An excessive attentional inertia could also be analogous to the response inertia which has been evoked to explain the sequential response deficits observed in frontal lesions. Luria, for example, has suggested that a pathological inertia of motor responses could explain the high frequency of perseverative and other errors in complex or sequential tasks [8]. Excessive inertia can also be linked to suggestions that frontal damage may affect release from proactive interference in memory tasks [10]. Future studies will need to examine the relationship between the different types of inertia in frontals. If they have common causes, it may facilitate progress in understanding the role of inertia as a fundamental characteristic of frontal cognitive deficits. Also, it may be interesting to investigate whether other types of frontal inertia problems are linked to an increased susceptibility to interference.

In the RSVP task, frontals also showed an important disruption in the identification of the first target at 10 letters/sec that is not present in normals and temporals. Although the rates used here did not permit quantitative evaluation of this disruption, previous studies indicate that, in normals, T1 disruption can be significant at rates higher that 12 letters/sec [7]. Whether the same mechanisms are responsible for the interference affecting T1 and that which affects T2 at lower rate is not yet clear. However, there is evidence that T1 interference is also related to the presence of distractors and that it is not simply due to a sensory masking phenomenon since it varies with the type of categorization performed [7]. The T1 interference observed in frontals is an additional sign of increased susceptibility to interference. This form of susceptibility appears to produce a failure of attentional engagement on T1 due to the overall pace of stimuli. If it is generalizable, this phenomenon may help quantify the capacity for attentional engagement under interference.

In summary, the performance of frontals in the RSVP task used here suggests that some of their attentional problems may have their roots in an increased susceptibility to interference, one effect of which is to exacerbate the inertia of target discrimination. The data also suggest that a systematic investigation of the different types of inertia and of the different types of susceptibility to interference in frontal patients may help to understand the underlying mechanisms of their cognitive problems.

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References

1. Anderson, S. W., Damasio, H., Dallas Jones, R. and Tranel, D. Wisconsin card sorting performance as a

measure of frontal lobe damage. *J. Clin. Exp. Neuropsychol.* **13,** 909–922, 1991.
2. Broadbent, D. E. and Broadbent, M. H. P. From detection to identification: Response to multiple targets in rapid serial visual presentation. *Percept. Psychophys.* **42,** 105–113, 1987.
3. Chun, M. M. and Potter, M. C. A two-stage model for multiple target detection in rapid serial visual presentation. *J. exp. Psychol.: Human Percept. Perform.* **21,** 109–127, 1995.
4. Duncan, J. The locus of interference in the perception of simultaneous stimuli. *Psychol. Rev.* **87,** 272–300, 1980.
5. Duncan, J., Ward, R. and Shapiro, K. Direct measurement of attentional dwell time in human vision. *Nature* **369,** 313–315, 1994.
6. Fuster, J. M. *The Prefrontal Cortex*. Raven Press, New York, 1989.
7. Lawrence, D. H. Two studies of visual search for word targets with controlled rates of presentation. *Percept. Psychophys.* **10,** 85–89, 1971.
8. Luria, A. R. Frontal-lobe syndromes. In *Handbook of Clinical Neurology*, P. J. Vinken and G. W. Bruyn (Editors), Vol. 2, pp. 725–757. North Holland, Amsterdam, 1969.
9. Milner, B. Some effects of frontal lobectomy in man. In *The Frontal Granular Cortex and Behavior*, J. M. Warren and K. Akert (Editors), pp. 313–334. McGraw-Hill, New York, 1964.
10. Moscovitch, M. Multiple dissociations of function in amnesia. In *Human Memory and Amnesia*, L. S. Cermak (Editor). Lawrence Erlbaum, Hillsdale, NJ, 1982.
11. Owen, A. M., Roberts, A. C., Polkey, C. E., Sahakian, B. J. and Robbins, T. W. Extra-dimensional versus intra-dimensional set shifting performance following frontal lobe excisions, temporal lobe excisions or amygdalo-hippocampectomy in man. *Neuropsychologia* **29,** 993–1006, 1991.
12. Raymond, J. E., Shapiro, K. L. and Arnell, K. M. Temporary suppression of visual processing in an RSVP task: An attentional blink? *J. exp. Psychol.: Human Percept. Perform.* **18,** 849–860, 1992.
13. Richer, F., Décary, A., Lapierre, M-F., Rouleau, I., Bouvier, G. and Saint-Hilaire, J. M. Target detection deficits in frontal lobectomy. *Brain Cognit.* **21,** 203–211, 1993.
14. Richer, F., Décary, A., Chouinard, M. J. and Lepage, M. Working memory capacity is not preferentially affected by frontal excisions. *Brain Cognit.* (in press).
15. Shapiro, K. L., Raymond, J. E. and Arnell, K. M. Attention to visual pattern information produces the attentional blink in RSVP. *J. exp. Psychol.: Human Percept. Perform.* **20,** 357–371, 1994.
16. Stuss, D. T. and Benson, D. F. *The Frontal Lobes*. Raven Press, New York, 1986.
17. Teuber, H. L. The riddle of frontal lobe function in man. In *The Frontal Granular Cortex and Behavior*, J. M. Warren and K. Akert (Editors), pp. 410–477. McGraw-Hill, New York, 1964.
18. Tyler, R. H. Disorders of visual scanning with frontal lobe lesions. In *Modern Neurology*, S. Locke (Editor), pp. 381–393. Little Brown, Boston, 1969.
19. Verin, M., Partiot, A., Pillon, B., Malapani, C., Agid, Y. and Dubois, B. Delayed response tasks and prefrontal lesions in man—Evidence for self-generated patterns of behaviour with poor environmental modulation. *Neuropsychologia* **31,** 1379–1396, 1993.
20. Wilkins, A. J., Shallice, T. and McCarthy, R. Frontal lesions and sustained attention. *Neuropsychologia* **25,** 359–365, 1987.