ECT in the elderly

Electroconvulsive therapy (ECT) remains one of the most controversial treatments currently used in psychiatric practice, despite its long-standing record of safety and efficacy. **Drs Sreedharan Amarjothi, Murali Krishna** and **Richard Barnes** discuss of the origins of ECT, how this form of therapy came to be incorporated in psychiatric practice and why it continues to be enigmatic to both medical practitioners and the general public.

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Historically, convulsive treatment developed from a believed antagonism between schizophrenia and epilepsy, and the observation that agitated patients with epilepsy seemed calmer after a fit. Initially 'therapeutic' convulsions were not induced by electricity. Dr William Oliver first reported treatment of melancholia with camphor-induced convulsions in the *London Medical Journal* in 1785. In 1934, Dr Ladislas von Meduna, superintendent of the Budapest Royal State Mental Hospital, successfully induced convulsions using intravenous metrazol. In Europe other researchers induced them by injections of insulin. However, these methods were slow, inconsistent and associated with serious adverse events. The first successful electroconvulsive treatment was described to the medical world in 1938 when two Italian psychiatrists, Ugo Cerletti and Lucio Bini, administered an electrical stimulus to a catatonic vagrant in Rome. With the introduction of anaesthetic induction agents, muscle relaxants and effective monitoring, the adverse physical effects of electroconvulsive therapy (ECT) have been significantly attenuated. The term 'electroconvulsive therapy' replaced Cerletti's more disturbing term, 'electroshock treatment'.

Mechanism of action

There is uncertainty about the precise mechanisms by which ECT works. Serotonin (5-hydroxytryptamine, 5-HT) is one of the most important neurotransmitters involved in depressive illness, and ECT alters several 5-HT-receptor subtypes in the central nervous system. These receptors in post-synaptic neurons are sensitised by repeated ECT, but those in pre-synaptic neurons (autoreceptors) are not changed. An autoreceptor is a receptor located on presynaptic nerve cell terminals and serves as a part of a feedback loop in signal transduction. It is sensitive only to those neurotransmitters and hormones that are released by the neuron in whose membrane the autoreceptor sits. In addition, ECT is known to increase the sensitivity of 5-HT3 receptors to serotonin in the hippocampus, resulting in an increase in release of neurotransmitters, such as glutamate and gamma-amino butyric acid. In contrast, ECT decreases the auto-receptor functions in noradrenergic neurons in locus coeruleus and dopaminergic neurons in substania nigra, resulting in an increase in release of noradrenaline and dopamine respectively[1].

Using positron emission tomography, Nobler *et al* (2001) demonstrated decreased glucose metabolism in patients after receiving ECT. This was particularly marked in frontal and parietal cortex[2]. Similar findings were observed in other studies and it was concluded that ECT reduces neuronal activity in some cortical regions and proposed this may have an antidepressant and (surprisingly) anticonvulsant effect. However, the finding that benzodiazepines and other anticonvulsant drugs, which bring about similar

Table 1. Clinical conditions known to response to ECT

Major depression associated with:
- strong suicidality
- marked psychomotor retardation
- depressive delusions
- life-threatening refusal of food or fluids

Severe mania resistant to pharmacological treatment

Affective and motor symptoms in people suffering from Parkinson's disease with severe disability despite medical treatment

Possible role in disorders such as:
- neuroleptic malignant syndrome
- Huntington's disease
- treatment-resistant epilepsy[8]

changes, fail to ameliorate depressive symptoms weakens this theory.

Indications

In the geriatric population, ECT is most commonly used for treating major depression[3]. It remains the most effective treatment available for severe depression, with a recovery rate in the region of 80 per cent[4]. It is well tolerated, even by very elderly patients[5]. ECT is particularly effective when depression is severe and the illness associated with strong suicidality, marked psychomotor retardation and depressive delusions, or is life-threatening because of refusal of food or fluids[6]. Several controlled trials have showed ECT superior to placebo, especially in depression with psychotic symptoms. Patients with severe depression respond faster to ECT as opposed to antidepressants. It is more effective than psychotropics in management of treatment resistant depression and in depression associated with psychotic symptoms[7].

ECT may be considered for severe mania associated with life-threatening physical exhaustion and when it has not responded well to pharmacological treatment. With the advent of effective psychotropics, this is a rare indication in clinical practice[8]. There is extremely limited evidence to suggest efficacy of ECT in schizophrenic illness in the absence of clear affective symptoms[9]. It is a safe adjunctive treatment for both affective and motor symptoms in people suffering from Parkinson's disease with severe disability despite medical treatment[10]. In catatonic states, ECT is indicated when treatment with short-acting benzodiazepine, such as lorazepam, has been ineffective. Recent research suggests a possible role in disorders such as neuroleptic malignant syndrome, Huntington's disease and treatment-resistant epilepsy[8] (*Table 1*).

Contraindications

There are few absolute contraindications but not many clinicians would use it in medical conditions with raised intracranial pressure. ECT should be avoided in the first three months following myocardial infarction or stroke. Uncontrolled hypertension, severe heart disease and intracerebral masses raise risk, and hypertension and cardiac failure should be treated optimally before ECT[11]. Dementia per se is not a contraindication for ECT. Other contraindications are on anaesthetic grounds.

Administration of ECT

All patients receiving ECT should have a detailed assessment of their physical health. A review of medical risk by a senior anaesthetist is mandatory before ECT. All patients should have their cognitive functions (particularly memory) and mental state assessed after each session, as well as checks for other side effects. Occasionally, in patients suffering from pre-existing cognitive impairment, detailed neuropsychological testing may be necessary[12].

ECT should be administered in a purpose-built suite. The patient receives an intravenous injection of anaesthetic agent and, once unconscious, given a muscle relaxant to modify the fit and minimise musculoskeletal injuries. The electroencephalogram (EEG), heart rate and other vital signs are monitored throughout the procedure[12]. A typical course of ECT consists of six to 12 treatments administered twice a week until improvement in target clinical symptoms is achieved. There is no advantage in prolonging a course beyond recovery[13]. The treatment may be discontinued if patients fail to show signs of improvement by six or eight treatments[14]. Older adults may respond slower, but older age is generally considered a positive predictive factor in response to ECT[15]. Symptom improvement during initial treatments, albeit transient, is a good prognostic factor.

Electrodes may be placed bilaterally (one above each temple) or unilaterally (one above the temple and one just to the same side of the occiput of the non-dominant hemisphere). The relative benefits of each are a matter of debate, though it is suggested that bilateral placement is more effective but with a greater risk of side effects. Unilateral ECT is indicated in individuals who experience unacceptable level of cognitive impairment or confusion following ECT.

A modified electrical current (high voltage, constant current and brief pulse) is then passed through the brain, inducing a grand mal seizure. The strength of the current applied depends on seizure threshold, which varies from patient to patient, so a technique known as stimulus dosing is used to determine the minimum necessary dose. Typically a current around 50 per cent greater than threshold is used as a treatment dose. When available, the most reliable method for documenting a seizure is by EEG. An EEG seizure of between 20–50 seconds is generally considered adequate, though this figure is essentially arbitrary[16] and there is no strict correlation between seizure length and outcome. Certainly longer seizures are not good predictors of efficacy.

Side effects

Although adverse cognitive effects of ECT have been reduced by the use of standardised ECT equipment delivering brief pulse stimulation (as opposed to sine wave stimulation) and convulsive seizures are modified using a muscle relaxant and anaesthetist, memory impairment is by far the most important and undesirable effect of ECT. Unilateral electrode placement over the non-dominant hemisphere also significantly reduces post-ECT memory loss.

Freeman describes four distinct types of memory impairment following ECT[14]:

> short-term retrograde amnesia — some patients experience a retrograde amnesia for events leading up to and during a course of ECT;

> retrograde amnesia for remote events — some patients certainly do complain of amnesia for events that are remote or stretching back several years. It has been shown that ECT can produce long-term impairment of personal memories;

> short-term anterograde amnesia — there is usually some degree of anterograde amnesia following ECT but it is short lived. This often presents with patients showing difficulty in retaining and new learning for few days or even a few weeks after the ECT;

> long-term permanent anterograde amnesia — though subjectively reported by some patients, it is difficult to ascertain how much ECT contributes to any permanent anterograde memory impairment. While ECT is clearly a component, the aetiology is likely to be multifactorial.

Other common side effects of ECT are headache, post-ECT confusion, clumsiness, nausea/vomiting, muscle aches and pains. Presently no convincing evidence indicates that either ECT or intermittent brief seizures, such as those induced during ECT, produce structural damage to the brain[17]. Despite above mentioned, side effects ECT is often well tolerated by patients including the very elderly[5].

ECT and mortality

The incidence of death occurring within 24 hours of treatment with ECT appears to be about one per 10,000 patients or one per 80,000 treatments[18]. This incidence does not differ significantly from the incidence of death in patients exposed to general anaesthesia alone. Most often, cause of death is a cardiovascular complication, including myocardial infarction, myocardial rupture or ventricular arrthymia.

ECT and psychotropics

The combined use of antidepressant medication and ECT does not appear to enhance the clinical benefits of ECT, although standard practice in the UK favours concomitant prescribing. It is wise to avoid agents with potent anticholinergic effects as they can significantly worsen cognitive impairment. Combining ECT with antidepressants like monoamine oxidase (MAO) inhibitors can lead to problems with blood pressure management during the ECT[12]. Drugs like benzodiazepines and anticonvulsants raise the seizure threshold leading to administration of higher doses of electrical stimulus or rendering the treatment ineffective. Commonly used antidepressants and antipsychotics may lower seizure threshold.

Predictors of outcome

Response to ECT is better in older adults[15]. In a relatively large number of studies, being elderly was positively associated with superior ECT outcome. ECT is more effective in depressive episode

Table 2. Positive predictors to ECT

Older adults

Depressive episode associated with:
> endogenous symptoms
> psychomotor retardation
> depressive delusions

Good response to ECT in the past

associated with endogenous symptoms, psychomotor retardation and depressive delusions. In addition patients who had responded well to ECT in the past, have relatively better outcomes[19]. (*Table 2*).

Several studies have noted that patients with a long duration of their current episode, ill-adjusted personality, hypochondriacal symptoms and neurotic traits are less likely to respond to ECT[19]. In the past, it was believed that as long as generalised seizures were of sufficient duration, maximal therapeutic effects would be obtained. It is now recognised, however, that seizure duration bears little relation to efficacy of ECT[20]. The predictive relationships between ECT outcome and age, episode duration and psychotic symptoms are of theoretical interest, they are of insufficient strength to guide treatment recommendations at individual level.

Maintenance ECT

The term 'maintenance ECT' refers to the use of ECT in the longer term as a prophylactic treatment to prevent recurrence. In several case studies, maintenance ECT has been reported to be efficacious, safe and well tolerated in the management of recurrent mood disorders in the elderly. It is claimed maintenance ECT may well reduce relapse, recurrence and rehospitalisation. Some elderly patients who are refractory or intolerant to pharmacotherapy can show a good response to maintenance ECT. The outcome depends greatly on the rate of compliance.

There is no good evidence to suggest that ECT has prophylactic value in preventing recurrence following symptomatic recovery. The benefit, tolerance and cognitive impairment from maintenance ECT needs further evaluation because the literature to date is limited to case

Table 3. Summary of NICE guidelines on ECT[8]

> Should only be used for the treatment of severe depressive illness, a prolonged or severe episode of mania, or catatonia, to gain fast and short-term improvement of severe symptoms, after all other treatment options have failed, or when the situation is thought to be life-threatening.

> A risk/benefit assessment for the individual should be made and documented. It should include the risks associated with the anaesthetic, whether the person has other illnesses, the possible adverse effects of ECT (particularly problems with memory) and the risks of not having the treatment.

> Doctors should be particularly cautious when considering ECT treatment for elderly, because they may be at higher risk of complications with ECT.

> The person should be re-assessed after every session of ECT. There should be ongoing checks for any signs of memory loss, and as a minimum, a check at the end of each course of treatment.

> The treatment should be stopped as soon as the person has responded, if there are any adverse effects or if they withdraw their consent.

> Should not to be used as a long-term treatment to prevent recurrence of depressive illness.

> Should not be used in the general management of schizophrenia.

> It is recommended that more than one course of ECT should be considered only for people who have severe depressive illness, catatonia or mania and who have previously responded well to ECT. As for the first course of treatment, it should be used only to gain fast and short-term improvement of severe symptoms after all other treatment options have failed or when the situation is thought to be life-threatening and after discussion of the risks and benefits with the service user and where appropriate their advocate or carer.

> NICE recommends that information leaflets to help people to make an informed decision about their treatment should be developed nationally and should be available in formats and languages that will make them accessible to a wide range of service users.

Key points

- ECT is an important, effective and safe treatment option for a variety of neuropsychiatric disorders.
- Psychotic, treatment-resistant or severe depression are the most common indications for ECT.
- ECT may be a life-saving intervention for some patients.
- ECT is well tolerated even by the very elderly.
- There is no good evidence to suggest ECT has prophylactic value in preventing recurrence following symptomatic recovery.

reports and anecdotal evidence. Controlled studies are needed. Both the National Institute for Health and Clinical Excellence (NICE) (*see Table 3*) and the Royal College of Psychiatry do not recommend maintenance ECT as part of standard practice.

ECT and consent

ECT cannot be given to a patient mentally capable of making a decision about their treatment and declines it. All patients being considered for ECT should have should have a full discussion with their clinical team about the pros and cons, side effects and likelihood of response. Written information may also be helpful. The doctor should adhere strictly to the recognised guidelines about consent, should not place any pressure on the patient and remind them they have the right to change their mind either for or against treatment at any time[8]. If discussion and informed consent are not possible at the time of treatment, any advance directive should be fully taken into account and patients' advocate or a carer should be consulted. Should a patient be unable to give valid consent, ECT can only be given following an assessment under the Mental Health Act and after an opinion from an appointed independent consultant psychiatrist[8].

Conclusion

In summary, ECT is an important, effective and safe treatment option for a variety of neuropsychiatric disorders; psychotic, treatment resistant or severe depression are the most common indications. ECT may be a life-saving intervention for some patients; and is well tolerated even by the very elderly. The invasiveness of the procedure and the adverse effects like memory loss are limiting variables in its use. Most of the innovations in ECT over the past 20 years have sought to diminish the side effects while maintaining benefits. There is no good evidence to suggest ECT has prophylactic value in preventing recurrence following symptomatic recovery.

Conflict of interest: Dr Barnes is a member of ECT committee of Royal College of Psychiatrists. The other authors have no conflict of interests to declare.

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