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BRIEF COMMUNICATION

Pattern of cognitive dysfunction in depressive patients during maintenance electroconvulsive therapy

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ABSTRACT

Background. Objective data regarding adverse cognitive deficits associated with maintenance electroconvulsive therapy (M-ECT) are lacking. This study examined the cognitive state of depressive patients during M-ECT.

Method. A cross-sectional study was carried out in 11 depressive patients in remission, all with a DSM-IV diagnosis of major depressive disorder. The mean number of previous ECT sessions was 36·1, and the mean intersession interval was 52·7 days. A group of 11 patients who had not received ECT was selected for comparison and matched for diagnosis, sex, age and years of schooling. All subjects were assessed using a complete neuropsychological battery including memory, attention and frontal function tests.

Results. Groups did not present differences in long delay verbal recall. Encoding of new information and results on the frontal function tests were significantly lower in the M-ECT patients.

Conclusion. Depressed patients preserve long-term memory, but suffer short-term memory impairment and frontal function alteration during M-ECT. Further longitudinal studies are necessary to determine the influence of M-ECT on non-memory functions and different memory subtypes.

INTRODUCTION

Maintenance electroconvulsive therapy (M-ECT) is an effective treatment for the prevention of relapse and recurrence in patients with major psychiatric disorders, especially mood disorders (Petrides *et al*. 1994; APA, 2001; Sackeim *et al*. 2001). It has proved to be a safe option particularly useful in drug-resistant patients (Rabheru & Persad, 1997; Tew *et al*. 1999) and in medically compromised subjects who are sensitive to the toxic effects of medication (Sackeim *et al*. 2001). M-ECT does not present any adverse physical effects other than those found in acute courses of ECT (Jaffe *et al*. 1990; Fink *et al*. 1996), but the risk of cognitive dysfunction associated with the treatment remains unknown. Previous data are based on case reports (Grunhaus *et al*. 1990; Devanand *et al*. 1991; Barnes *et al*. 1997), retrospective studies without a control group (Brodaty *et al*. 2000), subjective evaluations of cognitive dysfunction after treatment (Vanelle *et al*. 1994) or studies based on global cognitive screens such as the Mini-Mental State Examination (Thienhaus *et al*. 1990). Information has also been extrapolated from acute ECT cases: most reports have shown a transient memory dysfunction that disappears 3 to 6 months after the completion of treatment (Rami-González *et al*. 2001). Other cognitive functions such as attention or frontal functions have not been

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widely studied, though they appear to be less affected than memory after an ECT course (Calev *et al*. 1995). Consequently, no objective data are available on the adverse cognitive effects during continuation ECT sessions. Since the time between treatments is much longer during M-ECT, fewer cognitive side effects would be expected than during an acute course of ECT. The aim of this study was to determine the profile of cognitive deficits in depressive patients during an M-ECT protocol.

METHOD

Eleven consecutive drug-resistant patients participating in the M-ECT programme were selected for inclusion in the study by the Institute of Psychiatry and Psychology at the Hospital Clínic, Barcelona. Nine patients met DSM-IV criteria for recurrent major depressive disorder, and two for a single episode of major depressive disorder with melancholic features. The mean duration of illness was 12·8 years (S.D. = 9·2) and the mean number of depressive episodes was 3·1 (S.D. = 2·4). Mini-Mental State Examination scores were between 25 and 29 in all cases. The frequency of sessions was adapted to each patient's needs. The mean intersession interval in the sample was 52·7 days (S.D. = 16·8; range 30–90 days). Patients receiving more than one session per month were not included in the present study. The mean of total previous ECT sessions was 36·1, and the mean time of M-ECT was 27·2 months. Nine of the 11 finished their acute ECT course 6 months before assessment; in the two remaining patients 5 months had passed since the ECT course. Patients were treated with the standard bilateral frontotemporal electrode placement with a customized MECTA spectrum device. Anesthesia consisted of atropine (0·4 mg), succinylcholine and thiobarbital.

Eleven depressive subjects who had never received ECT were used as a control group, matched with the M-ECT patients regarding sex, age and years of schooling. A difference in age of ± 3 years was accepted. Six of these patients met criteria for recurrent major depressive disorder and five for a single episode of major depressive disorder with melancholic features. The mean duration of illness was 11·1 years (S.D. = 7·3) and the mean number of depressive episodes was 1·7 (S.D. = 0·8). Most patients were treated with psychotropic medications and the drug type used in both groups was similar. In the M-ECT group three subjects were treated with selective serotonin reuptake inhibitors (SSRI), three with selective noradrenaline reuptake inhibitors (SNRI), and three with tricyclic antidepressants. In addition, one patient was taking benzodiazepines, and one neuroleptics. In the control group, four patients were treated with SSRI, three with SNRI, two with tricyclics and two subjects were taking benzodiazepines.

Written informed consent was obtained from all patients and controls after the procedures had been fully explained. Clinical and cognitive assessment was administered to all subjects. ECT patients were assessed the day of the ECT session, in order to guarantee maximum intersession time. Clinical state was measured with the Hamilton Depression Rating Scale (HDRS) (Hamilton, 1967). All patients had been in remission (HDRS < 8) for at least 3 months before neuropsychological assessment. The cognitive battery included an assessment of global cognitive function by Mini Mental State (MMSE) (Folstein *et al*. 1975). Memory subtypes were evaluated with the Logical Memory test of the Wechsler Memory Scale (WMS) (Wechsler, 1945) and the Auditory Verbal Learning Test (RAVLT) (Rey, 1964). To assess the encoding of new verbal information or short-term memory we used Logical Memory part I and total learning of the RAVLT. Information retention or long-term memory was assessed by delayed recall at 20 min of the logical memory story and the words in the AVLT memory list. The Digits Forward test on the Wechsler Intelligence Scale for Adults (Wechsler, 1955) was used to assess selective attention and the Trail Making Test – part A (Army Individual Test Battery, 1964) assessed simple motor velocity in addition to attention. The Digit-Symbol Coding test from the WAIS was used to determine visuomotor speed. Frontal functions were assessed by the Trial Making Test (part B) (Army Individual Test Battery, 1964), the Tower of Hanoi (Goldberg *et al*. 1990) and the Digits Backward test, measuring mental flexibility, planning, and working memory respectively. Verbal fluency, measured by the FAS-test (Borkowski *et al*. 1967), was also used as an indication of frontal function.

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Table 1. *Characteristics and case-control comparisons using Wilcoxon Rank test*

	M-ECT group		Comparison group		Wilcoxon Rank test	
	Mean	(S.D.)	Mean	(S.D.)	*Z*	*P*
Characteristic						
Age (years)	63·9	(8·8)	63·8	(9·1)	−0·1	0·92
Education (years)	8·0	(2·4)	9·3	(2·9)	−1·4	0·17
HDRS	3·5	(2·7)	2·5	(2·7)	−1·6	0·17
MMSE	27·5	(1·6)	28·2	(1·7)	−0·9	0·32
Total ECTs, *N*	36·1	(11·1)				
Time in ECT treatment (months)	27·2	(17·7)				
Neuropsychological battery						
Memory						
RAVLT Total Learning	14·3	(4·7)	16·3	(4·2)	−0·8	0·406
RAVLT (% Verbal Recall in delay)	74·7	(27·9)	72·2	(20·4)	−0·3	0·721
Logical Memory (LM I)	5·2	(2·6)	8·1	(1·9)	−2·7	0·01
(LM I − delayed LM) (raw score)	1·3	(0·9)	2·4	(1·6)	−1·8	0·066
Frontal tests						
Trail Making 'B' (s)	219·0	(88·4)	152·7	(66·8)	2·4	0·035
FAS-test (total words)	20·6	(7·0)	27·9	(8·6)	−2·5	0·029
Digits Backward	3·4	(0·7)	4·3	(1·2)	−2·8	0·020
Digit Symbol Coding	19·4	(7·5)	31·4	(14·4)	−2·7	0·023
Tower of Hanoi	13·8	(5·9)	10·6	(3·7)	−1·2	0·233
Attention						
Trail Making 'A' (s)	91·6	(77·1)	67·4	(42·7)	−1·5	0·12
Digits Forward	5·0	(1·1)	5·1	(1·4)	−0·1	0·914

Statistical analysis was performed using SPSS for Windows V 9.0. The Wilcoxon Rank test was used for case–control comparison of cognitive measures. Non-parametric Spearman correlations were used to assess association within M-ECT group. Two-sided *P* values are given.

RESULTS

The mean age of the sample was 63·8 years (S.D. = 8·7; age range: 44–77). There were eight females and three males in each group. No differences in years of illness duration ($Z = -0{\cdot}2$, $P = 0{\cdot}84$) or number of depressive episodes ($Z = -1{\cdot}6$, $P = 0{\cdot}11$) were found between groups, nor regarding age, sex or years of schooling.

The M-ECT group scored lower on the encoding of new information, as expressed by raw scores on the Logical Memory (LM) I and Total Learning tests on the AVLT (Mitrushina *et al.* 1999). Only the LM I score was significantly lower. However, the M-ECT group obtained higher long-term memory scores than controls, but the two groups did not differ significantly in terms of variation in the direct scores between LM I and delayed LM or in percentage retention on the AVLT. The two groups also showed similar scores on Trial Making A and Digits Forward tests. With the exception of the Tower of Hanoi, all frontal test scores were significantly lower in the M-ECT group. Results are presented in Table 1.

There was no significant correlation between cognitive measures and number of total ECT sessions, total time in M-ECT, or time between treatments, or between cognitive measures and age, years of schooling, number of episodes or years of evolution of illness. The small sample size may have limited the power to find significant correlations. The dispersion graph was analysed visually, and no significant outliers were found.

DISCUSSION

No differences were observed in long-term memory or in attention function between depressed patients undergoing M-ECT and matched comparison subjects. However, encoding of new information and performance on most tests of frontal function was significantly impaired in our M-ECT patients. Compared with controls, M-ECT patients showed alternations in verbal fluency, mental flexibility, working memory and visuomotor speed.

Memory impairment is the main neuropsychological problem associated with acute ECT, but the specific subtypes of memory dysfunction

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associated with M-ECT remain unknown. The ability to consolidate information and recall it after a delay is clearly deficient after an ECT course (Frith *et al.* 1983; Hasse-Sander *et al.* 1988) and it is significantly affected for 3 to 6 months after an acute treatment. Long-term memory dysfunction is associated with the medial temporal lobe, though its precise mechanism remains unclear. Several explanations have been proposed: an abnormal long-term potentiation mechanism, excessive release of excitatory amino acids and the activation of their receptors such as N-methyl-D-aspartate (NMDA), decreased cholinergic transmission, increased cerebral blood pressure and reductions of regional cerebral blood may contribute to this transient memory dysfunction after an acute course of continuous ECT (Rami-González *et al.* 2001). Although any or all of these phenomena may be present in M-ECT, the absence of long-term memory dysfunction in our sample may be explained by the long interval between sessions during continuation ECT. Perhaps this long period between ECTs helps the recovery of the neurobiological bases involved in memory dysfunction. Interestingly, the M-ECT group obtained better scores than the matched group in long-term memory. Absence of an information retention dysfunction may improve the lifestyle of patients during M-ECT treatment and may aid their functional adaptation. It is well established that short-term memory is less affected than long-term memory after acute ECT. Within a few weeks of treatment, patients recover the ability to encode new information (Steif *et al.* 1986; Sackeim *et al.* 2000; Zervas *et al.* 1993). Taking evidence-based knowledge from previous ECT course studies as our starting point, we predicted that this memory subtype would not be disturbed as a consequence of M-ECT, since the mean intersession period was sufficient for short-term memory recovery. One possibility is that the encoding information dysfunction in our sample was influenced by the patients' inability to make associations in encoding, which may be influenced by frontal dysfunction (Shimamura *et al.* 1991).

Contrary to our expectations, the frontal functions of M-ECT patients were significantly impaired during treatment. These results must be considered in the context of the limitations of our study, which are common to M-ECT research in general. The design of experimental studies in this area is difficult, since M-ECT is a specific continuation treatment for certain patients. All patients were in remission and there was no difference in their clinical symptomatology at the moment of cognitive assessment. The small sample size probably prevented the emergence of statistical differences between certain relevant clinical variables, such as number of episodes. The fact that the M-ECT group were seriously ill and drug-resistant may have influenced the cognitive differences between the groups. A frontal lobe dysfunction is clearly implicated in depressive patients. The higher number of episodes and the more serious depression in the M-ECT subjects may partly explain the differences between the groups in frontal functions. However, all patients were in remission when neuropsychological assessment was made. The cognitive profile of depressive patients in remission is not clear (Austin *et al.* 2001). Some studies have found that seriously depressed patients show global memory dysfunction during remission (Abas *et al.* 1990; Marcos *et al.* 1994), but some found no residual deficits (Calev *et al.* 1986; Bazin *et al.* 1994); others have even reported improved functions after recovery from depressive symptoms (Peselow *et al.* 1991). However, few studies have found altered frontal functions in addition to memory dysfunction (Beats *et al.* 1996; Paradiso *et al.* 1997) in depressive patients in remission.

One of the major difficulties in the design of ECT cognitive studies is the influence of many factors that contribute to an individual's cognitive impairment. These factors include the effect of age, the interference of symptomatology, and the use of concurrent medications. In our study, all patients had been in remission for the 3 months prior to cognitive assessment to ensure that the influence of mood disorder on cognitive functions would be minimal. Medication and age were also similar in the two groups. The effects of the small differences between groups in tricyclic antidepressants used would only have contributed to memory dysfunction in M-ECT patients (Parde, 1994; Healy, 2001) but do not seem to explain frontal function differences. Therefore, none of the controlled factors described above seem able to explain the frontal function discrepancy between groups.

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The high number of ECT sessions in these patients is another factor which could perhaps be related to the frontal dysfunction. Very little information is available on the effects of ECT on non-memory functions (Calev *et al*. 1995). Studies reported to date are based on the acute course and report no effects (Small *et al*. 1986; Lawson *et al*. 1990) or fewer effects than on memory (Taylor *et al*. 1985; Calev *et al*. 1991). Recently, the APA stated that there is no evidence that ECT results in lasting impairments of executive functions (APA, 2001).

Although retrospective studies based on acute courses of ECT indicate that there is no memory impairment at long-term follow-up (Cohen *et al*. 2000) or any alteration in brain structure associated with ECT (Devanand *et al*. 1994; Ende *et al*. 2000), further specific studies are necessary to rule out the effects of M-ECT on non-memory cognition, especially on frontal functions.

The advanced age of many patients in our sample deserves some comment. Although ECT can be used safely and effectively in geriatric subjects, these patients are more vulnerable to adverse cognitive effects (Stoudemire *et al*. 1993; Zervas *et al*. 1993; Tew *et al*. 1999; Brodaty *et al*. 2000) and may be more susceptible to developing frontal alterations associated with ECT. Pettinati & Bonner (1984) evaluated mental flexibility in depressed geriatric patients with a history of ECT. In the Trail Making B test, the over-65s with a history of ECT performed worse than older patients who had never received ECT or younger depressed patients regardless of their ECT history. Even though we did not find a significant correlation between cognitive measures and age, further studies with different ages samples and with other diagnoses are necessary to clarify ECT effects on non-memory functions in certain patients.

In summary, results of the present study indicate cognitive profile of our M-ECT sample was characterized by normal long-term memory while frontal functions and short-term memory were impaired. However, further studies are required to establish the cognitive state in patients during M-ECT, as this will help to determine their quality of life and everyday functioning during treatment.

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