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ECT and cerebral atrophy

A COMPUTED TOMOGRAPHIC STUDY

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The case-notes of 41 elderly depressives who underwent computed tomography were examined and the ECT history of each patient was assessed. No association was found between ECT and global cortical atrophy or ventricular size, but a significant relationship was demonstrated between frontal lobe atrophy and ECT.

Key words: Depression – electro-convulsive therapy – computed tomography – cortical atrophy – frontal lobes.

The possibility that electro-convulsive therapy (ECT) has lasting effects on the brain has frequently been raised. Research has focussed on the long-term psychological effects of ECT especially with regard to memory (*Halliday et al.* (1968), *Squire & Chace* (1975); *Weeks et al.* (1980)). There is little information about morphological changes in the brain following ECT in man, although animal studies have showed structural changes in neurones and glial cells particularly in the frontal area following electrically induced convulsions (*Hartelius* (1952), *Ferraro et al.* (1946)). Computed tomography (CT) offers a non-invasive way of examining structural changes *in vivo*. In order to investigate any association between ECT and cerebral atrophy we re-examined the data of *Jacoby & Levy* (1980) who looked at the relationship of CT appearance to clinical state in 41 elderly patients with a primary diagnosis of affective disorder.

METHOD

The patients were 41 consecutive admissions to the psychogeriatric ward of the Bethlem Royal with a primary diagnosis of affective disorder. All 41 case-notes were traced and re-examined. Two patients with a history of excessive alcohol intake and one with a history of syphilis were excluded from the analysis. The case-notes of the remaining 38 patients were examined in order to determine the presence or absence of ECT in their treatment history and the number of applications. No patient had received ECT in the 6 months prior to computed tomography, and none had had a leucotomy or insulin coma therapy. It was impossible to calculate the exact number of ECTs received in every case as several patients had been treated at various other hospitals, often many years earlier. An estimate of the number of treatments was therefore calculated on the arbitrary basis of eight applications per course of treatment in cases where there was definite evidence that a course had been administered but the exact number of applications was not known.

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Table 1. *Presence or absence of frontal, parietal and insular atrophy in patients with and without a history of ECT*

	Atrophy					
	Frontal		Parietal		Insular	
	Absent	Present	Absent	Present	Absent	Present
No ECT	11	4	7	8	8	7
ECT	7	15	4	18	11	11
	$\chi^2 = 6.15$, 1 df, $P < 0.02$		$\chi^2 = 3.46$, 1 df, n.s.		$\chi^2 = 0.04$, 1 df, n.s.	

For analysis ECT was considered by presence or absence of ECT in the patient's history and estimated number of applications. Patients were assigned to one of six groups according to the estimated number of applications as follows: 1–6, 7–12, 13–24, 25–36, over 36.

The technique of scan analysis and assessment of cortical atrophy has been described in detail by *Jacoby et al.* (1980). Cortical atrophy was rated blindly by a neuroradiologist on a four-point scale for each of the five cortical areas – frontal, temporal, insular, parietal and occipital.

The relationship of CT changes to ECT was assessed by means of chi-squared test and the Mann-Whitney U test for non-parametric data.

RESULTS

Twenty-two patients (mean age 71.5) had received ECT and 15 patients (mean age 73.8) had not. Information was insufficient in one case who was excluded from further analysis. Twenty-nine out of 37 patients were rated as having some degree of cortical atrophy.

No relationship was shown between ventricular measures and ECT. However, there was an association between measures of cortical atrophy and ECT (Table 1).

Table 1 shows the relationship between history of ECT and atrophy in the frontal, parietal and insular regions of the brain. Temporal or occipital atrophy was present in only four patients and no statistical evaluation could be made. A chi-square test indicates a significant association between history of ECT and presence of frontal atrophy ($P < 0.02$) but not with insular atrophy. The association between ECT and parietal atrophy just failed to reach significance at the 0.05 level.

These differences were not due to age as there was no significant difference between the ECT-treated groups (mean age 71.5) and those not receiving ECT (mean age 73.8).

The majority of patients had received bilateral ECT. The number in whom it could be stated with certainty that they had received only unilateral ECT was too small for a valid comparison to be made between the two groups.

Table 2 shows the relationship of ECT to frontal atrophy in more detail.

Table 2. *Estimated number of ECT applications in patients with and without cortical atrophy in the frontal area*

	No. of ECT applications					
	0	1–6	7–12	13–24	25–36	36 +
No atrophy	11	2	1	2	0	2
Atrophy	4	2	5	3	2	3

Mann-Whitney U test = 100.5, two-tailed, $P < 0.05$.

The estimated number of ECT applications given to patients with and without cortical atrophy is shown.

The Mann-Whitney U test for non-parametric data showed that patients with frontal lobe atrophy had received more applications of ECT ($P < 0.05$).

DISCUSSION

The results suggest an association between history of treatment with ECT and cortical atrophy in the frontal region. One possible explanation for these findings is that ECT causes cortical atrophy. An alternative is that there may be a sub-group of patients with depressive symptoms who are more prone to develop frontal atrophy and who are also more likely to be given ECT for clinical reasons. Additionally, these patients might be relatively unresponsive to treatment, perhaps because of the organic changes observed here, and as a consequence might receive more ECT than the other group.

The relationship between ECT and cerebral atrophy has also been considered by *Weinberger et al.* (1979) who performed CT scans on 75 chronic schizophrenics. Measuring the width of fissures and sulci they found significantly greater cortical atrophy in 17 ECT-treated patients compared with 58 patients who had not received ECT ($P < 0.01$). The only attempt at a prospective study of the putative effect of ECT on brain structure observable on CT scans was undertaken by *Menken et al.* (1979). In a single case study of a 30-year-old woman who had 10 ECT applications over 45 minutes a CT scan performed 3 hours after the last application showed no 'haemorrhages or oedema', a study which, in our opinion, does not help to resolve the issue of the possible role of ECT in causing structural damage to the brain.

CONCLUSION

The *ad hoc* nature of this study and the difficulty in obtaining an accurate assessment of the number of applications of ECT do not permit us to claim an unequivocal association between ECT and structural change in the brain. Nevertheless, this is a question of such importance that, in our opinion, the finding of a relationship between frontal atrophy and ECT justifies this brief report. It emphasizes the need for a more detailed investigation, with larger numbers of patients including a younger age group.

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