A Preliminary Magnetic Resonance Imaging Study of ECT-Treated Depressed Patients

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Introduction

Until the introduction of magnetic resonance imaging (MRI), all in vivo human studies, except for a single case report (Menken et al. 1979), of the potential effects of electroconvulsive therapy (ECT) on brain structure had been retrospective comparisons of patients (with and without a history of prior ECT) who had had computerized tomographic scans. An association between prior ECT and lateral ventricular enlargement in schizophrenics (Weinberger et al. 1979) and cortical atrophy in elderly depressives (Calloway et al. 1981) was reported. Kolbeinsson et al. (1986) found that depressed patients had larger ventricle/brain ratios and greater cortical atrophy than normal controls but there was no association with prior ECT.

The first prospective study to use serial MRI brain scans (Coffey et al. 1988b) found no ECT-related structural brain changes in 10 patients, although a number of brain abnormalities were noted even at the pre-ECT scan. Similarly, Price et al. (1988) found no MRI scan changes with ECT in nine patients. We report a prospective investigation of possible ECT-related structural brain changes using serial MRI.

Methods

Seven psychotropic drug-free inpatients with major depressive disorder by the Research Diagnostic Criteria (Spitzer et al. 1978) and referred for ECT were studied. One subject (No. 1) had trifluoperazine treatment up to 3 weeks prior to ECT; all others had tricyclic antidepressant treatment which was discontinued at least 7 days prior to ECT. Exclusion criteria were a history of drug abuse, alcoholism, head trauma or other neurological condition, ECT in the preceding 12 months, or implanted medical devices (e.g., pacemakers, prosthetic valves, replaced joints) as these pose hazards during MRI. Pre-ECT screening included anesthesia and cardiology consultations, as well as blood work, spinal x-ray films, and any other pertinent tests. In addition to consent for anesthesia and ECT, separate informed consent for the present study was obtained. Patients underwent MRI scanning at the University of Michigan MRI Facility with a Diasonics (0.35 Tesla) or a GE (1.5 Tesla) MRI scanner. All pre-ECT scans were done with the Diasonics scanner, but in three patients (5,6 and 7) (Table 1) the post-ECT scans were done on the more powerful GE scanner. Nondominant unilateral ECT was given three times a

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week under anesthesia with intravenous methohexital (1 mg/kg body weight) and succinylcholine (0.75 mg/kg body weight). Continuous ventilation with a mixture of oxygen and room air was maintained during ECT to avoid anoxia. MRI scanning was repeated within 1 week after the end of the course of ECT.

Results

A radiologist (*A.M.A*), experienced in interpreting MRI studies of the brain and blind to the sequence (i.e., pre- or posttreatment) of scanning, read the T2-weighted MRI scans making judgments about ventricular size, cortical atrophy, and any other discrete lesions, focusing particularly on the relative differences between the two scans for each patient. Results are summarized in Table 1. Five patients showed multiple areas of signal hyperintensity located mainly in the periventricular white matter. These were found prior to ECT and remained unchanged following treatment. No other brain changes were found at the post-ECT scanning. Some subjects showed age-appropriate brain atrophy which remained unaffected after ECT.

Discussion

This pilot study found no acute ECT-related structural brain changes on MRI scanning; this agrees with similar prospective MRI studies (Coffey et al. 1988b, Price et al 1988). The patchy white matter lesions (PWML) noted are similar to those observed in other older populations studied with MRI (Bradley et al. 1984). Aging and cardiovascular risk factors (such as hypertension, diabetes) may play a role that is unclear. As the normative brain MRI data were acquired from populations that were not screened for depressive illness, a meaningful association of these findings and affective illness cannot be ruled out. It has recently been suggested that the white matter lesions are more frequent in late-onset than in early-onset depressives (Coffey et al. 1988a). Because older depressives often exhibit the other biological findings of depression (e.g., hypercortisolemia), these may be speculated to have a bearing on the PWML. Increased VBR in depressives has been correlated with mean 24-hr urinary-free cortisol (Kellner et al. 1984) but it is unknown if discrete focal brain changes may occur with exposure to corticosteroids.

Six of our seven patients had the dexamethasone suppression test (DST) prior to starting ECT and of these, five were in the nonsuppressor range. Though this requires cautious interpretation in light of the short drug-free period prior to the DST and does not in itself prove a hypercortisolemic state, it may possibly indicate a shift in hypothalamic pituitary adrenal axis functioning and be a lead for further investigation not only of the white matter lesions but

Table 1. Results of T2-Weighted MRI Scans

Pt	Age	Gender	No. ECT	Pre-ECT MRI	Post-ECT MRI	DST
1	64	M	10	Normal	No change	NS
2	62	F	9	Multiple PWML	No change	—
3	75	F	6	No atrophy, many PWML	No change	NS
4	71	F	7	Two PWML left side	No change	S
5	38	F	7	Normal	No change	NS
6	70	F	7	Mild atrophy, many PWML	No change	NS
7	79	F	10	Mild atrophy, many PWML	No change	NS

PWML = patchy white matter lesions; DST = dexamethasone suppression test; NS = nonsuppressor; S = suppressor.

of other changes in brain morphology among depressives.

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Lactate-Induced Electrolyte Changes in the Cerebrospinal Fluid of Rabbits

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Introduction

In 1967, Pitts and McClure reported that sodium lactate infusions could precipitate panic attacks in susceptible individuals. Since then, a number of studies have investigated the mechanism by which lactate induces panic attacks by measuring changes in plasma concentrations of calcium (Liebowitz et al. 1985), phosphate (Gorman et al. 1986), and chloride ions (George and Jimerson 1986). However, a differential effect of lactate on these measures has not been established between panicking and nonpanicking subjects. Furthermore, the effects of lactate on brain or cerebrospinal fluid (CSF) biochemistry

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