CHAPTER 28

ELECTROCONVULSIVE THERAPY (ECT)

Introduction

ECT is a treatment of psychiatric disorders in which a brief electrical current is passed through the brain of the anaesthetised patient using specialized apparatus. There is a convulsion which is modified by muscle relaxants.

Illustration. A recently superseded ECT machine.

ECT is a safe and most effective treatment of major depression and catatonia, among other disorders (Abrams 1997). It is also a contentious treatment. Negative attitudes and misconceptions abound among the general public (Dowman et al, 2005) medical students (Papakosta et al, 2005), and even psychiatrists (Gazdag et al, 2005). This mainly arises out of ignorance and attitudes change with experience or education/information.

It is unclear why ECT generates such negative attitudes. One factor may be our hard-wiring. There is an innate repugnance for certain biological actions. Convulsing, like vomiting, is not something we like to watch. There may be evolutionary factors. Convulsing, like vomiting, could indicate sickness and as sickness may be contagious, we may be genetically programmed to fear and avoid such situations. We avoid discussing the topic of convulsions, as people with epilepsy will testify. Some people with epilepsy contend the rest of us also avoid people with epilepsy.

History of ECT

ECT was first performed in Rome in 1938 (and has been in continuous use ever since). As with other events in science, it is always possible to find accounts of similar events in previous centuries.

In AD 46, Scribonius Largus described the application of electric torpedo fish to the head as a treatment for headache. In 1470, a Jesuit missionary in Ethiopia applied electric catfish to people (anatomical site unknown) as a means of expelling devils. In the 18th century electric eels were applied to the head (condition treated unknown). However, there is no clear history of the application of electricity to the head for the treatment of mental disorders before 1938.

Convulsions had been induced by other means for medical purposes at different times over the centuries. Paracelsus (1490-1541) administered camphor by mouth to induce convulsions in the treatment of mental disorders. In 1785 an account appeared in the London Medical Journal of camphor induced convulsions for the treatment of psychosis.

ECT emerged at an interesting time. Until the early 1920's little could be offered to people with serious mental disorders other than humane care. Then came a series of active treatments which encouraged optimism and set the scene for the development of ECT.

From around 1917 Julius Wagner-Jauregg (Professor of Psychiatry, Vienna) began treating the otherwise progressive and fatal general paresis of the insane (terminal syphilis) by infecting sufferers with malaria. (The malaria fever killed the spirochaetes which caused the syphilis.)

In 1933, Manfred Sakel (Vienna) announced the successful treatment of schizophrenia with insulin. Other psychiatrist had used insulin to stimulate appetite, however, Sakel sought to induce coma. In the process, some patients experienced seizures, and this may have been responsible for observed improvement.

In1934, Ladislaus von Meduna (1896-1964; Budapest) injected camphor into a person with schizophrenia with the intention of inducing convulsion; this was the first modern convulsive therapy.

Von Meduna had developed the theory of "biological antagonism", between epilepsy and schizophrenia. He believed the two conditions could not co-exist. This arose out of two observations. First, when a person with severe mental disorder had a seizure (for whatever reason) their mental state improved. Second, was an epidemiological mistake, the "observation" that people with schizophrenia did not suffer epilepsy.

Von Meduna published his results. But the induction of convulsions with camphor, and subsequent commercial agents was unpredictable and unsatisfactory. ECT has the advantages of immediacy and predictability.

In Rome, in 1938, stimulated by the success of von Meduna, **Ugo Cerletti** (1877-1963) assisted by Lucio Bini (1908-1964), supervised the first ECT treatment. The first patient, SE, was a 39 year old engineer from Milan who was found wandering the streets of Rome in a psychotic state. He received 11 treatments, obtained a good response and wrote to the doctors the following year thanking them for their treatment.

Illustration. Ugo Cerletti (1877-1963), supervised the first ECT treatment (1938).

The use of ECT spread rapidly around the world. It is now used more widely in major depression than in schizophrenia.

Improvements in technique

ECT has been in continuous use over the last 80 years. However, there have been technical improvements:

- The introduction of anaesthesia to ECT practice made the process less distressing for patients.
- Anaesthesia also allowed the application of muscle relaxants which reduced the strain on the musculoskeletal system, reducing injuries.
- Pre-oxygenation and assisted ventilation during recovery reduced side-effects.
- Electrical stimuli have been designed to produce therapeutic convulsions without the delivery of unnecessary electrical energy to the brain.
- A range of electrode placements from which to choose, depending on the clinical details of the particular case.
- Methods for monitoring brain and body activity before, during and after convulsions.
- Stimulus modifications – originally sinusoidal – now brief square waves, generally 1ms [some have used 0.3ms; Galletly et al, 2013]

Conditions treated

Major depressive episode

Major depression is the condition most commonly treated with ECT. It is especially indicated where drugs have failed or there is risk of suicide.

Active ECT has been shown superior to placebo ECT in many trials (e.g., Gregory et al, 1985). Further trials of this comparison are unnecessary.

ECT has also been found to be superior to the available antidepressant drugs in more than a dozen trials. A typical design is for patients were divided into two groups: one receiving active ECT and placebo medication, and the other receiving placebo ECT and active medication (Gangadhar et al, 1982). In this way ECT can be compared with and antidepressant medication, and both groups of patients received an active form of treatment.

Mania

Mania is a state of mood elevation or irritability and physical over-activity. Treatment may be a necessary to ensure food and fluid intake and prevent exhaustion and physical injury.

This is a difficult population to study for various reasons. Universal clinical experience is that ECT is an effective treatment and can be lifesaving.

ECT has been shown superior to lithium carbonate in acute mania (Small et al, 1988). (However, lithium carbonate alone is not a standard pharmacological treatment.)

Schizophrenia

As mentioned earlier, Meduna used camphor to induce convulsion in schizophrenia.

ECT is currently used in schizophrenia when there are marked catatonic features (Raveendranathan et al, 2012; Pompili et al, 2013) with limited food and fluid intake and when other psychotic symptoms are unresponsive to medication.

Postpartum disorders

A range of psychiatric disorders may develop following childbirth. The majority can be managed with support and the judicious use of medication.

Acute, severe disorders may develop, however, and mother may represent a danger to herself and/or the baby. As a generalization, the majority of the severe postpartum conditions are similar to an episode of major depression, and the remainder are psychotic episodes, with delusions and hallucinations.

ECT is useful in these severe conditions (Reed et al, 1999). ECT induces remission rapidly, thus, the risk to mother and baby rapidly passes, and breast-feeding and mother-baby bonding can be commenced without delay. ECT obviates high doses of various medications, thus minimizing the medication reaching the breast-fed baby.

Maintenance ECT

When medication has failed and ECT is necessary to induce remission in major depression, and medication fails to prevent relapse, maintenance ECT is considered (Frederiske et al, 2006). This is conducted on an outpatient basis. The frequency of ECT is determined by clinical response. Often, on completion of a course of ECT, when remission has been achieved, one ECT continues to be given at weekly intervals. This is usually gradually extended out to one treatment each 4 or 6 weeks (Gagne et al, 2000).

The National Institute for Clinical Evidence (2003) in the UK, does not recommend maintenance ECT. The American Psychiatric Association does, and there is a continuous, but modest, stream of publications (Nordenskjold et al, 2013).

The procedure

Preparatory work includes making an accurate diagnosis (disappointment and personality disorder, for example, do not respond to ECT), communication with the patient and family, anaesthetic assessment, and deciding on the most appropriate electrode placements.

Generally, the stimulus is applied using one of two electrode arrangements. In bilateral stimulation, one electrode is placed on either side of the forehead and the electricity passes through both sides of the brain. In unilateral stimulation, one electrode is attached to one side of the forehead and the second is placed further back on the scalp on the same side of the head. With unilateral stimulation the electricity remains predominantly on one side of the head. (However, when the convulsion commences, it extends to the other side of the brain.)

A third electrode placement has recently been described: ‘bifrontal’. Here, electrodes are placed on the forehead, above the eyes. Results have been very encouraging (Phutane et al, 2013). Theoretically, this could give the greater efficacy of bilateral ECT, and with a smaller region of the brain exposed to electricity, minimal cognitive side-effects.

Two sets of electrodes are attached to the patient to monitor the activity of the brain before, during and after ECT administration. One set is placed on the scalp (EEG) and the other on a limb. The observations assist in decision making regarding the adequacy of the physiological response.

The patient is lying on a trolley. An anaesthetist, psychiatrist and at least two nurses are present. The anaesthetist inserts a cannula, an anaesthetic nurse attaches ECG electrodes, and the psychiatrist and psychiatric nurse attach ECT, EEG and peripheral muscle electrodes.

The anaesthetic is administered. When muscle relaxation has occurred, the ECT stimulus is applied. This is a square wave with a pulse width of 1.0 millisecond. Using one popular device (Thymatron), the stimulus is delivered at a maximum frequency of 70 pulses per second. Therefore, in one second the stimulus runs for 0.14 second. The longest the stimulus can continue, using this device, is 8 seconds. Thus, with

maximum setting, the stimulus runs for a total time of a little over one second (1.12 seconds).

As mentioned, recently a pulse width of 0.3 millisecond (ultrabrief) has been reported [– while there are positive reports, one review (Galletly et al, 2013) expresses reservations].

The convulsion is much modified. Usually there is bending of the elbows and pointing of the toes. When the convulsion has stopped (generally less than 30 seconds) the patient is rolled onto the side and transported to the recovery room. The whole procedure from arrival to departure from the procedure room takes in the order of 10 minutes.

Electrode placement

As mentioned, there are two main electrode placements, bilateral and unilateral. [At the moment, bifrontal is mainly being used in specialized units, and will not be covered in this discussion.]

The most troublesome side effect of ECT is memory problems. Memory is not located in any one particular region of the brain - current wisdom is that memory depends on many regions of the brain being anatomically and functionally linked together. It is known that severe memory problems occur when structures on both sides of the brain are damaged, for example, when both left and right temporal lobes are destroyed.

There is evidence to indicate that bilateral ECT has a stronger antidepressant effect than unilateral ECT (UK ECT Review Group, 2003). However, bilateral ECT is also believed to be associated with greater temporary memory disturbance than unilateral ECT.

Evidence shows that delivering a substantially larger amount of electrical energy unilaterally than is required to simply trigger a convulsion (“seizure threshold”) can produce similar antidepressant effects as bilateral ECT, but with less memory disturbance (Sackheim et al, 1993). This “high dose unilateral ECT” is now the most often chosen form. However, when a maximum antidepressant effect is required, bilateral ECT may be necessary.

Dose determination

Current thinking is that optimum antidepressant effect is achieved with electrical doses well above the seizure threshold (Sackheim et al, 1993). [This thinking has recently been challenged (Lapidus, et al, 2013), but not yet disproved.]

There are two methods of determining a suitably high dose of charge. One is by first determining the “seizure threshold”. In this method a number of stimuli are applied, starting at a low level, and increasing the electrical energy of subsequent stimuli until a seizure is triggered. Treatment is then provided with a stimulus 2-3 times higher

than the seizure threshold. This is called the "stimulus titration method", and is favoured by many experts (Tiller and Ingram, 2006).

Alternatives include delivering a dose determined by age ("age-based dosing algorithm"; Abrams 2002a), or a fixed high dose (Abrams 2002b).

The jury is still out on whether the "stimulus titration method" or the "age-based algorithm" is the better method of dose determination. The APA Taskforce on the Practice of ECT (2001) approves both.

Peterchev et al (2010) have recently have criticized the use of a "summary metric" (charge) to describe the dose of ECT. They provide theoretical and empirical evidence that stimulus parameters (pulse amplitude, shape, and width and time frequency, directionality, polarity, and duration) exert unique neurophysiological effects. Recently electrode size has been shown to influence the physiological response (Deng et al, 2013). Thus, the optimal dosing paradigms remain to be determined, and will depend on more than the oversimplified "summary metric" of charge.

Death and ECT

Death during ECT is extremely rare. ECT is safer than dental extraction under anaesthesia. The few deaths which have occurred, have been a result of anaesthetic rather than the ECT complications. Searching 50 years of records, one death was found in 46,770 treatments (Kendall, 1977). There are less deaths among people with depression who are treated with ECT than among people with depression who are treated by other means (Avery & Winokur, 1978)

Permanent brain damage and ECT

ECT does not cause brain damage. Every possible investigation has been conducted including blood enzyme studies, imaging of the structure and chemical composition of the brain, and post mortem histological studies. No abnormalities have been detected which can be attributed to ECT.

Memory and ECT

Loss of memory strikes at the sense of autonomy and is fundamentally threatening to the individual.

Two recent developments have reduced the memory disturbance associated with ECT. First, the introduction of stimulation by brief (1 ms) square waves. Early ECT devices delivered sine waves, which have limited stimulation potential relative to the amount of energy they deliver, and the unnecessary energy greatly disturbed memory. Very recently, the use of ultrabrief pulses (0.3 ms) have been reported to further reduce memory problems (Rosa et al, 2013). Second, was the introduction of unilateral ECT, which is not usually associated with the subjective experience of memory difficulties (Squire and Slater, 1983).

In disentangling the effect of ECT on memory, other factors must be taken into account. Major depression per se, perhaps through distractibility and perhaps through the slowing of thought processes, has a detrimental effect on memory. Also, many antidepressants (the alternative treatment) may also have a mild, temporary, detrimental effect on memory. Thus people who suffer an episode of major depression may have a poor memory for this period of their lives whether they had ECT or not.

Using sophisticated neuropsychological testing methods, disturbance of memory can sometimes be demonstrated following ECT (Schulze-Rauchenbach et al, 2005).

Memory difficulty is the most commonly claimed side effect of ECT. Frequently, no objective evidence can be demonstrated. However, as Vamos (2008) points out, despite the low correlation, both perspectives must be taken into consideration.

The following summarize our present knowledge regarding ECT and memory:

- Memory difficulties may follow ECT, and while these usually subside within a few weeks, evidence indicates that some individual have long term difficulties.
- The modern brief square wave stimulus is less likely to produce memory difficulties than the now abandoned sine wave. This may be extended by the introduction of ultra-brief pulses.
- Unilateral ECT is associated with less memory difficulties than bilateral ECT
- The majority of patients who have unilateral ECT make no claim of memory difficulties.
- Most people who claim subjective memory difficulties post ECT have no objective difficulties on testing.
- When memory disturbance does occur, it is more for impersonal than important personal events.
- Depression per se and antidepressant medication are also associated with memory difficulties.

Case history, 1

Harold Watts was an accountant of 44 years of age, he was married to Ellen and the father of Josephine aged 21, who had recently married, and Paula aged 19, who had recently left home to live in a de facto relationship. Harold was brought to hospital by ambulance, accompanied by police, Ellen and a next-door neighbour.

Ellen had gone to investigate two loud noises in the garage. She had found Harold on the floor next to an overturned chair, apparently dead. She rushed to her friends next door and they ran back with her. By this time Harold was beginning to move and groan on the floor. They rang the ambulance. There was a belt tied to a rafter with the buckle end hanging down. The buckle was broken. It appeared Harold had tried to hang himself. The first noise Ellen heard may have been the jerking of the rafter or the chair falling over, and the second, some moments later, may have been when the buckle broke and Harold landed on the floor. It was unclear who called the police.

When the police arrived Harold was sitting in the living room saying that it was all a misunderstanding and that he did not need attention. The ambulance officers noted thick purple marks around his neck and that the whites of his eyes were pinkish. The police were shown the hanging belt and Harold was taken to hospital.

Harold was orientated in time, place and person and an X-ray of his neck revealed no bony abnormality. He could move all limbs and did not appear to have sustained any permanent physical damage. He cried and said he was just missing “the girls” since they both left home about the same time. Ellen, a neighbour, a hospital doctor and an ambulance officer were discussing the situation in the corridor. Ellen was saying she would take Harold home and perhaps they should take a holiday together, when a nurse passing his cubicle noticed Harold was attempting to strangle himself with the leads of a cardiac monitor. They rushed back, removed the leads and called a psychiatrist.

Harold had been drinking excessively over the last month. His appetite for food had decreased. He denied feeling depressed, but had been moved to tears when watching sentimental television programs. He had been preoccupied with thoughts of his dead parents and dead brother. He had found himself thinking about cemeteries and his own funeral. He then started to experience strong urges to kill himself. He could not explain these urges, nor could he guarantee he would not act on them.

Harold’s business affairs appeared to be with out a blemish and he denied any professional indiscretions or worries. “The girls” had left the home four months previously, but there had been no acrimony and they visited.

Harold was transferred to a psychiatric ward for observation, with a probable diagnosis of major depressive disorder. There was some uncertainty as he denied feeling depressed. However, depressed mood is not always a prominent complaint in major depression, in which case the term “masked depression” may be applied. Supporting the diagnosis of depression was the history of preoccupation with death and sad events, and self-destructive urges.

Within and hour of admission to the psychiatric ward Harold again performed self-destructive behaviour. He was being watched closely. He asked to go to the toilet and was allowed access to a specially designed facility which contained no cloth towels and no suspension points from which one could hang, and no sharp edges with which cutting could be performed. Soon after he had been left alone a heavy thud was heard. Harold was found on the floor outside the toilet cubicle in a pool of blood and with a large laceration on the top of his head. He had climbed up and stood on the wall of the toilet cubicle and divided down head first onto the floor. This was a resourceful and determined attempt and left no doubt that Harold was a danger to himself.

Harold’s head wound was sutured, his skull was X-rayed. There was no fracture. ECT was commenced next day. He immediately lost his suicidal urges. He revealed that he had been feeling guilt as if he was responsible for events which he heard about on the news, even events on the other side of the world. He had not admitted this when brought into hospital because he felt ashamed. He left hospital two weeks later, in remission, and returned to work.

Case history, 2

Hilda Durant was a 54 year old podiatrist who was married to Colin, an earth moving contractor. Colin took Hilda to their general practitioner who referred her to hospital for admission. She displayed psychomotor retardation (she moved very slowly, sat slumped in her chair, did not move her hands when talking, she was slow to answer questions, and her answers were slow and brief). He body and mind were working at her normal rate.

She admitted to depressed mood and some suicidal thoughts for at least two months. She had difficulty staying asleep, could not concentrate and lacked energy. She had a history of a similar episode five years previously which had responded to ECT and she and Colin had no hesitation in agreeing to another course.

Hilda responded well to the first and second treatments. Her sleep improved and she became more energetic and active. One the morning before the third she left the hospital and drowned herself in a nearby river.

With the benefit of hindsight, the ECT had helped the psychomotor retardation (slow movement and thinking) but had not yet eradicated the depressed mood and suicidal thoughts. This is not unique to ECT, and can occur with antidepressant medication. The remission of depression can be uneven; the last thing to improve is usually the low mood. There may be frustration on both sides when a patient begins to recover, with the hospital staff telling the patient they look better, and the patient protesting that he/she doesn't feel any better.

The case of Hilda Durant proves the old clinical adage that depressed patients with psychomotor retardation are at greatest risk when they are getting better.

Case history, 3

Betty Day was 35 years of age, twice divorced and living with an unemployed alcoholic man in rented accommodation. She was brought in to the Department of Emergency Medicine of a large hospital because of unruly behaviour in public.

She had given birth to two children, to different fathers, both children had been taken into care.

Betty had been to university, she had dropped out of second year Arts. He parents lived in a comfortable middle class suburb. There was no one else in Betty's current social circle who had attended university and she had no contact with her parents.

Her early life had been unremarkable, she was raised with a younger brother who was now living in another state. She had not been outgoing, but had been successful at a church girl's school. At university she started taking drugs and behaving in an aggressive, disinhibited and promiscuous manner. At first her parents thought this was because she was not ready for the greater freedom of university life and tried to

regulate her behaviour by increasing their supervision. She had been living in a flat, they insisted she move back home. She stayed up all night playing loud music and walked around the house naked. She did not study and within a fortnight she left home and slept on the floor of other student's rooms. Gradually, she became unpopular and unwelcome among the other students and she began frequenting working class pubs. She talked loud and continuously, she was often hoarse from talking and sometimes she could only keep quiet when she was drunk to the point of unconsciousness.

Betty was admitted to a psychiatric ward at 24 years of age when she suffered a brief episode of depression and scratched her wrists. She was thought to have a psychopathic personality disorder. She was given a small dose of an antidepressant medication and swung out of depression into a floridly manic state with overtalkativeness, loud disinhibited behaviour and racing thoughts. She was not euphoric, but irritable. In spite of her irritability she could agree that she was not her "normal self" and that she needed help to "slow down".

Various medications were tried. She developed a shin rash to the mood stabilizer carbamazepine. A combination of two others (lithium and sodium valproate) gave her only slight relief. She needed large doses of antipsychotic medication to control her mood elevation, and this caused large weight gain. She became a pathetic creature. From a successful church school girl she became an obese, frequently drunk, ostracised woman who could not stop talking and would sleep with any man who offered her affection. It seemed those who could tolerate her behaviour were those who were themselves drunk most of the time.

Betty became known to the police as a psychiatric patient and they began to bring her to hospital rather than charge her when they were called to control her unruly behaviour.

On this admission, because her chronic mania was unresponsive to all other treatments, she was offered a course of ECT. This had a good effect and she was discharged as a composed and cooperative person. Unfortunately, she soon relapsed, as medication alone could not maintain remission.

Betty readily agreed to a trial of maintenance ECT. After a course of 6 treatments as an inpatient she was discharged and had one treatment weekly for a month. The time between treatments was extended and finally she was managed on one treatment every 5 weeks. She remained well on this regime for years. At times she would need to have ECT more frequently, but then the time between treatments would again be extended.

She did not re-establish close contact with her parents. She remained overweight and talkative but she was able to largely abstain from alcohol. She entered a new stable de facto relationship, found work as a teacher's aid and was able to have one of her children returned to her care.

References

Abrams R. Electroconvulsive Therapy. 3rd Edition. Oxford University Press: New York. 1997.

Abrams R. Stimulus titration and ECT dosing. Journal of ECT 2002a; 18:3-9.

Abrams R. Electroconvulsive Therapy 4th Edn. Oxford University Press: New York, NY. 2002b.

APA. The Practice of Electroconvulsive Therapy: Recommendations for Treatment, Training and Privileging: A Task Force Report of the American Psychiatric Association. Washington, DC: American Psychiatric Association; 2001Treatment procedures; 125-196

Avery D, Winokur G. Suicide, attempted suicide, and relapse rates in depression. Archives of General Psychiatry 1978; 35:749-753.

Deng Z, Lisanby S, Peterchev A. Controlling stimulation strength and focality in electroconvulsive therapy via current amplitude and electrode size and spacing. U ECT 2013; 29: 321-331.

Dowman J, Patel A, Rajput K. Electroconvulsive therapy: attitudes and misconceptions. Journal of ECT 2005; 21:84-87.

Frederiske M, Petrides G, Kellner C. Continuation and maintenance electroconvulsive therapy for the treatment of depressive illness: a response to the national institute for clinical excellence report. Journal of ECT 2006; 22:13-17.

Gagne G, Furman M, Carpenter L, Price L, Efficacy of continuation ECT and antidepressant drugs compared to long-term antidepressants alone in depressed patients. American Journal of Psychiatry 2000; 157:1960-1965.

Galletly C, Clarke P, Paterson T, Rigby A, Gill S. Practical considerations in the use of ultrabrief ECT in clinical practice J ECT Sep 30 [Epub ahead of print]

Gangadhar B, Kapur R, Kalyanasundaram S. Comparison of electroconvulsive therapy with imipramine in endogenous depression: a double blind trial. British Journal of Psychiatry 1982; 141:367-371.

Gazdag G, Kocsis N, Tolna J, Lipcsey A. Attitudes toward electroconvulsive therapy among Hungarian psychiatrists. Journal of ECT 200521:204-207.

Gregory S, Shawcross C, Gill D. The Nottingham study. A double-blind comparison of bilateral, unilateral and simulated ECT in depressive illness. British Journal of Psychiatry 1985; 146:520-524.

Kendall R. The present status of electroconvulsive therapy. British Journal of Psychiatry 1981; 139:265-83.

Lapidus K, Shin J, Pasculli R, et al. Low-dose right unilateral ECT: effectiveness of the first treatment. J ECT 2013, in press

Lisanby S, Electroconvulsive Therapy for Depression. New England Journal of Medicine 2007; 357:1939-1945.

Loo C, Sainsbury K, Sheehan P, Lyndon B. A comparison or RUL ultrabrief pulse (0.3 ms) ECT and RUL ECT. International Journal of Neuropsychopharmalcology 2008; 11:883-890.

National Institute for Clinical Evidence. Guidance on the use of electroconvulsive therapy. Technological appraisal 59. London. 2003

Nordenskjold A, von Knorrint L, Ljung T et al, Continuation electroconvulsive therapy with pharmacotheherapy versus pharmacotherapy alone for prevention of relapse of depression: a randomized controlled trial. J ECT 2013, in press.

Papakosta V, Zervas I, Pehlivanidis A, Papadimitriou G, Papakostas Y. A survey of attitudes of Greek medical student toward electroconvulsive therapy. Journal of ECT 2005; 21:162-164.

Peterchev A, Rosa M, Deng Z-D, Prudic J, Lisanby S. ECT stimulus parameters: rethinking dosage. Journal ECT 2010; 26:159-174.
Phutane V, thirthalli J, Muralidharan K, et al. double-blind randomized controlled study showing symptomatic and cognitive superiority of bifrontal over bitemporal electrode placement during electroconvulsive therapy for schizophrenia. Brain Stimulation 2013; 6:210-217.
Pompili M, Lester D, Dominici G, et al. Indications for electroconvulsive treatment in schizophrenia: a systematic review. Schizophrenia Research 2013, in press.
Raveendranathan D, Narayanaswamy J, Reddi S. Response rate to catatonia to electroconvulsive therapy and its clinical correlates. European Archives of Psychiatry and Clinical Neurosciences 2012. [Epub ahead of print]
Schulze-Rauchenbach S, Harms U, Schlaepfer T, Maier W, Falkai P, Wagner M. Distinctive neurocognitive effects of repetitive transcranial magnetic stimulation and electroconvulsive therapy in major depression. British Journal of Psychiatry 2005; 186:410-416.
Sackheim H, Prudic J, Devanand D, Kiersky J, Fitzimons L, Moody B, McElhiney M, Coleman E, Settembrino J. Effects of stimulus intensity and electrode placement on the efficacy and cognitive effects of electroconvulsive therapy. New England Journal of Medicine 1993; 328:839-846.
Small J, Klapper M, Kellams J, Miller M, Milstein V, Sharpley P, Small I. ECT compared with lithium in the management of manic states. Archives of General Psychiatry 1988; 45:727-732.
Squire L, Slater P. Electroconvulsive therapy and complaints of memory dysfunction: a prospective three year follow up study. British Journal of Psychiatry 1983; 142:1-8.
Tiller J, Ingram N. Seizure threshold determination for electroconvulsive therapy: stimulus dose titration versus age-based estimations. Australian and New Zealand Journal of Psychiatry 2006; 40:188-192.
Reed P, Sermin N, Appleby L, Faragher B. A comparison of clinical response to electroconvulsive therapy in puerperal and non-puerperal psychoses. Journal of Affective Disorders 1999; 54:225-260.
Rosa M, Bueno C, Andrade M, et al. Ultrabrief (0.3ms) or brief (0.5 ms) pulses for right unilateral electroconvulsive therapy: is there a difference in seizure thresholds? J ECT 2013; 29:15-17.
UK ECT Review Group. Efficacy and safety of electroconvulsive therapy in depressive disorders: a systematic review and meta-analysis. Lancet 2003; 361:799-808.
Vamos M. The cognitive side effects of modern ECT: patient experience or objective measurement. J ECT 2008; 24:18-24.