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## Electroconvulsive Therapy

### Series Editor:

**Jerald Kay, MD**

*Professor and Chair, Department of Psychiatry, Wright State University School of Medicine, Dayton, OH*

### Contributing Authors:

**Victor R. Knapp, MD**

*Assistant Professor, Department of Psychiatry, Wright State University School of Medicine, Chief Clinical Officer, Twin Valley Behavioral Healthcare, Dayton, OH*

**David Bienenfeld, MD**

*Professor and Vice Chair, Director of Residency Training, Department of Psychiatry, Wright State University School of Medicine, Dayton, OH*

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# Electroconvulsive Therapy

Electroconvulsive therapy (ECT) is one of the most controversial treatments in modern psychiatry. It has been the subject of movies, documentaries, protests, and political referendums. Few treatments have come under such scrutiny or vilification. ECT's protagonists emphasize its safety and efficacy. ECT's antagonists claim that it causes irreversible brain damage and severe memory loss. Researchers in the field of ECT are enabling clinicians to address these issues in objective and meaningful ways. Apart from the rhetoric and polemics, a powerful science has emerged for the ECT practitioner and patient. The breadth of this work provides for a comprehensive discussion about efficacy, safety, side effects, patient selection, and treatment methods.

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## MECHANISM OF ACTION

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- **What causes the ameliorative action of electroconvulsive therapy?**

There is a wide array of ideas regarding ECT's action. However, because there are multiple, simultaneous changes induced by each ECT treatment and over a course of treatment, it is difficult to separate one change from another to determine a single locus of action. Currently, no unified theory exists as to how ECT exerts its action; a myriad of work looking at diverse systems exists in the literature. Contemporary theories regarding ECT's mode of action fall into 3 primary groups: 1) enhancement of monoamine transmission (eg, dopamine, norepinephrine, serotonin), 2) neurotrophic effects, and 3) anticonvulsant effect.

### MONOAMINE TRANSMISSION

A variety of serotonin receptor changes have been reported in studies measuring the effects of long-term ECT. Many or most of these involve animal studies, where applicability to the human brain is far from certain. These changes are shown in **Table 1**. Studies have suggested that the actual concentration of serotonin is not as critical or central a locus of action as in antidepressants.<sup>1</sup> Some studies indicate a relationship between the serotonergic system and the opiate system, suggesting that ECT may exert its action by affecting this system.<sup>2</sup>

A number of studies fail to show any convincing effect of ECT on norepinephrine turnover. Plasma cat-

echolamines increase in response to ECT but are not altered with repeated ECT. Mann et al<sup>3</sup> found that the degree of catecholamine increase correlated with clinical response. This suggests that ECT's antidepressant action may be related to its effect on catecholamine release via the sympathetic nervous system. However, this finding does not mean this effect is part of ECT's antidepressant action.<sup>1</sup>

ECT is useful in treating patients with Parkinson's disease, suggesting that it increases dopaminergic transmission in the basal ganglia. Indeed, most evidence does indicate that ECT causes an increase in dopaminergic activity. This seems to be mostly due to an increase in dopamine turnover, although a receptor effect cannot be excluded.<sup>1</sup>

### NEUROTROPIC EFFECTS

Using animal models, Duman et al<sup>4</sup> have suggested that ECT works by activating the adenylyl cyclase system and thereby increasing brain-derived neurotrophic factor (BDNF) and its receptor, TrkB, in the hippocampus and cerebral cortex. BDNF has been shown to increase synaptic strength, survival, and growth of adult neurons. BDNF has been shown to increase norepinephrine and serotonin turnover and sprouting of serotonergic terminals.<sup>1</sup> Duman et al<sup>4</sup> hypothesize that ECT reverses the atrophy of stress-vulnerable neurons or protects them from any further damage by regulation of these neurotrophic factors.

### ANTICONVULSANT EFFECT

Sackeim et al<sup>5</sup> report that clinical response to ECT is related to an increase in seizure threshold throughout a course of treatments. Using data from animal models, at least 3 possible mechanisms have been suggested: 1) ECT enhances GABAergic transmission that contributes to the increase in seizure threshold; 2) antagonism of GABAergic transmission is a key factor in seizure induction with ECT; and 3) ECT produces endogenous anticonvulsant substances, at least one of which has opioid-like properties.<sup>6</sup> The magnitude of the change in seizure threshold varies directly with the degree of clinical improvement.

Positron emission tomography scanning studies in conjunction with ECT also demonstrate that the increase in seizure threshold throughout a course of ECT varies directly with decreases in regional cerebral blood flow