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Electroconvulsive therapy

Electroconvulsive therapy (ECT), formerly known as electroshock therapy and often referred to as shock treatment, is a psychiatric treatment in which seizures are electrically induced in patients to provide relief from psychiatric illnesses.

It is the only currently used form of shock therapy in psychiatry.

ECT is often used with informed consent as a last line of intervention for major depressive disorder, mania and catatonia.

A round of ECT is effective for about 50% of people with treatment-resistant major depressive disorder, whether it is unipolar or bipolar.

Follow-up treatment is still poorly studied, but about half of people who respond relapse within 12 months.

Aside from effects in the brain, the general physical risks of ECT are similar to those of brief general anesthesia.

Immediately following treatment, the most common adverse effects are confusion and memory loss.

ECT is considered one of the least harmful treatment options available for severely depressed pregnant women.

A usual course of ECT involves multiple administrations, typically given two or three times per week until the patient is no longer suffering symptoms. ECT is administered under anesthetic with a muscle relaxant.

Electroconvulsive therapy can differ in its application in three ways: electrode placement, frequency of treatments, and the electrical waveform of the stimulus. These three forms of application have significant differences in both adverse side effects and symptom remission. Placement can be bilateral, in which the electric current is passed across the whole brain, or unilateral, in which the current is passed across one hemisphere of the brain. Bilateral placement seems to have greater efficacy than unilateral, but also carries greater risk of memory loss.

After treatment, drug therapy is usually continued, and some patients receive maintenance ECT.

ECT appears to work in the short term via an anticonvulsant effect mostly in the frontal lobes, and longer term via neurotrophic effects primarily in the medial temporal lobe.

Case series

Twenty-four patients with severe MDD and 14 healthy controls were enrolled in this study. Eight ECT sessions were conducted for MDD patients using brief-pulse square-wave signal at bitemporal locations. To investigate the regional cerebral blood flow in MDD patients before and after ECT treatments by resting-state functional magnetic resonance imaging (rs-fMRI), the patients were scanned twice (before the first ECT and after the eighth ECT) for data acquisition. Afterward, we adopted fractional amplitude of low-frequency fluctuations (fALFF) to assess the alterations of regional brain activity.

Compared with healthy controls, the fALFF in the cerebellum lobe, parahippocampal gyrus, fusiform gyrus, anterior cingulate gyrus, and thalamus in MDD patients before ECT (pre-ECT) was significantly increased. In another comparison, the fALFF in the cerebellum anterior lobe, fusiform gyrus, insula, parahippocampal gyrus, middle frontal gyrus, and inferior frontal gyrus in pre-ECT patients was significantly greater than the post-ECT fALFF.

Only two rs-fMRI scans were conducted at predefined times: before the first and after the eighth ECT treatment. More scans during the ECT sessions would yield more information. In addition, the sample size in this study was limited. The number of control subjects was relatively small. A larger number of subjects would produce more robust findings.

The fALFF of both healthy controls and post-ECT patients in cerebellum anterior lobe, fusiform gyrus, and parahippocampal gyrus is significantly lower than the fALFF of pre-ECT patients. This finding demonstrates that ECT treatment is effective on these brain areas in MDD patients ¹⁾.

A study of Sorri et al. from the Tampere University Hospital in Finland, included thirty patients suffering from major depressive disorder (MDD). Their serum and plasma brain derived neurotrophic factor (BDNF) levels were examined before electroconvulsive therapy (ECT) (baseline) and after the first, fifth, and last ECT session. The severity of the depression and the response to ECT were measured with Montgomery-Asberg Depression Rating Scale (MADRS).

Electroconvulsive therapy caused no significant changes in serum BDNF levels. Plasma BDNF levels decreased during the fifth ECT session between the baseline and the 2-hr samples (p = 0.019). No associations were found between serum or plasma BDNF levels and remission. The correlations between plasma and serum BDNF levels in each measurement varied between 0.187 and 0.636.

Neither serum nor plasma BDNF levels were systematically associated with the clinical remission. However, the plasma BDNF levels somewhat varied during the ECT series. Therefore, the predictive value of BDNF for effects of ECT appears to be at least modest ²⁾.

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