



ELECTROCONVULSIVE THERAPY

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ABSTRACT

Electroconvulsive therapy (ECT) was introduced in its current form by Cerletti and Bini in 1938. Since then it has been subjected to controversy and antipathy by the media and general public alike. Along with inadequate post-graduate training in psychiatry, in some developing countries like Pakistan, it is responsible for the widespread unacceptability of this treatment form among patients. Electroconvulsive therapy (ECT), as a non-invasive brain stimulation technique, is effective in treating severe psychiatric disorders.

Key Words: Controversy, therapy, stimulation, psychiatric.

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INTRODUCTION

Electroconvulsive therapy (ECT) is an effective therapy for severe major depressive disorder and other mood disorders. Especially among adults with treatment-resistant depression, generally defined as depression that does not remit or respond to one or more standard antidepressant pharmacotherapies, ECT stands out in its effectiveness. Whereas standard antidepressant therapies achieve a response of 16%–17% of patients with treatment-resistant depression, ECT achieves response rates of 50%–70% with such patients (**Weiner RD et al**). In 1947, one patient reported that they were given scientific papers to read on the various physical treatments for depression, including leucotomy, cardiazol and ECT. Their psychiatrist explained the process of ECT and answered questions to describe benefits and allay fears; therefore they were able to form their own opinion of treatment choice and knew what to expect during the treatment. The patient reported positive experiences of ECT in terms of the process and effectiveness. This sets a good standard for patient information and communication, but a recent review revealed that this is often not the standard that some patients experience [**Chakrabarti S et al 2010**].

However, there have been improvements since a 1976 review in which only 21% of patients reported that they were given adequate information prior to treatment [**Wheeldon TJ et al 1999**].

For example, in a 2004 study around 80% stated that the treatment had been fairly or very well explained [**Benbow SM et al 2004**], with 85% in a 2007 study stating that written information was helpful [**Rush G et al 2007**]. Fears about ECT treatment can be alleviated if a patient has the process and treatment fully explained to them by medical staff [**Kerr RA et al 1982**].

Despite considerable advances in the practice of electroconvulsive therapy (ECT), the benefits of this treatment remain a balance between effectiveness and the risk of cognitive side effects. It is therefore important for clinicians to understand the clinical indications and likely clinical benefits of ECT and have a clear understanding of the cognitive side effects.

REASONS OF ECT

Electroconvulsive therapy (ECT) can provide rapid, significant improvements in severe symptoms of several mental health conditions. (<https://www.mayoclinic.org/tests-procedures/electroconvulsive-therapy/about/pac-20393894>)

ECT is used to treat:

- **Severe depression**, particularly when accompanied by detachment from reality (psychosis), a desire to commit suicide or refusal to eat.
- **Treatment-resistant depression**, a severe depression that doesn't improve with medications or other treatments.
- **Severe mania**, a state of intense euphoria, agitation or hyperactivity that occurs as part of bipolar disorder. Other signs of mania include impaired decision-making, impulsive or risky behaviour, substance abuse, and psychosis.
- **Catatonia**, characterized by lack of movement, fast or strange movements, lack of speech, and other symptoms. It's associated with schizophrenia and certain other psychiatric disorders. In some cases, catatonia is caused by a medical illness.
- **Agitation and aggression in people with dementia**, which can be difficult to treat and negatively affect quality of life.

RISKS OF ELECTROCONVULSIVE THERAPY (ECT)

Modern electroconvulsive therapy (ECT) is generally considered a safe and effective treatment for severe, chronic depression and treatment-resistant depression, although it may occasionally be used to treat other conditions as well. Despite its general safety and efficacy, like psychiatric medications, it carries with it a number of side effects.

1. **Memory Loss:** Some people's memory loss is longer and greater with ECT. Some have trouble recalling events that occurred during the weeks leading up to treatment, or the weeks after treatment. (**John Hauser 2016**)
2. **Concentration and attention problems:** There was **no** evidence of long-term damage to concentration, attention, verbal and visual memory, or verbal fluency. There were no impairments of motor strength



and executive processing, even during the early (within 7-10 days) post-ECT period. (Neera Ghaziuddin, Donna Laughrin, Bruno Giordani, 2000)

3. **General Confusion:** Many people who undergo electroconvulsive therapy find that they experience a period of confusion after the procedure has been completed. You may forget why you're in the hospital, or even what hospital you're in

TREATMENT OR TECHNIQUE

Methohexital is considered the gold standard for induction of anaesthesia for ECT. It is a barbiturate that stimulates Gamma-aminobutyric acid (GABA) receptors which are the main inhibitory receptors in the brain. Methohexital produces anaesthesia for approximately 4 to 7 minutes, which is ideal for ECT. It does not affect seizure threshold and, compared to propofol, does not shorten seizure length. (Avramov MN et al 1995)

Methohexital causes moderate cardiac depression, which helps counteract the sympathetic discharge produced by a seizure. The initial dose is usually 1 to 1.5 mg/kg. (Martone CH et al 1991)

Etomidate is a unique drug that also works by activating GABA receptors in the brain. Studies have shown that it tends to produce a longer seizure compared to propofol. (Gazdag G et al 2004)

Etomidate does not cause cardiac depression, so the sympathetic discharge tends to be elevated when used as the sole anaesthetic for ECT. Supplementing with an intravenous opioid such as remifentanyl or a beta-blocker like esmolol to inhibit sympathetic discharge is common. Etomidate is known to cause adrenal suppression and should be avoided in patients with known adrenal insufficiency or critical illness. A dose of 0.3 mg/kg of etomidate is given to induce general anaesthesia, lasting around 5 to 10 minutes. (Upchurch CP et al 2017, Forman SA 2011)

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