INVITED ARTICLE

MYTHS AND REALITIES OF ELECTROCONVULSIVE THERAPY

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ABSTRACT

With its introduction in 1938 electroconvulsive therapy became a powerful form of treatment for depressive illness and the major psychoses. Its use over the years has declined with the development of effective antidepressant and antipsychotic drugs. It still has a place in psychiatric treatment where urgent resolution of symptoms is indicated in certain conditions. The mechanism by which it exerts its therapeutic effect remains unknown.

Keywords: *Electroconvulsive therapy (ECT)*

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INTRODUCTION

Electroconvulsive therapy (ECT) has been in use in psychiatry for more than fifty years. Despite being an effective form of treatment for some psychiatric disorders, controversies surrounding its use and mode of action still remain.

HISTORICAL BACKGROUND AND MYTHS

Early theories about the causation of physical and mental illnesses centred on the belief that illnesses were caused by evil spirits. Based on this, the treatment was to drive out such spirits by whatever means that one could think of. Electricity was one of those means.

Electricity has been used to treat medical ailments even before man discovered how to generate it. It has been recorded that in the year AD 46, Scribonius Largus used the torpedo fish to cure headaches and gout[1]. In the 16th century, Ethiopians were applying the electric catfish to mentally ill patients to expel "devils out of the human body, and it did torment spirits no less than men". In the mid-eighteenth century, Michael Shuppach gave something resembling a course of ECT to a Swiss farmer's wife diagnosed to be possessed of 8 devils. She was given 7 shocks on consecutive days; the 8th devil, said to be the biggest, was given such a severe shock that the patient fell to the floor in a faint. Apparently a cure resulted. Desbois de Rochefort, in a treatise of 1779, recommended the use of electricity for various nervous complaints, and described its successful use in a patient with grief reaction. The first patient to receive anything like 'shock treatment' was probably a London porter who had been ill for a year. In 1792 John Birch, surgeon to St. Thomas Hospital reported that 6 small shocks were passed through the brain in different directions on each of 3 successive days, following which the patient recovered and remained perfectly well for 7 years.

It is not known whether any of the shocks described above induced grandmal fits as seizures would certainly be observed and described. Freud too, at one stage, was reported to have experimented with an electric machine but soon put it aside, concluding that its effect was only that of "suggestion on the part of the physician". His machine, like the others, delivered only sub-convulsive shock.

Seizures were first recorded definitely by Weikhardt in 1798. He used injections of camphor to induce "vertigo and fits". At that time, with tuberculosis much more common in mental hospital patients, there was a great deal of interest in the relationship between physical and mental illness and in conditions thought to be symbiotic or antagonistic. There was, for example, some suggestion of symbiosis in that tuberculosis was rife among schizophrenics and, conversely, of antagonism in that schizophrenia and epilepsy rarely occurred together. It was a common observation that psychotic patients lost their symptoms or became more subdued after a spontaneous fit. Several physicians, including Nyiro in Hungary in 1937, attempted unsuccessfully to treat schizophrenia with the serum of epileptic patients. Shortly before this, in 1933-4, Lazlo von Meduna, also a Hungarian, had begun using intramuscular camphor in olive oil, and then a synthetic form of camphor (pentamethylentetrazol or 'Cardiazol') to treat schizophrenics, and it was reported that his results were dramatic. The reports interested Ugo Cerletti, Professor of Psychiatry at Rome. Together with Lucino Bini, they developed their first ECT machine. The first patient to receive ECT was a schizophrenic vagrant in 1938. A grandmal fit resulted on the second application of current and the patient was discharged completely recovered after a number of further treatments given over the next two months.

The electrical method of fit induction was generally adopted as it was more predictable and safe. Just like any new treatment in medicine that has impressive effects in some patients, ECT became widely and extensively used in the 1940s and early 1950s. At that time it was considered to be the most effective treatment available for almost all the acute psychoses - schizophrenic, depressive, manic and organic. The introduction of chlorpromazine in 1953 and imipramine in 1957, and the wide range of phenothiazines, tricyclic antidepressants and butyrophenones which followed them, provided effective drug therapies for many of these illnesses. The widespread prophylactic use of lithium carbonate and long-acting phenothiazines and thioxanthenes has prevented the psychoses from relapsing. As a result the clinical indications of ECT have been shrinking for the last 30 years and its declining use is well documented throughout the world.

PRESENT DAY REALITIES AND MYTHS

Does ECT work? How does it work? What conditions are amenable to treatment with ECT? Is unilateral ECT better than bilateral ECT? The realities or otherwise of these issues have concerned psychiatrists over the last four decades.

Does ECT work?

The most fundamental question that can be asked of any treat-

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ment is whether it works. In the case of ECT the issue was given added importance by a series of widely publicised assertions in the 1950s to the effect that it was an unproven treatment which if it did work at all, did so by frightening or punishing the patient or by damaging his memory. In fact, the evidence that ECT is an effective treatment, especially for depression, is strong and came from many sources[2]. When it first came into widespread use in the 1940s its effect were so dramatic that those who knew from personal experience what untreated melancholia had been like beforehand never doubted its efficacy. Slater (1951)[3] showed that its introduction was associated with a major reduction in mortality and duration of hospital stay and Post (1978)[4] recalled its almost miraculous effect on melancholic patients who had previously had to be tube fed twice a day for years on end.

Efficacy and use in Depressive Illness

Studies on the effectiveness of ECT have mainly been done on Depressive Illness. In the 1960s two large multicentre trials of the treatment of depressive illness were carried out, one in USA, the other in England. In both, over two hundred inpatients were randomly allocated to treatment with ECT, imipramine, phenelzine or placebo. In the former (Greenblatt et al, 1964)[5] the proportion of patients showing "marked improvement" after eight weeks was significantly higher for ECT than for any of the other groups - 76% compared with 50% for phenelzine and 49% for imipramine. In the latter (Medical Research Council Clinical Psychiatry Committee, 1965)[6] ECT was more effective than phenelzine and more effective than imipramine in women, though not in men. It also acted faster than the other drugs, so that at the end of five weeks the proportion of ECT patients who have been discharged from hospital was twice as high as for either drug.

The above studies were not double-blind trials. In the 1970s and 1980s six double-blind studies of the efficacy of ECT in depression were done in the UK. In each of these patients with severe depression were randomly allocated to treatment either with real or 'simulated' ECT (ie the full ECT regime except the actual passage of an electric current through the brain). Five of the studies demonstrated a clear therapeutic benefit from real ECT as opposed to the 'simulated' ECT[7-11]. In only one of them (Lambourn and Gill, 1978)[12] was real ECT not shown to be significantly superior to 'simulated' ECT. In this latter trial, brief pulse right unilateral ECT was used. It is possible that the technique was relatively ineffective.

Recovery from ECT does not imply a lifetime free from further depressive illness. ECT treats the episode, not the underlying inclination to develop the illness. Whether there will be a relapse of further episodes of depressive illness depends on the individual's personality makeup and life events. The presence of biological symptoms of depression such as early morning insomnia, loss of appetite and weight, loss of libido, fatigue, impaired concentration are the best indicator that ECT may be beneficial, more so if depressive delusions are expressed or psychomotor retardation is present (Brandon et al, 1984)[16]. On the other hand, depression without biological symptoms, especially if associated with high anxiety and bodily over-concern ('neurotic depression') is not likely to respond to ECT.

The main place of ECT is in primary depressive illnesses that are severe, long-lasting or life-threatening. It is also indicated in illnesses where there are medical reasons for seeking rapid resolution of the symptoms, or in those cases where antidepressant drugs are not producing the desired result though given in adequate dosages and for sufficient periods.

Efficacy and use in Schizophrenia

Though initially used extensively to treat schizophrenia, ECT is much less used for this condition since the development of effective antipsychotic drugs which are used to treat cases in the first instance.

A number of studies have shown that ECT may have a place in the treatment of schizophrenia. Turek (1973)[13] found that ECT plus a neuroleptic hastened the resolution of symptoms in acute schizophrenia, shortening hospital stay and decreasing re-admission. This was not a double-blind trial.

Taylor and Fleminger (1980)[14] conducted a double-blind controlled trial comparing ECT with simulated ECT in combination with phenothiazines. Thirty-six patients with schizophrenia who subsequently matched the research criteria were treated with chlorpromazine (300 mg daily) or its equivalent for a minimum of two weeks. At the end of that period, 20 who had not improved were randomly allocated to real or simulated ECT given three times a week for 8 to 12 treatments, in addition to the neuroleptic drugs. While at two, four and eight weeks, the group receiving real ECT plus chlorpromazine showed significantly greater improvement; by sixteen weeks, the differences had disappeared. The symptoms most improved after real ECT were passivity, persecutory delusions, delusional mood and thought interference. There was no significant advantage for those patients with marked depressive features.

Janakiramaian and Channabasavanna (1982)[15] studied the effectiveness of ECT given with either a low (300 mg per day) or high (500 mg per day) dose of chlorpromazine. There was some advantage when ECT was given with the low dose of chlorpromazine, but none when given to those on the high dose.

Brandon et al (1985)[16] compared the effects of real with simulated ECT in a resultant group of 19 selected schizophrenic patients who were randomly allocated to either treatment. Each patient was to receive eight treatments at the rate of two a week. Two in the simulated group were withdrawn in the first week because of deterioration. The remainder completed the trial. Established neuroleptic medication was not changed and new courses were not permitted. During the four-week trial, the real ECT group did better and the improvement was not due to an improvement of depressive symptoms. However, at 12 and 28 weeks' follow-up, there was no statistical difference between the two groups, as after the 4-week trial period the clinicians were free to prescribe whatever treatment they wish.

Todate, there is no research evidence that ECT alone is effective as a treatment of schizophrenia. In all the published trials it has been used in combination with neuroleptic drugs.

In present day practice, for schizophrenia, ECT is used mainly as an adjunct to neuroleptic medication, in cases where the patients have not responded to drug therapy and remain too ill to leave hospital. Though often said to be the most effective treatment for catatonia, there is little published evidence for this. In any case catatonia has become uncommon since the advent of neuroleptic drugs. There is no place for giving ECT to chronic schizophrenics with negative symptoms.

Efficacy and Use in Mania

Although used widely in the past, and successfully, for the treatment of mania, ECT is much less used now because it has been supplanted by drugs of proven efficacy such as the phenothiazines, butyrophenones and lithium.

Until recently only retrospective studies were done. McCabe & McCabe (1976)[17] in a controlled study showed that ECT was as effective as chlorpromazine.

Small et al (1986)[18] reported the preliminary findings of a prospective study of ECT and lithium. ECT and lithium were found to be equally effective in the treatment of mania with some slight advantages for the ECT group at eight weeks and

with fewer recurrences and re-admissions at follow-up. The study is complicated by the fact that all the patients had quite large doses of neuroleptic drugs and that the majority of the patients had been on lithium when they became ill.

Use of ECT in other conditions

Though it has been reported that ECT may be effective in puerperal psychosis and in certain delirious states there have been no controlled trials to support these observations. ECT may be used to treat secondary depression in conditions such as obsessive-compulsive disorder or anorexia nervosa when other anti-depressants are not effective but not for these primary conditions per se.

Mode of Action of ECT

The mechanism by which ECT exerts its beneficial effects is not known. Initially, various psychological theories were proposed postulating that it was the patient's emotional attitude to the treatment which determined its efficacy and these were reviewed by Miller (1967)[19]. Firstly, it was suggested that fear induced by the treatment was the effective agent; secondly, that if the patient regarded the shock as a punishment, his conscience would be assuaged and his guilt and depression relieved; and thirdly, that the stresses involved in the treatment caused regression of behaviour to infantile levels allowing the patient to resolve early conflicts. These theories are all myths and are inconsistent with the fact that it is the induction of a fit which is of therapeutic value. Cronholm and Ottoson (1960)[20] showed that artificially shortening the duration of the epileptic discharge with lidocaine makes the treatment less effective.

Other theories attribute the therapeutic effect to confusion and amnesia following ECT, but there is good evidence that the therapeutic effect and the mental side-effects can be dissociated as when unilateral ECT is given. Furthermore, Ottoson (1960)[21] has shown convincingly that the memory impairment is related to the degree of electrical stimulation; the therapeutic effect is independent of this but varies with the duration of the epileptic discharge.

Following ECT there is a marked increase in anterior pituitary secretion of prolactin, adrenocorticotrophic hormone (ACTH) and beta-endorphin. The fact that the secretion of thyroid-stimulating hormone and growth hormone (GH) is not increased suggests some specificity of action on hypothalamic control of the pituitary gland. Posterior pituitary secretion of vasopressin is also increased. Thus far, none of the hormonal changes has been shown to be a reliable correlate of clinical response to ECT.

Studies in experimental animals with electrically induced seizures (ECS) have shown changes in 5-hydroxytryptamine (5 HT, serotonin), noradrenergic and dopamine systems which appear to be consistent with the monoamine theory of depression in human beings. However, it has not been possible to put forward a coherent theory of the neurochemical mechanism of action of ECT.

Unilateral versus bilateral ECT

There is still considerable controversy as to whether unilateral ECT (U/ECT) or bilateral ECT (B/ECT) should be routinely recommended. It is a known fact that the memory impairment that occurs with U/ECT is substantially less. However, with newer machines that can deliver brief-pulse low energy stimuli, the memory impairment with B/ECT is less than it used to be.

In a review by d'Elia and Raotma (1975)[22] in which they looked through 29 studies, 15 of the studies reported the two methods to be equally effective, 13 reported an advantage for B/ECT and one an advantage for U/ECT. Studies published since that time have shown greater advantages for B/ECT. The evidence seems to be that a greater proportion of depressed patients receiving B/ECT improve, that the percentage improvement is larger and that fewer treatments are required (Abrams, 1983, 1986)[23,24]. There is still a place for U/ECT in patients where speed of action is less important than minimising the side-effect of memory impairment.

CONCLUSION

In reality ECT is an effective treatment for depressive illness and other forms of functional psychoses such as schizophrenia and mania especially where speed of action is indicated. One has to bear in mind that it treats the episode, not the underlying illness. Its use has generally declined over the years with the development of effective anti-depressants and anti-psychotic drugs.

Recent studies have shown that bilateral ECT appears to be more effective than unilateral ECT. The exact mechanism of action of ECT remains elusive. Early psychological theories of its mode of action are myths as it has been convincingly shown that the induction of a fit is necessary to produce a therapeutic effect. Experimental studies have shown that ECT produces hormonal and neurochemical changes in the brain. From all these studies researchers have not been able to propose a coherent theory of its mechanism of action though the major effects on 5HT and noradrenergic neurotransmission appear to be relevant. Further studies are needed in this field. Present day hypotheses of its mechanism of action may turn out to be myths of tomorrow.

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