INTRODUCTION

A number of neuropsychological studies have demonstrated that the frontal lobes play an important role in episodic memory. Evidence for this comes from studies showing that patients with lesions of the frontal lobes are impaired in free recall (Gershberg and Shimamura, 1995; Incisa della Rocchetta, 1986; Jetter et al., 1986; Stuss et al., 1994), as well as in memory for contextual information (Janowsky et al., 1989; Kesner et al., 1994; Kopelman et al., 1997; McAndrews and Milner, 1991; Milner et al., 1991). Initially, it was believed that recognition memory was relatively unaffected by frontal lobe damage (Jetter et al., 1986; Milner et al., 1991). In their meta-analysis, Wheeler et al. (1995) suggested that frontal lobe patients were more frequently impaired on free recall (80% of the cases) than on cued recall (50%), and that cued recall itself was more often impaired than recognition memory (8%). This was attributed to the greater demands that recall places on strategic retrieval processes, compared to recognition memory. Consistently, Gershberg and Shimamura (1995; see also Stuss et al., 1994) have demonstrated that the frontal lobe patients failed to initiate and use organizational strategies at encoding and at retrieval during free recall tasks.

However, recent case studies and group studies have shown that recognition memory can also be impaired following frontal lobe lesions. Moreover, it appears that there is some variability in the recognition performances of frontal lobe patients.

First, the presence of a recognition memory deficit following damage to the frontal lobes may depend on the kind of recognition test used. Parkin et al. (1994) reported that a patient (C.B.) who had suffered from a rupture of an anterior communicating artery (A CoA) aneurysm was impaired on recall tasks, performed well on forced-choice recognition memory tests, but showed lower scores on yes/no recognition tasks. This can be interpreted by reference to dual-process models of recognition memory (Mandler, 1980; Yonelinas, 1994), which suggest that at least two processes contribute to recognition memory: recollection and familiarity. Recollection has been described as a recall-like process, involving the conscious retrieval of an event together with its encoding context. By contrast, familiarity refers to knowing that an event has previously occurred, without any recollection. Parkin et al. (1994) suggested that C.B. was able to use familiarity to discriminate between targets and distractors presented simultaneously, but failed to recollect the items in the yes/no tasks. Consistently, a recent study by Bastin and Van der Linden (2003) has shown that familiarity makes a greater contribution to forced-choice than to yes/no recognition memory, whereas yes/no recognition memory tends to require more recollection.

Second, several case studies have documented a particular pattern consisting of relatively good recall performance (although marked by many intrusions) and impaired yes/no recognition memory performance, with a normal hit rate and a great amount of false recognitions (Delbecq-
retrieved information to a source (e.g., real processes are run, involving the attribution of the memory trace. Finally, post-retrieval monitoring is able to trigger the reactivation of the appropriate characteristics specific to the episode as possible, which does not overlap with the traces of similar episodes. At retrieval, the representation of the episode may be automatically reactivated if the information contained in the retrieval cue match sufficiently the memory trace. Otherwise, one can engage into strategic retrieval processes. These processes imply the building of a description of the episode may be automatically reactivated if the specific details of the event and its associated context into a coherent memory trace, encoding of the specific details of the event and its associated context into a coherent memory trace. Finally, post-retrieval monitoring processes are run, involving the attribution of the retrieved information to a source (e.g., real vs. imagined event) and the verification that what is retrieved is the target episode. In order to do so, one must set a criterion which determines the characteristics that the retrieved episode must have in order to be accepted as the sought-after episode (Derouesné et al., 1990; Parkin et al., 1996; Ward and Parkin, 2000). In those studies, interpretation of this pattern of recognition performance was made in light of the theoretical framework provided by constructive views of episodic memory (see Schacter et al., 1998, for a synthesis). According to these views, appropriate encoding of an episode necessitates the encoding of the specific details of the event and its associated context into a coherent memory trace.

By contrast, Parkin et al. (1993) (Parkin, 1997; Parkin et al., 1996, 1999; Ward, 2003; Ward and Parkin, 2000) suggested that the pathological false recognitions observed in yes/no tasks in two patients (J.B. and M.R.) with a left-sided frontal lesion was a consequence of an encoding deficit. Both patients had a normal hit rate and a great amount of false recognitions. Importantly, their productions of false recognitions were not affected by retrieval manipulations, such as taking the targets and the distractors from different categories or constraining the number of possible “yes” responses. By contrast, encoding manipulations, such as providing deep encoding instructions compared to free encoding, did improve their ability to reject distractors. Therefore, Parkin et al. (1999; Ward, 2003) and Ward and Parkin (2000) suggested that the disorder in these patients may affect the selection of the pertinent information to be encoded, leading to the creation of general memory representations which lack item-specific details.

Third, a pattern of verbal yes/no recognition memory impairment characterized by a poor hit rate and a high false recognition rate was found in a group of patients having a unilateral left frontal damage or a bilateral frontal damage (Stuss et al., 1994). In addition, their response bias did not differ from that of the controls. Stuss et al. suggested that, in these cases, this deficit could be related to either a mild residual aphasia – as evidenced by a reduced score on the Boston Naming Test – or to the fact that the damage extended to the basal forebrain, which is highly connected with the medial temporal lobe structures (see Brosutzky et al., 2000, for a review). In the former hypothesis, the mild language disorder may have interfered with the encoding of the verbal material. In the latter, the deficit may reflect an alteration of the memory processes depending on the medial temporal lobe, the damage of which is known to produce amnesia (Mayes, 2002).

Finally, Swick and Knight (1999) examined the performance of a group of patients with dorsolateral frontal lesions in a continuous yes/no recognition test and also recorded the electrophysiological neural activity of the patients during the memory test. The behavioral results indicated that the frontal patients had an increased false recognition rate, together with a normal hit rate. Moreover, their response bias was abnormally liberal. In addition, the patients as well as the controls did show event-related potential modulations which have been associated with memory retrieval, and more particularly with recollection (Düzel et al., 1997; Smith, 1993). As the event-related potential effect was normal in the frontal lobe patients, Swick and Knight suggested that the memory retrieval process itself was preserved. They proposed that the pattern of recognition memory impairment observed in these patients, with increased false recognitions and
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