

Electroconvulsive Therapy Resurrected: Its Successes and Promises After 75 Years

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Can J Psychiatry. 2011;56(1):3–4.

Clinical psychiatry lacks a coherent model of illness. The neuropathological models of the late 19th century failed to identify brain lesions that explained abnormal behaviours except for a few inherited genetic disorders such as Tay-Sachs disease. Understanding metabolic abnormalities, infectious diseases, and epilepsies demarcated illnesses for which specific treatments were soon found; these conditions quickly became wards of medical practitioners. Although psychodynamic theory next enthralled clinicians and dominated psychiatric thinking, biological roots encouraged experimentation and the 1917 report of the relief of neurosyphilis by malaria fever therapy earned the discoverer, Julius Wagner-Jauregg, the 1927 Nobel Prize in Medicine. Repeated comas and seizures with injections of insulin were serendipitously discovered to relieve symptoms of schizophrenia in 1933. A year later, chemically induced seizures effectively relieved catatonia, and, by 1938, the electrical induction of seizures replaced chemical inductions, was found to be safe and effective, and was universally adopted as the treatment standard. In 1935, frontal leucotomy was proposed to relieve obsessive thinking, earning the 1949 Nobel Prize for Medicine for Egas Moniz, its originator. Considering the prior paucity of effective treatments, these interventions took the profession and public by storm, dominating the psychiatric hospital scene in the 1940s and 1950s. The treatments were hailed as auspicious discoveries and applied widely throughout the world.¹

The introduction of psychoactive drugs quickly replaced these treatments and by the mid-1960s, fever therapy, insulin coma, leucotomy, and electroconvulsive therapy (ECT) disappeared, a fortunate change in the eyes of many critics who firmly believed that the patients had been abused by these interventions. By the late 1970s, however, as increasing numbers of patients with severe illness failed to improve, even with the cornucopia of new chemical elements and imaginative psychotherapies, experienced clinicians resurrected ECT and found it still effective.

We are 75 years into the ECT era and it is timely to assess our status. The treatment, once legislated against in many venues, is increasingly used. Recognition that the treatment requires special skills led to programs for certification of treatment centres

in the United Kingdom. (Elsewhere, the model of see one, treat one, teach one is commonplace.)

By the mid-1950s, muscle relaxation and sedation became features of the treatment. As anesthesiologists established their fiefdoms, ECT treatment teams of psychiatrist, anesthetist, and nurse became the standard of care. (In some countries where the cost of anaesthesia and the drugs is prohibitive, unmodified ECT is accepted.) Treatment devices modified and reshaped the available alternating electric currents and added instruments to monitor the electroencephalogram, cardiac, and motor aspects of the seizure. These modern methods made effective treatment possible in properly selected people with severe mental illness to remission rates greater than 85%.^{2–4} Well-designed studies of ECT and continuation treatments in the past 2 decades find the benefits to require continuation treatments after the immediate illness is resolved. ECT is unlike the relief afforded by surgery or specific antibiotics, an effective cure after treatment, but more akin to the endocrine replacements as in diabetes and hypothyroidism, requiring continuing treatments to sustain benefits.^{5,6} The changes in clinical practice are ably described in this issue by Dr Pascal Sienaert,⁷ who offers the reader a balanced image of what we know about ECT techniques. Present practices assure patients of effective and safe courses of treatment.

But the mechanism of action is puzzling. How does inducing grand mal seizures alter brain and body physiology and relieve the symptoms of the most severe psychiatric disorders? These are disorders of brain function but what is the pathophysiology? Where are we to look? As each new physiologic measure is devised, each is seen to change with seizures, and each leads to their consideration as the mechanism. Many explanations have been offered, but none has attracted continuing interest. Three models—the generalized seizure–electrophysiologic, the neuroendocrine–diencephalic, and the anatomic–ictal—are ably described by Dr Tom Bolwig.⁸

The first hypothesis recognizes the centrality of the seizure and its broad effects on brain function. The neuroendocrine hypothesis focuses on the consequences of the seizure in the release of neurohormones from centrencephalic nuclei and the recognition that the principal changes in responsive patients are

the vegetative and motor disturbances mediated by hormones. Another theory focuses on neurogenesis, the development of new functioning neurons and glia. Individual findings support each hypothesis but each awaits more concrete evidence of their relation to pathophysiology. Understanding the mechanism is the principal challenge in ECT research today.

The conflicted history of this treatment is puzzling. From its onset, it redeemed the mental normalcy of the most severely disturbed patients and despite risks, pain, and fright, the treatment was universally applied. With the introduction of sedation, muscle relaxation, and ventilation with oxygen, the actual procedure became benign. It is handicapped in that treatments must be repeated over weeks, and requires continuation treatments for the benefits to be sustained. That is the experience with many effective treatments—think, chemotherapy or radiation for cancer—treatments that have neither been maligned in the press and media nor shunned by the profession. The attacks on ECT have led to its broad stigmatization, marked by interdiction in some venues and rejection in many psychiatric treatment facilities—a practice that is unethical and unjust. Most surprisingly, its denigration is strongest within its own fraternity.^{9,10} ECT researchers in the United States and Europe are beating the drums to naysay the treatment, crying “memory loss, memory loss,” and disregarding the life-saving features. Their loathing of the treatment led to experiments meant to minimize its benefits, and lately to advance nonseizure brain stimulation as a replacement, with promises of less risk and equal efficacy. Transcranial magnetic stimulation (TMS) and vagus nerve stimulations (VNS) are lauded alternatives, the proponents forgetting that the essential element in convulsive therapy is the induction of seizures, not in the method of induction.¹¹ These wishes fail their promise. The latest reports find real TMS no more active in changing symptoms and illness behaviour than sham TMS.¹² A comparison of TMS and ECT finds no benefit for TMS and the customary benefit for ECT.¹³ The marketing of VNS far outran the evidence, and as practitioners applied the expensive treatment—surgical implantation of a stimulating device in the chest and snaking electrodes into the neck to reach the left vagus nerve—they found no antidepressant benefit and severe alteration of speech, quashing the initial enthusiasm. But the anti-ECT posture of the practitioners and blind enthusiasm for neurostimulation has led them to reprogram the focus and rename the national Association for Convulsive Therapy into the International Neurostimulation Society, divorcing themselves from the central features of the treatment’s success.¹⁴

The clinical experience with ECT has improved the delivery of care so that no absolute contraindication for the treatment is acknowledged and the efficacy for the principal psychopathologies—psychotic and melancholic depression, catatonia, malignant catatonia, and delirious mania—is well established with greater than 85% remission rates.²⁻⁴ Despite its

poor optics, despite its public and professional stigmatization, despite universal contempt, the treatment survives. It does so because it is effective and safe even in people who are the most severely and desperately ill and who have failed all other treatments. ECT deserves greater attention to its mechanism of action, offering the potential for greater understanding of the treatment and its underlying pathophysiology, and the best opportunities to understand the abnormal brain processes that underlie major psychiatric disorders and their remarkable resolution by inducing seizures.

References

- Shorter E. History of psychiatry. New York (NY): John Wiley & Sons, Inc; 1997.
- Abrams R. Electroconvulsive therapy. New York (NY): Oxford University Press; 2002.
- Fink M. Electroconvulsive therapy: a guide for professionals & their patients. New York (NY): Oxford University Press; 2009.
- American Psychiatric Association. Task Force on Electroconvulsive Therapy. The practice of electroconvulsive therapy: recommendations for treatment, training, and privileging. Washington (DC): APA; 2001.
- Kellner CH, Knapp RG, Petrides G, et al. Continuation ECT versus pharmacotherapy for relapse prevention in major depression: a multi-site study from CORE. *Arch Gen Psychiatry*. 2006;63:1337–1344.
- Kellner CH, Knapp R, Husain M, et al. Comparing bifrontal, bitemporal, and right unilateral electrode placement in ECT: a multisite study from CORE. *Br J Psychiatry*. 2010;196:226–234.
- Sienaert P. What we have learned about electroconvulsive therapy and its relevance for the practising psychiatrist. *Can J Psychiatry*. 2011;56(1):5–12.
- Bolwig T. How does electroconvulsive therapy work? Theories on its mechanism. *Can J Psychiatry*. 2011;56(1):13–18.
- Shorter E, Healy D. Shock therapy. A history of electroconvulsive treatment in mental illness. New Brunswick (NJ): Rutgers University Press; 2007.
- Ottosson J-O, Fink M. Ethics of electroconvulsive therapy. New York (NY): Brunner-Routledge; 2004.
- Fink M. Induced seizures as psychiatric therapy: Ladislav Meduna’s contributions in modern neuroscience. *J ECT*. 2004;20:133–136.
- George MS, Lisanby SH, Avery D, et al. Daily left prefrontal transcranial magnetic stimulation therapy for major depressive disorder. A sham-controlled randomized trial. *Arch Gen Psychiatry*. 2010;67:507–516.
- Eranti S, Mogg A, Pluck G, et al. A randomized controlled trial with 6-month follow-up of repetitive transcranial magnetic stimulation and electroconvulsive therapy for severe depression. *Am J Psychiatry*. 2007;164:73–81.
- Rasmussen KG, Kellner CH. Au revoir: Association for Convulsive Therapy. *J ECT*. 2009;25:231–232.

Manuscript received, revised, and accepted June 2010

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