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## ANOSOGNOSIA, INTRUSIONS AND 'FRONTAL' FUNCTIONS IN ALZHEIMER'S DISEASE AND DEPRESSION

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**Abstract**—The relationship between anosognosia of memory deficit, intrusions and 'frontal' functions was investigated in 12 Alzheimer (DAT) patients, 12 depressed patients and 24 normal controls. DAT and depressed patients could not be dissociated according to the proportion of intrusion they produced in memory tasks. However, regardless of their clinical diagnosis, patients with anosognosia produced significantly more intrusions than patients without anosognosia, and anosognosia of memory deficit was positively and strongly correlated to the tendency to produce intrusions. By contrast, there was no correlation between intrusions, anosognosia and patients' performance on frontal tasks except for Verbal Fluency. Whereas anosognosia of memory deficit seems indispensable for intrusions, frontal dysfunction must not be considered a necessary condition for intrusions or anosognosia.

**Key Words:** anosognosia; intrusion; memory; Alzheimer's disease; depression.

### INTRODUCTION

Intrusion, the unintentional production in a memory task of fictitious information where the correct information is, for whatever reason, lacking or inaccessible, is frequently observed in several clinical syndromes, including dementia [7, 14–16, 19, 22, 27, 34], major depression [16, 34], amnesia [7, 12, 15, 16, 31, 34] and also, on some occasions, in normal subjects [7, 12, 15, 16, 22, 31].

Since intrusions, like confabulation, reflect the tendency to provide an inappropriate response instead of the correct one, it has been argued that these two phenomena might share some basic common cognitive mechanism and that therefore intrusion can be considered a confabulatory-like behaviour [12, 15]. However, data concerning the impaired mechanism underlying confabulatory-like behaviours are far from being conclusive. Several investigators have discussed the relation between confabulation and awareness of deficit, or anosognosia, in patients with memory deficits (for a review see Ref. [38]).

Unawareness of deficit, anosognosia [1], is observed in several pathological conditions, including hemiplegia, blindness, hemianopia, aphasia, visual agnosia, prosopagnosia and amnesia. There is a general agreement that patients who confabulate not only are often unaware of the inappropriateness of their responses, but also that they are unaware of their

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memory deficit. However, while the available evidence is consistent with the idea that anosognosia is a necessary condition for confabulation, the reverse is not the case. In fact patients have been described in which the resolution of confabulatory state was not paralleled by an equivalent increase of awareness of the existence and severity of memory impairment. In addition, patients who are unaware of their memory deficit do not confabulate on every memory task where their performance is impaired.

On the basis of the fact that these symptoms are frequently associated with a pathology of the frontal lobe, it has been argued that frontally based control functions are impaired in both anosognosia of memory deficit [6, 36, 40, 54] and confabulatory-like behaviour [2, 26, 31, 33, 36, 43, 58].

Well-described phenomena among patients with memory disorders are the heightened susceptibility to the interfering effect of previously studied material on the recall of newly-learned information, i.e. proactive interference (PI) [9, 10, 60–62, 65], and the impairment to eliminate this effect under certain conditions, i.e. release from PI [8, 18, 28, 29, 42, 56, 63]. Prior list intrusions are usually the result of these deficits. Since great susceptibility to PI and failure to release from PI are memory disorders typical of Korsakoff amnesia, it has been argued [18, 42] that such disorders may result from the combination of Korsakoff's memory deficits and impaired frontal functions. However, recently Kopelman [32] did not find any significant correlation between Alzheimer and Korsakoff patients' ability to release from PI and their performance on any of eight 'frontal' tests.

This study addresses the relationship between intrusions, anosognosia and frontal functions in Alzheimer's and depressed patients. Lack of awareness of cognitive deficits, including memory impairment have been frequently reported in DAT, whereas in depression this issue has been little investigated.

## METHOD

*Subjects.* Forty-eight subjects participated in the present study: 12 depressed patients (DD; 6 female, 6 male); 12 patients with DAT (6 female, 6 male); 12 middle-aged normal controls (M-NC; 6 female, 6 male); and 12 elderly normal controls (E-NC; 6 female, 6 male). The M-NC subjects were utilised for comparison with DD patients; the E-NC subjects for comparison with DAT patients.

The DD patients, who carried a diagnosis of primary depression using the DSM-III—R criteria, were not receiving any pharmacological treatment for their illness.

The diagnosis of probable DAT was made according to the criteria suggested by McKhann and his associates [39]. A number of laboratory tests (e.g. CT brain scan, urinalysis, folate levels) were performed to rule out various viral, metabolic or traumatic causes of dementia. Patients with a history of severe head injury, alcoholism, or serious psychiatric illness were excluded from this group. Patients with a score of 5 or greater on the Hachinski scale [20] were excluded to reduce the possibility of including multi-infarct dementia.

Normal controls were either spouses of patients or other individuals who volunteered to participate in the research projects of our laboratory. Informed consent was obtained from all subjects participating to the study.

Table 1 shows the mean age, years of education, Mini-Mental Status Examination (MMSE) scores (Folstein *et al.*, 1975) for the four subject groups as well as the Blessed Dementia Rating Scale (BDRS) scores [5] and the Montgomery and Asberg Depression Rating Scale [41] for the two patient groups. One-way analyses of variance (ANOVAs) were performed on each of these demographic and psychometric variables. Significant group effects were found for age [ $F(3, 44)=9.87, P<0.0001$ ], MMSE scores [ $F(3, 44)=15.51, P<0.0001$ ], BDRS scores [ $F(1, 22)=68.72, P<0.0001$ ] and MADRS scores [ $F(1, 22)=67.32, P<0.0001$ ]. Group comparisons\* indicated that, as expected, the M-NC subjects were significantly younger than the E-NC subjects [ $F(22)=6.4; P<0.01$ ] and the DD patients were significantly younger than patients with DAT [ $F(22)=3.2; P<0.05$ ]. DD patients had MMSE scores significantly lower than M-NC [ $F(22)=3.5; P<0.05$ ], and DAT patients had MMSE scores significantly lower than E-NC [ $F(22)=11.3; P<0.0001$ ], but the two patient groups did not differ significantly

\*Sheffé's *F* test was used for all *post-hoc* comparisons in this study.

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