

## Review

# Status of ECT in modern psychiatry

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### **Introduction**

ECT or electroconvulsive therapy (electroplexy) as it is sometimes termed, has been in use for the treatment of psychiatric disorders since 1937. A large number of specialists and general practitioners working outside the field of psychiatry may have preconceived ideas or use of ECT in psychiatric treatment. Some are under the impression that its use is now obsolete, others seem to think that neuronal damage to the cerebrum would result from its use. Even some, psychiatrists have called ECT a barbaric form of treatment! These misconceptions are not warranted and its use continues in most parts of the world, though to a much lesser extent than in the nineteen sixties, due to the development of antidepressant and neuroleptic drugs. In fact in the USA a greater number of ECTs are given now by psychiatrists than ten years ago<sup>1</sup>.

### **Historical aspects**

In 1935 Von Meduna introduced convulsive therapies for the treatment of schizophrenia as he found that patients lost their symptoms after a spontaneous convulsion. He used camphor in oil given intramuscularly. As there was pain at the site of injection, metrazol (Cardiazol) was given intravenously to induce a convulsion. More recently hexafluorodiethyl ether (Indo-

klon) was used as an inhalant through a mask and vaporizer to produce a convulsion. Even today [some centres in USA and Europe use this form of convulsive therapy, together with muscle relaxants. A serious complication of this type of treatment has been postconvulsive vomiting. In 1938 Cerletti and Bini produced convulsions in patients, electrically stimulating the frontal lobes, using alternating current from a simple apparatus. The ECT was given without either anaesthesia or muscle relaxants. Patients developed fear without anaesthesia and due to the absence of muscle relaxants, collapse of vertebral bodies, fracture of vertebral transverse processes and dislocations of the mandible and shoulder joints occurred. Most of these were eliminated by proper positioning and holding of the patient during the convulsion. Unmodified form of ECT was used in this country till the late sixties.

At that time, ECT was thought to be a panacea for all forms of mental illness and its use became widespread and indiscriminate. The author has administered over 200 unmodified ECT per day at the mental hospital in the midsixties. Electroconvulsive therapy thus fell into disrepute, because this treatment is not useful for all forms of psychiatric disorders.

### **Technique — modified ECT**

Today the administration of modified ECT is a safe procedure. ECT is usually

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administered in the morning. The patient is kept fasting from midnight. Dentures and contact lenses are removed, and where there are loose teeth a sponge pad is inserted between the upper and lower sets of teeth. Anaesthesia best done by a consultant anaesthetist, is induced by intravenous thiopentone 0.25Gm in 5ml of sterile water + 1mg of atropine sulphate given slowly. The syringe is disconnected and 30 to 50mg of succinylcholine (Scoline) is given IV, through the same needle for muscle relaxation. An airway is inserted and a few puffs of oxygen given through a mask. The electrodes dipped in sodium bicarbonate or smeared with electro-jelly is placed bilaterally over the frontoparietal areas; an alternating current of 30-40 joules is passed at 120 volts for 0.1 to 0.5 seconds. In a properly modified convulsion only the toes should show movement. Further oxygenation should continue through a pressure bag, till spontaneous breathing resumes. Recovery takes place rapidly, though the patient may be somewhat confused. Using this method a number of patients could be treated on an outpatient basis.

### Clinical indications

1. Severe delusional depression.  
In this type of illness paranoid or nihilistic delusions are present. ECT together with antidepressant medication is very effective in treatment<sup>2</sup>.
2. Depressive illness where suicide is imminent and rapid alleviation of symptoms is important<sup>2</sup>.
3. Depressive illness that has not responded to a course of antidepressant medication carried out over a period of 6 to 8 weeks.

4. Schizophrenia. In schizophrenia or a relapse of the illness resistant to neuroleptic medication; and in the removal of the affective components of a severe schizophrenic attack, ECT together with neuroleptic medication have been used successfully<sup>3</sup>.

### Risks and contraindications

ECT is a surprisingly safe procedure. The mortality rate of ECT, in a survey of 259,000 treatments investigated by Barker and Baker, was 3 deaths per 100000 treatments<sup>4</sup>. The deaths followed myocardial infarction or ventricular arrhythmia where severe hypertension was a factor. Hesche and Roeder in Denmark obtained a similar mortality rate following ECT treatment<sup>5</sup>. With the introduction of muscle relaxants in ECT treatment, fractures have become virtually unknown. Acute respiratory disease carries a risk of disseminating the infection to other parts of the lung. There is an anaesthetic risk in a severe asthmatic who has been on recent prednisolone therapy. A vial of an intravenous cortisone preparation should be at hand in such cases together with aminophylline, to be given intravenously.

In an elderly patient with arteriosclerosis and hypertension there may be a transient memory defect for recent events, which may last usually upto about a month from the cessation of treatment. To minimize the recent memory defect unilateral placement of electrodes over the temporo-parietal area of the non dominant hemisphere was used (D'Elia and Raotma)<sup>6</sup>. However though the memory defect was marginally minimized, more ECT were needed to eliminate the

depression. Therefore the bilateral placement of electrodes is still favoured by British and American psychiatrists, though the Danish psychiatrists prefer unilateral placement<sup>7</sup>.

This controversy of ECT and memory defects is still going on. ECT invariably produces EEG changes. With bilateral electrode placement, paroxysmal delta activity appears in the frontal leads after 2 or 3 treatments. A minority of patients may develop spike foci. The EEG activity wanes rapidly with the cessation of treatment and returns to normal in 3 months (Klotz)<sup>8</sup>. According to Freeman and Kendell, 7% had memory defects, especially the retrograde component and retention of newly acquired information<sup>9</sup>. These transient minor memory defects are a much smaller price to pay, than a potentially dangerous depression which may eventually lead to suicide. Weeks, testing past personal memories of patients at 3 and 6 months intervals after bilateral ECT could not detect any changes<sup>10</sup>. A large number of electric shocks administered to laboratory animals did not show cerebral oedema, neuronal degeneration, gliosis or petechial haemorrhages. Bergsholm et al performed CT scans on 40 depressives, aged 26 to 87 years, before and after long courses of ECT, but could not detect any brain damage<sup>11</sup>. ECT is contraindicated in a case of recent myocardial infarction. It is also contraindicated in any form of organ failure. Organically determined coma is another contraindication. ECT should not be used in a recent case of head injury. Earlier, cerebral tumours were a contraindication. Recently ECT had been successfully administered to alleviate depression in a case of metastatic carcinoma by Dress-

ler and Folk<sup>12</sup>, Hsiao and Evans in a parietal meningioma<sup>13</sup>, & Greenberg Mofson & Fink in a case of meningioma<sup>14</sup>. We would consider the presence of raised intracranial pressure or cerebral aneurysms, absolute contraindications for ECT.

Insertion of a cardiac pacemaker is not a contraindication for the use of ECT, and neither is senility. Atre Vaidya and Jampala suggest that ECT is a safe and effective treatment for either depressive or manic symptoms associated with Parkinsonism<sup>15</sup>. Earlier ECT was not used in cases of Parkinsonism with depression.

#### **Mode of action of ECT**

Three powerful factors are operating when ECT is administered (a) passage of the electric current, (b) a period of unconsciousness and (c) a concerted effort in patient caring by the staff administering the ECT.

It has been demonstrated that the passage of an electric current through the brain results in the increase of the synthesis and utilization of epinephrine especially in the limbic system and the hypothalamus. This could account for the improvement in depressive mood disorders<sup>16</sup>. Before the advent of modern neuroleptic drugs, ECT was successfully used in the treatment of schizophrenia<sup>3</sup>. This would indicate that the current may be causing an increase in the sensitivity of the post-synaptic dopamine receptors. Bolwig showed a temporary breakdown in the blood brain barrier in animals after one electroshock and he also demonstrated the temporary permeability of small molecules to the blood brain barrier.

In doubleblind trials of ECT against simulated ECT in depressive patients, all clinicians agreed that ECT was far superior to simulated ECT<sup>17</sup>. The exception was the trial in Southampton carried out by Lambourn and Gill, where a significant difference between ECT and simulated ECT was not demonstrated<sup>18</sup>.

In a memorandum issued by the Royal College of Psychiatrists two major trials carried out by Greenblatt et al<sup>19</sup>, and the Medical Research Council<sup>2</sup> indicated the superiority of ECT over conventional antidepressant medication at 4 months, but this improvement levelled out at 6 months.

### Conclusion

Modified ECT is a safe and effective procedure in the treatment of certain psychiatric conditions. The only drawback being the occasional transient loss in recent memory especially in old people where long courses of frequent ECT have been administered.

### REFERENCES

1. American Psychiatric Association. Electroconvulsive therapy: 'Report of the task force on ECT of the American Psychiatric Association, Task force report 14. Washington D. C. 1978.
2. Medical Research Council Clinical Psychiatry Committee. Clinical Trial of the treatment of depressive illness. *B M J* 1965; **1**: 881-886.
3. Taylor P J, Fleming J J. ECT for schizophrenia. *Lancet* 1980; **i**: 1380-1382.
4. Barker J C, Baker A A. Deaths associated with electroplexy. *Journal of Mental Science* 1959; **105**: 339-348.
5. Hesche J, Roeder E. Electroconvulsive therapy in Denmark. *British Journal of Psychiatry* 1976; **128**: 241-245.
6. D'Elia G, Raotma H. Is unilateral ECT less effective than bilateral ECT? *British Journal of Psychiatry* 1975; **126**: 83-89.
7. Valentine M, Keddle K M G, Dunne D. A comparison of techniques in ECT. *British Journal of Psychiatry* 1968; **114**: 989-996.
8. Klotz M. Serial EEG changes due to electrotherapy. *Diseases of the Nervous System* 1955; **16**: 120-121.
9. Freeman C P, Kendall R E. ECT. Patient's experiences and attitudes. *British Journal of Psychiatry* 1980; **137**: 8-16.
10. Weeks D, Kendall R E. ECT Patients who complain. *British Journal of Psychiatry* 1980; **137**: 17-25.
11. Bergsholm P, Larsen J L, Rosendahl K, Holsten F. ECT and cerebral computed tomography. *Acta Psychiatrica Scandinavia* 1989; **6**: 566-72.
12. Dressler D M, Folk J. The treatment of depression with ECT, in the presence of brain tumour. *American Journal of Psychiatry* 1975; **132**: 1320-21.
13. Hsio J, Evans D L. ECT in a depressed patient after craniotomy. *American Journal of Psychiatry* 1984; **141**: 442-43.
14. Greenberg L B, Mofson R, Fink M. Electroconvulsive therapy in a delusional depressed patient with a frontal meningioma, a case report. *British Journal of Psychiatry* 1988; **153**: 105-107.
15. Atre-Vaidya N, Jampala V C. Electroconvulsive therapy in Parkinsonism with affective disorder. *British Journal of Psychiatry* 1988; **152**: 152-158.
16. Freeman A M, Kaplan H I, Saddock B J. Modern Synopsis of Psychiatry 11. U. S. A. Williams and Wilkins Co. 1977; 975.
17. Kendall R E. Present status of ECT, *British Journal of Psychiatry* 1981; **139**: 265-283.
18. Lambourn J, Gill D. A controlled comparison of simulated and real ECT. *British Journal of Psychiatry* 1978; **133**: 514-519.
19. Greenblatt M, Grosser G H, Weschler H. Differential response of hospitalized depressed patients to somatic therapy. *American Journal of Psychiatry* 1964; **120**: 935-43.