The effectiveness of electroconvulsive therapy: A literature review

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SUMMARY. **Aim** – To review the literature on the efficacy of electroconvulsive therapy [ECT], with a particular focus on depression, its primary target group. **Methods** – *PsycINFO, Medline*, previous reviews and meta-analyses were searched in an attempt to identify all studies comparing ECT with simulated-ECT [SECT]. **Results** – These placebo controlled studies show minimal support for effectiveness with either depression or 'schizophrenia' during the course of treatment (i.e. only for some patients, on some measures, sometimes perceived only by psychiatrists but not by other raters), and no evidence, for either diagnostic group, of any benefits beyond the treatment period. There are no placebo-controlled studies evaluating the hypothesis that ECT prevents suicide, and no robust evidence from other kinds of studies to support the hypothesis. **Conclusions** – Given the strong evidence (summarised here) of persistent and, for some, permanent brain dysfunction, primarily evidenced in the form of retrograde and anterograde amnesia, and the evidence of a slight but significant increased risk of death, the cost-benefit analysis for ECT is so poor that its use cannot be scientifically justified.

Declaration of Interest: Neither author has any financial conflicts of interest in relation to this paper.

KEY WORDS: ECT, evidence-based medicine, literature review, cost-benefit analysis.

Received 22.12.2009 – Final version received 11.03.2010 – Accepted 14.03.2010.

INTRODUCTION

The use of electricity to cause convulsions, in the hope of improving a person's mental health, is one of the most controversial issues in the mental health field. Paralleling the diverse and often strongly held beliefs about ECT, there are wide variations between and within countries in terms of usage, indications, modality, and degree of governmental or professional regulation (Asioli & Fioritti, 2000).

A recent editorial in the *British Journal of Psychiatry* celebrates 75 years of convulsive therapy, beginning with the work of Hungarian psychiatrist Laszlo Meduna. It reports that "despite the lack of evidence at this stage of therapeutic benefits, Meduna carried on with convulsive therapy", and that his "persistence was admirable" (Gazdag *et al*., 2009). The authors conclude that "ECT has saved and significantly improved the lives of tens of thousands of patients since the 1930s".

Since Meduna's day, however, it has been recognised that medical ineffectiveness is often the consequence of poor scientific research (Cochrane, 1972). There has been a global movement towards evidence-based medicine, defined as "the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients" (Sackett *et al*., 1996). Advocates of this approach assume that clinical decision-making should be informed by a hierarchy of knowledge, at the top of which stands data from placebo-controlled randomized controlled trials (Devereux & Yusuf, 2003; Cipriani *et al*., 2009). In keeping with this now well-established approach, this review of the effectiveness of ECT pays particular attention to comparisons of ECT and simulated-ECT [SECT], in which the usual general anaesthesia is administered but the electric shock is not.

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REVIEW METHODS

To ensure maximum possible inclusion of studies MEDLINE and PsycINFO were searched using the following combinations of keywords: 'electroconvulsive therapy' OR 'electroconvulsive treatment OR 'electroshock therapy' OR 'electroshock treatment' OR 'ECT' AND 'placebo' OR 'sham' OR 'simulated'. Reviews, meta-analyses, recent studies, and an independent review commissioned by the New Zealand government (Ministry of Health, 2004), were scanned to increase the detection rate. Only studies with human participants, presented in English, were included. Because there were only four depression studies with follow up-data (usually a requisite for demonstrating effectiveness) studies with data only for the treatment period (six) were included. This review also includes all the studies cited in a recent book in support of the conclusion that ECT is "a safe and effective treatment" (Shorter & Healey, 2007). The search identified eight meta-analyses in relation to depression (two of which also evaluated ECT for 'schizophrenia') and one that focused exclusively on schizophrenia. Besides using these meta-analyses to make the search as comprehensive as possible, they were also, themselves, reviewed, with particular attention to the accuracy of their reporting of studies and variation in their inclusion and exclusion of studies.

DOES ECT WORK?

When evaluating ECT, it is important that researchers observe methodological standards that have become widely recognised since the inception of the evidence-based medicine movement. Studies should be properly designed, with patients being randomised to treatment, and an adequate follow-up period using objective measures of outcome. In the case of ECT, it can be argued that the requirement to include a placebo control is particularly compelling because there is a prima facie case for assuming that applying electrical currents to the brain may be harmful. Hence, if researchers are to adhere to the first injunction in the Hippocratic oath (*primum non-nocere* – first do not harm; Gillon, 1985), they must demonstrate that the electroshock is a necessary component of the therapy, and that the procedure is not only effective but safe. To do this, researchers must compare gains made by ECT recipients with gains made by people who thought they received ECT but did not (Ross, 2006). Most studies claiming that ECT is effective fail to do this, including the NIMH-funded research by the Consortium on Research in ECT (eg Kellner *et al*., 2005; 2006).

Many studies report high 'response' rates for ECT. However, the UK ECT Review Group (2003) found that only 73 of 624 studies (12%) met their standards for inclusion in their review, adding: "The quality of reporting", of even this 12%, "was poor". Very few included a placebo condition, which is necessary to exclude the possibility that any observed improvement is the consequence of expectancy and hope in psychiatrists or patients.

For the first ten years this had not been feasible. Because of the frequent fractures cause by 'unmodified' ECT a disguisable placebo was impossible. In the early 1950s general anaesthesia was introduced. This 'modified ECT' could be evaluated by comparison with SECT control groups rendered unconscious but not given ECT. The failure of most studies over the next 60 years to follow this procedure is often justified in terms of the claimed ethical difficulties of withholding a treatment assumed to be effective and (despite claims that ECT is safe) imposing on a control group "a treatment which involves repeatedly rendering a control group unconscious" (Kendell, 1981). The *assumption* that ECT is effective is used to justify not using the method that can best determine whether it *is* effective.

In their 382 page book Shorter & Healy (2007) cite only four studies to support their claim that ECT is effective (other than four that relate to ECT preventing suicide – see below). All four are from the 1940s. Three of them (Kalinowsky, 1944; Myerson, 1941; Smith *et al*., 1942) had no control groups, vague or non-existent definitions of "recovery", and the people assessing "recovery" were either the hospital staff or unidentified (in the Myerson study none of the 'schizophrenics' improved). In the fourth (Tillotson & Sulzbach, 1945) a control group of "clinically comparable patients" improved less often (50%) than ECT recipients (80%), but there was no definition of "improved" and no mention of who decided who was "improved".

In the 1940s clinicians had became excited about the new treatment. Hope of recovery had returned to even the most depressing of institutions. Hope is a powerful placebo factor in psychiatric treatments, biological or psychological. It is important to clinicians and to patients. It can influence not just recovery itself but perceptions of recovery. Placebo effects in relation to ECT were acknowledged from the outset (see Brill *et al*., 1959). Neurologist John Friedberg suggested that in the early days "the influence of ECT was on the minds of the psychiatrists, producing optimism and earlier discharges" (Friedberg, 1976). Despite this possibility (which could not be evaluated without SECT), some early studies found lower

recovery rates for ECT recipients than for non-recipients (Karagulla, 1950) or no difference (Scherer, 1951).

The inadequacy of most ECT research continued throughout the rest of the 20th century. The methodological failings were not limited to failure to compare to a SECT group. In a *British Journal of Psychiatry* study, claiming that the proportions showing some improvement were 100% for depression and 98% for schizophrenia, the description of how improvement was measured was: "A record was kept of progress" (Shukla, 1981). A survey for the British Royal College of Psychiatrists simply gathered psychiatrists' opinions about improvement (Pippard & Ellam, 1981). (Despite the bias involved in asking psychiatrists to estimate patients' opinions, the number of patients deemed to believe they were "worse" after ECT was five times greater than that which the psychiatrists believed were worse).

COMPARISON WITH SIMULATED-ECT FOR DEPRESSION

It is hard to ensure that neither psychiatrists nor patients know who did and did not receive ECT, because of the confusion and headaches that frequently immediately follow ECT. In one study the patients in the SECT group, many of whom had had real ECT in the past, "believed that they were receiving some new variation on ECT" (Brill *et al*., 1959). The UK ECT Review noted that of 73 studies comparing ECT to drug treatment, no treatment or SECT "only two described the method of allocation concealment". So although comparison with SECT is the best research design it doesn't eliminate the possibility of placebo effects. Therefore even the very minimal positive benefits reported below may not have been caused by ECT.

Effectiveness during treatment period

Despite their claim to have conducted a "fair and comprehensive investigation of ECT", Shorter & Healy (2007) mention none of the studies reviewed next, the ones that best assess the effectiveness of ECT.

There have been ten studies comparing ECT and SECT for depression (Table I). Five found no significant outcome differences. One of these found identical response rates for ECT and SECT and concluded "The results suggest that the ECT pre-treatment procedure has an important therapeutic effect. This casts some doubt on current views of the effectiveness of electro-convulsive therapy" (Lambourn & Gill, 1978).

Of the five studies that did produce some significant findings, two invalidated their work, in terms of any lasting benefits, by giving real ECT to the SECT group after the first (Freeman *et al*., 1978) or third week (West, 1981). What these two studies can reasonably claim is that the ECT group improved faster than the SECT group (which also improved) early in the treatment, at least on some measures. In the Freeman *et al*. study there were no differences on the Beck Depression Inventory (in this study the raters were blind to group membership but the doctor giving the ECTs and SECTs, who obviously was not blind, was the lead researcher).

The third was the famous Northwick Park study (Johnstone *et al*., 1980). A prominent ECT advocate described it as "the most thoroughly designed and extensive trial of ECT's efficacy ever to be conducted in this country" (UK) but conceded that the "modest" difference found was "restricted to patients with delusions" and was "short-lived" (Kendell, 1981). There were no significant differences for two of the three subgroups of depressed patients: 'agitated' and 'retarded' (Nortwick Park ECT Trial, 1984). Furthermore, the positive finding for the 'deluded' subgroup was only perceived by psychiatrists. The ratings by nurses and by patients produced no significant differences for any of the three subgroups. The researchers themselves concluded that "The therapeutic benefits of electrically induced convulsions in depression were of lesser magnitude and were more transient than has sometimes been claimed" and "The results confirm that many depressive illnesses although severe may have a favourable outcome with intensive nursing and medical care even if physical treatments are not given" (Johnstone *et al*., 1980). A recent review of the effectiveness of SECT describes the Northwick Park study as "probably the best trial in terms of methodology and psychopathological characterization of patients" and comments on the fact that "rigorously defined endogenously depressed patients did exceptionally well with sham ECT, just as well as with real ECT. This needs explaining because it is common wisdom that endogenous (melancholic) depressions are not supposed to be placebo responsive. Perhaps melancholic patients in hospital do obtain considerable relief from milieu approaches" (Rasmussen, 2009).

The fourth study (Brandon *et al*., 1984) found significantly greater improvement in the ECT group, during the treatment period, for the 'retarded' and 'deluded' subgroups of depression, but not for the 'neurotic' subgroup. The fifth (Gregory *et al*., 1985) arguably provides the strongest evidence in favour of ECT. Although both the ECT and the SECT groups improved significantly by the end of the treatment, the ECT group improved significantly more than the SECT group.

Table I – *Studies comparing ECT and simulated-ECT for Depression.*

Study	Year	Significant Difference During Treatment	Significant Difference at Follow-Up
Brill *et al*.	1959	*[no data]*	no – 1 month
Harris & Robin	1960	no	*[no data]*
Fahy *et al*.	1963	no	*[no data]*
Wilson *et al*.	1963	no[1]	*[no data]*
Freeman *et al*.	1978	yes[2]	*[no data]*
Lambourn & Gill	1978	no	*[no data]*
Johnstone *et al*.	1980	yes[3] – 'deluded'[4]	no – 1 month
		no – 'agitated'	no – 6 months
		no – 'retarded'	
West	1981	yes[5]	*[no data]*
Brandon *et al*.	1984	yes – 'deluded'[4]	no – 2 months
		yes – 'retarded'	no – 5 months
		no – 'neurotic'	
Gregory *et al*.	1985	yes[6]	no – 1 month
			no – 2 months
			no – 6 months

1. Non significant on measure of current depression, significant on measure of depressive personality type. UK Review group concluded that the findings are not statistically significant
2. Study invalidated after one week by giving ECT to people in the simulated group. UK Review group concluded that the findings are not statistically significant.
3. Difference perceived by psychiatrists, but not by nurses or patients.
4. Subtypes of depression
5. Study invalidated after three weeks by giving ECT to people in the simulated group.
6. Both ECT and SECT groups improved significantly. ECT significantly greater improvement.

Effectiveness beyond the treatment period

None of the ten studies found significant differences beyond the end of treatment (Table I). Six did not follow up beyond treatment. Brill *et al*. (1959) found no significant differences after one month, concluding "It could very well be that the primary therapeutic agent is the psychological meaning of the treatment to the patient" and noting the "influence of the unusual amount of care and attention" involved. The Northwick Park study (Johnstone *et al*., 1980) found that even the one difference at the end of treatment (for one of three depression subtypes, observed by only one of three groups of raters) had disappeared four weeks later. It actually found a slight advantage for the SECT group. They added: "it is not possible to attribute the loss of advantage of real ECT [at the end of treatment] to differences in the subsequent treatment of the two groups". The other two studies that followed patients beyond the end of treatment found no differences between ECT and SECT at one or three months (Gregory *et al*., 1985) or at eight or 24 weeks (Brandon *et al*., 1984).

These findings are consistent with studies using less appropriate placebos. For example an early British Medical Research Council study had found that one week after the end of treatment ECT recipients were significantly more likely to have been discharged than patients given an inert medicine capsule (42% vs 25%) but that the difference was no longer significant four weeks after the end of treatment. By 20 weeks after the end of treatment 80% of ECT recipients and 88% of placebo recipients had been discharged (Medical Research Council, 1965).

This absence of evidence that ECT has any benefits beyond the treatment period is often countered by ECT advocates with the argument that this does not matter because the person can be treated subsequently with antidepressants. However, a well-designed study of subsequent pharmacotherapy followed up, for six months, the 159 of 290 (55%) patients with uni-polar depression who had shown improvement during ECT (Sackeim *et al*., 2001). In this randomized, double-blind trial, relapse rates were: anti-depressants and lithium – 39%, antidepressants alone – 65%, placebo – 84%. Thus even with the most effective (medical) relapse prevention strategy 45 of every 100 ECT recipients received no benefit in the first place and at six months a further 21 (55 X .39) had relapsed. Meanwhile the rate of sustained improvement that can reasonably be attributed to ECT at six months (the placebo group) is nine out of every 100 (55 multiplied by the 16% non-relapse rate in those receiving placebo medication). The authors concluded, "Our study indicates that without active treatment, virtually all remitted patients relapse within 6 months of stopping ECT".

Other ECT researchers have argued that the outcome data suggests that ECT must be continued on a maintenance basis if symptomatic improvement is to be sustained. Kellner *et al*. (2006), for example, examined out-

comes over six months of patients receiving a course of continuous ECT over a period of 6 months (shocks tapered from three times a week initially to monthly), either alone or in combination with pharmacotherapy. The authors concluded that both interventions "had limited efficacy, with more than half of patients either experiencing disease relapse or dropping out of the study".

Previous meta-analyses of comparisons with simulated-ECT for depression

Meta-analyses are important because they can produce a valid significant outcome from a series of non-significant findings if the original studies were underpowered. We identified eight meta-analyses comparing ECT and SECT for depression (Gabor & Laszlo, 2005; Greenhalgh *et al*., 2005; Janicak *et al*., 1985: Kho *et al*., 2003; Pagnin *et al*., 2004; Tharyan & Adams, 2005; UK ECT Review Group, 2003; van der Wurff *et al*., 2003). None cite any evidence that ECT is superior to SECT beyond the treatment period. All but Greenhalgh *et al*. (2005), however, claim that ECT is superior during the treatment period (although Kho *et al*. reported that there is no evidence for superior speed of action of ECT). Inspection of the meta-analyses reveals that the claim is questionable.

For example, Janicak *et al*. (1985) included six studies, only two of which were claimed to have produced significant differences between ECT and SECT. One was the West (1981) study in which ECT was given to members of the SECT group during the treatment period (see above). Their reporting of the other "significant" finding is incorrect. Ulett *et al*. (1956) studied patients with diverse diagnoses and should therefore be excluded from reviews regarding depression. Moreover, the study compared photoshock therapy (an early variant of shock therapy), ECT and SECT. Their actual findings were that 33% of the ECT group and 24% of the SECT group had shown "recovery" or "marked improvement" by the end of treatment. Janicak *et al*. (1985), however, incorrectly include data from the photoshock group, and a control group for that condition, to produce figures of 65% vs 35%. This is by far the largest study of the six in their meta-analysis and therefore this incorrect reporting (combined with the West study) largely accounted for the claimed overall effect size.

Pagnin *et al*. (2004) also include both the West *et al*. and Ulett *et al*. (1956) studies (but report the Ulett findings correctly as non-significant). Despite only one of seven studies (West, 1981) producing a significant difference, the studies do, when combined, find a significantly greater effect for ECT than for SECT at the end of treatment. The issue of differences beyond the end of treatment, however, is not even mentioned.

The UK ECT Review Group (2003) included six studies in their meta-analysis, including three which had found a significant difference during treatment. One of the three positive studies was, again, the West (1981) study in which ECT was given to some of the SECT group during the treatment period. The meta-analysis excluded four of the studies in Table I (Brandon *et al*., 1984; Brill *et al*., 1959; Fahy *et al*., 1963; Harris & Robin, 1960), three of which had found no benefit for ECT even during the treatment period. They report that only one study met their inclusion criteria for follow-up studies and found no significant difference. The study (West *et al*.) had not, in fact, reported any follow-up data.

The most recent systematic review was conducted for the UK's *National Health Service Research* and *Development Health Technology Assessment Programme* (Greenhalgh *et al*., 2005). The 170 page report concluded that "there is little evidence of the long-term efficacy of ECT" and that, even in the short-term, "low-dose unilateral ECT is no more effective than sham ECT", adding that the short-term gains from using higher doses or bilateral electrode placement "are achieved only at the expense of an increased risk of cognitive side-effects". The review found "no randomised evidence of the effectiveness of ECT in specific subgroups, including older people, children and adolescents, people with catatonia and women with postpartum exacerbations of depression or schizophrenia".

One of the eight meta-analyses focussed specifically on the effectiveness of ECT for its primary target group, the "depressed elderly" (van der Wurff *et al*., 2003). This Cochrane Systematic Review again found no evidence of ECT being effective beyond the treatment period. It identified only one study comparing ECT and SECT (O'Leary *et al*., 1994). This was a re-analysis of data from a study, by three of the reviewers (Gregory *et al*., 1985), which the reviewers described as having "major methodological shortcomings". It was concluded that "None of the objectives of this review could be adequately tested because of the lack of firm, randomised evidence".

Comparisons with Simulated-ECT for 'Schizophrenia'

Effectiveness during treatment period

The findings for patients diagnosed with 'schizophrenia' (for which ECT had originally been invented) are

remarkably similar. All but one of the early studies (e.g. Miller *et al*., 1953; Brill *et al*., 1959) found no significant differences between ECT and SECT. The exception did find short-term differences on psychotic symptoms but not on readiness for discharge (Ulett *et al*., 1956). A 2001 report by the American Psychiatric Association acknowledged that none of five pre-1980 ECT vs SECT studies found any differences, even in the short term. It claimed, however, that three later studies had demonstrated "a substantial advantage" for ECT (Abraham & Kulhara, 1987; Brandon *et al*., 1985; Taylor & Fleminger, 1980).

In all three studies, however, both groups were receiving anti-psychotic medication and any advantage, as we shall see, was again very short-term. An example of "substantial advantage" is the Leicester ECT Trial (Brandon *et al*. 1985). Both the ECT and the SECT groups improved on all four measures used. The real ECT group showed faster improvement on two of the four scales. 'Global psychopathology' did not differ at all. Another of these studies (Taylor & Fleminger, 1980) found that during treatment there was equal improvement in both groups but that ECT reduced general psychopathology faster than SECT for the first four weeks.

As was the case in a previously noted depression study (Johnstone *et al*., 1980), the psychiatrists in the Taylor & Fleminger (1980) study were the only ones to perceive any difference. Nurses and relatives did not. While this might have been because psychiatrists are better trained to rate symptoms, it is also possible that such differential findings are the result of wishful thinking on the part of psychiatrists. Another depression study found that psychiatrists saw improvement when the rest of the treatment team did not (Fahy *et al*., 1963). These findings suggest that the placebo effects of expectancy and hope do seem to be operating for psychiatrists.

A more recent Indian study of 36 people diagnosed with 'schizophrenia' found no differences in double-blind ratings on four symptom measures, after one, two, three or four weeks of treatment, between SECT and either bilateral or unilateral ECT (Sarita *et al*., 1998). A Nigerian study also failed to find significant differences between ECT (bilateral) and SECT at the end of treatment (Ukpong *et al*., 2002).

Effectiveness beyond the treatment period

The third study cited by the A.P.A. report as demonstrating "substantial advantage" (Abraham & Kulhara, 1987) found that both the ECT and SECT groups improved significantly during treatment but that the ECT group improved significantly more. It also found that ECT maintained its advantage over SECT at two and four weeks post-treatment. At eight weeks "the groups were similar" and the "advantage was totally lost with the passage of time". Using one-tailed t-tests had, however, doubled the chances of differences being judged significant. The only time that the more appropriate two-tailed test produces a significant difference is at two-weeks post-treatment.

In the other two studies any advantage for ECT over SECT had disappeared by the time of the first post-treatment assessment, at four weeks (Brandon *et al*., 1985; Taylor & Fleminger, 1980). Moreover, the Taylor & Fleminger study found that after four weeks the ECT group gradually deteriorated while the SECT group continued to improve, a pattern that was continuing 16 weeks after treatment. Similarly, Brandon *et al*. found that eight weeks after treatment ECT recipients had deteriorated on three of the four measures, but the SECT group continued to improve, overtaking the real ECT group on all four measures within two to six weeks. In this last study, improvements are graphically represented by change scores, thereby obscuring possible group differences at the outset. In fact, substantial baseline differences between the two groups can be discerned from the tables with, for example, the ECT groups scoring a mean of 9 (range 3-16) and the SECT group a mean of 12 (7-18) on the Montgomery-Asberg Schizophrenia Scale.

The recent Nigerian study that found no difference at the end of treatment also failed to find any significant differences 20 weeks later (Ukpong *et al*., 2002). Thus of eight follow-up studies (four on depression, four on 'schizophrenia') seven found no difference at the first follow-up and one, a 'schizophrenia' study (comparing ten ECT patients with ten SECT) found a significant difference at two weeks, but not four.

Previous meta-analyses of comparisons with simulated-ECT for 'schizophrenia'

None of three meta-analyses on schizophrenia report any evidence of any long term benefits (Greenhalgh *et al*., 2005; Painuly & Chakrabarti, 2006; Tharyan & Adams, 2005). A 2005 update of the Cochrane database found a short-term advantage for ECT over SECT but "no evidence that this early advantage for ECT is maintained over the medium to long term" (Tharyan & Adams, 2005). The same reviewers found that even in the short term ECT was less effective than antipsychotic medication. They concluded that "even after more than

five decades of clinical use, there remain many unanswered questions regarding its role in the management of people with schizophrenia".

DOES ECT PREVENT SUICIDE?

Despite the absence of benefits beyond the treatment period it can be argued that the faster recovery (in some studies only, for some subgroups only, perceived by some raters only, and usually followed by relapse) can prevent suicide. It is on the basis of this claim that it is argued that it is not safe to wait for the similar, but sometimes slower, recovery that occurs without ECT. The claim that ECT prevents suicide is a cornerstone of the case for ECT (e.g. Gazdag *et al*., 2009; Kellner *et al*., 2005; Prudic & Sackeim, 1999; Sharma, 1999; Shorter & Healey, 2007). There are, however, no suicide studies comparing ECT and SECT. The New Zealand Government's report (2004) found "no definitive randomised evidence that ECT prevents suicide".

A review of broad comparisons with the pre-ECT era found that "These studies provided little indication that the introduction of ECT had a clear long-term impact on suicide" (Prudic & Sackeim, 1999). There are a few studies comparing suicide rates of groups who have and have not had ECT (Table II). The most commonly cited as evidence that ECT prevents suicide are two papers by Avery & Winokur (1976). Their 1976 study recorded all deaths of 519 depressed people three years post-discharge. There were four suicides among the 257 who had received ECT (1.6%) and four among the 262 who had not (1.5%). Later, they published the figures for six months. All four of the non-treated suicides had occurred within the six months. The difference (0% vs 1.5%) was not significant (Avery & Winokur, 1978).

Table II – *Studies of Suicide Rates Comparing People treated and not treated with ECT.*

Study	Year	Diagnoses	Significant Difference
Ziskind *et al*.[1]	1945	Affective Psychoses	yes
Huston & Locher[2]	1948	Manic-depressive	yes
Bond	1954	Psychosis	no
Bond & Morris	1954	Manic-depressive	no
Avery & Winokur	1976	Depression	no[3]
Eastwood & Peacocke	1976	Depression	no
Avery & Winokur	1978	Depression	no[4]
Tsuang *et al*.	1979	Schizoaffective	no
Babigian & Guttmacher	1984	Depression	no
Milstein *et al*.	1986	mixed	no
Black *et al*.	1989	mixed	no
Sharma	1999	mixed	no[5]
Munk-Olsen *et al*.	2007	mixed	no[6]

1. 66% treated with Metrazol, 33% with ECT (unmodified)
2. unmodified ECT
3. three year follow-up, ECT = 1.6%, non-ECT = 1.5%
4. same sample as 3, at 6mths, ECT = 0, non-ECT = 1.5% (non-significant; p = .15)
5. inpatient suicides 3.4 times more likely to have received ECT than a matched control group who did not commit suicide
6. those who had completed ECT in the previous week nearly five times more likely to kill themselves than inpatients not treated with ECT

Like Avery and Winokur, most researchers find no difference in suicide rates between ECT and non-ECT groups. For example, a study of 1076 inpatients found no differences over two years between depressed people who received ECT (2.2%), anti-depressant medication (2.6%) or neither (1.9%) (Black *et al*., 1989). A comparison of 89 patients who had killed themselves with 89 matched controls found no significant difference in the percentage who had ever received ECT (80% and 76% respectively). There were also no significant differences between the two groups in terms of whether, at last contact, they had received ECT, antidepressants or neither (Bradvik & Berglund, 2000). In a subsequent study, the same researchers also found that while suicide attempts were, overall, less frequent after ECT than after antidepressant drugs, there were significantly more *severe* suicide attempts (defined as highly lethal suicide attempts, (i.e. requiring intensive care) following ECT than following antidepressants (Bradvik & Berglund, 2006).

Two studies did find a difference, both in the 1940s using unmodified ECT, and neither for depression. In the first, a study of 'affective psychoses', only one third of the treated group had received ECT; in the rest convulsions were induced with Metrazol (Ziskind *et al*., 1945).

(Two people died during ECT, one on the ECT table). The second study compared 80 people with 'manic-depressive psychosis' admitted to hospital before the introduction of ECT with 74 with the same diagnosis treated with ECT after 1940 (Huston & Locher, 1948). Six (7.5%) of the untreated group and one (1.4%) of the treated group killed themselves during a follow up period of between three and seven years. The study involved a form of ECT discarded 60 years ago. Furthermore, it is impossible to know whether the different suicide rates can be attributed to ECT. Prior to ECT the untreated group were more disturbed than the treated group. Twice as many (31% v 16%) were classified as 'severe illness', more (72% v 58%) came from disturbed families ('mental illness', alcoholism, criminality etc.) and more (52% v 34%) were men – who have a higher suicide rate.

In their book Shorter and Healey (like many others) base much of their support for ECT on the claim that it prevents suicide. They cite five studies to support this claim. One study is incorrectly cited twice with different authors. This is the Metrazol study (Ziskind *et al*., 1945). So the actual number of ECT studies is three. In addition to the other study from the 1940s described above Huston & Locher, 1948 they cite Avery & Winokur (1976) and a NIMH study (Kellner *et al*., 2005).

The NIMH study is not a study of suicide. It is a study of *thinking about* suicide (which might be justified on the grounds that completed suicides are a very rare event and difficult to investigate without very large samples). The study, which had no control group, found that people receiving ECT scored lower on the 4-point 'Expressed Suicidal Intent' section of the Hamilton Rating Scale for Depression during inpatient ECT treatment. 'High risk' was considered to be *either*: 3. active suicidal thoughts, threats, gestures *or* 4. serious suicide attempt. Immediately before ECT 13 (2.9%) "received a score of 4 for reporting a suicidal event during the current episode". None did so at the end of the three week treatment period. Since the treatment was for the 'current episode' one wonders why these 13 should no longer report these recent suicide attempts so soon after having reported them. Given the memory dysfunction following ECT (see below), perhaps they had forgotten them.

What would it mean if the risk of suicide *was* reduced during ECT treatment? ECT usually takes place in a hospital (especially for suicidal patients). Indeed these are precisely the extreme sorts of cases – acutely suicidal people in hospital who have stopped eating, drinking and communicating – that are highlighted to make the case for ECT. If these severely depressed inpatients are given ECT and do not kill themselves while in hospital, how can we be sure it was the ECT rather than the medical and nursing care they receive that saved them? The claim that ECT helps ensure that patients do not kill themselves *after* discharge, by alleviating depression, is, as we have seen, not supported by any research.

Shorter and Healy are far from alone in claiming that ECT prevents suicide. For example, the authors of the study critiqued above (Kellner *et al*., 2005) claim that in an earlier study "Both suicide and mortality were reduced with treatment". This study (Prudic & Sackeim, 1999) had not studied either mortality or suicide. Like the Kellner study it studied "suicidal ideation or intent"; again, only for the treatment period and with no comparison to SECT or any other type of control group.

Can ECT increase suicide risk?

Two studies have reported an association between ECT and increased suicide risk. A review of 149 inpatient suicides in Denmark found that inpatients who had received ECT were slightly more likely to have killed themselves than those who had not received ECT. Contrary to the claim that ECT saves lives by preventing the immediate, short-term risk of suicide, those who had completed ECT in the past week were nearly five times more likely to kill themselves than inpatients not treated with ECT (Munk-Olsen *et al*., 2007). The second study compared 44 suicides during or within two days of being in a psychiatric hospital with a control group of 43 inpatients who did not kill themselves, matched for age, gender and diagnosis (Sharma, 1999). Only two (4.7%) of those who did not kill themselves had had ECT within three months of discharge. However, seven of those who killed themselves (15.9%) had received ECT in the three months prior to suicide (three following completion of a course of ECT, two during a course of ECT, and one while receiving maintenance ECT). Because neither of these studies involved patients being randomised to treatment, it is possible that highly suicidal patients were more likely to receive ECT. Nonetheless, the findings hardly testify to an anti-suicide effect.

ECT-RELATED DEATHS

Does ECT decrease (non-suicide) mortality?

It has been agued, by some, that ECT reduces mortality risk. The claim here is that ECT somehow prolongs life

in other ways besides the unsubstantiated claim that it prevents suicide. Again the most frequently cited paper is Avery & Winokur (1976), which did not involve the random assignment of patients to conditions, and therefore could not exclude the likelihood that clinicians are unwilling to give ECT to patients with substantial physical health difficulties, particularly cardiac problems. Excluding suicides, 2.7% of those who had received ECT (with or without anti-depressants) had died over a three year period compared to 6.5% of those who had not received ECT. The only causes that differentiated the ECT from the non-ECT groups were cancer (0.7% v 1.5%) and myocardial infarction (0% v 2.3%). The authors do not suggest that ECT prevents cancer. In relation to myocardial infarction they argue that "adequate treatment effectively interrupts this aspect of the natural history of depression". Another study did not find a lower rate of deaths from myocardial infarction, but did for deaths from respiratory diseases (Munk-Olsen *et al*., 2007).

Another study (Philibert *et al*., 1995) found no difference in mortality rates of patients aged 65 or older in the first nine months but did find a significant overall difference over a ten year period (ECT 37%, non-ECT 45%) The causes of death were not cited. About half the non-ECT group had in fact received ECT prior to the study period. The authors acknowledged that those who had not received ECT "were more likely to have had prior myocardial infarction, to have coexisting chronic obstructive pulmonary disease and to have been initially hospitalized on the medical-psychiatric unit, an area for patients with both medical and psychiatric diagnoses, than those receiving ECT as their first treatment".

A study of 372 ECT recipients found that 18 (5%) died within two years of treatment, slightly greater than the 22 deaths in the 704 equally depressed patients (3%) who did not get ECT (Black *et al*., 1989). An Australian study (Brodarty *et al*., 2000) found a death rate among ECT recipients of 21% over two years in people aged 65 to 74 years.

Numerous methodological difficulties cloud the interpretation of these findings. However, given that none of the above studies employed either randomisation or SECT, and given that the previously reviewed trial data shows that the clinical advantages of ECT are at best short-lived, it seems almost impossible that any reduction in mortality can be attributed to a persisting effect on depression. It seems much more likely that clinicians are more willing to give ECT to patients who they judge to be relatively physically robust.

Can ECT cause death?

Textbooks and official reports claim that the risk of death from ECT is very small. For example the American Psychiatric Association (2001) report devoted just one of its 245 pages to deaths caused by ECT, which it called "General Issues". (This is one page more than the Shorter and Healey book). The report claimed: "Published estimates from large and diverse patient series over several decades report up to 4 deaths per 100,000 treatments." This is false. A report by the UK's Royal College of Psychiatrists (1977) had cited studies ranging from 4 to 9 per 100,000 treatments. The A.P.A. Report states, without citing any research, that: "A reasonable current estimate" is "1 per 10,000 patients or 1 per 80,000 treatments." This statement was repeated verbatim by a R.C.P. report three years later (Benbow, 2004).

The American Psychiatric Association (2001) report repeats the claim, made for decades, that the ECT death rate is about the same as that associated with general anaesthesia for minor surgery (which has been estimated at one per 13,000; Lagasse, 2002). This ignores the fact that even if this were true for an individual ECT treatment, the risk to each ECT recipient is likely to be much greater than that of minor surgery because they receive multiple treatments (eight on average). Paradoxically, it is precisely this risk that is referred to by those arguing that repeated general anaesthesia is too dangerous to warrant the use of SECT as a control.

Rather than extrapolate from the experience of patients receiving minor surgery (who may differ from psychiatric patients on a number of important health parameters such as consumption of cigarettes and adequacy of diet) it is important to examine data collected from those who have received ECT. Impastato (1957) reported 254 deaths caused by ECT and calculated a death rate of one per 1,000 patients overall and a death rate in people over 60 years old of one in 200, 50 times higher than the American Psychiatric Association claim. Frank (1978) reviewed 28 articles in which psychiatrists had spontaneously reported ECT-related deaths. Out of 130,216 ECT recipients there were 90 ECT-related deaths, one death per 1,447 people, seven times greater than the official claim.

Of 2,279 ECT recipients at the Mayo Clinic in Minnesota, 18 died within 30 days (Nuttall *et al*., 2004). The paper reporting this, in the *Journal of ECT*, claimed that "all deaths appear to be unrelated to ECT", despite six being "of unknown cause". Furthermore, one was a cardiac arrest (within two days of ECT) and one a stroke, two of the most common causes of death from ECT

(Kendell, 1981; Read, 2004). Excluding all six deaths of unknown cause, and including only these two, the rate would be one per 1,140 patients. Of 8,148 ECT recipients in Texas, seven died within 48 hours (Shiwach *et al*., 2001). Excluding the two "unlikely to have been related to ECT" this is a rate of one per 1,630. Eight more died within two weeks of "cardiac events". If these are included the rate becomes one per 627. A 1980 survey asked British psychiatrists to report ECT–related deaths (Pippard & Ellam, 1981). Including only deaths that occurred during or within 72 hours of treatment, there were four deaths in 2,594 patients. This is one per 648.5 people; 15 times greater than the American Psychiatric Association claim. Of the additional six that died within a few weeks of ECT two were from heart attacks and one from a stroke, two of the most common causes of death from ECT. Inclusion of these three deaths produces a rate of one death per 371 ECT recipients. In a Norwegian survey three of 893 women (one per 298) died as a result of ECT (Strensrud, 1958). It could not be determined whether the only death (four days after ECT) among 75 ECT recipients in France was ECT-related. This study, by anaesthetists, recorded one or more "complications" for 51 (68%), including 12 (16%) which were "potentially life-threatening" (Tecoult & Nathan, 2001).

All of these findings, all showing far greater levels of hazard than that claimed by official sources, depend predominantly on deaths being reported by those responsible for giving the ECT. A more objective measure was inadvertently provided in a study of patients' attitudes to ECT (Freeman & Kendell, 1980). The researchers wanted to interview 183 people, an average of one year after ECT. However, 22 (12%) were either dead or missing. Twelve were definitely dead. Four had killed themselves. Counting only the two deaths which occurred during ECT the mortality rate was 1 per 91.5 patients. This finding, over 100 times greater than the one per 10,000 American Psychiatric Association claim, is not mentioned by the American Psychiatric Association report (2001) or, to our knowledge, by any other report or review. The UK ECT Review Group (2003) mentions none of the studies reviewed above, showing higher rates than the American Psychiatric Association claim.

DOES ECT CAUSE MEMORY DYSFUNCTION?

Many medical interventions have adverse effects which must be included in a cost-benefit analysis. What, beyond the slight but significant risk of dying, are the costs which must be weighed against the possibility, for some patients, of short-term mood elevation or reduction in hopelessness and suicidal thinking? The effects of having sufficient electricity passed through your brain to cause a convulsion are many (Andre, 2008; Breggin, 2008; Rami-Gonzalez *et al*., 2001; Sackeim *et al*., 2007). Space does not permit a review of the literature on information processing speed and attention, nor on the many adverse emotional consequences (Johnstone, 1999). This review focuses on the most frequently cited cognitive effect, memory dysfunction.

A review identified four studies of memory loss at least six months post-ECT (n = 597), with a frequency range of 51% to 79%, and a weighted average of 70% (Rose *et al*., 2003). Four studies (n = 703) found a range for "persistent or permanent memory loss" of 29% to 55%, with a weighted average of 38%. The New Zealand Government report concluded that "ECT may permanently affect memory" (Ministry of Health, 2004) and bemoaned the "slowness in acceptance by some professional groups that such outcomes are real and significant in people's lives".

Retrograde Amnesia

Retrograde amnesia is the loss of memory for past events. Three facts are generally accepted:

i) Retrograde amnesia occurs to some extent in almost all ECT recipients,
ii) memory of events closest to the treatment are most affected, and
iii) some improvement occurs over time, with distant memories returning before recent ones (American Psychiatric Association 2001).

Even the American Psychiatric Association report (2001) acknowledges: "In some patients the recovery from retrograde amnesia will be incomplete, and evidence has shown that ECT can result in persistent or permanent memory loss".

Janis (1950) collected personal memories, from childhood to the present, from 30 people, 19 of whom then received ECT. Four weeks after ECT all 19 suffered "profound, extensive recall failures" that "occurred so infrequently among the 11 patients in the control group as to be almost negligible." Most were for the six months prior to ECT, but in some cases the loss was for events more than 10 years ago.

A larger study found that immediately after ECT memory gaps had been caused for a period spanning 25

years. This reduced to a three-year span seven months after ECT (Squire *et al*., 1981). Three years after ECT memory for events during the six months immediately prior to ECT remained lost (Squire & Slater, 1983).

Despite repeated claims, for 50 years, that ECT is safe, the first large-scale prospective study of cognitive outcomes following ECT did not occur until 2007. Prominent ECT advocate Harold Sackeim, *et al*. (2007), found that autobiographical memory was significantly ($p < .0001$) worse than pre-ECT levels both shortly after ECT and six months later. At both times the degree of impairment was significantly related to the number of shocks. Women and older people (both of whom are given ECT more frequently; Read, 2004) were particularly impaired. The impairment was also greater among those who received bi-lateral ECT rather than unilateral ECT (bilateral remains the most common form of ECT despite multiple previous findings of greater damage). Even using a conservative definition of two standard deviations worse than pre-ECT scores, 38 (12.4%) met the criterion for 'marked and persistent retrograde amnesia' (Sackheim *et al*., 2007).

A 1980 study produced the longest follow-up (Freeman *et al*., 1980). ECT recipients performed worse than non-recipients on ability to recall famous personalities from the 1960s and also on personal memories from early childhood. The average time since the last ECT was 8.4 years. Memory gaps after six months might, perhaps, be open to slight further filling in over time. After eight years the term "permanent", used by the American Psychiatric Association, New Zealand and U.K. reports, seems reasonable.

Anterograde Amnesia

Almost everyone receiving ECT suffers, as a result, anterograde amnesia, the inability to retain new information or recall events occurring after ECT. The Sackeim *et al*. (2007) study that found significant retrograde amnesia at six months, only found anterograde amnesia immediately after ECT. However, the American Psychiatric Association report (2001) cites 11 studies demonstrating anterograde amnesia in the first few weeks after ECT, concluding that during this time "returning to work, making important financial or personal decisions, or driving may need to be restricted". The report claims that "no study has documented anterograde amnesia effects of ECT for more than a few weeks". Studies showing that anterograde amnesia persists for four weeks (Feliu *et al*., 2008); two months (Porter *et al*., 2008; Squire & Slater, 1983) and three months (Halliday *et al*., 1968) might be considered consistent with "a few weeks." Two other studies are not. One found that ECT recipients were significantly impaired an average of 8.4 years after treatment, on retention of new information, such as repeating a paragraph of text (Freeman *et al*., 1980). The other found that ECT recipients scored worse than a non-ECT control group, on two memory tests used to assess brain damage, at both 10 and 15 years after ECT, which "suggests that ECT causes irreversible brain damage" (Goldman *et al*., 1972).

'Subjective' Memory Loss?

A common response has been to argue that memory dysfunction only occurs in those who don't recover from depression and that it is caused by the depression not the ECT. The term 'subjective memory loss' has become common in the literature. In 1995, however, McElhiney *et al*. (1995) identified five previous studies showing no significant relationship between anterograde or retrograde amnesia and clinical change after ECT. Their own study found that retrograde amnesia was related to the ECT and not to mood state before or after ECT. Another study (Neylan *et al*., 2001), which acknowledged that "the memory loss for events immediately preceding, during and after the treatment course can be permanent", found "no significant correlation between the change in depression rating and the change in any of the 12 cognitive measures". A recent review concluded that "There is no evidence of a correlation between impaired memory/cognition after ECT and impaired mood, much less a causal relationship" (Robertson & Pryor, 2006). The only large scale prospective study found no relationship between severity of depression and 19 of the 22 cognitive measures employed (Sackeim *et al*., 2007). Even if there was a correlation the causal relationship might have been in the other direction. It can be depressing to lose one's memory.

BRAIN DAMAGE

Evidence that the adverse effects of ECT are not imaginary or subjective' is provided by studies documenting brain damage (Breggin, 1984; 2008; Frank, 1978; Friedberg, 1976; 1977; Sterling 2000). In what is best described as a diatribe against "the old myth about ECT and brain damage" Shorter & Healy (2007) cite (amid studies of convulsive therapies prior to ECT or of ECT on animals) just one human ECT study (Coffey *et al*., 1991). This had found no structural changes (measured only by

ventricular enlargement) but had found an increase in subcortical hyperintensity due to cerebrovascular disease.

In the 1940s it was accepted that ECT worked precisely because it does cause brain damage and memory deficits. In 1941, Walter Freeman, who exported ECT from Europe to the U.S., wrote: "The greater the damage, the more likely the remission of psychotic symptoms. … Maybe it will be shown that a mentally ill patient can think more clearly and more constructively with less brain in actual operation" (Freeman, 1941). The paper was entitled "Brain damaging therapeutics". Myerson (1942) explained: "There have to be organic changes or organic disturbances in the physiology of the brain for the cure to take place. I think the disturbance in memory is probably an integral part of the recovery process".

In the 1940s and 1950s autopsies consistently provided evidence of brain damage, including necrosis (cell death). A review in the *Lancet* described ECT-induced haemorrhages and concluded that "all parts of the brain are vulnerable – the cerebral hemispheres, the cerebellum, third ventrical and hypothalamus" (Alpers, 1946). A review of the first twenty years of autopsies concluded: "damage to the brain, sometimes reversible but often irreversible, occurred in the course of electric shock treatments" (Allen, 1959). In 1974, Karl Pribram, head of Stanford University's Neuropsychology Institute wrote "I'd rather have a small lobotomy than a series of electro-convulsive shock … I just know what the brain looks like after a series of shock – and it's not very pleasant to look at" (Pribram, 1974).

More recently, CT scans have revealed increased frontal lobe atrophy amongst ECT recipients (Calloway *et al*.,1981; UK ECT Review Group, 2003). One review, which acknowledged that "both anterograde and retrograde memory impairment are common," actually documents the various forms of neurobiological dysfunction underlying the subtypes of ECT-induced memory dysfunction: Retrograde amnesia is a consequence of electrochemical dysfunction of the limbic-diencephalic subcortical areas involved in information retrieval, while in anterograde amnesia the medial temporal lobe is most affected (Rami-Gonzalez *et al*., 2001).

CONCLUSION

An earlier review, by one of the current authors (Read, 2004), concluded that: "There is no evidence at all that the treatment has any benefit for anyone lasting beyond a few days. ECT does not prevent suicide. The short-term benefit that is gained by some simply does not warrant the risks involved".

Two subsequent books (Andre, 2008; Breggin, 2008), and a review (Ross, 2006), have reached similar conclusions. A critique of the book which included the 2004 review (Read, 2004), by an eminent British psychiatrist (who found fault with other chapters), stated "Having at one time being involved in ECT research I found it difficult to fault John Read's account of this literature" (Crow, 2004).

Since the 2004 review there have been no findings that ECT is effective, but significant new findings confirming that the brain damage, in the form of memory dysfunction, is common, persistent and significant, and that it is related to ECT rather than to depression.

Few of those exposed to the risks of memory loss, and to the slight but significant risk of death, receive any benefit even in the short-term. There is no evidence at all that the treatment has any benefit for anyone beyond the duration of treatment, or that it prevents suicide. The very short – term benefit gained by a small minority cannot justify the significant risks to which all ECT recipients are exposed.

The continued use of ECT therefore represents a failure to introduce the ideals of evidence-based medicine into psychiatry. This failure has occurred not only in the design and execution of research, but also in the translation of research findings into clinical practice. It seems there is resistance to the research data in the ECT community, and perhaps in psychiatry in general. The reasons are beyond the scope of this review (Bentall, 2009; Doroshow, 2006). The recent British Journal of Psychiatry editorial acknowledges, however, that ECT "stimulated biological psychiatry" and "powerfully reinforced the belief in somatic treatment in psychiatry" (Gazdag *et al*., 2009).

ECT produces powerful placebo effects. A recent review (Rasmussen, 2009), which reported "an unexpectedly high rate of response in the sham [SECT] groups", concluded that "The modern ECT practitioner should be aware that placebo effects are commonly at play". It seems, however, that clinicians find it hard to recognise placebo effects even (perhaps especially) when they occur in front of them. A recent example is Parker's (2009) response to new evidence that anti-depressant medications have little benefit beyond placebo (Kirsch *et al*., 2008), in which every conceivable explanation for the findings is considered other than the power of placebo effects. Under these circumstances, practitioners may be reluctant to respond appropriately to negative cost-benefit analyses of therapies in which they have invested considerable time and effort and which they genuinely believe are safe and effective.

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