

Committee for Truth in Psychiatry
P.O. Box 1214
New York, NY 10003

To: Dockets Management Branch
Food and Drug Administration
5630 Fishers Lane (Room 1061)
Rockville, MD 20852

CITIZEN PETITION

The Committee for Truth in Psychiatry submits this petition pursuant to 21 CFR 10.30 to request the Commissioner of Food and Drugs to maintain the electroconvulsive therapy device (882.5849) in Class III for all indications.

Part A. SPECIFIC ACTION REQUESTED

The FDA is charged with the responsibility of categorizing medical devices according to the degree of risk they pose and the degree of oversight they require. The electroconvulsive therapy (ECT) device is a pre-amendments device which has never been tested for safety or efficacy; for which manufacturers have never presented any evidence of safety or efficacy although ordered to do so by the FDA on August 14, 1995 (FR 60: 41984); and for which no Premarket Application has been called for or submitted.

On September 4, 1979, the FDA classified the ECT device into Class III under the Federal Food, Drug and Cosmetic Act (FR 44:51777), identifying eight risks to health including brain damage and memory loss.

FDA's original classification was correct. Since that time, more valid scientific evidence has accumulated as to the risks of the device. Since that time, the manufacturers have not conducted a single study nor submitted any evidence showing that the original classification was incorrect. Under Section 515(i) (21 U.S.C. 360e(i)) of the Safe Medical Devices Act of 1990 (SMDA), manufacturers of ECT devices were required to submit all safety and effectiveness information known to them by August 14, 1997. No manufacturer ever submitted anything in response to the order.

We are asking FDA *not* to take an action: we are asking that it *not* reclassify the Device to Class II. We ask that it maintain the device as Class III under Section 515(i) of the SMDA. This petition is necessary because FDA has begun the process leading to reclassification.

Another way of stating our request is that we are asking that the ECT device be regulated in the same manner as other medical devices. Currently, this is not the case.

Part B. STATEMENT OF GROUNDS

The Committee for Truth in Psychiatry, founded in 1984, is the only national organization of patients who have been treated with the ECT device. We all had ECT without being truthfully informed of the risks of the device, and as a result we all suffered permanent harm to varying degrees (including memory loss, cognitive disability, and brain damage). The majority of our members lost our ability to work and to contribute to society due to ECT. We are fully familiar with the nature and history of FDA's regulatory proceedings regarding the ECT device. Obviously, we have a vital interest in protecting future patients from the preventable harm we experience.

On September 5, 1990, FDA published a proposed rule to reclassify the ECT device to Class II for depression. However, this proposed rule was never acted upon and was withdrawn by FDA on April 21, 2003.

We are aware that FDA is currently considering acting unilaterally, in the absence of any evidence or petition from the manufacturers or any other parties, to reclassify the ECT device based on a selective "literature review". An internal committee has been convened for this purpose. We are aware that reclassifying a device in the absence of *any* evidence from manufacturers is highly unusual if not totally unprecedented. FDA has never before reclassified a Class III device based solely on its own selective review of *some* of the literature on the device.

(N.B: For examples of what a rigorous *systematic* literature review looks like, see Source Documents #32 and #35.)

Section 513(3) (21 U.S.C. 360(e)) of the Act provides that device classifications may be changed by regulation only when there is "new information" supporting the change. Any reclassification is required to consist of "valid scientific evidence" as defined in section 513(a)(3) of the act (21 U.S.C. 360c(a)(3)) and 21 CFR 860.7(c)(2). This valid scientific evidence must be publicly available.

According to 21 CFR Ch 1 860.7 (c)(2), FDA recognizes five forms of valid scientific evidence:

- (1) Well-controlled investigations
- (2) Partially controlled studies
- (3) Studies and objective trials without matched controls
- (4) Well-documented case histories conducted by qualified experts
- (5) Reports of significant human experience with a marketed device.

A selective literature review does not constitute valid scientific evidence.

FDA has specified no criteria for inclusion of studies and has no plans to identify and make publicly accessible those studies not selected for inclusion. By its very nature, such a review can be tailored to any position. At all times since the American Psychiatric Association (the lobby for the device users) began its campaign to change the classification of the ECT device in the early 1980s, FDA has stated its intention to reclassify the device to Class II. There is no reason to think the agency has changed its position, and thus every reason to believe the "literature" can and will be selected to support that position.

Valid scientific evidence---now even more than in 1979 when the FDA correctly classified the device in Class III---shows that the device, when manufactured correctly and used as directed for any indication, presents an unreasonable risk of injury or harm. Further, its risks far outweigh its benefits, which are less than previously thought.

We have diligently searched for new evidence unfavorable to our petition. Our criteria were as follows:

- 1) Studies conducted by researchers free of financial, career, or other conflict of interest
- 2) Valid scientific evidence as defined above
- 3) Studies not previously considered by FDA
- 4) Studies documenting ECT's safety and efficacy:
 - a) Brain-imaging studies settling the question of whether ECT causes brain pathology or damage in the negative; or
 - b) Studies documenting the full return of memory, memory ability, and cognitive function to normal after treatment with the ECT device; or
 - c) Studies documenting long term (i.e. lasting more than one month) benefit from ECT, or its ameliorative effect on suicide risk or rate.

We found no such evidence.

We submit some of the new, valid scientific evidence supporting our petition in an appendix. The evidence is of the following nature:

The Manufacturers' Silence. This speaks for itself.

FDA's Own Files. More than 40 volumes of evidence is contained in the FDA's own files, Docket #82P-0316. We do not believe that anyone on the current FDA staff has read the entire file. Much of the material postdates the FDA's last evaluation of the device in 1990. A representative of our organization has read all the volumes and reports that more than 90% of the comments oppose the reclassification of the ECT device to Class II. Almost all who support reclassification are psychiatrists who use ECT. There are hundreds of reports of significant human experience with the device in the file. 97% of persons who identify as former ECT patients oppose reclassification, most of them on the grounds that it caused them permanent memory loss and/or disability.

A representative sample of these documents is included herein: Source Documents 10, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50

New Evidence for Permanent Memory Loss: Source Documents Nos. 7, 9, 11, 12, 13, 16, 18, 19, 20, 21, 23, 24, 25, 26, 27, 28, 29, 31, 32, 37

New Evidence for Permanent Memory or Cognitive Disability: Source Documents Nos. 1, 7, 8, 9, 11, 12, 13, 15, 16, 18, 19, 20, 21, 24, 25, 26, 27, 28, 29, 32, 37

New Evidence for Brain Damage: Source Documents Nos. 8, 9, 11, 14, 16, 22, 33, 34, 37

New Evidence of Lack of Efficacy: Source Documents Nos. 3, 6, 17, 25, 29, 30, 32, 35, 36, 37

New Evidence of Mortality and Morbidity: Source Documents Nos. 2, 4, 5, 29, 37

Sections 501(f), 513, and 515(b) of the act (21 U.S.C. 351(f), 360c, and 3603(b)), taken together, establish as a general requirement that a preamendments device that FDA has classified into Class III is subject, in accordance with Section 515 of the Act, to premarket approval. Premarket approval is appropriate for a device that has never been subjected to safety testing by the manufacturers nor subjected to any independent safety evaluation. It is crucial where there is decades of scientific evidence documenting permanent, serious adverse effects including death, and no evidence for longterm efficacy of the device in question.

We realize it may be awkward and challenging for the FDA to have to call for Premarket Approval Applications from manufacturers who have thus far been unresponsive to the agency, and who have successfully evaded the regulatory process for decades. However, this is exactly the reason why the United States government created the FDA: to regulate the drug and device manufacturers in the interest of protecting the public health. We realize that the device users' lobby, the American Psychiatric Association, has successfully pressured the FDA for over 20 years to prevent a call for PMAs. We believe that together, these factors help account for FDA's unwillingness to regulate the ECT device as all other medical devices. However, the agency has a legal duty and responsibility to ensure the safety of the American public. The ECT device must not be treated any differently from other medical devices because it has a well-financed and influential user lobby behind it.

Part C. ENVIRONMENTAL IMPACT

Not applicable.

Part D. CERTIFICATION

The undersigned, in her capacity as Director of the Committee for Truth in Psychiatry, certifies that, to the best of her knowledge and belief, this petition includes all data, information, and views on which the petition relies, and that it includes representative data and information known to the petition which are unfavorable to the petition.

Respectfully submitted,

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Petition to Maintain the ECT Device in Class III

Committee for Truth in Psychiatry

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Source Documents

Source Documents

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BRIEF COMMUNICATION

Pattern of cognitive dysfunction in depressive patients during maintenance electroconvulsive therapy

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ABSTRACT

Background. Objective data regarding adverse cognitive deficits associated with maintenance electroconvulsive therapy (M-ECT) are lacking. This study examined the cognitive state of depressive patients during M-ECT.

Method. A cross-sectional study was carried out in 11 depressive patients in remission, all with a DSM-IV diagnosis of major depressive disorder. The mean number of previous ECT sessions was 36.1, and the mean intersession interval was 52.7 days. A group of 11 patients who had not received ECT was selected for comparison and matched for diagnosis, sex, age and years of schooling. All subjects were assessed using a complete neuropsychological battery including memory, attention and frontal function tests.

Results. Groups did not present differences in long delay verbal recall. Encoding of new information and results on the frontal function tests were significantly lower in the M-ECT patients.

Conclusion. Depressed patients preserve long-term memory, but suffer short-term memory impairment and frontal function alteration during M-ECT. Further longitudinal studies are necessary to determine the influence of M-ECT on non-memory functions and different memory subtypes.

INTRODUCTION

Maintenance electroconvulsive therapy (M-ECT) is an effective treatment for the prevention of relapse and recurrence in patients with major psychiatric disorders, especially mood disorders (Petrides *et al.* 1994; APA, 2001; Sackeim *et al.* 2001). It has proved to be a safe option particularly useful in drug-resistant patients (Rabheru & Persad, 1997; Tew *et al.* 1999) and in medically compromised subjects who are sensitive to the toxic effects of medication (Sackeim *et al.* 2001). M-ECT does not present any adverse physical effects other than those found in acute courses

of ECT (Jaffe *et al.* 1990; Fink *et al.* 1996), but the risk of cognitive dysfunction associated with the treatment remains unknown. Previous data are based on case reports (Grunhaus *et al.* 1990; Devanand *et al.* 1991; Barnes *et al.* 1997), retrospective studies without a control group (Brodaty *et al.* 2000), subjective evaluations of cognitive dysfunction after treatment (Vanelle *et al.* 1994) or studies based on global cognitive screens such as the Mini-Mental State Examination (Thienhaus *et al.* 1990). Information has also been extrapolated from acute ECT cases: most reports have shown a transient memory dysfunction that disappears 3 to 6 months after the completion of treatment (Rami-González *et al.* 2001). Other cognitive functions such as attention or frontal functions have not been

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widely studied, though they appear to be less affected than memory after an ECT course (Calev *et al.* 1995). Consequently, no objective data are available on the adverse cognitive effects during continuation ECT sessions. Since the time between treatments is much longer during M-ECT, fewer cognitive side effects would be expected than during an acute course of ECT. The aim of this study was to determine the profile of cognitive deficits in depressive patients during an M-ECT protocol.

METHOD

Eleven consecutive drug-resistant patients participating in the M-ECT programme were selected for inclusion in the study by the Institute of Psychiatry and Psychology at the Hospital Clinic, Barcelona. Nine patients met DSM-IV criteria for recurrent major depressive disorder, and two for a single episode of major depressive disorder with melancholic features. The mean duration of illness was 12.8 years (s.d. = 9.2) and the mean number of depressive episodes was 3.1 (s.d. = 2.4). Mini-Mental State Examination scores were between 25 and 29 in all cases. The frequency of sessions was adapted to each patient's needs. The mean inter-session interval in the sample was 52.7 days (s.d. = 16.8; range 30–90 days). Patients receiving more than one session per month were not included in the present study. The mean of total previous ECT sessions was 36.1, and the mean time of M-ECT was 27.2 months. Nine of the 11 finished their acute ECT course 6 months before assessment; in the two remaining patients 5 months had passed since the ECT course. Patients were treated with the standard bilateral fronto-temporal electrode placement with a customized MECTA spectrum device. Anesthesia consisted of atropine (0.4 mg), succinylcholine and thio-barbital.

Eleven depressive subjects who had never received ECT were used as a control group, matched with the M-ECT patients regarding sex, age and years of schooling. A difference in age of ± 3 years was accepted. Six of these patients met criteria for recurrent major depressive disorder and five for a single episode of major depressive disorder with melancholic features. The mean duration of illness was 11.1 years (s.d. = 7.3) and the mean number of depressive

episodes was 1.7 (s.d. = 0.8). Most patients were treated with psychotropic medications and the drug type used in both groups was similar. In the M-ECT group three subjects were treated with selective serotonin reuptake inhibitors (SSRI), three with selective noradrenaline reuptake inhibitors (SNRI), and three with tricyclic antidepressants. In addition, one patient was taking benzodiazepines, and one neuroleptics. In the control group, four patients were treated with SSRI, three with SNRI, two with tricyclics and two subjects were taking benzodiazepines.

Written informed consent was obtained from all patients and controls after the procedures had been fully explained. Clinical and cognitive assessment was administered to all subjects. ECT patients were assessed the day of the ECT session, in order to guarantee maximum inter-session time. Clinical state was measured with the Hamilton Depression Rating Scale (HDRS) (Hamilton, 1967). All patients had been in remission (HDRS < 8) for at least 3 months before neuropsychological assessment. The cognitive battery included an assessment of global cognitive function by Mini Mental State (MMSE) (Folstein *et al.* 1975). Memory subtypes were evaluated with the Logical Memory test of the Wechsler Memory Scale (WMS) (Wechsler, 1945) and the Auditory Verbal Learning Test (RAVLT) (Rey, 1964). To assess the encoding of new verbal information or short-term memory we used Logical Memory part I and total learning of the RAVLT. Information retention or long-term memory was assessed by delayed recall at 20 min of the logical memory story and the words in the AVLT memory list. The Digits Forward test on the Wechsler Intelligence Scale for Adults (Wechsler, 1955) was used to assess selective attention and the Trail Making Test – part A (Army Individual Test Battery, 1964) assessed simple motor velocity in addition to attention. The Digit-Symbol Coding test from the WAIS was used to determine visuomotor speed. Frontal functions were assessed by the Trail Making Test (part B) (Army Individual Test Battery, 1964), the Tower of Hanoi (Goldberg *et al.* 1990) and the Digits Backward test, measuring mental flexibility, planning, and working memory respectively. Verbal fluency, measured by the FAS-test (Borkowski *et al.* 1967), was also used as an indication of frontal function.

Table 1. Characteristics and case-control comparisons using Wilcoxon Rank test

Characteristic	M-ECT group		Comparison group		Wilcoxon Rank test	
	Mean	(s.d.)	Mean	(s.d.)	Z	P
Characteristic						
Age (years)	63.9	(8.8)	63.8	(9.1)	-0.1	0.92
Education (years)	8.0	(2.4)	9.3	(2.9)	-1.4	0.17
HDRS	3.5	(2.7)	2.5	(2.7)	-1.6	0.17
MMSE	27.5	(1.6)	28.2	(1.7)	-0.9	0.32
Total ECTs, N	36.1	(11.1)				
Time in ECT treatment (months)	27.2	(17.7)				
Neuropsychological battery						
Memory						
RAVLT Total Learning	14.3	(4.7)	16.3	(4.2)	-0.8	0.406
RAVLT (% Verbal Recall in delay)	74.7	(27.9)	72.2	(20.4)	-0.3	0.721
Logical Memory (LM I)	5.2	(2.6)	8.1	(1.9)	-2.7	0.01
(LM I - delayed LM) (raw score)	1.3	(0.9)	2.4	(1.6)	-1.8	0.066
Frontal tests						
Trail Making 'B' (s)	219.0	(88.4)	152.7	(66.8)	2.4	0.035
FAS-test (total words)	20.6	(7.0)	27.9	(8.6)	-2.5	0.029
Digits Backward	3.4	(0.7)	4.3	(1.2)	-2.8	0.020
Digit Symbol Coding	19.4	(7.5)	31.4	(14.4)	-2.7	0.023
Tower of Hanoi	13.8	(5.9)	10.6	(3.7)	-1.2	0.233
Attention						
Trail Making 'A' (s)	91.6	(77.1)	67.4	(42.7)	-1.5	0.12
Digits Forward	5.0	(1.1)	5.1	(1.4)	-0.1	0.914

Statistical analysis was performed using SPSS for Windows V 9.0. The Wilcoxon Rank test was used for case-control comparison of cognitive measures. Non-parametric Spearman correlations were used to assess association within M-ECT group. Two-sided *P* values are given.

RESULTS

The mean age of the sample was 63.8 years (s.d. = 8.7; age range: 44-77). There were eight females and three males in each group. No differences in years of illness duration ($Z = -0.2$, $P = 0.84$) or number of depressive episodes ($Z = -1.6$, $P = 0.11$) were found between groups, nor regarding age, sex or years of schooling.

The M-ECT group scored lower on the encoding of new information, as expressed by raw scores on the Logical Memory (LM) I and Total Learning tests on the AVLT (Mittrushina *et al.* 1999). Only the LM I score was significantly lower. However, the M-ECT group obtained higher long-term memory scores than controls, but the two groups did not differ significantly in terms of variation in the direct scores between LM I and delayed LM or in percentage retention on the AVLT. The two groups also showed similar scores on Trail Making A and Digits Forward tests. With the exception of the Tower

of Hanoi, all frontal test scores were significantly lower in the M-ECT group. Results are presented in Table 1.

There was no significant correlation between cognitive measures and number of total ECT sessions, total time in M-ECT, or time between treatments, or between cognitive measures and age, years of schooling, number of episodes or years of evolution of illness. The small sample size may have limited the power to find significant correlations. The dispersion graph was analysed visually, and no significant outliers were found.

DISCUSSION

No differences were observed in long-term memory or in attention function between depressed patients undergoing M-ECT and matched comparison subjects. However, encoding of new information and performance on most tests of frontal function was significantly impaired in our M-ECT patients. Compared with controls, M-ECT patients showed alternations in verbal fluency, mental flexibility, working memory and visuomotor speed.

Memory impairment is the main neuropsychological problem associated with acute ECT, but the specific subtypes of memory dysfunction

associated with M-ECT remain unknown. The ability to consolidate information and recall it after a delay is clearly deficient after an ECT course (Frith *et al.* 1983; Hasse-Sander *et al.* 1988) and it is significantly affected for 3 to 6 months after an acute treatment. Long-term memory dysfunction is associated with the medial temporal lobe, though its precise mechanism remains unclear. Several explanations have been proposed: an abnormal long-term potentiation mechanism, excessive release of excitatory amino acids and the activation of their receptors such as N-methyl-D-aspartate (NMDA), decreased cholinergic transmission, increased cerebral blood pressure and reductions of regional cerebral blood flow may contribute to this transient memory dysfunction after an acute course of continuous ECT (Rami-González *et al.* 2001). Although any or all of these phenomena may be present in M-ECT, the absence of long-term memory dysfunction in our sample may be explained by the long interval between sessions during continuation ECT. Perhaps this long period between ECTs helps the recovery of the neurobiological bases involved in memory dysfunction. Interestingly, the M-ECT group obtained better scores than the matched group in long-term memory. Absence of an information retention dysfunction may improve the lifestyle of patients during M-ECT treatment and may aid their functional adaptation. It is well established that short-term memory is less affected than long-term memory after acute ECT. Within a few weeks of treatment, patients recover the ability to encode new information (Steif *et al.* 1986; Sackeim *et al.* 2000; Zervas *et al.* 1993). Taking evidence-based knowledge from previous ECT course studies as our starting point, we predicted that this memory subtype would not be disturbed as a consequence of M-ECT, since the mean intersession period was sufficient for short-term memory recovery. One possibility is that the encoding information dysfunction in our sample was influenced by the patients' inability to make associations in encoding, which may be influenced by frontal dysfunction (Shimamura *et al.* 1991).

Contrary to our expectations, the frontal functions of M-ECT patients were significantly impaired during treatment. These results must be considered in the context of the limitations of our study, which are common to M-ECT

research in general. The design of experimental studies in this area is difficult, since M-ECT is a specific continuation treatment for certain patients. All patients were in remission and there was no difference in their clinical symptomatology at the moment of cognitive assessment. The small sample size probably prevented the emergence of statistical differences between certain relevant clinical variables, such as number of episodes. The fact that the M-ECT group were seriously ill and drug-resistant may have influenced the cognitive differences between the groups. A frontal lobe dysfunction is clearly implicated in depressive patients. The higher number of episodes and the more serious depression in the M-ECT subjects may partly explain the differences between the groups in frontal functions. However, all patients were in remission when neuropsychological assessment was made. The cognitive profile of depressive patients in remission is not clear (Austin *et al.* 2001). Some studies have found that seriously depressed patients show global memory dysfunction during remission (Abas *et al.* 1990; Marcos *et al.* 1994), but some found no residual deficits (Calev *et al.* 1986; Bazin *et al.* 1994); others have even reported improved functions after recovery from depressive symptoms (Peselow *et al.* 1991). However, few studies have found altered frontal functions in addition to memory dysfunction (Beats *et al.* 1996; Paradiso *et al.* 1997) in depressive patients in remission.

One of the major difficulties in the design of ECT cognitive studies is the influence of many factors that contribute to an individual's cognitive impairment. These factors include the effect of age, the interference of symptomatology, and the use of concurrent medications. In our study, all patients had been in remission for the 3 months prior to cognitive assessment to ensure that the influence of mood disorder on cognitive functions would be minimal. Medication and age were also similar in the two groups. The effects of the small differences between groups in tricyclic antidepressants used would only have contributed to memory dysfunction in M-ECT patients (Parde, 1994; Healy, 2001) but do not seem to explain frontal function differences. Therefore, none of the controlled factors described above seem able to explain the frontal function discrepancy between groups.

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The high number of ECT sessions in these patients is another factor which could perhaps be related to the frontal dysfunction. Very little information is available on the effects of ECT on non-memory functions (Calev *et al.* 1995). Studies reported to date are based on the acute course and report no effects (Small *et al.* 1986; Lawson *et al.* 1990) or fewer effects than on memory (Taylor *et al.* 1985; Calev *et al.* 1991). Recently, the APA stated that there is no evidence that ECT results in lasting impairments of executive functions (APA, 2001).

Although retrospective studies based on acute courses of ECT indicate that there is no memory impairment at long-term follow-up (Cohen *et al.* 2000) or any alteration in brain structure associated with ECT (Devanand *et al.* 1994; Ende *et al.* 2000), further specific studies are necessary to rule out the effects of M-ECT on non-memory cognition, especially on frontal functions.

The advanced age of many patients in our sample deserves some comment. Although ECT can be used safely and effectively in geriatric subjects, these patients are more vulnerable to adverse cognitive effects (Stoudemire *et al.* 1993; Zervas *et al.* 1993; Tew *et al.* 1999; Brodaty *et al.* 2000) and may be more susceptible to developing frontal alterations associated with ECT. Pettinati & Bonner (1984) evaluated mental flexibility in depressed geriatric patients with a history of ECT. In the Trail Making B test, the over-65s with a history of ECT performed worse than older patients who had never received ECT or younger depressed patients regardless of their ECT history. Even though we did not find a significant correlation between cognitive measures and age, further studies with different ages samples and with other diagnoses are necessary to clarify ECT effects on non-memory functions in certain patients.

In summary, results of the present study indicate cognitive profile of our M-ECT sample was characterized by normal long-term memory while frontal functions and short-term memory were impaired. However, further studies are required to establish the cognitive state in patients during M-ECT, as this will help to determine their quality of life and everyday functioning during treatment.

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Univ. of Iowa Psy Hosp, Iowa City
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Does Treatment Influence Mortality in Depressives?

A Follow-up of 1076 Patients with Major Affective Disorders

Donald W. Black, M.D., M.S., George Winokur, M.D.,
 Emmanuel Mohandoss, M.S., Robert F. Woolson, Ph.D.,
 and Amelia Nasrallah, M.A.

ECT treatment were dead within 2 yr 12 per cent (3.2%)

This article reports mortality risk among 1076 Iowans with major affective disorders (705 primary unipolar, 219 secondary unipolar, and 152 bipolar depressives) compared to that of the general population. Patients were divided into four treatment groups depending on primary mode of therapy during the index admission; the groups included electroconvulsive therapy (ECT), adequate antidepressants, inadequate antidepressants, and neither treatment. All patients in the sample had an increased risk for an early death. A high risk for suicide was found for patients within each individual treatment group during the follow-up, especially the first 2 years when 69.4% ($n = 25$) of total suicides occurred. There were no significant differences in the risk for suicides, or deaths from all causes combined, among patients in the four treatment groups. Furthermore, mortality did not differ between patients having a lifetime history of ECT and patients never having had ECT. We conclude from a short-term follow-up of depressives that mode of therapy received in the hospital has minimal influence on subsequent mortality, including suicide.

KEY WORDS: Depression, electroconvulsive therapy, antidepressant medication, mortality, and suicide.

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Do modern psychiatric treatments help prevent premature deaths? Do modern psychiatric treatments help prevent suicide? These provocative questions have been asked repeatedly since effective treatments for the major psychiatric illnesses were developed beginning with electroconvulsive therapy (ECT). Few answers have been provided. Although several early studies on convulsive therapies (ECT or metrazol) were encouraging [1-3], other reports were not [4, 5]. More recently, two studies found lower death rates in depressives [6] and schizoaffectives [7] treated with ECT, but suicide rates were unchanged. Three additional studies since 1976 have not shown ECT to reduce suicide rates in depressives, either [8-10].

Confounding the effect of somatic treatment on death rates has been the independent trend in general mortality and suicide rates. In the past, both natural and unnatural causes of death were highly excessive [11, 12, 14], but now death from suicides and accidents is primarily responsible for the excess [13, 14]. Death from natural causes in psychiatric patients has been declining, however, most likely due to improvements in the availability and efficacy of general medical care, and deinstitutionalization, and may no longer be excessive [11, 13, 15]. Because natural causes of death may no longer be excessive, any protective effect that ECT may have had in the past in preventing these deaths may now be unimportant. Any effect that ECT might have on preventing suicide could still be critical, however. Also of concern is whether antidepressants, particularly tricyclics, might actually increase death rates, due either to their demonstrated effects on vasculature and cardiac conduction at both therapeutic and supratherapeutic dosages [16, 17] or to their le-

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(7.4% of the other 42-54% were dead within 2 yrs)

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thality in overdose. Certainly, patients with easy access to them may be at increased risk for overdoses. There are conflicting data as to whether antidepressants cause sudden death [18, 19], but no data indicating that antidepressants increase suicide rates. One study failed to find evidence of increased mortality associated with the use of psychotropic drugs, including antidepressant medication, but did not look specifically at suicide [20].

We undertook this study to thoroughly evaluate the possible influence of treatment on mortality. We wondered whether it is still possible to demonstrate that ECT-treated patients have lower mortality than patients receiving other treatments. We were able to address these questions through review of the case notes of 2054 patients admitted to our hospital who had an affective disorder. Because different affective disorders may be associated with different suicide rates, we chose to look at a subset of depressed patients demonstrated to have similar suicide rates [21] so that diagnosis would not confound the results. Further, we chose to focus on death rates during the first 2 years after hospital discharge as this portion of follow-up has been shown to be the period of greatest risk for premature death, especially suicide [22, 23].

METHODS

Between January 1, 1970, and December 31, 1981, 2054 patients with an affective disorder were admitted to the University of Iowa Psychiatric Hospital. Their charts were carefully reviewed by a master's level psychologist (A.N.) for demographic, diagnostic, treatment, and outcome data. Patients were re-diagnosed by applying DSM-III criteria to the case notes. Only patients from Iowa ($n = 1167$) satisfying DSM-III criteria for major depressive disorder or bipolar affective disorder, depressed type, were included in this study. To eliminate the possibility that physical illness could further confound the results, we excluded 91 patients who had concomitant organic mental disorders or serious medical illnesses that were potentially life-threatening. Serious medical illnesses would have included, for example, cancer or chronic obstructive lung disease, but not well-controlled hypertension or dysfunctional uterine bleeding. This left a final sample of 1076 subjects: 705 primary unipolar, 219 secondary unipolar, and 152 bipolar depressives.

The patients were subdivided into four mutually exclusive treatment groups depending upon primary mode of therapy during index hospitalization. (1) patients who received ECT during index hospitalization ($n = 372$); (2) patients who received an adequate trial of antidepressants ($n = 180$); (3) patients who received an inadequate trial of antidepressants ($n = 317$); and (4) of a group receiving neither ECT nor antidepressants ($n = 207$). Treatment assignment was left to the ward psychiatrists. Patients receiving ECT had, on average, 9.0 modified treatments using either sinusoidal wave or brief pulse stimulation. Typically, patients had no other therapy during the course of ECT but practice varied among clinicians. An adequate trial of antidepressants was defined as a minimum of 4 weeks of antidepressant therapy with at least 2 weeks of imipramine 150 mg/day or its equivalent. All patients receiving antidepressants at a lower dose or for less time were defined as having had an inadequate trial of antidepressants. The last treatment group included patients who received neither antidepressants nor ECT during the index hospitalization. These patients, however, may have received other treatments, including other somatic (e.g., anxiolytics, lithium carbonate) or psychosocial interventions (i.e., individual or family therapy). A minority of patients in all treatment groups received antipsychotic medication and, although this treatment was noted, the amount of antipsychotics given was not quantified. The ECT treatment group included only those who had ECT during the index hospitalization, although antidepressants may have been administered before or after ECT. Patients in the antidepressant treatment groups received antidepressants in the hospital and may have received them prior to hospitalization as well. They did not receive ECT, however. Further details of the study are available elsewhere [24].

We then matched the list of 1076 patients with Iowa death certificates issued between January 1, 1970, and December 31, 1983. Multiple identifiers (i.e., name, age, sex, date of birth, etc.) were used to facilitate the electronic matching process. We learned that 103 patients had died during the follow-up. Deaths were classified as either suicide (ICD-9 E950 to E969) or nonsuicide (all other death codes) [25].

Expected numbers of death were calculated using mortality tables adjusted for age, sex, and follow-up time among the patients. These tables were de-

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veloped using vital statistics and census data for Iowa. We adjusted for length of follow-up because study subjects were not all followed for the same amount of time. For example, a person followed 10 years would have a greater cumulative risk for mortality than someone followed 1 year. This method is more fully described elsewhere [22].

Expected and observed numbers of deaths were compared using the Freeman-Tukey-corrected chi square. The Freeman-Tukey correction was used because it is more conservative than the regular chi square and many of our expected numbers were so small. Standardized mortality ratios (SMRs) were calculated and represent the ratio of observed to expected mortality. An SMR greater than 1 means that observed death exceeds expectation. Ninety-five percent confidence limits were calculated for the SMRs using Byar's method [26].

RESULTS

Of 1076 patients, 372 (34.6%) received ECT, 180 (16.7%) received adequate antidepressants, 317 (29.5%) received inadequate antidepressants, and 207 (19.2%) received neither ECT nor antidepressants during the index hospitalization. Using a four-way chi square, there were significant differences among the groups on age, marital status, prior episodes, prior suicide attempts, precipitating factors, delusions, and recovery at discharge. There were no differences in sex or suicidal ideations. Patients receiving ECT were older than the others, were more likely to be married (probably because of their advanced age), tended to have more delusions, and were less likely to have attempted suicide. Patients within the two antidepressant groups were similar except that patients receiving adequate antidepressants were more likely to have had prior

episodes of illness. The group of patients receiving neither treatment differed from the other groups. These patients were younger, were less likely to be married, nearly two thirds had reported factors precipitating their depressions, nearly one-half had prior suicide attempts, and few were reported as receiving drug prophylaxis. At hospital discharge, patients receiving ECT were more likely to have recovered than patients in the other treatment groups.

Thirty-six suicides were identified in the record-linkage and comprise 3.3% of the study sample (Table 1). The following percentage of the total sample size for each diagnostic group committed suicide: ECT 3.2, adequate antidepressant 2.8, inadequate antidepressant 3.5, and neither treatment 3.9. There were no significant differences for the unadjusted (crude) suicide rates among the treatment groups ($\chi^2 = 0.944$, $df = 3$). Suicides as a percentage of the total deceased were ECT 23.5, adequate antidepressant 33.3, inadequate antidepressant 50.0, and neither treatment 53.3.

Table 2 shows the distribution of the 103 deaths by treatment group and portion of follow-up. Forty (38.8%) deaths occurred during the first 2 years of the follow-up. During this portion of the follow-up, general (all cause) mortality was significantly excessive compared with expectation for the groups receiving ECT and inadequate antidepressants. For the entire follow-up period, the mortality is excessive for the groups receiving ECT or neither treatment. There are no significant differences between SMRs among the four treatment groups, as demonstrated by overlapping confidence intervals.

Twenty-five (69.4%) suicides occurred during the first 2 years of follow-up (Table 3). This was particularly obvious in the inadequate antidepressant group; 10 of 11 suicides occurred during this phase of follow-up. SMRs in each treatment category are

Table 1. Suicide Among Deceased at Follow-up

Treatment Group	Observed Deaths			Observed Suicides		
	N	N	(%)	N	Percent of Total	Percent of Deceased
ECT	372	51	(13.7)	12	3.2	23.5
Adequate antidepressant	180	15	(8.3)	5	2.8	33.3
Inadequate antidepressant	317	22	(6.9)	11	3.5	50.0
Neither treatment	207	15	(7.2)	8	3.9	53.3
Total	1076	103	(9.6)	36	3.3	35.0

Table 2. Observed and Expected Number of Deaths During Follow-up

Treatment Group	N	Observed Deaths	Expected Deaths	Chi-square Test (<i>df</i> = 1)	<i>p</i> Value	Standardized Ratio	95% Confidence Limits
First 2 Years After Hospital Discharge							
ECT	372	18	8.22	7.73	<0.01	2.19	(1.30-3.46)
Adequate antidepressant	180	5	2.27	2.28	NS	2.20	(0.71-5.13)
Inadequate antidepressant	317	13	4.42	9.18	<0.005	2.94	(1.56-5.03)
Neither treatment	207	4	2.14	1.30	NS	1.87	(0.50-4.79)
Total Follow-up Interval							
ECT		51	33.81	7.18	<0.01	1.51	(1.12-1.99)
Adequate antidepressant		15	10.82	1.49	NS	1.39	(0.78-2.29)
Inadequate antidepressant		22	20.21	0.19	NS	1.09	(0.68-1.65)
Neither treatment		15	7.99	4.54	<0.05	1.88	(1.05-3.10)

significantly greater than expectation and during the follow-up. Although the SMRs for the inadequate antidepressant and the neither treatment groups were greater than the SMRs for those receiving ECT or adequate antidepressants, the 95% confidence intervals overlapped considerably.

Four-way comparisons of the treatment groups with respect to six important and potentially confounding variables (sex, age, marital status, duration of hospitalization, duration of illness, delusions) were performed using a statistical procedure called "exact test for 2×4 contingency table" developed by Freeman and Halton [27]. We used this analysis since the number of deaths (or suicides) was small. For the first 2 years after hospital discharge, none of the variables show an association with treatment groups either for suicide or for general mortality. Logistic regression and propor-

tional hazards models were also considered for these statistical analyses. However, we believe that the limited number of deaths would not support the use of such sophisticated models. Sample sizes were judged to be adequate for controlling one or two stratification variables. The number of suicides is particularly small, and even in the comparisons among treatments exact, rather than chi-square, tests were required for analysis.

DISCUSSION

The data demonstrate that affective disorders are associated with high risk for mortality. More than one third of all deaths reported in this study are suicides. Although the proportion of patients with major affective disorders who commit suicide is

Table 3. Observed and Expected Number of Suicides During Follow-up

Treatment Groups	N	Observed Deaths	Expected Deaths	Chi-square Test (<i>df</i> = 1)	<i>p</i> Value	Standardized Mortality Ratio	95% Confidence Limits
First 2 Years After Hospital Discharge							
ECT	372	8	0.10	21.53	<0.001	80.00	(34.45-157.64)
Adequate antidepressant	180	3	0.04	7.02	<0.01	75.00	(15.07-219.08)
Inadequate antidepressant	317	10	0.05	29.05	<0.001	200.00	(95.74-367.80)
Neither treatment	207	4	0.04	9.99	<0.005	100.00	(26.90-256.02)
Total Follow-up							
ECT		12	0.32	30.91	<0.001	37.50	(19.35-65.51)
Adequate antidepressant		5	0.14	11.49	<0.001	35.71	(11.51-83.31)
Inadequate antidepressant		11	0.23	29.05	<0.001	47.83	(23.84-85.57)
Neither treatment		8	0.16	20.70	<0.001	50.00	(21.53-98.53)

about 15% of suicides [28]. Future studies in

Literature

The purpose of this study was to compare the effectiveness of different treatments for major affective disorders. The study included 1,076 patients who were followed up for a period of 2 years. The results showed that ECT was significantly more effective than antidepressants in reducing the risk of suicide. The study also found that the combination of ECT and antidepressants was more effective than either treatment alone. The study was limited by the small number of suicides and the lack of a control group. The results suggest that ECT should be considered as a first-line treatment for major affective disorders with a history of suicide.

Table 4.

Follow-up
0-6 mo
≥6 mo
≥1 yr
≥18 mo
≥2 yr
Total
There is

about 15% in long-term follow-ups, the percentage of suicides is much higher in a short-term study [23, 28]. Further, the study shows no significant differences in death rates among four treatment groups.

Literature Survey

The purpose of this study was to determine whether specific treatment categories were associated with a differential risk for suicide, which had been suggested by early studies. Ziskind and colleagues followed 197 patients with affective psychoses, mostly manic-depression, for a mean of 40 months. Eighty-eight had received convulsive therapy (ECT or metrazol); 109 had refused convulsive therapy, had symptoms too mild to warrant the treatment, or had a contraindication to ECT. There were 13 deaths in the control patients, with 9 by suicide compared with 3 deaths with 1 suicide in the convulsive therapy patients. Huston and Locher [2] compared patients with involuntal psychosis treated with ECT with those receiving conservative therapies. None of the patients in the convulsive therapy groups committed suicide but 13% of those receiving conservative therapies did. Unfortunately, the follow-up periods differed for the different groups complicating subsequent data interpretation. In a later report on manic-depressive illness [3], these same authors found that ECT-treated patients followed for a mean of 36 months had a 1% suicide rate while the control patients followed for a mean of 82 months had a 7% suicide rate. Milstein and co-workers [10] recently reexamined these studies and noted that although they are suggestive of a beneficial effect, only the data from Ziskind and colleagues [1] yielded a statistically significant finding with a Fisher's exact probability of 0.029. The results of the current study are different from the results of

Ziskind and colleagues [1] and Huston and Locher [2], but are consistent with the conclusions of Eastwood and Peacocke [8], Babigian and Guttman [9], and Milstein and colleagues [10]. We found significant, but similar risk for suicide among patients in all treatment categories. Suicide as a percentage of the total number of patients who died differs between groups but this is easily explained on the basis of age. Patients in the ECT group were older than the patients in other groups and would be more likely to die from natural causes. SMRs in the different treatment groups (Table 3) might suggest that ECT and adequate antidepressants might carry a lower risk for suicide, but this could not be demonstrated statistically. However, expected values as small as those reported in Table 3 make any conclusions tentative. Clearly, one must be cautious in basing interpretations on such small numbers.

Since there was no difference in suicide rates during the first 2 years after hospital discharge, was it possible that a shorter follow-up might reveal a difference between groups? Perhaps the protective effect of a treatment is shorter lived. We looked at this possibility (Table 4), but found no discernible trend in the pattern of suicides. There was also no apparent association between treatment group and follow-up interval.

Avery and Winokur Data

The Avery and Winokur [6] report merits further discussion. This paper has been widely cited as demonstrating that adequate somatic treatment reduces death rates in depressives. In fact, these investigators found that among five treatment groups, patients receiving ECT had lower total mortality than patients receiving inadequate antidepressant therapy, or patients receiving neither ECT

Table 4. Distribution of Suicides in the Four Treatment Groups During Follow-up

Follow-up Interval ^a	ECT (n = 372)	Adequate Antidepressant (n = 180)	Inadequate Antidepressant (n = 317)	Neither Treatment (n = 207)
0-6 mo	6	1	5	3
≥6 mo <1 yr	0	1	3	0
≥1 yr <18 mo	0	0	2	0
≥18 mon <2 yr	2	1	0	1
≥2 yr	4	2	1	4
Total	12	5	11	8

^a There is no significant difference with the 2 × 4 exact test, when the follow-up interval is divided into a period of <2 yr and a period ≥2 yr, $p = 0.236$.

nor antidepressants, but not than patients receiving adequate antidepressants, or ECT in addition to antidepressants. Avery and Winokur also found that death from natural causes, particularly myocardial infarction, was significantly more frequent in a group of patients receiving inadequate antidepressants or neither ECT nor antidepressants, compared with an adequately treated group (i.e., patients receiving ECT or adequate antidepressants). No differences were found for suicide rates among the five treatment groups.

Several methodological problems complicate the interpretation of the Avery and Winokur report. First, the authors looked at mortality in a diagnostically heterogeneous group including manic-depressives, involutional melancholics, psychotic depressives, schizoaffectives, and neurotic depressives according to classifications in DSM-I or -II. Thus, the diagnostic composition of the five treatment groups differed substantially. For example, the ECT group was heavily weighted towards manic-depressives, involutional melancholics, and psychotic depressives (a group we would now consider as primary or endogenous depressives); and the group receiving neither treatment was heavily skewed towards neurotic depressives (a group we would now consider as secondary or nonendogenous depressives). Because mortality may be diagnosis-specific, combining such widely divergent groups may lead to improper comparisons. However, as we have previously reported, within the major affective disorders suicide rates may not be diagnosis specific [21].

Next, there was no direct control for the presence of medical illness. It is well known that many psychiatric patients suffer from serious medical illnesses [29, 30] and that the combination of a physical and mental illness leads to referral bias [11, 13]. Further, patients who are physically ill may be less likely to receive aggressive somatic treatment from clinicians worried about potential medical complications or drug interactions. As a result, patients

with medical illnesses who are predisposed to an early death by virtue of their physical condition would probably receive inadequate treatment or no somatic treatment. The result is to spuriously inflate mortality in those treatment groups. In our own sample, we excluded 91 persons with organic mental conditions or serious physical illnesses. We excluded patients with organic mental disorders because these conditions, along with physical illnesses, are associated with high risk of premature death from natural causes [31]. Had these 91 patients been included, 59 (64.8%) would have been assigned to our inadequate antidepressant group or to the group receiving neither treatment. Of the remaining 1076 patients, only 524 (48.7%) were assigned to these two treatment groups yielding a highly significant difference ($\chi^2 = 8.74$, $df = 1$, $p = 0.003$).

Long-term Effect of ECT

If there is no significant difference in mortality rates between treatment groups during a 2-year follow-up, might ECT exert a long-term protective effect? This is what the data of Tsuang and colleagues [7] suggested for schizoaffective illness. To test this possibility, we compared mortality in all patients who had received ECT at any time during their lives ($n = 566$) with those who had never received ECT ($n = 510$). The results are presented in Tables 5 and 6. There are no differences in the percentage of patients who committed suicide. There is a difference in proportion of deceased but this may be the result of the older age of the ECT patients, increasing the number of deaths from natural causes. There are also no significant differences in SMRs among treated and untreated patients from suicide. One implication drawn from these data is that ECT, although effective in terminating an episode of depression [24], has little effect on long-term consequences of the illness, including mortality. Therefore, the effect of ECT is probably short-lived.

Table 5. Suicide Among Deceased at Follow-up: Patients With a Lifetime History of ECT Versus Patients Never Receiving ECT

Treatment Group	Total Deceased			Suicides		
	N	N	Percent of Total	N	Percent of Total	Percent of Deceased
ECT	566	70	12.4	19	3.4	27.1
Non-ECT	510	33	6.5	17	3.3	51.5

Table 6. ECT Versus

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Table 6. Observed and Expected Number of Deaths and Suicides During the Total Follow-up: Patients With a Lifetime History of ECT Versus Patients Never Receiving ECT

Treatment Groups	N	Observed Deaths	Expected Deaths	Chi-square Test (df = 1)	p Value	Standardized Mortality Ratio	95% Confidence Limits
Total Mortality							
ECT	566	70	51.10	6.10	<0.025	1.37	(1.07-1.73)
Non-ECT	510	33	21.70	4.86	<0.05	1.52	(1.05-2.13)
Suicides							
ECT		19	0.47	50.84	<0.001	40.43	(24.33-63.11)
Non-ECT		17	0.38	45.83	<0.001	44.74	(26.04-71.63)

Concerns and Caveats

Because ECT and antidepressants are efficacious [32, 33], we might expect them to lower suicide rates, at least in a short-term follow-up. However, during a relatively short follow-up we found no statistically significant difference in general mortality or suicide rates among treatment groups, nor did a lifetime history of ECT appear to have any influence on mortality. One would like to believe that effective treatments would decrease the likelihood of an early death. Possibly, suicide rates would have been much higher had somatic treatments not been used at all. We cannot state categorically that ECT or antidepressants failed to prevent suicides; our methods may not be sensitive enough to measure the effect of adequate treatment on individual patients. As Murphy has observed, "a saved life is a statistical non-event" [34, p. 573]. It is reassuring, however, that death rates are not higher in the antidepressant groups because antidepressants are associated with cardiovascular disturbances, are reported to lead to sudden death, and can be lethal in overdose.

A potential criticism of the analysis is that the treatment groups are inherently unequal, so that it may be inappropriate to draw comparisons among them. As treatment selection was left to the clinician, group assignment was nonrandom and, as we noted earlier, the four groups differ substantially in many respects, including age, age of onset, psychosis, suicide attempts, and aftercare. We have attempted to correct for some of these inequalities. First, we restricted our sample to diagnostic groups shown to have similar suicide rates. We deleted from our sample medically ill persons. We also used SMRs to correct for age, sex, and duration of follow-up. Regardless, some may argue that even with

appropriate statistical safeguards, the groups remain unequal, that patients receiving ECT are simply "sicker" than patients receiving antidepressants, or no somatic treatment, and that such patients may have higher suicide rates. It is curious that the ECT group had fewer prior suicide attempts. Review of the literature, however, shows that suicide in depressed persons has not been clearly associated with clinical symptoms [35, 36], diagnostic subtype [21, 36], or psychosis [37, 38]. According to our results, suicide may also be unrelated to treatment.

We must emphasize that the results pertain to a highly select sample—depressives hospitalized at a tertiary care facility. The results should not be generalized to other groups, including outpatients. Indeed, most psychiatric patients are not hospitalized [39]. In particular, the results should not be generalized to never-treated outpatients. By virtue of entering the hospital, all our patients received "treatment," although some may not have had an active biologic therapy. Other treatments are provided in hospitals, including individual or group psychotherapy, milieu therapy, and activities and occupational therapies, whose effect on mortality cannot easily be assessed. Unfortunately, we also had no way to control for prophylactic treatments. This important variable may well affect mortality rates. Future studies will need to address this issue, as it is clear that drug prophylaxis affects relapse rates among the mood disorders.

In the past, lack of somatic treatment may have had dire consequences. A patient hospitalized in 1940 suffering a depressive stupor and not receiving a convulsive therapy (ECT or metrazol) may have developed fluid and electrolyte imbalance, stasis ulcers, or tendon contractures or may have aspirated and contracted pneumonia. These problems

may have been difficult to manage in an institutional setting. Furthermore, intravenous rehydration was uncommon and antibiotics were unavailable. Nowadays, if a psychiatric patient were to present with severe psychiatric symptoms such as stupor, and have no biologic therapy, it is less likely that the condition would be life threatening. Good nursing care, careful maintenance of fluid and electrolyte balance, and prompt treatment of intercurrent infections would be routine. In most cases, the patient would improve spontaneously. Therefore, active biologic treatments, such as ECT, may not be deemed as "lifesaving" now as in the past.

Another limiting factor in this study is the sample size, particularly the small number of suicides. Since there were only 25 suicides during the first 2 years of follow-up, the power to detect even modest differences between the four treatment groups is quite limited: hence the failure to claim a difference must be stated with some reservation.

Finally, it is important to realize that although ECT and antidepressants may not reduce death rates, their value is not diminished. They are effective treatments that can shorten, abort, or prevent depressions. As coronary artery bypass grafts (CABG) can reduce severe anginal pain and improve exercise tolerance [40], ECT or antidepressants can reduce the mental anguish and somatic discomfort associated with depression. And as CABG may not increase survival in the cardiac patient (except for certain groups of patients with three-vessel disease, and for patients with left main coronary artery disease) [41], ECT or antidepressants may not increase survival in patients with unipolar or bipolar depression. Nonetheless, these treatments may improve the quality of life.

5 CONCLUSIONS

We were unable to confirm earlier reports that treatment with ECT or adequate amounts of antidepressants are associated with lower mortality in depressed persons. In fact, neither general (all cause) mortality rates nor suicide rates varied significantly among treatment groups. Therefore, we conclude that, among hospitalized depressives, mode of therapy during the index hospitalization has little relationship to subsequent mortality. After 50 years of experience with ECT and 30 years with antidepressants, we still have inadequate informa-

tion regarding the impact of these treatments on death rates. Studies designed to address these issues would probably be impossible to implement. Ideally, such a study would involve a prospective follow-up of large numbers of patients, randomly assigned to different treatment groups, including placebo. The data analysis would need to take other treatments into account, including drug prophylaxis and psychosocial interventions. The legal, social, and ethical problems associated with such a project make it highly unlikely that it could ever be carried out. Nevertheless, studies of suicide and other causes of mortality will continue to be important because prevention of an untimely death is the ultimate goal of treatment.

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The first part of the paper discusses the importance of the study of the history of the United States. It is argued that a knowledge of the past is essential for a full understanding of the present. The author then goes on to discuss the various factors that have shaped the development of the United States, including the role of the government, the economy, and the culture.

The second part of the paper discusses the role of the government in the development of the United States. It is argued that the government has played a crucial role in shaping the country's history, from the founding of the nation to the present day. The author then goes on to discuss the various ways in which the government has influenced the development of the country, including through its policies, its actions, and its institutions.

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Continuation Pharmacotherapy in the Prevention of Relapse Following Electroconvulsive Therapy

A Randomized Controlled Trial

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ELECTROCONVULSIVE THERAPY (ECT) is usually administered to patients with severe and medication-resistant major depression.¹ The number of ECT procedures performed in the United States exceeds coronary bypass, appendectomy, or hernia repair.² While the response rate to ECT in major depression is high,^{1,3} relapse is a key problem.⁴ Naturalistic studies show that the relapse rate during the 6 to 12 months following ECT exceeds 50%.⁵⁻¹⁵

Electroconvulsive therapy is the only somatic treatment in psychiatry that is typically discontinued following response, yet patients untreated following ECT response have high rates of relapse.¹⁶⁻¹⁹ Studies in the 1960s suggested that continuation therapy with a tricyclic antidepressant (TCA) or monoamine oxidase inhibitor markedly reduced the 6-month post-ECT relapse

Context Electroconvulsive therapy (ECT) is highly effective for treatment of major depression, but naturalistic studies show a high rate of relapse after discontinuation of ECT.

Objective To determine the efficacy of continuation pharmacotherapy with nortriptyline hydrochloride or combination nortriptyline and lithium carbonate in preventing post-ECT relapse.

Design Randomized, double-blind, placebo-controlled trial conducted from 1993 to 1998, stratified by medication resistance or presence of psychotic depression in the index episode.

Setting Two university-based hospitals and 1 private psychiatric hospital.

Patients Of 290 patients with unipolar major depression recruited through clinical referral who completed an open ECT treatment phase, 159 patients met remitter criteria; 84 remitting patients were eligible and agreed to participate in the continuation study.

Interventions Patients were randomly assigned to receive continuation treatment for 24 weeks with placebo (n=29), nortriptyline (target steady-state level, 75-125 ng/mL) (n=27), or combination nortriptyline and lithium (target steady-state level, 0.5-0.9 mEq/L) (n=28).

Main Outcome Measure Relapse of major depressive episode, compared among the 3 continuation groups.

Results Nortriptyline-lithium combination therapy had a marked advantage in time to relapse, superior to both placebo and nortriptyline alone. Over the 24-week trial, the relapse rate for placebo was 84% (95% confidence interval [CI], 70%-99%); for nortriptyline, 60% (95% CI, 41%-79%); and for nortriptyline-lithium, 39% (95% CI, 19%-59%). All but 1 instance of relapse with nortriptyline-lithium occurred within 5 weeks of ECT termination, while relapse continued throughout treatment with placebo or nortriptyline alone. Medication-resistant patients, female patients, and those with more severe depressive symptoms following ECT had more rapid relapse.

Conclusions Our study indicates that without active treatment, virtually all remitted patients relapse within 6 months of stopping ECT. Monotherapy with nortriptyline has limited efficacy. The combination of nortriptyline and lithium is more effective, but the relapse rate is still high, particularly during the first month of continuation therapy.

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See also p 1346 and Patient Page.

rate.¹⁶⁻¹⁸ Post-ECT monotherapy with antidepressant medication is now standard.^{9,20-23} However, the evidence supporting this practice is flawed, and the

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recent naturalistic studies document high relapse rates.

Post-ECT continuation pharmacotherapy has been based on 3 studies conducted in the 1960s.¹⁶⁻¹⁸ A primary goal of those studies was to determine whether concurrent treatment with TCAs or monoamine oxidase inhibitors reduced the number of ECT treatments needed. Following ECT, patients continued taking active medication or placebo or no subsequent treatment. Using 6-month follow-up periods, the findings were consistent. Patients who received a TCA or monoamine oxidase inhibitor during and following ECT had a relapse rate of approximately 20%, compared with 50% in the control groups. There are major concerns about this research.^{4,24} At that time, ECT was a treatment of first choice.^{25,26} Relevance for continuation therapy in medication-resistant ECT responders is uncertain. Second, some patients likely benefited from the concurrent antidepressant during ECT, and continued to benefit from the medication as continuation therapy. Since use of ECT now centers on medication-resistant patients,^{1,21,27} the relevance of this early research is questionable.

We conducted a randomized, double-blind, placebo-controlled trial of continuation pharmacotherapy following ECT response. The treatments were a TCA (nortriptyline hydrochloride), combination treatment with nortriptyline and lithium carbonate, or placebo. A placebo-controlled trial following ECT had never been conducted in the United States. This trial was justified since the relapse rates in recent follow-up studies³⁻¹⁵ often exceeded those seen with placebo in the controlled investigations from an earlier era.¹⁶⁻¹⁸ A placebo-controlled trial was also justified by our hypothesis that TCA monotherapy, the best documented treatment in post-ECT relapse prevention,¹⁶⁻¹⁸ has limited efficacy. Monotherapy with nortriptyline was tested since (1) early research suggested that TCA continuation therapy was effective in relapse prevention¹⁶⁻¹⁸; (2) concern that newer agents, such as selective serotonin reuptake inhibitors

(SSRIs), may be less effective than TCAs in treatment of the severe episodes characteristic of ECT patients²⁸⁻³³; and (3) given the widespread use of SSRIs and other newer agents as first-line treatments, a low probability that ECT responders would have received an adequate TCA trial during the episode.³⁴ We hypothesized, however, that the nortriptyline-lithium combination would be most efficacious, given the evidence that combined TCA-lithium treatment is particularly effective in medication-resistant major depression,³⁵⁻⁴¹ and the supposition that regimens effective in the acute treatment of medication-resistant major depression exert protective effects as continuation treatment. Nortriptyline-lithium was also selected since few ECT remitters would have received this treatment during the episode.^{34,42}

METHODS

Study Site and Study Participation

The study was conducted at the Carrier Foundation (Belle Meade, NJ), a private psychiatric hospital, and at university-based psychiatric facilities of the University of Iowa (Iowa City) and Western Psychiatric Institute and Clinic (WPIC; Pittsburgh, Pa). The New York State Psychiatric Institute (NYSPI; New York) was the coordinating and monitoring center. Using the *Schedule for Affective Disorders and Schizophrenia*,⁴³ patients met the research diagnostic criteria⁴⁴ for major depressive disorder. They had a pretreatment score of 21 or higher on the Hamilton Rating Scale for Depression (HRSD; 24-item scale).⁴⁵ Patients were excluded if they had a history of bipolar disorder, schizophrenia, schizoaffective disorder, nonmood disorder psychosis, neurological illness, alcohol or drug abuse within the past year, ECT within the past 6 months, or severe medical illness that markedly increased the risks of ECT (eg, unstable or severe cardiovascular conditions, aneurysm or vascular malformation susceptible to rupture, severe chronic obstructive pulmonary disease).

Participants were recruited from those clinically referred for ECT. Over a 6-year period (1993-1998), 349 patients con-

sented and participated in the pre-ECT screening (FIGURE 1). Patients who met inclusion/exclusion criteria for the open ECT phase were completers if they received at least 5 treatments or ended ECT earlier due to response and did not receive any psychotropic medication during the ECT course other than lorazepam (≤ 3 mg/d). Of the 59 patients who did not contribute to ECT outcome data, 17 patients were dropped before ECT due to diagnostic exclusions; 14 patients could not be withdrawn from psychotropics before ($n=7$) or during ($n=7$) ECT; 12 patients terminated ECT against medical advice prior to the fifth treatment; 9 developed an intercurrent illness so ECT was not initiated ($n=2$) or was interrupted ($n=7$) (all before the fifth treatment); 6 patients withdrew consent before ECT; and 1 dropped below the inclusion threshold (HRSD score of 21) before starting ECT. Only 2 of 59 dropouts (prohibited medications) should have contributed to ECT efficacy analyses, but end point evaluations were not obtained.

To enter the continuation trial, patients had to achieve at least a 60% reduction in HRSD scores relative to pre-ECT baseline, with a maximum score of 10 both at an assessment within 2 days of ECT discontinuation and reassessment 4 to 8 days following ECT termination, while free of psychotropic medication. Since the extent of residual symptoms is predictive of relapse following antidepressant treatment,^{46,47} the remitter criteria were particularly stringent. These criteria required both a substantial symptomatic reduction and a low absolute score both immediately and 4 to 8 days following ECT. Patients with medical contraindications to nortriptyline or lithium were excluded. Patients provided separate informed consent for participation in the ECT and continuation pharmacotherapy phases, and capacity to consent was assessed at each time point. The institutional review boards at each enrollment site and the NYSPI approved the study. Assuming a relapse rate of 50% with placebo, the goal was to enroll at least 25 patients in each randomized treatment condition to have

at least an 80% probability of detecting a significant advantage in relapse time for an active treatment in a primary, intent-to-treat, parametric survival analysis.

Study Design

Patients were withdrawn from psychotropic medications, other than lorazepam (up to 3 mg/d) as needed, before starting ECT. Methohexital (0.75-1.0 mg/kg) and succinylcholine chloride (0.75-1.0 mg/kg) were the anesthetic medications, with preadministration of an anticholinergic agent (0.4-6 mg of atropine or 0.2-4 mg of glycopyrrolate). Based on clinical judgment, patients received either right unilateral or bilateral ECT, using the d'Elia⁴⁸ or bifrontotemporal²¹ placements, respectively. Electroconvulsive therapy was given 3 times per week with a customized MECTA SR1 device (MECTA Corp, Lake Oswego, Ore), which had double the maximal charge output of commercial devices in the United States. Seizure threshold was quantified at the first treatment using empirical titration.⁴⁹ For right unilateral ECT, dosage at subsequent treatments exceeded initial threshold by at least 150%. Patients who did not show substantial improvement to right unilateral ECT within 5 to 8 treatments were switched to bilateral ECT. To be considered adequate, minimal seizure duration was 20 seconds of motor or 25 seconds of electroencephalogram manifestation.²¹ Length of the ECT course was determined on clinical grounds.

The ECT remitters were randomized to 3 continuation pharmacotherapy groups, stratified by classification of the index episode as psychotic depression; medication-resistant nonpsychotic depression; and nonpsychotic depression without medication resistance. Medication resistance was rated using the Antidepressant Treatment History Form.^{43,50} Medication-resistant nonpsychotic patients had to have received at least 1 adequate antidepressant trial prior to ECT. Patients with psychotic depression were not further stratified by resistance classification since only 4 (4.3%) of 92 such patients received an adequate

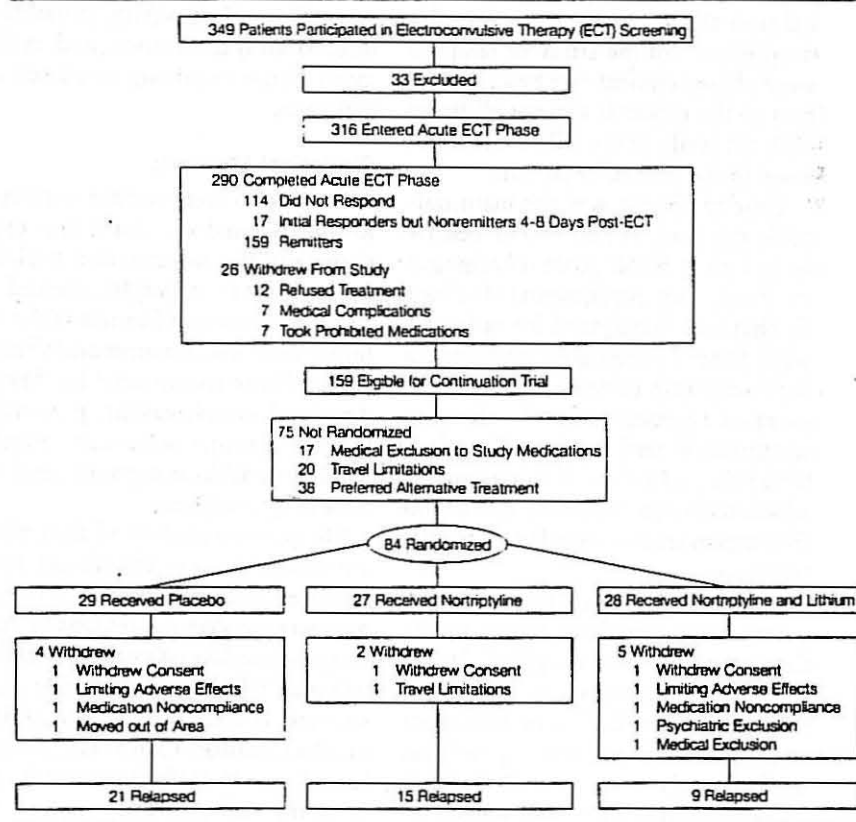
combination antidepressant-antipsychotic trial during the episode.⁴²

Using a randomly permuted block procedure consisting of blocks of 6 patients (within site and the 3 strata), each treatment condition was equally represented. The study psychiatrist who completed the Antidepressant Treatment History Form communicated the patient classification to the pharmacist who assigned the next available patient number within the stratum. Only the site pharmacist, the study coordinator at NYSPI, and the NYSPI laboratory conducting plasma level assays had access to the randomization code. The randomization code was generated by the study coordinator at NYSPI based on the randomization tables provided by Fleiss.⁵¹ Treatment teams, outcome assessors, and data analysts were blind to treatment assignment.

Medication was administered in sealed capsules containing 25 mg of nortriptyline, 300 mg of lithium, or mi-

crocrystalline cellulose (placebo). The capsules containing nortriptyline or lithium were distinct in appearance, and each was matched with placebo capsules identical in size, weight, appearance, and taste. Each patient was given 2 sets of pills. On the first study day, 50 mg of nortriptyline or its placebo and 600 mg of lithium or its placebo were administered. Blood samples were obtained 24 hours later and estimates were determined for the oral dose needed to produce steady-state levels of 100 ng/mL of nortriptyline and 0.7 mEq/L of lithium.⁵²⁻⁵⁴ On days 3 and 4, depending on the estimate, oral doses were adjusted and maintained until plasma levels were again taken on days 9 through 11. The goal was to maintain nortriptyline levels between 75 and 125 ng/mL and lithium levels between 0.5 and 0.9 mEq/L. During the 24-week trial, plasma levels were determined on 10 occasions. A yoked-control procedure was used, with a

Figure 1. Participant Flow



psychiatrist at NYSPI reporting simulated nortriptyline and lithium values for patients receiving placebo, based on matching by sex, age, and weight with patients who were receiving active medication.

Patients were evaluated at weekly intervals for the first 4 weeks, at 2-week intervals for the next 8 weeks, and at 4-week intervals for the remaining 12 weeks. They were contacted by telephone at weekly intervals between visits. Clinical ratings during the continuation phase were obtained by the same blinded evaluator (continuous rater) who evaluated patients throughout the ECT course. During the continuation trial, a blinded study psychiatrist assessed adverse effects and vital signs, adjusted medication or placebo dosage (based on plasma levels reported by NYSPI and adverse effects), and completed clinical ratings. To evaluate the adequacy of the blinding, patients guessed their treatment assignment as placebo, nortriptyline, or nortriptyline-lithium at study exit. Patients who dropped out of the study or relapsed were offered clinical care by a psychiatrist at the research site not affiliated with the study or the follow-up evaluation of the particular patient.

Time to relapse was the main outcome measure. The criteria for relapse were a mean HRSD score (continuous rater and study psychiatrist) of at least 16 that was maintained for at least 1 week (over 2 consecutive visits) and a mean absolute increase of at least 10 points at 2 consecutive visits relative to continuation trial baseline. These criteria reflected a clinical worsening for which most clinicians would abandon the current treatment in favor of an alternative.

At the pre-ECT evaluation, a research nurse completed ratings on the Cumulative Illness Rating Scale³⁵ to assess medical comorbidity. At all major time points (pre-ECT, post-ECT, start of continuation trial [day 0], week 12, week 24, and relapse), the HRSD, Clinical Global Impression,³⁶ and Global Assessment Scale⁴³ scores were completed by the continuous rater and the

study psychiatrist. At each site, intraclass correlation coefficients for the 2 raters exceeded 0.97, 0.93, and 0.90 for HRSD, Clinical Global Impression, and Global Assessment Scale scores, respectively. A site-independent, time-blind clinician at NYSPI rated 239 videotapes of continuous rater interviews conducted at random intervals during the ECT and continuation phases. The intraclass correlation coefficients were 0.97, 0.96, and 0.95 for HRSD, Clinical Global Impression, and Global Assessment Scale scores, respectively. The HRSD, Clinical Global Impression, and Global Assessment Scale scores reported below are the continuous rater evaluations.

At each visit in the continuation phase, a blinded study psychiatrist completed the Treatment Emergent Symptom Scale.³⁶ Forty-eight possible adverse effects were rated for severity, relationship to study medication, and action taken. Clinically significant adverse effects were defined as those rated as moderate in severity, possibly related to study medication, and, at minimum, those requiring increased surveillance.

Statistical Methods

Patients who met remitter criteria following ECT and who did or did not participate in the continuation trial were compared in demographic, clinical, and previous treatment features with *t* tests for continuous measures and χ^2 analyses for dichotomous variables. The randomized continuation pharmacotherapy groups were compared on baseline variables using analyses of variance or χ^2 analyses.

The primary analysis of the continuation trial used survival analysis for right-censored failure-time data. A simultaneous regression model was fit to the relapse-time data using the Weibull distribution.^{10,15} Covariates in the regression model were the randomized treatment condition (3 levels), strata (3 levels), sex, and HRSD score at the start of the trial. In a secondary analysis, ECT treatment modality (right unilateral only vs right unilateral and bilateral ECT vs

bilateral ECT only) and number of ECT treatments were added as additional covariates. To confirm the findings from the parametric analysis regarding treatment group differences, nonparametric estimates of the survival distribution function for each group were computed, using the Kaplan-Meier method³⁷ and contrasted with the log-rank test (Mantel-Cox).³⁸

Early in the study, 1 site (Carrier Foundation) was closed when the hospital discontinued its research division, so another site (University of Iowa) was added late. These 2 sites entered 21 patients in the continuation trial compared with 63 patients at WPIC. To determine whether the effects were not unique to WPIC, the Carrier Foundation and the University of Iowa were pooled for analysis. A site term (WPIC vs Carrier Foundation and University of Iowa) was entered into both secondary parametric and nonparametric survival analyses.

To assess the adequacy of pharmacotherapy, separate analyses of variances were conducted on the last plasma levels for nortriptyline and lithium obtained in completers (24-week or time of relapse), using the assayed values for active medication and the simulated values for placebo, and treatment group (3 levels) and relapse status as between-subject factors. A logistic regression was conducted on the patients' guess of treatment condition with relapse status and actual treatment assignment as predictors.

RESULTS

Of the 290 patients who completed the ECT phase, 159 (54.8%) patients were remitters (TABLE 1 and Figure 1). There was no difference among the sites in remitter rate ($\chi^2_1=3.75$, $P=.15$). Immediately following ECT, 17 patients (5.9%) met initial remitter criteria, but not at the 4- to 8-day reassessment. The remitter rate may have been negatively influenced by the stringency of the remission criteria and the fact that 262 patients (90.3%) started with right unilateral ECT, with the minimum dosage only 150% above seizure thresh-

old. Subsequent research has shown that the efficacy of right unilateral ECT improves at a higher dosage relative to seizure threshold.^{15,30}

Of the 159 remitters, 84 (52.8%) patients entered the randomized continuation trial. Of the 75 remitters who did not participate, 22.7% had medical exclusions for nortriptyline or lithium; 26.7% had travel limitations; and 50.7% preferred treatment by their referring physician, were receiving other medications or ECT, or were unwilling to receive placebo.

Comparisons of remitters who did or did not enter the continuation trial yielded no differences in pre- or post-ECT HRSD, Clinical Global Impression, or Global Assessment Scale scores, number of episodes, duration of current episode, number of ECT treatments, strength of the most potent antidepressant trial during the index episode, sum or average potency of all trials, number of trials, or number of adequate trials. The groups also did not differ in sex, race, history of previous ECT, use of right unilateral or bilateral ECT, or classification of medication resistance. Trial participants were younger (mean [SD], 57.4 [17.2] years) than nonparticipants (64.2 [16.3] years) ($t_{157}=2.54$; $P=.01$); had more previous psychiatric hospitalizations (2.4 [2.6] than nonparticipants (1.5 [1.6]) ($t_{157}=2.82$; $P=.005$); a higher rate of psychotic depression (41.7% vs 16.0%) ($\chi^2_1=12.54$, $P<.001$); and less total medical burden (Cumulative Illness Rating Scale score, 6.1 [4.2] vs 8.0 [3.9]) ($t_{157}=2.91$; $P=.004$). The medical exclusions for the continuation trial and travel limitations likely accounted for the higher age and greater medical burden of nonparticipants.

The continuation treatment groups were compared in demographic and clinical features (TABLE 2). There were no significant differences.

Eleven (13.1%) of the 84 patients dropped out of the trial before completing 24 weeks or meeting relapse criteria. The reasons for noncompletion are described in Figure 1. Dropout rates were evenly distributed among the 3

treatment groups (4 placebo, 2 nortriptyline, and 5 nortriptyline-lithium).

The overall model in the parametric analysis on survival time was significant (likelihood ratio, $\chi^2_6=27.3$; $P<.001$) (TABLE 3). The treatment groups differed markedly ($P<.001$). Both nortriptyline alone ($P=.01$) and nortriptyline-lithium ($P<.001$) were su-

prior to placebo in survival time, and nortriptyline-lithium was superior to nortriptyline alone ($P=.04$).

The Kaplan-Meier survival function was computed for each treatment group (FIGURE 2). Across the sample, 45 (61.6%) of 73 completers relapsed. This confirmatory nonparametric analysis yielded a log-rank χ^2_2 of 9.12 ($P=.01$).

Table 1. Number of Patients at Each Site Who Completed Electroconvulsive Therapy (ECT), Remitted With ECT, and Entered and Completed the Continuation Trial

Site	ECT Completer	ECT Remitter	Entered Continuation Trial	Continuation Trial	
				Dropout	Relapse
Carver Foundation	66	43	16	3	8
University of Iowa	22	12	5	0	2
Western Psychiatric Clinic and Institute	202	104	63	8	35
Total	290	159	84	11	45

Table 2. Patient Characteristics for Continuation Treatment Groups*

Variable	Placebo Only (n = 29)	Nortriptyline and Placebo (n = 27)	Nortriptyline and Lithium Carbonate (n = 28)
Age, mean (SD), y	55.8 (13.6)	57.2 (19.8)	59.2 (18.3)
Women, %	69.0	70.4	60.7
Pre-ECT Hamilton Rating Scale for Depression, mean (SD)	34.9 (8.4)	36.1 (8.2)	34.9 (8.5)
Psychotic, %	44.8	37.0	42.9
Medication resistant, %†	48.3	44.4	50.0
Selective serotonin reuptake inhibitor	31.0	33.3	37.0
Tricyclic antidepressant	17.2	11.1	18.5
Monoamine oxidase inhibitor	10.3	0	0
Other antidepressant	3.4	14.8	11.1
Tricyclic antidepressant lithium carbonate	6.9	0	0
Cumulative Illness Rating Scale, mean (SD) total	4.8 (3.4)	7.3 (4.4)	6.3 (4.6)
Episode duration, median, wk	31.0	24.0	25.0
No. (%) of previous episodes‡	2.3 (2.6)	2.4 (2.0)	2.8 (2.2)
History of previous ECT, %	41.4	48.1	46.4
Age at onset, mean (SD), y	40.7 (18.1)	38.1 (17.3)	38.0 (17.4)
Total ECT treatments, mean (SD)	10.2 (2.9)	10.8 (5.2)	10.7 (3.5)
Total right unilateral ECT treatments, mean (SD)	7.7 (3.0)	7.4 (3.4)	6.6 (2.9)
Hamilton Rating Scale for Depression, mean (SD)§	5.0 (2.7)	5.6 (3.1)	6.0 (3.1)
Clinical Global Impression severity, mean (SD)§	1.6 (0.6)	1.7 (0.6)	1.8 (0.6)
Global Assessment Scale, mean (SD)§	74.8 (6.1)	73.2 (7.1)	73.2 (6.5)

*ECT indicates electroconvulsive therapy.

†Adequacy of each medication trial given during the index episode before ECT was evaluated with the Antidepressant Treatment History Form. Each trial was rated on a scale ranging from 0 to 5, with a score of 3 the threshold for classification as medication resistant. To be considered an adequate trial, the threshold for sufficient dosage corresponded to a minimum of 200 mg/d imipramine equivalents for tricyclic antidepressants and 20 mg/d for fluoxetine. The threshold for sufficient duration was a minimum of 4 weeks at or above the threshold for sufficient dosage. To be classified as resistant, patients with psychotic depression had to receive an adequate antidepressant trial and at least 3 weeks of concurrent treatment with an antipsychotic medication, with a dosage of at least 400 mg/d chlorpromazine equivalents.

‡An upper limit of 10 episodes was used.

§Measured at day zero, which was the start of the continuation trial.

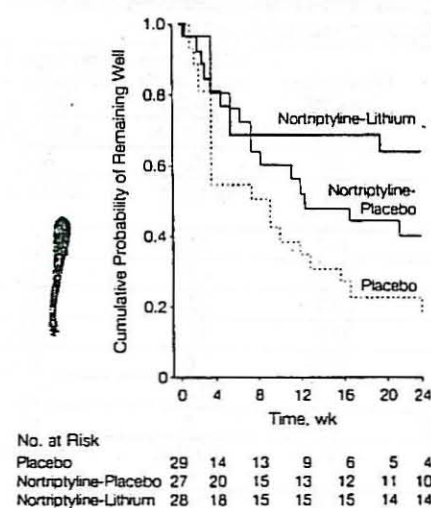
Table 3. Parametric Survival Analysis on Time to Relapse*

	Coefficient (SE)	df	χ^2	P Value
Treatment group				
Nortriptyline	0.96 (0.39)	1	6.18	.01
Nortriptyline and lithium carbonate	1.75 (0.47)	1	13.67	<.001
Total		2	14.74	<.001
Medication resistance/psychosis				
Psychotic depression	0.79 (0.37)	1	4.57	.03
Nonpsychotic nonresistant	0.34 (0.56)	1	0.37	.54
Total		2	4.59	.10
Sex†	-0.98 (0.43)	1	5.23	.02
Hamilton Rating Scale for Depression‡	-0.15 (0.06)	1	5.40	.02

*Within the treatment groups, the statistical comparisons for the nortriptyline alone and combined nortriptyline-lithium treatment conditions are against the placebo condition. Within the subgroup strata, the tests for psychotic depression and nonpsychotic depression, nonresistant subgroups are against the nonpsychotic, medication-resistant subgroup.

†Values are 1 for men and 2 for women.

‡Measured at day zero.

Figure 2. Kaplan-Meier Estimates

Proportion of patients who remained well during the continuation trial, for patients randomized to treatment with placebo (n=29), nortriptyline alone (n=27), and combination nortriptyline and lithium carbonate (n=28).

The relapse rates for completers were 84.0% (21/25) for placebo (95% confidence interval [CI], 70%-99%); 60.0% (15/25) for nortriptyline (95% CI, 41%-79%); and 39.1% (9/23) for nortriptyline-lithium (95% CI, 19%-59%). Only 1 patient relapsed while taking nortriptyline-lithium after 5 weeks, while relapse steadily continued with placebo and nortriptyline throughout the 24-week trial (Figure 2). Nonparametric survival analyses comparing each active treatment condition with placebo yielded a

significant effect for nortriptyline-lithium ($\chi^2=8.52$; $P=.004$), but only a trend for nortriptyline ($\chi^2=3.33$; $P=.07$).

The parametric survival analysis indicated that across the treatment conditions, medication-resistant nonpsychotic patients had a higher relapse rate than patients with psychotic depression. The relapse rates were 50.0% for psychotic patients (n=28), 55.6% for nonpsychotic patients without medication resistance (n=9), and 72.2% for nonpsychotic medication-resistant patients (n=36). The significant effect of sex was due to a higher relapse rate among women (77.8%) than men (53.6%). Patients who relapsed had higher mean (SD) HRSD scores at trial entry (6.0 [3.1]) than patients who did not relapse (5.0 [2.8]). There were no additional significant effects in the parametric survival analysis when treatment with right unilateral, right unilateral and bilateral, or bilateral ECT ($P=.89$), and number of ECT treatments ($P=.96$) were entered as additional terms.

Study site (WPIC vs combined Carrier Foundation and University of Iowa) was entered as a term in both the parametric and nonparametric survival analyses. There were no site effects. The relapse rates at WPIC for placebo, nortriptyline, and nortriptyline-lithium were 88.9%, 60.0%, and 41.2%, respectively, and for the combined Carrier Foundation and University of Iowa they were 71.4%, 60.0%, and 33.3%, respectively.

The high rate of relapse across the treatments could have been due to excessively sensitive relapse criteria. Clinical ratings at continuation trial entry and end point were compared as a function of relapse status (TABLE 4). Relapsed patients showed marked symptomatic worsening. Fifteen (33%) of the 45 relapsed patients were hospitalized and received ECT, 6 patients (13%) received outpatient ECT, and all other relapsed patients (53%) were switched to other pharmacotherapies. The severity of relapse did not differ among the continuation treatments.

No effects approached significance in the analyses of variances of nortriptyline and lithium levels on final visit. At final visit, the mean (SD) nortriptyline level was 89.9 (38.2) ng/mL for the nortriptyline group, 89.2 (32.2) ng/mL for the nortriptyline-lithium group, and the simulated levels reported for the placebo group averaged 93.0 (27.5) ng/mL. For lithium, the levels were 0.59 (0.2) mEq/L for the nortriptyline-lithium group, with simulated levels of 0.54 (0.2) mEq/L and 0.62 (0.2) mEq/L for the nortriptyline and placebo groups, respectively. Relapse was not associated with nortriptyline or lithium plasma levels.

A 1-way analysis of variance indicated that the treatment groups did not differ in the average number of clinically significant adverse effects ($F_{2,40}=0.13$; $P=.88$). For the placebo, nortriptyline, and nortriptyline-lithium groups, the mean (SD) number of significant adverse effects per patient was 1.24 (1.8), 1.42 (1.7), and 1.21 (1.3), respectively. An analysis of variance in the completer sample (with treatment group and relapse status as between-subject factors) yielded no significant effects. The mean (SD) number of significant adverse effects among patients who relapsed (1.48 [1.7]) did not differ from nonrelapsed patients (1.32 [1.6]) ($t_{70}=0.39$; $P=.70$). TABLE 5 presents the clinically significant adverse effects experienced by at least 3 patients.

At study exit, 63 of the 73 completers guessed their treatment assignment. The logistic regression analysis

yielded a modest association between the treatment assignment and the patients' guesses ($\chi^2_4=9.68$; $P=.05$) and a more robust association with relapse status ($\chi^2_2=8.17$; $P=.02$). Only 1 (4%) of the 25 patients who did not relapse believed he/she was treated with placebo, while this was true of 16 (42.1%) of the 38 patients who did relapse. Of the patients treated with placebo, 50% believed they received only placebo, while 31.8% and 18.2% believed that they had received nortriptyline and nortriptyline-lithium, respectively. For the nortriptyline group, the guesses were 29.4% for placebo, 23.8% for nortriptyline, and 52.4% for nortriptyline-lithium. For nortriptyline-lithium, these guesses were 5.0%, 30.0%, and 65.0%, respectively. While the patient blinding was imperfect, relapse status was a more powerful determinant of the guesses. The distributions overlapped considerably among patients treated with nortriptyline and nortriptyline-lithium.

COMMENT

Early research, based on first-choice use of ECT for major depression, indicated that half of the patients remain well in the 6 months following response without continuation therapy.¹⁰⁻¹⁸ We found that the relapse rate for placebo-treated patients was 84%. This suggests that the prognosis following ECT is more guarded today. Given the shift in use of ECT for severe, recurrent, and medication-resistant patients with higher risk of relapse,^{11,15,60} almost universal relapse should be expected without effective continuation therapy.

The early research suggested that continuation monotherapy with a TCA reduced the relapse rate to approximately 20%.¹⁰⁻¹⁸ We found that the relapse rate with nortriptyline continuation monotherapy was 60%, above the original projections for placebo. While TCAs are believed to be among the most effective antidepressant agents,^{27,30,33} our findings indicate that the efficacy of post-ECT TCA continuation monotherapy is not acceptable. Similarly, in a naturalistic study, Flint and Rifat¹

Table 4. Clinical Ratings as a Function of Relapse Status

	Mean (SD) Nonrelapse (n = 28)		Mean (SD) Relapse (n = 45)	
	Baseline	End Point	Baseline	End Point
Hamilton Rating Scale for Depression	5.0 (2.8)	4.8 (3.8)	6.0 (3.1)	24.1 (6.8)
Clinical Global Impression	1.6 (0.5)	1.4 (0.7)	1.7 (0.6)	4.1 (0.6)
Global Assessment Scale	74.5 (6.3)	77.9 (8.7)	73.3 (6.4)	49.3 (9.2)

Table 5. Clinically Significant Adverse Effects for the Continuation Treatment Groups*

Adverse Effect	No. (%)			
	Total Sample (N = 84)	Placebo Only (n = 29)	Nortriptyline and Placebo (n = 27)	Nortriptyline and Lithium Carbonate (n = 28)
Drowsiness/tiredness	19 (22.6)	5 (17.2)	8 (29.6)	6 (21.4)
Insomnia	10 (11.9)	5 (17.2)	3 (11.1)	2 (7.1)
Constipation	7 (8.3)	0 (0)	5 (18.5)	2 (7.1)
Anorexia/decreased appetite	6 (7.1)	4 (13.8)	0 (0)	2 (7.1)
Depressive affect	6 (7.1)	2 (6.9)	3 (11.1)	1 (3.6)
Syncope/dizziness	6 (7.1)	2 (6.9)	3 (11.1)	1 (3.6)
Dry mouth	5 (6.0)	2 (6.9)	3 (11.1)	0 (0)
Nausea/vomiting	5 (6.0)	2 (6.9)	1 (3.7)	2 (7.1)
Hypotension	4 (4.8)	1 (3.4)	0 (0)	3 (10.7)
Dermatologic	3 (3.6)	1 (3.4)	1 (3.7)	1 (3.6)
Diarrhea	3 (3.6)	1 (3.4)	1 (3.7)	1 (3.6)
Headache	3 (3.6)	1 (3.4)	1 (3.7)	1 (3.6)
Nasal congestion	3 (3.6)	0 (0)	3 (11.1)	0 (0)
Peripheral edema	3 (3.6)	0 (0)	0 (0)	3 (10.7)

*Clinically significant adverse effects were defined as those rated by the study psychiatrist on the Treatment Emergent Symptoms Scale as at least moderate in severity, at least possibly related to study medications, and as requiring at minimum greater surveillance or a dosage change. Data are presented for the clinically significant adverse effects experienced by at least 3 of the 84 patients.

found that continuation monotherapy with a TCA was ineffective in preventing relapse in psychotically depressed patients who responded to ECT.

The relapse rate for the combination of nortriptyline-lithium was 39.1%, which was superior to placebo and nortriptyline monotherapy. Similar results were reported in a naturalistic study at NYSPI, in which relapse rates over 1 year were markedly lower among ECT remitters who received TCA-lithium continuation therapy (35.3%) compared with patients who received continuation treatment with other pharmacological regimens (67.9%).¹⁵ It was noteworthy that the lithium levels in the present study were at the low end of what is considered the therapeutic range for acute or maintenance treatment (0.5-1.2 mEq/L).^{62,63} This suggests that in combination with nortriptyline,

lithium levels may only need to be greater than 0.5 mEq/L to prevent post-ECT relapse.

This study could not determine whether the advantage of the TCA-lithium combination was due to lithium alone or the synergism of lithium with the TCA. The only placebo-controlled trial of lithium following ECT in unipolar patients found that lithium did not have protective effects during the first 6 months following ECT.^{64,65} Thus, it is likely that the advantage of nortriptyline-lithium was due to additive or synergistic effects and not lithium alone. Our findings encourage the use of nortriptyline-lithium as post-ECT continuation therapy. It is unknown whether similar protective effects would be obtained with a mood stabilizer other than lithium or antidepressants other than nortriptyline (in combination with

lithium). This issue is important since SSRIs and other newer antidepressant agents have better tolerability than TCAs and are now more commonly used.

Patients with higher HRSD scores at the start of the continuation trial had shorter survival time. This is consistent with several studies of relapse during continuation pharmacotherapy following response to antidepressant medications^{46,47} or ECT.⁸ Thus, concerted attempts should be made to maximize symptomatic improvement in patients receiving ECT. Women were more prone to relapse during the continuation phase. There is inconsistent evidence from naturalistic studies of a higher relapse/recurrence rate among women.^{14,66-70} Studies of patients with psychotic depression suggested a high post-ECT relapse rate.^{6,7} However, regardless of the treatment producing remission, no previous controlled study has compared relapse rates in psychotic and nonpsychotic depressed patients. We found that psychotically depressed patients had a lower relapse rate than medication-resistant nonpsychotic patients. Several studies have shown that medication resistance is especially predictive of post-ECT relapse.^{8,15,60} It is also possible that compared with medication-resistant nonpsychotic patients, patients with psychotic depression had less Axis II (personality disorder) pathology and better interepisode function. There is evidence that the post-ECT course is poorer in patients with significant Axis II pathology.^{71,72}

The major finding was that treatment with the nortriptyline-lithium combination produced a substantially lower relapse rate than treatment with placebo or nortriptyline alone. Nonetheless, the relapse with nortriptyline-lithium was high (39.1%). Two alternative strategies, which are not mutually exclusive, should be tested.⁴ Both strategies are suggested by the observations that relapse is heavily skewed to the period immediately following ECT. During the acute treatment phase, there is a several week delay before antidepressant and mood stabilizing agents exert

therapeutic effects.⁷³ Further, the abrupt discontinuation of effective somatic treatment is associated with potentiation of relapse,⁷⁴⁻⁷⁶ which is standard in terminating an ECT course. One strategy is to taper ECT over a few weeks, as is commonly done with pharmacological treatments, providing symptom suppression during the most vulnerable period. Second, the antidepressant medication used in continuation therapy may be started during the course of ECT, followed by post-ECT addition of lithium. All controlled studies in which ECT was combined with an antidepressant medication focused on whether response to ECT was improved,¹⁶⁻¹⁹ and not whether this strategy reduced post-ECT relapse. Nonetheless, a low post-ECT relapse rate was seen in studies in which patients began taking an antidepressant at the start of the ECT course.¹⁶⁻¹⁹ Thus, these 2 adjunctive strategies raise the possibility that the advantage seen with the nortriptyline-lithium therapy may be further improved and that the problem of the high rate of early relapse with continuation pharmacotherapy following ECT could be resolved.

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Morbidity in electroconvulsive therapy

E. Tecoult & N. Nathan

Background and objective To assess retrospectively the complications and morbidity of electroconvulsive therapy.

Methods Complications occurring in 75 patients during 612 electroconvulsive therapy procedures under propofol anaesthesia were reviewed by data analysis.

Results At least one complication occurred in 51 patients (68%) during the course of their treatment. Among these complications, 12 were potentially life-threatening: one patient developed angina pectoris, another aspiration pneumopathy, there were two incidences of bronchospasm, three hypoxic episodes ($SpO_2 < 92\%$ with $FiO_2 = 1$) and five severe episodes of laryngospasm which caused hypoxia. Twenty-five patients (33%) were confused for more than 2 h after the electroconvulsive therapy. Confusion recurred in 10 patients (13%) after several sessions of electroconvulsive treatment. Six patients had a traumatic complication, with one requiring surgery.

Conclusion Our results, compared with other studies, suggest that electroconvulsive therapy is not a low-risk procedure, with a particularly high rate of respiratory complications that may have been previously overlooked. Therefore, ambulatory anaesthesia may not be appropriate on a regular basis for most of these patients.

Introduction Go to:

Although general anaesthesia has been used since 1963 to provide humane conditions for electroconvulsive therapy (ECT) [1], there are few studies about its complications and their implications for the anaesthesiologist [2-6]. Moreover, some authors have suggested that anaesthesia for ECT is associated with lower standards of care than for other non-surgical anaesthetic procedures [7,8]. Anaesthesia for ECT is associated with specific problems such as repeated exposures and attendant risks. It is performed outside the operating room and pharmacological interactions between anaesthetic drugs, psychiatric medications and convulsions are numerous [1]. Finally, the patients are often elderly and unable to relate their medical problems coherently.

Over the past few years, some authors have suggested that ECT had a lower morbidity rate than antidepressant drugs [4,9] and have suggested that this practice is unsuitable on an outpatient basis [10]. It is estimated that 1% of the general population is suffering from bipolar disorders [11], one of the most common pathologies treated with ECT. It has also been suggested that ECT can be a useful treatment for some forms of neurological disease, e.g. epilepsy and Parkinsons disease [12]. All this implies that potentially a considerable number of patients might receive ECT. Thus, it is useful to re-evaluate its safety. However, surprisingly few studies have tried to quantify its risks. Most studies have focussed on a class of patients with a specific risk factor such as age or cardiopathy. The aim of the present study was to examine the rate and type of complications observed during and after ECT in an unselected group of psychiatric patients, in order to assess the feasibility of ECT on an outpatient basis.

Methods Go to:

The 612 ECT treatments performed in our institution on 75 patients between October 1st 1996 and September 30th 1997 were retrospectively reviewed. The median number of ECT treatments per patient was eight (1-23), with four patients receiving two series of eight treatments and 10 receiving a maintenance ECT each month after an initial course of treatment.

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Anaesthesia for ECT was provided according to the French legal standards and the recommendations of the French Society of Anaesthesiology and Critical Care. A preanaesthetic evaluation was performed at least 3 days before the first ECT, and a standard evaluation form completed by the anaesthesiologist. Additionally a full blood count, blood electrolytes, blood urea and activated partial thromboplastin time were determined for all patients. An electrocardiograph, a chest radiograph and a cardiologist evaluation were undertaken for patients older than 40 years or those with known cardiovascular pathology. A dental examination was made by a stomatologist and a dental cast was taken to provide a dental protector specific for each patient.

None of the ECT courses was conducted on an outpatient basis or those for maintenance ECT, except in five patients and for 25 ECT treatments. The patients fasted from midnight. Although no premedication was given regular cardiac medications were given. ECT sessions were conducted from 08:00 to 12:00 hours, twice a week. Patient monitoring was performed with an electrocardioscope, an electroencephalogram, a pulse oximeter and a non-invasive blood pressure device, and dental protectors were inserted into the patients' mouths. After preoxygenation, anaesthesia was induced with propofol (1-1.5 mg kg⁻¹) and a Guedel oral airway was inserted if required. In five patients, with severe osteoporosis or prosthetic joints, succinylcholine (1 mg kg⁻¹) i.v. was also administered. Then, the patient was firmly held by the nursing staff to prevent any trauma and the bilateral ECT stimulation (Thymatron DG, Somatics) was applied by the psychiatrist. After the end of the period of convulsions, manual ventilation of the lungs with oxygen 6-8 L min⁻¹ was performed until spontaneous respiration was resumed. After the patient had opened his/her eyes, he/she was taken to the recovery room, given oxygen 3 L min⁻¹ and monitored using pulse oximetry and an automatic blood pressure device until the anaesthesiologist authorized discharge. A standardized record was then completed by the anaesthesiologist. Blood pressure, pulse rate and mood were monitored every 1 to 3 h for the next 8 h by the nursing staff in the psychiatric department. The anaesthesia and clinical records of ECT were retrospectively analysed by an independent observer. Complications were considered as severe when they were life threatening (such as laryngospasm, bronchospasm, inhalation pneumonitis, severe angina pectoris or hypoxaemia despite an enriched oxygen supply) or they potentially impede discharge on the same day. No files were missing.

Results Go to:

There were 17 men and 58 women. The median age for men was 47 years (36-88 years), and their median weight was 87 kg (60-100 kg). The median age for women was 62 years (25-76 years), and the median weight was 58 kg (43-90 kg). Amongst the patients, 22 (29%) were class ASA I, 43 (57%) ASA II, seven were (9%) ASA III and three were (4%) ASA IV; 34 patients (45%) were older than 60 years, and 25 (33%) had previously received one or more courses of ECT. The psychiatric diagnosis was depression resistant to medication in 56 patients, unipolar and bipolar disorders for five and eight patients, respectively, schizophrenia for three patients and undetermined for three patients. Patients also received various psychiatric medications (lithium, tricyclic antidepressants, neuroleptics, paroxetine and benzodiazepines) which changed several times during the course of the study period. The past medical history of the patients is summarized in Table 1.

One hundred and twenty-six complications (20.6% of the ECT procedures) occurred in 51 patients (68% of the patients) (Table 2). Some patients experienced the same type of complication after several different ECT sessions: 11 patients had the same complication twice, seven patients, three times, one patient, four times and five patients, five times (Table 3). Fourteen patients suffered from two different complications, and six from three (Table 4). Twelve potentially life-threatening complications (2% of all the ECT procedures) occurred in seven patients (9% of the patients) (Table 4).

Neuropsychiatric complications

Eight patients (10%) complained of headaches and in three patients, headaches reappeared after several (three or more) ECT episodes. Three patients had nausea, one patient suffering from this after several ECTs. Twenty-five patients (33%) suffered from confusion for more than 2 h after ECT. In 10 of them (13%) the confusion recurred after subsequent ECT. Nine patients (12%) were agitated for more than an hour after ECT and in three the agitation reappeared after the next ECT. One patient was agitated after a maintenance ECT session and was confused after another. One patient recovered consciousness only 15 min after ECT.

Traumatic complications

In six patients (8%) traumatic complications occurred; all of them had previously had ECT. Three patients had dental damage despite the use of dental protection in all and succinylcholine in one of them. Poor dental condition had previously been noted on the anaesthesia evaluation form for all these patients. One patient had a split lip and another a lacerated tongue. One patient with rheumatoid arthritis suffered a fractured humerus which required surgical repair.

Respiratory complications

Respiratory problems occurred in six patients (8%) but none during the three first ECTs. They were considered as life threatening in five patients (Table 5). Patient no. 1 was carefully watched on the nights before ECT to prevent him from smoking and eating and no adverse respiratory event occurred. The course of ECT was initially interrupted in patients nos. 2 and 5, but because medical treatment of depression was inefficient it was decided to attempt ECT again. In addition to their usual antisialogogue medication, they were given metoclopramide, cimetidine and sodium citrate as premedication. ECT was performed in the seated position and cricoid pressure was maintained during the procedure. These patients did not suffer from any other respiratory incident. Patient nos. 3, 4 and 6, did not receive any other ECT.

Cardiovascular complications

Eleven patients (15%) had hypertension after ECT and required treatment (three of these patients more than 2 h after the ECT) and two patients after two or more ECTs. Among them, eight had not suffered previously from hypertension. Four patients developed severe bradycardia - 20 beats min⁻¹ - immediately after the convulsions: this resolved after atropine i.v. administration. One patient had a sinus dysrhythmia, which terminated spontaneously. One patient, a 42-year-old, with previously untreated hypertension, developed ST segment changes and chest pain after his fourth ECT. He was transferred to the cardiology department and the ECT course was discontinued. He reported later having previously had chest pain during exercise, although he had not mentioned it during the preanaesthesia assessment.

Allergic reactions

One patient suffered from a skin rash after her sixth ECT. Propofol was then changed to etomidate for future sessions.

Miscellaneous complications

One patient aged 42 years, died at home from pneumonia 4 days after ECT. She had previously had several bowel obstructions due to the large amount of antipsychotic medication she was taking. Unfortunately, we have no information to judge whether the pneumonia was related to ECT or not. In five patients central venous access was required for parenteral feeding (cachexia) as peripheral venous access was impracticable. One of these patients suffered from a pneumothorax and another one from catheter-induced septicaemia.

Discussion

In this series of ECT under general anaesthesia, 51 patients (68%) were affected by a complication and among them, seven (9%) had one or more potentially life-threatening events. The general morbidity of ECT had been previously estimated between 0.3% and 0.4% [1], but confusion, and headaches were not considered as significant complications by some authors [2,4,13]. On the contrary, studies rating confusion and agitation as complications reported morbidity rates up to 54%, which is similar to our morbidity rate [14]. Other authors consider psychiatric complications and memory impairment as part of a normal ECT course [15]. Their occurrence is increased by the use of bilateral stimulation and simultaneous psychiatric medication [15,16] such as in the present study explaining the high confusion rate. Older patients are also prone to develop confusion after ECT. Out of 138 patients, Burke and his colleagues [2] observed 18% and 35% complication rates in patients, respectively, under and above 80 years of age with higher confusion rates in older patients (13% vs. 18%). Casey and his colleagues [6] found 22.7% complications when patients were over 75 years of age. In patients over 65 years, Cattan and his colleagues [3] noted that repeated confusion was the most frequent complication.

In cases of outpatient ECT, confusion and agitation are serious problems, as confused patients have a risk of falls (14% of iterative falls in patients between 65 and 80 years and 36% above 80 years) [3], wounds and fractures, escape from their family, as well as violence [2]. Confusion can also mask somatic problems such as bladder distension as well as more serious events such as cardiac ischaemia [17]. Thus, confusion precludes any ambulatory ECT. Moreover, all the serious complications including confusion, occurred after three or more uneventful ECT procedures. Burke and his colleagues [2] have reported the absence of correlation between the number of ECTs and the occurrence of complications. They suggest that if a patient had previously received an ECT without any complications, this does not imply that subsequent ECT remains at low risk. In the present study, the organization of ECT courses on an outpatient basis would have thus been possible only in a very limited number of patients.

Cardiovascular complications

Other studies suggest that cardiovascular complications are the main components of the morbidity rate and are favoured by pre-ECT cardiovascular status [18]. In the study by Rice and his colleagues [5], 19.2% of major complications occurred in patients with a cardiovascular risk factor whereas in only 14.8% without. In another study, out of 40 patients with previous cardiovascular disease, 22 had a cardiovascular complication, eight of which were potentially life threatening (chest pain, ECG modification, asystole) [4]. A threefold increase in cardiovascular complications was also found in patients over 80 years of age (36%) [3]. However, in this study cardiovascular complications were not the main factor contributing to morbidity and only one patient suffered from a serious cardiovascular complication, mainly because he failed to report his previous cardiac symptoms. This case outlines the specific difficulties of the assessment of psychiatric patients who might forget important parts of their past medical history.

Respiratory complications

Contradicting previous epidemiological studies, a high rate of respiratory events was observed in this study. Respiratory complications are amongst the most common complications of general anaesthesia and seizures [19]. Burke and his colleagues [2] found two instances of aspiration pneumonia among 138 ECT patients. Cattan and his colleagues [3] reviewed the medical records of 81 older patients (65 years of age and over), who underwent ECT at a university-affiliated private geriatric hospital, to evaluate the safety and efficacy of this treatment for depression in the young-old (65-80 years) compared with the old-old age group (over 80 years). They found that patients over 80 years had significantly more cardiovascular complications and falls (95% confidence interval) and tended to have a worse ASA-scale rating and a somewhat less successful outcome. Because all patients with pulmonary aspiration do not show clinical symptoms [20], the incidence may be underestimated, especially in confused patients. Thus, Wayne and his colleagues [21] compared chest radiographs before and after ECT in 12 patients. They found abnormalities in three patients: two had atelectasis of the lower left lobe and one had pulmonary oedema. None of the patients had dyspnoea. Thus, it seems that respiratory complications occur quite often after ECT and perhaps were unnoticed in some previous studies because of the lack of appropriate monitoring. The use of pulse oximetry is not mentioned in some studies although patients received oxygen before and during the ECT procedures [2,4]. McCormick and Saunders [8] found a decrease in SpO₂ < 90% after ECT in 17% of the patients breathing air during the recovery period, which is similar to the present study. However, the incidence of laryngospasm is far higher than the 5.6% and 8.9% rate already observed during general anaesthesia [22]. Out of the six patients with a respiratory complication, five were recorded probably because of specific risk factors such as a broken preoperative fast (patient 1), hiatus hernia (patients 2 and 5) and gastro-oesophageal reflux. Berrios and Sage [23] noted that among 76 patients, 21 broke the preanaesthetic fast and most often during the 3 h before ECT. However, the relationship between hiatus hernia and gastro-oesophageal reflux is still debated. In our series of patients, one of the patients with hiatus hernia did not develop any adverse respiratory event. The incidence of gastro-oesophageal reflux has been estimated between 14.8% and 15.9% during general anaesthesia with a facemask, and between 18.8% and 11.1% during the recovery period [24,25]. Studies of anaesthesia using a facemask for other procedures do not report such a high incidence of laryngospasm or coughing [25]. However, the depth of anaesthesia for ECT is only light which is a known factor contributing to increased reactivity of the larynx. Combined psychiatric medications may also enhance the risk of gastric fluid regurgitation. The upper and lower oesophageal sphincter pressures are decreased by benzodiazepines [25,26]. Tricyclic antidepressants reduce gastric secretion [27], but can induce hiatus hernia [28]. Although neuroleptic drugs increase the lower oesophageal pressure [29], they may induce laryngopharyngeal dyskinesia and death after food inhalation in conscious patients has been reported [30]. Thus, the laryngospasm, coughing or pneumopathy observed in patients 3, 4 and 6 could have also been linked to inhalation of saliva [31].

The management of ECT in patients with hiatus hernia, or at risk of inhalation, is controversial. The sole use of succinylcholine may not prevent pulmonary aspiration, as it increases the intragastric pressure and decreases the upper oesophageal pressure [31,32]. The most effective way to prevent pulmonary aspiration is endotracheal intubation with cricoid pressure, but, to our knowledge, there have been no studies on elective

tracheal intubation for ECT except an anecdotal case report of ECT for a pregnant patient [33]. Elective tracheal intubation is unlikely to be considered in patients with difficult intubation, for example patient no. 5 in our series. Antacid and antiemetic premedication, the application of cricoid pressure and administration of ECT in the sitting position were simple methods with few side-effects that could be applied to any patient but deserves evaluation.

Traumatic complications

Traumatic complications during ECT can be prevented with short-acting muscle relaxants although one of our patients had a tooth broken despite the use of succinylcholine. Since the publication of the French National Guidelines for Electroconvulsive Therapy, succinylcholine 1 mg/kg is now given to all patients unless contraindicated. In conclusion, the morbidity associated with ECT may have been underestimated. This study addresses the specific problem of pulmonary aspiration and respiratory complications during ECT, although the effects of most psychiatric drugs on gastric emptying and oesophageal tone are well known. Because of the high incidence of post-ECT confusion (and potential associated complication), or the risk of broken preoperative fasting, anaesthesia should not be performed regularly on an outpatient basis. The occurrence of complications despite previous uneventful ECT must be kept in mind to inform patients of the risk of unanticipated admission after an ECT scheduled on an outpatient basis.

Acknowledgments Go to:

We would like to address special thanks to Dr J. Pratt and Mrs C. Pailley for their helpful comments.

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Image Previews

Effects of Medical Comorbidity on Post-Treatment Survival

Barry S. Fogel, M.D.

11

Recovery is also related to the intrinsic mortality of the primary illness, in addition to the mortality of any coexisting illness. Major depression is a common disorder

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Nevertheless, the mortality of patients with depression and what one must take is a diminished expectation of the high rate of medical illness.

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among the elderly and results in a significant amount of mortality if left untreated. Roth² found a mortality rate of 14.8% at 6-month follow-up in a group of 54 depressed patients, all of whom were over age 60. More recently, Avery and Winokur³ compared the mortality of patients with depression, age 60 or older, who had been treated with ECT with those who had been treated with neither ECT nor antidepressants and found 3-year mortality rates of 5.1% and 21.1%, respectively.

The association between affective disorder and mortality in the general population has been a major focus of a 16-year investigation by Murphy et al.⁴ A summary of outcome that combined mortality from any cause with psychiatric morbidity revealed that 75% ($n = 41$) of the individuals 60 years old or older who had depression, anxiety, and/or affective disorder at baseline had a poor outcome at follow-up. Overall, 82% of the sample with depression at baseline had a poor outcome 16 years later.

In an effort to measure mortality in a psychiatric population, Martin et al.⁵ established the mortality status for 494 subjects during follow-up. A significant excess in mortality was observed among patients with diagnoses of antisocial personality disorder, alcoholism, schizophrenia, drug addiction, organic brain syndrome, homosexuality, and secondary affective disorder. Excess mortality was not observed among patients with primary affective disorder, using the distinction that secondary affective disorder was preceded or accompanied by a life-threatening, serious, or incapacitating medical illness. It is of interest that an excess of mortality was not found in any psychiatric category in the two oldest age groups, 65–74 years and 75–84 years.

Nevertheless, when considering the mortality of patients who have major depression and who are over the age of 80, one must take into account the statistically diminished expectation of survival because of the high rate of comorbid, age-associated medical illness. This factor alone contrib-

utes to the unique aspects of depressive disorders in this age group and has a likely effect on prognosis.

This nonexperimental study was part of a historical cohort study of hospitalized patients with mood disorder. Roughly 12% of admissions to our Psychiatric-Medical Inpatient Unit at Rhode Island Hospital are over age 80. This contrasts with the 5.2% of the general population who are over age 80. Rhode Island has the fourth highest percentage of residents over age 65 (14.7%), after Florida (17.8%), Pennsylvania (14.8%), and Iowa (14.8%).¹

METHODS

We included in a chart review all patients hospitalized between 1974 and 1983 at the Rhode Island Hospital who were over the age of 80 when admitted and who had a discharge diagnosis of any of the following: major affective disorders (from DSM-II⁶), involuntional melancholia (from DSM-II), affective psychosis (from ICD-8⁷), depressive disorder (from ICD-9⁸), major depression (from DSM-III⁹), or atypical depression (from DSM-III). Patient charts that had clear historical or clinical criteria for DSM-III-R¹⁰ definition of major depression were included and labeled as the index admission.

A sample of 10 records, 5 with and 5 without criteria for major depression, was reviewed separately by two independent raters to establish the reliability of the method of subject selection; raters agreed on all 10 charts. Treatment modalities were then ascertained. Patients receiving ECT were initially evaluated separately ($n = 37$). This includes 9 patients who were intolerant of medications. The others received ECT because of severity of symptoms, treatment refractoriness, psychotic features, or a good past response to ECT.

To observe and quantify the survival of all patients over age 80 following inpatient treatment for major depression, we identified a comparison group of patients who

ECT in the Oldest Old

were also hospitalized for major depression but did not receive ECT ($n = 28$). This group consisted of 12 patients from Rhode Island Hospital and, as a way to increase the number in this reference group, 16 patients from Fuller Memorial Hospital, an 82-bed private psychiatric hospital in southern Massachusetts.

Hospitalizations for clear and/or established organic brain syndromes (DSM-II), depressive neurosis (DSM-II), dysthymia (DSM-III), or organic affective disorder (DSM-III) were excluded in both groups. Length of stay (in days) in hospital was determined from the charts as was the length of follow-up, which was calculated from the date of index admission.

Information pertaining to next of kin and/or relatives, subsequent admissions, and residence status was collected from the chart. Letters were addressed and sent to the patient with reference to our intended study and purpose. Interview was via telephone survey (by D.K.) using the following hierarchy of interviewees: patient, spouse, child, sibling, physician, friend, other professional caretaker. If a person on the list was unavailable, deceased, or unable to give complete information, the next person on the hierarchy was invited to contribute information. Interviews were stopped when the standardized semistructured interview was complete, when the list of interviewees was exhausted, or when there was a lack of cooperation. Attempts were made to ascertain the following information: pre- and posthospitalization residence status, recurrence of depressive features, the need for rehospitalization (psychiatric and/or medical), medications (in-hospital, posthospital, current), condition on discharge, additional courses of ECT, and a subjective report on present functional status. Recurrence rates were determined by subjective report during the follow-up interview. Patients or interviewees were asked if the depression recurred.

If a patient was deceased, the date and cause of death was determined. In six of the

deceased, the date and cause of death were obtained through the Rhode Island State Department of Vital Statistics. There were three patients in the non-ECT group on whom follow-up information was not available.

Survival experience for all patients was then compiled for analysis. For this study, survival was measured from the index admission date to either the time of telephone interview or to the time of death. Estimation of the survival curve was calculated by analysis of the survival data as described by Kaplan and Meier.¹¹ The essential concept of this "actuarial" survival curve involves the recording of survival times for n individuals and r of these times exceed a specified time, t , where r refers to the number of surviving individuals at risk. The natural estimate of the probability of surviving more than t units would be r/n . The Kaplan-Meier methodology produces a nonparametric estimate of survival when all the survival times are not exactly known. For instance, if death has not occurred by t , then survival time must exceed t and the resulting survival curve reflects this probability.

Comparison of the Kaplan-Meier curves for both treatment groups was calculated by the log-rank or Mantel-Haenszel test,^{12,13} which is designed to detect differences between survival curves from two groups. This test is especially useful when the survival rate in one group is consistently higher than the corresponding survival of another group and the ratio is constant over time. The log-rank test was chosen as the test for survival comparison because it gives equal importance to all deaths within comparison groups, as opposed to the generalized Wilcoxon test, which attaches greater importance to earlier rather than later deaths. The goal of this study was to view longitudinal survival with different points of entry.

RESULTS

Table 1 provides a summary of the demographic data for the two comparison groups.

TABLE 1. Characteristics given for depressed patients

Total ($N = 65$)
Male ($n = 17$)
Female ($n = 48$)
Mean age, years, at index admission
Male
Female
Length of follow-up, median/interquartile

We found a total of 80 years old or older hospitalized for depression were treated with medication. The non-ECT group consisted of 28 patients (15), trazodone (1), chloral hydrate (2), maprotiline (1), and nomifene (1).

Table 2 summarizes the results. At the end of the study, there were 31 individuals remaining, 16 male to 15 female (2.9:1) and non-ECT (2.9:1) corresponds to the cause of death. Included cancer (1), pneumonia (1), aspiration, or uremia or congestive intestinal obstruction ($n = 1$), cerebral stroke ($n = 1$), and suicide ($n = 1$), at the end of the study.

The mean number of days between admission and death was 2.9. Two patients withdrew from the study. One patient developed CHF and was not continued. The interval between admission and death was 1 month. The number of days between admission and death was 1 month.

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TABLE 1. Characteristics of patients over 80 given ECT and non-ECT treatment for depression

	ECT	Non-ECT
Total ($N = 65$)	37 (56.9%)	28 (43.1%)
Male ($n = 17$)	9	8 (26.1%)
Female ($n = 48$)	28	20 (73.9%)
Mean age, years,		
at index admission	83.0	82.5
Male	83.0	82.1
Female	83.0	82.6
Length of follow-up, months		
median/interquartile range	34/43	30/35

We found a total of 65 patients who were 80 years old or older when they were hospitalized for depressive illness. Thirty-seven were treated with ECT and 28 were treated primarily with medication. Treatment of the non-ECT group comprised tricyclic antidepressants ($n = 20$), benzodiazepines ($n = 15$), trazodone ($n = 6$), neuroleptics ($n = 5$), chloral hydrate ($n = 2$), lithium carbonate ($n = 2$), maprotiline ($n = 1$), carbamazepine ($n = 1$), and nomifensine ($n = 1$).

Table 2 summarizes treatments and outcomes. At the endpoint of the study there were 31 individuals (47.7%) alive. The female to male ratio of those living at the endpoint (2.9:1) was similar in the ECT (3:1) and non-ECT (2.8:1) groups and corresponds to the baseline rate (2.8:1). The cause of death in the 34 individuals included cancer ($n = 8$), "failure to thrive," (i.e., pneumonia, septicemia, dehydration, aspiration, or urosepsis; $n = 8$), cardiac arrest or congestive heart failure (CHF; $n = 7$), intestinal obstruction ($n = 2$), renal failure ($n = 1$), cerebral vascular accident ($n = 1$), suicide ($n = 1$), and unknown ($n = 6$).

The mean number of ECTs was 7.9 ± 2.9 . Two patients had only 2 ECTs: one patient withdrew consent, and the other developed CHF and died before ECT could be continued. The median value for the interval between ECT and death was 20 months with an interquartile range of 45 months. The number of ECT treatments for

TABLE 2. Treatment and outcome of patients over 80 given ECT and non-ECT treatment for depression

	ECT	Non-ECT
Mean length of stay, days		
Median/interquartile range	32/20	19/14
At Rhode Island Hospital	32/20	30/15
At Fuller Mem. Hospital	na	18/9
Living at 1 year, %	73.0	96.4
Mortality, %		
At 1 year	27.0	3.6
At 3 years	48.6	33.3
Number living at endpoint of study ($n = 31$)	8	23
Male (25.8%)	2	6
Female (74.2%)	6	17
Mean age, living, years	87.5	85.0
Male	84.5	85.9
Female	88.5	84.6
Recurrence		
Yes = 27 (41.5%)	20 (54.1%)	7 (25.0%)
No = 28 (43.1%)	8 (21.6%)	20 (71.4%)
Unknown = 10 (15.4%)	9 (24.3%)	1 (3.6%)
Rehospitalization		
Yes	9	3
No	11	4
Unknown	8	1
Additional ECT		
Yes	6	na
No	23	na
Unknown	8	na
Residence after hospitalization		
Nursing home	18	8
Own home	17	15
Unknown	1	5

Note: For a description of outcome measures, see text. Patients who had a recurrence are incorporated in rehospitalization category.

any one patient was not significantly correlated with the likelihood of recurrence.

We found an overall recurrence rate of 41.5%, with 54.1% in the ECT group and 25.0% in the non-ECT group. The mean age at index admission of those who had recurrence of depression was 83.0 years, compared with a mean age of 82.8 at admission for those who did not have recurrence. Lasting recovery was achieved in 43% of our patient population (22% in the ECT and 71%

ECT in the Oldest Old

TABLE 3. Survival rates of patients over 80 given ECT and non-ECT treatment for depression

	Survival Rate, %		
	1 year	2 years	3 years
ECT	73.0	54.1	51.4
Non-ECT	96.4	90.5	75.0

in the non-ECT group), which is considerably greater than the study of Baldwin and Jolley¹⁴ (21.9%) and the two studies by Post^{15,16} (30.8% and 26.1%, respectively). If recurrence was measured by the rehospitalization rate, then the difference between the two groups would be less significant (41% for the ECT group and 15% for the non-ECT group). "Recurrence yet not hospitalized" could be considered to be a "soft" relapse, the significance of which should be questioned. The rehospitalization rate from this study coincided with the rehospitalization rate for geriatric depression in the study by Colenda et al.¹⁷

The estimated survival curves for the ECT group, the non-ECT group, and the normal population (from U.S. Census data) are shown in Figure 1. As can be seen, the "curve" is derived from horizontal sections, which represent the intervals of time that deaths were not observed, and vertical steps, which represent the number of deaths recorded at *t* months. The analysis of survival differences for the two treatment populations revealed constant differences across time. The significance level using the Mantel-Haenszel calculation is $0.005 > P(\chi^2 > 14.20) > 0.001$; this supports the conclusion that there are true differences in the survival of the two groups.

Table 3 gives the 1-, 2-, and 3-year survival rates after discharge from hospital. At 1 year we established a 73.0% survival rate for the ECT group and a 96.4% survival rate for the non-ECT group. At 3 years, the survival rate of the ECT group was 51.4% compared with 75.0% for the non-ECT group. These survival rates are lower than those of the group age 60 years or older

treated with ECT ($n = 39$) in the study of Avery and Winokur,³ who found a 97.4% 1-year survival rate and a 94.9% 3-year survival rate.

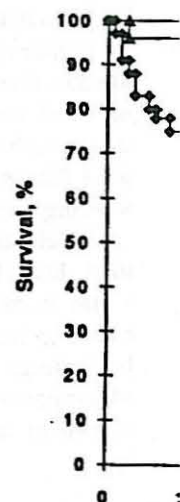
It can be seen from Table 3 that the survival of the ECT group had a higher rate of decline between the first and second year than between the second and third year. This suggests that mortality from combined depression and unstable medical illness is highest in the first 2 years following treatment, after which survival parallels the aged-matched general population. ECT did not appear to interfere with the normal propensity for survival in our elderly sample, and this corresponds to the findings of Gaspar and Samarasinghe¹⁸ and the conclusion of Benbow¹⁹ that ECT is safe in the medically compromised elderly.

DISCUSSION

The findings of this study suggest that patients over 80 years old who receive ECT for major depression are at increased risk for death over the 2 years following treatment. The mortality rates for these patients exceed the rates for patients of comparable age treated in a hospital without ECT, and both exceed published rates for mortality following depression in younger patients.^{3,20} The variance was not, however, due to the use of ECT and had more to do with patient characteristics.

Although it was not the ultimate intent of this study to measure treatment efficacy, attention was paid to the possibility for selection and treatment-monitoring biases. The selection of subjects was solely by age, illness, and hospitalization status and, while there were a few subjects who could not be reached during follow-up, our ability to recruit participants was good to excellent. Accuracy and reliability of historical information was likely subject to both interviewer bias and recall bias, an inherent limitation of this and most other historical cohort studies. Underreporting or over-

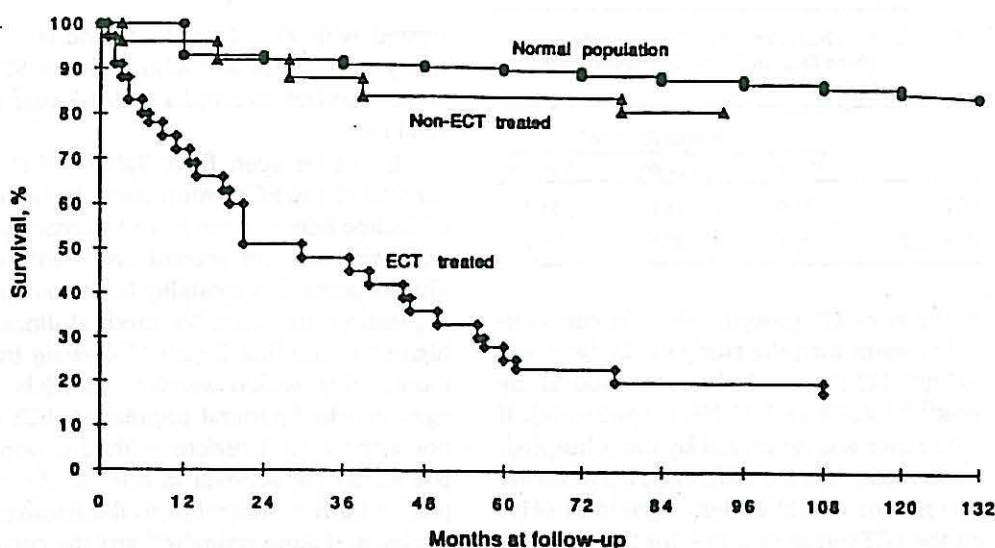
FIGURE 1. Kaplan-Meier survival curves for ECT and non-ECT groups.



reporting of clinical endpoints is also a concern in this type of study. The comparison is strengthened by the fact that subjects refused to participate in the study, and the same time period was used for both groups.

The pretreatment of both groups was similar, and any of the other factors that could affect survival were controlled for. The most obvious cause of medical illness was depression. The medically debilitated elderly, which was a high-risk population, had a high length of hospitalization rate, and tended to be in a nursing home or hospital. The Medical Island Hospital

FIGURE 1. Kaplan-Meier actuarial survival curve



reporting of clinical information by the respondent is also a source of potential bias in this type of study design. The validity of the comparison in this study can be strengthened by the fact that none of the subjects refused to participate and the courses of illness for both groups were similarly monitored, as was the follow-up procedure. In addition, all subjects were hospitalized and treated during roughly the same time period, thereby keeping treatment practices standard.

The pretreatment clinical characteristics of both groups were more important than any of the other measured variables. The most obvious covariates were the presence of medical illness, the need for close ancillary medical attention, and severity of depression. The ECT group was both medically debilitated and mentally more ill, which was supported by the increased length of hospital stay, the higher recurrence rate, and the increased need for extended care in a nursing home. The patient populations in both hospitals differed, reflecting the clinical function of each hospital. The Medical-Psychiatry Unit at Rhode Island Hospital focuses on patients with

comorbidity, whereas Fuller Memorial Hospital has a lower threshold for medical/psychiatric comorbidity.

For this reason the mortality and survival of the non-ECT group cannot be compared with the ECT group with statistical significance, yet these rates should be compared as a measure of the effect of physical illness on outcome. One way to strengthen this and other similar studies would be to record the number of medical diagnoses or the number of total medications and then make adjustments in the analysis. Nevertheless, our finding that those depressed patients with diminished survival were more likely to be suffering from chronic physical illness coincides with the study of Murphy,²¹ who found that of 124 elderly depressed patients, 49% of the "poor outcome" group had major chronic physical health problems compared with 29% in the "good outcome" group.

The group of elderly patients who received ECT could be considered to be at a relatively higher risk because of their advanced age and physical illness, yet we did not find an increase of ECT-related mortality or morbidity in these very old individuals.

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This is in contrast to Alexopoulos et al.,²² who found medical problems arising during ECT to be significantly more common in those over age 65. We conclude that ECT is effective and useful in patients over age 80 with the same margin of safety as in younger populations, as long as attention is given to the optimal management of comorbid medical illness. People over age 80 can be treated successfully and safely with ECT.

In addition, and contrary to Babigian and Guttmacher,²⁰ who found that women aged 75 years treated with ECT had substantially decreased mortality compared with age-matched non-ECT-treated groups, we found increased mortality in the ECT-treated group. The increased mortality in our study can be viewed as a reflection of the prevalence of severe physical illness in the group of patients who received ECT. The small number of patients in this study did not allow for calculation and comparison of cause-specific deaths in proportion to the standard, nondepressed population. All deaths, rather than cause-specific deaths, were included for estimation of relative survival. It would be important to establish whether treatment of depression with ECT has a positive effect on the mortality from and comorbidity of medical diseases.

The number of patients suffering from "first episode of depression" at the index admission vs. a history of "recurrent depressive episodes" was not ascertained, and this may identify patients with more refractory depressive disorders. It is also unclear how many of the index admissions and readmissions were preceded by apparent or occult medical illness. In some cases, the physical

illness was manifest, but in others, an underlying problem, such as an occult cancer, may have increased the severity and refractoriness of depression before the medical illness was apparent.

In this study the survival characteristics varied with the type of treatment, which is not to say that the treatment directly affected survival. The variance is a function of the clinical heterogeneity of our patient population and it appeared that the non-ECT group had the same survival rates as the general population. The psychiatric follow-up data, while limited, show that those patients who did not die had a reasonable chance of remaining in the community and avoiding rehospitalization for recurrent depression.

It became clear during the initial data collection of the non-ECT group that the survival characteristics of both groups could not be reliably compared because the ECT group was, in general, a sicker group. The high mortality rate in the ECT group suggests that patients over 80 who are referred for ECT have more physical illness and depressive symptomatology caused by physical distress than those who can be successfully treated with medication. These patients deserve particularly close follow-up because they are at a higher risk for life-threatening physical illness. In addition, further ECT outcome studies in this age group must give meticulous attention to medical comorbidity as a prognostic factor.

This article was presented by Dr. Kroessler at the Fifth Congress of the International Psychogeriatric Association, August 18-23, 1991, in Rome, Italy.

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Does Electroconvulsive Therapy Prevent Suicide?

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Summary: To examine the issue of whether or not electroconvulsive therapy (ECT) protects against suicidal death, we followed a complete population of 1,494 adult hospitalized psychiatric patients for 5-7 years. During that time there were 76 deaths, of which 16 or 21% were by suicide. Cause of death was not significantly related to age, gender, or research diagnosis. Patients who committed suicide were more apt to have received ECT than those who died from other causes, but this difference was not significant. A control group of living patients matched for age, sex, and diagnosis had very similar exposures to ECT, which further indicates that ECT does not influence long-term survival. These findings combined with a close examination of the literature do not support the commonly held belief that ECT exerts long-range protective effects against suicide.

Key Words: Electroconvulsive therapy—Suicide.

At the recent Consensus Development Conference on Electroconvulsive Therapy (ECT) sponsored by the National Institutes of Health and Mental Health, there was much argument concerning whether ECT does or does not reduce the risk of suicide. At first, this concern would appear to be superfluous as ECT is known to be an effective form of treatment for severe depression and other illnesses that are associated with a significantly elevated risk of suicide. The conference report (Consensus Development Conference, 1985) states that "the immediate risk of suicide (when not manageable by other means) is a clear indication for consideration of ECT." However, factual data in support of this contention are not readily obtainable.

Studies by Tsuang et al. (1979) and Avery and Winokur (1976) often are quoted as showing that ECT is associated with lower mortality rates than is drug therapy or institutional care in the treatment of patients with schizoaffective disorder or depression. However, their data show reduced mortality from *all* causes but no significant reduction in suicidal death per se. Avery and Winokur (1976) found that death from suicide was not different in patients receiving ECT compared with those receiving

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other treatment modalities. Later, these same authors (1978) demonstrated that patients who were treated with ECT made significantly fewer suicide attempts over a 6-month follow-up period than did patients who did not receive ECT. However, Babigian and Guttmacher (1984) failed to demonstrate that ECT exerts a protective influence against suicidal death. Eastwood and Peacocke (1976) did not find an interrelationship between suicide, hospital admissions for depressive illness, and ECT.

Review of the early literature also reveals conflicting findings. Ziskind et al. (1945) reported that treatment with ECT or pentylenetetrazol (Metrazol) reduces death from suicide. Huston and Locher (1948a) found that none of their patients with involutional melancholia treated with ECT committed suicide, whereas 13% of untreated patients did. The same authors reported a lower rate of suicide in manic depressive patients treated with ECT than in untreated patients (1948b). However, two subsequent studies (Bond, 1954; Bond and Morris, 1954) found no significant protective effect of ECT against suicide in patients with either involutional psychosis or manic depressive illness.

FOLLOW-UP STUDIES

In an effort to cast light on this still unresolved question, we report our findings from follow-up studies of a series of 1,494 patients. They consisted of all consecutive adult admissions to Larue D. Carter Memorial Hospital during the years 1965-72. Further details concerning the facility and patient sample appear elsewhere (Small et al., 1984). From contacts with families and attending physicians and cross-referencing of patients' names listed on Indiana death certificates, we ascertained that 76 patients had died during the 5- to 7-year follow-up period. Thus, 5.1% of the total sample had died by the time of follow-up, and of these, 16 or 21% were the result of suicide. Causes of death were examined in relation to age, sex, retrospective research diagnosis (Feighner et al., 1972), and whether or not the patient had received ECT during the index hospitalization or at any time in the past. These data are summarized in Table 1.

Neither age nor gender was significantly related to suicidal versus nonsuicidal deaths. There were no significant associations with research diagnoses grouped in terms of affective disorder, schizophrenia, or other conditions. Forty-four percent of the patients who committed suicide had been treated with ECT during the index hospital admission, whereas 32% of patients who died from other causes had received ECT. These differences were not statistically significant.

In view of these negative findings, we next evaluated a control group of patients who were still alive at follow-up. The patients comprising this group were individually and exactly matched for sex and research diagnosis (Feighner et al., 1972) with those who had died. They also were matched for age as closely as possible and for date of admission to the hospital. When we examined the ECT experience of these living matched control patients and compared them with those of the patients who had died, we found no statistically reliable differences (Table 1).

DISCUSSION AND CONCLUSION

The results of this retrospective study do not support the contention that ECT exerts long-term protective effects against suicide. Although not statistically significant, more

n	Males/female:
n	%
Research diag	
Affective	
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Other	
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TABLE 1. Patient characteristics by outcome

	Patients who died		Living controls matched to death	
	Suicide	Other	Suicide	Other
n	16	60	16	60
Males/females				
n	6:10	23:37		
%	38:63	38:62		
Research diagnosis (n%)				
Affective	4 25	21 35		
Schizophrenic	4 25	12 20		
Other	8 50	27 45		
Mean age (yrs)	32	43	31	44
ECT during index admission (n%)				
Yes	7 44	19 32	8 50	21 35
No	9 56	31 68	8 50	39 65
ECT: index plus history (n%)				
Yes	8 50	24 40	9 56	29 48
No	8 50	36 60	7 44	31 52

of the patients whose death was ascribed to suicide had received ECT during their index hospital admission than those who died from other causes (44 vs. 32%). Similarly, when their previous ECT experience was added, more patients who died as a result of suicide had received ECT (50 vs. 40%). The matched control group revealed very similar percentages, suggesting that ECT has minimal impact on long-range survival.

To consider the early studies demonstrating that ECT exerts a protective effect against suicidal death, the published data must be reworked to determine whether differences were significant. Ziskind et al. (1945) followed ~ 200 patients for a mean of 40 months (range 6-69 months). Eighty-eight patients were treated with either Metrazol or ECT. The remaining 109 patients either refused convulsive therapy ($n = 43$), had symptoms too mild to warrant this treatment ($n = 50$), or had a condition contraindicating ECT ($n = 16$). There were 13 deaths in the control patients with 9 by suicide, compared with 3 deaths with 1 suicide in the convulsive therapy patients. These data yield a Fisher's exact probability of 0.029, indicating a significant association between treatment/nontreatment and suicide/other causes of death. However, the conditions of the 16 patients with contraindications to ECT and whether they contributed disproportionately to the suicides are unknown.

Huston and Locher (1948a) compared patients with involutional psychosis untreated and treated with ECT. They found that none of the patients in the convulsive therapy group committed suicide, whereas 13% of those untreated did. Interpretation of this study is complicated by the fact that they followed the ECT-treated patients for a mean of 36 months (range 1-48 months) and the untreated patients for 77 months (range 2 days to 180 months). In a subsequent report on manic depressive psychosis treated with ECT or not, the same authors (1948b) found that the ECT-treated patients, followed for a mean of 36 months, had a 1% suicide rate, while the control patients, followed for a mean of 82 months, had a 7% suicide rate. Examining the association

of ECT/no ECT and death from suicide/other causes yielded a nonsignificant probability using Fisher's exact method. In studies of patients with involutional psychosis (Bond, 1954) and manic depressive illness (Bond and Morris, 1954) examined 5 years after treatment with ECT or no treatment, analysis of these data does not reveal a significant protective effect against suicide of ECT compared with nontreatment.

Thus, we are able to point to only one study, the very early report of Ziskind et al. (1945), which indicates a significant protective effect of ECT against suicide. The remainder of the evidence is overwhelmingly negative. It appears to us that the undeniable efficacy of ECT to dissipate depression and symptoms of suicidal thinking and behavior has generalized to the belief that it has long-range protective effects. In one sense, it is reassuring that this very effective somatic therapy does not exert long-reaching influences on future behavior; in another, it is disappointing that it does not.

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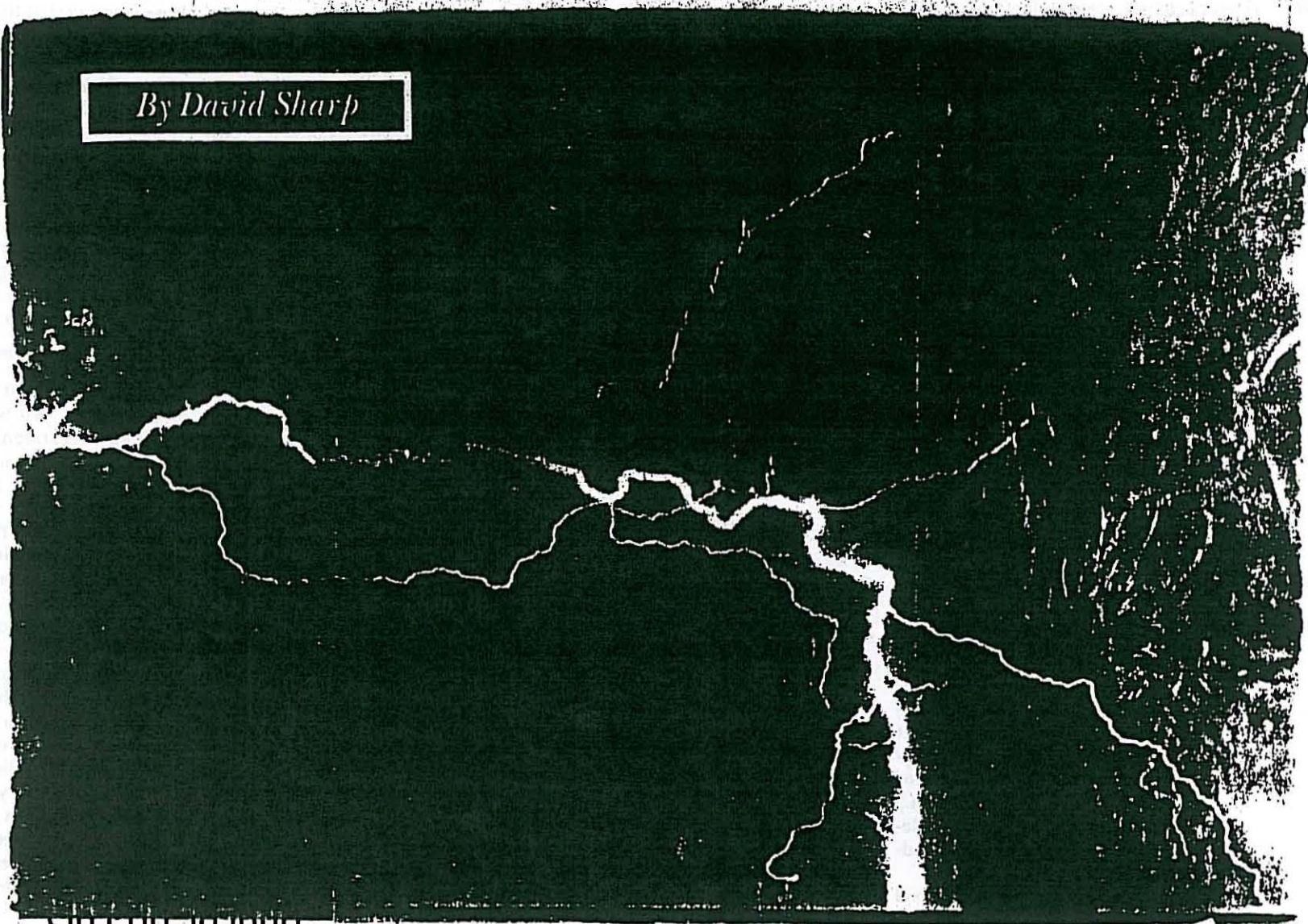
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A BOLT FOR THE BLUES

Is it safe? Does it work? A personal encounter with electric shock treatment

By David Sharp



DAVID SHARP (7)

SPECIAL REPORT

NOVEMBER 1991—JANUARY 1992

LAST FALL A 34-YEAR-OLD EAST COAST attorney we'll call Colleen Tate became one of the estimated 100,000 Americans a year who undergo modern medicine's most radical method of treating the severely depressed: electroconvulsive therapy, or ECT. During a four-week period beginning in late September, Tate re-

ceived eight electric shocks in an attempt to end her three-year bout with depression—the same malady that had plagued her mother for many years. In December, a neuropsychologist asked Tate a series of simple, largely biographical questions—including many of those that follow—as part of a battery of tests administered to gauge her overall mental condition. The news wasn't good. In his report the psychologist stated that Tate had performed "below expectations for someone of her educational and occupational attainment."

What's your trouble?

Tate first became depressed in 1987. No mere case of the blahs, the sense of hopelessness that afflicted her grew so all-encompassing that suicide seemed preferable to the bleaker prospect of living in permanent gloom. She was ready to do anything to escape what she thought of as "a dark hole with no way out." Finally, in September 1990, she decided to follow her psychiatrist's advice and try ECT.

Shock treatment was developed more than 50 years ago and has been used on such famous folk as

Ernest Hemingway, Jacqueline Onassis (if you believe the Kitty Kelley biography of her), and former Missouri senator Thomas Eagleton, whose well-publicized history of mental illness led to his resignation as a 1972 vice-presidential candidate. Over time the procedure has become so technically routine that many people, like Tate, get the treatments as outpatients, with not much more fuss than if they were going in for a tooth extraction. Even so, ECT maintains a low profile as medical procedures go, since most people who have it—fearing the social stigma of being labeled shock patients—aren't exactly eager to advertise the fact. Tate, a corporate trial attorney who graduated from law school with honors in 1986, had a relatively easy time keeping her appointments secret. She told her office colleagues that she'd be out sick for a month or so because of a thyroid condition.

Why did you come here?

Tate matched the textbook description of an ECT candidate in two key respects. First, she was experiencing prolonged suicidal depression; not just a transitory fit of low spirits; second, she'd gone through a medicine cabinet's worth of antidepressant drugs without success. In the view of some psychiatrists, ECT is the next logical step when pharmaceuticals fail. In fact, proponents of ECT claim a success rate of roughly 80 percent, though no one can really explain why it works. The simplest theory is that flooding the brain's 10 billion neurons with electricity is physiologically comparable

to fixing a loose connection in your TV by smacking the set with your fist. Whatever the reason, it's known that a trauma to the brain, whether induced by an electric current or a 12-car pileup, can leave the recipient feeling remarkably relaxed and comfortable. That's why nurses and orderlies have long joked that at any hospital the head-injury ward ranks with the maternity ward in having some of the most cheerful patients around.

What kind of place is this?

On her first visit for treatment, Tate lay stretched out on a hospital bed as an anesthesiologist slipped a needle into her arm. Moments later she was unconscious, her body as flaccid as an empty laundry bag, and a psychiatrist and a nurse went to work. The nurse held two electrodes shaped like miniature barbells against Tate's head; the psychiatrist adjusted the dials on a piece of electronic equipment about the size of a stereo receiver. Then he pushed a button on its face, and for one second the amount of electricity it takes to turn on a 100-watt light bulb pulsed through Tate's brain.

What month is this?

Tate spent the four weeks of treatment and several weeks afterward in a thick cloud of mental confusion, unable to think clearly or recall recent events—including the fact that she had been shocked. "Trying to remember things was like looking into muddy water," she says. Partial

amnesia is a common side effect of ECT, but experts disagree on how extensive the amnesia can be expected to be. ECT experts, in fact, fall into two contentious camps: those who say that the therapy's effects on memory are neither extensive nor permanent and those who believe that large chunks of a patient's past can be irreversibly wiped out—a risk Tate claims her psychiatrist hadn't mentioned. Two weeks after her last treatment, she felt the disorientation begin to lift. Even more encouraging, her depression had also disappeared. She looked forward to returning to the career she loved as soon as the remaining fuzziness cleared up.

Have you ever seen me before?

In November, just weeks after her last shock treatment, Tate's plans suddenly changed when she got a phone call at home. She didn't recognize the caller's voice, but the woman talked as if they knew each other. Politely playing along, Tate wondered, *Is this somebody I should remember?* Later she mentioned the puzzling conversation to her husband. He looked at her oddly and explained that the stranger on the phone had been the secretary at Tate's law office, a woman she'd worked with for several months.

After that Tate discovered other holes in her memory. Her husband showed her photos of a vacation they had taken together the previous year, but she recalled nothing about it. He reminded her that she had broken her ankle; Tate asked how it had happened. He described

Her husband showed her photos of a vacation they had taken together, but she recalled nothing about it. He reminded her that she had broken her ankle; Tate asked how it had happened.

the suicidal depression she had battled back from; she remembered little of it. Even worse, the memory loss was like a fire that had not only decimated the contents of her mental warehouse but also wiped out the inventory list. Once, thinking it would be safe to see someone she knew well from work, Tate went to lunch with a law colleague who had lost a lot of weight a few months earlier. She told him how good he looked, and in surprise he reminded her that he had been thin for months. Didn't she remember? "Oh, I guess I forgot," Tate answered and quickly changed the subject. Episodes like that one made her stop going anywhere on weekends without her husband, out of dread that she'd run into someone she knew but couldn't recall.

Who are you?

Her worst moment came when she tested her legal knowledge, only to discover that it too had been shredded. Law books she'd read meant nothing to her. The cases she'd worked on since 1989 were a blur. She had even forgotten how to

perform some of the most basic court procedures. Her legal mind was so muddled that her ability to practice law, and the self-esteem that went with it, were in shambles.

For an assembly-line worker or a toll-booth operator, such memory loss might well be a price worth paying to banish depression. But for Tate, losing the power to remember became the cerebral equivalent of an opera singer's losing her voice. "Maybe it's the lawyer's ego in me speaking," says Tate, "but everything I was is now gone."

Have you ever been here before?

Tate went back to her psychiatrist and told him about the huge gaps in her memory. His response was to blame the depression, not the ECT. The reason she couldn't remember some events in the past, he told her, was that she didn't *want* to remember them; her emotional despair had been that painful. Tate refused to accept this argument, particularly since she had had no memory problems before the treatments. Eventually she contacted other people who'd undergone shock therapy

and found that many of them too had forgotten large swaths of their lives—the people they'd met, the jobs they'd performed, the skills they'd mastered. She also learned one other fact that struck her like a two-by-four: although their lost memories sometimes didn't return, their depression often did.

If you could have one wish, what would it be?

Eight months after undergoing ECT, Tate has mixed feelings about the future. She refers to herself as a *former* trial attorney but clings to the hope that one day her memory will return at a level that will permit her to represent clients in court again. For now, she's considering a career in legal research, a field in which memory and reaction time don't make the difference between winning and losing a case. "It's easier for me to write and research than to take depositions and ask questions, because I can't think that quickly," she says. She has already stepped down from her job as a corporate trial attorney; she told her colleagues that she'd suffered se-

vere memory loss as a result of treatment for the thyroid condition she had told them about earlier.

Tate has also begun taking antidepressants again. She takes them in hopes that they, along with the lingering effects of last year's ECT, will keep her old gloom at bay. "Whenever I notice I'm unhappy—just normally unhappy—I'm on full alert to monitor myself," she says. She has also thought through her options in case the depression does return. Colleen Tate, once a woman who would do anything to escape her emotional dark hole, makes a different vow today. "I would never have ECT again," she says. "I know that it was beneficial in some respects. I'm no longer depressed; I can have fun now. But I'm a totally different person."

Few things in life are worse than losing your ability to recover from depression. But one of them, Tate now believes, is losing yourself. ★

DAVID SHARP, a contributing editor of this magazine, lives in Norris, Tennessee.

Can ECT Permanently Harm the Brain?

Donald I. Templer and David M. Veleber
Clinical Neuropsychology (1982) 4(2): 62-66

Literature relevant to the question of whether ECT permanently injures the brain was reviewed. Similar histological findings of epileptics and patients who had received ECT were discussed. Experimental research with animals seems to have demonstrated both reversible and nonreversible pathology. Psychological test findings, even when attempting to control for possible pre-ECT differences, seem to suggest some permanent cognitive deficit. Reports of spontaneous seizures long after ECT would appear to point to permanent brain changes. Human brain autopsies sometimes indicate and sometimes do not indicate lasting effects. It was concluded that vast individual differences are salient, that massive damage in the typical ECT patient is unlikely, and that irreversible changes probably do occur in some patients.

This review centers around five areas germane to the question of whether electroconvulsive therapy (ECT) causes permanent brain pathology. Relatively indirect evidence is provided by two of these areas, the brain condition of epileptics and the examination of animal brains after experimental ECT. The other three areas are psychological testing findings with history of many ECTs, spontaneous seizures, and autopsy findings. The review does not concern the extensive literature that shows that ECT temporarily impairs cognitive functioning. Such literature eventually shows impairment beginning with the first ECT and becoming progressively worse with succeeding treatments. Improvement occurs following the course of ECT, sometimes with the tested functioning actually being higher than the pretreatment level, which is presumed to have been impaired by psychopathology such as thought disorder and depression. Reviews of this literature can be found elsewhere (American Psychiatric Association, 1978; Campbell, 1961; Dornbush, 1972; Dornbush and Williams, 1974; Harper and Wiens, 1975), as can reviews indicating that the unilateral ECT (applied to the right side) in increasing usage in recent years causes less impairment than bilateral ECT (American Psychiatric Association, 1978; d'Elia, 1974; Hurwitz, 1974; Zamora and Kaelbing, 1965). This literature is really not very relevant to the central issue of our review. It has never been disputed that cognitive impairment occurs after ECT. Even the most fervent and excathedra defenders acknowledge that "temporary" impairment occurs. It is the issue of permanency that has been controversial.

THE BRAINS OF EPILEPTICS

It would seem that if an epileptic grand mal seizure produces permanent brain changes, then an electrically induced convulsion should also do so. In fact, inspecting the evidence

with respect to epileptics may provide us with a conservative perspective in regard to ECT since the latter could produce damage from the externally applied electrical current as well as from the seizure. Experimental research with animals has shown that the electric shocks (not to the head) produce more deleterious effects in the central nervous system than any other locality or system of the body. More pertinent are the studies of Small (1974) and of Laurell (1970) that found less memory impairment after inhalant induced convulsions than ECT. And, Levy, Serota and Grinker (1942) reported less EEG abnormality and intellectual impairment with pharmacologically induced convulsions. Further argument provided by Friedberg (1977) is the case (Larsen and Vraa-Jensen, 1953) of a man who had been given four ECTs, but did not convulse. When he died three days later, a subarachnoid hemorrhage was found in the upper part of the left motor region at the site where an electrode had been applied.

A number of post-mortem reports on epileptics, as reviewed by Meldrum, Horton, and Brierley (1974) have indicated neuronal loss and gliosis, especially in the hippocampus and temporal lobe. However, as Meldrum et al. pointed out, on the basis of these post-mortem reports, one does not know whether the damage was caused by the seizures or whether both were caused by a third factor intrinsic to the epilepsy. To clarify this issue, Meldrum et al. pharmacologically induced seizures in baboons and found cell changes that corresponded to those in human epileptics.

Gastaut and Gastaut (1976) demonstrated through brain scans that in seven of 20 cases status epilepticus produced brain atrophy. They reasoned that "Since the edema and the atrophy were unilateral or bilateral and related to the localization of the convulsions (unilateral or bilateral chronic seizures), the conclusion can be drawn that the atrophic process depends upon the epileptic process and not on the cause of the status."

A common finding in epileptics and ECT patients is noteworthy. Norman (1964) stated that it is not uncommon to find at autopsy both old and recent lesions in the brains of epileptics. Alpers and Hughes (1942) reported old and recent brain lesions associated with different series of ECT.

ANIMAL BRAINS

There are a number of articles concerning the application of ECT and subsequent brain examination in animals. In the 15 study review of Hartelius (1952), 13 of the 15 reported pathological findings that were vascular, glial or neurocytological, or (as was generally the case) in two or three of these domains. However, as Hartelius pointed out, inferences of these studies tended to be conflicting because of different methods used and because of deficient controls. The research that Hartelius himself carried out was unquestionably the outstanding study in the area with respect to methodological sophistication and rigor. Hartelius employed 47 cats; 31 receiving ECT, and 16 being control animals. To prevent artifacts associated with the sacrificing of the animals, the cerebrums were removed under anesthesia while the animals were still alive. Brain examinations were conducted blindly with respect to ECT vs. control of subject. On a number of different vascular, glial, and neuronal variables, the ECT animals were significantly differentiated from the controls. The animals that had 11-16 ECTs had significantly greater pathology than the animals that had received four ECTs. Most of the significant differences with respect to reversible type changes. However, some of the significant differences pertained to clearly irreversible changes such as shadow cells and neuronophagia.

PSYCHOLOGICAL TEST FINDINGS WITH HISTORY OF MANY ECTS

There have been several studies regarding the administration of psychological tests to patients with a history of many ECTs. Unfortunately, all were not well controlled. Rabin (1948) administered the Rorschach to six chronic schizophrenics with a history of from 110 to 234 ECTs. Three patients had 6, two had 4, and one had 2 Piotrowski signs. (Piotrowski regards five or more as indicating organicity.) However, control subjects were not employed. Perlson (1945) reported the case of a 27-year-old schizophrenic with a history of 152 ECTs and 94 Metrozol convulsions. At age 12 he received an IQ of 130 on the Stanford Achievement Test; at age 14 an IQ of 110 on an unspecified general intelligence test. At the time of the case study, he scored at the 71st percentile on the Otis, at the 65th percentile on the American Council on Educational Psychological Examination, at the 77th percentile on the Ohio State Psychological Examination, at the 95th percentile for engineering freshman on the Bennett Test of Mechanical Comprehension, at the 20th percentile on engineering senior norms and at the 55th percentile on liberal arts students' norm on a special perception test. These facts led Perlson to conclude that convulsive therapy does not lead to intellectual deterioration. A more appropriate inference would be that, because of the different tests of different types and levels and norms given at different ages in one patient, no inference whatsoever is justified.

There are two studies that provide more methodological sophistication than the above described articles. Goldman, Gomer, and Templer (1972) administered the Bender-Gestalt and the Benton Visual Retention Test to schizophrenics in a VA hospital. Twenty had a past history of from 50 to 219 ECTs and 20 had no history of ECT. The ECT patients did significantly worse on both instruments. Furthermore, within the ECT groups there were significant inverse correlations between performance on these tests and number of ECTs received. However, the authors acknowledged that ECT-caused brain damage could not be conclusively inferred because of the possibility that the ECT patients were more psychiatrically disturbed and for this reason received the treatment. (Schizophrenics tend to do poorly on tests of organicity.) In a subsequent study aimed at ruling out this possibility, Templer, Ruff, and Armstrong (1973) administered the Bender-Gestalt, the Benton, and the Wechsler Adult Intelligence Scale to 22 state hospital schizophrenics who had a past history of from 40 to 263 ECTs and to 22 control schizophrenics. The ECT patients were significantly inferior on all three tests. However, the ECT patients were found to be more psychotic. Nevertheless, with degree of psychosis controlled for, the performance of the ECT patients was still significantly inferior on the Bender-Gestalt, although not significantly so on the other two tests.

SPONTANEOUS SEIZURES

It would appear that if seizures that were not previously evidenced appeared after ECT and persisted, permanent brain pathology must be inferred. There have been numerous cases of post-ECT spontaneous seizures reported in the literature and briefly reviewed by Blumenthal (1955, Pacella and Barrera (1945), and Karliner (1956). It appears that in the majority of cases the seizures do not persist indefinitely, although an exact perspective is difficult to obtain because of anticonvulsant medication employed and the limited follow-up information. another difficulty is, in all cases, definitively tracing the etiology to the ECT, since spontaneous seizures develop in only a very small proportion of patients given

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The first part of the document is a letter from the President of the United States to the Congress, dated January 1, 1861. The letter is signed by James Buchanan and is addressed to the Senate and House of Representatives. The letter discusses the state of the Union and the recent events leading to the secession of the Southern States. The President expresses his regret over the secession and his hope for a peaceful resolution of the crisis. The letter also discusses the President's actions to maintain the Union and his belief in the Constitution. The letter is a significant historical document that provides insight into the President's perspective on the crisis at the time.

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this treatment. Nevertheless, the composite of relevant literature does indicate that, at least in some patients, no evidence of seizure potential existed before treatment and post-ECT seizures persist for years.

An article that is one of the most systematic and representative in terms of findings is that of Blumenthal (1955) who reported on 12 schizophrenic patients in one hospital who developed post-ECT convulsions. Six of the patients had previous EEGs with four of them being normal, one clearly abnormal, and one mildly abnormal. The patients averaged 72 ECTs and 12 spontaneous seizures. The time from last treatment to first spontaneous seizure ranged from 12 hours to 11 months with an average of 2 and 1/2 months. The total duration of spontaneous seizures in the study period ranged from 1 day to 3 and 1/2 years with an average of 1 year. Following the onset of seizures, 8 of the 12 patients were found to have a clearly abnormal, and 1 a mildly abnormal EEG.

Mosovich and Katzenelbogen (1948) reported that 20 of their 82 patients had convulsive pattern cerebral dysrhythmia 10 months post ECT. None had such in their pre-treatment EEG. Nine (15%) of the 60 patients who had 3 to 15 treatments, and 11(50%) of the 22 patients who had from 16 to 42 treatments had this 10 month posttreatment dysrhythmia.

HUMAN BRAIN AUTOPSY REPORTS

In the 1940s and 1950s there were a large number of reports concerning the examination of brains of persons who had died following ECT. Madow (1956) reviewed 38 such cases. In 31 of the 38 cases there was vascular pathology. However, much of this could have been of a potentially reversible nature. Such reversibility was much less with the 12 patients who had neuronal and/or glial pathology. The following are the comments pertaining to the neuronal and glial pathology and the amount of time between last treatment and death: "Gliosis and fibrosis" (5 months); "Small areas of cortical devastation, diffuse degeneration of nerve cells", "Astrocytic proliferation" (1 hour, 35 minutes); "Small areas of recent necrosis in cortex, hippocampus and medulla", "Astrocytic proliferation" (immediate); "Central chromatolysis, pyknosis, shadow cells (15 to 20 minutes); "Shrinking and swelling, ghost cells", "Satellitosis and neuronophagia" (7 days); "Chromatolysis, cell shrinkage". "Diffuse gliosis, glial nodules beneath the ependyma of the third ventricle" (15 days); "Increased Astrocytes" (13 days); "Schemic and pyknotic ganglion cells" (48 hours); "Pigmentation and fatty degeneration, sclerotic and ghost cells", "Perivascular and pericellular gliosis" (10 minutes); "Decrease in ganglion cells in frontal lobes, lipid pigment in globus pallidus and medial nucleus of thalamus", "Moderate glial proliferation" (36 hours); "Glial fibrosis in marginal layer of cortex, gliosis around ventricles and in marginal areas of brain stem, perivascular gliosis in white matter" (immediate); "Marginal proliferation of astrocytes, glial fibrosis around blood vessels of white matter, gliosis of thalamus, brain stem and medulla" (immediate). In one case the author (Riese, 1948), in addition to giving the neuronal and glial changes, reported numerous slits and rents similar to that seen after execution. Needless to say, patients who died following ECT are not representative of patients receiving ECT. They tended to be in inferior physical health. Madow concluded, on the basis of these 38 cases and 5 of his own, "If the individual being treated is well physically, most of the neuropathological changes are reversible. If, on the other hand, the patient has cardiac, vascular, or renal disease, the cerebral changes, chiefly vascular, may be permanent."

CONCLUSION

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A wide array of research and clinical based facts that provide suggestive to impressive evidence in isolation, provide compelling evidence when viewed in a composite fashion. Some human and animal autopsies reveal permanent brain pathology. Some patients have persisting spontaneous seizures after having received ECT. Patients having received many ECTs score lower than control patients on psychological tests of organicity, even when degree of psychosis is controlled for.

A convergence of evidence indicates the importance of number of ECTs. We have previously referred to the significant inverse correlations between number of ECTs and scores on psychological tests. It is conceivable that this could be a function of the more disturbed patients receiving more ECTs and doing more poorly on tests. However, it would be much more difficult to explain away the relationship between number of ECTs received and EEG convulsive pattern dysrhythmia (Mosovich and Katzenelbogen, 1948). No patients had dysrhythmia prior to ECTs. Also difficult to explain away is that in Table I of Meldrum, Horton and Brierley (1974), the nine baboons who suffered brain damage from experimentally administered convulsions tended to have received more convulsions than the five that did not incur damage. (According to our calculations, $U=9$, $p < .05$) And, as already stated, Hartelius found greater damage, both reversible and irreversible, in cats that were given 11 to 16 than in those given 4 ECTs.

Throughout this review the vast individual differences are striking. In the animal and human autopsy studies there is typically a range of findings from no lasting effect to considerable lasting damage with the latter being more of the exception. Most ECT patients don't have spontaneous seizures but some do. The subjective reports of patients likewise differ from those of no lasting effect to appreciable, although usually not devastating impairment. The fact that many patients and subjects suffer no demonstrable permanent effects has provided rationale for some authorities to commit the non-sequitur that ECT causes no permanent harm.

There is evidence to suggest that pre-ECT physical condition accounts in part for the vast individual differences. Jacobs (1944) determined the cerebrospinal fluid protein and cell content before, during, and after a course of ECT with 21 patients. The one person who developed abnormal protein and cell elevations was a 57-year-old diabetic, hypertensive, arteriosclerotic woman. Jacobs recommended that CSF protein and cell counts be ascertained before and after ECT in patients with significant degree of arteriosclerotic or hypertensive disease. Alpers (1946) reported, "Autopsied cases suggest that brain damage is likely to occur in conditions with pre-existing brain damage, as in cerebral arteriosclerosis." Wilcox (1944) offered the clinical impression that, in older patients, ECT memory changes continue for a longer time than for younger patients. Hartelius (1952) found significantly more reversible and irreversible brain changes following ECT in older cats than younger cats. Mosovich and Katzenelbogen (1948) found that patients with pretreatment EEG abnormalities are more likely to show marked post-ECT cerebral dysrhythmia and to generally show EEGs more adversely affected by treatment.

In spite of the abundance of evidence that ECT sometimes causes brain damage, the Report of The Task Force on Electroconvulsive Therapy of the American Psychiatric Association (1978) makes a legitimate point in stating that the preponderance of human and animal autopsy studies were carried out prior to the modern era of ECT administration that included anesthesia, muscle relaxants, and hyperoxygenation. In fact,

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animals which were paralyzed and artificially ventilated on oxygen had brain damage of somewhat lesser magnitude than, although similar patterns as, animals not convulsed without special measures. (Meldrum and Brierley, 1973; Meldrum, Vigourocex, Brierley, 1973). And it could further be maintained that the vast individual differences stressed above argue for the possibility of making ECT very safe for the brain through refinement of procedures and selection of patients. Regardless of such optimistic possibilities, our position remains that ECT has caused and can cause permanent pathology.

1. The first part of the paper discusses the importance of the study of the history of the United States. It is argued that a knowledge of the past is essential for a full understanding of the present and for the development of a sound policy for the future. The author points out that the study of history is not only a means of acquiring knowledge, but also a means of developing a sense of responsibility and a sense of civic duty.

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SPECIAL ISSUE

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Challenging the Therapeutic State, Part Two:

Further Disquisitions on the Mental Health System

David Cohen, Editor
Université de Montréal

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ECT: Sham Statistics, the Myth of Convulsive Therapy, and the Case for Consumer Misinformation

Douglas G. Cameron

World Association of Electroshock Survivors

This paper emphasizes that, contrary to the claims of ECT experts and the ECT industry, a majority, not "a small minority," of ECT recipients sustain permanent memory dysfunction each year as a result of ECT. The paper exposes the convulsion hypothesis, upon which ECT is allegedly based, as mythological. Finally, through hidden and comparative electrical parameters, it exposes the extreme destructive power of today's "new and improved" ECT devices.

The purpose of this paper is threefold: to identify misleading or false information on memory damage disseminated by electroconvulsive/electroshock therapy (ECT/EST) device manufacturers as well as by the American Psychiatric Association (APA); to provide historical and mathematical proof that convulsive therapy is a myth; and to show that modern ECT/EST devices are much more powerful, not less powerful, than ECT/EST devices of the past.

ECT is the passage (for 0.1 up to 6 seconds), usually from temple to temple through the frontal lobes, of electric current, for the purpose of inducing therapeutic "grand mal" convulsions. Follow-up studies about the effects of ECT in which recipients themselves evaluate the procedure are both rare and embarrassing to the ECT industry. The outcomes of these studies directly contradict propaganda regarding permanent memory loss put forth by the manufacturers of ECT devices in the United States (Somatics, MECTA, cor, and Medcraft), upon whom physicians and the public rely for information, much as the public relies upon pharmaceutical companies for information on drugs.

Requests for reprints should be sent to Douglas G. Cameron, World Association of Electroshock Survivors, P.O. Box 141, San Marcos, Texas 78667

One of the first and best prospective follow-up studies on ECT recipients was conducted over 40 years ago by Irving Janis (1950). He merely asked ECT recipients personal, mainly biographical questions before they underwent ECT, then again several weeks and months later. In all cases, whether or not the recipients themselves recognized memory loss, they had forgotten much of their personal history. Unpublished conversations with many of Janis' patients six months or one year later (Davies, Detre, and Egger, 1971) led him to conclude the memory loss was long-term, perhaps permanent.^{1,2} This is just as the majority of patients have claimed since ECT's inception in 1938 (Brody, 1944; Brunschwig, Strain, and Bidder, 1971; Squire and Slater, 1983).

Few other similar studies were performed until Freeman and Kendell's (1980) investigation. In the meantime, doctors (not patients) concluded that ECT was "successful" and provided "marked improvement" with "minimal side-effects" (Bender, 1947; Chabaszinski, 1978). Freeman and Kendell's study was prompted by patients who, on BBC radio, described ECT as the most fearful and terrifying experience of their lives. Freeman and Kendell set out to prove that patients were "unafraid" of the treatment. They recounted the following:

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We were surprised by the large number who complained of memory impairment [74%]. Many of them did so spontaneously, without being prompted, and a striking 30 percent felt that their memory had been permanently affected. (1980, p. 16, italics added)

In this study, shock survivors were "invited" back to the same hospital where they had been shocked and many were interviewed by the same doctor who had shocked them. Some of these persons, when asked if they were afraid of the treatment, might have been reticent to admit the treatment was indeed frightening. Even the authors acknowledge this intimidation factor: "It is obviously going to be difficult to come back to a hospital where you have been treated and criticize the treatment that you were given in a face-to-face meeting with a doctor What is less certain is whether there was a significant number of people in the midground who felt more upset by ECT than they were prepared to tell us" (1980, p. 16). In any case, almost a full third did complain of permanent memory loss: an astonishing number considering the circumstances.

¹Years after Janis' 1950 study, Marilyn Rice (see below) contacted Irving Janis, and in a personal telephone interview, Janis explained how, one year later, he had followed up his 1950 study (unpublished) and how its results appeared reliable.

²Only Squire, Slater, and Miller (1981, p. 93) have repeated the Janis prospective study. Even after two years, and even with reminder cues, 50% of the ECT recipients in this study still could not recall specific autobiographical events spontaneously recalled before ECT. This does not preclude the possibility that autobiographical events which could be "remembered" after two years, might simply have been re-learned rather than recalled.

Squire and his colleagues conducted what are perhaps the best known studies on ECT and memory loss. Squire and Slater (1983) report that "55% felt that their memories were not as good as those of other people of the same age and that this was related to their having received ECT" (p. 5). The average reported memory loss was 27 months' duration for the entire group, and for the 55% who felt they had sustained injury, it was 60 months. Using various cognitive tests, Squire and Slater could not "find" evidence for the latter figure, but they estimated an "authentic" average eight month gap in memory even after three years. Squire (1986, p. 312) also conceded that his tests may not have been sensitive enough.

Both Janis and Squire concluded that 100% of ECT recipients they tested sustained at least some permanent memory loss, even though some patients denied such loss. Squire's "authentic eight month gap" after three years was that reported by the 55% in their study who felt ECT had damaged their memory. Interestingly, after three years, the 45% who felt ECT had not injured their memories reported an even larger average persistent gap, of 10.9 months (Squire and Slater, 1983). A control group of depressed patients reported a five month gap as a result of depression alone. None was administered ECT, and no one in the group reported any gap in memory three years later (In fact, control subjects' memories had cleared only a few months into the experiment). Consequently, Squire and Slater concluded that there existed some actual permanent memory gap as a result of ECT, even for ECT recipients denying such an effect.³

The Committee For Truth In Psychiatry, founded by Marilyn Rice in 1984, includes approximately 500 ECT survivors in the United States, who suffer from permanent memory loss as a direct result of ECT. The Committee has the sole aim of convincing or forcing mental health authorities to give truthful informed consent regarding ECT.⁴

³That Squire and Slater selected the permanent gap to be the smaller one may indicate bias. Also, after three years, the larger gaps originally reported may only have appeared reduced (e.g., to eight and 10.9 months). Squire and Slater's conclusion that 100% of their subjects suffered an ECT induced average eight month permanent gap in memory is unquestionably the most conservative conclusion one may draw from their data. In any case, both studies indicate that patients under-report rather than over-report treatment induced permanent memory loss.

⁴Larry Squire himself administered Marilyn Rice a battery of cognitive tests as part of a malpractice suit she brought, in which she charged that years of her memory were permanently erased by ECT (Squire was hired by the defense). In a personal interview with the author, she related that she passed all of Squire's tests easily and in fact, regarded them as absurd. Throughout her lifetime, Marilyn contended that eight shock treatments had eliminated, in addition to treasured personal memories, all the mathematical and cumulative knowledge of her twenty years with the Department of Commerce in Washington D.C., where she co-ordinated vital statistics and activities concerning the National Budget (Frank, 1978). In spite of her claims, the results of Squire's tests were successfully used in court to prove her memory "intact" and she lost her malpractice suit. Rice, who died in 1992, lobbied the Food and Drug

Misinformation from the ECT Manufacturers

An insidious source of misinformation about ECT's effects on memory are videotapes marketed by some of the ECT device manufacturers (Somatics, MECTA) and made available to patients, family members, and shock facility professionals in the United States and Canada. There are no disclosures in these videos identifying either Somatics or MECTA as manufacturers of ECT devices (Fink, 1986; Grunhaus, 1988).

MECTA's (1987) video for professionals, *Health Information Network*, features a panel of "experts," Richard Weiner of Duke University, Harold Sackheim of New York State Psychiatric Institute, and Charles Welch of Harvard Medical School, each interviewed in turn. Welch says: "I tell my patients they may experience a temporary loss of memory during the time they're having the treatments and for several weeks after that." In another MECTA video designed for individuals and family members, the narrator is slightly more honest: "[W]e know that 80 to 90 percent of the patients who received bilateral ECT will report that their memory has recovered within 3 to 6 months after the treatment, while 10 to 20 per cent may report a change in the quality of the memory" (Grunhaus, 1988).

Another educational video prepared by Somatics features Max Fink (1986), leading proponent of ECT in the United States. Fink states:

The usual thing that patients complain about and the family complains [about] is the patient has a loss of memory and that occurs in every patient. Every patient has a loss of memory for the treatment itself . . . Now when we give a patient treatment over three or four weeks they tend to have a fuzzy idea of what happened in the hospital. But [other than] the treatments themselves, the patients do not forget what happened in their early life, they don't forget what happened in their childhood, they don't forget the telephone, they don't forget the names of their children, they don't forget their work, and they have no difficulty in learning these things after the treatment is over when they're better . . . Now some doctors and some people have said "Well, electroshock erases the mind and it's like erasing a blackboard." That's nonsense. If there is any erasure, it is for the events during the hospital. In many ways we're very grateful that patients forget that. After all, it's not a pleasant time of your life. For a depressed patient to be in the hospital, it's not pleasant and if they forget that, that's fine.

Administration (FDA) and state legislatures to mandate warnings of permanent memory loss and brain damage. Her influence on state legislatures may have been demonstrated by the recent 1993 Texas legislation, S.B. 205, which mandates a fresh signature by the patient and a fresh discussion with the patient on the "possibility of permanent irrevocable memory loss" before each individual treatment (not series) [see Cameron, 1994].

Misinformation from the American Psychiatric Association

In 1990, the APA published recommendations from an ECT Task Force aimed at specifying the "standard of care" regarding the administration of ECT throughout the United States (APA Task Force, 1990). Weiner, Fink, and Sackheim, who appear on the previously mentioned MECTA and Somatics videos, are three of the six members of the Task Force. Fink has admitted in a court deposition to receiving royalties from videos created and marketed by Somatics (*Aubrey vs Johns Hopkins Hospital*, 1991). Psychiatrist Richard Abrams, the most frequently referenced author in the Task Force Report, owns Somatics (Breggin, 1992, p. 13). Psychiatrist Barry Maletzky, one of the authors cited in the Report, is viewed in one MECTA video "pitching" that company's device to potential purchasers (Maletzky, 1987). Numerous videos, books and brochures created or marketed by these companies are mentioned in the appendix of the Task Force Report. The names and addresses of all four ECT device manufacturers are also listed. The APA Task Force Report on ECT might more appropriately be deemed *The Manufacturers' Task Force Report on ECT*.⁵

In a sample informed consent form appended to the Task Force Report, the following statement (which has appeared in numerous scientific and professional articles) appears: "A small minority of patients, perhaps 1 in 200, report severe problems in memory that remain for months or even years" (APA, 1990, p. 158; Foderaro, 1993, p. A16). The number, however, has unclear origins. This author located only two "one in 200" estimates in the ECT literature. One mention comes from a book by Fink (1979, p. 52), who states:

Spontaneous seizures are a rare manifestation and may be considered evidence of persistent altered brain function. From a review of various reports, I estimate that post ECT organic syndrome, including amnesia and tardive seizures to persist in one in 200 cases.

Fink provides no specific references or data for his estimate.⁶ Even so, the figure again appears in the appendix of his book, in a sample of informed consent (p. 221). The other "one in 200" estimate this author located comes from an Impastato (1957) study, but rather than citing cases of permanent memory loss, Impastato is citing the death rate for ECT recipients over 60

⁵The APA apparently gathered most of its facts from the device manufacturers or those closely connected with the products; in turn, the FDA obtained most of its information from the APA (APA, 1990; FDA 1990).

⁶Fink's unsubstantiated statistic was brought to my attention by shock survivor Linda Andre, Director of Committee For Truth In Psychiatry.

years of age. Another inaccurate statement in the Task Force Report was noted by Breggin (1992, p. 14). Citing the Freeman and Kendell (1980) study, the Report states that "a small minority of patients" report persistent deficits. Unless 30% is a small minority, the APA is misinforming the public.

One finding stands out from follow-up studies, including those without conspicuous intimidation factors (Brunschwig, Strain, and Bidder, 1971; Janis, 1950; Small, 1974; Squire, 1986; Squire and Chace, 1975; Squire and Slater, 1983): a majority of subjects continue to believe they were permanently injured due to ECT. The "small minority" statistic put out by the ECT industry, by the APA, and further emulated by the FDA, has no factual basis.

Patients' claims of years of permanent memory erasure as a result of ECT, then, are invalidated by "cognitive tests." Squire and Slater's (1983) estimate of an "authentic" eight month memory gap is transformed by manufacturers into "memory changes of events prior to, during, and immediately following the treatment" (MECTA Corporation, 1993, p. 84). Unfortunately, phrases similar to these by the manufacturers, which suggest that memory loss is narrowly restricted, have come to be regarded as sufficient by numerous state Medical Disclosure Panels. Consequently, potential patients clearly receive inadequate information regarding memory loss and ECT as part of informed consent (see, for example, Texas Department, 1993, p. 2; Texas Medical Disclosure Panel, 1993, p. 14). As has been shown, more persons (the majority of ECT recipients) are convinced they are suffering permanent memory dysfunction as a result of ECT, and the memory gap is much wider (at least 8 months) than is currently reported or implied within their various informed consent protocols by the manufacturers of ECT devices, the APA, and various mental health authorities. Past and potential ECT recipients were and are being grossly misinformed.

The Myth of Convulsive Therapy

It has now become fashionable to declare brain damage from ECT a thing of the past because of "new refinements" in the procedure and in the machines (Coffey, 1993; Daniel, Weiner, and Crovitz, 1983; Foderaro, 1993; Kellner, 1994; Weiner, Rogers, and Davidson, 1986a). Breggin (1979, 1991) has debunked these "new and improved" claims, yet it appears that the strongest arguments in favor of ECT are the "new and improved" brief pulse machines. The implication that the sine wave device of old has been replaced by the brief pulse device of present lurks behind much of the continued use of ECT. The remainder of this paper shall examine the "new and improved" brief pulse device in light of the original aim and purpose of ECT.

Von Meduna introduced the concept of convulsive therapy in the 1930s (see von Meduna, 1938; Mowbray, 1959). He believed that a "therapeutic" or "anti-schizophrenic" effect could be obtained from the chemical induction of

grand mal seizures. In 1938, Cerletti and Bini introduced electroshock treatment (EST), or convulsions induced without chemicals. The convulsion appeared to be eliciting what later came to be described as an "anti-depressant effect" (Alexander, 1953, p. 61). While "patients" were at first intimidated and terrified, after a series of ECT they appeared more co-operative, docile, apathetic, or in some cases even cheerier toward their physician. These "improvements" (as short-lived then as now), appeared to validate von Meduna's convulsion theory.

From the onset, the treatment also produced severe memory problems, openly acknowledged as brain damaging effects by any of a myriad of published papers during that era (Brody, 1944; Ebaugh, Barnacle, and Neuburger, 1942; Sakel, 1956; Salzman, 1947). At the time, both the "anti-depressant" effect and the memory dysfunction were attributed to the convulsion. Gaining almost instant popularity among European psychiatrists, the machine was soon introduced into the United States, and by 1950 as many as 175,000 people annually may have been administered enforced ECT (Cohen, 1988; Robie, 1955).

A handful of professionals rejected the idea of brain damage as treatment (Delmas-Marsalet, 1942; Liberson, 1946; Wilcox, 1946; Will, Rehfeldt, and Newmann, 1948). One of them was Paul H. Wilcox, who by 1941 had concluded that the "therapeutic" effect of EST could be successfully separated from its brain damaging effects (Alexander, 1953, pp. 61-62; Friedman, Wilcox, and Reiter, 1942, pp. 56-63). Wilcox's own theory of electrostimulation challenged Meduna's theory. According to Wilcox (1946, 1972), perhaps it was simply electric stimulation of the brain which created the anti-depressant effect. Providing the correct dosage of non-convulsive electrical stimulation to the brain might elicit the therapeutic effects without the brain damaging convulsion.

This "non-convulsive therapy" failed to elicit the "therapeutic" effect (Impastato, 1952). However, in his quest to determine the ideal electrical dosage, Wilcox discovered that the strength of an electrically induced grand mal seizure did not depend upon any more electricity than that required to induce the seizure (Alexander, 1953, p. 64; Sulzbach, Tillotson, Guillemin, and Sutherland, 1943, p. 521). This meant that "adequate" convulsions could be induced with much lower dosages of electricity than had previously been used, and that the Cerletti-Bini devices were utilizing much more electricity than needed to induce such convulsions (Friedman, 1942, p. 213). Cerletti and Bini's device, then, was not an electroconvulsive device, but an electroshock device.

Wilcox reasoned that even if convulsions were necessary for the "anti-depressant" effect, by inducing convulsions with the least electricity dosage possible, side effects might be reduced or eliminated (Friedman et al., 1942;

Impastato, Frosh, and Robertiello, 1951). Wilcox set out to build the first "true" ECT machine, which he completed in 1942 (see Friedman, 1942). By ECT Wilcox meant electrically induced "adequate" grand mal convulsions; utilizing electrical dosage minimally above seizure threshold.⁷

To build his machine, Wilcox collaborated with an electrical engineer named Reuben Reiter. Following Wilcox's instructions, Reiter first operationalized Wilcox's minimal dosage concept into a direct current (DC) device, as opposed to the Cerletti-Bini alternating current (AC) device. The power of the new Wilcox-Reiter machine was thus immediately reduced by half. Wilcox was able to induce equal or "adequate" grand mal convulsions (of at least 25 seconds' duration) with his new machine, showing the Cerletti-Bini EST apparatus culpable of electrical overkill (Friedman, 1942, p. 218). The Wilcox-Reiter machine approached the challenge of threshold convulsions differently than other devices: from below rather than above threshold (Impastato, Berg, and Gabriel, 1957). The machine depended upon the cumulative effect of the electricity in order to induce a convulsion, at the first indications of which the current was immediately abated. Wilcox, Friedman, and Reiter turned the switch on and off manually as fast as possible during an application,⁸ which further reduced the current (Friedman, 1942, p. 219; Weiner, 1988, p. 57, Figure 3). Finally, in 1942, Wilcox and Friedman developed unilateral ECT (Alexander, 1953, p. 62; Friedman, 1942, p. 218), a method to reduce seizure threshold, allowing even more reductions in electrical dosage. That usually consists of placing one electrode on the temple and the other on top of the head so that a single frontal lobe of the brain is shocked. Unilateral ECT is often touted today as a "new and improved" methodology (Weiner, 1988, p. 59).

These methods and refinements greatly reduced the dosage of electricity required to induce an "adequate" convulsion. Wilcox now attributed memory loss and brain damage to such excess electricity (Alexander, 1953, p. 62). The Cerletti-Bini EST device utilized up to 125 volts of electricity and up to 625 milliamperes of current, compared to between 20 and 40 volts and an average of 40 milliamperes for the Wilcox-Reiter ECT device (Alexander, 1953, p. 62; Impastato et al., 1951, p. 5).

Correspondingly, the Wilcox-Reiter device greatly reduced, but did not eliminate, side effects. This was shown in EEG studies comparing the Wilcox-Reiter with the Cerletti-Bini. For example, Wilcox (1946) and others

(Liberson, 1949; Proctor and Goodwin, 1943) found a positive relationship between electrical dosage and abnormal or slow brain wave activity and memory dysfunction. Brain damage and memory dysfunction did indeed appear to be more a product of electricity than of convulsion.

Weiner (1988) criticizes the early comparative EEG studies as compromised by the possible use of unilateral ECT and other variations. Still, the relationship between memory impairment, brain damage and electrical dosage has been corroborated by various early and more recent studies (Alexander and Löwenbach, 1944; Cronholm and Ottosson, 1963; Dunn, Giuditta, Wilson, and Glassman, 1974; Echlin, 1942; Essman, 1968; Gordon, 1982; Liberson, 1945a; Malitz, Sackheim, and Declina, 1979; McGaugh and Alpern, 1966; Reed, 1988; Squire and Zoukounis, 1986). Many of these studies compared the effects of electricity to those of other convulsive stimuli on brain tissue. The results implicated the electricity much more than the convulsion. Specific observations as a result of applying even sub-convulsive dosages of electricity to the brain include retrograde amnesia in animals (McGaugh and Alpern, 1966); constriction of arteries, arterioles, and capillaries passing through the meninges of the brain (Echlin, 1942); metabolic changes in the brain chemistry of animals (Dunn et al., 1974); permeability of the blood brain barrier (Aird, Strait, and Pace, 1956); and other evidence of brain damage or its effects. According to an APA Fact Sheet (1992) on ECT, spontaneous seizures, even lasting up to 90 minutes, do not cause brain damage. Breggin (1979, p. 118) also notes, in his review on electrical damage to the brain, that "although convulsions of all kinds can cause biochemical disturbances in the brain, experienced researchers in the field believe that a case has been made for the electrical current as the main culprit."

First Brief Pulse

Also in the early 1940s, another psychiatrist, W.T. Liberson, who accepted von Meduna's theory, was inspired by the Wilcox discoveries to devise yet another method by which to reduce electrical dosage. Liberson (1945b, 1946, p. 755) is credited with producing the first "brief pulse" (BP) ECT device, using a systematically and continuously interrupted current. Because of the interruptions, each pulse of electricity becomes briefer than standard sine wave (SW) or relatively non-interrupted "wall" current. A single standard SW is 8.33 milliseconds (msec) long, compared to 1.0 msec for a single standard BP. The Wilcox-Reiter DC device cut the number of waves in half compared to the Cerletti-Bini AC device. Liberson adopted Wilcox's previous modifications and introduced electronically systematic continuous interruptions in the current as well (not merely the less efficient manual interruptions introduced by Wilcox), so that each individual pulse now became briefer.

⁷Thus, the Americans Wilcox and Friedman, not the Italians Cerletti and Bini, produced the world's first ECT device. The experiment with reduced electrical current was repeated in France that same year (Delmas-Marsalet, 1942).

⁸In that sense, the Wilcox-Reiter ECT device should also be credited with being the first brief pulse device (see below).

For a time, Liberson's BP device was the one using the least electrical dosage and thus causing the least amount of memory damage (Alexander, 1953, p. 62; Liberson, 1945b, 1946, p. 755; Liberson and Wilcox, 1945). Both Wilcox's and Liberson's devices were ECT machines, in that their purpose and successful function was to induce constant strength grand mal convulsions with minimal dosages of electricity (Alexander, 1953, p. 64). However, could these new machines produce the same therapeutic or anti-depressant effect as the Cerletti-Bini devices? Did adequate convulsions without the higher electrical dosages still "work"? Would von Meduna's convulsion theory prove correct?

Brief Pulse Fails

Despite the advantages of the Liberson ECT device, physicians in clinical practice did not use it widely. Brief pulse devices may have been slightly more expensive to build. Also, the earliest BP device emitted such low electrical dosage that unconsciousness was sometimes induced by the convulsion rather than by the electricity. In these instances the ECT recipient remained conscious until the convulsion, resulting in even more apprehension than in unmodified (without anaesthesia) high dosage SW EST (Liberson, 1948, p. 30). The problem was corrected by a slight increase in the pulse width or by the utilization of sodium pentothal or both (Liberson, 1948, pp. 30, 35).⁹ Some psychiatrists believed fear to be a necessary dimension of the procedure and so increased apprehension may not have been a negative factor for physicians in using the device (Cook, 1940; Liberson, 1948, p. 37). However, most clinicians complained that the same anti-depressant effect attainable with high dosage EST devices could not be achieved with Liberson's low-current BP ECT device (Impastato et al., 1957, p. 381). Many psychiatrists were not convinced the treatment worked without the higher dosage of electricity and its accompanying side effects. In fact, since the treatment appeared less effective with reduced side effects, many practitioners held side effects to be desirable, an integral part of the treatment itself (Alexander, 1955).

Although Liberson claimed complete therapeutic success with his device, he soon began proposing more treatments per series — in fact, as many as thirty (Liberson, 1948, p. 38). Rationalizing, Liberson proposed "a relatively great number of BST (brief stimulus) treatments in order to 'consolidate' the therapeutic results . . . As [BP] treatments are not followed by as much organic disturbance as with the classical ones, one should be particularly

eager not to stop the treatments too early" (Liberson, 1948, p. 36). Liberson failed to explain why, if the "anti-depressant" effect was a product of the adequate convulsion, a greater number of individual treatments would be required.

As early as 1948 then, it was known that, even with potent seizures, the "anti-depressant" effect at low electrical dosages was simply not satisfactory.¹⁰ Liberson (1946, p. 755) must have understood that electricity was the true "therapeutic" agent, but rather than publish findings showing von Meduna's convulsion theory weakened considerably, he focused instead on making his BP ECT device "work." After calling for more and more treatments, he recommended longer doses of BP ECT (Liberson, 1945b), eventually marketing a machine which allowed the current to flow between the temples for a full five seconds (compared to between 0.5 and one second previously) [see Weiner, 1988, p. 59, Figure 6]. The Liberson device could no longer be called an ECT, but was now an EST device. Next, although Liberson had already increased the wave length duration from 0.3 to between 0.5 and one millisecond¹¹ (Weiner, 1988, p. 57), his newer BP model offered adjustable wave lengths from between 1.5 to two milliseconds. The current was eventually stepped up to between 200 and 300 milliamps and, finally, Liberson returned to AC — doubling the original power.

All these modifications, of course, defeated the original purpose of the BP experiment: to induce adequate seizures at just above threshold electrical dosage. But even as Liberson continued increasing the "anti-depressant" effect of his BP machines by augmenting the dosage of electricity in various ways, the machines still lacked the power of the original or newer Cerletti-Bini style EST devices. Physicians everywhere seemed to prefer the higher dosage machines for their greater effectiveness (Cronholm and Ottosson, 1963; Page and Russell, 1948). Eventually, Liberson stopped increasing the power of his own device any further.

No one, including Liberson, mentioned that the convulsion theory might have been shown false, that adequate convulsions by themselves did not appear to produce a "therapeutic" effect. Nor did anyone suggest that it was electroshock that psychiatrists preferred, not minimal dosage electroconvulsion at all. By the mid-1950s, the Liberson BP ECT series disappeared forever from the marketplace.

¹⁰One might argue that barbiturates prompted Liberson to enhance electrical components as seizure threshold increases with barbiturate use. While this might explain some increases in electrical parameters, it does not explain increased numbers of treatment nor does it explain the eventual abandonment of minimal stimulus devices both here and abroad (see below).

¹¹This initial increase in wave length was developed to induce unconsciousness in the "patient" through electricity rather than convulsion (Liberson, 1948, p. 30).

⁹Eventually, with the introduction of informed consent, all unmodified EST (without exception terrifying to recipients) was replaced with anesthetized EST. Fear associated even with modified EST continues to baffle practitioners today (Fox, 1991).

The Wilcox-Reiter Device

Just as Liberson originally adopted the Wilcox-Reiter modification of DC in lieu of AC, Wilcox and Reiter soon incorporated Liberson's electronic BP principle into their own device. Wilcox and Reiter held one additional advantage: a cumulative sub-convulsive technique culminating in just above threshold seizures. This allowed the Wilcox-Reiter devices to surpass even Liberson's BP in ability to induce grand mal convulsions with the least electricity possible. The Reuben Reiter Company (producer of the Wilcox-Reiter machine) continued to produce such ECT devices into the 1950s.

Even so, by 1953, it was apparent that the Wilcox-Reiter ECT "electro-stimulators" also began to decline in popularity and could not compete with the more powerful Cerletti-Bini style American EST machines (i.e., Radha, Lectra, and Medcraft). In December 1956, at the Second Divisional Meeting of the APA in Montreal, Canada, psychiatrist David Impastato¹² and his colleagues made this announcement:

These currents (unilateral currents of the previous Reiter machines) evoke convulsions after three to five or more seconds of stimulation. In view of this, we may call such convulsions threshold convulsions....The fracture rate is moderately reduced when these currents are used, but apnea, post-convulsive confusion and agitation and subsequent memory changes are greatly reduced. In spite of these advantages, the use of unidirectional currents has not found favor in all quarters because a number of observers feel that with these currents more treatments than with AC currents are needed to effectuate a remission or to quickly bring under control such abnormal behavior as unmanageable agitation and suicidal drives. The psychiatrist of this faith therefore continues to use the old AC current machines and makes the best of the undesirable side actions. (Impastato et al., 1957, p. 381)

This announcement was, in effect, the unprecedented concession that the Wilcox-Reiter experiment with ECT had failed; that adequate convulsion alone had not, according to clinicians everywhere, created the desired antidepressant effect Wilcox, Friedman, Reiter, and Liberson had hoped for, 15 years earlier. ECT had failed and EST had emerged victorious. Almost all manufacturers of the popular SW devices recognized the "adequate dosage" precept. The more powerful their machines became, the more "effective," and commercially successful.

There was at this time no FDA, no physician adverse effect reporting system, no psychiatric survivor led civil rights movement, no informed consent requirements. In short, there was no one but the ECT investigator him/herself to announce that ECT had failed and that EST was producing the desired effects. It remained only for the investigator to report that there was

¹²Impastato had introduced several of the earlier Wilcox-Reiter models and was probably an undeclared paid consultant to Reiter.

no possibility of administering EST without the damaging effects, as both the damage and the "therapeutic" effect appeared to be the result of suprathreshold dosages of electricity. But neither Wilcox, Friedman, nor Reiter made any such announcement. Rather than challenge colleagues who were damaging the brains of thousands of persons yearly, Wilcox and Reiter, after voicing half-hidden resentment through Impastato's announcement and publication (Impastato et al., 1957) [see footnote 12] against those who failed to use the safer unidirectional minimal current ECT devices, then allowed Impastato and colleagues to introduce the newest Wilcox-Reiter machine, the Molac II, a Cerletti-Bini style SW AC device, capable of administering convulsions many times over seizure threshold. This was, in effect, the first deliberately designed Wilcox-Reiter EST apparatus.

The Molac II was announced as having a superior feature over "old" Cerletti-Bini style machines, a millisecond of high voltage current (around 190 volts) in order to render the person unconscious before delivering two to three seconds of AC current at around 100 initial volts. Ironically, Impastato and colleagues, just before the announcement of the new Molac II, had railed against the side effects of the "classic Cerletti-Bini EST machine," attributing them to "excessive current used" (Impastato et al., 1957, p. 381). There was no reason to believe the current intensity of the new device was any lower and whereas the original Cerletti-Bini machine could administer current up to five tenths of a second, the new Molac II had no timer at all. The recommended duration of each treatment was between two and three seconds, but this was left completely up to the doctor's discretion. The black button could be held down indefinitely!

After designing the least dangerous machine in history, Wilcox and Reiter had now designed the most dangerous EST machine in history, completely discarding their minimal dosage, adequate convulsion precept of ECT. Ironically, the Impastato et al. (1957) paper ended by claiming that Molac II recipients tested on the "Proteus Maze" did no worse than those who had been treated with previous minimal dosage machines, a contradiction of everything Wilcox, Friedman, and Reiter stood for and had maintained for the previous 17 years. Since December, 1956, there have been no ECT devices produced in America. The same experiment ended similarly in Europe (see footnote 7).

The Case for Consumer Misinformation

In 1976, due to the actions of a California group of psychiatric survivors, Network Against Psychiatric Assault (NAPA), the psychiatric survivor movement scored a major victory (Hudson, 1978, p. 146). NAPA had attained for the state of California the first semblance of informed consent for EST in

the United States (perhaps the first semblance of informed consent anywhere for persons labeled "mentally ill"). At least 30 other states enacted similar rule changes within the next few years. Psychiatrists in state institutions had to begin asking patients if they wanted EST. In these institutions, where EST had been predominantly administered up to this time, shock was, for a period at least, largely abandoned. At about this time too, shock devices came under the scrutiny of the FDA. It was time for the shock industry to take a different approach.

Also in 1976, psychiatrist Paul Blachley helped launch an attempt to make shock respectable again in America. A major part of a campaign to alter and improve the now very negative image of shock came in the form of "new and improved" EST devices, specifically the resurgence of Liberson's BP machine. Blachley's new company, Monitbred Electro Convulsive Therapy Apparatus (MECTA), was soon followed by Somatics, Elcor, and Medcraft in producing the "safer wave form," or BP ECT, devices.¹³ With these newer devices, hospitals began, as standard procedure, to anaesthetize patients, the great majority of whom were now private hospital patients with insurance.

A recent *New York Times* article lauded the "modern" brief pulse models as "improved," and having modifications "like reduced doses of electricity" (Foderaro, 1993, p. A16). Recently, the television show *48 Hours* featured psychiatrist Charles Kellner of the Medical University of South Carolina, who regularly administers electric shock. Kellner (1994) stated: "Well, it's such a different treatment now that there's almost no comparison . . . It really is a different treatment now . . . Having the seizure is the therapeutic part of ECT; probably about one fifth of the electricity that was used in the old days . . ." Such claims are false or misleading: the new BP devices are neither lower stimulus nor lower current devices than the older, or even the newer, SW models.

All other electrical components being equal, simple unmitigated BP (systematic interruptions of SW current) does in fact lead to reduced electrical dosages. However, aware that convulsions alone, induced by simple BP, are ineffective, manufacturers of modern BP devices amplify all other electrical components in order to compensate for the interruptions. Therefore, modern "souped up" BP apparatuses re-equal the cumulative electrical charges of the

Cerletti-Bini style SW in every respect. For instance, 100 percent power of standard SW will emit the same 500 millicoulombs of electrical charge as 100 percent power of a modern BP machine such as Somatics Thymatron DG. While one would expect reduced charges with BP, in fact, the old standard SW, i.e., Medcraft's 1950 model, emits slightly less charge than the modern day BP Thymatron DG. This would not be possible without electrical compensation of BP devices.

This compensation is accomplished in the following ways:

(a) *The frequency is increased.* Frequency is the number of pulses of electricity per second flowing past a given point. Although sine waves are "wider" than brief pulses, they are emitted at a constant rate of 120 per second. In comparison, modern BP devices can emit up to 180 pulses per second of electricity (e.g., MECTA's SR-2 and JR-2), or up to 200 pulses (e.g., Elcor's MF-1000).

(b) *The current is increased.* Current can be defined as electron flow per second and is measured in amperes or milliamperes (mA). The "old" SW devices deliver between 500 and 600 mA of current. The "new" BP Thymatron DG by Somatics delivers 900 mA constant current, the MECTA SR/JR devices, 800 mA, and the Medcraft B-25 BP up to 1000 mA or one full ampere.

(c) *Duration is increased.* Duration is the amount of time the current flows through the brain. Maximum duration of modern BP machines is four to six times the maximum duration of the older SW models.

(d) *Wave lengths can be increased* in most modern BP devices. The Elcor MF-1000, for instance, has adjustable brief pulses from a typical one msec up to an atypical two msec. A standard SW is 8.33 msec.

(e) *Alternating current is used.* In spite of the fact that both Liberson and Wilcox utilized DC successfully to induce adequate grand mal convulsions, modern BP devices utilize AC.

Thus modern BP devices are made to equal the charge¹⁴ of SW devices in every consideration with respect to percent of energy utilized. In addition, they surpass the "older" SW machines in energy output (joules), or actual power emitted.¹⁵ The following electrical features account for this increase:

¹³Two companies (Medcraft and Elcor) continue to manufacture the older Cerletti-Bini style SW devices, both more powerful than Cerletti and Bini's original SW device renowned for brain damage and memory loss (Impastato et al., 1957) and upon which Wilcox and Liberson attempted to improve. Cerletti and Bini's original device emitted a maximum 120 volts for a maximum of 0.5 seconds. Medcraft's "modern" SW device, unchanged since its 1953 model, the BS 24 (now the BS 24 III), has a maximum potential of 170 volts and emits a current for up to one full second (Weiner, 1988, p. 56; Medcraft Corporation, 1984). Today's SW devices, as well as modern day BP devices, are EST devices.

¹⁴By charge is meant the cumulative amount of electricity which has flowed past a given point at the end of an electron transaction.

¹⁵Using a straight-forward mathematical formula, the power of the new brief pulse devices can be verified by calculating joules (or the more familiar "watts" as in a lightbulb), the measure of actual energy emitted (voltage is potential energy or power). All four companies (e.g., MECTA, 1991, p. 13) do list their devices as 100 joule maximums in all 4 brochures, but the manufacturers' calculations are based on a typical resistance of 220 ohms (ohms are the measure of resistance, here, of the skull and brain, to current flow). However, the true maximum joules or watts for all modern day BP devices is much higher than the estimate reported by

(a) Much higher voltages are utilized. For example, the Thymatron DG utilizes up to 500 volts; the MECTA SR/JR, up to 444 volts; the new Medcraft up to 325 volts; and the Elcot MF-1000 up to 500 volts. Compare this to between 120 volts maximum for the oldest sine wave models and 170 volts maximum for modern SW devices.

(b) Constant current and continually increasing voltages are properties of all modern BP devices. Constant current means that the current never fluctuates or descends. This unique feature of BP devices is accomplished by higher and increasing voltages, a characteristic not found in SW devices. The constant lower voltage in the latter results in gradually decreasing currents. Just as the resistance of a wooden wall can eventually slow down and overpower an electric drill, so the human skull gradually slows down current. Modern BP devices maintain a constant current of about one ampere throughout the full four to six seconds it is emitted, making these devices the most powerful in ECT/EST history.

The tremendous energy output of modern BP devices (see footnote 15), the best measure of the machine's potential destructiveness, is a well-kept manufacturer's secret. The modern day BP devices are more than four times as powerful as the older SW devices, and about two and a half times as powerful as modern day SW devices. In fact, today's "new and improved" BP device is over eight times more powerful than the original Cerletti-Bini device renowned for permanent memory loss and upon which Wilcox and Liberson attempted to improve. Modern day BP devices have not been shown to be cognitively advantageous to SW devices in any modern study, and the few studies which have claimed cognitive advantages with modern day BP could not be replicated by other researchers (see Squire and Zola-Morgan, 1986; Weiner, Rogers, and Davidson, 1986a, 1986b).

Conclusion

Contrary to the claims put forth by the four manufacturers of EST devices, the evidence reviewed in this paper clearly shows that the majority of EST

the manufacturers. For SW devices, the formula is: joules = volts x current x duration, or joules = current squared x impedance x duration. For BP devices, the formula is: joules = volts x current x the π x wave length x duration, or joules = current squared x impedance x the π x wave length x duration. All four manufacturers utilize the latter in lieu of the former formulas, deriving the 100 joule maximums for their BP machines. Utilizing the former formulas, however, which give us non-theoretical amounts, we find that the Thymatron DG BP is capable of emitting 250 joules or watts of electricity; the MECTA SR/JR BP models, 256 joules; the Medcraft B-25 BP, 273 joules; and the Elcot device even more. Compare these energy emissions with the following typical analogy: the standard SW device can light up a 60 watt light bulb for up to one second. (Modern SW devices can light up a 100 watt light bulb for up to one second.) Modern BP devices can light up the same 60 watt light bulb for up to four seconds.

recipients report damage as a result of EST. EST recipients — whether or not they report memory loss — do, in fact, sustain actual permanent memory loss, averaging at least eight months, as a result of the procedure.

Modern day BP devices are not "lower current" machines, as most proponents claim. Through electrical compensation, they equal SW devices in every respect, and emit far greater energy. The results of studies claiming cognitive advantages using modern day BP over SW have not been replicated. Any advantage of the original BP device has been attenuated in modern day devices.

Hundreds of studies conducted between 1940 and 1965 (Correllis and Meyer, 1954; Hartelius, 1952; Heilbrunn and Weil, 1942; McKegney and Panzetta, 1963; Quandt and Sommer, 1966) demonstrating brain damage have been criticized as "old." However, since that time, the machines have only become more powerful. Thus few studies are "old" or irrelevant.

Most experts agree that current, and not convulsion (APA, 1992; Breggin, 1979, pp. 114, 122; Dunn et al., 1974; Sutherland et al., 1974) is responsible for long-term memory loss and severe cognitive dysfunction. Von Meduna's "therapeutic convulsion" is a myth, convincingly disconfirmed by early minimal stimulus convulsion experiments. Memory dysfunction and the "therapeutic" effect — which appear to be products of electricity — may well be inextricably related.

All four manufacturers continue to claim their devices are convulsive therapy devices. Nevertheless, because some of the Wilcoxian principles of the past are being rediscovered today, and because the efficacy of threshold convulsions is questionable (APA Task Force, 1990, pp. 28, 86, 94), a few BP manufacturers and researchers who collaborate with the manufacturers have gained enough confidence to call for even more powerful electrical devices — under the unsubstantiated claim that BP suprathreshold dosages of electricity are safer than SW suprathreshold dosages (Glenn and Weiner, 1983, pp. 33-34; MECTA, 1993, pp. 13, 14; Sackheim, 1991). For instance, Gordon (1980) rediscovered the adequateness of grand mal convulsions administered at low electrical dosages. Gordon (1982) later reiterated that high doses of electricity cause irreversible brain damage. Unaware of the lost history, Gordon suggested using minimal stimulus machines to induce convulsions. Deakin (1983) responded that minimal stimulus machines would be misguided, alluding to Robin and De Tissera's (1982) important double-blind study which demonstrated that current is the factor in ECT efficacy — not convulsions.¹⁶ Sackheim, Declina, Prohovnik, Portnoy, Kanzler, and Malitz (1986)

¹⁶Ex-lobbyist Diann'a Loper, who suffers from severe grand mal epilepsy as a result of EST, worked on the passage of S.B. 205 in Texas. Her neurologist John Friedberg called Diann'a's seizures the worst he had witnessed. Even so, I noted Diann'a never suffered extensive long-term memory loss as a result of her seizures, but she had side effects exactly like those

and Sackheim (1987) published studies corroborating the relevancy of electrical dosage to efficacy, and Sackheim restated this theme in a lecture delivered in New York in 1992 (Sackheim, 1992). Today's manufacturers are quietly leaning away from von Meduna's convulsion theory, away from the concept of adequate convulsions at minimal dosage and toward an unobtrusive attempt to legitimize adequate or suprathreshold electrical dosages.¹⁷ These tendencies, coupled with the power of modern BP devices, should lead to re-appraisal of the devices world-wide.

Manufacturers may have parted from the convulsion theory exemplified by just above seizure threshold devices of the past, to what might be just above damage threshold devices of the present, and if not forced to stop and prove the safety of their devices (allowing for even more powerful machines), might be embarking upon just above agnosognosic threshold apparatuses of the future.

In summary, modern electric shock machine companies are attempting to redefine safety from the original convulsion concept of "just above seizure threshold" to "safer wave form." The Food and Drug Administration must re-scrutinize today's SW and BP devices, withdrawing their "grandfathered in" status under convulsive therapy devices. Because they utilize an entirely different principle, and because they are suprathreshold devices rather than convulsion-dependent devices, all modern day BP and SW EST device manufacturers must be required to prove machine safety to the Food and Drug Administration, prior to further utilization of new machines. All modern day SW and BP EST devices are more powerful than early instruments. Modern day BP suprathreshold devices have not proved safer than SW suprathreshold devices. Side effects have been convincingly identified as products of electricity. These facts warrant the elimination of all EST machines from the marketplace.

described by the manufacturers — temporary confusion, headache, temporary memory loss, and sometimes permanent loss of an event immediately surrounding (within minutes — not months) the seizure. On the other hand, as a result of EST, Diann'a has memory loss spanning years, as well as permanent memory retention problems. (My own experience with EST, resulting in permanent loss of both my high school and college educations, parallels Diann'a's and many thousands like us [Cameron, 1991]). Manufacturers typically describe the less egregious effects of epilepsy or convulsions when describing "side effects" of EST, characteristically ignoring the effects of the one factor not present in spontaneous seizures — the electricity. Diann'a (along with the author) is Director of World Association of Electroshock Survivors (WAES), which seeks to prohibit EST world-wide.

¹⁷This is best exemplified through unilateral ECT. Originally utilized by Wilcox and Friedman to induce the most minimal stimulus threshold seizures possible (Alexander, 1953, p. 62; Liberson, 1948, p. 12), unilateral ECT is used by modern manufacturers to induce the highest electrical dosages possible (Abrams and Swartz, 1988, pp. 28-29) in order to achieve efficacy.

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SEP 17 1991

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September 6, 1991

To whom it may concern:

I was hospitalized, voluntarily, from January 30 to February 20, 1991, during which I had eight electroshock treatments. The second treatment was with bilateral electrode placement; the others were all unilateral right-hemisphere electrode placement.

Why Choose ECT?

My reason for wanting electroconvulsive therapy was quite simple: I was out of other alternatives. The antidepressant drug that had worked for me most of the time for over a year--the only one of the various drugs I had tried that *did* work--was no longer working, and I had reached the maximum dose that my body could tolerate. Psychotherapy had been very helpful in many ways, but even my therapist, an excellent clinical psychologist, agreed that I needed immediate medical intervention at that point, and supported my decision to get ECT. Various "alternative" treatments and therapies had failed to provide any relief for me. And even the best community support systems reach their limits of effectiveness when the source of the problem is neurochemical malfunctions (regardless of etiology) that simply do not respond to psychosocial care.

I certainly wish there had been less risky and drastic options available. But there *wasn't* anything else for me to do--except kill myself, which of course was an option I held in reserve if it turned out that the ECT didn't work. It did work; it unquestionably saved my life.

I want to state at the outset, though, that I am not an apologist for ECT or for those who provide it. I know that ECT *can* be a safe and effective treatment for many people--and that it can have very serious adverse effects for some of us. Most medical research concludes that prolonged or severe adverse effects are very rare, but for those of us who do experience them--whether we represent one out of two or one out of two million ECT recipients--they are very real, debilitating, and even life-threatening. I also see some critical problems with current practices in the use of ECT, and I believe that some reforms in those practices could significantly mitigate the harm some of us suffer from this treatment.

Effectiveness

The ECT got rid of my depression completely for three weeks. After that, my depression began returning intermittently, and by six weeks post-treatment, it was constant again--and has been ever since (i.e. for the last five months). In accordance with current "received wisdom," I continued to take antidepressant

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medication during and after my ECT, and have continued psychotherapy.

Somatic Effects

Various research has failed to find evidence of *structural* damage to the nervous system from ECT. I think it is certainly credible that there would be no such damage.

ECT *does*, however, create *changes* in the central nervous system--this, of course, is precisely why it is effective. Moods, feelings, memories and actions are, after all, the expression of neurological and biochemical processes in the central nervous system.

In addition to the therapeutic effect of eliminating my depression, my ECT had a number of other effects, my subjective experience of which seems to be clear evidence of changes in my central nervous system. Some of these effects were positive, and some were benign. For example, since my ECT, I now have a degree of dexterity with my left hand that far surpasses my previous ability. Interestingly, none of the clinicians I've asked about this has any idea of why this kind of change would result from ECT. They are equally bemused by all the other post-ECT changes, gains and losses I've experienced in my moods, aptitudes and feelings.

Unfortunately, very little is known about *what* changes in the central nervous system do result from ECT, or why it has a therapeutic effect when it does.

Short-term Effects

While some of the unintended effects I experienced from ECT were positive, others were decidedly horrific--and far more severe than the predictable "confusion" and "memory loss."

Most people--including the mentally healthy--get confused from time to time. The confusion I experienced immediately after each ECT treatment was different enough from routine confusion that it should probably have a different name. I'm not talking about not knowing whether it was morning or afternoon--I'm talking about not even knowing *what* I was, let alone who or where I was. But this ill effect usually passed within an hour or so after the treatment.

The "confusion" I continued to experience for about six weeks after my last treatment was another matter. I was unable to organize or contextualize thoughts and feelings. I couldn't discern any logic, either internal or externally imposed, in my thoughts and perceptions. I was unable to perceive or create any hierarchy among thoughts, feelings, and physical sensations. Everything I thought, said or wrote was an incoherent stream of consciousness, with the result that I was utterly unable to communicate appropriately or effectively. This amounted to a severe occupational and social disability.

I happened to have known before my ECT that the right and left hemispheres of the brain have certain different specialized functions. During one of my post-ECT visits with my regular psychiatrist, I told him I was still experiencing the disabling "confusion." He made the observation that since the right

hemisphere is where the functions of thought organization and contextualization are controlled, these functions could be disrupted by right-hemisphere electroshock, and that this potential disruption wasn't given adequate attention by practitioners.

"Memory" isn't just a data bank of pieces of information that we might or might not care to use at any given moment. Memory pretty much covers everything we know and feel, and need to know and feel, to function--on every level. I lost knowledge, skills, abilities and feelings of all kinds, and these losses made it impossible to function in work, routine activities, self-care, relationships, etc.

My memory loss included, at various times for about six weeks following my ECT, not knowing who people were, let alone what their names were; not being able to figure out how to put on my clothes; not having even the most basic job skills necessary to perform my usual work, let alone any concept of what that work was about; not being able to drive my car, let alone figure out where I was going; not knowing where anything was in my home; etc.

I found myself wondering things such as: What is that thing [the machine that I later recognized as my vacuum cleaner], and what is it for? I wonder if there's any way I can get my floor clean? Is cleaning floors something that normal people do, or am I being strange to want to clean my floor? Who lives in that house across the street? Did I used to know who lives there? Am I the kind of person who *would* have known who lived there? What kind of person am I anyway? What did I used to believe, and would I believe the same things now?

I am also having to re-learn *how* to remember--I lost the process itself of remembering and learning.

The most agonizing after-effects I had during those six weeks were: sudden dramatic and unpredictable mood swings; loss of superego; loss of all feelings of love and affection for the people and things I used to love, and of motivation for the work I used to love doing; loss of certain kinds of aesthetic pleasure and of pleasure from physical movement; and, most agonizing of all, an overwhelming sense that I had lost my *self*.

At least a couple times every day, I found myself screaming and writhing uncontrollably. When there was verbal content to my screams, it consisted of such things as: "What have they *done* to me?!" "They've *destroyed* me!" "My *self* is gone!" "I can't *feel*!" "They've turned me into a *monster*!" "They should have *killed* me instead!"

For me, many of these effects were a far worse agony than the depression which was successfully treated. During this period of time, on several occasions, I came much closer to actually killing myself than I had come before. This was partly because the intensity of the anguish was so overwhelming, and also because the functions of my superego and my feelings of closeness to other people, which had previously acted as a brake on my suicidal inclinations, were gone.

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However, I was progressively recovering from some of the ECT effects, and I hadn't been recovering at all from the preceding depression. Otherwise, I'm sure I would have killed myself during that immediate post-ECT period to escape the indescribable horror that was more harrowing than anything I'd ever felt before.

I took LSD once, over 20 years ago. My post-ECT experience felt most analogous to a protracted real bumner of an acid trip, interspersed with some periods of feeling very pleasantly "high."

Bi-lateral shock

I had at the outset consented only to 6-8 *unilateral* shock treatments. I specifically did not want any shock treatments with bilateral electrode placement because: 1) I had read that there is a higher incidence and severity of adverse effects with bilateral shock; 2) I had read that there was no evidence that bilateral electrode placement produces any greater therapeutic benefit than unilateral placement; and 3) I didn't want my left (dominant) hemisphere exposed to direct shock at all.

The bilateral shock I had was administered not only without my informed consent, but over my explicit objection--and without any other legal authorization, for that matter (i.e., I had been found competent to consent, and no one else was legally authorized to make treatment decisions for me).

The immediate aftermath of the bilateral shock included a subjective anguish and depression that was worse than the depression which had preceded my decision to get ECT in the first place. This mood lasted for the next two days, until the next (unilateral) treatment. The afternoon after the bilateral treatment, I wrote in my journal, "I'm more depressed today than I've been in a long time--if I weren't in the hospital, I'd probably kill myself today."

It was also immediately after that bilateral treatment that I first felt the lack of any pleasure in music that had previously been very pleasurable to me, and the loss of feelings of love, care and affection for people I had previously had those feelings for. These two effects did not occur after the first (unilateral) shock, but they have persisted ever since that one bilateral shock, even during the periods of time when I wasn't depressed.

Six months later

I am now living with the deadly depression and the debilitating residual effects of the ECT. After the first six weeks post-treatment, my progress in recovering from the adverse effects came to a standstill.

In the six months since my ECT, I have been able to work only a few days altogether. There are still significant gaps in my memory of the past, and I am still often unable to learn and remember in the present. Since cognitive and memory impairments have always also been symptomatic of my depression, I know that some of these difficulties may be attributable to the depression. However, I'm convinced that most of these dysfunctions are a

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result of the ECT, both because they are qualitatively different, and because they are far worse now than they were before the ECT.

My biggest apprehension before getting ECT was that it might impair my intellectual functioning. Well, obviously, it did--but that seems relatively insignificant to me now. There are two other losses which may be unimaginable to most people because they involve feelings that are usually taken for granted as inherent parts of the human experience.

The first is the inability to feel certain kinds of aesthetic pleasure, in a way that is characteristically different from the loss of pleasure that is usually symptomatic of major depression. Interestingly, I can still feel pleasure from some kinds of aesthetic stimuli. But I am utterly unable to feel aesthetic pleasure from music that used to be very pleasing to me and from static visual stimuli. When I hear music that I used to love and see the beautiful sights in my environment that used to evoke a feeling of *perceiving* that beauty, I have none of those old feelings--I feel as though I am sucking on a pinched straw with my eyes and ears to try to extract from these stimuli the responses they used to create.

By far the worst loss is the absence of feelings of love and affection for the people in my life. This too is different from the apathy, irritability, and social withdrawal that often characterize depression. What I have lost now is all sense of caring, concern, closeness and connection with the people I used to love, the people whose love for me used to be the last thing keeping me alive when I was feeling most suicidal. *This* is the most profound loss of my *self*.

Informed Consent

I am deeply perplexed and outraged by pervasive inadequacies in both the processes of obtaining "informed consent" and the content of information provided to prospective ECT recipients. A psychiatrist who has been very active in fighting against regulation of the use of ECT has lamented that California's legal requirements on informed consent for ECT are the most restrictive for any medical procedure anywhere in the U.S. In my own case, those requirements were not even followed. But even if they had been, they would still leave a lot to be desired by way of protecting consumers' rights. I shudder to think how unprotected consumers must be in other states if California's requirements are the strongest.

The content of the information given to me, on the basis of which I was to consent to ECT, was woefully inadequate. What I already knew, and recall being informed of, was that memory loss and confusion, usually of short duration, were common effects of ECT, and that other more serious adverse effects--such as cardiac arrhythmia, hypotension and death--were possible, but very rare, complications of this procedure. I wasn't informed that the debilitating effects I have since experienced from ECT were possible outcomes.

I strongly believe that the information about ECT which is given to the client, and on which his/her consent is to be based, should be as comprehensive as possible, and should let the client

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know that extreme psychological pain is one possible result. There is, of course, a wide range of variation in individual responses to ECT, and in the immediate and longer-term adverse effects that different people experience. I have no idea how typical or atypical my own experiences are, so the particulars of the information I wish I'd had may be completely idiosyncratic. The point is, though, that any of us who consider having ECT should be informed that there is a wide range of *possible* adverse effects, including affective, as well as cognitive, impairments.

I especially wish I had been forewarned that recuperation from the treatment can be a very difficult and time-consuming process. I wasn't prepared for the possibility that I might be disabled for months by the residual effects of the ECT. It would have helped me enormously to have had ahead of time the advice I got only weeks later, from another psychiatrist, who suggested that the best way for me to cope with my continuing disability and the recovery process was to go very slowly, be very patient, not expect much from myself, and ask for a lot of support.

I understand the psychology of minimizing information about possible harmful effects, on the assumption that the "power of suggestion" may have a negative influence on a client who otherwise might not have a negative experience. However, I believe it is a grave disservice to the client to withhold such information. For those of us who do have agonizing after-effects from ECT, it is a doubly horrific experience when not having been forewarned.

In their efforts to defend the use of ECT--whether motivated by individual financial gain or the best-intentioned altruistic feeling for their patients--some psychiatrists fail utterly to comprehend how psychologically crippling and emotionally devastating the after-effects of ECT can be.

It may well be the case that many physicians who administer ECT might not even be aware of these effects, especially in the absence of comprehensive and systematic documentation of their occurrence, and in the absence of followup examination by the physician. For example, whenever I reported any of the adverse effects to my attending physician in the hospital, his response was to categorize all my experiences as manifestations of the predictable "memory loss and confusion," and to assure me that they would be of short duration. However, the worst of these effects didn't even become manifest until I left the hospital and tried to return to my life. Since that physician never did any followup on my progress after I left the hospital, he has no way of knowing what the longer-term effects of my ECT have been or how severely they have disabled me.

Much more specific information should be provided to clients on things like the average length and range of time it takes to recover memory; the percentage of people who never fully recover memory or other cognitive functioning; the percentage of people who experience affective impairments and the kinds and severity of these impairments, etc. I know that some of this information is not currently available, but it should be, and research should be done to find it.

There are certain qualities of subjective experience that researchers can't quantify and that those who haven't felt them really can't adequately describe. That's why one of the things I'd like to see is a significant amount of input from ECT recipients and survivors in the information that is provided to people who are considering ECT.

The National Institute of Mental Health's *Consensus Development Conference Statement on "Electroconvulsive Therapy"* (Vol. 5, No. 11, 1985) includes the recommendation that, for a number of reasons, informed consent be an ongoing and repeated process throughout the course of ECT treatment, and that such periodic reviews should be initiated by the physician. I wholeheartedly concur with this recommendation. Because it was not followed during my own treatment, my autonomy, and my right to consent or withhold consent, were *de facto* taken away from me for the duration of the process, after my initial consent before the first treatment.

One of the reasons this is important is that a client may decide, on the basis of initial adverse effects, that the potential benefits no longer outweigh the risks, and may decide to withdraw consent for further treatments. Given the likely memory loss and cognitive impairment, which are especially acute within hours or days of the electroshock, the client may not even remember having given consent or being entitled to withdraw it.

My Conclusions

From what I've said, it may sound as though I'm opposed to ECT. I'm not. Even knowing what I know now about my own adverse after-effects, I still think it was probably the right decision for me to have ECT. That treatment did, undeniably, save me from suicide. However, if I had known before I had ECT what I know now, and if I had decided that death was preferable, I would have felt that being subjected to ECT without my consent was a profound and unforgiveable violation of my self.

With that in mind, I'm going to go out on a limb... It is my tentative opinion that, especially given the inherent invasiveness and potential psychological toxicity of ECT, no one, under any circumstances, should ever be subjected to ECT without his/her fully informed consent. I realize that not many people would agree with me on this point, but I would go so far as to argue that ECT should not be administered without one's consent even if it is known with certainty that suicide will otherwise result. Having been myself in the position of having suicide or ECT as the only options available to me, I feel entitled to make that argument. And if I were faced with the choice today, I would unhesitatingly prefer death to ECT.

My opinion is also based on everything I've heard and read from people who were given ECT without their consent and have resented it, often very bitterly, ever since. The reason this opinion is tentative, though, is that there may be some people who were given ECT without their consent and later appreciated that that had happened. If this is the case, intensive research should be done to find any possible variables that might be

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helpful in predicting who is and who is not likely to consent "after the fact," as it were--because it is unacceptable to continue sacrificing those who are harmed by involuntary ECT, on the basis of statistical probabilities or wishful thinking.

My hope is that the question will not continue to be whether ECT should be permitted or banned, because for some people in some circumstances, it is truly the last means they have for saving their lives. What I hope is that some essential questions will be answered; that protocols for the use of ECT and related processes will be improved, standardized and monitored; that psychiatric patients' rights to self-determination and decision-making in their own treatment will be given the paramount status they deserve; and that eventually, less toxic and more effective treatments--and even cures or preventions--will be found for the devastating maladies which are currently treated so imperfectly by ECT.

Sincerely,

Karen Rian, Ph.D.

Time to Abandon Electroconvulsion as a Treatment in Modern Psychiatry

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ABSTRACT

This review examines the evidence for the current use of electroconvulsive therapy (ECT) in psychiatry. The history of ECT is discussed because ECT emerged with no scientific evidence, and the absence of other suitable therapy for psychiatric illness was decisive in its adoption as a treatment. Evidence for the current recommendation of ECT in psychiatry is reconsidered. We suggest that ECT is an unscientific treatment and a symbol of authority of the old psychiatry. ECT is not necessary as a treatment modality in the modern practice of psychiatry.

Keywords: | electroconvulsive therapy; treatment uses and abuses

INTRODUCTION

Berrios¹ has thoroughly documented the history of electroconvulsive therapy (ECT). We suggest that in both the 19th and 20th centuries the social context in which ECT emerged, rather than the quality of the scientific evidence, was decisive in determining its adoption as a treatment.

The medical literature is a virtual graveyard for inadequately tested preparations that die ignominiously after a brief moment of glory. Egas Moniz won a Nobel Prize in medicine for the prefrontal lobotomy, targeted at patients in whom ECT had failed. Clearly, psychiatrists abandoned all forms of shock treatment except ECT because of the empiric nature of such therapy and the lack of a credible explanation why it should work.

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The prime bases of validation for ECT are vague statements about "clinical experience." Since the introduction of antipsychotics and antidepressants, the number of people subjected to ECT has undoubtedly declined, yet it is still used by some psychiatrists as the ultimate weapon. The proponents of ECT have to preserve the integrity of its use by having more training and better technology and claiming that ECT has proved its worth in clinical "experience." Thomas Szasz wrote that electricity as a form of treatment is "based on force and fraud and justified by 'medical necessity'." "The cost of this fictionalization runs high," he continued. "It requires the sacrifice of the patient as a person, of the psychiatrist as a clinical thinker and moral agent." Some people who have had ECT believe that they were cured by it; this fact indicates that they have so little self-control over the conditions of their lives that they must be shocked by an electric current to discharge their responsibilities.

When ECT became an emotional issue in psychiatry because of pressure groups, various bills were introduced by legislators in the United States. Professional societies and colleges—the task force of the American Psychiatric Association³ and the Royal College of Psychiatrists memoranda^{4,6}—have tried to study the subject and survey ECT use. Despite these efforts, ECT is and will remain controversial.

SHOCK AND TERROR AS THERAPY

Terror as a therapy for insanity has been used since antiquity, and as late as the 19th century, the insane were submerged in cold water to terrify them with the prospect of inevitable death.

While using insulin as a sedative in Viennese drug addicts, Sakel⁸ observed that accidental overdose resulted in coma or epileptic fits. In a burst of nonscientific theorizing, he wrote: "I began with the addict. I observed improvements after severe epileptic fits.... Those patients who had previously been excited and irritable suddenly became contented and quiet after this shock.... The success I had achieved in treating addicts and neurotics encouraged me to use it in the treatment of schizophrenia or major psychoses."

Meduna used camphor-induced fits on psychiatric patients in a Hungarian state mental hospital after unsuccessful attempts by Nyiro, his superior, to treat schizophrenia by injections of blood from epileptics. Meduna later employed Cardiazol-induced shock. Nyiro's and Meduna's convulsive therapies were based on the view that a neurobiologic opposition existed between epilepsy and schizophrenia. Meduna abandoned his theory of schizophrenia and epilepsy and later wrote "We are undertaking a violent onslaught...because at present nothing less than a shock to the organism is powerful enough to break the chain of noxious processes that lead to schizophrenia."

Psychiatrists of that era who used this form of shock therapy believed that the fear and terror produced were therapeutic because—the "feeling of horror" before the onset of convulsion following injection of camphor, pentetrazol, triazole, picrotoxin, or ammonium chloride rendered the patients different after the experience.¹⁰

ELECTRICITY AS THERAPY

Extensive literature is available on the use of electricity as a therapy and the induction of epilepsy by electric current.¹¹ In ancient Rome, Scribner Largus tried to cure the emperor's headache with an electric eel. In the 16th century, a Catholic missionary reported that the Abyssinians used a similar method to "expel devils out of human body." Aldini treated two cases of melancholia in 1804 by passing galvanic current through the brain. In 1872, Clifford Allbutt in England applied electric current to the head for treatment of mania, dementia, and melancholia.

In 1938, Ugo Cerletti obtained permission to experiment with electricity on pigs in a slaughterhouse. "Except for the fortuitous and fortunate circumstances of pigs' pseudo-butcherery," he wrote, electroshock would not have been born.¹² Cerletti did not bother to obtain permission to experiment on the first human subject, a schizophrenic who after the initial shock said "Non una seconda! Mortifere." (not again; it will kill me). Cerletti nevertheless proceeded to a higher level and a longer time, and so ECT was born. Cerletti admitted that he was frightened at first and thought that ECT should be abolished, but later he started to use it indiscriminately.

In 1942, Cerletti and his colleague Bini advocated the method of "annihilation," which consisted of a series of (unmodified) ECTs many times a day for many days. They claimed good results in obsessive and paranoid states and in psychogenic depression. In fact, Cerletti had discovered nothing, as both electricity and fits were already known. No scientist, he believed that he discovered a panacea, reporting success with ECT in toxemia, progressive paralysis, parkinsonism, asthma, multiple sclerosis, itch, alopecia, and psoriasis.¹² By the time of his death in 1963, neither Cerletti nor his contemporaries had learned how ECT worked. The inheritors of ECT continue the same lack of understanding today.

Insulin coma and pentetrazol-induced fits, heretofore treatments of choice for schizophrenia, are not therapies any longer, and ECT is not a treatment for schizophrenia. The fact of the matter is that the pioneers of all these shock treatments contributed nothing to the understanding of mental illness, which contemporary psychiatrists are still trying to comprehend and treat on a scientific basis.

ELECTRICITY, CONVULSIONS, THE BODY, AND THE BRAIN

For its proponents, ECT is a relatively simple procedure. Electrodes are attached to the subject's head, either at the temples (bilateral ECT) or at the front and back of one side (unilateral ECT). When the current is turned on for 1 second, at 70 to 150 volts and 500 to 900 milliamperes, the power produced is roughly that required to light a 100-watt bulb. In a human being, the consequence of this electricity is an artificially induced epileptic fit. Modified ECT was introduced as a humane improvement on earlier versions of convulsive therapy to eliminate the elements of fear and terror. In modified ECT, muscle relaxant and general anesthesia are supposed to make the patient less fearful and feel nothing. Nonetheless, 39% of patients thought it was a frightening treatment.¹³ These induced fits are associated with many physiologic events, including electroencephalographic (EEG) changes, increased cerebral blood flow, bradycardia followed by tachycardia and hypertension, and throbbing headache. Many patients report temporary or prolonged loss of memory, a sign of acute brain syndrome.

Since early in the history of ECT, we have known that insulin coma or pentetrazol shock can cause brain damage.¹⁴ Bini reported severe and widespread brain damage in experimental animals treated with electroshock.¹⁵ EEG studies showed generalized slowing following ECT that takes weeks to disappear and may persist even longer in rare cases.¹⁶ Calloway and Dolan raised the issue of frontal lobe atrophy in patients previously treated with ECT.¹⁷ The memory deficits after ECT may persist in some patients.¹⁸

Fink, an advocate of ECT, argues that the risks of ECT amnesia and organic brain syndrome are "trivial"¹⁹ and can be reduced by hyperoxygenation, unilateral ECT over the nondominant hemisphere, and the use of minimal induction currents.²⁰ Earlier, Fink had indicated that post-ECT amnesia and organic brain syndrome were "not trivial." ECT advocates blame the modification for decreasing the efficacy of the treatment.²¹ In the United States, the issue of unilateral ECT reflected class differences. In Massachusetts in 1980, ECT was bilateral in 90% of patients in public hospitals and in only 39% of patients in private hospitals.²²

Templer compared the issue of ECT brain damage to that of boxing. He wrote that "ECT is not the only domain in which change to the human brain is denied or de-emphasized on the grounds that this damage is minor, occurs in a very small percentage of cases or is primarily a matter of the past."²³

There has been less scientific investigation into the effect of ECT on other body functions and morbidity. Various animal studies showed significant results that may be important in psychoimmunology--an area of investigation that is more neglected in psychiatry than in any other field of medicine. Although it is difficult to move from an animal model to the human system, animal models frequently demonstrate the role of a range of variables in disease onset. Rats subjected to electrical stress showed significant diminution in the strength of their lymphocyte response that could not be explained by an elevation in adrenal corticosteroids. Even adrenalectomized rats had a similar decrease in lymphocyte response after electric shock²⁴; other studies have confirmed immunologic change following electric shock in animals.

USE AND ABUSE OF ECT IN SCHIZOPHRENIA

Initial claims that cardiazol convulsions and insulin coma were successful in the treatment of schizophrenia were not universally shared. Some researchers found that these interventions were worse than no treatment.²⁵

For more than 50 years, psychiatrists used ECT as therapy for schizophrenia, even though there is no evidence that ECT alters the schizophrenic process.²⁷ In the 1950s, ECT was reported to be no better than hospitalization alone²⁸ or anesthesia alone.²⁹ At the beginning of the 1960s, the era of ECT in schizophrenia was fast drawing to a close as ECT abuses were brought to light by patients and pressure groups. In 1967, however, Cotter described symptomatic improvement in 130 schizophrenic Vietnamese men who refused to work in a psychiatric hospital and received ECT at a rate of three shocks per week.³⁰ Cotter concluded that "the result may simply be due to patients' dislike and fear of ECT," but he further claimed that "the objective of motivating these patients to work was achieved."³⁰

Finally, Gregory and colleagues⁴³ compared simulated ECT with actual unilateral or bilateral ECT. Real ECT produced faster improvement but no difference between the treatments was apparent 1, 3, and 6 months after the trial. Only 64% of patients completed this study; 16% of the patients withdrew from bilateral ECT and 17% from simulated ECT.

From the West and the Northwick Park trials, it appears that only delusional depression responded more to real ECT, and this view is held by ECT proponents today. A study by Spiker et al, showed that in delusional depression amitriptyline and perphenazine were at least as good as ECT. After a series of ECT for his depression and just before committing suicide, Ernest Hemingway said, "Well, what is the sense of ruining my head and erasing my memory, which is my capital, and putting me out of business." His biographer remarked that "it was a brilliant cure but we lost the patient."⁴⁵

ECT AS AN ANTISUICIDAL

Despite the lack of an acceptable theory as to how it works, Avery and Winokur⁴⁶ regard ECT as a suicide preventive, although Fernando and Storm⁴⁷ later found no significant difference in suicide rates between patients who received ECT and those who did not. Babigian and Cuttmacher⁴⁸ found that the mortality risk after ECT was higher soon after hospitalization than in patients who did not receive ECT. Our own study⁴⁹ of 30 Irish suicides from 1980 to 1989 showed that 22 patients (73%) had received a mean of 5.6 ECTs in the past. The explanation that "ECT induces a transient form of death and thus perhaps satisfies an unconscious desire on the part of the patient, but this has no preventative effect on suicide; indeed it reinforces suicide in the future."⁴⁹ Many psychiatrists today concur that ECT as a suicide preventive does not hold up.

THE PSYCHIATRIST'S DILEMMA: TO USE OR NOT USE ECT

Some psychiatrists justify the use of ECT on "humanistic grounds and as a means of controlling behaviour" against the wishes of the patient and family.⁵⁰ Even Fink admits that the catalogue of ECT misuses is depressing but suggests that the guilt lies with the abusers and not the instrument.⁵¹ The editor of the *British Journal of Psychiatry* considered it "inhuman" to administer ECT without asking the patient or the relative, even though Pippard and Ellam showed that this was common practice in Britain. Not long ago, ECT administration in Great Britain was described as "deeply disturbing" by a *Lancet* editorial writer, who commented that "it is not ECT which brought psychiatry into disrepute; psychiatry has done just that for ECT."⁵² Despite efforts to preserve the integrity of the treatment, in Great Britain and in most public hospitals worldwide consultant psychiatrists order ECT and a junior doctor administers it. This maintains the belief of institutional psychiatry that electricity is a form of treatment and prevents the junior psychiatrist from being a clinical thinker.

Levenson and Willett⁵³ explain that to the therapist using ECT it may seem unconsciously like an overwhelming assault, which may resonate with the therapist's aggressive and libidinal conflict.

Most contemporary psychiatrists consider the use of ECT in schizophrenia as inappropriate, but some believe that ECT is at least equal to other therapies in this illness.³¹

ECT in Depression

In the 1960s, advocates of ECT were not able to provide evidence that it is therapeutic in schizophrenia but were nevertheless convinced that electricity and fits are therapeutic in mental illness and vigorously defended the use of ECT in depression. Their rationale came from studies in the United States³² and Britain.³³

In the US study,³² patients were pooled from three hospitals. In hospitals A and C, ECT was as good as imipramine; in hospitals B and C, ECT equaled placebo. The results showed that ECT was universally effective in depression, regardless of type: 70% to 80% of depressed patients improved. The study also showed, however, a 69% improvement rate after 8 weeks of placebo. Indeed, Lowinger and Dobie³⁴ reported that improvement rates as high as 70% to 80% can be expected with placebo alone.

In the British study,³³ hospitalized patients separated into four treatment groups: ECT, phenelzine, imipramine, and placebos. No differences were observed in male patients at the end of 5 weeks, and more men who received placebo were discharged from the hospital than those treated with ECT. Skrabanek³⁵ commented about this most quoted study: "One wonders how many psychiatrists read more than the abstract of these studies."

The Royal College of Psychiatrists memorandum mentioned earlier was in response to a report of ECT abuse in depression. The memorandum declared that ECT is effective in depressive illness and that in "depressed patients" there is suggestive, if not yet unequivocal, evidence that the convulsion is a necessary element of the therapeutic effect. Crow,³⁶ on the other hand, questioned this widely held view.

In the late 1970s and in the 1980s, with uncertainty continuing and further work needed, seven controlled trials were carried out in Britain.

Lambourn and Gill³⁷ used unilateral simulated ECT and unilateral real ECT in depressed patients and found no significant difference between the two.

Freeman and associates³⁸ used ECT in 20 patients and achieved a satisfactory response in 6; a control group of 20 patients received the first two of six ECT treatments as simulated ECT, and 2 patients responded satisfactorily.³⁸

The Northwick Park Trial showed no difference between real and simulated ECT.³⁹

Gangadhar and coworkers⁴⁰ compared ECT and placebo with simulated ECT and imipramine; both treatments produced equally significant improvements over 6 months follow-up.

In a double-blind controlled trial, West⁴¹ showed that real ECT was superior to simulated ECT, but it is not clear how a single author carried out a double-blinding procedure.

Brandon et al⁴² demonstrated significant improvements in depression with both simulated and real ECT. More important, at the end of 4 weeks of ECT, consultants were unable to guess who received real or simulated treatment. The initial differences with real ECT disappeared at 12 and 28 weeks.

Studies that examined attitudes of psychiatrists toward ECT found marked disagreement among clinicians about the value of this procedure.^{55,56} Thompson et al⁵⁷ reported that ECT use decreased 46% between 1975 to 1980 in the United States, with no significant changes between 1980 to 1986. Fewer than 8% of all US psychiatrists use ECT, however.⁵⁸ A very recent study⁵⁹ on the characteristics of psychiatrists who use ECT found that female practitioners were only one-third as likely to administer it as were their male counterparts.⁵⁹ The proportion of female psychiatrists has been rising steadily and if the gender gap continues, this could hasten the end of ECT.

CONCLUSION

When ECT was introduced in 1938, psychiatry was ripe for a new therapy. Psychopharmacology offered two approaches to the pathogenesis of mental disorders: to investigate the mechanism of action of drugs that ameliorate the disorder and to examine the actions of drugs that reduce or mimic the disorder. In the case of ECT, both approaches have been pursued without success. Chemically or electrically induced fits have profound but short-lived effects on brain function, ie, acute organic brain syndrome. Shocking the brain causes increases in levels of dopamine, cortisol, and corticotropin for 1 to 2 hours after the convulsion. These findings are pseudoscientific, as there is no evidence that these biochemical changes, specifically or fundamentally, affect the underlying psychopathology of depression or other psychoses. Much of the improvement attributed to ECT is an effect of placebo or, possibly, anesthesia.

From the earliest uses of convulsive therapy, it was recognized that the treatment is unspecific and only shortens the duration of psychiatric illness rather than improves the outcome.⁶⁰ Convulsive therapy based on the old belief of shocking the patient into sanity is primitive and unspecific. The claim that ECT has proved its usefulness, despite the lack of an acceptable theory as to how it works, has also been made for all the unproven therapies of the past, such as bloodletting, which are reported to produce great cures until they are abandoned as useless. Insulin coma, cardiazol shock, and ECT were treatments of choice in schizophrenia, until they, too, were abandoned. For ECT to remain as an option in other psychoses transcends clinical and common sense.

When an electrical current is applied to the body by tyrannical rulers, we call this electrical torture; however, an electrical current applied to the brain in public and private hospitals by professional psychiatrists is called therapy. Modifying the ECT machine to reduce memory loss and giving muscle relaxants and anesthesia to make the fit less painful and more humane only dehumanize users of ECT.

Even if ECT were relatively safe, it is not absolutely so, and it has not been shown to be superior to drugs. This history of ECT, its abuse, and resultant public pressure are responsible for its increasingly lower use.

Is ECT necessary as a treatment modality in psychiatry? The answer is absolutely not. In the United States, 92% of psychiatrists do not use it despite the existence of an established journal entirely devoted to the subject to give it scientific respectability. ECT is and always will be a controversial treatment and an example of shameful science. Even though some 60 years have been spent defending the

treatment, ECT remains a revered symbol of authority in psychiatry. By promoting ECT, the new psychiatry reveals its ties to the old psychiatry and sanctions this assault on the patient's brain. Modern psychiatry has no need of an instrument that allows the operator to zap a patient by pressing a button. Before inducing a fit in a fellow human being, the psychiatrist as clinician and moral thinker needs to recall the writings of a fellow psychiatrist, Frantz Fanon⁶¹: "Have I not, because of what I have done or failed to do, contributed to an impoverishment of human reality?"

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Memory Complaint After Electroconvulsive Therapy: Assessment with a New Self-Rating Instrument

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Memory complaints before bilateral electroconvulsive therapy (ECT), 1 week after ECT, and 6 months after ECT were assessed in 35 patients using a newly developed self-rating scale. Memory complaints that occurred 1 week after ECT differed quantitatively and qualitatively from memory complaints that occurred before ECT. Six months later, memory complaints qualitatively resembled the complaints reported 1 week after ECT and differed sharply from those reported before ECT. It was suggested that a patient's impression of his memory is altered by bilateral ECT and that this altered impression persists in gradually diminishing form for at least 6 months after a typical course of treatment. Since the self-rating instrument used here appeared to differentiate between memory complaints associated with depression (before ECT) and memory complaints associated with amnesia (1 week after ECT), this instrument may be useful in a variety of settings where there is interest in human memory function.

INTRODUCTION

Complaints of poor memory are common in psychiatric and neurological patients, but their significance is often difficult to determine. Self-reports of

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memory function can be discrepant with the results of memory tests (Kahn *et al.*, 1975; Cronholm and Ottosson, 1963). For example, in depressed elderly patients memory complaints appeared to be related more to degree of depression than to performance on memory tests (Kahn *et al.*, 1975). Conversely, patients receiving electroconvulsive therapy (ECT) who were clinically improved often denied memory impairment despite the fact that memory impairment could be documented by formal testing (Cronholm and Ottosson, 1963).

We recently reported that memory complaints were common 6 to 9 months after a course of bilateral ECT, being reported by 60% to 70% of patients interviewed (Squire and Chace, 1975; Squire, 1977). Yet, formal tests have failed to demonstrate persisting memory impairment after bilateral treatment (Squire and Chace, 1975; Harper and Wiens, 1975). Since memory complaints are common in depressed patients (Ianzito *et al.*, 1974; Marsella *et al.*, 1973), it has been difficult to know whether memory complaints after ECT are related to recurring depressive symptoms, to a sense of impaired memory that was present even before treatment; or indeed, whether complaints are in any way related to or caused by ECT.

A method for distinguishing memory complaints that are related to depression from memory complaints related to the effects of ECT could help understand the ECT process and could provide a tool for the assessment and interpretation of memory functions in a variety of conditions. It has been shown previously that depression and the acute amnesia associated with ECT exert qualitatively distinct effects on memory (Sternberg and Jarvik, 1976; Cronholm and Ottosson, 1961). This finding suggested that depression and amnesia might also affect memory complaints differently. Here we describe a new self-rating instrument which can differentiate memory complaints that occur before ECT, when patients are presumably depressed, from memory complaints that occur shortly after a course of ECT, when patients are amnesic. We have applied this self-rating instrument to the problem of understanding the memory complaints that occur many months after ECT.

METHOD

Subjects

The subjects of the prospective follow-up study were 46 depressed psychiatric inpatients at six hospitals who had been prescribed a course of bilateral ECT. Of the 46 patients originally included in the study, 11 were lost to long-term follow-up. Seven could not be located, two died, one declined to be interviewed, and one was subsequently prescribed a course of maintenance ECT. For the remaining 35 patients (27 female) the specific diagnoses as recorded

Memory Complaint After ECT

on admission were primary depressive, depressed (11), (4), schizo-affective disorder, phrenia, or depression secondary. Twenty-one of the 35 patients received ECT during the past previously, 12 had received (mean = 8 years). The remaining ECT, respectively, during the 25 and 64 (mean = 41), with

An additional group of These subjects had been psychotic to 10 months previously (mean course of bilateral ECT. There or severe depression (8), manic (4), involuntional melancholia subjects had received ECT previously. These 19 subjects were between average of 13.1 years of education

Bilateral treatment was following medication with (130-150 V for 0.6-1.0 sec). cases the patient was described seizure. Decisions concerning individual psychiatrists. Persons in treatments (mean = 11.1). Patients to 20 treatments (mean = 10

An 18-item self-rating, asked subjects to compare the before hospitalization (Table 9-point scale from -4 (worst) to +4 (better than ever before). somewhat different way. They had heard patients make a information about how depressed

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on admission were primary affective disorder or severe depression (12), manic-depressive, depressed (11), involutional melancholia (7), depressive neurosis (4), schizo-affective disorder (1). Patients with neurological disorders, schizophrenia, or depression secondary to alcoholism or drug-abuse were excluded. Twenty-one of the 35 patients had not received ECT before, and none had received ECT during the past year. Of the 14 patients who had received ECT previously, 12 had received one course of ECT from 1 to 16 years previously (mean = 8 years). The remaining two had received two and three courses of ECT, respectively, during the same period. Patients were between the ages of 25 and 64 (mean = 41), with an average of 12.7 years of education.

An additional group of 19 subjects (15 female) was studied retrospectively. These subjects had been psychiatric patients at the same six hospitals from 6 to 10 months previously (median = 7 months), and had received a prescribed course of bilateral ECT. Their diagnoses had been primary affective disorder or severe depression (8), manic-depressive, depressed (4), neurotic depression (4), involutional melancholia (2), schizo-affective disorder (1). Four of these subjects had received ECT prior to that time; 15 had never received ECT before. These 19 subjects were between the ages of 26 and 64 (mean = 42), with an average of 13.1 years of education.

Electroconvulsive Therapy

Bilateral treatment was administered three times a week on alternate days following medication with atropine, methohexital sodium, and succinylcholine (130-150 V for 0.6-1.0 sec). Electrode placement was temporal-parietal. In all cases the patient was described by his physician as having a modified grand mal seizure. Decisions concerning the number of treatments were made by the individual psychiatrists. Persons in the first group ($n = 35$) received from 5 to 21 treatments (mean = 11.1). Persons in the second group ($n = 19$) had received 6 to 20 treatments (mean = 10.0).

Tests and Procedure

An 18-item self-rating scale of memory functions was constructed that asked subjects to compare their memory now to their memory during the period before hospitalization (Table I). For each item, subjects rated themselves on a 9-point scale from -4 (worse than ever before), through 0 (same as before), to +4 (better than ever before). Each item inquired about memory functions in a somewhat different way. The wording of items was derived from remarks we had heard patients make about their memory before and after ECT, and from information about how depression and amnesia can differentially affect memory.

Table 1. Self-Rating Scale of Memory Function^a

1. My ability to search through my mind and recall names or memories I know are there is
2. I think my relatives and acquaintances now judge my memory to be
3. My ability to recall things when I really try is
4. My ability to hold in my memory things that I have learned is
5. If I were asked about it a month from now, my ability to remember facts about this form I am filling out would be
6. The tendency for a past memory to be "on the tip of my tongue," but not available to me is
7. My ability to recall things that happened a long time ago is
8. My ability to remember the names and faces of people I meet is
9. My ability to remember what I was doing after I have taken my mind off it for a new minutes is
10. My ability to remember things that have happened more than a year ago is
11. My ability now to remember what I read and what I watch on television is
12. My ability to recall things that happened during my childhood is
13. My ability to know when the things I am paying attention to are going to stick in my memory is
14. My ability to make sense out of what people explain to me is
15. My ability to reach back in my memory and recall what happened a few minutes ago is
16. My ability to pay attention to what goes on around me is
17. My general alertness to things happening around me is
18. My ability to follow what people are saying is

^aEach item began with the statement "compared to before I began to feel bad and went to the hospital. . . ." Thus, both before ECT and after ECT patients were asked to rate their memory now compared to before they began to feel bad. In this way, the before-ECT test attempted to assess memory problems of recent onset, presumably related to depression. After-ECT tests attempted to assess residual effects of depression and the effects of ECT on memory. For each item, patients rated themselves on a 9-point scale ranging from -4 (worse than ever before), through 0 (same as before) to +4 (better than ever before). Here the items have been ordered according to the magnitude of the difference in score obtained before ECT and 1 week after ECT (Fig. 1). Item 1 produced the largest difference, and Item 18 produced the smallest difference.

The first group ($n = 35$) was given the self-rating scale 1 to 2 days before the first treatment of the series, 1 week after the completion of the series, and again about 6 months after the completion of treatment (range 5-9 months, median = 6). For the 1-week test, 8 patients were visited in the hospital; 27 had been discharged and were visited in their homes. The location of testing did not affect the results. The difference between the self-rating scores of patients tested at home and patients tested at the hospital did not approach significance ($F < 1.0$). For the 6-month test, 34 of the 35 original patients were visited in their homes. The second group ($n = 19$) was given the self-rating scale on one occasion 6 to 10 months (median = 7) after their course of bilateral ECT. Seventeen of the 19 subjects were visited in their homes. Two had been readmitted and were tested in the hospital.

Fig. 1. Self-ratings of memory function (on a -4 to +4 scale) before and 1 week after a course of bilateral ECT. The order of test items (1-18) is same as shown in Table 1.

Figure 1 shows the results 1 week after ECT ($n = 35$). The difference in scores obtained before and 1 week after ECT produced the smallest difference in scores for the 18 test items. This difference was significant ($F = 11.4, p < 0.01$). For purpose of comparison, the mean age = 42; mean = 13 years, and were asked to rate their memory. The average score on the self-rating scale was different from zero ($t = 0.1$) for the patient group before ECT (F considered their memory to be no different from before ECT). At this time, the average SEM ± 0.1 (items \times test occasion) revealed that some items to the effects of ECT indicated that some items to the effects of ECT significant interaction between 0.01). Thus, before-ECT patients

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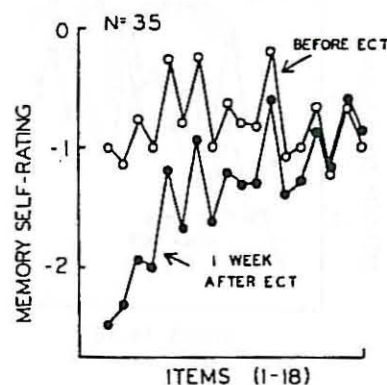
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Fig. 1. Self-ratings of memory function (on a -4 to +4 scale) before and 1 week after a course of bilateral ECT. The order of test items (1-18) is the same as shown in Table 1.



RESULTS

Figure 1 shows the results with the self-rating scale before ECT and one week after ECT ($n = 35$). The items (1-18) have been ordered along the abscissa just as they appear in Table 1, according to the magnitude of the difference in scores obtained before and after ECT. Thus, Item 1 to the extreme left produced the smallest before-after difference. These results make three general points about the experience of memory dysfunction before and after ECT: (i) Patients clearly had memory complaints before ECT. At this time, the average score on the self-rating scale was -0.80 and the average $SEM \pm 0.29$ for the 18 test items. This score was significantly below the zero level ($t = 11.4, p < 0.01$). For purposes of comparison, 20 hospital employees (16 female; mean age = 42; mean = 13 years of education) also took the self-rating scale and were asked to rate their memory now compared to 1 year ago. This group's average score on the self-rating scale was -0.05 ± 0.06 , a score not measurably different from zero ($t = 0.1$) and significantly different from the score of the patient group before ECT ($F = 6.1, p < 0.02$). Thus, before-ECT patients considered their memory to be poorer than normal subjects. (ii) One week after ECT memory complaints were present, but patients rated their memory worse than before ECT. At this time the average score on the self-rating scale was -1.4 and the average $SEM \pm 0.33$ for 18 items. A two-way analysis of variance (items \times test occasion) revealed a significant effect of test time on memory self-ratings ($F = 4.3, p < 0.05$). (iii) The pattern of scores obtained before and after ECT indicated that some items were apparently more sensitive than other items to the effects of ECT. This conclusion followed from the findings of a significant interaction between item scores and testing occasion ($F = 2.2, p < 0.01$). Thus, before-ECT patients had an approximately equivalent score across

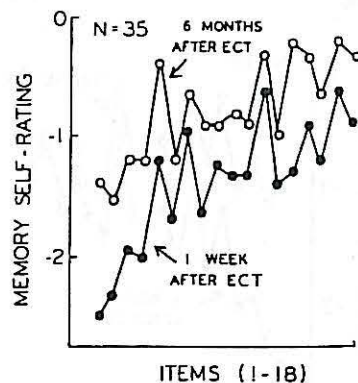


Fig. 2. Self-ratings of memory function (on a -4 to +4 scale) 1 week and 6 months after a course of bilateral ECT. The order of test items (1-18) is the same as shown in Table 1.

all items. By contrast, 1 week after-ECT patients rated themselves as worse on some items than on others.

Having established that patients had one pattern of memory complaint before ECT and a different pattern of complaint after ECT, it was possible to ask whether memory self-ratings obtained 6 months after ECT resembled the before-ECT pattern or the after-ECT pattern. Figure 2 shows the self-rating scores 6 months after ECT for the same 35 patients. Here the 6-month scores have been presented together with the 1-week scores so that they might be more easily compared. At 6 months after ECT, the average self-rating score was -0.8 , vs. -1.4 at 1 week after ECT, and a two-way analysis of variance (items \times test occasion) revealed that scores at 6 months were significantly improved compared to scores at 1 week ($F = 8.1$, $p < 0.1$). These data indicate that patients consider their memories to have improved between 1 week and 6 months after ECT. The pattern of complaint 6 months after ECT appeared similar to the pattern observed 1 week after ECT.

Figure 3 depicts more clearly the relationship between the self-rating scores obtained 6 months after ECT and the self-rating scores obtained ear-

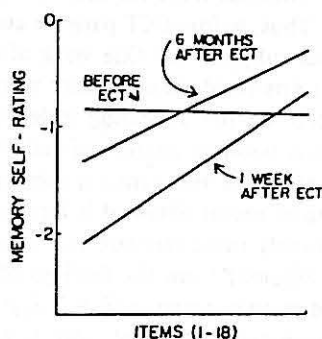


Fig. 3. Self-ratings of memory function before, 1-week after, and 6 months after a course of bilateral ECT. Scores for test items 1-18 are here represented as best-fitting lines across the scores for all test items.

Memory Complaint After ECT

lier. Here three best-fitting lines (from before ECT, 1 week after ECT, and 6 months after ECT) are shown (squares). These data illustrate that the pattern of memory complaint after ECT resembled the pattern observed before ECT, but differed sharply from the pattern observed 1 week after ECT. This conclusion followed from a comparison of the pattern of memory complaint between before-ECT and 1 week after-ECT ($F = 10.1$, $p < 0.01$), whereas the comparison between before-ECT and 6 months after-ECT did not approach significance ($F = 1.8$, $p > 0.1$).

Patients who had no memory complaint before ECT and patients who had no memory complaint 1 week after ECT (mean age = 41) had similar patterns of memory complaint 6 months after ECT ($F = 1.8$, $p > 0.1$). Six patients who had received ECT before and who had received better than patients with no memory complaint before ECT.

In the absence of a control group, the results are interpreted. It is possible that the pattern of memory complaint previously experienced by patients with memory complaints are more willing to experience this sequence of events. Patients who received ECT before will always have long-term memory complaints and who will have long-term memory complaints in two subgroups, now in two subgroups.

Age and number of test scores (Kahn *et al.* 1971) memory complaints on 18 items (0.1). However, the distribution may not have been sufficient; 46% of the 35 patients received 8-12 treatments.

To determine whether memory complaints had been influenced in an individual patient, the scores of these 35 patients were examined on only one occasion 7 months after ECT. The scores were nearly identical in every item (for $n = 35$) and -0.7 for all 18 items ($n = 35$) this line could be drawn for the second group.

Further examination of the scores on some items had a similar pattern to the scores on other items had a similar pattern.

Here three best-fitting lines have been constructed through the scores from before ECT, 1 week after ECT, and 6 months after ECT (method of least squares). These data illustrate that the pattern of complaint 6 months after ECT resembled the pattern of complaint observed 1 week after ECT and differed sharply from the pattern of complaint observed before ECT. This conclusion followed from the finding that the interaction (items \times test occasion) between before-ECT and 6-month scores was highly significant ($F = 2.2, p < 0.01$), whereas the comparable interaction between 1-week and 6-month scores did not approach significance ($F = 0.7, p < 0.3$).

Patients who had had prior experience with ECT ($n = 14$, mean age = 41) and patients who had never received ECT prior to the present course ($n = 21$, mean age = 41) had similar self-rating scores before ECT and 1 week after ECT ($F < 1.8, p < 0.1$). Six months after ECT, these two subgroups also had a similar pattern of self-rating scores (items \times subgroup, $F = 1.2, p < 0.2$), but patients who had received ECT in the past rated their memory as somewhat better than patients without prior ECT experience ($F = 4.1, p < 0.06$).

In the absence of additional information, this finding cannot be clearly interpreted. It is possible that persons who have had ECT before and who have previously experienced a complete sequence of amnesia and recovery from amnesia are more willing to rate their memory as good than persons who have not experienced this sequence. It is also possible that any group that has never had ECT before will always contain some individuals who respond poorly to ECT and who will have long-lasting memory complaint. Additional follow-up of these two subgroups, now in progress, may clarify these issues.

Age and number of treatments, two factors that can influence memory test scores (Kahn *et al.*, 1975; Harper and Wiens, 1975) did not correlate with memory complaints on any occasion before or after ECT (all r 's $< 0.22, p > 0.1$). However, the distribution of ages and number of treatments in this study may not have been sufficiently broad to provide a good test of these correlations; 46% of the 35 patients were 35–45 years of age, and 63% of them had received 8–12 treatments.

To determine whether self-rating scores obtained 6 months after ECT had been influenced in any way by repeated testing, we compared the 6-month scores of these 35 patients with the scores of the 19 patients who were tested on only one occasion 7 months after ECT. The scores of these two groups were nearly identical in every respect. The average self-rating scores were -0.80 (for $n = 35$) and -0.76 (for $n = 19$). The best-fitting lines constructed from the scores for all 18 items were nearly superimposable. Thus, for the first group ($n = 35$) this line could be described by the relationship $y = 0.061X - 1.38$; for the second group ($n = 19$), $y = 0.065X - 1.38$.

Further examination of Fig. 3 indicates that at 6 months after ECT, scores on some items had not yet recovered to the before-ECT level. Yet, scores on other items had apparently recovered to or exceeded the before-ECT level.

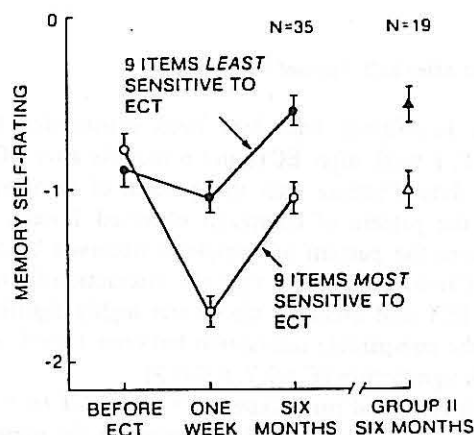


Fig. 4. Self-ratings of memory function before, 1 week after, and 6 months after a course of bilateral ECT. The nine test items labeled as most sensitive to ECT are the first nine items in Table I. These items were the nine items producing the largest difference in self-rating scores before ECT vs. 1 week after ECT. Group II ($n = 19$) was tested on one occasion 7 months after a course of bilateral ECT.

This point is illustrated in a different way in Fig. 4, where the 18 items have been separated into two equal groups. One group consists of the nine items most sensitive to the acute effects of ECT on memory. These items are the first nine items in Table I and the left-most items in Figs. 1, 2, and 3. The second group consists of the nine items least sensitive to the acute effects of ECT. These are Items 10–18 in Table I and the right-most items in Figs. 1, 2, and 3. Figure 4 shows that at 6 months after ECT the average scores for the nine items most sensitive to ECT remained significantly below the before-ECT level ($t = 2.8, p < 0.01$). Moreover, at 6 months after ECT the average score for the nine items least sensitive to ECT was significantly above the before-ECT level ($t = 2.7, p < 0.01$). To determine whether the results obtained from this somewhat arbitrary grouping of test items had any generality, we followed this same procedure with the independent group of 19 subjects. Figure 4 indicates that nearly identical results were obtained with this group. Taken together, these results suggest that some memory complaints, as measured by Items 1–9, were more severe at 6 months after ECT than before ECT. As measured by Items 10–18, other complaints were less severe after ECT than before ECT.

Finally, an attempt was made to assess the importance to the subjects of their persisting complaints by asking them to select one of five statements that best described their circumstance.

Memory Complaint After

Severity of Memory

1. No problem
2. Only an occasional problem
3. Minor problems occur frequently
4. Many problems disturbing and frequently
5. Severe problems interfere with everything I do

This tabulation indicates that the most disturbing memory problem reported was "minor problems occur frequently."

A new self-rating interpretation of memory complaints 1 week after treatment were presumably (Squire *et al.*, 1973). Since after a course of 1976; Cronholm and altered pattern of memory related to amnesia that persist several

Memory complaints compared to 1 week after ECT qualitatively after ECT and described before ECT. bilateral ECT. Unilateral ECT (Squire, 1977) with less memory memory complaint course of memory than ECT.

Severity of Memory Problem	Before ECT	1 Week after ECT	6 Months after ECT
1. No problem	9	8	6
2. Only an occasional minor problem	7	2	8
3. Minor problems but they occur frequently	9	4	10
4. Many problems that are disturbing and that occur frequently	10	14	9
5. Severe problems that interfere with almost everything I do	0	7	2

This tabulation indicated that 1 week after ECT the average report was "many disturbing memory problems." Before ECT and at 6 months after ECT the average report was "minor but frequent problems."

DISCUSSION

A new self-rating instrument has been described for the assessment and interpretation of memory complaints. The test appears to discriminate between memory complaints that occur before ECT and memory complaints that occur 1 week after treatment is completed. Memory complaints reported before ECT were presumably related to depressive illness (Ianzito *et al.*, 1974; Marsella *et al.*, 1973). Since memory dysfunction can easily be demonstrated 1 week after a course of bilateral treatment (Harper and Wiens, 1975; Squire *et al.*, 1976; Cronholm and Blomquist, 1959), it seems reasonable to suppose that the altered pattern of memory complaints observed 1 week after ECT was largely related to amnesia. We have used this method to assess memory complaints that persist several months after a course of bilateral ECT.

Memory complaints were present 6 months after ECT, but diminished compared to 1 week after ECT. The memory complaints reported 6 months after ECT qualitatively resembled the pattern of memory complaints observed 1 week after ECT and differed sharply from the pattern of memory complaints observed before ECT. It must be emphasized that these findings apply only to bilateral ECT. Unilateral ECT, which affects memory test scores less than bilateral ECT (Squire, 1977; Harper and Wiens, 1975), would be expected to be associated with less memory complaint. A long-term follow-up study of unilateral ECT and memory complaint is now in progress. It should also be of interest to assess the course of memory complaint in psychiatric patients receiving treatments other than ECT.

The specific items that demonstrated persisting memory complaint (Items 1-9) seemed to differ from items that did not (10-18). Several of the first nine items asked about the ability to learn and retain new material or to recall previously learned material. Several of the second nine items asked about the ability to attend, to hold information in memory across short time intervals, or to recall material from the remote past. It is interesting to note that the amnesic syndrome typically spares immediate memory function (e.g., Items 13, 14, 16, 17, 18) and memory of the distant past (e.g., Items 10, 12), but can affect learning (e.g., Items 4, 8), delayed recall (e.g., Items 5, 9), and memory for the more recent past. Thus, Items 1-9 may ask about experiences likely to be associated with amnesia. Items 10-18 may ask about experiences more likely to be associated with depression. Whereas it is not clear that each of the 18 items was affected differently by depression and amnesia, or that the two sets of items (1-9 and 10-18) are each measuring just one factor, the results before and after ECT nevertheless suggest that these two sets of items can differentiate between memory complaints due to depression and memory complaints due to amnesia.

The results from the self-rating scale described here do not constitute strong evidence for persisting memory dysfunction, because formal memory tests have indicated that memory functions substantially recover by 6 months after ECT. New learning capacity and memory for events that occurred many years previously appear to be fully recovered by 6 months after ECT (Squire and Chace, 1975; Squire *et al.*, 1980; Squire, 1980). Memory for events that occurred 1 to 2 years before ECT recovers substantially (Squire *et al.*, 1975; Squire and Cohen, 1978; Squire, 1980), although the possibility has remained that lasting impairment may occur for some information acquired during this time period (Squire *et al.*, 1980; Janis, 1950).

This discrepancy between memory complaint and the results of formal testing cannot yet be completely resolved. Nevertheless, the results with the self-rating instrument appear to answer certain questions about the experience of memory dysfunction and its causes. In the present case, it seems clear that (i) memory complaints long after ECT are qualitatively different from memory complaints that occur before ECT. Therefore, these complaints cannot be explained as recurrence of psychiatric illness, low self-esteem, or as a long-standing tendency to complain about memory; (ii) in a variety of respects, memory complaints long after ECT resemble complaints reported shortly after ECT, at a time when amnesia can be demonstrated with formal tests. Memory complaints might therefore be based on this earlier experience and reflect a persisting tendency to question whether memory functions have fully recovered. This information should be useful in counseling patients about memory problems. In addition, the methods described here may prove useful in evaluating other examples of memory complaint such as those associated with head injury, psychotropic drugs, or normal aging.

We thank Neal Con-
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memory complaint (Items 5, 9), and memory experiences likely more than each of the other two factors, the results of items can differ and memory com-

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Patients' perspectives on electroconvulsive therapy: systematic review

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Abstract

Objective To ascertain patients' views on the benefits of and possible memory loss from electroconvulsive therapy.

Design Descriptive systematic review.

Data sources Psychinfo, Medline, Web of Science, and Social Science Citation Index databases, and bibliographies.

Study selection Articles with patients' views after treatment with electroconvulsive therapy.

Data extraction 26 studies carried out by clinicians and nine reports of work undertaken by patients or with the collaboration of patients were identified; 16 studies investigated the perceived benefit of electroconvulsive therapy and seven met criteria for investigating memory loss.

Data synthesis The studies showed heterogeneity. The methods used were associated with levels of perceived benefit. At least one third of patients reported persistent memory loss.

Conclusions The current statement for patients from the Royal College of Psychiatrists that over 80% of patients are satisfied with electroconvulsive therapy and that memory loss is not clinically important is unfounded.

Introduction

Electroconvulsive therapy is generally indicated for depression that is resistant to treatment. The procedure, which involves the application of electrodes to the head to induce a convulsion, is carried out under general anaesthetic. Although electroconvulsive therapy is less commonly used today than in the past, over 11 000 patients receive it in England annually.¹ Nearly one fifth of patients receive treatment under a special section of the Mental Health Act 1983.

The Royal College of Psychiatrists' fact sheet states that more than eight out of 10 depressed patients who receive electroconvulsive therapy respond well.² "Electroconvulsive therapy is the most effective treatment for severe depression and people ... report that it makes them feel 'like themselves again' or that 'life is worth living'." Although reviews on attitudes to electroconvulsive therapy in the 1980s concluded that patients found treatment beneficial and that they were satisfied with it, this is currently opposed by individual patients and groups.³ We aimed to examine the

sources of this controversy and to assess the debated distinctions between efficacy, effectiveness, and satisfaction.⁴ Efficacy is restricted to what can be measured in a controlled clinical trial, often over a short period. It will not necessarily predict the effectiveness of a treatment in a real life situation, still less will it predict satisfaction. For instance, a systematic review of randomised controlled trials investigated evidence of the efficacy of electroconvulsive therapy as measured by symptom scales completed by a mental health professional.⁴ But these ratings may not be the same as perceptions of relief of symptoms by patients themselves. For example, in one study similar numbers of patients were regarded as improved by themselves and by health professionals, but in 20% (n=13) of cases these were different individuals.⁵

Patients' perceptions of benefit are likely to be based on broader considerations than just the relief of symptoms. They may take into account the amount and length of time symptoms are relieved (clinical benefit) as well as any side effects. One side effect is memory loss. The Royal College of Psychiatrists' fact sheet states that while memory of recent events may be affected by electroconvulsive therapy, "in most cases this memory loss goes away within a few days or weeks although some patients continue to experience memory problems for several months. As far as we know, electroconvulsive therapy does not have any long term effects on your memory or intelligence."² Some patients, however, report severe and longstanding memory losses after electroconvulsive therapy, and these will influence decisions on the risks and benefits of treatment.

Despite these disagreements there has been little systematic study of patients' views about the effectiveness and safety of electroconvulsive therapy. We aimed to ascertain patients' attitudes on the perceived benefit of treatment, as distinct from clinically rated outcome, and reported memory loss after treatment.

Methods

We searched the databases Psychinfo, Medline, Web of Science, and the Social Science Citation Index for papers and reports of patients' views on treatment with electroconvulsive therapy (see bmj.com for search terms). Bibliographies were also hand searched. Articles were excluded that concerned lay or

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professional opinion, children or adolescents, or where not all the patients had received treatment.

Of the 27 papers identified, 26 were authored by academics or researchers and conducted in psychiatric facilities. A reference group enabled us to identify nine reports written either by patients or in collaboration with them. The work of Communicate, the user group at the Maudsley hospital, is awaiting publication, but we had access to its raw data. Although our searches included global sources, articles written by patients were confined to the United Kingdom in all but one case.

Analysis

We calculated the proportion of patients with positive responses to questions on effectiveness of treatment and the 95% confidence intervals. Positive responses were defined as an affirmative response to the statements "electroconvulsive therapy is helpful" or "I would have electroconvulsive therapy again." A Forrest plot was produced on the raw (proportion) scale as to whether electroconvulsive therapy was considered helpful, with normal approximation standard errors.

The research studies were rated on four methodological variables. These were selected from either previous research (setting and interviewer), preliminary analysis of the data (interval between treatment and interview), or the social science literature.⁸

Interval between treatment and interview

We considered the interval between treatment and interview because the benefits of treatment may be short lived and side effects only apparent later. The scores were: 0 for during course of treatment or maintenance treatment; 1 for within four weeks or predischARGE; 2 for 1-6 months; and 3 for more than six months.

Number of questions

As a few brief questions are likely to produce less engagement than a more exploratory list of questions, we scored: 1 for five or less questions; 2 for 6-14 questions; and 3 for 15 or more questions.

Complexity of interview

With simple response options there was less scope for patients to express their opinions whereas multiple choice questions or semistructured interviews allowed more complex opinions to be recorded. The scoring system was: 1 for dichotomous responses; 2 for simple Likert scales; 3 for complex Likert scales or multiple choice; and 4 for a semistructured interview.

Setting of interview and status of interviewer

Conducting an interview has been shown to influence the willingness of patients to be critical about services.⁹ They are more likely to be critical when interviewed by a fellow patient in a neutral setting. Because the setting and status are always highly correlated, we amalgamated them into one category. The scoring system was: 1 for inpatients; 2 for same hospital or treating doctor; 3 for non-treating doctor or at home; 4 for day care or voluntary sector; and 5 for source independent of health services, and choice of setting.

Logit models were fitted to assess associations between positive responses and methodological characteristics and the distinction between clinical and patient studies. SPSS version 10 and Stata version 7 were used for the analyses.

Results

In 16 studies patients were asked if they found electroconvulsive therapy helpful and in 12 studies they were

Table 1 Details of perceived benefit of electroconvulsive therapy, date, sample size, and four scored methodological variables of studies eliciting patients' views on treatment. Values are numbers (percentages; 95% confidence intervals) of patients unless stated otherwise

Study	Country	Sample size	Perceived benefit of treatment		Methodological variables			
			Helpful	Would have again	Interval since treatment*	No of questions†	Complexity of interview‡	Setting of interview§
Freeman and Kendell 1980 ¹⁰	United Kingdom	156	129 (78; 71 to 84)	98 (59; 51 to 67)	3	3	4	2
Hughes et al 1981 ⁷	United Kingdom	72	59 (83; 73 to 91)	52 (72; 60 to 82)	2	3	2	2
Kerr et al 1982 ¹¹	Australia	88	64 (73; 62 to 82)	Not available	2	3	1	3
Aperia 1986 ¹²	Norway	30	21 (70; 51 to 85)	19 (63; 44 to 80)	3	2	3	2
Benbow 1988 ¹³	United Kingdom	54	39 (73; 58 to 84)	37 (69; 54 to 80)	1	3	2	1
Szuba et al 1991 ¹⁴	United States	25	19 (76; 55 to 91)	18 (72; 51 to 88)	1	1	2	1
Rogers and Pilgrim 1993 ¹⁵	United Kingdom	231	99 (43; 36 to 50)	Not available	3	3	3	4
Riordan et al 1993 ¹⁶	United Kingdom	37	21 (56; 39 to 73)	25 (67; 50 to 82)	2	3	4	3
Pettinati et al 1994 ¹⁷	United States	78	Not available	78 (98; 91 to 100)	1	1	1	1
United Kingdom Advocacy Network 1995 ¹⁸	United Kingdom	308	92 (30; 25 to 36)	55 (18; 14 to 23)	3	3	2	5
Mental Health Foundation 1997 ¹⁹	United Kingdom	107	32 (30; 21 to 40)	Not available	3	2	3	5
Bernstein et al 1998 ²⁰	United States	52	43 (83; 70 to 92)	41 (79; 65 to 89)	0	2	1	1
ECT Anon 1998 ²¹	United Kingdom	200	58 (29; 23 to 36)	Not available	3	Not given	Not given	5
Whealdon et al 1999 ²²	United Kingdom	150	121 (81; 74 to 87)	115 (77; 70 to 84)	1	1	1	1
Goodman et al 1999 ²³	United States	24	Not available	20 (82; 63 to 95)	0	3	1	2
Pedler (MIND) 2000 ²⁴	United Kingdom	418	150 (36; 31 to 41)	Not available	3	3	3	4
Manic Depression Fellowship 2002 ²⁵	United Kingdom	97	29 (30; 21 to 40)	Not available	3	3	3	5
Communicate†	Pending	45	20 (44; 30 to 60)	18 (41; 26 to 56)	2	3	1	4

*0=During course or maintenance electroconvulsive therapy; 1=within four weeks or predischARGE; 2=1-6 months; 3=more than six months.

†1=Five questions or less; 2=6-14 questions; 3=15 or more questions.

‡1=Dichotomous; 2=simple Likert scale; 3=complex Likert scale or multiple choice; 4=semistructured interview.

§1=Inpatient; 2=same hospital or same treating doctor; 3=non-treating doctor or at home; 4=voluntary or collaborative, day care or choice; 5=source independent of health services, choice of setting.

†Patient study.

asked if they would have the treatment again (table 1). The level of positive responses varied widely between studies (tests for heterogeneity: $\chi^2=370$, $P<0.001$, for treatment helpful, $\chi^2=256$, $P<0.001$ for would have treatment again). The Forrest plot for "helpful" shows that the patient led and collaborative studies report the lowest levels of positive responses; there was, however, an overlap in the confidence intervals (figure and table 1).

A funnel plot showed no evidence of publication bias among the clinical studies. No systematic relation was found between perceived benefit and the country, or region of the United Kingdom, where the research was undertaken.

Methodological variables

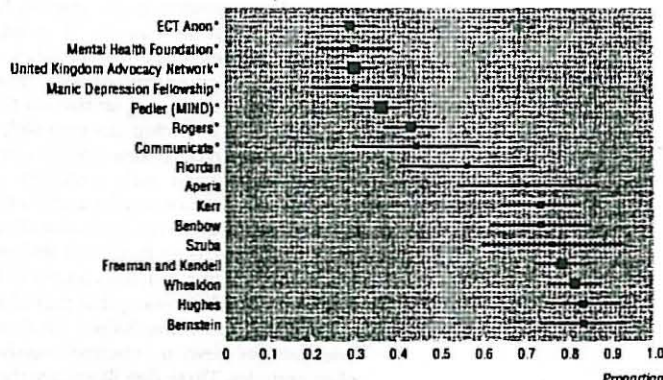
The number of questions, complexity of the interviews, and the interval before interview were intercorrelated (between number of questions and both the other two variables $r=0.54$, between interval and complexity $r=0.75$). The clinical studies tended to use fewer questions, less complex schedules, and a shorter interval, although the difference in complexity was not significant (see table 1).

Studies where the interviews were conducted soon after treatment, in hospital settings, by the treating doctor, were more likely to report positive views of electroconvulsive therapy (table 2). Studies with low complexity schedules, few questions, and a short interval were also associated with high perceived benefit. In the case of treatment considered helpful there was a clear hierarchy in setting, as coded from studies of inpatients (coded 1) to studies based in the community (coded 5).

When the analyses were repeated for the clinical studies alone, the effects were in the same direction and of a similar magnitude. Because of reduced sample sizes, fewer associations were significant. Within clinical studies, the number of questions remained significantly associated with treatment considered helpful, and complexity and interval were associated with whether the patient would have treatment again. In multivariate models, only setting remained significant.

Persistent memory loss

Of the 35 studies, 20 considered memory loss as a consequence of electroconvulsive therapy. Thirteen were excluded because data were not given or the interval between treatment and questions about memory loss was less than six months. In four of the remaining studies, respondents were asked specifically whether they had experienced persistent or permanent memory



Proportions of patients who would find electroconvulsive therapy helpful, by study. Lines indicate approximate 95% confidence intervals; size of box indicates precision. *Patient study

Table 3 Numbers (percentages) of patients reporting memory loss, by study

Study	Memory loss	
	Strict definition*	Loose definition†
Freeman and Kendall 1980 [§]	48/166 (29)	131/166 (79)
Kerr et al 1982 ^{††}	26/88 (30)	—
Pedler (MIND) 2000 ^{‡‡}	176/418 (42)	—
Squire and Slater 1983 ^{§§}	17/31 (55)	—
Pettinati et al 1994 ^{††}	—	40/78 (51)
Communicate‡	—	23/45 (52)
United Kingdom Advocacy Group 1995 ^{§§}	—	225/308 (73)

*Persistent or permanent memory loss.

†Any memory loss reported.

‡Patient study.

loss, and in four studies any reported memory problem was sought (one study reported on both; table 3).

The rate of reported persistent memory loss varied between 29% and 55%, but, unlike levels of perceived benefit, the rate did not seem to depend on whether studies were clinical or patient based, with relatively high levels being reported by both types of study.

Discussion

The methods used to elicit patients' views on electroconvulsive therapy influence the reporting of perceived benefit and willingness to repeat treatment. Variation in levels of perceived benefit was also related to the source of the research. Patient led studies reported lower rates of perceived benefit than clinical studies. This might be attributed to a selection bias,

Table 2 Associations between positive responses and methodological variables of patients' responses to electroconvulsive therapy. Values are odds ratios (95% confidence intervals) unless stated otherwise

Study characteristic	Treatment helpful (n=16)	P value	Would have treatment again (n=12)	P value
Schedule†:				
Interval between treatment and interview (scale 0-3)	0.542 (0.491 to 0.597)	<0.001	0.482 (0.422 to 0.551)	<0.001
Complexity of interview (scale 1-4)	0.804 (0.733 to 0.880)	<0.001	0.918 (0.815 to 1.035)	0.161
No of questions (scale 1-3)	0.353 (0.288 to 0.433)	<0.001	0.508 (0.432 to 0.597)	<0.001
Setting‡:				
Same hospital or treating doctor	0.861 (0.592 to 1.253)	0.435	0.727 (0.528 to 1.017)	0.063
Non-treating doctor or some at home	0.311 (0.154 to 0.629)	<0.001	0.863 (0.419 to 1.773)	0.688
Collaborative	0.198 (0.137 to 0.285)	<0.001	0.276 (0.146 to 0.530)	<0.001
Patient led	0.124 (0.092 to 0.169)	<0.001	0.090 (0.063 to 0.129)	<0.001

*Per unit increase in scale.

†Compared with inpatient.

with patient studies only selecting people who were antagonistic to treatment. The study by Communicate, the user group at the Maudsley Hospital, is, however, a prospective one, where the interview schedule was clearly stated to come from a patient group. This study still reports lower rates of satisfaction than any of the clinical studies, indicating that even with a prospective design, patient led or collaborative research finds lower rates of satisfaction with treatment. Our findings suggest the difference may be attributed to a tendency for clinical studies to take place soon after treatment, to use medical assessors in clinical settings, and to use brief questionnaires with low complexity for responses.

Qualitative data collected as part of a wider review supports the above conclusions but show, in addition, how patients' views on electroconvulsive therapy are often complex. These data illuminate the way in which patients make decisions about electroconvulsive therapy by weighing the risks and benefits of treatment. Most of the studies we reviewed used simple response categories that did not allow this complex trade-off and other attitudes to be described. One hypothesis is that many patients are not simply for or against the treatment or even are neutral about it. The concept of satisfaction and its measurement are also subject to these criticisms of oversimplification. Future research should include qualitative measures with representative samples of patients who have received electroconvulsive therapy.

Electroconvulsive therapy is a complex intervention comprising many stages and the involvement of many staff. Patients may have varying views about these different stages. As the literature we reviewed relied on global ratings, however, it was not possible to investigate each stage independently. The exception was the information and consent stage, which will be reported later.

Memory loss

Although the studies did not use consistent definitions or standardised ratings for memory loss, levels were between 29% and 79%. The levels were not determined by whether studies were clinician led or patient led, but the two types of study did differ in their analyses and interpretation of findings. Patient led research typically presents numerical results and illustrates these with quotations to show what the data mean in terms of patients' lives, whereas clinical researchers tend to undertake further statistical analysis of the data, sometimes ignoring the original data. For example, one study asked participants to assess the statement that "electroconvulsive therapy permanently wipes out large parts of memory."¹¹ The study then reported that people who had never received treatment were more likely to endorse this statement than those who had received it. It did not, however, comment on the finding that one third of those who had received treatment agreed with the strongly worded statement.

Another study controlled for depression in the analyses and found that memory loss continued to be significant.²⁷ Nonetheless, the authors concluded that long term memory loss was an important problem for only a small group of people and were doubtful about the causative role of electroconvulsive therapy.

The findings relate to the experience of persistent memory loss. Routine neuropsychological tests have

CONCLUSIONS

Around 11,000 people receive electroconvulsive therapy in England annually.

Controversy exists as to whether treatment is beneficial and whether patients are satisfied with it.

Patient views have never been systematically reviewed.

RECOMMENDATIONS

At least one third of patients reports significant memory loss after treatment.

Routine neuropsychological tests assess members of the public address the types of memory loss reported by patients.

Reported patient satisfaction with electroconvulsive therapy depends on the methods used to elicit a response.

been used in studies of electroconvulsive therapy to establish objective measures of memory loss and concluded that there was no evidence of persistent memory loss. It would seem that these are the studies on which the Royal College of Psychiatrists based its findings. The studies, however, typically measure the ability to form new memories after treatment (anterograde memory). Reports by patients of memory loss are of the erasing of autobiographical memories or retrograde amnesia. Thus the risks reported by patients do not appear in clinical assessments.

Controversy between medical opinion and patient organisations

We found possible sources of controversy between professional bodies and some patients and patient organisations. The levels of perceived benefit differed between patient led and clinician led studies because different methods were used and because in many cases these methods did not allow an adequate description of the complexity of subjective experience. Even where findings, such as persistent memory loss, did not differ between patient led and clinician led studies, the interpretations may have differed radically. It is therefore not surprising that disputes can arise between professionals and patients and that organisations should emerge that provide support and a forum for those who feel their treatment has not been beneficial.

Conclusion

Although clinical trials concluded that electroconvulsive therapy is an effective treatment,⁶ measures of efficacy did not take into account all the factors that may lead patients to perceive it as beneficial or otherwise. Studies of treatment are needed that are able to investigate a range of outcomes valued by patients. Important among these are factors that impact on effectiveness and satisfaction. Also important is loss of autobiographical memory, which is widely described but insufficiently systematically investigated.

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Lateral Cerebral Ventricular Enlargement in Chronic Schizophrenia

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To investigate if cerebral ventricular enlargement is associated with chronic schizophrenia, computerized tomography scans from 73 psychiatric patients were compared with 56 asymptomatic volunteers all less than 50 years old. Ventricular size was significantly greater in the subgroup of 58 chronic schizophrenic patients than in the controls. Of the chronic schizophrenic patients, 40% were outside the control range; 53% exceeded 2 SDs of the control mean. Neither duration of illness nor length of hospitalization correlated with ventricular size. The 44 chronic schizophrenic patients who had never been treated with electroshock therapy (EST) had larger ventricles than controls. A group of seven nonchronic schizophrenic patients also had enlarged ventricles; the eight patients who were either schizoaffective or nonschizophrenic did not differ from controls. This study shows that lateral cerebral ventricular enlargement is associated with chronic schizophrenia; it suggests that this is not a result of treatment.

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The observation that some chronic schizophrenic patients have enlarged cerebral ventricles dates back at least 50 years to Jacobi and Winkler,¹ who evaluated 19 patients with pneumoencephalography and diagnosed "internal hydrocephalus" in all but one. In more than 30 other pneumoencephalography studies of schizophrenic patients, cases of ventricular enlargement were found. Huang² and Bliss³ have briefly reviewed some of this extensive but relatively ignored literature. This pneumoencephalography finding, however, has been difficult to interpret because most of these studies used biased patient selection, inadequate controls, unspecified or nonuniform diagnostic criteria, and inadequately validated standards for normal ventricular size. In addition, changes in ventricular morphology may result from the pneumoencephalography procedure itself.^{4,5}

Postmortem neuropathological studies, primarily during the first half of the century, failed to confirm ventricular enlargement and led to the widely held notion that structural brain changes do not occur in schizophrenia. Recent studies, however, have shown that cerebral ventricles usually are smaller at autopsy than during life.^{6,7} Agonal premortem events, coincident illnesses, edema, and postmortem extraction and fixation procedures all may obscure premortem ventricular enlargement, especially if such

enlargement is only modest.⁸

The advent of computerized tomography (CT) introduced a noninvasive and more reliable method of assessing ventricular morphology during life.^{9,10} In the only other controlled study of schizophrenic patients evaluated by this method, larger ventricles were found in 17 chronically institutionalized patients as compared with eight normal controls.^{10,11} This finding has been questioned, however, because of the small control group, the advanced age of the patients (mean age, 58 years), possible effects of prolonged institutionalization (mean number of in-hospital years, 32), and possible effects of somatic treatment (eg, drugs and EST).^{12,13} The present study was designed to answer some of these questions.

SUBJECTS AND METHODS

Patients

Seventy-three psychiatric inpatients and recently discharged outpatients in treatment at St Elizabeths Hospital, Washington, DC, had CT scans. The patients were either volunteers in a clinical research division or patients from a general adult psychiatric unit that serves a designated catchment area. Only patients younger than age 50 years were asked to participate. The nature of the procedure was described and consent was obtained. Of 41 patients on the research wards during the six-month study period, 38 consented and cooperated with the procedure. Of 50 available patients in the general psychiatric unit, 35 consented and cooperated.

Diagnosis was determined by the ward psychiatrist prior to the CT scan, using Research Diagnostic Criteria (RDC).¹⁴ The diagnoses fell into the following categories: 65 cases of schizophrenia; four of schizoaffective disorder; and three of affective disorder. One patient's condition was diagnosed as being mental retardation (etiology unknown). The schizophrenic cases were then categorized by course of illness as follows: acute (present episode, including onset period, of less than six months' duration, with complete recovery from previous episodes) (three patients); subacute (present episode of less than one year's duration, with complete recovery from previous episodes) (four patients); subchronic (present episode of less than two years' duration or incomplete recovery from prior episodes) (16 patients); and chronic (more than two years of continuous illness) (42 patients). Length of illness, defined as current age minus the age of onset, and years spent living on a psychiatric inpatient ward were also determined. All schizophrenic patients, except for the acute ones, also met the criteria for schizophrenia proposed by Feighner et al.¹⁵ No patients had evidence of a neurological disorder except for two with oral-facial dyskinesia thought to be drug-induced. A complete blood cell count, VDRL, and laboratory studies to determine the values for total protein, albumin, calcium, phosphorus, cholesterol, glucose, BUN, uric acid, creatinine, total bilirubin, alkaline phosphatase, and SGOT, and a urinalysis were performed at least once within a year of the scan for each patient, and the results were either negative or within normal limits. The research patients had

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Table 1.—Ventricular Sizes for Control Groups

Age, yr*	Subjects†	
	Present	Barron et al ²³
20-29	3.1 ± 2.2 (N = 28)	3.1 ± 0.6 (N = 15)
30-39	4.1 ± 2.6 (N = 17)	4.2 ± 0.8 (N = 15)
40-49	3.8 ± 2.0 (N = 11)	4.4 ± 0.8 (N = 15)
Total population	3.5 ± 2.3 (N = 56)	3.9 ± 0.9 (N = 45)

*Age factor for present controls nonsignificant (one-way analysis of variance).

†Ventricular size expressed by mean ventricular brain ratio ± SD.

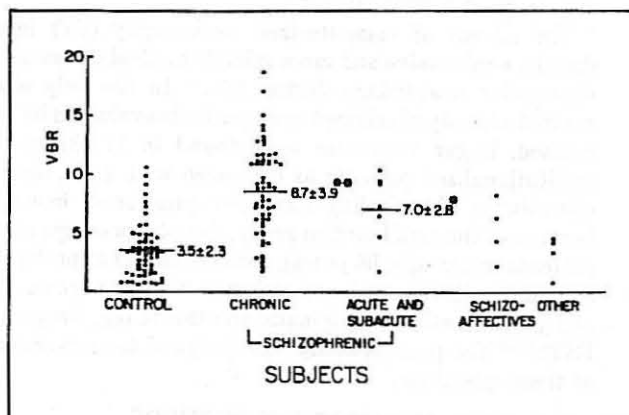


Fig 1—Ventricular size for control subjects and patients grouped by diagnosis. Bar represents mean ventricular brain ratio (VBR) ± SD. Asterisk indicates comparison to control group ($P < .006$) (Mann-Whitney U Test); double asterisk, comparison to control group ($P < .0001$).

thyroid function tests whose results were also normal. The results of Wechsler Adult Intelligence Scale (WAIS) testing done within one year of the CT scan were available for 19 patients.

Control Subjects

The control group consisted of 56 healthy, asymptomatic volunteers less than 50 years of age who had undergone head CT scanning for other research projects at the National Institutes of Health, Washington, DC. Forty-eight had first-degree relatives with Huntington's disease but had no clinical manifestations of the illness themselves. The remaining eight were volunteers between the ages of 40 and 50 years who were free of similar genetic risk. By linear measurements (according to the method of Enzmann and Lane²⁴), the CT scans of the control group could not be differentiated from those of a group of normal volunteer subjects with no known genetic risk for Huntington's disease."

Methods

All CT scans were performed with a 160 × 160 head scanner at the National Institutes of Health. A minimum of eight and a maximum of 12 cuts were taken for each subject; most scans consisted of ten to 12.

The CT slice showing the ventricles at their largest (through the body of the lateral ventricles in all except five controls where the body of the lateral ventricles was not visible) was selected for each subject. The chosen cuts, in the form of either self-developing prints or transparent film, were coded and mixed randomly for blind measurement. One person, using a fixed-arm planimeter, measured the area of the lateral ventricles and the area of the intracranial space in each cut. This involved tracing the perimeter of the lateral ventricles and the inner table of the skull, respectively. The instrument determines area by mechanical integration. Each area was traced five times and the mean used. Dividing the

ventricular area by the intracranial area and multiplying by 100 produces a ventricular brain ratio (VBR). This method for determining ventricular size was used because it has been shown to correlate well with computer-derived ventricular volume.¹⁴ Linear measurements (eg, Evans' ratio¹⁵ or Huckman's measure²⁰) were not used because they correlate poorly with ventricular volume and are less reliable.^{18,21,22} Expressing ventricular size as a ratio controls for magnification variance and provides a simple numerical value. To assess reliability, the same evaluator selected ten scans of varied ventricular size for repeated measurement at a later date. The correlation coefficient for original and repeated measures was 0.98. The mean difference in absolute magnitude was 0.7 VBR units (range, 0.06 to 1.29). Except where stated, all statistical tests for significance were two-tailed *t* tests.

RESULTS

Control Subjects

The VBRs for the control group categorized by age are given in Table 1. Age is not a significant determinant of ventricular size in this group. In Table 1, this group is compared with the one published by Barron et al,²³ the only other series of asymptomatic volunteers evaluated by the same method. Ventricular size does not differ significantly between these groups. The values for normal ventricular size agree with results from the four other studies²⁴⁻²⁷ that used comparable methods but "symptomatic" normals; however, the present control group defines a slightly broader normal range. It is possible that finding some subjects with ventricles larger than those seen in other studies of normals results from the inclusion of some subjects with preclinical Huntington's disease.

Patients

The following factors were not significant determinants of patient ventricular size: race, sex, patient location (ie, whether research or general psychiatric patients), and history of alcohol abuse (six patients). The difference in VBR between patients with a diagnosis of chronic schizophrenia (N = 42, mean VBR ± SD = 8.9 ± 4.0) and subchronic schizophrenia (N = 16, mean VBR ± SD = 8.1 ± 3.4) was not statistically significant; therefore, these groups were combined and are hereafter referred to as the chronic schizophrenic patients. Figure 1 illustrates the distribution of ventricular size for diagnostic groups and controls.

Chronic Schizophrenics.—The difference in ventricular size between chronic schizophrenic patients and control subjects is highly significant ($P < .0001$, Fig 1). Of the chronic schizophrenics, 23 (40%) are outside the control range; 31 (53%) are beyond 2 SDs of the control mean. This level of significance holds for each decade compared individually; age, itself, is not a significant factor in ventricular size for this group of chronic schizophrenic patients (Table 2). The distribution of VBR values does not differ significantly from a Gaussian distribution ($\chi^2 = 11.3$, $df = 7$).

Ventricular size does not correlate significantly with either length of illness (Fig 2) or duration of hospitalization (Fig 3). Of the chronic schizophrenic patients, 13 were ill for less than five years (mean VBR ± SD = 8.0 ± 4.0); ten were ill for more than 20 years (mean VBR ± SD = 10.0 ± 4.6). Ventricular size does not differ significantly between these groups. Comparisons of years of illness and hospitalization for the chronic schizophrenic and

Table 2.—Ventricular Size by Age for Controls and Chronic Schizophrenic Patients*

Age, yr	Controls	Chronic Schizophrenics
<30	3.1 ± 2.2 (N = 28)	8.1 ± 3.7 (N = 30)
>30, <40	4.1 ± 2.6 (N = 17)	9.3 ± 4.2 (N = 15)
>40, <50	3.8 ± 2.0 (N = 11)	9.4 ± 3.6 (N = 13)

*Two-way analysis of variance: age factor, $F = 1.19$ ($P = .31$); group factor, $F = 65.44$ ($P < .0001$).

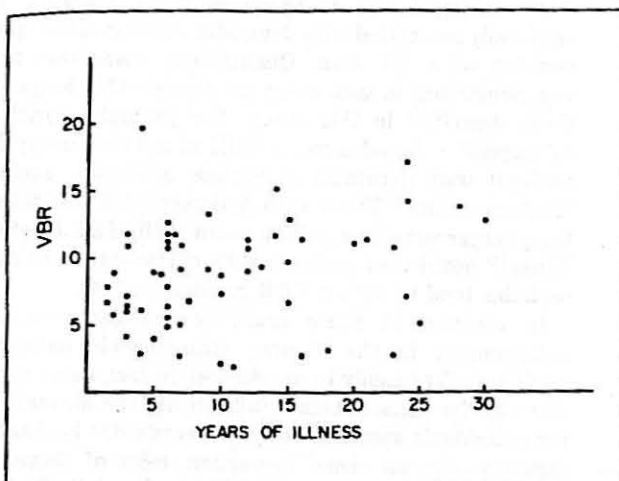


Fig 2.—Correlation of ventricular brain ratio (VBR) with length of illness for chronic schizophrenic patients (Pearson $r = .21$; nonsignificant).

nonschizophrenic patients are further evidence against a primary role for these factors. The mean length of illness in years \pm SD is 10.6 ± 7.7 for the 58 chronic schizophrenics, whereas the four nonschizophrenics had a mean length of 11.2 ± 5.3 . The mean length of hospital stay was 6.5 ± 7.1 years for the chronic schizophrenics, and it was 7.9 ± 5.6 years for the four nonschizophrenics. These intergroup comparisons were nonsignificant (Mann-Whitney U Test). Thus, the lengths of illness and hospitalization for the nonschizophrenics are considerable and not significantly different from those for the chronic schizophrenics.

A history of electroshock therapy (EST) is associated with larger ventricles. The 16 patients who had received EST had larger ventricles (9.9 ± 5.2) than the 57 patients who had not (7.5 ± 3.3) ($P < .04$). However, EST does not account for the ventricular enlargement seen in the chronic schizophrenic patients. When the chronic schizophrenic patients with a history of EST are excluded and the remaining (44 patients, 7.9 ± 3.3) are compared with controls, the high degree of statistical significance persists ($P < .0001$).

Within one year of their scans, 19 patients had completed WAIS testing. These were all performed as part of a routine evaluation and not as follow-up for suggested deficits. Table 3 gives the results of this testing. On all WAIS subtests, as well as the full scale, chronic schizophrenic patients performed significantly worse than the other patients. Among the chronic schizophrenic patients, no significant correlation could be demonstrated by linear regression analysis for any WAIS subtest and ventricular size.

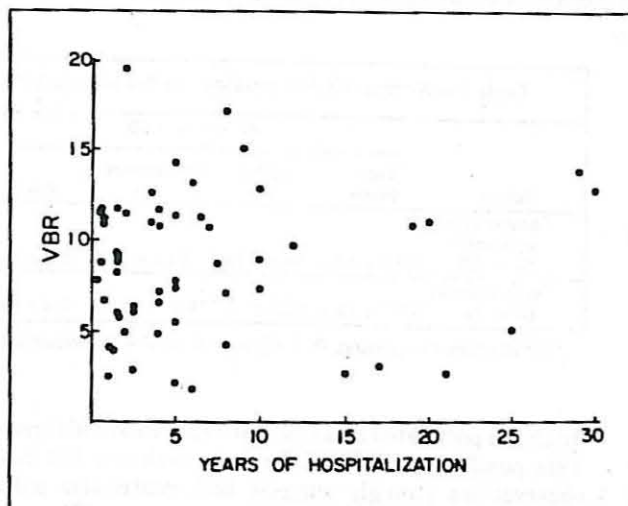


Fig 3.—Correlation of ventricular brain ratio (VBR) with length of inpatient hospital stay for chronic schizophrenic patients (Pearson $r = .05$; nonsignificant).

Other Patients.—The acute and subacute schizophrenic patients have been grouped together in Fig 1 because they are small in number and because they do not differ significantly with respect to ventricular size ($P = .1$, Mann-Whitney U Test). Comparing this combined group to controls, however, may be misleading because only the subacute patients differ significantly from the control subjects (Mann-Whitney U Test, $P < .004$). Neither the schizoaffective nor the nonschizophrenic patients had significantly larger ventricles than the control subjects.

COMMENT

This investigation demonstrates that lateral cerebral ventricular enlargement is associated with chronic schizophrenia. Ventricular size, determined quantitatively from CT scans, is significantly greater in the chronic schizophrenic patients than in the asymptomatic control subjects. This finding challenges the widely held belief that structural brain abnormalities do not occur in schizophrenia and demands careful interpretation and further research.

The etiology and clinical importance of this finding are unknown. Since the data do not differentiate between cause and association, it is important to consider the possibility that ventricular enlargement may relate not to the illness but to its treatment.

Impact of Treatment

Institutionalization.—Chronic institutionalization could theoretically lead to ventricular enlargement if it predisposed patients to malnutrition, intercurrent infections, metabolic deficiency states, or other stigmata of neglect. Although prospective studies are needed to evaluate this possibility conclusively, the present investigation found no support for it. None of the patients in this study had been confined to so-called back wards and neglected. None were clinically malnourished or suffering from other major illnesses. More importantly, the length of hospitalization did not correlate significantly with ventricular size.

Medication.—Neuroleptic medication is another important part of the treatment of chronic schizophrenia that

Table 3.—Wechsler Adult Intelligence Scale Results*

Group	IQ Scores \pm SD			V - P
	Full-Scale	Verbal (V)	Performance (P)	
Chronic schizophrenics (N = 14)	78.4 \pm 17.4	83.1 \pm 16.6	74.8 \pm 17.4	8.4 \pm 8.7
Other patients (N = 5)	106.8 \pm 12.3	109.0 \pm 6.6	102.4 \pm 15.3	6.6 \pm 9.4

*For all group comparison, $P < .05$, except for V - P comparison.

might be postulated as a cause of ventricular enlargement. This possibility is also difficult to evaluate, but several observations strongly suggest that ventricular enlargement occurs independent of drug treatment. First, Johnstone et al^{10,11} included in their study four patients who had never received phenothiazines. These patients had significantly enlarged ventricles ($P < .01$). Second, many of the patients with abnormal ventricles described in the pneumoencephalography studies also had never received neuroleptics.^{1,2,28-30}

Third, the present study found no significant correlation between length of illness, a variable closely related to the amount of drug treatment, and ventricular size. In retrospective studies of patients who have discontinued treatment records and have been in and out of numerous hospitals, it is impossible to accurately reconstruct the amount of neuroleptics consumed. The duration of illness, however, is probably closely correlated with the amount of drugs received, especially in patients who have an unremitting illness. The lack of a correlation between this variable and ventricular size suggests that drug treatment did not exert a cumulative dose-related effect in this group of patients. It does not exclude the possibility that ventricular enlargement was the result of idiosyncratic drug toxicity or of high doses of neuroleptics administered for short periods of time. It is important to note, however, that several schizophrenic patients had received high doses of neuroleptics for prolonged periods of time and did not have enlarged ventricles.

Fourth, the four patients in this study who were not schizophrenic, although too small in number to adequately control for drug and institutionalization effects, serve as an interesting comparison group. These patients had received antipsychotic doses of neuroleptic drugs for behavioral control during most of their treatment. Their durations of illness and hospitalization were equivalent to those of the chronic schizophrenic patients. Their ventricular sizes, however, clustered around the normal mean.

EST.—This is another somatic treatment that could possibly cause ventricular enlargement. Although EST was associated with larger ventricles, significant ventricular enlargement ($P < .0001$) was found in patients who had never received EST. Thus, its role in this study is unclear. Either EST further enlarged the ventricles of the patients treated with it, or it was used with greater frequency in patients who tended to have larger ventricles. The results of the few studies that have examined the effect of EST on ventricular size militate against the former premise. Johnstone et al¹⁰ found no EST effect. The pneumoencephalography studies, although problematic in determining

ventricular enlargement in schizophrenia, were suited to investigate the effects of EST. Of the five studies^{2,28-31} that compared ventricular size in treated and untreated patients, four^{2,28,29,31} found no difference.

Brain Morphology in Schizophrenia

Ventricular enlargement is a relatively nonspecific change in brain morphology. It occurs to some extent with advanced age²³ and is found in many diseases. It is commonly associated with dementia where it often appears striking on a CT scan. Quantitative measurements of ventricular size in such cases are considerably larger than those described in this study. For example, Synek and colleagues^{24,25} found a mean VBR of approximately 16 in patients with dementia of various etiologies, excluding "hydrocephalus." Those with hydrocephalus had substantially larger ventricles, with a mean VBR of 36. Jacobs and Kinkel²⁶ noted that patients with normal-pressure hydrocephalus tend to have a VBR greater than 25.

In contrast to these dramatic changes, ventricular enlargement in the chronic schizophrenic patients is modest and can easily be overlooked. In fact, mean ventricular size for these patients falls within the normal range for individuals approximately 70 years old.²³ Perhaps this explains why, on visual inspection, most of these scans were interpreted as being "within normal limits." Two neuroradiologists who independently "read" all films agreed on the diagnosis of ventricular enlargement in only ten cases (all chronic schizophrenics), and most of these were called "mild" or "borderline." These ten cases were distributed among 23 chronic schizophrenics with a VBR greater than 10, indicating an inconsistent correlation between subjective readings and actual ventricular area.

The 58 chronic schizophrenic patients in this study were, by definition, suffering from unremitting symptoms. Ventricular-size abnormalities in this group may not be typical of all patients with the diagnosis of chronic schizophrenia. Evidence is accumulating that with respect to various genetic, laboratory, clinical, and treatment variables, unremitting schizophrenic patients may have a different illness than patients who experience relapses and remissions.³² Whether ventricular enlargement is more characteristic of the unremitting illness merits further investigation.

Even within this patient sample, ventricular enlargement is not characteristic of every chronic schizophrenic. In 60% of the patients, ventricular size is within the control range. It is logical to question, therefore, whether those patients outside the normal extreme are a unique subgroup of chronic schizophrenic patients. A current research approach to the heterogeneity typical of populations of schizophrenic patients is to look for factors that may categorize them into more homogeneous groups.³³ Perhaps, ventricular size will prove useful in this respect. The ventricular-size distribution for this group of patients, however, does not easily lend itself to such subdivision. The distribution is not bimodal but normal, suggesting, instead, that ventricular size may be linked to a general characteristic of the group.

The results of this study conflict with the widely held assumption that cerebral morphology is normal in chronic schizophrenia. Although a CT scan abnormality is not a

histopathological diagnosis, ventricular enlargement in the absence of obstruction to CSF circulation implies cerebral atrophy. If a process resulting in cerebral atrophy occurs in some cases of chronic schizophrenia, neuroanatomical changes should be observable. It is widely assumed that neuropathological studies of schizophrenia failed to demonstrate such changes. (These studies have been reviewed elsewhere.³⁵⁻³⁹) In fact, numerous pathological findings were described, but most proved to be either nonspecific or not present in all patients with the diagnosis. Since the theoretical focus of these earlier studies was on finding a specific anatomical basis for schizophrenia, nonspecific and variable findings were not considered relevant. Two recent studies, looking primarily for nonspecific cerebral degenerative changes, found such pathology to be quantitatively increased in schizophrenic patients.⁴⁰⁻⁴¹

The assumptions that neuropathological findings in schizophrenia are important only if they represent the pathogenesis of the disorder and that they should be both specific and the same in all patients with the diagnosis may have misguided the interpretation of earlier findings. In light of these CT scan findings and the current conception that schizophrenia is not a single disease entity, the

chapter on neuropathology and schizophrenia may need to be rewritten. Perhaps, the gross and light microscopic pathology associated with chronic schizophrenia is subtle, nonspecific, and variable, like the clinical manifestations^{33,34,42} and like the changes in ventricular size seen with CT scanning.

CONCLUSION

Lateral cerebral ventricular enlargement on CT imaging was found to be associated with chronic, unremitting schizophrenia in a large group of patients. This finding was not shown to be the result of treatment. Postmortem studies are prone to artifacts that probably interfere with the observation of mild ventricular enlargement. Important questions for further research are raised.

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Paul Teychenne, MD, and Giovanni DiChiro, MD, made eight CT scans of normal volunteers available to us. Giovanni DiChiro, MD, also assisted in the implementation of this study and reviewed the scans and the manuscript. Paul O'Brien helped with the statistical analyses and Ann Reifman provided editorial assistance.

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ECT, Blood Pressure Changes and Neuropsychological Deficit

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Twenty psychiatric in-patients were studied before and after five electroconvulsive treatments for major depression. There were significant memory and neuropsychological changes after treatment, and significant elevations in depression rating scores, but they did not correlate with various measures of blood pressure elevation during treatment. The importance of ECT-induced amnesia is discussed.

Most recognise that memory dysfunctioning electroconvulsive therapy (ECT) is a side-effect, which is irrelevant to its efficacy (Otto, 1969). Unilateral, electrode placement was introduced to minimise the memory impairment found so with bilateral treatments. Unilateral ECT has been shown to be an effective treatment (Rasmussen, 1975) that does not produce the memory loss found with the standard treatment (Scam, 1968). Cognitive dysfunction in memory, is virtually the only side-effect of ECT, as it is currently unclear how ECT causes amnesia. A few investigators have attempted to elucidate mechanisms of post-ECT cognitive impairment, one hypothesis is that increased permeability which occurs during ECT is responsible.

Others (1977) demonstrated an increase in blood to brain of a test substance following seizures in humans. Hamilton (1979) showed learning and immediate memory impairment after ECT in a group of 27 in-patients. In a further attempt to account for any impaired cognitive function, methodological uncertainties in the study were a clear interpretation of the data. A significant correlation between performance on the Benton Visual Retention Test (Benton, 1963) and the degree of BP elevation. We have also now addressed the question of whether the rise in BP during the treatment is related to impairment of memory dysfunction and neuropsychological impairment which often occurs

Method

Patients were selected from daily admissions to the psychiatric unit of a private, university-affiliated hospital in November of 1983. Patients who were referred for ECT by their consultant and who

agreed to this form of treatment were potential candidates. The selection of bilateral ECT was the responsibility of the treating psychiatrist, with no input from the study group. Other inclusion criteria were age between 18 and 65 years, a diagnosis of primary affective disorder, depressed type (Feighner *et al.*, 1972) with less than 24 months' duration of symptoms, and a minimum Hamilton Depression Rating (HRS) (Hamilton, 1960) score of 18 (first 17 items).

The subjects were 20 psychiatric in-patients, five male and 15 female, 18 white and two black, between the ages of 21 and 63 (mean = 43.75). Those who gave written informed consent for participation were given the HRS, the Mini-Mental State Examination (MMSE) (Folstein *et al.*, 1975), a Personal Data Sheet (PDS) adapted from an instrument devised by Bidder *et al.* (1970) and a neuropsychological screening battery (NPB) devised by one of the current authors (RSD). This battery measured a number of different neuropsychological functions including orientation, digit span, spatial discrimination, grammatical comprehension, object naming, mental computation, finger identification, verbal fluency, temporal memory, verbal abstraction, visual memory, tactile discrimination, verbal expression, patterns, rhythm, verbal construction, written expression, reading, and construction. All of these measures were administered by a psychiatrist before the first treatment. Patients were also given the Beck Depression Inventory (BDI) (Beck, 1962) a self-rating scale. The same instruments were used to assess each patient, 24 to 48 hours after their fifth ECT, by a second psychiatrist who was blind to the blood pressure data.

For the treatments, all patients were pre-medicated with atropine sulphate (range: 0.4 to 1.0 mg), and were given 100% oxygen by facial mask just before the treatment. At the time of treatment, data were collected including BP before and after the administration of methohexitone (range: 35 to 80 mg) and succinylcholine (range: 25 to 60 mg); BPs were also recorded during the clonic phase of the seizure and at 30-second intervals for two minutes or more after cessation of visible seizure activity, to ensure that a peak BP had been attained and that it was returning to the pre-ECT baseline level. To minimise inter-rater variability of measurement, the BPs were all taken by the same doctor (BGK), using a standard sphygmomanometer. The duration of each peripheral seizure was also recorded. The ECT machine was the Medcraft 2. The voltage-intensity for each

TABLE I
Significant changes in test measures before and after five ECT (paired t-test)

	Pre-ECT mean S.D.	post-ECT mean S.D.	P <
Hamilton Depression Rating Scale Scores	26.58 ± 5.80	9.85 ± 8.14	.001
Beck Depression Inventory Scores	30.00 ± 10.76	12.50 ± 15.01	.001
Mini-Mental State Exam: Backward Spelling Errors	0.00 ± 0.00	0.55 ± 0.99	.05
Neuropsychological Battery: Verbal Fluency	8.80 ± 3.50	6.35 ± 2.89	.01
Temporal Memory	0.65 ± 0.49	0.20 ± 0.41	.001
Visual Memory	1.65 ± 0.67	0.90 ± 0.91	.001
Self Assessment of Mood	3.45 ± 1.85	5.83 ± 2.00	.001

stimulation (range: 100 to 170 mg) and the total number of stimulations necessary to elicit a seizure were recorded; the stimulus duration was one second in all cases.

The change of scores on the individual memory and neuropsychological test items before and after the five treatments were correlated with various parameters of BP and with depression scale scores, using Spearman's rho. For the Personal Data Sheet, an independent, blind rater (RSD) determined the similarity of the patient's verbatim response to individual personal data items before and after treatment on a five-point scale, with '1' indicating a completely different response after ECT or an inability to recall the requested information and '5' indicating an identical response before and after treatment. Paired t-tests were used to determine changes in depression scale scores, individual MMSE items, and individual neuropsychological test measures, before and after five ECTs. Patients were also asked to rate their mood and memory globally on a ten-point scale, with '1' indicating "the worst your mood/memory has ever been" and '10' indicating "the best your mood/memory has ever been". These estimates were obtained both before and after ECT.

Results

A number of items of the MMSE and the NPB were significantly affected by ECT. As expected, there was a significant improvement in the HRS scores, the BDI scores, and the self-rated global assessment of mood. The self-rated assessment of memory was not significantly different before and after the five treatments. The significant changes before and after ECT are recorded in Table I: significant deficits occurred in backward spelling (MMSE), verbal fluency (NPB), temporal memory (NPB), and visual memory (NPB).

However, none of these significant changes correlated with any measure of BP elevation. A number of different BP variables were used in the analysis; these included the mean peak elevation over pre-treatment level, the mean peak BP during the five treatments, the mean percentage rise over the pre-treatment level, the peak systolic BP, and the peak diastolic pressure. In order to correct for a possibly elevated pre-treatment pressure due to anxiety, which could obscure a significant finding, a second baseline level was also constructed to determine the mean BP during the days in hospital before the treatment, and

this baseline was then used to determine the elevation during treatment. This correction did not influence the findings: there were still no significant correlations of any of these BP parameters with memory or neuropsychological deficits. Nor was age a significant factor in either the memory deficits or the BP elevations. However, patients aged over 65 had been excluded from the study.

Other significant correlations are listed in Table II. The HDS scores after treatment correlated with the self-rated assessment of mood, but not before treatment. The self-rated assessment of memory correlated significantly with the BDI scores after treatment, but as with the HDS scores, not before treatment. There was also a strong correlation between the HDS and the BDI scores.

Discussion

The hypothesis of the study, that significant memory and neuropsychological deficits following ECT correlate with the rise in BP during the treatment, was not supported. Thus, our data did not support the finding of Hamilton *et al.* (1979). However, the study designs and neuropsychological test measures in these two studies were rather different. For example, they studied patients three to four hours after the treatment, while we did so 24 to 48 hours post-ECT. We chose that time interval so as not to confound the effects of the treatment itself with the effects of anaesthesia or the acute effects of a central seizure. It is, of course, conceivable that neuropsychological deficits which were present three to four hours after ECT could have disappeared one or two days later. We also used different memory and neuropsychological measures than those in the former study. We too found that visual memory was affected by ECT, but did not utilise the Benton Visual Retention test; although our measure of visual memory deficit did not correlate with BP parameters, it is possible that the Benton is more sensitive to such vascular changes.

The small number of subjects ($n = 20$) may account, in part, for the lack of significant findings. Another confounding factor may be the fixed

TABLE II
Significant correlations between psychological measures (Spearman's rho)

	n	r	P ≤
Hamilton Depression Rating Scale Scores × Beck Depression Inventory Scores	33	0.85	.01
Memory self-assessment (post-ECT) × Beck Depression Inventory Scores (post-ECT)	14	-0.57	.05
Mood self-assessment (post-ECT) × Hamilton Depression Rating Scale Scores (post-ECT)	20	-0.57	.01

number of treatments. We deliberately elected to study patients after five treatments for two reasons: (1) the different number of treatments each patient received on clinical grounds would have added a known confounding factor in terms of data analysis; and (2) to be consistent with the research design of other investigators (Squire & Chance, 1975). However, it is conceivable that five was too small a number of treatments to find a correlation between cognitive impairment and BP elevations. This possibility is rendered less likely though by the fact that we did find significant cognitive impairments after only five treatments, and yet the impairments did not correlate with the BP data. Another possibly confounding factor may be that our patients did not have a typical rise in BP after ECT. Our patients had a mean peak pressure of 181 mm Hg per treatment, which represents a mean rise per patient of 45 mm Hg over pre-ECT levels. This is

consistent with the reviews of the pertinent data by Fink (1979) and by Pitts (1982) for modified ECT, and does not seem to be of substantial concern.

Since cognitive impairment is such an important side-effect of bilateral ECT, it seems important to attempt to define as carefully as possible which aspects of the treatment are responsible for the deficit. Although our data do not support the idea that treatment-related BP increases are an essential aspect in ECT-related cognitive impairment, it is important to continue to search for the cause or causes of the impairment. If this important side-effect could be eliminated or even modified, it could only be a service to patients undergoing this effective treatment.

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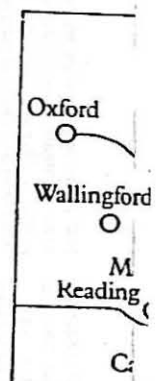
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PREVENTABLE BRAIN DAMAGE

Brain Vulnerability and Brain Health

Donald I. Templer
Lawrence C. Hartlage
W. Gary Cannon
Editors



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CHAPTER 8

ECT AND PERMANENT BRAIN DAMAGE

Donald I. Templer

Electroconvulsive therapy (ECT) is a very controversial treatment. It is a topic for which it is difficult to obtain an objective perspective because emotional undercurrents tend to run strong. It may, in this respect, be comparable to other emotionally laden issues such as ethnic differences in IQ and the bad effects of marijuana. Friedberg (1977), an outspoken critic of ECT, attributed the rise of ECT in the 1930s to the authoritarian political era in Europe in which 275,000 "inmates" in German psychiatric hospitals were starved, beaten, drugged, and gassed to death. On the other hand, Shukla (1981) stated, "Despite abhorrence in some quarters, it is still being practiced as one of the cheapest and safest, and yet one of the most effective, therapeutic techniques in the whole of medical science" (p. 569).

Hoffmann (1986) provided a scholarly discussion of the philosophical differences between those who favor and those who are opposed to ECT. He said that the former have a paternalistic philosophy and those who oppose it have libertarian and Kantian assumptions. He argued, "Paternalism does not pay much attention to patient education or self-esteem and libertarian ethics do not consider patient pain, fear, or dependency." He said that traditionally medicine has operated from a paternalistic point of view and that the attack on ECT can be viewed as arising from the valuing of freedom and autonomy in our society, plus the fact that politically educated persons have little tolerance for obligatory government rule. However, it is here noted by the present author that a study indicated that the psychiatrists and other mental health professionals who were more favorably disposed toward ECT were more experienced and knew more correct facts about ECT (Janicak, Mark, Trimakas, & Gibbons, 1985).

The use of ECT in the United States is decreasing. In fact, there was a 46% decrease from 1975 to 1980. However, even in 1980 there were 33,384 psychiatric patients given ECT (Thompson & Blain, 1987). In California, legislation in 1975 severely restricted the use of ECT. Nevertheless, from 1977 to 1983, 18,627 patients received a total of 99,425 ECT treatments in California, with little year-to-year variation (Kramer, 1985). ECT is far from becoming an obsolete treatment modality. And, because controlled research has demonstrated its efficacy, and

because it is especially valued in the recalcitrant cases of depression that do not respond to antidepressant drugs, it is not going to become an obsolete treatment unless and until more effective antidepressant drugs are developed. Janicak, Davis, Gibbons, Ericksen, Chang, and Gallagher (1985) published a meta-analysis that showed ECT to be clearly superior to the tricyclic antidepressants, the monoamine oxidase (MAO) inhibitors, simulated ECT, and placebo for severe depression.

This review covers eight areas relevant to the issue of permanent brain damage caused by ECT. These are (a) subjective report long after ECT, (b) human brain autopsy reports, (c) animal brain studies, (d) the brains of epileptics, (e) spontaneous seizures, (f) psychological test findings in patients with history of many ECT, (g) CT scan findings, and (h) magnetic resonance imaging (MRI) findings.

It is important that the reader be aware of the importance of distinguishing between the modern era of ECT administration with hyperoxygenation, muscle relaxation, and general anaesthesia, and ECT administration before the 1960s, which was less safe for the brain. A number of researchers and authorities have emphasized this distinction (Janicak, Mark, Trimakas, & Gibbons, 1985; Weiner, 1979; d'Elia & Raotma, 1975; Kendell & Pratt, 1983).

It is also important for the reader to bear in mind that unmodified ECT is often administered in third world countries (Weiner, 1984). The brains of poor people in poor countries also deserve protection. Shukla (1981) stated that in India, because of the shortage of anesthesiologists, most psychiatric centers, even in teaching centers, often have to use unmodified ECT that is followed by severe confusion. In India, ECT is used much more often than in the United States and is the mainstay of treatment for schizophrenia.

SUBJECTIVE REPORT

It is common knowledge that most patients complain of memory impairment during and after their course of ECT. There have been at least four studies that have investigated subjective reports of memory deficit long after it is expected that this impairment should have dissipated.

Freeman, Weeks, and Kendell (1980) placed a notice in a local newspaper in the United Kingdom asking for participation of subjects who had ECT at any time in their lives. In addition to the 13 subjects thusly recruited, there were 12 subjects who had been identified as complainers of impairment and referred by local psychiatrists. There were two main sorts of memory complaints. One was forgetfulness of such things as faces, names, phone numbers, and messages. The other was that of holes or gaps in past memories. Furthermore, these subjects' scores on neuropsychological tests were inferior to those of control persons. Needless to say, the generalizability of these findings is very limited because of

the subject selection process. Nevertheless, these findings do mesh with other studies concerning the memory complaints of patients who had a past history of ECT.

One hundred and sixty-six patients who had ECT from 1 to 7 years before were interviewed. Although a clear majority of the patients viewed the treatment as beneficial, 30% stated they believed the ECT produced lasting memory impairment (Freeman & Kendell, 1980).

Squire and Slater (1983) followed up 31 patients 3 years after ECT. Eighteen (58%) of the respondents said they did not think their memory was as good as for most people their age. Seventeen of these 18 persons attributed their memory difficulty to ECT.

In summary, there is a good accumulation of evidence that many patients complain of memory impairment attributed to their ECT years before. The authors of these studies pointed out that these reports do not provide conclusive evidence that such impairment actually exists. Nevertheless, these reports do legislate against a completely confident bill of health for ECT.

ANIMAL BRAIN STUDIES

Perhaps the most reasonable omnibus generalization is that many animal studies have been carried out, and that some authors have reported permanent damage and some authors have not reported permanent brain damage. In the 15-study review of Hartelius (1952), 13 of the 15 reported pathological findings that were vascular, glial, or neurocytological—or (as was generally the case) in two or three of these domains. However, as Hartelius pointed out, inferences of these studies tended to be conflicting because of different methods used and because of deficient controls. The research that Hartelius himself carried out was unquestionably the outstanding study in the area with respect to methodological sophistication and rigor. Hartelius employed 47 cats, 31 receiving ECT and 16 being control animals. To prevent artifacts associated with the sacrificing of the animals, the cerebrums were removed under anesthesia while the animals were still alive. Brain examinations were conducted blindly with respect to ECT versus control subject. On a number of different vascular, glial, and neuronal variables, the ECT animals were significantly differentiated from the controls. The animals that had 11–16 ECTs had significantly greater pathology than the animals that had received four ECTs. Most of the significant differences were with respect to reversible-type changes. However, some of the significant differences pertained to clearly irreversible changes such as shadow cells and neuronophagia.

The preponderance of human and animal autopsy studies were carried out prior to the modern era of ECT administration that included anesthesia, muscle relaxants, and hyperoxygenation. In fact, animals that were paralyzed and artificially ventilated on oxygen had brain damage of somewhat lesser magnitude

than, although similarly patterned as, animals not convulsed without special measures (Meldrum & Brierley, 1973; Meldrum, Vigourocex, & Brierley, 1973).

Needless to say, the generalization from these studies to humans is most difficult because of the great variation in stimulus parameters and other properties of the ECT, the different types of animals, and varying sophistication of design. Nevertheless, there does seem to be one generalization that applies to both animals and humans. It is possible to cause definite permanent brain damage through ECT, and it is possible to administer ECT with minimal or no damage. It is not a matter of whether ECT can produce permanent damage but a matter of in what circumstances it occurs.

HUMAN BRAIN AUTOPSY REPORTS

In the 1940s and 1950s, there were a large number of reports concerning the examination of brains of persons who had died following ECT. Madow (1956) reviewed 38 such cases. In 31 of the 38 cases, there was vascular pathology. However, much of this could have been of a potentially reversible nature. Such reversibility was much less with the 12 patients who had neuronal and/or glial pathology. In one case, the author (Riese, 1948), in addition to giving the neuronal and glial changes, reported numerous slits and rents similar to that seen after execution. Needless to say, patients who died following ECT are not representative of patients receiving ECT. They tended to be in inferior physical health. Madow concluded, on the basis of these 38 cases and five of his own, "If the individual being treated is well physically, most of the neuropathological changes are reversible. If, on the other hand, the patient has cardiac, vascular, or renal disease, the cerebral changes, chiefly vascular, may be permanent" (p. 347).

An interesting autopsy case report was presented by Lippmann et al. (1985). An 89-year-old woman with a long history of psychiatric illness died in 1982 after a documented history of 1250 bilateral treatments beginning in the 1920s. There was also some unsubstantiated evidence of her having received 800 additional ECTs. The authors stated that the moderate cerebral atrophy was consistent with her age and did not show old focal ischemic lesions or any evidence of brain injury resulting from the ECT. The author of the present chapter does believe that these clinical observations, even though based on an apparently nonblind determination, do argue in favor of the brain safety of ECT, especially since many of her treatments were administered prior to the modern era (1960 to present) of ECT administration. However, I raise the question of this woman's aging processes masking the ECT effects upon the brain many years earlier. I note that the authors stated that examination of the frontal lobes failed to reveal the sites of the cannula used in her prefrontal lobotomy in 1953.

CT SCANS

Calloway, Dolan, Jacoby, and Levy (1981) found no significant relationships between history of ECT and CT-scan-determined atrophy and ventricle size. However, a positive significant relationship between ECT and frontal lobe atrophy was found. Borderline significance was obtained with parietal atrophy. However, the authors appropriately raised the possibility that frontal lobe atrophy could have been present before ECT and in some way contributed to the patients receiving ECT.

Calloway, Dolan, and Jacoby (1988) found frontal lobe atrophy assessed by CT scans in 15 of 22 elderly depressed patients who had a history of ECT in contrast to four of 15 control patients without a history of ECT.

Weinberger, Torrey, Neophytides, and Wyatt (1979) found that those patients who had received ECT had significantly higher ventricle brain ratios than patients with no history of ECT.

One study found no relationship between CT scan assessed ventricular enlargement and number of life history of ECT in 27 bipolar patients (Pearlson et al. 1984). However, ECT was a minor part of this study and the authors did not specify how many patients received ECT. The details of ECT administration were also not specified. However, since the patients were from 18 and 40 years of age and presumably living in the United States, a reasonable assumption is that they received modern era administration with oxygenation, sedation, and general anaesthesia.

Kendell and Pratt (1983) presented CT findings on 12 patients who had a history of from 14 and 398 and a median of 94 ECT which were predominantly to the nondominant hemisphere. In two cases, CT scans were performed before history of ECT. In five cases, scans were obtained early in the course of treatment after two to six treatments. In all 12 patients, examinations were made at the end of therapy, which had lasted from over 1-40 years. Neither blind assessment of CT scans nor ventricle measurement pointed to effects of ECT upon the brain. Any increase in atrophy over the years was described as minimal and either bilateral or equally ipsilateral and contralateral to the treated hemisphere. The authors concluded that the absence of CT changes cannot exclude damage but that it is encouraging that CT showed no evidence of this occurring with prolonged courses of ECT taking place over widely varying period of time.

Kolbeinson, Arnoldson, Petruson, and Skulason (1986) found that 22 patients with a history of ECT did not differ in CT scan findings from control patients without a history of ECT. Neither atrophy scores nor ventricle brain ratios differentiated the two groups.

One patient was given a CT scan the day before and 3 hours after multiple ECT that consisted of 10 ECT in a period of 45 minutes (Menken, Safer, Goldfarb, & Varga, 1979). The patient was very confused, disoriented with

respect to time and place, and amnesic for events before the day of ECT. Nevertheless, no CT changes were observed. The findings would appear to point to the safety of the ECT. However, the present author is willing to entertain an alternative explanation. If the CT did not reflect the massive acute brain syndrome with gross disorientation, then it may not be capable of detecting minor changes in patients months or years after the ECT. Perhaps the CT scan is not the most optimal tool for ruling out brain changes resulting from ECT.

A reasonable generalization may be that CT scans have failed to provide a definitive perspective with respect to the matter of permanent brain damage.

MAGNETIC RESONANCE IMAGING

Coffey and colleagues (1988) reported on magnetic resonance imaging before and after ECT administered to nine depressed patients. Blind raters' assessments showed no significant differences between pre- and postECT in cortical atrophy and global comparison. There were also no significant changes in ventricle brain ratios. Furthermore, patients with preexisting brain disease showed no worsening. However, the authors did state: "Still these observations need to be confirmed in a larger number of subjects with techniques that will quantitate even subtle brain changes which might otherwise not be detected by qualitative clinical assessments. Further studies should also include patients with histories of previous ECT (to evaluate any potential cumulative effects) and should involve long-term follow-up studies including both subjective and objective measures of memory function" (p. 706).

A case report of a multiple sclerosis patient with magnetic resonance imaging before and after ECT is reassuring. There was no evidence of changes in white matter lesions visualized on spin-echo images (Coffee, Weiner, McCall, & Heinz, 1987).

In summary, the two studies using magnetic resonance imaging did not provide evidence of permanent brain damage resulting from ECT. However, more studies are needed.

PSYCHOLOGICAL TESTING WITH PAST HISTORY OF MANY ECTS

Goldman, Gomer, and Templer (1972) administered the Bender-Gestalt and the Benton Visual Retention Test to schizophrenics in a VA hospital. Twenty had a past history of from 50 to 219 ECTs, and 20 had no history of ECT. The ECT patients did significantly worse on both instruments. Furthermore, within the ECT groups there were significant inverse correlations between performance on these tests and number of ECTs received. However, the authors acknowledge that

ECT-caused brain damage could not be conclusively inferred because of the possibility that the ECT patients were more psychiatrically disturbed and for this reason received the treatment. (Schizophrenics tend to do poorly on tests of organicity.) In a subsequent study aimed at ruling out this possibility, Templer, Ruff, and Armstrong (1973) administered the Bender-Gestalt, the Benton, and the Wechsler Adult Intelligence Scale to 22 state-hospitalized schizophrenics who had a past history of from 40 to 263 ECTs and to 22 control schizophrenics. The ECT patients were significantly inferior on all three tests. However, the ECT patients were found to be more psychotic. Nevertheless, with degree of psychosis controlled for, the performance of the ECT patients was still significantly inferior on the Bender-Gestalt, although not significantly so on the other two tests.

Thus, the research using psychological tests with patients with history of many ECTs does suggest permanent impairment. However, one should bear in mind that retrospective studies do not permit the same confidence as do prospective studies. Also, the ECT in these studies was administered before the modern era of ECT.

BRAINS OF EPILEPTICS

It would seem that if an epileptic grand mal seizure produces permanent brain changes, then an electrically induced convulsion should also do so. In fact, inspecting the evidence with respect to epileptics may provide us with a conservative perspective in regard to ECT because the latter could produce damage from the externally applied electrical current as well as from the seizure. Experimental research with animals has shown that electric shocks (not to the head) produce more deleterious effects in the central nervous system than any other locality or system of the body. More pertinent are the studies of Small (1974) and of Laurell (1979) that found less memory impairment after inhalant-induced convulsions than ECT. Also, Levy, Serota, and Grinker (1942) reported less EEG abnormality and intellectual impairment with pharmacologically induced convulsions. Further argument provided by Friedberg (1977) is the case (Larsen & Vraa-Jensen, 1953) of a man who had been given four ECTs, but did not convulse. When he died 3 days later, a subarachnoid hemorrhage was found in the upper part of the left motor region "at the site where an electrode had been applied" (p. 18).

A number of postmortem reports on epileptics, as reviewed by Meldrum, Horton, and Brierley (1974) have indicated neuronal loss and gliosis, especially in the hippocampus and temporal lobe. However, as Meldrum et al. (1974) pointed out, on the basis of these postmortem reports, one does not know whether the damage was caused by the seizures or whether both were caused by a third factor intrinsic to the epilepsy. To clarify this issue, Meldrum et al. (1974) pharmacologically induced seizures in baboons and found cell changes that corresponded to those in human epileptics.

Gastaut and Gastaut (1976) demonstrated through brain scans that in seven of 20 cases status epilepticus produced brain atrophy. They reasoned, "Since the edema and the atrophy were unilateral and bilateral and related to the localization of the convulsions (unilateral or bilateral chronic seizures), the conclusion can be drawn that the atrophic process depends upon the epileptic process and not on the cause of the status" (p. 18).

A common finding in epileptics and ECT patients is noteworthy. Norman (1964) stated that it is not uncommon to find at autopsy both old and recent lesions in the brains of epileptics. Alpers and Hughes (1942) reported old and recent brain lesions associated with different series of ECT.

SPONTANEOUS SEIZURES

The reports of spontaneous seizures, which appeared in the pre-1960s ECT era, probably do not constitute one of the more definitive domains. However, this section is included to increase breadth of perspective.

It would appear that if seizures that were not previously evidenced appeared after ECT and persisted, permanent brain pathology must be inferred. There have been numerous cases of postECT spontaneous seizures reported in the literature and briefly reviewed by Blumenthal (1955), Pacella and Barrera (1945), and Karlner (1956). It appears that in the majority of cases the seizures do not persist indefinitely, although an exact perspective is difficult to obtain because of anticonvulsant medication employed and the limited follow-up information. Another difficulty is, in all cases, definitively tracing the etiology to the ECT, since spontaneous seizures develop in only a very small proportion of patients given this treatment. Nevertheless, the composite of relevant literature does indicate that, at least in some patients, no evidence of seizure potential existed before treatment and postECT seizures persist for years.

An article that is one of the most systematic and representative in terms of findings is that of Blumenthal (1955) who reported on 12 schizophrenic patients in one hospital who developed postECT convulsions. Six of the patients had previous EEGs with four of them being normal, one clearly abnormal, and one mildly abnormal. The patients averaged 72 ECTs and 12 spontaneous seizures. The time from last treatment to first spontaneous seizure ranged from 12 hours to 11 months, with an average of 2½ months. The total duration of spontaneous seizures in the study period ranged from 1 day to 3½ years, with an average of 1 year. Following the onset of seizures, eight of the 12 patients were found to have a clearly abnormal, and one a mildly abnormal EEG.

Masovich and Katzenbogen (1948) reported that 20 of their 82 patients had convulsive pattern cerebral dysrhythmia 10 months post-ECT. None had such in their pretreatment EEG. Nine (15%) of the 60 patients who had three to 15 treatments, and 11 (50%) of the 22 patients who had from 16 to 42 treatments

($\chi^2 = 10.68$; $p < 0.01$, according to our calculations) had this 10-month post-treatment dysrhythmia.

SYNTHESIS

There seems to be little doubt that ECT always produces an acute brain syndrome and that such remits over time. There seems to be little doubt that ECT has, at least in the past, caused permanent brain damage in some patients and has the capacity to continue to do so. There also seems to be little doubt that modern era ECT has greater brain safety than that administered prior to the 1960s. It appears that the overwhelming majority of persons who currently receive ECT in the United States do not suffer from massive cognitive deficits caused by the ECT.

What percentage of persons who receive ECT suffer some permanent impairment? What are, if any, the long-term effects of ECT in the "typical" or "average" ECT patient? Can we tell most of our patients there is absolutely and positively no danger of any permanent brain changes? These are the sort of questions for which we cannot provide confident answers. The present author believes that the difficulties in answering such questions are similar to the questions regarding whether or not alcohol and alcoholism are associated with brain pathology. We do know that a small amount of alcohol produces changes in the brain in all alcoholics and in all normal drinkers. We also know that all or almost all of these effects rather quickly dissipate. We also know that some alcoholics have massive and permanent brain pathology, for example, as seen in Korsakoff's syndrome. We know that a large percentage of newly abstinent alcoholics suffer from neuropsychological deficits. We know that in many of these patients there is improvement in neuropsychological testing over time and in some patients even a retrenchment in cortical atrophy. However, when we attempt to supply the details to answers about the typical or average alcoholic, or even the specification of who are average or typical alcoholics, the situation becomes less clear. This is the difficult situation we face with ECT patients. Some authors argue that ECT is hazardous to the brain and others argue it is safe. I believe they are both right.

The crucial questions at this point in time are those centered around in whom and in what circumstances are the risks higher and lower. We are able to make some generalizations. There is research evidence that type of ECT administration does have an effect upon degree of confusion and amnesia. Higher levels of stimulus intensity, stimulus waveforms that are relatively inefficient in seizure-eliciting properties, and bilateral electrode placement are associated with greater confusion and amnesia (Sackeim, Decena, Prohovnik, Malitz, & Resor, 1983; Cronholm & Ottosom, 1963; Ottosom, 1960; Valentine & Dunne, 1969; Welner, Rogers, Welch, Davison, Welr, Cahill, & Squire, 1983; Sackeim, Portney, Neeley, Steif, Decema, & Malitz, 1986; Squire & Slater, 1978).

A convergence of evidence indicates the importance of number of ECTs. We have previously referred to the significant inverse correlations between number of ECTs and scores on psychological tests. It is conceivable that this could be a function of the more disturbed patients both receiving more ECTs and doing worse on tests. However, it would be much more difficult to explain away the relationship between number of ECTs received and EEG convulsive pattern dysrhythmia (Mosovich & Katzenelbogen, 1948). No patients had dysrhythmia prior to ECT. Also difficult to explain away is that in Table 1 of Meldrum, Horton and Brierley (1974), the nine baboons who suffered brain damage from experimentally administered convulsions tended to have received more convulsions than the five that did not incur damage. (According to our calculations, $U = 9$, $p < 0.05$.) And, as already stated, Hartelius found greater damage, both reversible and irreversible, in cats that were given 11 to 16 than in those given four ECTs.

Throughout this review the vast individual differences are striking. In the animal and human autopsy studies there is typically a range of findings from no lasting effect to considerable lasting damage with the latter being more of the exception. Most ECT patients do not have spontaneous seizures, but some do. The subjective reports of patients likewise differ from those of no lasting effect to appreciable, although usually not devastating, impairment. The fact that many patients and subjects suffer no demonstrable permanent effects has provided rationale for some authorities to commit the nonsequitur that ECT causes no permanent harm.

There is evidence to suggest that preECT physical condition accounts in part for the vast individual differences. Jacobs (1944) determined the cerebrospinal fluid protein and cell content before, during, and after a course of ECT with 21 patients. The one person who developed abnormal protein and cell elevations was a 57-year-old diabetic, hypertensive, arteriosclerotic woman. Jacobs recommended that CSF protein and cell counts be ascertained before and after ECT in patients with significant degree of arteriosclerotic or hypertensive disease. Alpers (1946) reported, "Autopsied cases suggest that brain damage is likely to occur in conditions with pre-existing brain damage, as in cerebral arteriosclerosis" (p. 369). Wilcox (1944) offered the clinical impression that, in older patients, ECT memory changes continue for a longer time than for younger patients. Hartelius (1952) found significantly more reversible and irreversible brain changes following ECT in older cats than younger cats. Mosovich and Katzenelbogen (1948) found that patients with pretreatment EEG abnormalities are more likely to show marked post-ECT cerebral dysrhythmia and to generally show EEGs more adversely affected by treatment.

RECOMMENDATIONS

It is recommended that more research be carried out on the safety and the hazards of ECT. Research on the unmodified ECT given in the developing

countries of the world would seem to be especially important. The present author does not have the credentials to make recommendations concerning the brain safety precautions that should be followed. However, I here present the recommendations of Frankel et al. (1978) and those of Weiner (1984).

Weiner (1984) recommended that a careful analysis of risks and benefits be determined; that the possibility of persistent memory defects should be part of the informed consent procedure; that ordinarily unilateral nondominant electrode placement should be used; that EEG monitoring should be carried out; that instruction in sophisticated use of ECT should be in psychiatric residency programs and continuing education opportunities; that inspections of ECT equipment should be made; that the public should be better informed about ECT; and that more research be carried out.

Frankel et al. (1978) recommended that the patients receive a thorough pretreatment medical examination; that there be designated ECT and recovery room areas with availability of equipment, drugs, and personnel in the event of cardiopulmonary or other complications; that ECT be administered with anesthesia and muscle relaxant drugs and ventilatory assistance with a positive pressure bag and 100% oxygen, with EKG, blood pressure and pulse rate monitoring, and with appropriate electrode placement and electrical parameters; that ECT only be used in those conditions for which ECT efficacy has been established; that medical contraindications be considered; that the severity and unremitting nature of the patient's suffering and incapacitation and unresponsiveness to other treatments be taken into account; and that proper informed consent be obtained.

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A Controlled Comparison of Simulated and Real ECT

By J. LAMBOURN and D. GILL

SUMMARY. Two groups of 16 patients with depressive psychosis took part in a controlled evaluation of electro-convulsive therapy (ECT). One group received six brief pulse unilateral shocks under conventional anaesthesia and muscle relaxation; the second group underwent the same procedure without receiving shocks. Outcome was assessed by a separate investigator using the Hamilton Rating Scale for Depression under double-blind conditions. The results showed that this form of ECT was only superior to the control treatment for one item in the scale, a finding which could have occurred by chance. 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 in general, and of brief pulse unilateral ECT in particular.

Introduction

Electroconvulsive therapy (ECT) was introduced to psychiatry before controlled trials of treatment were widely used. Although it has been in clinical use for over 40 years and is accepted as a highly effective therapy, particularly for depressive psychosis, there is little proof that either the passage of electricity or the resultant convulsion are the important components of treatment.

Four methods have been used to investigate the efficacy of ECT. Cronholm (1960) found the effectiveness of ECT with normal fit length superior to that of ECT where fit length had been shortened. Lancaster (1958), during his study of unilateral ECT, found that in 32 cases where ECT had failed to produce a convulsion the improvement in depression scores was significantly less. Others have compared ECT favourably with pharmacotherapy (Robin, 1962; Medical Research Council, 1965), and active ECT has been compared with a simulated procedure in which shocks have not been given (Wilson, 1963; McDonald, 1966; Brill, 1959).

Only the last-mentioned method takes account of the non-specific therapeutic effect of the ECT procedure, which is independent of the

shock itself. Unfortunately, the results found were conflicting and open to criticism.

Wilson compared ECT plus imipramine; ECT plus placebo imipramine; placebo ECT plus imipramine; placebo ECT plus placebo imipramine. The author himself admitted the inadequate size of the double placebo group, which contained only six patients, two of whom made a good recovery, and another did well. In the epilogue, reference was made to ECT and imipramine proving equally effective in equivalent dosage, but the author could not assess their superiority over placebo.

McDonald performed a similar study but with only four in the simulated ECT group. In his paper, their outcome was concealed in the data of those who received placebo amitriptyline, but as a combined group they did worse than those who received amitriptyline or real ECT ($P < 0.05$).

Using a mixed diagnostic group, Brill found no statistically significant difference in outcome with straight ECT; ECT plus succinylcholine; ECT plus thiopentone; thiopentone alone; nitrous oxide anaesthesia alone. This was true for the depressed patients in the group also. Because of these doubts, another comparison of

active and simulated ECT was necessary.

Me

Patients

Following Ethical Committee approval, 32 patients with depressive psychosis referred for ECT were screened. Both inpatients and outpatients were included, but those with organic disorder were excluded. All patients who had received ECT in the three months prior to entry were excluded.

Procedure

Psychotropic drugs were discontinued the night before the first treatment. Each patient was given a simulated treatment first. The group was made up of 16 patients, 8 in each group, by procedure based on age, sex, and social class, so that the two groups were comparable on these variables. All patients received a standardized anaesthetic regimen for extremes of physiological state: sodium, 70 mgm; suxamethonium, 1.2 mg/kg; and atropine, 1.2 mg. Patients then received oxygen before the electrical treatment. The right temporo-parietal electrode was used (Lancaster (1958)). The treatment given to the control group was the same as that given to the patients who did not receive ECT. Those in the active ECT group received a pulse stimulus of approximately 1000 Hz using an Ectron Duopulse Model M1 electrically and mechanically. The conclusion of the project was to produce a bilateral seizure on every occasion. Patients were ventilated until a pulse had been established.

The control group, however, received a simulated procedure involving nursing care and expectation of a beneficial treatment. The referring doctors were not aware of the patient from the study and had not been achieved.

active and simulated ECT was deemed necessary.

Method

Patients

Following Ethical Committee approval, and having obtained informed consent, all right-handed patients with a diagnosis of depressive psychosis referred for ECT at Knowle Hospital were screened. Both in-patients and out-patients were included, but those with other psychiatric or organic disorder were excluded, as were those who had received ECT within the preceding three months.

Procedure

Psychotropic drugs (see Table I), except benzodiazepine hypnotics, were stopped the night before the first treatment. Allocation of each patient to a simulated or active ECT group was made by a constrained random procedure based on age (over or under 45) and sex, so that the two groups were balanced for these variables. All patients received a standardized anaesthetic regime (with dose modified for extremes of physique) of methohexitone sodium, 70 mgm; suxethonium cation, 50 mgm; and atropine 1.2 mgm intravenously. All patients then received four ventilations with oxygen before the electrodes were applied to the right temporo-parietal position described by Lancaster (1958). The only difference in treatment given to the placebo group was that they did not receive an electrical stimulus. Those in the active ECT group received a brief pulse stimulus of approximately 10 Joules from an Ectron Duopulse Mk. 4, which was checked electrically and mechanically before and at the conclusion of the project. This was noted to produce a bilateral modified convulsion on every occasion. Patients in both groups were then ventilated until spontaneous respiration had been established.

The control group, therefore, received an elaborate procedure involving loss of consciousness, nursing care and attention, and the expectation of a beneficial outcome. The treatments were given three times weekly and referring doctors were at liberty to withdraw any patient from the study if adequate improvement had not been achieved.

Assessments

These included:—

- (i) the Hamilton Rating Scale for Depression (Hamilton, 1960), completed by D.G. (who was blind to which treatment was being given) prior to and one day after 6 treatments and again one month later.
- (ii) a global assessment of improvement by the referring doctor one day after 6 treatments,
- (iii) days in hospital and treatments received in the month of follow up.

As a final check that the code had not been broken, referring doctors were asked to state which treatment they thought their patients had received.

Results (Table II)

The scores on the Hamilton Scale were found to be skewed, so non-parametric statistics were used in the analysis. The Wilcoxon matched-pairs signed-rank test (Siegal, 1956) was used, and as the hypothesis did not predict the direction of the result a two-tailed test was appropriate.

The overall outcome for the 32 patients in this study was quite good, only 5 failing to make any improvement after six 'treatments' given over a period of two weeks. These 5 patients all improved during the one-month follow-up period, and although 6 other patients were lost from the study one can conclude that the prognosis of depressed patients in an active treatment program is good. The contribution of spontaneous remission during this study remains an unknown factor because of the lack of a totally untreated control group.

Discussion

In this group of patients suffering depressive psychosis, six brief pulse unilateral ECT's did not produce a significantly superior therapeutic effect when compared with a simulated procedure. There could be several reasons for these results other than a conclusion that the electrical stimulus/convulsion component of ECT is an unimportant part of the ECT procedure. The diagnosis of depressive psychosis

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TABLE I
Demographic and pre-treatment assessment

										Assessments	
Age	Male	Female	Previous admissions for depression	Previous courses of ECT	Carney prognostic index depressive psychosis	Carney ECT indicator good	Carney ECT indicator bad	Anti-depressant medication prior to study	Pre-treatment Hamilton rating	Hamilton rating	Har Out impro 1-33 34-66 67-100
Real ECT group											
38			1	1				0	64	56	
60			4	3				75 mg. 1/52	50	28	
								Imipramine			
55			5	3				100 mg. 12/52	54	26	
								Amitriptyline			
44			4	1				0	52	18	+
56			3	2				75 mg. 2/52	72	2	+
								Imipramine			
50			0	0				0	32	12	+
53			0	0				0	36	2	+
36			4	4				0	52	10	+
59			10	10				0	42	8	+
52			0	0				0	46	8	+
64			2	1				0	58	16	+
								75 mg. 4/52			
60			1	0				Maprotiline			
								75 mg. 24/52	52	56	
69			2	2				Imipramine			
								45 mg. 8/52	30	20	
44			6	0				Phenelzine			
								2 mg. 2/52	58	46	
68			1	1				Flupenthixol			
63			1	1				0	76	56	
								100 mg. 4/52	22	12	+
								Imipramine			
Total	7	9	871	44	29	9	5	8	796	Total	376
Mean			54.4	2.75	1.8				50	Mean	24
Simulated ECT group											
42			1	1				100 mg. 8/52	70	38	+
								Dothiepin			
60			5	2				75 mg. 4/52	32	8	+
								Clomipramine			
63			2	2				0	48	43	
37			6	5				0	38	38	
54			7	5				150 mg. 12/52	54	68	
								Amitriptyline			
54			3	1				0	72	40	+
56			1	0				0	46	18	+
41			3	3				0	60	12	+
52			3	1				0	56	6	+
59			2	2				0	58	62	+
63			0	0				0	66	18	+
60			1	1				0	56	26	+
52			0	0				0	74	31	+
								150 mg. 12/52			
43			1	0				Amitriptyline			
								200 mg. 6/52	70	70	
66			1	0				Amitriptyline			
54			0	0				0	30	10	+
								0	34	10	+
Total	7	9	856	36	23	12	8	5	864	Total	498
Mean			53.5	2.25	1.4				54	Mean	31

TABLE I—Continued

Assessments after six treatments

Assessment after a further month

Anti-depressant medication prior to study	Pre-treatment Hamilton rating	Hamilton rating	Hamilton Outcome improvement 1-33%+ 34-66%++ 67-100%+++	Referring Doctors' global assessment of outcome	Referring Doctors' attempt to break blind code	Extra ECT after study	Extra antidepressant after study	Hamilton rating	Hamilton improvement 1-33%+ 34-66%++ 67-100%+++
Real ECT group									
0	64	56	+	+	Placebo	7	Amitriptyline	2	+++
mg. 1/52	50	28	++	++	N.K.	—	Imipramine	Lost	Lost
ipramine									
mg. 12/52	54	26	++	++	Placebo	—	Amitriptyline	42	+++
itriptyline									
0	52	18	++	+++	N.K.	—	0	16	+++
mg. 2/52	72	2	+++	+++	Active	—	0	14	+++
ipramine									
0	32	12	++	+++	N.K.	—	0	0	+++
0	36	2	+++	++	N.K.	—	0	Lost	Lost
0	52	10	+++	+++	N.K.	—	Amitriptyline	10	+++
0	42	8	+++	+++	Active	3	Amitriptyline	10	+++
0	46	8	+++	+++	N.K.	—	0	14	+++
mg. 4/52	58	16	+++	++	N.K.	—	Amitriptyline	Lost	Lost
aprotiline									
mg. 24/52	52	56	0	0	N.K.	5	0	14	+++
ipramine									
mg. 8/52	30	20	+	++	N.K.	5	Clomipramine	20	+
azine									
mg. 2/52	58	46	+	+	N.K.	5	Clomipramine	16	+++
ipenthixol									
0	76	56	+	+	N.K.	6	Amitriptyline	30	++
mg. 4/52	22	12	++	+	Placebo	—	Dothiapien	20	+
ipramine									
8	796	Total	376	+++ 6	+++ 6	Correct 2 31 ECT's Incorrect 3 6 patients N.K. 11	10 patients	208	+++ 8
	50	Mean	24					16	
Simulated ECT group									
mg. 8/52	70	38	++	+	N.K.	—	0	10	+++
othicpen									
mg. 4/52	32	8	+++	+++	Placebo	—	Clomipramine	14	++
ipramine									
0	48	43	+	+	N.K.	7	0	8	+++
0	38	38	0	+	N.K.	7	0	14	+++
mg. 12/52	54	68	0	0	N.K.	5	Amitriptyline	8	+++
itriptyline									
0	72	40	++	+++	N.K.	—	Amitriptyline	Lost	Lost
0	46	18	++	++	N.K.	4	Amitriptyline	4	+++
0	60	12	+++	+++	Active	—	0	30	++
0	56	6	+++	++	Placebo	—	Imipramine	0	+++
0	58	62	0	0	Placebo	—	0	50	+
0	66	18	+++	+++	Active	4	0	Lost	Lost
0	56	26	++	+++	Active	4	MRC Trial Tab	Lost	Lost
mg. 12/52	74	31	++	++	Active	—	Amitriptyline	12	+++
itriptyline									
mg. 6/52	70	70	0	0	Placebo	5	0	10	+++
itriptyline									
0	30	10	+++	+++	Active	—	Mianserin	0	+++
0	34	10	+++	++	N.K.	—	Amitriptyline	4	+++
5	864	Total	498	+++ 6	+++ 6	Correct 4 36 ECT's Incorrect 5 7 patients N.K. 7	9 patients	164	+++ 9
	54	Mean	31					12.6	

TABLE II

Hamilton rating scale for depression—scores before and after six treatments: ECT versus placebo (absolute change in scores)

Hamilton Rating Scale for Depression	Mean change		Wilcoxon test		
	ECT	Control	Z	P. Tail	N/T
Total Score	25.86	23.12	-0.31	0.38	16/62
Items					
1. Depressed mood	3.0	2.5	0.31	0.38	12/35
2. Guilt	2.0	1.76	0.43	0.33	12/35.5
3. Suicide	3.38	3.32	0.59	0.28	15/21.5
4. Insomnia (initial)	1.12	1.26	-0.22	0.41	11/30.5
5. Insomnia (middle)	0.38	1.62	-1.957	0.025	13/17.5*
6. Insomnia (delayed)	1.12	1.50	-0.40	0.34	11/28.5
7. Work and interest	2.62	2.12	0.08	0.47	12/38
8. Retardation	0.76	1.40	-1.19	0.12	8/9.5
9. Agitation	1.26	0.62	1.16	0.12	11/20
10. Anxiety (psychic)	2.76	1.76	0.91	0.18	13/32.5
11. Anxiety (somatic)	2.5	0.62	1.61	0.0537	12/18.5
12. Somatic symptoms gastro-intestinal	1.26	1.38	-0.31	0.38	6/9
13. Somatic symptoms general	1.12	0.62	0.90	0.18	12/27.5
14. Genital symptoms	0.88	1.26	-0.56	0.29	10/22
15. Hypochondriasis	0.62	-0.50	1.86	0.031	7/3*

* P. Two-tail significant at the 0.10 level.

might have been inaccurate, but we relied on the referring consultants' diagnosis, and as 77 per cent reliability has been found between psychiatrists using this criterion (Kreitman, 1961) this procedure was felt to be justified. The selection of out-patients might reflect the referring consultants' opinion that these had a better prognosis than patients admitted, and the randomization of two out-patients to the placebo group but not the active treatment group might be argued to have introduced a bias. This is not borne out, as the two out-patients made only a mean 38 per cent improvement and therefore slightly disadvantaged the placebo group. It could also be argued that only mildly depressed patients were referred for the study. As all the patients receiving ECT were screened, and only six patients fulfilling the research criteria did not enter the study, this is difficult to defend. The possibility was examined that a sub-group of patients did well but their responses were masked by our presentation of mean results; the distribution of good responses was similar between the groups, and no clinical features distinguished them. The Carney diagnostic index for depressive illness (1965) was

found to predict the outcome of treatment poorly in both groups. It has been argued that unilateral ECT is less effective than bilateral ECT (Royal College of Psychiatrists, 1977), and despite argument to the contrary (D'Elia, 1975), it is impossible to generalize the results of this study to include other techniques of administration. Valid criticism can be made that assessment after only two weeks was too early to allow the full therapeutic effect of ECT to develop, and that the arbitrary application of six treatments was not ideal (Barton, 1973). The referring clinicians were, however, able to add extra ECT or medication afterwards, and there was no difference in outcome between the groups one month later. That part of the study was unfortunately not blind, and it is difficult to interpret the findings meaningfully owing to the loss of six patients in that time.

Overall improvement on the Hamilton Scale showed a small trend in favour of ECT, and it is possible that if a larger sample of patients had been treated this difference would have been significant. Nevertheless, only two of the individual items in the scale were significant, one in favour of ECT and one in favour of the

control treatment, result occurred by chance.

The implication of the effectiveness of unilateral in previous investigations the attendant procedure administration of an mystique associated with treatment. Further studies are therefore indicated to placebo effect, particularly over a longer period, using parameters and electrode

In a recently published (1978) it was found that sinusoidal stimulus wave superior to a simulated interpretations of both the presented here are correct of unilateral and bilateral sinusoidal and brief previously re-examined.

Acknowledged

We wish to thank Mr C. Je statistical analysis, Dr Peter encouragement, also the consultants Hospital for their cooperation.

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absolute change in scores)

oxon test

p.	N/T
Tail	
1.38	16/62
1.38	12/35
1.33	12/35.5
1.28	15/21.5
1.41	11/30.5
1.025	13/17.5
1.34	11/28.5
1.47	12/38
1.12	8/9.5
1.12	11/20
1.18	13/32.5
1.0537	12/18.5
1.38	6/9
1.18	12/27.5
1.29	10/22
31	7/3*

treatment poorly
ied that unilateral
eral ECT (Royal
7), and despite
Elia, 1975), it is
ults of this study
f administration.
that assessment
early to allow the
to develop, and
of six treatments
) The referring
o add extra ECT
d there was no
the groups one
e study was un-
t is difficult to
ully owing to the

: Hamilton Scale
of ECT, and it is
of patients had
ould have been
by two of the
were significant,
favour of the

control treatment, results which could have occurred by chance.

The implication of these findings is that the effectiveness of unilateral brief pulse ECT shown in previous investigations is due in large part to the attendant procedures associated with the administration of an anaesthetic and the mystique associated with an unusual form of treatment. Further studies with simulated ECT are therefore indicated to explore this apparent placebo effect, particularly in patients treated over a longer period, using a range of stimulus parameters and electrode placements.

In a recently published study (Freeman, 1978) it was found that bilateral ECT using a sinusoidal stimulus waveform was significantly superior to a simulated ECT placebo. If the interpretations of both that study and the one presented here are correct, then the equipotency of unilateral and bilateral ECT, given with both sinusoidal and brief pulse stimuli must be seriously re-examined.

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ECT: II: Patients who Complain

By C. P. L. FREEMAN, D. WEEKS and R. E. KENDELL

SUMMARY Twenty-six subjects who complained of permanent unwanted effects following ECT were compared with two groups of control subjects on a battery of 19 cognitive tests. Many statistically significant differences were found in cognitive functioning, mostly attributable to the level of depression or medication in the complainers. However, after analysis of variance co-variance some differences still remained, indicating impaired cognitive functioning in the ECT complaining group.

The aim of the study was to identify a group of people who had specific complaints about electroconvulsive therapy (ECT), to catalogue their complaints and to assess their cognitive function. Results on a battery of cognitive tests were compared with results from a group of matched normal volunteers.

Methods

With the cooperation of the local evening newspaper (circulation 140,000 approx.), an article was written, entitled "Is there any harm in shock treatment?". At the end of the article readers who thought that ECT had had an adverse effect on them were asked to contact one of the authors:

So if YOU have had ECT, no matter how recently or how long ago, and reckon it has had an adverse effect on you, the group would be grateful if you would help by allowing them to test your memory and ability to think quickly, and see how you compare with other people. It would only take about an hour or so one afternoon . . . and there are no shocks in store. That's a promise!

We also asked consultants in the hospital to let us know of any patient who had complained about ECT.

Each complainer was given an unstructured interview by either C.P.F. or R.E.K. A note was made of their complaints, time and number of treatments, and whether they would willingly have ECT again. An attempt was made to

assess their mental state at interview to see if they were clinically depressed or otherwise ill and a note was made of their drug treatment, if any. This rough assessment was supplemented by completion of the Wakefield depression self-rating scale (Snaith *et al*, 1971) and the Middlesex Hospital questionnaire (Crown and Crisp, 1966). (All references are at the end of Paper III).

Subjects were tested for cognitive function by D.W. who did not know the nature of their complaints. A battery of 19 tests was used, as described with literature references at the end of Paper III. They covered visual design, verbal and spatial positional learning, verbal and visual memory, and there were two tests of remote memory, tests of delayed recall and recognition, a test of the ability to link faces with names, and tests of perceptual aptitude and concentration.

The subjects also filled in the Broadbent cognitive failures questionnaire which gives a self-rating of the subject's memory and concentration difficulties.

Controls—A group of volunteers who had not had ECT, and most of whom had not been psychiatric patients, were tested in exactly the same way. These were group-matched with the ECT complainers for age, sex, social class, educational level and intelligence. These volunteers were also obtained via an article in the same evening newspaper which asked for people

who would be prepared to help out with research projects at the Royal Edinburgh Hospital.

The samples—Twenty-eight people replied to the newspaper article, 10 men and 18 women. One woman had Alzheimer's disease and was attending the hospital as a day patient. She had insisted on coming when her husband brought the article to her attention. She was interviewed but was not testable.

Of the remaining 27, 14 had specific complaints about ECT (newspaper complainers), and 13 had misunderstood the article (newspaper non-complainers), and attended because they thought we wanted to have any views on ECT. They had either good or neutral things to say about the treatment. On closer questioning most had one or two very minor complaints about the treatment.

Twelve patients were identified via psychiatrists in the area, (hospital complainers), as they had told their doctors that ECT had produced enduring unwanted effects.

Results

The majority of complainers were women: 22 to 5 men (see Table I). There were only minor differences between the groups, except that the hospital complainers had last had ECT much more recently than either of the newspaper groups.

Nature of complaints

Case summaries are given in the Appendix. The commonest complaint by far was about some type of memory impairment. There were two main types of memory complaint: everyday forgetfulness such as forgetting faces or names, forgetting 'phone numbers or messages, forgetting things when going shopping; and secondly, holes or gaps in past memories.

Most subjects accepted that there might be poor memory for the time of their illness and course of ECT. Their complaints were of loss of periods, usually some months before ECT but occasionally afterwards. One subject complained he could not remember an annual summer holiday, another a wedding which occurred in months after ECT. The amount of distress the memory impairment caused varied considerably, but most found it irritating rather than incapacitating.

Other complaints were of epilepsy, patient 7, severe episodic pain (patients 7 and 21), personality change (patients 9 and 16), difficulty in knitting and fine hand function (patient 12), poor concentration (patients 22, 24 and 26). Many subjects had more than one complaint.

In all these cases the subjects definitely related the onset of the complaint to a course of ECT.

Only one complainant was against ECT in principle (No. 4). She felt it was a senseless and

illogical thing to pass an electric current across peoples' brains when they were depressed.

Of the total of 26 complainers 4 said they would have ECT again, 13 said they would never have it again under any circumstances and 9 said they were doubtful and it would depend on the circumstances, such as how depressed they were or whether antidepressants had failed. All the non-complainers said they would have ECT again.

Thus we did not attract any cranks or politically motivated complainers by our enquiries or, if we did, we didn't detect them. All but one of the subjects put their complaints in a reasonable balanced way, they seemed genuinely concerned by their difficulties and often relieved when told the results of their test scores. We did not get the impression that people were exaggerating their complaints or 'faking bad' on the cognitive test results.

Comparisons on non-cognitive tests

The subjects as a whole rated themselves as more depressed than the matched volunteer controls on the Wakefield scale. They also scored more highly than the volunteers on the Middlesex Hospital questionnaire (MHQ) on both total score and all subscales except hysterical personality. They rated themselves as having more cognitive failures on the Broadbent questionnaire. (See Table II). ECT complainers ($n = 26$) scored as more distressed on the same tests than ECT non-complainers ($n = 13$). (See Table III).

As drug taking varied greatly from subject to subject both in amount and type of drug, each subject was crudely rated on a score of 0-4 on the amount of psychotropic drugs taken. (Example: nitrazepam 5 mg taken the night before would score 1; diazepam 5 mg t.d.s. would score 2; amitriptyline 150 mg daily would

TABLE II
Comparison of ECT subjects with normal volunteers by mean scores

	ECT subjects N = 39	Normals N = 33	Significance
Wakefield self-rating scale	17.2	7.9	P < 0.001
Middlesex Hospital questionnaire			
Total symptom score	42.3	24.2	P < 0.001
Sub scales			
Free floating anxiety	10.1	5.5	P < 0.001
Phobic fear	6.3	3.3	P < 0.001
Obsessive symptoms and personality	9.7	6.8	P < 0.001
Somatization complaints	7.7	5.0	P < 0.001
Depression	8.5	4.6	P < 0.001
Hysterical extravert personality	4.4	3.9	NS
Broadbent cognitive failures questionnaire	73.9	63.9	P < 0.01

TABLE III
Relative illness of ECT complainers vs non-complainers

	ECT complainers (N = 26)	ECT non-complainers (N = 13)	Significance
Wakefield	19.1	13.2	P < 0.005
Middlesex Hospital questionnaire	43.3	40.4	P < 0.001
Medication	2.3	0.8	P < 0.005

TABLE I
Means of personal variables for the groups

	Newspaper non-complainers N = 13	Newspaper complainers N = 14	Hospital complainers N = 12	Normal volunteer controls N = 53
Male: Female	7:6	3:12*	2:10	1:2.3
Age (years)	56.8	50.6	52.7	52.9
Social class	3.1	2.9	2.8	2.7
Education in years of schooling	10.3	11.5	11.3	11.2
Total no of ECT	10.1	13.9	9.9	Not applicable
Time in years since ECT	12.8	9.3	2.6	Not applicable
IQ	104	104	102	108

* One woman untestable (Alzheimer).

score 3; diazepam 30 mg daily, barbiturates in doses of 200 mg daily, major tranquillizers if more than 100 mg daily of chlorpromazine or its equivalent would all score 4. Using this measure the complainers were taking more drugs than the non-complainers.

Thus on all measures of symptoms and medication the complainers scored more than the non-complainers and the subjects as a whole scored more than the normal volunteer controls. The non-complainers' scores were closer to the normal volunteers than to the complainers.

Comparisons on cognitive tests

When all ECT subjects were compared with the normal controls they were significantly impaired on eight tests, (See Table IV) and not impaired on eleven. They were slower than controls and their retention was poor; they couldn't remember a spoken paragraph of text as well; they couldn't put names to faces as well. They scored poorly on memories of their own past and on remembering personalities since the 1950s. In general, the test results appeared to match the subjects' complaints.

Despite rating themselves as more depressed, more anxious etc., and being on drugs, they

did as well as the matched volunteers on the majority of tests. Their new learning, (visual spatial and verbal), was not impaired and the remembered personalities from the 1930's, 1950's as well as controls.

Removing the 13 non-complainers from the ECT group and then comparing the complainers with normal controls alters the picture very little. The difference on personal remote memory becomes non-significant because the N is smaller and the means remain the same. Complainers were significantly better than non-complainers on one test and worse on one (Table V).

Summary of group comparisons

The picture emerges of a group of patients who have had ECT, who rate themselves as more depressed, having more symptoms in general and currently receiving more medication, and who perform significantly worse on number of cognitive tests than a group of volunteer controls. They also tend to be more impaired than a small group of non-complaining subjects who have also had ECT (See also Table VI).

A crucial question therefore arises: How

TABLE V
Comparison of ECT complainers vs ECT non-complainers

Test	ECT complainers (N = 26)	ECT non-complainers (N = 13)	Significance
Verbal learning	26.9	21.4	P < 0.005
Famous personalities of 1960's	11.4	12.3	P < 0.05
Logical memory	9.1	10.2	P < 0.05

TABLE VI
Comparison of ECT non-complainers with normal volunteer controls

Test	ECT non-complainers	Volunteer controls	Significance
Movement time (m/sec)	304	267	P < 0.005
Verbal learning	21.4	23.9	P < 0.005*

* ECT non-complainers less impaired

much of the poor performance of the complainers is due to their level of depression, and medication?

Analysis of variance

To try to answer this question the test results on all tests by all subjects and controls were put into an analysis of variance/covariance matrix with level of medication, level of depression, total symptom score on MHQ, age and social class as covariates. The object was to determine how much of the variance in test scores could be accounted for by these five variables, and whether having allowed for this the test results which had discriminated between subjects and controls still did so. We examine the previously significant differences test by test.

(a) Decision time and Movement time: These are measures of speed. Level of medication had a very large effect on results and level of depression a significant effect. There were smaller contributions from age and MHQ scores. When these factors were allowed for there was no significant difference between complainers and controls on either test.

(b) Famous personalities of 60's and 70's:

All five covariates had an effect and when they were allowed for the significant difference between controls and complainers disappeared.

(c) Logical memory test: The level of significance increases, so some of the covariates must have been operating in the direction of reducing any difference. In other words, the difference between complainers and controls becomes greater when the five covariates are allowed for.

(d) Face-name test: Social class was a significant covariate. All the other covariates had little effect and the difference between the complainers and controls remained significant, P < .05.

(e) Verbal learning: Medication had little effect on this test. The Wakefield score and total symptom score of the MHQ both had large effects and age had some effect. When all five covariates were allowed for the difference between complainers and controls remained significant, P < .05.

(f) Personal remote memory: All covariates had some effect on this test and when they were all allowed for the

TABLE IV
All ECT subjects vs normal volunteers on cognitive tests

Test	ECT subjects N = 39	Volunteers N = 53	Significance
Personal remote memory	38.8	40.5	P < .05
Logical memory	9.5	12.5	P < .001
Famous personalities from past 1960's	11.6	14.4	P < .001
1970's	13.9	15.8	P < .001
Verbal memory sensitivity	2.7	4.0	P < .001
Face-name connection	5.7	7.1	P < .02
Decision time (m/secs)	445.6	353.7	P < .001
Movement time (m/secs)	365.9	258.3	P < .001

(t test independent)

There were non-significant differences on:

Delayed recall, Delayed recognition, Famous personalities from past 1930's, 40's and 50's, Verbal learning, Spatial positional learning, Visual design learning, Anomalous sentence repetition, Perceptual aptitude, Incidental visual memory.

difference between complainers and controls just missed significance at $P < .05$.

Individual test results

So far we have only considered group comparisons on cognitive testing. Although there were a number of statistically significant differences between the means of the groups, when translated into clinical terms these differences are all small.

When the scores of individual subjects are examined there are some large deficits on some tests. A few patients scored well into the organic range on some measures. Sometimes there was a probable explanation for these deficits. For instance in patient 1, and possibly in patient 5, alcohol could be implicated. Patient 20 was taking large amounts of psychotropic medication. Patient 10 was on a considerable amount of medication and was very anxious. Patients 24, 26 and 27 were clinically depressed. However in a number of patients, particularly numbers 2, 14, 16 and 23, there seemed to be no ready explanation for their poor test results. They were virtually symptom-free, not taking drugs and as far as we could tell had no history of brain damage or excessive alcohol consumption.

The most convincing complainers who had no obvious explanation for their poor memory appeared to have nothing in common. They had not had excessive amounts of ECT, nor had their ECT been more recent than the other complainers, nor, as far as we knew were there any complications during their treatment. There were no comments in the case-notes about things going wrong such as prolonged hypoxia, missed fits, stuns, or excessive applications of electricity.

Discussion

The findings of this study must be interpreted with caution. We have not shown that ECT causes permanent memory impairment, though our results are compatible with this possibility. The study was designed as a descriptive one. What we have done is to describe in some detail a self-selected group of patients who complained about enduring unwanted effects of ECT. We have found that members of this

group do have some areas of impaired cognitive function, but on the majority of tests they performed as well as control subjects. On the tests where they were impaired, much of the impairment could be accounted for by other factors such as their level of depression and their level of medication. However, even when these factors and three other variables were taken into account not all the difference could be explained.

We are left with the fact that on three of a large battery of tests the ECT complainers performed significantly worse than the controls. Although these results are statistically significant their practical significance is less certain. The differences on test scores were not great when the groups as a whole were compared, and it is not possible to say whether the differences are certainly due to the ECT, or to something else which had happened in the period since the end of treatment. The length of time since the last course of ECT varied from nine months to thirty years and in the group that answered the newspaper advertisement the mean time since their last ECT was ten years.

There are two possible explanations for our findings. The first is that ECT does indeed cause some lasting impairment of memory in a small proportion of the people who receive it. The second is that our ECT complainers were simply people whose memories came in the lower half of the normal range, or had some mild impairment of memory for other reasons, and mistakenly attributed these failings to the treatment they had received years before. One man, for example, had a history of heavy drinking and had fallen down stairs and concussed himself on four occasions.

In our study on patients' attitudes to ECT (see Paper I, p. 12), we found that 12 per cent of patients agreed with the statement that "My memory now is better than ever". Had our newspaper article been worded differently it is conceivable that we could have attracted a group of people who had had ECT but whose memory was better than average.

What is clear is that the present subjects themselves clearly linked their memory impairment with having had ECT. Some were quite emphatic that their memory had been

average or above average beforehand. In a number of cases the memory disability had become apparent shortly after the course of ECT and had remained constant over many years. It may be that ECT does cause some degree of permanent memory impairment in a small proportion of the patients who receive it, but we consider that our own and other comparisons of carefully matched groups of patients receiving ECT and drug treatment indicate fairly convincingly that ECT does not normally produce such enduring effects on memory, though they do not prove that it never does so. It would, however, require a very large scale, and probably multicentre, prospective study to detect impairments that only affected, say, one patient in a hundred.

All references and the address of the authors will be found after Paper III.

APPENDIX:

Case Histories of Complainers

Numbers 1-14 were obtained through the newspapers, the rest from consultant psychiatrists.

1. *Male. Age 48: I.Q. 98. ECT 2 courses 1960-1972 for severe depression. Complaints—Slight but persistent difficulty in remembering numbers and names. Cognitive function—Impaired on nearly all tests, particularly remote memories, face-name test.*

Impression—Not depressed or otherwise ill when seen. Past history of alcohol consumption amounting to 50 pints of beer per week. Four episodes of concussion requiring overnight admission to hospital. Diastolic BP 120 mm Hg. Seems to be considerably underestimating his deficits.

2. *Male. Age 53: I.Q. 116. ECT 1 course 1973, 5 treatments for depression. Complaints—Forgetful of names, gets easily sidetracked and forgets what he was going to do. One particular hole in his memory. Can't remember going to a wedding a few months ago, 6 years after ECT. Cognitive function—Poor on personal remote memory and on face-name, delayed recall impaired.*

Impression—Slightly anxious but not now depressed. Correct assessment of his deficits, for which there is no obvious reason.

3. *Male. Age 48: I.Q. 125. ECT 1 course of 27 ECT in 1972. Complaints—Two particular holes in his memory. One a few months before, the other a few months after ECT. Now has generally poor memory. Sure that memory was good before but doesn't know whether to attribute loss to ECT or illness. Wouldn't*

have ECT again. *Cognitive function—Good. Verbal learning somewhat impaired.*

Impression—Severe obsessional neurosis of 20 years standing. Takes 30 mg diazepam daily plus L. tryptophan. Scored highly on Wakefield depression inventory. Holes in memory probably would not be picked up on our tests, otherwise did better on tests than his complaints would suggest.

4. *Female. Age 19: I.Q. 90. ECT 1 course of 5-8 treatments when aged 16. Would never agree to have ECT again. Complaints—Very against ECT. No complaints about her own experience of treatments, but feels that it is a senseless and illogical thing to give people shocks across their brain. No memory complaints. Not anti-psychiatry in general. Cognitive function—Within normal range except for mild impairment of verbal memory.*

Impression—From her history she clearly had considerable adolescent problems. In the past she had cut her wrists and taken overdoses. ECT may well have been an inappropriate treatment. We were puzzled by her strength of feeling about ECT.

5. *Female. Age 57: I.Q. 96. ECT 1 course 1962. Would have ECT again if doctor recommended it. Complaints—Inadequate separation from other patients at time of treatment. Poor memory; has to write things down more than she used to. Not distressed by this. Cognitive function—Moderate impairment on a number of tests. Face-name, verbal memory, mental set shifting. Also slow on reaction time, cube analysis and card dealing.*

Impression—Main complaint was that her GP had made her alcoholic by suggesting she take a sherry at night to help her sleep. Claims she has now been abstinent for four years, and is a stalwart A.A. member. Her memory complaints and cognitive function were congruent.

6. *Female. Age 58: I.Q. 123. ECT 2 courses in 1967 and 1974. Would have ECT again if very depressed. Complaints—Gaps in memory going back 20 years, prior to last ECT. Not a serious problem. Not sure if it was her age. Otherwise no memory complaints. Cognitive function—Entirely within normal range except for personal remote memory which was 1 SD below mean.*

Impression—Intelligent veterinary surgeon. Entirely well at present. Complaints and test results congruent.

7. *Female. Age 48: I.Q. 100. ECT 18 treatments in 1962 following puerperal depressive illness. Doesn't know if would agree to ECT again. Complaints—2 grand mal fits 3 months after ECT followed by a large number of what were probably temporal lobe attacks. All fits stopped when her tricyclic medication was stopped. Intermitent severe pain radiating from*

her left temporo-mandibular joint to whole of left side. Told by neurologist 14 years ago that ECT may have damaged trigeminal nerve. *Cognitive function*—Delayed recall and decision time mildly impaired. Otherwise entirely normal.

Impression—Epilepsy seems definitely temporally related to ECT and antidepressant treatment. Current neurological opinion is that her unusual facial radiating pain could be temporomandibular arthrosis. Patient says that she wouldn't have associated pain with ECT unless neurologist had suggested it.

8. Female. Age 60: I.Q. 114. ECT 12 treatments in 1970. Would readily have ECT again. *Complaints*—Memory impairment. Says she was known in her bridge club as the 'computer' because of her good memory. Now has to write things down, and misplaces her keys and jewellery. *Cognitive function*—In middle of normal range for age and intelligence.

Impression—She takes at least 60 mg chlorthalidopoxide, imipramine 75 mg, thioridazine 50 mg and nitrazepam 10 mg daily out of habit. She was entirely well, leading a full and active social life but would seem to be dependent on her many drugs.

9. Female. Age 63: I.Q. 106. ECT 63 treatments in Canada in 1950. Wouldn't have ECT again. *Complaints*—Regards all current and past troubles as due to ECT including need for flupenthixol injections. Convinced that ECT has changed her personality and made her miserable. *Cognitive function*—Gross impairment of ability to learn visual designs. Decision and movement time both slow.

Impression—Fairly typical chronic schizophrenic who accepts that she had a mental illness before ECT but blames the chronicity of her illness and personality change on ECT.

10. Female. Age 35: I.Q. 88. ECT 6 bilateral treatments in 1969. Would have ECT again if drugs didn't work. *Complaints*—Difficult to remember phone messages. Gets mixed up when people tell her things. Dates this from ECT. Still has a good memory for faces. *Cognitive function*—Sentence repetition, and verbal memory impaired. Slow decision time, Porteus maze tests poor in both speed and errors.

Impression—Pleasant, very anxious woman with mild phobic illness. Takes diazepam 10 mg daily. Cognitive test results compatible with complaints.

11. Female. Age 33: I.Q. 89. ECT 6 bilateral treatments in 1977. Wouldn't have it again. *Complaints*—Multiple complaints about almost everyone who had tried to help her; psychiatrists, social workers, housing department etc. She felt ECT had generally made her worse but couldn't elaborate. Her memory was worse. She couldn't remember what her children told her. *Cognitive function*—Definite impairment on face-name test. Spatial learning and sentence

repetition mildly impaired. Decision and movement time slow.

Impression—A rather dramatic lady dressed all in white. Was tearful throughout the interview. Says she was completely crippled by her anxiety symptoms. In comparison with all her other complaints those about ECT were trivial.

12. Female. Age 57: I.Q. 110. ECT 1 course of ECT in 1969. Wouldn't have ECT again. *Complaints*—Difficulty knitting. She keeps making mistakes and has to knit slowly. Cannot retain things that her daughter and friends tell her. She feels her memory, progressively getting worse and that this can't be her age. *Cognitive function*—Moderate impairment of logical memory and decision time.

Impression—Manic depressive well maintained on lithium and tranylcypromine. Completely well when interviewed. Marked tremor, presumably from lithium, which may account for her knitting difficulty. *Cognitive function* objectively less impaired than her complaints would suggest.

13. Female. Age 67: I.Q. 118. ECT 7 bilateral ECTs four years before testing. *Complaints*—Memory impaired since ECT. *Cognitive function*—Gross impairment of visual design learning. Decision and movement time slow.

Impression—Moderately depressed and anxious when seen. Diagnosed as schizophrenic in past. It appears to have some residual symptoms.

14. Female. Age 52: I.Q. 102. ECT 1 course of ECT 18 years before testing. *Complaints*—Terrible memory since. At present wonders if it might be due to her age but has always blamed ECT. *Cognitive function*—Poor personal remote memory. Poor face-name test. Modest impairment on spatial positional learning and verbal learning.

Impression—Not ill or depressed when tested. Do seem to have some definite impairment which is not accountable for by drugs or depression.

15. Female. Age 40: I.Q. 96. ECT 1 course of treatments 14 months before testing. *Complaints*—Poor memory since ECT. Couldn't give example. *Cognitive function*—Only very mild impairment of decision time and delayed recall.

Impression—Anxious, rather obsessional lady. She attends day hospital. Not obviously ill. Rated here as moderately depressed.

16. Female. Age 63: I.Q. 102. 30 ECT 8 years before testing, for depressive illness. *Complaints*—Permanently damaged by ECT. Not very specific about how she was damaged, gave impression that her personality was changed. Denies any memory impairment. *Cognitive function*—Grossly impaired verbal learning. Face-name, sentence repetition and perceptual aptitude impaired.

Impression—Not psychiatrically ill now. The sort of woman who might attract the label hysterical personality disorder from some psychiatrists. Test results not explained by drugs or symptoms.

17. Female. Age 65: I.Q. 95. ECT 2 courses 9 months and 2 years before testing. Approximately 13 ECT. *Complaints*—Had thought memory impairment was permanent but now beginning to doubt this as her memory has recently improved. *Cognitive function*—Gross impairment of verbal and visual design learning. Verbal memory impaired. Face-name test and mental set shifting, sentence repetition all impaired. Decision time and movement time both very slow.

Impression—Only mildly depressed when tested. Surprising degree of impairment. In normal/mildly depressed range on Wakefield. Memory functioning so poor at one stage thought to be dementing.

18. Female. Age 45: I.Q. 115. ECT 1 course nine months before testing. Would have it again. *Complaints*—Memory still affected. Forgets where she puts things, can't remember names. *Cognitive function*—Very slow on card dealing; sentence repetition and delayed recall impaired.

Impression—On lithium, amitriptyline and trifluoperazine with marked side effects of drowsiness, slurred speech. Considering this she did remarkably well.

19. Female. Age 62: I.Q. 118. ECT 4 bilateral ECTs 2 years before testing and 1 course many years ago. *Complaints*—Memory permanently affected. *Cognitive function*—Definite impairment on face-name, spatial and visual learning decision and movement time slow.

Impression—Still depressed, rates herself highly on Wakefield and analogue scales.

20. Male. Age 55: I.Q. 101. ECT 2 courses 5 years before testing. *Complaints*—Multiple. Memory poor and gets confused, to such an extent that he loses jobs. Muscle aches and pains across chest. Believes all definitely due to ECT. *Cognitive function*—Careless impulsive errors on some tests. Face-name test very impaired, mental set shifting and visual incidental memory impaired.

Impression—A withdrawn, isolated and lonely man prone to bouts of depression. Takes amitriptyline 150 mg daily. Not depressed when tested.

21. Female. Age 39: I.Q. 94. ECT 6 unilateral ECTs 8 months prior to testing. Wouldn't have it again.

Complaints—Slight but definite memory impairment. Can't concentrate as well. *Cognitive function*—Verbal learning and verbal memory impaired. Mental set shifting very impaired.

Impression—Mildly depressed when tested. Chronically depressed and rather disillusioned with all psychiatric treatment.

22. Female. Age 44: I.Q. 96. ECT 6 bilateral treatments 20 months prior to testing. *Complaints*—Mild but definite memory impairment. Agrees it fluctuates with her mood. *Cognitive tests*—Verbal learning mildly impaired. Otherwise normal.

Impression—Not depressed when tested. 23. Male. Age 47: I.Q. 89. ECT 4 bilateral ECTs 2 years previously. Wouldn't have it again. *Complaints*—Poor memory, can't concentrate. *Cognitive function*—Impaired logical memory, spatial learning and mental set shifting.

Impression—Chronically depressed and anxious man. Still quite severely depressed when tested.

24. Female. Age 68: I.Q. 112. ECT 3 courses 1958, 1971 and 1972 (13 treatments). *Complaints*—Memory poor for everyday events: messages, faces. *Cognitive function*—Impaired on sentence repetition, face-name, verbal learning and memory, mental set shifting and positional learning.

Impression—Not depressed or otherwise ill when tested. Poor results not obviously explained.

25. Female. Age 55: I.Q. 116. ECT 16 bilateral ECTs 3 years previously. *Complaints*—All aspects of memory, learning and retention. Can't do her job as well because of it. *Cognitive function*—Very impaired on sentence repetition and verbal memory. Verbal learning impaired. Decision and movement time slow.

Impression—Chronically depressed. Scored 27 on Wakefield.

26. Female. Age 49: I.Q. 95. ECT 1 course of 6 bilateral ECT 19 months before testing. *Complaints*—All aspects of memory. Holes in memory from past. Can't retain things, has to make lists. *Cognitive function*—General poor performance. Face-name, delayed recall, personal remote memory. Mental set shifting and logical memory all impaired.

Impression—Unhappy lady with chronic marital problems. Scored highly (28) on Wakefield.

Electroconvulsive Therapy and Complaints of Memory Dysfunction: A Prospective Three-Year Follow-up Study

LARRY R. SQUIRE and PAMELA C. SLATER

SUMMARY: Self-reports of memory problems have been evaluated prospectively in depressed patients receiving bilateral ECT or unilateral ECT, and in depressed patients receiving treatments other than ECT. Depressed patients did not complain of poor memory at seven months after hospitalization. Compared to bilateral ECT, right unilateral ECT was associated with only mild memory complaints. At three years after treatment approximately one-half of the persons who had received bilateral ECT reported poor memory. These reports seemed to be influenced by three factors: (1) recurrence or persistence of conditions that were present before ECT; (2) the experience of amnesia initially associated with ECT and a subsequent tendency to question if memory had ever recovered; and (3) impaired memory for events that had occurred up to six months before treatment and up to about two months afterwards.

Electroconvulsive therapy (ECT) is a safe and effective treatment for depressive illness (Fink, 1979; Turek and Hanlon, 1977). Since memory impairment is its major side-effect (Squire, 1982; Harper and Wiens, 1975), considerable attention has been directed to evaluating its severity and duration. Memory impairment is greater after bilateral ECT than after right unilateral ECT (Squire and Slater, 1978; Reichert *et al.*, 1976; Fromholt *et al.*, 1973), and cumulates across treatments (Bidder *et al.*, 1970; Reichert *et al.*, 1976). As measured both by tests of remote memory and by tests of new learning capacity, memory functions gradually improve after treatment is completed. By several months after treatment, the ability to acquire new material is substantially normal (Squire and Chace, 1975). Memory for information acquired prior to treatment also recovers, but persisting memory loss can occur for material acquired near the time of treatment (Squire *et al.*, 1981).

Despite this evidence concerning recovery of memory functions after ECT, it has been recognized that this recovery is not always reflected in patients' own reports of their memory abilities. In one study of bilateral ECT, 10 of 16 patients reported at six to nine months after treatment that their memory was not as good as it used to be (Squire and Chace, 1975). In another study of 166 patients who had received either bilateral or unilateral ECT about one year previously, 30 per cent agreed with the statement that their

memory had 'never returned to normal after ECT' (Freeman and Kendell, 1980). In an effort to understand these memory complaints, we recently reported the findings of a prospective study of 35 individuals prescribed bilateral ECT who were assessed with a newly developed memory self-rating instrument on three occasions: before ECT, one week after ECT, and five to nine months later (Squire *et al.*, 1979). The results several months after ECT reflected a continuing experience of amnesia rather than an experience of depression. It was suggested that a patient's impression of his memory is altered by bilateral ECT and that this impression persists for several months after treatment.

These findings raise additional questions about the impact of ECT:

(1) Is this impression of impaired memory permanent or does it gradually subside?

(2) Does this impression of impaired memory imply perceived difficulty in new learning ability long after ECT or might it apply to a perceived gap in memory around the time of treatment?

(3) What is the impact of these memory complaints on patients' attitudes towards ECT?

To answer these questions we have completed a three-year prospective follow-up study of patients prescribed bilateral ECT. For purposes of comparison, prospective follow-up data up to seven months after hospitalization have also been collected for patients

Subjects

Bilateral ECT (Table 1)

This group was originally composed of 35 in-patients at five local hospitals who had been prescribed a course of bilateral ECT and who had been followed prospectively up to several months after their course of treatment (Squire *et al.*, 1979). Of these 35 patients, 5 were lost to our three-year follow-up. Two could not be located, 1 declined to be interviewed and 2 were excluded because they had received an additional course of ECT during the follow-up interval. One additional patient, who had been excluded from the original study because a test one week after ECT could not be given, was now included—making a total of 31 in the follow-up group. The diagnoses recorded on admission by the patients' various psychiatrists were primary affective disorder or severe depression (17); manic depressive illness, depressed phase (9); depressive neurosis (4); and schizoaffective disorder (1). For the three-year follow-up all individuals were first contacted by letter, then by telephone to arrange an appointment, and were subsequently visited in their homes. All interviewing was conducted by the same person.

Right unilateral ECT

This group consisted of 28 in-patients at six local hospitals who had been prescribed a course of right unilateral ECT. The diagnoses as recorded on admission were primary affective disorder or severe depression (19); manic-depressive illness, depressed phase (5); depressive neurosis (2); schizoaffective disorder (1); and unspecified personality disorder (1).

Depressed patients

This group consisted of 19 psychiatric in-patients at one of the participating hospitals who had been admitted for treatment of depressive illness. The specific diagnoses recorded on admission were primary affective disorder or severe depression (14); manic depressive illness, depressed phase (2); neurotic depression (2); and schizoaffective disorder (1).

Electroconvulsive therapy

Treatment was administered three times a week on alternate days following medication with atropine, methohexital sodium and succinylcholine. Decisions concerning the number of treatments were made by the patients' psychiatrists. In all cases, patients were described as having a modified grand mal seizure. For bilateral ECT, electrode placement was bi-temporal.

130–170 V for 0.6–1 sec). The remaining four patients received their treatments with a Reiter-Cedak Model SOS, a machine which delivers a series of unidirectional brief pulses. For right unilateral ECT, electrode placement followed the method described by D'Elia (1974) ($n = 19$) or McAndrew *et al.* (1967) ($n = 9$). All patients received their treatment with a Medcraft machine (140–170 V for 0.6–1 sec). The effects of right unilateral ECT on memory have been reported to be similar despite wide variation in electrode placement (D'Elia, 1976; D'Elia and Widepalm, 1974).

Test and procedures

Test 1: Memory self-rating scale

All 18 test items have been presented previously (Squire *et al.*, 1979). Subjects respond to 18 items that ask them to rate various aspects of memory functions e.g. 'now compared to before I began to feel bad and went to the hospital'. Ratings are made on a nine-point scale from -4 (worse than ever before) through 0 to +4 (better than ever before). For patients prescribed bilateral ECT, this scale had previously been administered one to two days prior to treatment, one week after the course of treatment and again seven months later (range 5–9 months, mean = 6.8 months). In the present study, 31 of the original 35 patients were tested about three years after ECT (range 25–41 months, mean = 34.7 months). For 28 patients prescribed right unilateral ECT, this scale was administered one to two days prior to treatment, one week after the course of treatment and again about seven months later (range 5–13 months, mean = 6.5 months). For 19 depressed patients not receiving ECT, the scale was administered during initial hospitalization and again about seven months later (range 6–10 months, mean = 7.4 months).

Test 2: Time line

This method of illustrating the temporal aspects of memory loss has been adapted from Barbizet (1970, p. 126). Subjects are shown a horizontal line 101 in. long and told that the line is intended to represent time. The line is labelled at several points from right to left: now, two years after ECT, one year after ECT, time of ECT (with the month and year written in for each subject), one year before ECT, two years before ECT, five years before ECT, ten years before ECT and more than ten years before ECT.

Subjects are asked to indicate on the line any periods of time that they have difficulty remembering, either prior to or after ECT. To obtain a similar estimate

for 31 patients prescribed bilateral ECT, time estimates were obtained before treatment, seven months after treatment and at the three-year follow-up.

Test 3: Structured interview

A structured interview was constructed that asked 12 questions about the ECT experience (see Table 1). This interview was administered to the group prescribed bilateral ECT on the occasion of the three-year follow-up.

Results

Fig 1 shows the results with the self-rating scale during the time of hospitalization to seven months later.

TABLE 1
Subject characteristics for electroconvulsive therapy (ECT) groups and control*

	Bilateral ECT ($n = 31$)	Right unilateral ECT ($n = 28$)	Depressed patients ($n = 19$)
Mean age at follow-up	45 (28–68)	40 (17–68)	45 (23–62)
Sex	25 F	22 F	13 F
Years of education	12.5	12.6	12.5
Number of treatments	10.9 (5–21)	9.2 (5–18)	
Number of patients with no ECT history prior to study**	19	18	19

* Values are expressed as means, ranges are in parentheses.

** None of the patients had received ECT during the preceding year. Twelve bilaterally treated (BL) patients and nine right unilaterally treated (RUL) patients had received one prior course of treatment, on average 6.7 years (BL patients) and 10.7 years (RUL patients) previously. One RUL patient had received two courses, four years ago and nine years ago.

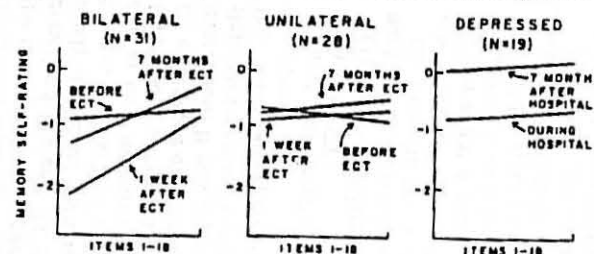


FIG 1.—Self-ratings of memory (on a -4 to +4 scale) by patients prescribed bilateral ECT or right unilateral ECT, and by a control group of depressed patients not receiving ECT. The self-rating scale was administered on two or three occasions as shown. The order of items appearing on the test was random. Here the group means obtained for each of the 18 items have been ordered according to the difference in the means obtained before and one week after bilateral ECT. Items to the left of each panel yielded the largest difference, and items to the right yielded the smallest difference. Data are here represented as best-fitting lines across the group means for all test items.

degree of memory complaints before ECT, either in terms of overall mean score ($F < 1.0$ [2, 75], $P > 0.1$) or in terms of the slopes of the regression lines ($F = 1.6$ [2, 75], $P > 0.1$). The average scores were -0.78 (bilateral ECT), -0.78 (unilateral ECT), and -0.77 (depressed group). These scores were all well below the zero level ($t > 10$, $P < 0.01$), confirming previous findings that depression is associated with quantifiable self-reports of poor memory.

Secondly, bilateral ECT, but not right unilateral ECT, produced initially a level of memory complaint well beyond that produced by depression alone. For bilateral ECT, the mean self-rating score changed from -0.78 before treatment to -1.47 one week after treatment ($F = 4.7$ [1, 29], $P < 0.04$). For unilateral ECT, the corresponding change was from -0.78 to -0.73 ($F = 0.4$ [1, 27], $P > 0.1$). Thus at one week after treatment, bilateral ECT was associated with a greater degree of self-reported memory impairment than unilateral ECT ($F = 5.7$ [1, 56], $P < 0.05$).

Thirdly, one of the effects of ECT was to change the pattern of self-ratings so that some items were rated worse than others. Thus for both bilateral and right unilateral ECT, the slope of the regression line was significantly different one week after ECT from that before ECT (for bilateral ECT, $F = 10.4$ [1, 29], $P < 0.01$; for unilateral ECT, $F = 9.7$ [1, 27], $P < 0.01$). Since patients are demonstrably amnesic one week after a course of bilateral ECT as measured by formal tests (Squire *et al.*, 1976; Cronholm and Bloomquist, 1959), the slope of the regression line observed at this time has been taken to reflect an amnesic pattern of performance. In this regard, it is significant that the slope of the regression line one week after bilateral ECT was greater than one week after unilateral ECT ($F = 9$ [1, 56], $P < 0.01$).

Fourthly, in marked contrast to the similarity between groups before ECT, the three groups differed from each other seven months later with respect to the severity of memory complaints ($F = 3.5$ [2, 75], $P < 0.05$). The average score at this time was -0.83 (bilateral ECT), -0.55 (unilateral ECT) and $+0.12$ (depressed group). The depressed group exhibited a significant improvement over their previous score ($F = 23.9$ [1, 18], $P < 0.01$). The bilateral ECT group were also improved over their score one week after ECT ($F = 7.5$ [1, 29], $P < 0.01$). Whereas the average scores for each ECT group were now similar to the before-ECT scores, they remained below the zero level ($t > 7$, $P < 0.01$).

Finally, the pattern of memory complaints reported seven months after ECT was different from that before ECT. The data reported here provide additional evidence that patients' estimates of their own memory abilities at seven months after treatment have been

affected to some degree by ECT. This conclusion follows from the finding that the slopes of the regression lines for the two ECT groups differed from their slopes before treatment (bilateral ECT: $F = 6.45$ [1, 30], $P < 0.02$; unilateral ECT: $F = 11.97$ [1, 27], $P < 0.01$). Both slopes were in fact indistinguishable from the slopes at one week after treatment ($F_s < 1$, $P > 0.1$).

Fig 2 shows the results for patients prescribed bilateral ECT, who were administered the self-rating scale an additional time three years after their course of treatment. Patients who received bilateral ECT seem to have experienced some persisting change in the perception of their memory abilities. This point follows from the finding that the scores at seven months and three years were similar ($F < 1$ [1, 30], $P > 0.1$), and that the slopes of the two regression lines were also similar ($F = 1.3$ [1, 30], $P > 0.1$). Moreover, the overall memory self-rating score at three years remained below the zero level ($t = 10.6$, $P < 0.01$) and significantly lower than the score of the depressed group at seven months after hospitalization ($F = 8.9$ [1, 48], $P < 0.01$).

Fig 3 shows how the 31 patients prescribed bilateral ECT described the time periods that they had difficulty remembering. The bars depict the median response of the group just prior to ECT (top), seven months after ECT (middle) and three years after ECT (bottom). The median time period affected was calculated separately for the months leading up to ECT and for the months after ECT. Prior to ECT, patients expressed difficulty remembering a median of five months into the past. Seven months after ECT, the median response was two years before ECT and three months after ECT. Three years after ECT, the median response was six months before ECT and two months after ECT.

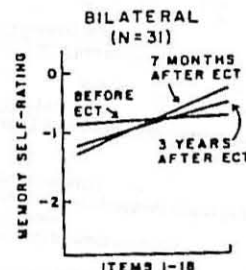


Fig 2.—Self-ratings of memory before, seven months after and three years after bilateral ECT. Scores are here represented as best-fitting lines across the scores for all test items.

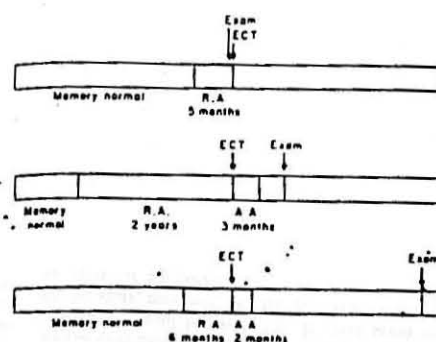


Fig 3.—Estimates of time periods that are difficult to remember obtained before (top bar), seven months after (middle bar) and three years after (bottom bar) bilateral ECT ($N = 31$). Shaded areas represent the median time period perceived as affected both from the period before ECT (i.e., RA retrograde amnesia) and after ECT (i.e., AA anterograde amnesia). Since the first time estimate was obtained just prior to ECT (top bar), the five months perceived as affected at that time presumably reflects memory problems associated with depressive illness.

The results obtained with these time lines make three general points about the experience of memory problems after bilateral ECT. First, the six-month retrograde amnesia (RA) reported at three years after treatment was significantly less than the two-year RA reported at seven months after treatment ($T = 17.1$, $P < 0.01$, Wilcoxon matched pairs, signed ranks test), and not measurably different from the five months perceived as affected before ECT was given ($T = 276$, $P > 0.1$). Shrinking RA has been reported routinely in the clinical neurological literature, particularly in the case of head trauma (Barbizet, 1970; Russell and Nathan, 1946; Benson and Geschwind, 1967), and has been demonstrated in the case of ECT using formal tests (Squire *et al.*, 1981).

Secondly, the anterograde amnesia (AA: loss of new learning capacity) associated with ECT was stable from seven months after ECT to three years afterwards. This finding corresponds to what should be expected for patients who gradually recover from a period of AA. Though the capacity for new learning returns, memory for the period during which this capacity was affected does not return, presumably because memories were not being established in the normal way during the affected period.

Thirdly, the typical individual prescribed bilateral ECT did not express difficulty at follow-up in remembering day-to-day events or events from the recent

past. Indeed, only 1 of 31 persons reported this experience. In addition, 4 other persons reported an intermediate experience in which memories up to the present time were described as 'hazy', but not 'blank' or 'impossible to remember'.

Table 11 shows the results of the structured interview. The data show that roughly half of the 31 individuals interviewed had a positive attitude about ECT. For example, 45 per cent (14) had no complaint about ECT as far as their memory was concerned. Fifty-five per cent (17) felt that their memories were not as good as those of other people of the same age and that this was related to their having received ECT. These two groups were of similar age (47 vs. 43) and had received similar numbers of treatments (10.5 vs. 11.3). In an effort to describe the characteristics of those persons who reported memory problems at three years after ECT, responses in the structured interview were examined separately for the 17 who reported memory problems and for the 14 who did not. There was a significant association between reports of memory problems and the report that ECT either did not help at all or helped for no longer than three months ($\chi^2 = 9.2$, $P < 0.01$). There was also a significant association between reports of memory problems and the response that ECT would not be requested if the same condition occurred again ($\chi^2 = 9.2$, $P < 0.01$).

We next examined the time line data (Fig 3) to determine if those individuals reporting memory problems at three years after ECT also reported a longer period of time that was difficult to remember than individuals not reporting memory problems. Using the data from Fig 3, no difference was found in the affected time periods (for the 17 with reported memory problems, median RA = 6 months, median AA = 2 months; for the 14 without reported memory problems, median RA = 8.4 months, median AA = 2.5 months).

We next compared scores on the memory self-rating scale (test 1) obtained three years after ECT by the 17 individuals with reports of memory problems at that time and the 14 individuals with no complaints. As might have been expected, these comparisons revealed significantly lower scores for the subjects who reported memory problems (mean = -1.62) than for those who did not (mean = -0.02) ($F = 15.6$ [1, 29], $P < 0.01$). The slopes of the two sets of scores across the 18 test items were similar ($F = 2.36$ [1, 29], $P > 0.1$).

Finally, we sought to compare these two groups on some objective measure of memory performance. Most of the subjects had previously participated in follow-up studies of remote memory functions conducted about six months after treatment, and had taken one or more of the following three tests: remote

TABLE II
Responses to structured interview three years after bilateral electroconvulsive therapy (ECT) (N = 31)

1. Did the treatment cause pain?		7. Would you agree to ECT with your doctor's recommendation?	
None	67.7%	Yes	51.6%
Slight or moderate	22.6%	No	41.9%
Excruciating	6.5%	Maybe	6.5%
No memory	3.2%		
0-4 scale: mean score =	0.5	8. Would you recommend ECT to relatives or children?	
2. What was the reason you received ECT?		Yes	45%
Depression	87.1%	No	55%
Other	9.7%		
No memory	3.2%	9. Given choice, would you choose ECT or antidepressant drugs for 3-6 months?	
3. How much did ECT help?		ECT	48.3%
Did not help	38.7%	Drugs	45.2%
Helped a little	29%	Neither	6.5%
Helped a lot	32.3%	10. Has ECT caused you embarrassment?	
0-9 scale: mean score =	4.5	No	58.1%
4. How long did helpful effect last?		Yes, because of memory problems	19.3%
To present time	42%	Yes, other reasons	22.6%
12 months	3.2%	11. Did you have memory problems during first week outside hospital?	
1-3 months	16.1%	Yes	87%
Not applicable	38.7%	No	13%
5. How did experience of ECT compare with expectation?		12. Do you think your memory is as good as for most people your age?	
As expected	35.5%	Yes	42%
Memory problems worse	9.7%	No	58%
More scary or stressful	3.2%	13. Do you feel ECT had anything to do with how your memory is now?	
Less scary or stressful	25.8%	Yes	54.8%
More effective	3.2%	No	3.2%
Less effective	22.6%	Not applicable	42%
6. Would you ask for ECT again?			
Yes	45%		
No	55%		

memory for current events (Squire *et al.*, 1981); detailed recall of former one-season television programmes (Squire *et al.*, 1981); and memory for temporal order of former television programmes (Squire *et al.*, 1976). Data from these tests were available for 13 of the 17 persons who reported memory problems (mean = 1.7 tests per subject) and 12 of the 14 persons who did not (mean = 1.3 tests per subject). To compare performance across tests, all scores for each test were converted to z scores. The two groups performed similarly on these tests (13 of those with reported memory problems, $z = -0.05$; 12 of those without reported memory problems, $z = +0.03$; $t = 1.0 [24]$, $P > 0.1$).

Taken together, these data indicate that 55 per cent (17) of our sample responded negatively to the direct question, 'Is your memory as good as for other persons your age?' and attributed their memory problem to ECT. Compared to persons not reporting memory problems, these persons also reported significantly more memory problems on the self-rating scale. In an interview, these persons stated that ECT was significantly less helpful than did persons who did not report

memory problems, and they indicated significantly less often that they would request ECT again if the same condition recurred. Finally, with respect to remote performance assessed about six months after treatment, they did not differ from persons without memory complaints.

Discussion

Whereas the depressed group reported no memory problems at all at follow-up, both ECT groups reported a negative average self-rating that was no better than the self-rating score before ECT (Fig 1). This result could be interpreted as evidence for a persisting effect of ECT on memory self-reports, but this conclusion cannot be a strong one. There may have been pre-existing differences in the characteristics of the patients in the three treatment groups that influenced their memory self-ratings seven months after treatment. For example, patients prescribed ECT might initially have been more depressed than patients not prescribed ECT. This point could be settled conclusively by a study in which patients were randomly assigned to treatment groups.

About half the patients prescribed bilateral ECT subsequently felt that their memory had never returned to normal; and even when all the patients prescribed bilateral ECT were considered as a group, ECT changed the quality or pattern of memory complaints in a lasting way. We believe these persisting reports of memory problems reflect the influence of three different factors.

In the first place, if there was recurrence or persistence of some of the conditions present before ECT, then these conditions could contribute to a general depression of self-rating scores across all test items. That there might be a place for this factor in understanding memory complaints is supported by the association between memory complaints and the feeling that ECT did not help.

Secondly, the pattern of memory complaints reported up to three years after bilateral ECT resembled the pattern of complaints reported at one week after treatment when patients were amnesic, and it differed from the pattern of complaints reported before ECT when patients were depressed. If only those 17 persons who had memory complaints are considered, then the pattern of these complaints at three years after treatment was clearly of the 'amnesic type'. One way of understanding how such complaints could occur long after ECT is to suppose that they are based on the experience of amnesia initially associated with ECT and reflect a persisting, and perhaps altogether natural, tendency to question whether memory functions have fully recovered.

Thirdly, the findings with the time line at three years after treatment (RA = six months, AA = two months) suggest that reports of memory problems may be veridical, in that they can refer to a gap of several months around the time of treatment. A similar interpretation of memory complaints has been suggested by Freeman and Kendell (1980). The estimate of AA obtained here matches rather well estimates of the duration of AA obtained with formal tests (cf. Squire, 1982; Freeman and Kendell, 1980 and references therein). The estimate of six months' RA, however, may have been influenced to some degree by the earlier effects of depression, since nearly the same estimate (five months) was obtained even before treatment. Importantly, the data from the time lines (Fig 3) suggest that memory complaints long after bilateral ECT are usually not complaints about new learning capacity or complaints about memory for the recent past.

We cannot determine whether these three factors are the only ones that deserve consideration or, if so, how they should be weighted. It does seem worth emphasizing, however, that the available data provide no basis for supposing that ECT is associated with

a permanent loss of memory functions, beyond what is represented by the time line data: i.e. an RA of about six months and an AA of two months. At the same time, even this degree of amnesia is substantial and is of concern to many patients. Right unilateral ECT is considered to be as therapeutically effective as bilateral ECT (D'Elia and Raotma, 1975; Strömberg, 1973; but see discussion by Abrams, 1982); yet unilateral ECT is associated with markedly less memory impairment (Squire and Slater, 1978; Reichert *et al.*, 1976; Fromholt *et al.*, 1973). The present study indicates that the effects of right unilateral ECT on memory are also of less concern to persons who receive the treatment. This information should be useful in counselling patients about the risks and benefits of ECT and in reaching informed choices about possible alternative treatments.

Acknowledgments

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ILLUSTRATION

...TREATED WITH
ELECTROCONVULSIVE
THERAPY,

**SHE
WAS**

SHOCKED

TO DISCOVER HOW MUCH OF HER
MEMORY WAS WIPED OUT

WHICH ALLERGY DRUGS SHOULD YOU REALLY "ASK YOUR DOCTOR" ABOUT?
WHAT THEY'LL EAT ON THE WAY TO MARS • COCKING AN EYEBROW AT BILBERRY
A REVOLUTION IN TOOTH CARE • CAN A PILL PROTECT YOU FROM SKIN CANCER? WELL, NO

*Electroconvulsive
therapy helped
to treat her
intractable,
dangerous
depression.
But the author
was surprised to
find out how
much of her
memory was
wiped out.*

BY ANN LEWIS

ILLUSTRATIONS BY
TIM TEEBKEN

I've been asked over and over again whether undergoing electroconvulsive therapy—also known as ECT or shock therapy—was a good decision. And whether I would have ECT again under the same circumstances.

The only honest answer I can give is that I have no idea. To say whether ECT was the right treatment for me, I would have to compare my life before ECT to my life now. And I simply cannot remember life before ECT. In particular, I cannot remember much about the two years leading up to my ECT treatments. That period, along with much of the preceding years, is memory that I lost in exchange for the hoped-for benefits of ECT.

That loss was huge and painful and potentially crippling. And yet, when my therapist describes how I was just before ECT, I believe that ECT was probably the best option at the time. He says that I was spiraling down into a depression that wouldn't lift. He says that I was contemplating suicide. And I believe him. While I don't remember that particular depression, I remember others—many paralyzing episodes of depression in my 37 years of living with mental illness.

My therapist also says that I was failing to respond to medications. And that I also believe. While I cannot remember specific experiences with the plethora of drugs I've tried over the years, I do know that I tried so many because I was constantly searching for one that would finally work.

See **SHOCKED** Page 16

SHOCKED. From Page 14

I had 18 ECT treatments over a six-week period beginning in May 1999. Based on some vague recollections and on what I've been told, here's what happened: Three times a week I rose at dawn to be at the hospital first thing; I sat in a crowded waiting room until my name was called. Then I put on a hospital gown, lay down on a gurney and was wheeled into an operating room designated for ECT patients. Full anesthesia was administered intravenously, and the next thing I knew I'd be waking up in the recovery room, ready to be taken home, where I'd sleep for the rest of the day.

My boyfriend and my mother shared the burden of caring for me. On the days between treatments, she says, we sometimes went to museums, malls and restaurants. She says that I was a zombie, unable to make even the smallest decisions. My boyfriend says I asked the same questions over and over again, unaware that I was repeating myself.

Right after my last treatment—my mother made a note of this in her diary for July 8—I woke up. I can liken this only to what I expect a person coming out of a coma experiences. I felt like a newborn, seeing the world for the first time. But unlike the common notion of first sight as a thing of splendor and awe, for me it was complete frustration.

While I couldn't recall how I had felt before ECT, I couldn't imagine it was any worse than what I was experiencing now.

Every little thing told me that I had no memory. I couldn't remember who had given me the beautiful picture frames or the unique knickknacks that decorated my home. My clothes were unfamiliar, as were the jewelry and trinkets I had owned for years. I didn't know how long I had had my cat or who my neighbors were. I couldn't remember which foods I liked or what movies I had seen. I didn't remember people who greeted me on the street or others who called me on the telephone.

A former news junkie, I was especially frustrated to realize that I didn't even know who the president was or why someone named Monica Lewinsky was famous. I was shocked when I found out about the impeachment hearings.

And I couldn't remember my boyfriend, although he practically lived with me. There was evidence all over the apartment that we loved each other, but I didn't know how or when we had met, what we liked to do together or even where we liked to sit while watching television. I didn't even

remember how he liked to be hugged. Starting from scratch, I had to get to know him again while he had to accept the frustrating loss of what we once had together.

While continuing to battle my mental illness—ECT is no instant cure—I had to relearn how to live my life.

I didn't know my parents had moved. I had to be "reminded" about that great sub shop in Bethesda and about my favorite restaurant, the Lebanese Taverna. I spent 15 minutes in the cracker aisle in Safeway until I recognized the box of my favorite crackers, Stone Wheat Thins. I retrieved some clothes only by going to seven different cleaners to ask if they had an overdue order belonging to Lewis. Just yesterday I lost a contact lens: I've been wearing contacts for at least 10 years, but I have no idea who my eye doctor is, so replacing the lost one will be another tedious challenge.

And I couldn't remember my boyfriend, although he practically lived with me.

Socializing was the hardest part of my recovery, since I had nothing to contribute to a conversation. While I had always been sharp-tongued, quick-witted and sarcastic, I now had no opinions: Opinions are based on experience and I couldn't recall my experiences. I relied on my friends to tell me what I liked, what I didn't like and what I'd done. Listening to them trying to reconnect me to my past was almost like hearing about someone who had passed away.

Before ECT I had been working for a legal concern in the District where the environment was exciting and the people were fun. That's what I've been told, anyway. Just before undergoing my treatment I informed my employer of my disability and requested time off. I estimated that I would need two weeks, unaware that the ECT would eventually stretch on for six weeks and that I would need months to recover.

As the weeks passed, I missed going to work, though I realized I had forgotten the names of major clients I had dealt with daily and even the names of the computer programs I had used routinely. And I couldn't recall the names—or the

faces—of the people I had worked beside—people who had been to my house and with whom I had traveled frequently.

I didn't even know where my office building was located. But I was determined to get my life back on track, so I dug up all my work materials and began studying to catch up with my old life.

Too late: My therapist's request that the firm accommodate my extended absence failed. The company claimed that for business reasons it had been obliged to put someone else into my position and asked where my personal belongings should be sent.

I was devastated. I had no job, no income, no memory and, it seemed, no options. The thought of looking for a job scared me to death. I couldn't remember where I had saved my resume on my computer, much less what it actually said. Worst of all—and this is probably the most familiar feeling among those who suffer from depression—my self-esteem was at an all-time low. I felt completely incompetent and unable to handle the most minor of tasks. My resume—when I finally found it—described a person with enviable experiences and impressive accomplishments. But in my mind I was a nobody with nothing to hold onto and nothing to look forward to.

Perhaps due to these circumstances, perhaps due to my natural biological cycles, I fell back into depression.

Those first months after ECT were horrible. Having lost so much, I was facing another bout of depression—just what the treatments had been intended to correct. It wasn't fair and I didn't know what to do. Restoring my memory—or trying to accept its permanent loss—became the focus of my therapy sessions. I couldn't recall how badly I had felt before the treatment, but I knew now that I was desperate and completely demoralized.

At the edge of hopelessness, I somehow committed myself to hanging in there—not for me, but for the family members and friends who were working hard to make my life better. Daily thoughts of suicide were something I learned to ignore. Instead, I focused on making it through each day. I managed to get out of bed each morning and drive to the coffee shop, where I forced myself to read the entire newspaper, even if I couldn't remember much of what I had read. It was exhausting, but after a few weeks I was reading books and running errands. Soon I re-entered the world of com-

Shock Therapy: Positive and Negative Charges

The extensive memory loss described by Ann Lewis in the accompanying article reinforces some of the widespread negative impressions about electroconvulsive therapy. Even supporters of ECT acknowledge that memory loss is a common side effect, though they say it is typically far less severe than that reported by Lewis.

Juan Saavedra, the Bethesda psychiatrist who treated Lewis before she underwent ECT, says he generally considers this therapy only for a very old person who would have trouble tolerating medication or for a person who is "in danger of suicide [where] you really cannot wait for the antidepressants to be effective." In discussing this as an option, he says, "my approach will be to say that the most important thing is preservation of life."

"There is always a lot of fears, and it's understandable" in light of publicized cases of "people who have been mistreated," says Saavedra, who adds that in his experience the majority of patients who are urged to receive ECT agree to do so.

"There is no way to predict" the degree of memory loss from ECT, Saavedra says. "Every treatment has its possibilities of something going wrong," but ECT is "a very safe procedure these days."

Not nearly safe enough. In the view of those who believe ECT remains more dangerous than it's worth.

"The shock induces an electrical storm that

obliterates the normal electrical patterns in the brain, driving the recording needle on the EEG up and down in violent, jagged swings. This period of extreme bursts of electrical energy often is followed by a briefer period of absolutely no electrical activity. . . . The brain waves become temporarily flat, exactly as in brain death, and it may be that cell death takes place at this time."

That's the view of another Bethesda psychiatrist, Peter Breggin, in his book "Toxic Psychiatry." Breggin's Web site, breggin.com, is only one of many (ect.org, antipsychiatry.org, banishock.org, etc.) that warn about the nasty repercussions of ECT.

Last year's Surgeon General's Report on Mental Health gave ECT's opponents little solace, though it did acknowledge some of the scientific mysteries and past misuses of the therapy since it was developed in the 1930s:

"ECT consists of a series of brief generalized seizures induced by passing an electric current through the brain by means of two electrodes placed on the scalp. . . . The exact mechanisms by which ECT exerts its therapeutic effect are not yet known. . . .

Accumulated clinical experience—later confirmed in controlled clinical trials . . . —determined ECT to be highly effective against severe depression, some acute psychotic states and mania. No controlled study has shown any other treatment to have superior efficacy to

ECT in the treatment of depression."

On the issue of memory loss, the report suggests that most patients are far less affected than Lewis was: "The confusion and disorientation seen upon awakening after ECT typically clear within an hour. More persistent memory problems are variable. Most typical . . . has been a pattern of loss of memories for the time of the ECT series and extending back an average of six months, combined with impairment with learning new information, which continues for perhaps two months following ECT."

The report also reiterated the medical establishment's conclusion that ECT is a worthwhile tool for treating certain mental disorders:

"Although the average 60 to 70 percent response rate seen with ECT is comparable to that obtained with pharmacotherapy, there is evidence that the antidepressant effect of ECT occurs faster than that seen with medication, encouraging the use of ECT where depression is accompanied by potentially uncontrollable suicidal ideas and actions. However, ECT does not exert a long-term protection against suicide. Indeed, it is now recognized that a single course of ECT should be regarded as a short-term treatment for an acute episode of illness."

Or as Saavedra said last week, "ECT doesn't cure anything."

—Tom Graham

puters and e-mail and the Web. Little by little, I was reconnecting to the world.

I also attended therapy religiously. The therapist's office was a safe place where I could admit just how bad I was feeling. Thoughts of suicide were a normal part of my life, but I felt it would be unfair to share those dark feelings with family and friends.

Through the Depression and Related Affective Disorders Association, I joined a support group, which became central to my recovery. There I realized that I was not alone in my plight and for once I had friends to whom I could talk honestly. Nobody was shocked to hear what the voice in my head was telling me.

And I began to run and exercise again. Before ECT I had been training for my first marathon. After, I couldn't run even a mile. But within a few months I was covering long distances, proud of my accomplishment and grateful for an outlet to deal with my stress.

In October I tried a new medication for depression, Celexa. Maybe it was this drug, maybe it was my natural cycle, but I began to feel better. I experienced days where death wasn't on my mind, and then I experienced days where I actually felt good. There was even a turning point when I began to feel hopeful, like something good could actually happen in my life.

The most poignant moment occurred a month after I changed medications. My therapist asked, "If you always felt the way you do today, would you want to live?" And I honestly felt that the answer was yes. It had been a long time since I had felt like living instead of dying.

It's close to a year now since I finished my ECT treatments. I am working full-time. I see my therapist only once every two to three weeks. I still attend DRADA meetings regularly. My memory is still poor. I cannot recall most of the two years before ECT, and memories prior to that

My therapist asked, "If you always felt the way you do today, would you want to live?" And I honestly felt that the answer was yes.

time must be triggered and dug out of my mental archives. Remembering requires a great deal of effort, but my mind is sharp once again.

Friends and family say that I am less gloomy than I was, cheerful and less brash. They say I've softened a bit, though my basic personality has indeed returned. In part I attribute my gentler attitude to the truly humbling experience of having my self disappear. In part I attribute it to the loss of my well-honed vocabulary: I was reluctant to speak up when I couldn't find the right words. But in greatest part I attribute my change to a renewed desire for peace in my life. I am now dedicated to managing my depression and living a satisfying life day by day. I feel that if I can make the best of the moment, then the future will take care of itself.

As for my boyfriend, we're getting to know each other again. I'll be forever grateful for how he cared for the sudden stranger he met after my treatments.

Would I undergo ECT again? I have no idea. Where medication does not work, I believe the doctors' judgment that ECT is still the most effective treatment. For people who are sick enough to be considered for ECT—as I was—I believe the benefits justify the potential loss of memory. Losing my memory, my career, my connections to people and places may seem too much to bear, but I see all that as not a huge price to pay for getting better. What I lost was enormous, but if it is health I have gained, that is obviously far more valuable than what I lost.

While this year has been the hardest of my life, it has also provided me with a foundation for the next phase of my life. And I truly believe that this next phase will be better. Perhaps it will even be great. With a medication that seems to be working, a strong network of support and the ability to move forward, my life looks promising. I've learned to hang in there when it seemed impossible and to rebuild from a significant loss. Both are difficult. Both are painful. But both are possible. I am living

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B

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Brit. J. Psychiat. (1982), 141, 608-613

Comparison of Unilateral and Bilateral ECT: Evidence for Selective Memory Impairment

D. FROMM-AUCH

Summary: Review of studies from the past 16 years employing quantitative measurement of memory functions before and after ECT revealed the following trends: impairment of non-verbal memory functions after less than five unilateral non-dominant ECTs; improvement of non-verbal memory functions after five or more unilateral non-dominant ECTs; no change or improvement of verbal memory functions with unilateral non-dominant ECT; consistent impairment of verbal functions with unilateral dominant ECT, and impairment of both verbal and non-verbal functions with bilateral ECT. The relative lack of impairment in memory functions with unilateral non-dominant ECT is consistent with the theory of asymmetrical hemispheric disorganization in affective disorders, and supports the choice of unilateral non-dominant ECT over bilateral or unilateral dominant ECT in the treatment of depression.

In the early years of electroconvulsive therapy (ECT), loss of memory was believed to be an integral part of the therapeutic effect, and hence the relationship between ECT and memory impairment was thought to be positive. Research has since demonstrated that clinical improvement is not correlated with memory deficits (Fink, 1974; Korin *et al.* 1956), and that unilateral non-dominant ECT, although of equal efficacy to bilateral ECT in ameliorating depression, produces less cognitive and memory impairment (Lancaster *et al.* 1958; Martin *et al.* 1965; Levy, 1968; Squire and Slater, 1978; d'Elia and Raotma, 1975; Zinkin and Birchnell, 1968; Dornbush *et al.* 1971; for review see Harper and Weins, 1975) and a lower percentage of abnormal EEG recordings at four days after treatment (Sutherland *et al.* 1969).

Recent research (Kronfol *et al.* 1978) has further suggested that rather than impairment, selective improvement occurs on tasks more dependent upon non-dominant hemisphere processing, such as visuo-spatial problems. These researchers studied 18 depressed patients neuropsychologically, prior to and after the first and eighth treatment of unilateral ECT. They found that non-dominant hemispheric functions, which were more frequently abnormal in the neuropsychological tests before ECT, improved with either dominant (DOM) or non-dominant (NDOM) ECT when depression was ameliorated. On this basis, it was concluded that in depression, non-dominant hemispheric functions are initially disturbed and ECT, instead of being deleterious to these functions, tends

to improve them. Kronfol and his colleagues did not include a bilateral ECT group and therefore selective improvement in non-verbal functions with bilateral ECT was not examined.

At least three interesting questions arise from this research: Have other studies demonstrated a selective improvement in non-verbal functions with unilateral ECT? Is this trend demonstrated following bilateral ECT? How do verbal memory functions covary with bilateral and unilateral ECT? To consider these questions, the present paper is a review of previously published research which investigated quantitative memory changes in samples of psychiatrically depressed patients, following bilateral and unilateral ECT.

Method

A Medlars II (1967) search covering the last 16 years was performed, using the key words 'electroconvulsive therapy' and 'psychological tests'. Inclusion criteria for studies were: that subjects were psychiatric patients with a diagnosis of depression; that quantitative measurement of memory or learning effects was carried out both before and after ECT; and that there was comparison of bilateral with unilateral ECT, or of unilateral DOM with unilateral NDOM ECT. Some of the earlier studies using the Wechsler Memory Scale (WMS) do not publish the subtest scores separately, and hence the verbal and non-verbal performance respectively could not be determined. In those cases, if the WMS quotient improved after ECT, both the

verbal and non-verbal portions are assumed to have improved.

The relationship between the following factors and test results was examined—the time between last ECT and testing, the number of ECTs and the percentage of females within each study. Attenuation of post-ECT confusion and depression may be indicated by the first two factors respectively, while gender may be important in the ECT response and laterality effect. The number of subjects within each study was also examined, so that equal weight would not be applied for each result.

Results

Twenty-two studies fulfilled the inclusion criteria (Table 1); most of them compared either unilateral DOM and NDOM ECT or bilateral with NDOM ECT. The most striking trend is that non-verbal memory functions, relative to pre-ECT testing, which appear to deteriorate with 1-4 NDOM ECTs, significantly improve after a minimum of five NDOM ECTs (lower right side of Table). Verbal memory remains unchanged or significantly improves with NDOM ECT, regardless of the number of treatments, in all but two studies (Strain *et al.* 1968; Fromholt

TABLE 1
Post-treatment changes in memory functions following bilateral and unilateral ECT

Research articles	n	Females	Latency to test time post-ECT	No. of ECTs	Bilateral ECT		Unilateral dominant ECT		Unilateral non-dominant ECT	
					Verbal	Non-verbal	Verbal	Non-verbal	Verbal	Non-verbal
Berent <i>et al.</i> (1975)	24	100%	5 hrs.	1	—	—	x	n.s.	n.s.	x
Annett <i>et al.</i> (1974)	32	84%	½ hr.	1	—	—	x	n.s.	n.s.	n.s.
Zinkin <i>et al.</i> (1968)	102	74%	0-3 hrs.	1	x	x	—	—	n.s.	n.s.
d'Elia <i>et al.</i> (1976)	20	75%	3 & 6 hrs.	2-3	—	—	x	n.s.	n.s.	x
Squire & Slater (1978)	72	74%	6-10 hrs.	1, 3 & 5	x	x	—	—	n.s.	x
Costello <i>et al.</i> (1970)	30	67%	28-31 hrs.	4	x	—	x	—	0	—
Halliday <i>et al.</i> (1968)	52		4 days	4	0	x	x	n.s.	0	x
Robertson & Inglis (1978)	48	55%	10-14 hrs.	4	x	x	—	—	0	x
Robertson & Inglis (1973)	20	70%	10-14 hrs.	4	x	x	—	—	0	x
Dornbush <i>et al.</i> (1971)	40		24 hrs.	4-5	x	n.s.	—	—	n.s.	n.s.
Strain <i>et al.</i> (1968)	106	71%	36 hrs.	4-12	x	n.s.	—	—	x	n.s.
Fraser & Glass (1980)	29	76%	24 hrs.	5	n.s.	0	—	—	n.s.	n.s.
Cohen <i>et al.</i> (1968)	24	100%	5-8 hrs.	5	x	x	x	n.s.	n.s.	x
Zamora <i>et al.</i> (1965)	28	43%	30-36 hrs.	5	—	—	x	—	0	—
Small <i>et al.</i> (1972)	19	63%		5	—	—	x	n.s.	0	0
Cannicott & Waggoner (1967)	24		2 hrs.	5	x	—	—	—	n.s.	—
Fromholt <i>et al.</i> (1973)	100	61%	24 hrs.	6	x	0	—	—	x	0
Jackson (1978)	34	0%	½ hr.	6	x	x	x	n.s.	n.s.	x
Sutherland <i>et al.</i> (1969)	57		36 hrs.	6	x	0	x	0	0	0
Weeks <i>et al.</i> (1980)	51	67%	1 week	5-8	n.s.	n.s.	—	—	0	0
Kronfol <i>et al.</i> (1978)	18	66%	5 hrs.	8	—	—	n.s.	0	n.s.	0
Martin <i>et al.</i> (1965)	40		24 hrs.	10	x	x	—	—	0	0

x = Significant decrease in post-ECT performance of at least $P < .05$

0 = Significant increase in post-ECT performance of at least $P < .05$

— = Functions not assessed

n.s. = Non-significant change

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et al. 1973). Bilateral and DOM ECT generally results in a decreased or non-significant change for both verbal and non-verbal functions. Improvement of verbal memory after bilateral ECT is reported in one study (Halliday *et al.* 1968), while improvement of non-verbal memory occurs in three studies employing bilateral ECT (Fraser and Glass, 1980; Fromholt *et al.* 1973; Sutherland *et al.* 1969) and in two studies employing DOM ECT (Sutherland *et al.* 1969; Kronfol *et al.* 1978). When they occur, positive changes in verbal functions are found after a minimum of four treatments (bilateral and NDOM ECT only), while positive changes in non-verbal functions are obtained after a minimum of five treatments (bilateral, DOM and NDOM ECT). Verbal memory functions are not found to improve with DOM ECT, in contrast to improvement of non-verbal functions with NDOM ECT, and less consistently, with bilateral and DOM ECT.

Of the 91 group performance measures in Table I (30 for bilateral ECT; 20 for DOM ECT, and 41 for NDOM ECT), ten are exceptions to the following trends: impairment of both verbal and non-verbal functions with bilateral ECT (Halliday *et al.* 1968; Fraser and Glass, 1980; Fromholt *et al.* 1973; Sutherland *et al.* 1969); impairment of non-verbal functions with DOM ECT (Sutherland *et al.* 1969; Kronfol *et al.* 1978); no change or improvement in verbal functions with NDOM ECT (Strain *et al.* 1968; Fromholt *et al.* 1973), and improvement in non-verbal functions after a minimum of five NDOM ECT treatments (Cohen *et al.* 1968; Jackson, 1978).

Two other studies fulfilled the inclusion criteria, but the results are not comparable to the findings reported above, due to the research paradigm used (Cronin *et al.* 1970) or the type of data analysis (Bidder *et al.* 1970). Nevertheless, both of these studies demonstrate verbal memory loss with bilateral ECT and non-significant change in non-verbal memory with unilateral NDOM and bilateral ECT.

The latency to test time after ECT does not appear to influence these findings, since the time varies from .5 hours to 100 hours within the 1-5 treatment category. However, it does appear important for the five-or-more treatment group of studies. One of two studies which does not show improvement of non-verbal functions after six unilateral NDOM ECT (Jackson, 1978) tested the patients half an hour after the sixth ECT treatment. This is also the only study which has an all-male sample. The lowest percentage of females in the studies for which this information is available is 43 per cent, with a mean of 73 per cent.

Discussion

Despite differences in methodology, treatment techniques and behavioural tests, several trends

emerge from the results reviewed. These are: impairment of non-verbal functions with less than five NDOM ECTs; improvement of non-verbal functions after a minimum of five NDOM ECTs; unchanged or improved verbal memory functions with NDOM ECT; impairment of verbal functions with DOM ECT; and impairment of verbal and non-verbal functions with bilateral ECT.

Improvement of non-verbal functions with NDOM ECT after five or more treatments suggests that attenuation of cognitive deficits parallels amelioration of depression. With fewer treatments, and presumably less attenuation of depression, the disruptive effects of the ECT predominate, and selective impairment rather than improvement is shown. This evidence is consistent with the theory of asymmetrical dysfunction in affective disorders. On the basis of a synthesis of findings from many diverse areas within neuroscientific research, Flor-Henry (1973; 1976; 1978a, b; 1979) concludes that the neural substrate of emotion is predominately non-dominant and that the depressive phase of the manic-depressive syndrome is manifested when cerebral disorganization is more pronounced for the non-dominant hemisphere.

The findings of others, however, are not consistent with this. A dysfunctional left hemisphere has been implicated in at least some forms of depression (e.g. Hommes and Panhuysen, 1971). The asymmetry of emotional response found in humans, i.e. strong emotion with right hemisphere activation and inhibition of the left hemisphere (Gainotti, 1972; Perria *et al.* 1961; Rossi and Rosadini, 1967; Ross and Mesulam, 1979; Dimond *et al.* 1976) also fits within this theoretical framework. In depression, an imbalance of hemispheric activation, presumably due to perturbation of the right hemisphere, normalizes following ECT, which in turn results in decreased depressive symptomatology and concurrent selective improvement of visuospatial functions.

Exceptions to the above five major trends are reported in eight studies. Halliday *et al.* (1968) found an improvement in verbal learning with four bilateral ECTs, while Fraser and Glass (1980), Fromholt *et al.* (1973) and Sutherland *et al.* (1969) found an improvement in non-verbal functions after five, six, and six bilateral ECTs respectively. It is of interest that the five studies with the largest number of subjects show either a non-significant change or else improvement of non-verbal functions, after a minimum of five bilateral ECTs. Similarly, Sutherland *et al.* (1969) and Kronfol *et al.* (1978) found selective improvement of non-verbal functions after six and eight DOM ECTs respectively. This improvement in non-verbal memory function with DOM and bilateral ECT, although less consistent, is similar to the trend noted after a minimum

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of five NDOM ECTs, and therefore may also be reflective of differential improvement of memory function with attenuation of depression.

Strain *et al* (1968) and Fromholt *et al* (1973), investigating 106 and 100 subjects respectively, were the only researchers to find a decrease in verbal memory with NDOM ECT. Strain *et al* (1979) found that paired Associate Learning test scores were significantly reduced ($P < .001$) from pre-ECT levels, for both the bilateral and unilateral ECT groups, although the bilateral group showed more impairment ($P < .05$). Similarly, Fromholt *et al* (1973), in an analysis of intra-group changes for the NDOM and bilateral ECT groups, showed reduced scores on Associate Learning ($P < .01$) from the WMS. It is difficult, however, to reconcile these data with those of other researchers: the divergent findings are based on the same or similar verbal learning tasks, i.e. paired associate word learning, similar latency to test time, number of ECTs, placement of electrodes, and percentage of females. One possible explanation is based on handedness, since the handedness of the sample is not indicated by Fromholt *et al*. A sufficient number of patients with primarily right hemisphere language functions could have influenced the group results: i.e. unilateral ECT to the hemisphere primarily responsible for speech would result in reduced verbal memory scores, as illustrated by the results following DOM ECT.

Cohen *et al* (1968) and Jackson (1978) are the only authors who found a decrease in non-verbal memory with five and six NDOM ECTs respectively. The first, although finding a decrement in the retention of both forms and words across DOM, NDOM, and bilateral ECT groups, conclude that the ECT-produced decrements were not as large for the Forms as for the Words. In addition, the decrement shown on Words by the DOM ECT groups exceeded that shown on Forms by the NDOM ECT groups. This is consistent with the trend appearing in the other studies reviewed in this paper. However, the reason for the decrement as opposed to improvement is not clear. Latency to test time and the gender ratio of the sample may have influenced the results of Jackson (1978), who tested his all-male patient sample half an hour after the treatment. Contamination of the results with post-ECT confusion may account for the negative findings. The evidence from numerous demographic studies indicates that the rate of depression is much higher for females than males, in the order of 2 or 3:1 (Baron, 1981; Polonio, 1966; Rosenthal, 1970; McCabe, 1975), making the sample in Jackson's study atypical of depressed patients.

Despite these exceptions, the studies reviewed suggest that unilateral NDOM ECT produces less

impairment of verbal memory, compared to unilateral DOM or bilateral ECT, a conclusion drawn by other researchers. An additional trend suggests selective improvement of non-verbal functions with NDOM ECT after a minimum of five treatments, a finding which is less consistent for DOM ECT and bilateral ECT. The relative lack of impairment in memory functions with NDOM ECT, coupled with the results from the last 20 years indicating the equal efficacy of NDOM and bilateral ECT treatment (for review see d'Elia and Raotma, 1975), cogently argues for the choice of unilateral NDOM ECT over bilateral ECT, in direct contrast to much of the present practice in Great Britain (Pippard and Ellam, 1981).

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He says: "The data is difficult to interpret."

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ECT and cerebral atrophy

A COMPUTED TOMOGRAPHIC STUDY

S. P. CALLOWAY, R. J. DOLAN, R. J. JACOBY AND R. LEVY

The case-notes of 41 elderly depressives who underwent computed tomography were examined and the ECT history of each patient was assessed. No association was found between ECT and global cortical atrophy or ventricular size, but a significant relationship was demonstrated between frontal lobe atrophy and ECT.

Key words: Depression - electro-convulsive therapy - computed tomography - cortical atrophy - frontal lobes.

The possibility that electro-convulsive therapy (ECT) has lasting effects on the brain has frequently been raised. Research has focussed on the long-term psychological effects of ECT especially with regard to memory (*Halliday et al.* (1968), *Squire & Chace* (1975), *Weeks et al.* (1980)). There is little information about morphological changes in the brain following ECT in man, although animal studies have showed structural changes in neurones and glial cells particularly in the frontal area following electrically induced convulsions (*Hartelius* (1952), *Ferraro et al.* (1946)). Computed tomography (CT) offers a non-invasive way of examining structural changes *in vivo*. In order to investigate any association between ECT and cerebral atrophy we re-examined the data of *Jacoby & Levy* (1980) who looked at the relationship of CT appearance to clinical state in 41 elderly patients with a primary diagnosis of affective disorder.

METHOD

The patients were 41 consecutive admissions to the psychogeriatric ward of the Bethlem Royal with a primary diagnosis of affective disorder. All 41 case-notes were traced and re-examined. Two patients with a history of excessive alcohol intake and one with a history of syphilis were excluded from the analysis. The case-notes of the remaining 38 patients were examined in order to determine the presence or absence of ECT in their treatment history and the number of applications. No patient had received ECT in the 6 months prior to computed tomography, and none had had a leucotomy or insulin coma therapy. It was impossible to calculate the exact number of ECTs received in every case as several patients had been treated at various other hospitals, often many years earlier. An estimate of the number of treatments was therefore calculated on the arbitrary basis of eight applications per course of treatment in cases where there was definite evidence that a course had been administered but the exact number of applications was not known.

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Table 1. Presence or absence of frontal, parietal and insular atrophy in patients with and without a history of ECT

	Atrophy					
	Frontal		Parietal		Insular	
	Absent	Present	Absent	Present	Absent	Present
No ECT	11	4	7	8	8	7
ECT	7	15	4	18	11	11
$\chi^2 = 6.15, 1 \text{ df}, P < 0.02$ $\chi^2 = 3.46, 1 \text{ df}, \text{n.s.}$ $\chi^2 = 0.04, 1 \text{ df}, \text{n.s.}$						

For analysis ECT was considered by presence or absence of ECT in the patient's history and estimated number of applications. Patients were assigned to one of six groups according to the estimated number of applications as follows: 1-6, 7-12, 13-24, 25-36, over 36.

The technique of scan analysis and assessment of cortical atrophy has been described in detail by *Jacoby et al.* (1980). Cortical atrophy was rated blindly by a neuroradiologist on a four-point scale for each of the five cortical areas - frontal, temporal, insular, parietal and occipital.

The relationship of CT changes to ECT was assessed by means of chi-squared test and the Mann-Whitney U test for non-parametric data.

RESULTS

Twenty-two patients (mean age 71.5) had received ECT and 15 patients (mean age 73.8) had not. Information was insufficient in one case who was excluded from further analysis. Twenty-nine out of 37 patients were rated as having some degree of cortical atrophy.

No relationship was shown between ventricular measures and ECT. However, there was an association between measures of cortical atrophy and ECT (Table 1).

Table 1 shows the relationship between history of ECT and atrophy in the frontal, parietal and insular regions of the brain. Temporal or occipital atrophy was present in only four patients and no statistical evaluation could be made. A chi-square test indicates a significant association between history of ECT and presence of frontal atrophy ($P < 0.02$) but not with insular atrophy. The association between ECT and parietal atrophy just failed to reach significance at the 0.05 level.

These differences were not due to age as there was no significant difference between the ECT-treated groups (mean age 71.5) and those not receiving ECT (mean age 73.8).

The majority of patients had received bilateral ECT. The number in whom it could be stated with certainty that they had received only unilateral ECT was too small for a valid comparison to be made between the two groups.

Table 2 shows the relationship of ECT to frontal atrophy in more detail.

Table 2. Estimated number of ECT applications in patients with and without cortical atrophy in the frontal area

	No. of ECT applications					
	0	1-6	7-12	13-24	25-36	36 +
No atrophy	11	2	1	2	0	2
Atrophy	4	2	5	3	2	3

Mann-Whitney U test = 100.5, two-tailed, $P < 0.05$.

The estimated number of ECT applications given to patients with and without cortical atrophy is shown.

The Mann-Whitney U test for non-parametric data showed that patients with frontal lobe atrophy had received more applications of ECT ($P < 0.05$).

DISCUSSION

The results suggest an association between history of treatment with ECT and cortical atrophy in the frontal region. One possible explanation for these findings is that ECT causes cortical atrophy. An alternative is that there may be a sub-group of patients with depressive symptoms who are more prone to develop frontal atrophy and who are also more likely to be given ECT for clinical reasons. Additionally, these patients might be relatively unresponsive to treatment, perhaps because of the organic changes observed here, and as a consequence might receive more ECT than the other group.

The relationship between ECT and cerebral atrophy has also been considered by Weinberger *et al.* (1979) who performed CT scans on 75 chronic schizophrenics. Measuring the width of fissures and sulci they found significantly greater cortical atrophy in 17 ECT-treated patients compared with 58 patients who had not received ECT ($P < 0.01$). The only attempt at a prospective study of the putative effect of ECT on brain structure observable on CT scans was undertaken by Menken *et al.* (1979). In a single case study of a 30-year-old woman who had 10 ECT applications over 45 minutes a CT scan performed 3 hours after the last application showed no 'haemorrhages or oedema', a study which, in our opinion, does not help to resolve the issue of the possible role of ECT in causing structural damage to the brain.

CONCLUSION

The *ad hoc* nature of this study and the difficulty in obtaining an accurate assessment of the number of applications of ECT do not permit us to claim an unequivocal association between ECT and structural change in the brain. Nevertheless, this is a question of such importance that, in our opinion, the finding of a relationship between frontal atrophy and ECT justifies this brief report. It emphasizes the need for a more detailed investigation, with larger numbers of patients including a younger age group.

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Editorial

Memory and ECT: From Polarization to Reconciliation

Discussions of the cognitive effects of electroconvulsive therapy (ECT) have been polarized for decades. Critics of the treatment often claim that patients only seem improved after ECT because they are "punch drunk"—too confused to maintain a depressed state (Sterling, 2000). Others contend that profound and permanent amnesia is common and a clear sign that the treatment causes brain damage (Frank, 1990). Still others have charged that the adverse effects are more pervasive than retrograde amnesia, with ECT impairing the most complex of human cognitive functions, i.e., intelligence, creativity, judgment, foresight, etc. (Breeding, 2000).

In contrast, practitioners and researchers often state that the adverse cognitive effects of ECT are transient. Within a few weeks of the acute treatment course, cognitive function is restored. If any residual deficit is acknowledged, it is restricted to gaps in memory for events that occurred close in time to the treatment. Some state that the memory loss is limited to the period of the treatment, while others extend this to a period of a few weeks or months surrounding the ECT course. Complaints of pervasive and persistent memory loss have often been attributed to causes other than ECT, typically persistent psychiatric disability.

Both views are out of keeping with clinical experience and research. Scores of studies have failed to find an association between clinical outcome and the depth of any cognitive deficit during or following ECT (Sackeim, 1992). People do not get better because they are confused or amnesic. To the contrary, many cognitive domains, including "intelligence," improve shortly following ECT (Sackeim et al., 1992). On the other hand, virtually all patients experience some degree of persistent and, likely, permanent retrograde amnesia. A series of recent studies demonstrates that retrograde amnesia is persistent, and that this long-term memory loss is substantially greater with bilateral than right unilateral ECT (Weiner et al., 1986b; McElhiney et al., 1995; Lisanby et al. [in press]; Sackeim et al. [in press]). It has also become clear that for rare patients the retrograde amnesia due to ECT can be profound, with the memory loss extending back years prior to receipt of the treatment.

As a field, we have more readily acknowledged the possibility of death due to ECT than the possibility of profound memory loss, despite the fact that adverse effects on cognition are by far ECT's most common side effects. Individual differences and hypersensitivity to side effects characterize virtually all medical procedures and pharmacological treatments. That ECT would have an especially narrow range of amnesic effects would be a remarkable exception.

Undoubtedly, reaching consensus on this fundamental issue has been impeded by the fact that memory complaints are subjective and can have multiple determinants. Some of the neuropsychological deterioration seen after ECT is due to natural progression of an

underlying illness. In young patients, seemingly irreversible cognitive decline may accompany the first manifestation of a psychotic disorder (Wyatt, 1991). When ECT is used early in the treatment of such patients, the precipitous cognitive decline is at times wrongly attributed to this therapeutic intervention. Similarly, ECT may unmask an underlying dementia in older patients.

It is also the case that in all populations studied (normal, neurological, psychiatric), current mood state is the most important correlate of subjective evaluation of memory function (Coleman et al., 1996). We believe that our memory (and other cognitive functions) are less intact when we are depressed. On the other hand, regardless of the population studied, subjective evaluations and objective measures typically show poor association (Sackeim and Stern, 1997).

Another complication is that some patients with persistent memory complaints following ECT have no treatment-related deficits. Rather, the subjective experience of cognitive deficit is related to ongoing psychopathology. While there is compelling evidence that this occurs with some frequency (Freeman et al., 1980), for understandable reasons the profession has not emphasized this phenomenon. In the consent form recommended by the 1990 APA Task Force Report on ECT (American Psychiatric Association, 1990), it was acknowledged that a minority of patients report severe memory problems, with the comment that, "The reasons for these rare reports of long-lasting impairment are not fully understood" [p. 158]. Some of the reasons were understood, but it is uncomfortable for the field to be perceived as "blaming the victim," and attributing memory complaints to unresolved psychiatric disturbance, even if true.

However, aside from each of these possibilities, some patients experience profound memory loss due to ECT. Most ECT practitioners have encountered fully credible patients who are distressed by the magnitude of their persistent post-ECT amnesia. Skeptics will argue that complaints of memory loss do not necessitate true disability, and that we have no objective "dipstick" to verify that memory is truly impaired. On the other hand, there is no dearth of patients who have received ECT who believe that the treatment was valuable, often life saving, who are not litigious, who return to productive activities, and yet report that a large segment of their life is lost. These patients often report a classic temporal gradient in their retrograde amnesia, with the memory loss most accentuated for the time period (months to years) closest in time to the treatment, with sparing of more remote memories. It is hard to imagine that such reports of a classic retrograde amnesic syndrome, with sparing of other cognitive functions, are simply fabricated. Attributing these subjective deficits to ongoing psychopathology or natural disease progression would seem disingenuous and defensive.

There have been few personal accounts of the amnesia following ECT (Wolfe, 1969). In this issue of *The Journal of ECT*, Anne B. Donahue provides a compelling description of the nature and impact of the persistent memory loss she experiences. In many ways this is a courageous statement, acknowledging the clinical benefit of the treatment, and alerting the field about the mismatch between our efforts to assess objectively cognitive alterations and the phenomenology of the memory loss. Donahue's paper also underscores the public relations fallout and, more critically, the turmoil to individuals that result when former patients experience chronic and pervasive memory loss and yet the field denies the possibility of its occurrence.

Fortunately, the tide has turned. The field has greater awareness of the common am-

nostic effects of the treatment, and reconciliation is occurring with the experience of exceptional patients with substantial and sustained memory loss. The newly revised APA Task Force Report (APA, in press) on ECT states:

In many patients the recovery from retrograde amnesia will be incomplete, and there is evidence that ECT can result in persistent or permanent memory loss. Owing to a combination of anterograde and retrograde effects, many patients may manifest persistent loss of memory for some events that transpired in the interval starting several months before and extending to several weeks following the ECT course. There are individual differences, however, and, uncommonly, some patients may experience persistent amnesia extending several years prior to ECT. Profound and persistent retrograde amnesia may be more likely in patients with preexisting neurological impairment and patients who receive large numbers of treatments, using methods that accentuate acute cognitive side effects (e.g., sine wave stimulation, bilateral electrode placement, high electrical stimulus intensity).

This change in attitude and understanding compel closer clinical and research attention to the cognitive effects of treatment. The papers in this special issue highlight some of the key unanswered questions.

TREATMENT TECHNIQUE AND AMNESIA

It has become increasingly clear that the sophistication with which ECT is conducted impacts not only on short-term cognitive effects, but also on the likelihood of long-term persistent changes. Lerer and colleagues review the effects of treatment schedule (using bilateral ECT) on adverse cognitive effects. This work (Lerer et al., 1995; Shapira et al., 1998) has demonstrated a principle regularly used by clinicians. Increasing the interval between treatments reduces the magnitude of cognitive impairment. In terms of long-term consequences, the choice of electrode placement (right unilateral versus bilateral ECT) may be more consequential than the electrical dosage administered and perhaps the treatment schedule (Weiner et al., 1986b; Sackeim et al., 1993; McElhiney et al., 1995; Lisanby et al., in press; Sackeim et al., in press). It appears that high dosage right unilateral ECT is as effective as robust forms of bilateral ECT, but has significantly less probability of resulting in marked and persistent retrograde amnesia (Abrams et al., 1991; Sackeim et al., in press; McCall et al., in press). Further refinements of ECT technique may additionally limit cognitive side effects. Perhaps the most attractive possibility is shortening the width of the brief-pulse stimulus. The pulse widths most commonly used are an order of magnitude longer than that needed for neuronal depolarization, and thus necessarily involve stimulation after neurons have fired (Sackeim et al., 1994).

Some practitioners have held the view that the focus of ECT research in the last two decades on optimizing stimulus dosing and waveform, electrode placement, and spacing of treatments was largely academic. High intensity treatment (e.g., high fixed dosage bilateral ECT) is the least complicated to administer and has the highest probability of efficacy. Given the view that all adverse cognitive effects are transient, with rapid resolution, for some there was little incentive to adopt new treatment methods. As recent research has consistently demonstrated that treatment technique impacts on the magnitude of persistent memory loss, this position becomes difficult to defend.

INDIVIDUAL DIFFERENCES AND ADVERSE COGNITIVE EFFECTS

It would be comforting to attribute all the negative cognitive outcomes with ECT to poor technique. However, regardless of how ECT is performed there are individual differences. Using the same technique, clinicians regularly encounter patients who respond to ECT without any cognitive alterations (or, indeed, may show resolution of preexisting cognitive deficits during and following the ECT course) as opposed to patients who develop delirium. Why?

Over the 65 years of use of convulsive therapy, there have been scores of studies examining the patient characteristics (phenomenology, clinical history, treatment history, biology) that predict therapeutic outcome (Scott, 1989; Nobler and Sackeim, 1996). Essentially, there has been one systematic report on the patient characteristics that predict short- and long-term cognitive outcome after ECT (Sobin et al., 1995). That study suggested that patients with pre-ECT global cognitive impairment and those with prolonged disorientation in the postictal state have more profound short- and long-term retrograde amnesia. This would suggest that treatment techniques be "softened" especially for patients with these characteristics. However, practitioners routinely face issues of this type that are unexplored. Does preexisting neurological illness (stroke, Parkinson's disease, dementia, etc.) predispose to long-term cognitive deficits? What is the contribution, if any, of comorbid substance abuse, concurrent antidepressant or antipsychotic pharmacotherapy, cardiac illness (low cardiac output), benzodiazepine use, etc., to post-ECT cognitive deficits? We have no answers to these questions.

PREVENTION AND TREATMENT OF COGNITIVE DEFICITS

The side effects of many pharmacological treatments are actively treated (e.g., anticholinergics for neuroleptic-induced extrapyramidal symptoms). Electroconvulsive shock (ECS) is the most common procedure used to induce amnesia in animals to screen pharmacological compounds for protective effects on memory. Our estimate is that between 50–100 compounds have shown benefit in ECS models (Krueger et al., 1992). For example, in this issue Andrade and colleagues review research on herbal preparations that ameliorate the cognitive effects of ECS in animal models (Joseph et al., 1994; Faruqi et al., 1995; Andrade et al., 1995; Vinekar et al., 1998), and discuss the strengths and weaknesses of animal models in generalizing to human ECT.

The interest of the pharmaceutical industry in using ECS as a screening method for identifying compounds with promemory effects is not to develop adjunctive medications for ECT. The ECT market is too small, and the predominant aim has been to develop medications for the treatment of dementing disorders (Krueger et al., 1992). Consequently, only a handful of studies have tested pharmacological adjuncts for protective effects in ECT (Stern et al., 1991; Prudic et al., 1999).

Concerted research in this area has the potential for making an important clinical contribution, as well as advancing our understanding of the neurobiology of ECT's amnesic effects. One example illustrates these possibilities. There is considerable interest in the notion that ECT results in altered glutamatergic transmission, particularly in prefrontal and medial temporal lobe structures (Morinobu et al., 1997; Pilc et al., 1998; Hiroi et al., 1998), and that this increased excitatory transmission contributes to amnesic effects

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(Chamberlin and Tsai, 1998). Long-term potentiation (LTP) has been commonly viewed as a model of memory formation, and ECS results in long-term disruption of LTP in the dentate gyrus (Stewart et al., 1994; Stewart and Davies, 1996). The NMDA antagonist, ketamine, protects against this disruptive effect (Stewart and Reid, 1994), raising the possibility that use of ketamine as an anesthetic, as opposed to the standard short-acting barbiturates, or use of other glutamatergic antagonists may have a protective effect on cognition (Reid and Stewart, 1997).

THE NATURE AND NEUROBIOLOGY OF ADVERSE COGNITIVE EFFECTS

There are additional goals for future research on the cognitive consequences of ECT. We need to 1) better characterize the nature of memory deficits (i.e., what is forgotten), 2) better characterize the neural systems implicated in these amnesic effects, particularly the role of prefrontal versus medial temporal lobe memory systems, and 3) determine the impact of ECT on neurocognitive functions other than memory (Calev et al., 1995).

It has been commonly thought that the memory deficits following ECT reflect medial temporal lobe dysfunction (Squire, 1981; 1986a; 1986b; Sackeim, 1992). The most prominent deficits are anterograde amnesia (rapid forgetting of newly learned information) and a temporally graded retrograde amnesia. ECT patients do not show deficits in priming, skill acquisition, or other types of procedural (nondeclarative) memory (Cohen and Squire, 1980; Squire et al., 1984; Graf et al., 1984; Squire et al., 1985). The rapid forgetting rate (Squire, 1981), preserved metamemory ("feeling of knowing") (Shimamura and Squire, 1986), and other features (Squire, 1982) distinguish the amnesia following ECT from that due to diencephalic lesions or Korsakoff's syndrome. This pattern, largely restricted to episodic, declarative memory, suggests that the underlying disturbance is one of consolidation and/or retrieval (Squire and Alvarez, 1995). The reversibility of amnesia, with the recovery of memories over time, particularly implicates an impaired retrieval process. The established role of medial temporal lobe structures in memory processes (Shimamura and Squire, 1987; Nadel and Moscovitch, 1997), the low threshold for afterdischarge and seizure elicitation in the hippocampus (Ajmone Marsan, 1972; Bragin et al., 1997), and the disruption by ECS of hippocampal processes implicated in memory (e.g., LTP) (Reid and Stewart, 1997) support the view that medial temporal lobe dysfunction is key.

However, there is hardly any physiological evidence linking medial temporal lobe dysfunction to the memory deficits following ECT. In this issue, we report that the development of EEG (electroencephalographic) theta activity in left frontal and temporal sites is associated with greater retrograde amnesia for autobiographical information, partially supporting the medial temporal lobe hypothesis. In contrast, there is consistent evidence that ECT exerts its most profound physiological effects in prefrontal cortex, as assessed by reductions in cerebral blood flow (Rosenberg et al., 1988; Silfverskiöld and Risberg, 1989; Nobler et al., 1994) and metabolic rate (Volkow et al., 1988; Guze et al., 1991), and the induction of EEG slow-wave activity (Fink and Kahn, 1956; Weiner et al., 1986a; Sackeim et al., 1996). Thus, there is the paradox that the most prominent cognitive effects are linked to a different brain region than the most pronounced physiological effects. There is a compelling need to examine associations between the magnitude of

cognitive effects and regional alterations in functional brain activity (e.g., metabolic rate) and biochemical parameters.

It is noteworthy that the classic deficits associated with hippocampal damage are a profound anterograde amnesia and a less marked retrograde amnesia (Russell and Nathan, 1946; Milner, 1970; Damasio et al., 1985). In contrast, ECT results in a rapidly resolving anterograde amnesia and persistent retrograde amnesia (Squire, 1986a; Weiner et al., 1986b; Sackeim et al., in press). In addition, the retrograde amnesia following hippocampal damage is believed to be greater for autobiographical than public (impersonal) events (Nadel and Moscovitch, 1997). We have recently shown that the opposite is the case following ECT (Lisanby et al., in press). Both in the short and long term, patients who received ECT had denser amnesia for events in the world (public knowledge) than for events in their own lives. Frontal lobe damage can result in profound retrograde amnesia (Stuss and Benson, 1986; Kopelman, 1992; Moscovitch, 1994; Shimamura, 1994), in some comparisons as great as temporal lobe pathology (Kopelman et al., 1999), and presumably due to the disruption of retrieval processes. In amnesic patients (with brain damage), anterograde and retrograde memory loss are often weakly associated, and there is evidence that tests of frontal lobe function can covary with the magnitude of retrograde amnesia (Kopelman, 1991). Thus, a reasonable argument can be made that our traditional view that the (retrograde) amnesic effects of ECT result from functional disruption of medial temporal lobe structures is wrong, and the retrograde amnesia may, in fact, have an important frontal lobe involvement.

Resolving this issue, while of obvious importance to our understanding of the neurobiology of retrograde amnesia, is also of clinical significance. The development of alternative electrode placements, such as the bifrontal (Lawson et al., 1990; Letemendia et al., 1993; Bailine et al., 2000) and the asymmetric (Swartz, 1994) techniques, are predicated on the notion that avoidance of temporal lobe stimulation minimizes adverse cognitive effects, while frontal lobe stimulation preserves efficacy. If prefrontal changes subserve the retrograde amnesia these efforts may be largely in vain.

The prefrontal cortex is linked to a variety of "executive functions," including working memory (holding information online), logical reasoning and abstraction, set shifting, temporal organization of behavior, planning, memory for the context of events, and inhibition of competing, prepotent responses (Baddeley, 1986; Stuss and Benson, 1986; Goldman-Rakic, 1987; Diamond, 1990; Fuster, 1990). Tasks assessing prefrontal functions may load on different dimensions than tasks presumed sensitive to medial temporal lobe function (episodic, declarative memory), and there is some evidence that performance on prefrontal tasks predicts the adequacy of memory for the source or context of information (Glisky et al., 1995) and retrograde amnesia (Kopelman, 1991). Executive functions are fundamental to organizing one's life and controlling behavior, yet there has been little investigation of the impact of ECT on this domain (Jones et al., 1988).

SUBJECTIVE EXPERIENCE OF COGNITIVE EFFECTS

In this issue, Prudic and colleagues summarize what is known about patients' own assessments of the effects of ECT on cognition. It appears that over time there has been a detectable shift. In older studies, largely using sine wave stimulation, a long-term detrimental impact was observed, especially with bilateral ECT (Squire et al., 1979;

Squire and Slater, 1983). Modern studies report that within a few days of ECT the vast majority of patients evaluate their memory as improved (Sackeim et al., 1993; Sackeim et al. (in press). This shift may be attributable to the advances in ECT technique (use of titration, brief pulse stimulation, etc.).

However, we should not be sanguine. ECT research has mainly relied on a single instrument to obtain self assessments of memory function, the Squire Subjective Memory Questionnaire (SSMQ) (Squire et al., 1979). The SSMQ is limited in the dimensions of metamemory that it examines, and is extraordinarily complex in its instructions. Patients are asked to rate their current functioning for discrete cognitive activities relative to their functioning before the onset of the index episode of depression. Perhaps not surprisingly, it has been shown that a substantial number of responses to the SSMQ are of doubtful validity. It is not infrequent for patients to state that their current cognitive function a few days after ECT is superior to that before the onset of the depressive episode, an unlikely phenomenon (Coleman et al., 1996). Broader-based assessment techniques are needed. It is especially surprising that direct and simple inquiries about whether ECT has had a positive or detrimental effect on memory have not been used in recent research. An older literature illustrated that such direct inquiries were effective in distinguishing ECT waveforms (Medlicott, 1948) and electrode placements (Cannicott, 1962; Fleminger et al., 1970).

Prospective patients, family members, and the public often want to know the frequency with which patients report substantial memory impairment following ECT. While we believe that such reports are infrequent, there is little objective evidence to support this judgment or to even broadly estimate base rates. Indeed, our estimates of the probability of death with ECT are based on a more secure empirical foundation (Abrams, 1997) than our estimates of marked subjective memory loss. This should be a readily resolvable issue, and calls for a large sample study in community settings.

In short, as the quality and sensitivity of neurocognitive research in ECT have improved, increasing evidence has accumulated that some degree of persistent memory loss is common. As the dialectical political battles of the 1960s and 1970s recede, there is greater acceptance and acknowledgment by the profession that ECT may infrequently result in extensive retrograde amnesia. At the clinical level, this shift in perspective highlights the need for practitioners to update what is communicated in the consent process and to monitor cognitive outcomes. This shift also presents many challenges for research, the most important of which is to further reduce or eliminate these adverse effects of ECT.

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Electroconvulsive Therapy and Memory Loss: A Personal Journey

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Summary: The cause for the significant gap between research and anecdotal evidence regarding the extent of some memory loss after electroconvulsive therapy (ECT) has never been adequately explained. A patient's development of awareness and self-education about her severe side effects from ECT raises questions regarding many current assumptions about memory loss. ECT-specific studies, which conclude that side effects are short term and narrow in scope, have serious limitations, including the fact that they do not take into account broader scientific knowledge about memory function. Because of the potential for devastating and permanent memory loss with ECT, informed consent needs significant enhancement until advancing research on both improved techniques and on better predictive knowledge regarding memory loss progresses to making a greater impact on clinical applications. Follow-up care and education in coping skills need to be a regular part of ECT practice when patients do experience severe effects.

Key Words: Electroconvulsive therapy—Memory loss.

INTRODUCTION

Occasionally, I feel bitter. More often, it is a sadness, a sense of a deep loss that may not even have had to happen. It is a grief that keeps deepening over time, because there is hardly a week that goes by that I do not discover yet another part of my life that is lost somewhere in my memory cells.

Despite that, I remain unflagging in my belief that the electroconvulsive therapy I received in the fall of 1995 and then the spring of 1996—33 treatments, initially unilateral and then bilateral—may have saved not just my mental health, but my life. If I had the same decision to make over again, I would choose ECT over a life condemned to psychic agony, and possible suicide. Like a heart patient who has to choose the risks of surgery over the risks of heart attack or stroke; like the cancer victim who must choose the horrible side effects of chemotherapy over certain death to the disease—I live with and accept the price I paid to break the stranglehold of a seemingly intractable and severe depression.

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Perhaps it is all as simple as that: a medical cost-benefit analysis between treatment with its side effects, or remaining with alternate, less effective treatments. As with any other illness and its treatments, new research and developments that reduce risks and increase effectiveness do not always trickle down to the front lines of practice as quickly as they should.

ECT, however, is different in several critical respects. It has a history of extended public controversy that may well exceed that of any other longstanding medical treatment, a controversy that would appear unjustified by the treatment's clear efficacy, or even its early abuses. There is an aura of dishonesty about the side effects: discrepancies between official positions and numerous personal testimonies of more severe problems that are discounted or left unexplained.

My long-term memory deficits far exceed anything my doctors anticipated, I was advised about, or that are validated by research. To the contrary, either I am one in a thousand, a complete anomaly, to be able to document memory loss still remaining after 3 years and extending as far back as incidences eight to nine years ago, or the profession in general, after all these years of treatment with ECT, has still failed to identify and come to grips with the true potential risks.

While the more distant incidents may be random events, they are hardly insignificant ones: hosting and driving Mother Teresa for a full day visit to Los Angeles in 1989; the dinner reception for my National Jefferson Award in Washington, D.C., in 1990, where I met and sat beside my co-honoree, General Colin Powell; my brother's wedding in 1991—the list goes on, and keeps growing as people bring up references to the past in casual conversations.

Human memory seems to me to be one of the most precious aspects of our personality, since our memories are so critical to who we are and how we see ourselves and others. The memories of our past give us an understanding of where we fit in the world. I have experienced more than a "cognitive deficit." I have lost a part of myself.

THE JOURNEY OF AN EXPERIENCE WITH SEVERE MEMORY LOSS

The greatest anxiety about ECT has been described as the fear of memory loss (Bernstein et al., 1998). As Fink (1997) has noted, "The effects of ECT on memory and cognition contribute to public fears. ECT does affect the mind—that unique and delicate essence of our individuality that distinguishes one human being from another..."

It has taken several years for me to gain the kind of perspective on coping with my memory loss to allow a reflection such as this. The lack of any referral, follow-up care, or general information forced me both into my own efforts at research and my own struggle through the process of developing coping skills. As I look back now, almost 3 years since the start of my first series of treatments in September of 1995, I recognize that while gaining information ultimately helped in understanding, it also contributed to feelings of anger and betrayal that complicated phases of the recovery process.

I had been easily treated on medication for two prior episodes of depression in 1987 and 1989; my 1993 relapse had failed to respond to medication trials. ECT was presented as an uncomplicated and low-risk alternative, with only short-term memory effects to be concerned about.

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I was not particularly anxious about my memory loss. I "knew" from my doctors that my memory would mostly recover within 6 months, so I was very casual, almost flippant, about the side effects. Throughout that fall, my mood was fairly positive, and since it was a temporary effect, it was something to make fun of among friends: jokes from them, "You mean you don't remember that \$500 I loaned you?" or my mock insistence that it was their memory that was impaired, not mine. "I know for a *fact* that we've never been to this restaurant before."

Perhaps more important to my light attitude was the fact that I had no concept of how much information was gone from my past. It may seem obvious, yet it becomes a truism that may cast one bit of light on those impairments that are not reported by patients when research follow-up is only done in the first few months: You cannot be aware of something that is missing. It is only through the gradual process of hearing others talk about the miscellany of life that one rebuilds the knowledge, though not necessarily the memory, of events past. Until that process develops, the vacuum remains unknown and unknowable, so the panic and sense of loss do not occur immediately. When, as in my case, follow-up assessment is not routinely done, the severe losses may remain unknown to the treating physicians, and any care for coping is thus left undeveloped.

It affected my relationships with newer, more casual friends in a very different way. I simply did not remember the status of our relationship. In addition, the gap in time caused by the gap in the corresponding memory period made it seem like far more time had passed than was real. I was not prepared to discuss ECT with them, and without being able to explain uncertain overtures, I was not comfortable approaching them. Most of these friends knew basically about my illness, and would have waited to hear from me, not wanting to intrude. The relationships with these people basically drifted away. Public stigma over mental health has been reduced somewhat in recent years, and it is not difficult for me to reveal my disability anymore, but ECT remains in a class of its own. I have encountered stunned silence or even horror.

As the 6-month marker came and went with only partial recovery of my recollection for past events, my focus began to change. I was again not doing as well emotionally, which affected my positive attitude. In addition, some mental health advocacy groups that were hosting a disability information day at the Vermont statehouse had asked me to put together a revised fact sheet on ECT. Feeling inadequately prepared, I did some superficial research.

I was completely stunned by the discrepancies I found. While multiple studies found any long-term amnesia to be extremely rare (as summarized by Sackeim (1992), informal accounts, advocacy group information, and newspaper exposés described extensive and broad-based risks (Breggin, 1979; Cauchon, 1995; Vermont Protection and Advocacy, 1996). Hearing claims such that ECT caused brain damage were terrifying to a layperson when discovered without yet knowing the questionable professional standing and credibility of the sources.

I had in fact experienced significant and long-term impairment that I could easily distinguish from ordinary memory fallibility. Yet as I reviewed what I had found, it seemed clear that comprehensive efforts to assess long-term adverse effects had not been made. I found repeated acknowledgment that more research was needed on memory loss (Culver et al., 1980; Weiner, 1984; Kaplan and Sadock, 1989; Calev, 1991; Sackeim, 1992; Devanand et al., 1994).

Despite the controversy and lack of a secure foundation in research, there appeared to be a general consensus among most experts, writers for lay audiences, and practitioners, which all grossly minimized my experience: 1) that virtually all impairment is reversed within 6 months, and 2) while there may be evidence for rare cases of more permanent loss, such loss existed only for spotty autobiographical memories from the few months before or after ECT (Squire and Slater, 1981; Frith et al., 1983; Kaplan and Saddock, 1989; Calev et al., 1991; Janicak et al., 1991; Papolos and Papolos, 1992; Sackeim, 1992; Ablow, 1993; Salmans, 1995; Sobin et al., 1995; Coleman et al., 1996).

Anecdotal evidence suggesting greater permanent impairment was often dismissed on the basis that other causes were more likely: the effect of depressive illness itself, the heightened sensitivity to loss being used to misidentify normal memory loss caused by such processes as aging, and exaggeration of effects for reasons such as perceived secondary gains of having a cognitive disorder (Squire and Slater, 1983; Weiner, 1984; Weiner, 1989; Sackeim, 1992; Sobin et al., 1995; Coleman et al., 1996). Research such as that of Squire (1981) and Weiner et al. (1986) suggesting the possibility of more persistent deficits, as I would later learn, was considered no longer persuasive since it was based upon comparisons with outdated sine wave technology, failed to take into account the independent effects of depression, or was considered methodologically inadequate (Calev et al., 1991b; Devanand et al., 1994; Coleman et al., 1996).

As shaken as I was to suddenly feel like an involuntary game piece in the center of a quasiscientific, quasipolitical debate, I was also intrigued. My discussions with family and friends shifted from jokes to serious efforts to pin down information. I began to initiate many more questions about events of the past, and thus to learn more and more about the extent of my amnesia. I also began to recognize the variation in some of the ways I was either recalling, or possibly falsely recalling, different memories. At times, particularly early on, full recall clearly did occur, whether spontaneously or based on a reminder stimulus despite an initial gap. There are other major past events for which memory has never returned. In the summer of 1992, for example, 3 years before my first treatment, my parents built their retirement home adjacent to my log cabin. Based on the skills I had learned building my own cabin, I volunteered to do all of the plumbing for their six-bedroom, six-bathroom, three-story house. I have several photos of my father and myself triumphantly completing the first basement main hook-up. Yet I have no recall whatsoever of this massive undertaking.

Between retrieval and total loss lies an unknown cross-over range of partially filled-in, partially reconstructed, or possibly completely "created" memories through external information planting, which then becomes mistaken as an actual memory. These obviously are very difficult to distinguish. Shortly after my last treatments in June of 1996, I remembered some sense of importance attached to something having happened in Oklahoma City. When I asked about it, my brother filled me in about the events a year earlier, and it was all completely new to me. As time went by, however, and I have seen pictures of the tragedy, it seems to me that I have regained vague memories—but only those which correspond to the pictures.

Similarly, in March of 1996, friends from as far away as Missouri gathered for a weekend reunion at my house for my 40th birthday. I have a full photo album: the evening dance, visiting the sugarhouse, the morning pancake breakfast. I had been relatively sure that I remembered the weekend, despite it being sandwiched between my fall and spring

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ECT series. But then I was told about all the fun we had sledding that weekend, the great airline fiasco one family endured, the serious hand injury a friend incurred on the rope swing—there are no pictures of these events, and it has become clear that the only memories I have are of those things that do exactly match the pictures. Are the memories completely suspect as false creations, or is a photo trigger more effective than a verbal trigger in bringing back actual memories?

The basic research I had uncovered on ECT side effects made no effort to distinguish among the many variables of human memory. It is relatively clear that the brain both routinely loses or has a break in the process of retrieval from long-term to working memory (Harrell et al., 1992), but also creates memory. Fiush (1996) points out the degree to which "we can be misled about our own memories" (see also, Crowley and Underwood, 1998; Payne and Blackwell, 1998). I had some limited familiarity with the work of Elizabeth Loftus on memory from research I had done years earlier as a young lawyer assisting in a murder case involving mistaken identity, and I knew the field had been pioneered when the issue of retrieved versus falsely created memory first became a debate in eyewitness identification cases and then in childhood sexual abuse prosecutions several decades ago, as noted by Alpert (1996). I had been fascinated then by what I learned about the brain and the inherent unreliability of our memory mechanisms.

As time goes by, earlier memories cued back after ECT seem more and more real to me, regardless of whether they ever were. Schooler (1996) observes that information integrated into memory can be held "with as much confidence as real memories" (see also Hirt et al., 1998; Moscovitch, 1989). This question is not a part of the studies assessing recovery of memory from ECT. For instance, while Sackeim (1992) summarizes the generally accepted description of the effects of ECT as being that, "The retrograde amnesia will often show a more gradual reduction, with substantial *return of memory* for events that were seemingly 'forgotten' immediately following the treatment course," (emphasis added), memory researchers such as Toglia (1996) point out instead that "The constructive nature of memory is sufficient to create recollections that are essentially entirely false" (see also Schooler, 1996).

The lack of connection between these fields also meant that I began a search that would last years to try to get memory assessment and help, when the resources should have been well known and available—and I should not have been the one to have to identify the need.

After that first significant experience of looking at basic ECT research, I became more anxious to gain a fuller understanding of what had happened to me. By the spring of 1997, I was in a more stable remission, and became eager to learn more about what was and was not known in whatever additional research I could track down. In doing so, I came to understand more fully the disparity in my case from standard statements about ECT, and I was increasingly frustrated in seeing how limited the data seemed on what the more serious effects could be.

Improved research was clearly not an easy task, particularly with the difficulty of devising tests to confirm the often random or isolated memory losses reported by many patients. The personal nature of perceptions, the complexity of human memory, and the processes of encoding, retrieval, and normal forgetting (Kellner, 1996; Cowley and Underwood, 1998; Payne and Blackwell, 1998), and the question of whether at the time testing is typically done the patient has gained an adequate sense of the degree of memory

loss (Coleman et al., 1996) are only part of the challenges. There is also the difficulty in pinpointing what an individual's prior memory would accurately recall, particularly in a person with depressive symptoms (Sackeim and Steif, 1988; Sackeim, 1992; Sobin et al., 1995) (including the possibility that those with specific preexisting impairments are more at risk for more severe impairments, as well as more likely to have subjective complaints) (Sackeim, 1992; Sobin et al., 1995; Coffey, 1996). Finally there are the limitations of scientific research in general, as summarized by Sackeim (1992), including the use of tools with inadequate psychometric properties, the issue of the breadth of cognitive functions evaluated, the need for a wider range of ecologically valid assessments, review of the aberrant processes involved, and the limitations of intergroup comparisons pooling data across individuals, so that outliers with more persistent deficits may be missed since "within any research program such individuals would be too rare for meaningful analysis," or so that effects with a low incidence of occurrence will wash out (Weiner et al., 1986a).

Despite the challenges, it seemed incredible to me that more had not been done. How could it be possible that ECT had been practiced for so long without a better grasp of its side effects? How could the research be so apparently limited in its focus and assumptions? Why was there no greater effort to understand *why* ECT caused cognitive losses, how often they occurred, and how severe they might be? Perhaps more than anything, how could I be experiencing what I was, if all these experts were saying it wasn't so? I felt that I was being mocked by science.

As the year continued, I began to care much more intensely on a deeply emotional level about what had happened to me. To what degree was I a different person, someone I did not even really know, because I had lost so much memory of my past? Finding out more and filling in the gaps suddenly became an urgent matter, and I pressed family and friends to distraction about events I might remember. The more I heard, the more I realized what I had lost, as one topic led to another. In the period between my two treatment series, and for 1 to 2 years before the beginning of the first, it became clear that I had near-total retrograde amnesia. It was more spotty but still significant for years earlier than that. Every new incident continued to shake me, and I did not know how to cope with these gaps and my reactions.

By that fall, a year and a half after ending ECT, I had finally regained a more stable period of remission, and I began to work on my own to develop a more gentle and constructive perspective. It occurred to me that there were times I needed to remember that I had amnesia—specifically, for one, when asked about my medical history. I had noticed a series of tiny bumps on my forehead and thought they were pimples, then later saw in a mirror that it was a scar from stitches. I had to ask my doctor to trace back through my files to find the record of a fall and stitches in 1994.

I needed to remember not to deny events based on my own belief—not to forget that I might have forgotten. I have to keep vigilant to not prejudge someone as lying or misleading simply because I forgot that the fault could have been my memory. Early on, I attacked my local bank for negligently bouncing a check. They had to show me my own signature before I believed that the check came from an account that I had closed. I had to alter my everyday thinking patterns, have a suspicion towards every recall. Sometimes it gave me a sense of being an outsider looking in to my own past world.

As I finally began to reach an equilibrium, an acceptance of what happened and how

to learn to live with it, I have become more relaxed in dealing with everyday situations that continue to arise. I find people almost universally helpful when typical encounters occur.

Woman on street (*in chance meeting*): "Anne! How wonderful to run into you. How are you?"

Anne (*rapid assessment: This is a person once well known, not a passing acquaintance who can be handled by bluffing through a conversation*): "Well, hello! Listen, I need to fill you in on something. I've been ill and a treatment I received has blocked my memory for several years back. I have to be honest. I have no idea who you are."

Woman: "Oh! Well, I'm Catherine S., from our time working together in New York in 1986."

Anne (*much relieved*): "Of course! Seeing you here out of time and place just threw me off. I remember now." (As well I did, from 9 years prior. I had just never expected to see her here in Burlington.)

Anne (*continues*): "Well, it's great to run into you here. What brings you to Burlington?"

Catherine: "I live in Burlington, remember?"

Anne: "No, I never knew that."

Catherine: "Well, actually, you did know that. We've had lunch together here several times over the past few years, and I've been out to visit you. It must be that treatment you mentioned."

I have never had a negative reaction to this kind of honesty. I do not necessarily go into a further explanation if I am having a passing encounter, but I do feel free to do so when there is time and supportive interest from the listener.

Despite acceptance and a growing comfort level in talking openly, despite the emotional outlet for anger through the development of my academic interest, and despite working through the experience of losing part of my sense of self, I remain bothered by a sense of incompleteness. It is obvious that if there is a serious side effect after heart surgery, there is follow-up intervention. The patient is checked for residual bleeding.

I had not been checked for residual bleeding. I feel left hanging—that nothing was ever comprehensively tested, recorded, or analyzed by the psychiatric profession and those involved in my care to evaluate my side effects: not just to intervene and to help me, but also to learn from my results. This should be routine when initial response shows significant cognitive impairment, as mine did. If it is done more adequately in other situations, the information, regardless, has not been collected and shared. No wonder the establishment has a different sense of the side effects. They don't ask.

I think that this lingering feeling of abandonment of care by the psychiatric profession, both as an individual and in a deeper sense on behalf of my peers, is strongly related to the part of me that still feels so damaged by my memory loss.

CONCLUSION

My story is my own—what happened to me, and the care I did or did not receive cannot automatically be assumed to apply to the practice of ECT in the U.S.A. today or to the follow-up care delivered when severe side effects result. The broader existence of activist groups of former patients who, for whatever reasons, are disgruntled by their results

sufficiently to attack the treatment itself, should also contribute to giving pause as to potential adequacy of care explanations for the vitriol of their distrust. While such attacks may be poorly founded, they are part of a landscape that both interferes with a vital psychiatric tool and helps to illustrate the pressing need for an improved response.

My own experience, and the research I pursued as a result, has led me to three major conclusions regarding areas which should receive greater attention:

1. Make a more concerted effort to conduct research that can better explain the discrepancies between the "official" consensus and the reported experiences of those uncounted and unaccounted for "outliers" like me.
2. Reduce the gaps in clinical knowledge.
3. Improve information on side effects and resources for aftercare for patients.

Weiner's (1984) call for precisely the *type* of information on the "nature, incidence and severity of possible persistent memory deficits... [including] large, well controlled prospective studies with long term follow-up" that I found so lacking and distressing, remains unanswered.

Research on improving efficacy and reducing side effects in the future is not the same as evaluating past and current status, and despite the efforts of the American Psychiatric Association (see 1990 Task Force report) to increase knowledge about the more potentially severe range of effects, there remains a near constant circulation of highly uninformed descriptions denying a problem [see, for example, Internet Web public medical information page stating, "The memory disturbance that has alarmed the public is relatively minor and temporary." (Mental Health Infosource, 1999)]. Paul Fink is quoted in a practitioner's study guide as saying during a practice guidelines discussion, that "The biggest reason people don't want to use it [ECT] has to do with memory loss. I am of the school that believes there is a transient confusional state and no memory loss" (Study Guide on Practice Guidelines, 1994) [statement later privately withdrawn (P. J. Fink, personal communication, Dec. 14, 1998)].

The juxtaposition of misstatements about the extent of possible memory loss continues the perception that side effects are being described dishonestly, to one extreme or the other. It is a part of what makes ECT different from other medical care, since the discrepancies "keep the controversy raging" (Center for Mental Health Studies, 1998), can be a factor in refusing treatment, and have resulted in public efforts to have it banned or to have informed consent statutorily defined (see impact upon state practices, Herman et al., 1995; and samples of legislation passed or pending, Cal. S.5326; Col. R.S.13-20-400; Texas 578.000; Vermont House of Representatives bill, H.12, 1999). My attempt at gathering accurate information went far beyond a typical patient's, yet I gained little more than the fear and confusion generated by grossly conflicting and limited data.

There are also serious gaps in clinical knowledge. If the treating psychiatrist is not fully aware of the degree of memory effects, he or she may also not recognize how critical the latest developments in techniques may be. I cringe when I review the ongoing research evolving, and recognize that even without an explanation for how and why extensive deficits do occur, they might have been avoided for me with insights on appropriate treatment. Practice levels currently often fall short of scientific updates, with Sackeim (1998), for instance, reporting that an estimated 30% of practitioners are still using the

substantially higher-risk, pre-1985 procedure of dosing at a uniform, high level. Other practices exceeding recommended guidelines are documented by Reid et al. (1998). Thus my medical cost-benefit analysis in accepting ECT treatment was skewed from the start by the fact that the existing professional statements on potential risks did not match the actual risks presented by current mainstream practice.

The final issue is the information provided to the patient. As Sackeim (1992) notes, even though the reasons for the discrepancy between objective testing and subjective reports are unknown, "...in informing patients about ECT, it is important to relate that a few individuals report profound and long-lasting cognitive impairment that they attribute to this treatment modality." Kellner (1998) appropriately suggests that the key to improved informed consent is "a middle ground that does not appear defensive": disclosure both of ECT's powerful, lifesaving effects and its serious side effects, dealing with it in a way that eases apprehension and allows an informed choice between typically brief impaired functioning and a return to health (Kellner, 1996). While this goal has been clear at least since the 1990 Task Force report of the American Psychiatric Association, his belief that, "Nowadays, we *do* tell patients what to expect and everyone is better for it," (Kellner, 1996) is not yet a universal reality.

Because ECT involves a series of treatments during which the cost-benefit ratio continues to change and the patient's ability to participate in informed decision-making often continues to improve, while at the same time, memory of the original consent may become impaired (Consensus Conference, 1985), potentially contributing to patient perceptions that side effects were worse than expected (Bernstein et al., 1998), a better record available later to the patient of his or her own participation in the consent process (such as offering to audiotape or videotape, or having a family member or friend present), as well as written information for a follow-up cognitive assessment plan if needed, should also be provided (as an example of work with coping skills, see Harell, 1992). None of this was offered to me, and it was the lack of information, as much as the actual effects, which made recovery so difficult.

In addition, as Kellner (1996) so well summarizes, "Preparing a patient for the predictable, expectable, and largely stereotyped effects of ECT on memory and other domains of cognition is honest, necessary and helpful. It leads to realistic expectations for the treatment, and can help the patient and family prepare for the post-ECT period. Disappointment and fear are decreased and some practical steps towards restoration of memory (coaching, list-making and 'filling in' by family and friends) can be planned."

Without these advances—more comprehensive research regarding causes and rates of the most severe instances of memory loss, better transmission of new clinical information to practitioners, and more comprehensive, accurate information and follow-up for patients—a vital tool in the battle against life-threatening affective disorders will remain underutilized. It is a major social loss that should not have to be that way.

If sharing my own experiences of successful treatment but deeply troublesome side effects can help in that cause—if my voice is heard, and heard to speak for others like me—then my own sense of damage and abandonment will be assuaged. It will give my experience a value in the lives of others. It will not help my own memory to return, but it will ease the pain of the feeling that the damage may have been unnecessary to achieve the results.

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The first part of the paper discusses the importance of the study of the history of the United States. It is argued that a knowledge of the past is essential for a full understanding of the present. The author then goes on to discuss the various factors that have shaped the development of the United States, including the role of the government, the economy, and the culture. The paper concludes by suggesting that a study of the history of the United States is not only a valuable academic exercise, but also a necessary one for anyone who wishes to understand the world in which we live.

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Abstract **ADVERSE PSYCHOLOGICAL EFFECTS OF E.C.T.**

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Abstract:

Results Although it is known that a proportion of people find ECT distressing to receive, these adverse psychological reactions are little understood. Twenty people who reported having found ECT upsetting were interviewed about their experiences in detail. A variety of themes emerged, including feelings of fear, shame and humiliation, worthlessness and helplessness, and a sense of having been abused and assaulted. This had reinforced existing problems and led to distrust of psychiatric staff. Few had felt able to tell professionals of the strength of their reactions, implying a possible hidden pool of trauma. Implications for the practice of ECT are discussed.

Discussion

References

Author

Although ECT (electroconvulsive therapy) is widely used in depression and some other conditions, it continues to attract controversy. Disagreement mainly centres around the possibility of memory loss and intellectual impairment, with the generally accepted official view being that "As far as we know, ECT does not have any long-term effects on your memory or your intelligence", (Royal College of Psychiatrists, 1997.) Although the debate about cognitive impairment has received much attention, (Breggin 1991, Frank 1990, Friedberg 1976), the question of possible unwanted psychological effects has, until recently, been almost totally neglected. No mention is made of them in most summaries of adverse effects, such as that in Weiner and Krystal (1994.) The ECT handbook contains a single paragraph referring briefly to pre-treatment anxiety (Royal College of Psychiatrists, 1995.) This omission has been commented on both by psychiatrists: "Doctors who give ECT have shown remarkably little interest in their patients' views of the procedure and its effects on them, and only recently has this topic received any consideration in the literature" (Abrams, 1997), and by service users: "What is never discussed in the literature is the profoundly damaging psychological effects ECT can have" (Lindow, 1992).

This is in contrast to earlier, mainly psychoanalytic, theorising about the psychological impact of ECT. Psychogenic theories of ECT's action were summarised in a review article by Cook (1944). Earlier belief in the therapeutic effects of fear had been largely replaced by theories about the healing nature of this symbolic death and re-birth. There was speculation along Freudian lines that the fit "by its severe motor manifestations 'discharges' large amounts of energy inherent in the destructive and death drives and unloads them in a....harmless manner." Gordon (1948) listed twenty-three possible psychological explanations of ECT's effects, such as the destruction of narcissistic protective patterns and the eroticising of the body. Some clinicians believed that these and other hypothesised reactions, such as the relief from guilt and self-punishment following the experience of "a sadistic, real attack", made the conjunction of ECT with psychoanalysis a particularly fruitful one

(Weigart 1940 in Boyer 1952.) Boyer includes a lengthy case history in which the young female client equates ECT in fantasy not only with death and re-birth, but also with intercourse, castration and impregnation, with ultimately favourable results in her therapy.

On a less positive note, Abse and Ewing (1956) noted that conscious attitudes towards ECT are "time and again", in long-term therapy, succeeded by feelings that it was cruel and destructive. There is "a revival of threatening and punitive parental figures" who are often, like the physician, initially credited with good intentions. The ECT appears to arouse anxiety and fear, while at the same time holding out hope of forgiveness and a fresh start. Wayne (1955) noted that certain aspects of the procedure may evoke unconscious meanings in both doctor and patient; for example, "It has all the characteristics of an overwhelming assault....and this can be documented by the reactions of some patients who have had this treatment." Fisher, Fisher and Hilkevitch (1953) investigated the conscious and unconscious attitudes towards ECT in 30 psychotic patients, and concluded that "the majority of patients found electric shock to be a traumatic experience." D.W. Winnicott (1947) argued that psychological reactions to ECT often compounded patients' difficulties and defences; for example, obsessional people might need to become even more controlled.

An exception to these analytically-oriented accounts is Warren's (1988) description of the implications of ECT for the self and for family relationships. In her interviews with ten women admitted to a state hospital in California between 1957 and 1961 and their relatives, there was uniform confusion and bewilderment at the loss of memory in everyday life. Sometimes this forgetfulness, for example of previous hostile outbursts, was welcome to their husbands. Fear of future ECT stopped some women from confiding emotional upsets, and family relationships were subtly altered all round.

With the general decline of psychoanalytic influences on psychiatry, theorising and research in this area appears to have been abandoned until Gomez's survey (1975) of side-effects in 96 ECT patients. Findings from this and other attitude studies (for example, Freeman and Kendall, 1980; Hughes et al., 1981; Kerr et al., 1982) were reviewed in Freeman and Cheshire (1986.) Subsequent studies by Malcolm (1989), Szuba et al. (1991), Riordan, Barron and Bowden (1993), and Pettinati et al. (1994) used essentially the same format of asking patients to respond to questions or complete checklists about their attitudes to and experience of ECT. The conclusions from this series of investigations can be summarised as follows:

- *Most people appear to find ECT helpful (varying from 83% in Hughes et al to 56% in Riordan et al.)

- *Most people also report side-effects, (around 80% in all studies), with memory impairment complained of most frequently, and headaches and confusion mentioned less frequently.

- *Most people do not seem to find ECT particularly frightening to receive (Freeman and Kendall; 50% less so than a visit to the dentist.) However, a majority does

experience some level of anxiety (74% in Gomez, 69% in Riordan et al.), and a significant minority reports much stronger reactions; (13.1% said it was so upsetting that they would not want it again, Freeman and Kendall; 14.3% say it was more upsetting than surgery, Pettinati et al; 23.7% agreed with the statement that ECT is a barbaric, inhumane treatment, Kerr et al.).

*Most people do not report other anxieties about ECT, although a minority does mention worries about brain damage. Death, personality change and being anaesthetised are also feared by some.

*Most people who have had ECT are profoundly ignorant about the whole procedure, and say that they were given no or inadequate explanations. (69% did not know that ECT involved a convulsion, Hughes et al.. Only 21% said they were given a good explanation of the procedure, Freeman and Kendall.) It is not clear how much these findings were influenced by memory loss.

(Two other studies produced broadly similar results, but are not directly comparable to those described above because scores for each item were averaged across all responses. See Calev et al., 1991, and Baxter et al., 1986.)

In summary, these studies would seem to justify Freeman and Kendall's (1980) often-quoted conclusion that patients find ECT "a helpful treatment and not particularly frightening." However, there are reasons for believing that the picture may be more complicated than this.

Firstly, there are the limitations acknowledged by Freeman and Kendall, which may apply to some extent to all these psychiatrist-led investigations: "It is obviously going to be difficult to come back to a hospital where you have been treated and criticise the treatment that you were given in a face-to-face meeting with a doctor." Earlier researchers certainly found such factors to be relevant: "The majority of

the patients seemed to be motivated by the idea that any criticism that they might make of shock would in an indirect sense be a criticism of the psychiatric staff....patients expressed themselves sincerely only after the interviewers spent considerable time in establishing a relationship." (Fisher et al., 1953.)

Secondly, there is the unusual degree of compliance noted by several investigators, who were puzzled by patients' willingness to agree to ECT despite being anxious and ill-informed: "We were left with the clear impression that patients would agree to almost anything a doctor suggested" (Freeman and Kendall, 1980.) Referring to the same phenomenon, Riordan et al (1993) suggested, "This may reflect a high level of trust, or a resigned lethargy, in part reflecting mental state, but also a feeling of lack of involvement in their own management". Freeman and Kendall (1980) quote a particularly striking example: "Two patients who misunderstood the initial appointment letter came fully prepared to have a course of ECT. Neither had been near the hospital for nine months and both were quite symptom-free." Little attempt was made to explore the meaning of this kind of behaviour, but it does raise the question of whether the absence of criticism reflects satisfaction, or merely learned helplessness and passivity.

Thirdly, there is the fact that a minority of people in all the studies did express very strong negative feelings about ECT, although this has been obscured by focussing on the majority view. In the only paper that acknowledges this as a problem, Fox (1993) describes how a "difficult-to-elicited, etiologically obscure, and currently under-recognised 'pathological' fear of treatment develops in some proportion of patients undergoing ECT.....Fear of E.C.T. merits further study."

Fourthly, there are several recent surveys carried out by investigators from outside the hospital setting which paint a much less reassuring picture. In the first one, UKAN (United Kingdom Advocacy Network) received 306 replies to a questionnaire distributed through UKAN- affiliated groups, Mindlink and Survivors Speak Out (both the last being service-user run organisations.) Overall, 35.1% described ECT as "damaging" with another 16.5% saying it was "not helpful." Although 30.1% found that it was helpful or very helpful, those who did not were likely to express very strong views against it, using words like "brutal", "barbaric" and "degrading." Psychological after-effects included loss of confidence, dignity and self-esteem; fear of hospitals and psychiatry; anger and aggression; loss of self; and nightmares. Similar themes emerged from a series of semi-structured interviews with 516 psychiatric patients contacted through MIND (Rogers, Pilgrim and Lacey, 1993.) While 43% found ECT helpful or very helpful, a large minority (37.1%) said it was unhelpful or very unhelpful, with a high proportion of the latter group strongly condemning it. Psychological effects included fear, flashbacks and nightmares. The same themes emerged from two smaller surveys by two researchers who had had ECT themselves, (Wallcraft 1987, Lawrence 1997) and from MIND's (1995) survey on "Older Women and ECT." In addition, the recently-formed organisation ECT Anonymous has collected several hundred reports from people who say that ECT has had a variety of disabling physical and psychological effects on them. However, respondents from all these sources were self-selected and might show a bias towards greater dissatisfaction.

In summary, all of the more recent research acknowledges that a proportion of people have very strong reactions against ECT, although very little is known about the nature of, and reasons for, these adverse psychological effects. The differences between the reported rates of adverse reactions (varying from 13.1% in the hospital-based surveys to 35.1% in the others) also remain puzzling.

While some of the earlier accounts may seem far-fetched, they do make an important point that has been over-looked in most subsequent surveys, that "there are crucial psychodynamic events involved in...organic therapy" (Abse and Ewing, 1956) and that attitudes can influence the outcome of the treatment.(Fisher et al 1953, Hillard and Folger 1977). Clearly, we need to know more about the meanings that ECT carries for a certain number of recipients, and which make it such a traumatic event for them. This may also throw some light on issues such as compliance and its possible effects on participants' responses. In order to investigate these areas, the existing questionnaires and pre-structured checklists of possible reactions need to be complemented by an approach that allows a detailed, in-depth exploration of the experiences of those people who find ECT a distressing event, entirely separate from the hospital setting. For these reasons a qualitative design was used in the present

study.

PARTICIPANTS

The study targeted people who had had negative reactions to ECT. They were recruited by posters and flyers asking, "Have you been given ECT? Did you find it upsetting or distressing in any way?" which were distributed through local mental health voluntary organisations. Twenty-two people contacted the researcher, and twenty were eventually found to fit the criteria. There were twelve women and eight men, with an age range of 27 to 63. One of the men was a female-to-male transgender. Ten were unemployed, and ten were involved in voluntary or paid work. Two described themselves as mixed race and the rest described themselves as white.

Participants were not always able to be precise about the details of their treatment, but nine of them reported that they had had more than one course of ECT, and six had had at least one course under section. The most recent course of ECT was 2-5 years ago for five participants; 5 to 10 years ago for five participants; 10 to 20 years ago for 6 participants; and 20 to 30 years ago for four participants.

It can be seen that within the overall category of adverse reactions to ECT, participants represented a wide range of backgrounds and treatment circumstances.

METHOD

The aims of the investigation were explained to the participants, and confidentiality was assured. The researcher emphasised that she had no current connections with psychiatric teams. Participants were invited to take part in a semi-structured interview at a place and time convenient to them, concerning all aspects of their experiences of ECT. Interviews were tape-recorded and transcribed, and a thematic analysis was performed on the results.

RESULTS

Themes can be organised under the following main questions:

What were the circumstances in which you came to have ECT?

Participants described their mental states at the time mainly in standard psychiatric terms, for example:

"I'm diagnosed as manic-depressive, and in those years I did suffer from some form of depression rather than mania, and I suppose I went into such deep depression that they thought ECT would help to get me out of it."

"I was just really depressed and I was getting a bit manic as well, and I didn't seem to be responding to the medication, and they said I should have a course of ECT."

However, as the interviews progressed, more complex background situations

emerged:

"I always knew I had problems that were emotionally-based, to do with my life. And although I'd gone in partly under the influence of drugs, LSD, I also knew when I was growing up that I had some problems."

"I was a very mixed –up and distressed person, and then my closest friend was killed six weeks after I got married....and my world fell apart."

"I was in nursing...One day I was a student, the next day I was qualified and in charge of a ward, which I wasn't trained to do.... I was just too young for the job."

"If I look back on what caused the depression and what caused me to try to take my life, it was quite normal, average things...a divorce, I had two children, I was three months pregnant when I left...holding three jobs down, mundane jobs, trying to keep it going really. I was worn out, absolutely worn out."

What kind of explanation of ECT were you given?

A problem here, as with other questions that asked for specific details about events, was that many participants had uncertain recall due to the effects of ECT itself: As in other surveys, nearly everyone felt that explanations had been completely inadequate or lacking altogether, and that there had been minimal opportunity for discussion.

"I don't remember anything being explained. I think they just said they were going to attach these things. I don't remember any discussion beforehand."

"She said, 'I don't think the Valium's doing you any good, so I'll put you on ECT.'"

Why did you agree to have ECT?

Six of the participants had had ECT under section on at least one occasion. The answer to the puzzling question raised by other researchers, of why the others consented despite inadequate explanations and the fact that many of them already had doubts based on the experiences of relatives or other patients, lies in their feelings of extreme desperation and powerlessness.

"I was so ill, I felt so desperate, I didn't know which way to turn. I was just looking for answers as to why I was so strange, so peculiar."

"I wasn't in a fit state to make any of those decisions. We were just grasping at straws, trying to find an answer."

"If you're at your wits' end and they've drugged you up to the eyes you don't question....you're not thinking straight anyway."

This desperate desire to get better was often coupled with a tendency towards compliance and a strong assumption that "doctor knows best". Moreover, participants felt they could not risk alienating these powerful people who seemed to

hold the key to their cure:

"I was a very compliant young woman, I was very frightened of everybody and that was part of the problem...I wouldn't have known how to object, it wasn't on the horizon. You didn't disagree with doctors, you did what they said."

"You believed that whatever they were going to do was going to work, you believed what you were told really."

"He is the one with the power, he is the one ultimately that has the answer...if that's the only help you're getting you've got to hang on to it."

A man who ended up completing his course of ECT despite his own reluctance and encouragement from the nursing staff to refuse it, put it like this:

"It was like, the consultants and the psychiatrists have such a powerful influence over you. In one sense your life is in their hands and it's wanting to please them, I suppose, because...part of depression is losing your sense of self really, and you're so easily influenced and so easily willing to accept authority."

One woman found that her refusal to have further courses of ECT was, in fact, respected. Others who were able to be assertive were not so fortunate:

"They asked me if I would agree to it, but they did say if I refused they'd go ahead with it anyway...being forced to stay there is bad enough but being forced to have something that you don't want is ten times worse, so I did agree, yes."

"Now what so often happens in psychiatric hospitals is, it's not the psychiatrist that forces you to have it. Long before that happens you get confronted by staff nurses who are very anxious to stop hassle...so what they do, they see that you're weak and vulnerable and they say, 'You'd better sign', just like that."

"I said immediately that I didn't want it, and I pointed out that the previous consultanthad said to me that she didn't think I was an appropriate case for ECT.....and he (the consultant) got into a real huff basically and got up and walked out of the room...I felt absolutely devastated. I just burst out crying and didn't know what was going to happen to me, or whether they were going to section me, or what."

In summary, nearly all participants wanted to emphasise how far their apparent agreement was from being fully informed consent:

"I wasn't physically taken to the suite or anything, I walked there on my own, but I felt it was forced on me."

What was the actual experience of ECT like?

Six people said that ECT was not particularly frightening to receive, although one woman attributed this to the numbing effects of her medication. All the other

participants reported a very high level of fear, with a lack of accurate information sometimes supplemented by observation of other patients who had had ECT and by their own imaginations:

"I really didn't know what to expect, so I was absolutely terrified...I imagined great big metal things being put each side of my head and, like, sparks coming out, thunder and lightning, and my whole body shaking."

"When you'd been on the ward there were certain people who had had ECT and all the other people were very scared by this....you would see them afterwards when they couldn't remember who they were and were very confused and had terrible headaches and weren't themselves at all."

All this generally produced a high level of anticipatory anxiety:

"I remember the very first time I had it, walking down to the ECT (suite) from the ward and I remember feeling very agitated, sick and scared. And when I got into the waiting room there, I came to a standstill. I couldn't go through with it, I didn't want it. They talked to me and said I'd signed the consent form and I was under section."

"As they wheeled you in you'd see what they used, they'd put some gel on it, they didn't even hide it from you.... You were scared, yes."

"I can remember sitting in the room waiting for treatment and looking at some of the other people who were there as well and I suppose it was almost like a pre-execution room really... We were all sitting there in complete silence. I remember reading in something, I think a hospital pamphlet, (that) it was just like going to the dentist, which is completely absurd....It's not like going to the dentist."

One participant reported that the reality was not quite as terrifying: However, the terror of the other participants remained or even increased as the course continued, and many found the immediate after-effects equally devastating:

"I thought maybe second time around it'll be much easier and I won't feel so scared and terrified, but it was just the same, if not a bit more."

"You dread it, your heart starts pumping, here we go again. Horrible, absolutely terrifying....It's like going to your death, your doom, isn't it."

"I was absolutely convinced they were trying to kill me...you know, I was so bad and evil, all they could do was get rid of me." (A woman who was psychotic at the time.)

"They could be doing anything, you don't know what they are doing....you get paranoid and think they are trying to poison you, or do weird experiments or something like that." (A woman with a diagnosis of paranoia.)

"Afterwards I felt as if I'd been battered...I was just incapacitated, body and

mind, like a heap of scrunched-up bones."

"....Pains in your head and the memory loss, and sometimes I used to have a bruise. I'd be dribbling, I looked insane....I felt terrible, I was only 22 and I must have looked 82. I just couldn't do anything."

When asked what was the most frightening aspect of receiving ECT, participants most commonly mentioned feelings of being helpless and out of control, and worries about long-term damage.

"It's a horrible sensation. You feel like a zombie, they could do what they want with you when you've had that and you would do it, because you don't know no different."

"It was the whole treatment, being carted off. I felt like a slave, taken away to this little room and put on a bed. No control, it was awful."

"You can't get it out of your head, how would you end up? ...you'd be so brain dead you wouldn't know what you were doing."

"What I was most concerned about wasn't the fact that it was unpleasant at the time, it was how it was going to affect me for the rest of my life....I remember feeling very disorientated and feeling that I'd been damaged for life."

For several, ECT was a confirmation that they were truly mad, and had reached the last option:

"It seemed to reflect how ill I was, the fact that he was saying I had to have ECT this time...this was the last desperate thing that they do."

"It was because this was the last resort...so what is there left, annihilation or what?"

"I knew I wasn't crazy. I knew what had happened." (After ECT) "I was beginning to think maybe I am mad...I must be mad to have ECT."

What other emotional or psychological effects has ECT had on you?

Fear is the only psychological reaction to ECT that has been investigated to any extent. However, these participants described a complex range of emotional responses including feelings of humiliation, increased compliance, failure, worthlessness, betrayal, lack of confidence and degradation, and a sense of having been abused and assaulted:

"It made me feel like a cabbage, like I wasn't worth anything at all. All I could do was sit around all day."

"It was like I was a non-person and it didn't matter what anybody did to me."

"I suppose I saw myself as worthless for a long time...almost being an empty person and having to start again, having to build up a personality, having to build yourself up."

"It's horrible to think that these people, doctors and nursing staff, are going to see you having a fit. It's degrading."

"I knew that the only way I could get out would be by being insignificant...by being a very good patient, and it worked. I wasn't any better, I felt quite terrible."

"I suppose as a woman, I feel...a lot of stuff was reinforced. You know, being the gender I am, it feels like you have to comply even more."

"It made me feel like a freak, and it's only since I've talked about that with a therapist about two years ago that I've got over that feeling."

"This psychiatrist had built this relationship with me, so I trusted him and then he did that (prescribed ECT). ... This chap had been clued up enough to realise he needed to build my trust, but didn't appear to be clued up enough to know that giving somebody electric shocks to their head might actually damage that trust...ECT I feel is just such a betrayal, this frightened young woman and they do that. Terrible."

"It's a really horrible feeling...a sense of failure, and what's wrong with me that I'm not getting better."

"It felt like I had been got at, yes, hashed, abused, as if my brain had been abused. It did feel like an assault."

Most people said that they did not mind others knowing that they had had ECT. For some, though, the perception by them and others that ECT is an intervention reserved for the extremes of madness, produced a strong sense of shame and stigma:

"I was deeply, deeply ashamed of having ECT.this was real serious stuff, this was a mad person."

"People can't imagine what on earth situation you need to be in, that you need to be electrically shocked. So they imagine that you must have been some kind of absolute raging animal or something to need that."

"I have told a couple of people in the past and they think for you to have ECT you must really be off your rocker."

ECT was experienced by several participants not just as a sign of madness, but also as a punishment for and confirmation of badness.

"At that time I was completely convinced I was being punished for something...I thought, well, I must have done something wrong to be treated like this."

"Maybe if I had been good or if I hadn't done this or that, I wouldn't be punished. Yes, I thought it's a form of abuse, a punishment."

Three of the women identified themselves as survivors of child sexual abuse. Of these, two drew explicit parallels between these early experiences and the experience of being given ECT, in terms of the emotions experienced at the time, confusingly mixed feelings towards both psychiatrists and original abusers, and inability to deal with their own powerful feelings of helplessness and rage afterwards:

"It certainly felt, 'Do what you like', and that's something I felt as a child, that I had no power, there was no way I could stop anyone doing whatever they wanted to me, so rather than get hurt I'll let them do it and maybe they'll like meespecially because it was men doing it, the men actually operating the machinery or whatever, and I can remember it was men putting the needle in. Yes, again there would have been no way I would have said I don't want this.....And then just sort of lying there, feeling really frightened and yet completely passive. So it was like all trapped, all my emotions were trapped anyway and my feelings were trapped, so it was all trapped inside. And on the other hand not caring what happened to me."

"I've had physical abuse as a child and I've had sexual abuse as a child and mental abuse as a child. I suppose I did think about it a couple of times going through the ECT, that this was some form of abuse, being put on you when you don't want it, or being more or less said that you've got to have it...I sometimes feel very angry to the people involved, that I can't get back at them or take revenge at them. So that I don't do that, I self-harm, I cut myself."

(LJ) *"Who do you want to get back at?"*

"Sometimes it's the doctors, the professionals, sometimes it's the abusers that have abused meI always tend to turn it in on myself. I've been told many times by doctors and counsellors, 'You've got to stop turning it on yourself', but I don't....It's like I feel I need to punish myself, maybe all the abuse is all my fault."

Although this investigation did not specifically seek to investigate the effects of ECT on memory, nearly all participants spontaneously reported some degree of loss. While acknowledging that medication and depression itself can affect the memory, they nevertheless believed that ECT had also been an important factor, and this caused much concern:

"Sometimes it really affects me, I break out in a cold sweat. Have I really got brain damage?"

"It's not the thought disorder that's disturbing me now, it's the damage done by the ECT...I've probably got another 50 years to go, and I thought, well, I'm going to be damaged for the rest of my life."

Some participants had lost large pieces of their lives, which was particularly upsetting where the memories involved young children:

"My memory is terrible, absolutely terrible. I can't even remember Sarah's first steps, and that's really hurtful...losing the memory of the kids growing up was awful."

"I can't remember when they started junior school, I can't remember when they left infant school. Now those are things you remember, they're highlights...and I'm quite resentful really to think that my ex-husband has got more memories of my children and did pretty well nothing to help."

The commonest complaints were inability to follow films, books or TV programmes, and problems with facial recognition. These disabilities were both frustrating and embarrassing. Less tangible was the general loss of sense of self described by a few participants:

"I can be reading a magazine and I get halfway through or nearly to the end and I can't remember what it's about, so I've got to read it all over again. Same with a film or a programme on the telly."

"I can understand the individual sentences but when it comes to taking in the whole story, you don't know what the hell's going on really...I like reading and I find it very irritating."

"People would come up to me in the street that knew me and would tell me how they knew me and I had no recollection of them at all...very frightening."

"It happens all the time. It's tiny little things, which on their own don't really matter, but it's this permanent sense of something that you've lost."

"It's a void, I can't describe it, and there's also a feeling of something fundamental that I don't even know what it is missing....just like an intrinsic part of me that I feel isn't there and it was once....Part of it feels like there was a real death of something, something died during that time."

Did ECT have any beneficial effects?

Nine people said that ECT had given them at least some temporary relief from depression, or in one case from hearing voices, although all but two of these felt that the costs had far outweighed the benefits. Two other participants reported a paradoxical effect:

"I felt I'd reached the absolute rock bottom and I couldn't go any further. Everything had been tried...Perhaps I felt the ECT gave me permission to get better."

"In a very bizarre sort of way, because the treatment and the abuse was so terrible, it made me come to my senses. I've got to get my act together, I've got to help myself."

Two of the nine believed that ECT had "worked" by triggering a high mood. A man with a diagnosis of manic-depression described how ECT had several times precipitated a change from suicidal depression to elation:

"I felt fantastic... Basically it puts you high, so you need the help then, that's when you need the help. Not, 'aren't you doing well, how are you feeling on a scale of one to ten,' 'oh about eight or nine, good I can get a job', 'are you, oh fantastic, go out and do it then.' Because you're sick, still sick."

A woman who also responded dramatically described it like this:

"I felt as though I had become a completely different person... I felt as if I had just totally gone off my head. I was totally dependent on the ward and everything and all of a sudden I think ECT had blasted me into this other reality. And some positive things did come out of it because I went out and I worked for a year and I was discharged from hospital... It was at a very high cost, obviously. You feel you've got to adapt to this new person that you are.... For a year or two afterwards I felt very mad... I felt I'd lost the person I used to be.... Too happy, really, too sort of split off from the side that was there before I had ECT, that all disappeared completely."

Nine years later, this woman felt that she had still not entirely reclaimed her real self.

Did you tell anyone how you felt about ECT?

Most participants had felt unable to tell psychiatrists or other professionals of the strength of their feelings about ECT, for the same reasons that prevented them refusing to have it in the first place. The few who tried to hint at their reluctance and terror felt they had been met with little response:

LJ "Did you explain to anyone how traumatic it had been for you?"

"No, I didn't dare. They had complete control over you, they could lock you up. You can't be angry with them. People who are, get a really bad time."

"Once or twice I've been able to say that I think it's a waste of time... and they say you've got to complete the course now, you've got to go through to the end and it's best for you and you're not in any fit state at the moment to know what you want. It's like the power's taken away from you all the time."

"I can remember asking him (the consultant) about what happened about me coming round (from the ECT) crying, and telling him I felt really frightened having it. And he certainly didn't acknowledge the fact it was frightening."

"I always said I wasn't feeling any better, but they started saying towards the end they thought I was feeling better, and I discovered a lot later that on my notes they invented that the ECT had been a successful treatment, and there was no way I was any better.... At the end of the treatment I had a meeting with the consultant who said he thought I was biologically cured of depression... The implication was, I suppose, that all the other things were just personal things I'd got to sort out."

It is perhaps not surprising that the experience of ECT had left some participants with a lasting distrust of mental health professionals and hospitals:

"When I was in hospital last time I was terrified that they were going to give it to me again. They promised they wouldn't, but can I trust them, can I trust them? I was terrified, I hated walking across the room where they did it."

"It was a useful lesson really. It's not sensible in this world to tell psychiatrists of your, what they call 'delusional systems', and in fact I never told them another one."

(This woman was feeling suicidal around the time of the interview, but had deliberately not told her community psychiatric nurse. She had previously had ECT under section.): "They've only got to mention the word hospital to me and I freak out...when I go into hospital, I won't trust nobody in there, because my mind runs away with me. Are they going to force me to have ECT? ... I know the staff on the ward, I've been there so many times, but each time I've been and come away, when I have to go back again I try and build that trust up all over again."

Many participants were very unhappy with other aspects of their psychiatric care, such as the use of medication. However, a number of them made the point that there is something qualitatively different about ECT: the idea of putting electricity through someone's head carries powerful symbolic meanings which still apply no matter how caringly the intervention is delivered. It can be experienced as a brutal assault on your very self:

"I think to tie somebody up and zap them with electricity....it goes back to the days of Frankenstein, doesn't it."

"Well, it's an assault on your head, isn't it? It's an assault on who you are, you are in your head. And yet you've gone to them expecting them to heal you."

"I would have thought anyone would be apprehensive about something like that, especially when they are messing about with your brain. That's the centre of your being, isn't it?"

"They make it all nice, they're nice to you when you go into the room, they pamper you a bit...talking to you very personably (sic) and all they want to do is jolt you with a thousand volts.....It goes back to the Jews, doesn't it, who went into this room and had a nice shower."

What other forms of help would have been more appropriate instead of ECT?

Nearly all participants were convinced, looking back, that ECT and all its disadvantages could have been avoided had the right kind of counselling and support been available instead:

"It was so obvious that one of the things I needed help with was grieving for this

friend. I needed to be given some way of knowing that I belonged to the human race."

"You used to say what you thought your troubles was, and she was nice, this doctor I had, and she would talk back and explain everything to me...If I could have carried on with her, on Valium, I would never have had ECT."

"There was one nurse who was actually a trained counsellor and about three or four years ago I was quite ill and there were things I wasn't disclosing to anybody, not even friends or whatever, and when I was in hospital I managed to talk to her and it all came out, and that was like a step forward."

"Although at that particular time I was very very psychotic, I needed to be allowed to be mad, but be somewhere with human decency and not be so restricted...I needed someone to talk to more than anything."

"Somebody sitting down with me in a room on your own, talking to you when you needed it...There were so many people on the ward and only three nurses, so you didn't get a lot of attention."

Ten of the twenty participants had ultimately been able to take up a variety of occupations including student, caretaker, and voluntary or paid worker in the mental health field. Two of the ten felt that they had recovered largely by their own efforts. The other eight had finally found the help they needed through a mixture of counselling/therapy, self-help groups and support from other service users:

"I've had private therapy on and off for about four or five years which I pay for, so that's helped a lot."

"I ultimately found the answer at a tranquilliser withdrawal group. I work for them and we all help and encourage each other, support each other and it's brilliant. And you have to build back your self-esteem, your self-worth, it doesn't just happen....and it's fantastic."

"I had so much inspiration from other people who were further on (at a support group), and I really just got involved and started helping out there and becoming a bit more empowered...I just knew that's what I wanted to do, try and help other people in the way that that helped me."

A common theme in this group was how anger at their treatment had turned their earlier compliance and conformity into assertiveness and a determination never to let others take control over them again:

"It taught me a lesson...always to question, never ever believe professionals, never assume because the doctor is a professional that he knows better than I do about my pain. I'm dreadful in a doctor's surgery. I do honestly make sure I get my time, I need to know what's going on. Never let them control me again like they did."

"It's really starting to come through now...angry at the way you've been treated by

people over the years, doormat, really put upon. I'm really starting to realise how badly at times I've been treated and now I'm changing that and putting my foot down and speaking out about things I'm not very popular, but that's too bad."

"I just feel...very angry, and basically I know my rights so much now, I'm in charge."

But most people still had unresolved feelings about ECT, in some cases many years later:

"Certainly if I do talk or read about ECT it does bring back all these horrible memories of the actual treatment. I always get the same symptoms, headaches, nausea and things." (23 years on.)

"I had absolutely terrifying lucid dreams. I couldn't explain to you how terrifying they are, it's just beyond words. I started telling this therapist about them to try and make sense (of them) and I always described this feeling as if I was having electricity...Terrible sensations, feeling like I was just about to die, and very, very lucid dreams, not like ordinary ones, where I wasn't sure if I was awake or asleep."

"This is one of the problems, when I feel I'm bitter towards this person, perhaps I'm not on Jesus's side...perhaps he hasn't accepted me because I hold this grudge." (A man with strong religious beliefs who was angry with the nurse who had put pressure on him to have ECT.)

"I do feel very angry, and sometimes I just have to stop myself dwelling on it because if I do I just get very angry. It's difficult to know what to do with that anger."

What are your overall views about ECT?

All the participants except one were very clear that they themselves would refuse ECT if they were ever offered it again. The exception was a man who said that he would consent as a "very, very last resort" if he ever became ill again.

One person thought that there was a place for ECT for some people, and thirteen others thought that people should be able to make their own informed decision on the matter. This was a conclusion generally put forward with some reluctance, with two participants adding that in their personal opinion it should be banned. The six remaining participants had no hesitation in calling for a universal ban even if some individuals wanted to have it.

"I think it's up to the individual really. I wouldn't touch it ever, even if I was really ill...I think if people gave you full information, a lot of people wouldn't have it."

"Personally I think there should be a ban, but until that happens I suppose if users feel it might benefit them, then go ahead, but I'd like to see in the next few years a total ban worldwide."

"It is not justifiable to give people something that harms their brains and gives them an epileptic fit on the NHS. It's just not, in my view, an ethical way to proceed."

Most participants expressed their overall views on ECT in strong terms. They saw it as a blunt instrument that produced brain damage without dealing with the person's real problems:

"It's like being hit on the head by a hammer, that's the way I would describe it...How do I know they're getting the right area and don't kill cells in a different area? It's a crude tool."

"Well, it deadens your brain, doesn't it? That's what it does."

"They didn't have the time and they didn't have the staff and so I think ECT is just a quick way, a quick job, less expensive."

"It's short-term relief...obviously until you find a solution to the problem it's just going to recur and you're going to keep on having ECT."

"I think it is barbaric giving it to people on the scale that it is. And I've never actually met anyone who said it had done them any good, so...I don't know where this eight out of ten figure comes from." (The proportion of people benefiting from ECT, according to this man's consultant.)

"Quite barbaric, really, barbaric to put electric shocks through people's heads."

"I think it works by causing brain damage...It knocks out the memory...so being unable to remember the unpleasant feelings, you are less able to feel depressed."

"When you think that shock treatment is a form of torture, then you can see the relationship...It's very extreme and it's abusive. Well, it's not a treatment really, is it, it's just a violation of a person's body."

"To be treated physically for something that isn't a physical complaint...I do object to that for emotional, psychic, spiritual problems."

"It is inhuman and inhumane."

DISCUSSION

Since this study specifically targeted those with a negative experience of ECT, the results cannot be taken as representative of all ECT recipients. However, the study does confirm that for a certain proportion of patients, ECT is a deeply and lastingly traumatic experience. Few participants doubted the good intentions of the professionals; as one of them put it, "I don't think the psychiatric system is made up of bad people wanting to harm people." Unfortunately, the fact that professionals genuinely believe that they are acting in the patient's best interests by prescribing ECT does not guarantee that the patient will experience the intervention as beneficial. This investigation provides ample evidence that organic therapies do carry

meanings, and that these meanings, filtered through the individual's own background/context and interpretations, influence how such therapies are experienced. Having said this, we must be careful not to discount the possibility that some of their concerns also have a factual basis; for example, that ECT does cause definite cognitive impairment, and that anxiety about brain damage is not just a psychological phenomenon but an understandable response to a real danger.

Although participants represented a wide range of treatment circumstances, the themes that emerged from their accounts were remarkably similar. There are a number of areas of particular concern to mental health professionals. Firstly, there is the fact that ECT may be undermining therapeutic work in ways that professionals are unaware of. One woman appreciated her psychiatrist's sensitive attempts to build a relationship with her, but lost all trust in him when he subsequently prescribed ECT. Another was encouraged to direct her anger outwards, while simultaneously being forced to undergo a treatment that increased her anger and self-blame to the point of self-harm.

Secondly, ECT may actually exacerbate existing psychological problems. Some participants who already believed themselves to be bad, saw ECT as confirming this. Several women who saw unassertiveness as having been part of their problems, received the message that they must comply and keep quiet. A man whose religious beliefs had caused him great conflict was deeply worried about his unresolved anger about ECT. In addition, ECT seemed to feed into two women's delusional beliefs; one was convinced that she was being killed, while another thought that "weird experiments" were being carried out on her. Feelings of shame, failure, badness, unworthiness, self-punishment and helplessness are common features of depression, and insofar as ECT reinforces them, it will obviously be unhelpful. Perhaps most worrying were the cases of the two women survivors of sexual abuse who clearly experienced ECT as a re-abuse. Given that an estimated 50% of women in psychiatric hospitals have suffered sexual and/or physical abuse in childhood, (Williams and Watson 1994), and also given that ECT is most commonly used on women, this raises the disturbing possibility that a number of patients are, in effect, being re-abused in the name of treatment.

Thirdly, ECT may be leaving some people with a distrust of psychiatric services that undermines any future attempts to form therapeutic relationships. They may be both unhelped_ perhaps even in a worse state_ and at the same time harder to reach.

It is important to appreciate how powerless and vulnerable psychiatric patients perceive themselves to be in relation to the professionals. The apparent willingness to consent to ECT remarked upon by other researchers may merely be a case of desperation and compliance temporarily overcoming terror and reluctance. Similarly, what seems like a successful outcome may simply be conformity and a fear of confiding one's true feelings to professionals.

Powerlessness, control and conformity were themes that constantly recurred in the participants' responses. They came for help feeling confused, helpless and desperate. The help they were offered was experienced as a further loss of power and control which left them even less able to protest and assert themselves than before. None of

them had felt able to convey the strength of their feelings about ECT to mental health professionals, implying a possible hidden pool of distress that is unlikely to be picked up by hospital-based surveys; hence, perhaps, the disparity in reported rates of psychological trauma after ECT.

The most optimistic outcomes were for those who were ultimately able to direct their anger outwards, reverse their previous pattern of compliance and take control of their lives again. That they were able to do this despite rather than because of their treatment, and mainly with help from outside the psychiatric services, is a matter for profound concern.

What lessons can be learned about the use of ECT from this survey?

Standards for the administration of ECT are still very variable, as the most recent audit (Duffett and Lelliott, 1998) indicates. The participants in this study particularly objected to lack of discussion beforehand, seeing trolleys and equipment as they waited, overhearing people being given ECT, and distant or offhand staff attitudes. All this could be remedied relatively easily, in line with measures already suggested by other researchers, but at the risk of being seen as hypocrisy or window-dressing; it is the central fact of having electricity passed through your head that was so unacceptable to these participants. Not only did this carry powerful symbolic meanings, it was also seen as irrelevant and damaging. The superficial adoption of psychiatric terminology ("manic-depression", "psychotic" and so on) disguises the fact that participants believed they had broken down for reasons which a physical intervention obviously could not address. This mismatch of models, with the professionals offering biomedical explanations and treatments while the patients tend to prefer psychosocial ones, has been noted by other researchers (Rogers, Pilgrim and Lacey, 1993.)

Also problematic is the call for fuller information on both positive and negative effects. The issue of what counts as accurate information about ECT is still controversial, although these participants are in line with some critics in believing that it can cause long-term brain damage. (Breggin, 1991; Frank, 1990.) Whether or not they were correct in reporting that no one had discussed ECT adequately with them, it seems clear that they would consider many current factsheets (for example that produced by the Royal College of Psychiatrists 1997) a highly misleading portrayal of possible cognitive and psychological consequences.

Whatever the true figures about adverse reactions to ECT, professionals obviously need to be very alert to the expression of fear or distress and to take such feelings very seriously, since such patients are likely to find ECT not only unhelpful, but actually damaging. It should be emphasised that consent can be withdrawn at any time, even after signing the form. The most constructive overall response may be to heed the call for much more access to counselling and general emotional support as an alternative to ECT. This is consistent with other recent surveys of service user views on treatment, for example those by MIND (1993), and the Mental Health Foundation (1997).

For some, the present findings will raise the question of whether there is a place for

ECT at all. If up to a third of people will suffer a serious adverse psychological reaction to ECT, and if there is no way of identifying these individuals in advance, the ratio of costs to benefits may begin to seem unacceptably high. As always, more research is needed. However, this should not be an excuse for complacency about the experiences of those for whom the description of ECT as "a helpful treatment and not particularly frightening" is profoundly untrue.

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Self-Ratings of Memory Dysfunction: Different Findings in Depression and Amnesia*

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ABSTRACT

An 18-item self-rating test of memory functions was administered to two patient groups: seven patients with amnesia resulting from Korsakoff's syndrome and six other amnesic patients. These results were compared to results obtained previously for depressed psychiatric inpatients ($n=19$) and depressed inpatients prescribed electroconvulsive therapy (ECT) ($n=35$). The latter group was tested both before and 1 week after completion of the course of ECT. One pattern of memory self-ratings was reported by the two groups of depressed patients. These two groups reported an approximately equivalent level of impairment across all test items. A different pattern of memory self-ratings was reported by the two groups of amnesic patients and by the group tested after ECT. These patients reported considerably more impairment on some items than others, such that performance was not equivalent across test items. Finally, the patients with Korsakoff's syndrome underestimated their memory problems, reporting a less severe impairment than the other amnesic patients. The results show that the memory problems experienced in depression and in amnesia are distinguishable with self-assessment techniques. In addition, the similarity between memory self-ratings reported by patients after ECT and by amnesic patients supports the idea that memory complaints after ECT reflect primarily the experience of amnesia. Self-rating forms like the one described here may have useful application to many diagnostic groups where questions arise about the nature of reported memory problems.

Memory dysfunction is a common symptom of psychiatric and neurological illness, often occurring in the context of broader cognitive impairment. The symptoms of memory impairment can differ depending on the precipitating

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condition, e.g., whether memory impairment is caused by depression, dementia, or amnesia (Butters, 1984; Squire, 1986). Specialized neuropsychological tests have been successful to some extent in identifying different patterns of memory impairment and distinguishing among them (Cronholm & Ottosson, 1961; Sternberg & Jarvik, 1976; Weingartner, Grafman, Boutelle, Kaye, & Martin, 1983). For example, tests can distinguish the memory impairment exhibited prior to a course of bilateral electroconvulsive therapy (ECT), when patients are depressed, from the impairment exhibited shortly after ECT when patients are amnesic (Cronholm & Ottosson, 1961).

Assuming that patients themselves experience these two conditions differently, it should be possible to develop a self-rating instrument that discriminates between them, i.e., a test that separates the memory complaints of depression from the memory complaints associated with amnesia. Such a test might then be useful in evaluating other examples of memory complaints, such as those associated with head injury, pseudodementia, or normal aging.

Recently, we constructed an 18-item self-rating instrument that yielded a different response profile before and 1 week after a prescribed course of bilateral ECT (Squire, Wetzel, & Slater, 1979). Because the before-ECT response pattern was also found in a group of depressed psychiatric inpatients not prescribed ECT (Squire & Slater, 1983), this response pattern is probably typical of depressed patients and not limited to those particular patients who are about to receive ECT. Less is known about the after-ECT response profile. It could reflect some combination of the amnesia associated with ECT together with psychiatric illness. Alternatively, it could reflect primarily amnesia. To address this question, it would be useful to administer the self-rating form to neurological patients with amnesia.

Neuropsychological testing suggests that the amnesia following ECT resembles the amnesia associated with neurologic injury or disease (Squire & Shimamura, 1986). However, amnesic patients have seldom been studied with self-assessment techniques. In two studies, low correlations were reported for memory-impaired or elderly subjects between self-assessments of memory function and performance on memory tests (Sunderland, Harris, & Baddeley, 1983; Zelinski, Gilewski, & Thompson, 1980). Yet some amnesic patients have considerable insight into their condition (Kaushall, Zetin, & Squire, 1981; Shimamura & Squire, 1986).

The present study compared the memory self-ratings obtained previously from psychiatric patients (Squire et al., 1979; Squire & Slater, 1983) to the memory self-ratings reported by two new groups: six amnesic patients who had had either an anoxic or ischemic episode ($n=5$) or a penetrating brain injury (patient N.A.); and seven patients who were amnesic as the result of alcoholic Korsakoff's syndrome. Patients with anoxic-ischemic amnesia and patient N.A., but not patients with Korsakoff's syndrome, have good metamemory skills, e.g., they are accurate at predicting their own performance on memory tests (Shimamura & Squire, 1986). It was therefore expected that the first group

of amnesic patients should be able to make more accurate self-ratings than the patients with Korsakoff's syndrome.

METHODS

Subjects

Patients prescribed bilateral ECT ($n = 35$). All were inpatients at one of five hospitals in San Diego County (Squire et al., 1979), where they were prescribed ECT for relief of depressive illness. Patients with neurological disorder, schizophrenia, or depression secondary to alcoholism or drug-abuse were excluded from the study. Twenty-one of the 35 patients had not received ECT before, and none had received ECT during the past year. Twelve of the 14 patients who had previously received ECT had received one course of ECT from 1 to 16 years previously (mean = 8 years). The remaining two had received either two or three courses of ECT during the same period. Treatment was administered three times each week on alternate days following medication with atropine, methohexital sodium, and succinylcholine. Electrode placement was bitemporal. Thirty patients received their treatments with a Medcraft machine (sine wave, 130-170V for 0.6-1 sec.). The remaining five patients received their treatments with a Reiter - Cedak Model SOS, a machine which delivers a series of unidirectional brief pulses. The patients' psychiatrists made all decisions concerning the number of treatments, which averaged 11.1 for this group (range = 5-21 treatments). Table 1 presents additional information.

Depressed patients ($n = 19$). All were inpatients at one of the hospitals where ECT patients were tested (Squire & Slater, 1983).

Patients with Korsakoff's syndrome ($n = 7$). Extensive neuropsychological data for six of these seven patients and for alcoholic control subjects have been reported (Squire & Shimamura, 1986). The full-scale WAIS IQ score for the seven patients averaged 103.4 (range 91-114), and their average Wechsler Memory Scale (WMS) score was 81.3 (range 69-93). In normal subjects, the WMS score is roughly equal to WAIS IQ. For the patients, immediate and delayed recall (12 min) of a short prose passage averaged 4.3 and 0 segments, respectively (21 segments total). Copy and delayed recall (12 min) scores for the Rey-Osterrieth figure averaged 26.3 and 2.7, respectively (36 segments total). Paired associate learning of 10 unrelated noun-noun pairs on three successive trials averaged 0.4, 0.2, and 1.3. Free recall of 15 words averaged 3.0, 3.9, 4.4, 4.1, and 4.6 on five successive study/test trials (Rey Auditory Verbal Learning Test). For yes/no recognition of 15 old words and 15 new words, the average score on five successive study/test trials was 20.2, 24.4, 24.1, 25.5, and 26.4. The Dementia Rating Scale Score averaged 129.4 points out of a possible 144 points (Mattis, 1976). Most of the points were lost on the memory subportion and the initiation-perseveration subportion of the test (7.0 points on memory and 4.4 points on initiation-perseveration). Independent neurological examination and neuropsychological screening indicated that memory impairment was the only notable deficit of higher cortical functions. All patients could draw a cube and a house in perspective, and none had aphasia or apraxia.

Six additional amnesic patients. Extensive neuropsychological data have been reported for four of these six patients and for control subjects (Squire & Shimamura, 1986). Patient A.B. became amnesic in 1983 following a cardiac arrest with unconsciousness; patient G.D. became amnesic in 1983 following a hypotensive episode that occurred

episodes that occurred during a seizure; patients W.H. and M.G. became amnesic in 1986 following an ischemic event. The sixth patient was N.A., who has been severely amnesic for verbal material since 1960 when he sustained a stab wound to the brain with a miniature fencing foil (Kaushall et al., 1981; Teuber, Milner, & Vaughan, 1968). As a group, these six patients obtained a full scale WAIS score of 120.5 (range 104-129) and a WMS score of 93.7 (range 81-105). Immediate and delayed recall (12 min) of a short passage averaged 6.0 and 0 segments, respectively. Five of the patients, excepting N.A., averaged 28.8 and 4.4 for copy and delayed recall (12 min) of the Rey-Osterrieth figure. N.A., whose memory impairment is primarily for verbal material, scored 33 for his copy and 17 after a 12-min delay. For all six patients, paired-associate learning of 10 unrelated noun-noun pairs on three successive trials averaged 5.5, 6.5, 6.7, 6.8, and 6.3 on five successive study/test trials. For yes-no recognition of 15 old words and 15 new words, the average score on five successive study/test trials was 24.0, 25.1, 26.2, 26.5, and 27.8. The Dementia Rating Scale Score averaged 136.2 points out of 144. Most of the points were lost on the memory subportion of the test (5.5 out of 7.8 points). Memory impairment was the only detectable deficit of higher functions. All six patients could draw a cube and a house in perspective, and none had aphasia or apraxia.

Test and Procedures

Subjects responded to 18 items that asked them to rate their memory ability in several ways (Table 2). Ratings were made on a 9-point scale from -4 through 0 to +4. Each item asked subjects to judge their memory now, compared to an earlier indicated time period. Depressed patients, patients tested prior to bilateral ECT, and patients tested 1 week after bilateral ECT were asked to rate each item by comparing their current ability level to "before I began to feel bad and went to the hospital". The 13 amnesic patients were asked to compare their current ability level to "before my memory problems began".

Table 1

Demographic Data for Patient Groups

Groups	ECT	Depressed	Korsakoff	Anoxic-Ischemic	Case NA
Sex					
Male	8	6	5	4	1
Female	27	13	2	1	-
Age (years)					
Mean	41.3	44.4	55.4	53.8	48
SD	11.2	12.6	9.9	7.2	-
Range	25-64	23-62	43-70	46-64	-
Education (years)					
Mean	12.7	12.5	11.4	15.6	13
SD	2.1	1.6	1.62	3.3	-
Range	9-18	9-16	9-14	13-21	-

SD = Standard deviation.

Because of the relatively small number of amnesic patients, each patient was given the self-rating scale on two different occasions separated by an average interval of 70 days. The score for each patient was the average score obtained on each item. One of the amnesic patients, W.H., was available for only one testing occasion.

RESULTS

Figure 1 (top) shows the results obtained with the self-rating scale before ECT and 1 week after ECT ($n=35$). To display the data, the test items (1 through 18) were ordered according to the self-ratings obtained after ECT. Thus item 1 (to the far left in Figure 1) yielded the lowest score and item 18 yielded the highest

Table 2.

Self-Rating Scale of Memory Functions^a

1	My ability to search through my mind and recall names I know are there is	11
2	I think my relatives and acquaintances now judge my memory to be	1
3	My ability to hold in my memory things that I have learned is	8
4	My ability to recall things when I really try is	4
5	The tendency for a past memory to be 'on the tip of my tongue,' but not available to me is	13.5
6	My ability to remember the names and faces of people I meet is	5
7	My ability to know when the things I am paying attention to are going to stick in my memory is	2.5
8	My ability to remember things that have happened more than a year ago is	12
9	My ability now to remember what I read and what I watch on television is	6.5
10	My ability to make sense out of what people explain to me is	17.5
11	My ability to remember what I was doing after I have taken my mind off it for a few minutes is	2.5
12	My ability to pay attention to what goes on around me is	10
13	If I were asked about it a month from now, my ability to remember facts about this form I am filling out would be	6.5
14	My ability to recall things that happened a long time ago is	17.5
15	My ability to reach back in my memory and recall what happened a few minutes ago is	9
16	My ability to follow what people are saying is	13.5
17	My general alertness to things happening around me is	15
18	My ability to recall things that happened during my childhood is	16

^a For each item subjects were asked to judge their ability as it seemed now compared to an earlier, specified time period. Subjects used a 9-point scale, ranging from -4 (worse than ever before), through 0 (same as before), to +4 (better than ever before). The items are ordered according to the score obtained 1 week after ECT. Item 1 produced the lowest score (mean = -2.5), and item 18 the highest score (mean = -0.6). Items 5 and 6, and items 17 and 18 were tied. The column of numbers to the right shows how the items would have appeared if the ordering had been done according to the responses of the 6 non-Korsakoff amnesic patients.

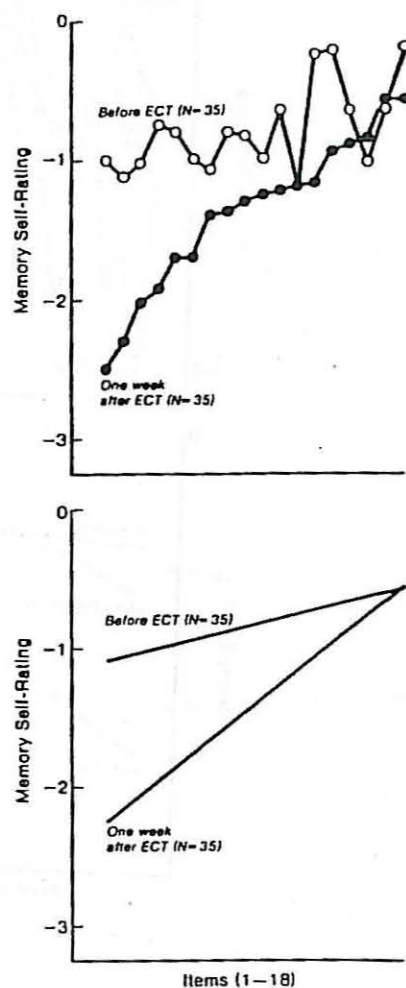


Figure 1 (top). Self-ratings of memory functions before and 1 week after bilateral ECT, as assessed by an 18-item test. (Bottom). The same data are represented as best-fitting lines across the scores for all 18 test items. The order of the items, from left to right, is shown in Table 2.

score. Next, best fitting lines were constructed through each set of self-ratings (Figure 1, bottom). The slopes and the 95% confidence limits for the slopes were as follows: before-ECT, slope = $.033 \pm .03$; after-ECT, slope = $.100 \pm .01$. An analysis of variance with tests for linear trends showed that memory complaints were greater overall after ECT than before ECT ($F[1,34] = 4.3, p < .05$) and that the linear trends were different ($F[1,578] = 22.5, p < .001$). Thus, after ECT memory impairment was experienced as both more severe and qualitatively different than before ECT.

Figure 2 shows the results for depressed patients, amnesic patients with Korsakoff's syndrome, and other amnesic (non-Korsakoff) patients. To permit comparison with the data for ECT patients, these data have been displayed just as in Figure 1, i.e., the order of the items is the same in the two figures. The top

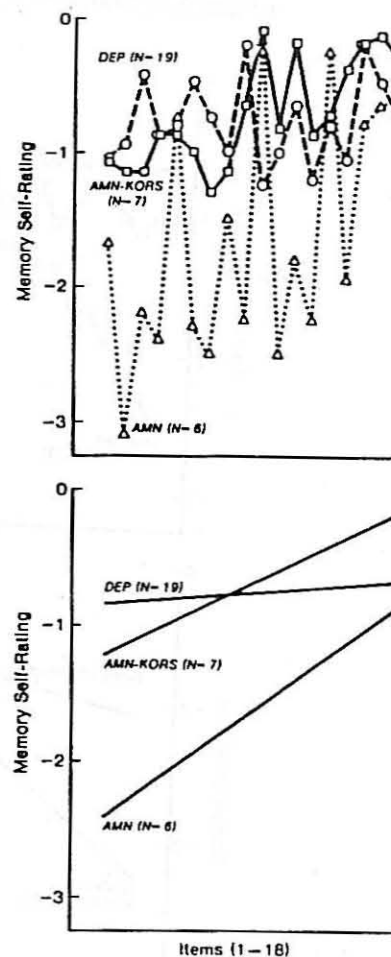


Figure 2 (top). Self-ratings of memory functions on an 18-item test, as reported by depressed patients (DEP), amnesic patients with Korsakoff's syndrome (AMN-KORS), and a group of non-Korsakoff amnesic patients (AMN). (Bottom). The same data are represented as best-fitting lines across the scores for all 18 test items. The order of the items, from left to right, is shown in Table 2.

- portion of Figure 2 shows the average scores for each item, as reported by the three groups. The lower portion of Figure 2 shows best fitting lines through each set of self-ratings. The slopes and 95% confidence limits for the slopes were as follows: depressed patients, slope = $.010 \pm .03$; Korsakoff patients, slope = $.060 \pm .02$; amnesic (non-Korsakoff) patients, slope = $.090 \pm .08$. These data can be summarized by stating that the pattern of memory problems reported by the depressed patients resembled those obtained before ECT and that the pattern of memory problems reported by the amnesic patients resembled those obtained after ECT. The patients with Korsakoff's syndrome, however, reported less severe memory impairment than did the other amnesic patients.

Separate comparisons between groups revealed no difference between depressed patients and before-ECT patients (for the between-groups comparison, which assessed the overall severity of memory complaint, $F[1.52 < 1.0, p > 0.11$; for the comparison of linear trends, which assessed the pattern of memory complaint, $F[1, 884] = 1.22, p > .1$). Similarly, the scores of the two amnesic groups resembled the scores of the after-ECT group (for the between-groups comparison of Korsakoff patients and after-ECT patients, $F[1, 40] = 1.51, p > 0.1$; for the between-groups comparison of non-Korsakoff amnesic patients and after-ECT patients, $F[1, 139] = .18, p > 0.1$; for comparison of linear trends, both $Fs < 2.4, ps > 0.1$).

In contrast, the two groups of amnesic patients differed from both the before-ECT patients from the depressed patients not prescribed ECT, especially with respect to the pattern, i.e., the slope, of the self-ratings. First, the depressed patients differed from the non-Korsakoff amnesic patients (for the between-groups comparison, $F[1, 23] = 5.7, p < .05$; for the comparison on linear trends, $F[1, 391] = 11.6, p < .001$). The depressed patients also differed from the Korsakoff patients in terms of the slope of the self-ratings (for comparison of linear trends, $F[1, 408] = 5.1, p < .05$). Second, the scores of patients tested before ECT differed from those of the non-Korsakoff amnesic patients (for comparison of linear trends, $F[1, 663] = 5.5, p < .05$; the between-groups comparison was short of significance, $F[1, 39] = 2.5, p = .12$). Similar comparisons between the before-ECT patients and the Korsakoff patients were not significant ($Fs < 1.4, ps > 0.1$).

The patients with Korsakoff's syndrome tended to report less severe memory impairment than the other six amnesic patients ($F[1, 11] = 4.1, p < .07$). Nevertheless, the pattern of complaints reported by these two groups was similar ($F[1, 187] = 1.6, p > 0.1$).

We considered that the pattern of memory complaints observed, i.e., the slope of the best-fitting line through the ordered scores from the 18 test items, might vary considerably depending on the method used to order the test item scores. This possibility seems unlikely for several reasons. First, the pattern of memory complaints observed before and after ECT did not change noticeably when the items were ordered according to the score obtained after ECT (as in Figure 1, this study) instead of according to the magnitude of the before ECT-after ECT difference score, as was done in a previous study (see Figure 1, Squire et al., 1979).

Second, the 18 items were also ordered according to how the non-Korsakoff amnesic patients responded (see right-most column in Table 2). Although there were some differences, this item order was rather similar to the order that resulted when the items were ranked according to the after-ECT scores ($r = .51, p < .05$). Moreover, when the results for all groups were compared using this new item order as a basis for constructing response profiles and best-fitting lines, the findings were similar to those just described. That is, depressed patients and before-ECT patients reported similar memory self-ratings (for

linear trends, $ps > 0.1$). In addition, the self-ratings reported by these two groups differed from those reported by the two amnesic groups (for four comparisons of linear trends, all $ps < .05$, except Korsakoff patients vs. before-ECT patients).

To determine the reliability of the self-rating responses made by the amnesic patients, we calculated the correlation between their responses on the two separate test occasions. Specifically, average group scores for each item were used to determine the correlation between the two sets of 18 items. For the amnesic (non-Korsakoff) patients, $r = .80, p < .001$; for the Korsakoff patients, $r = .37, p > 0.1$. This finding shows that the non-Korsakoff amnesic patients rated their memory consistently on both test occasions; however, the Korsakoff patients were not able to rate their memory in a consistent fashion. These patients did rate the overall severity of their memory impairment similarly on the two occasions ($F[1, 6] = 0.3, p > 0.1$); but the pattern of memory impairment was rated differently (for comparison of linear trends, $F[1, 16] = 12.8, p < .001$). In contrast, the non-Korsakoff amnesic patients were consistent across the two testing occasions, both with respect to the magnitude of their rated impairment ($F[1, 4] = 1.6, p > 0.1$) and with respect to the pattern of the impairment ($F[1, 68] = .09, p > 0.1$).

DISCUSSION

The amnesic patients reported an experience of memory dysfunction clearly different from that of depressed patients. It resembled instead the experience reported by psychiatric patients one week after a course of bilateral ECT. Amnesia is easily detectable one week after ECT (Cronholm & Bloomquist, 1959; Squire, 1984; Weeks, Freeman, & Kendell, 1980). Accordingly, it seems reasonable to suppose that the memory self-ratings obtained after ECT are attributable primarily to amnesia rather than to some other factor or combination of factors.

Amnesic patients with Korsakoff's syndrome reported a less severe memory impairment than did the six other amnesic patients. Moreover, the self-ratings of the patients with Korsakoff's syndrome did not contrast as sharply as those of the other amnesic patients with the self-ratings reported by depressed patients. These findings occurred despite the fact that, as assessed by quantitative tests of memory function, the patients with Korsakoff's syndrome were as severely impaired as the other amnesic patients (see Subjects section; also see Squire and Shimamura, 1986). For example, the patients with Korsakoff's syndrome recalled an average of 4.0 words out of 15 on each of five successive learning trials, and they recognized an average of 24.1 out of 30 words across five learning trials. The corresponding scores for the six (non-Korsakoff) amnesic patients were 6.3 (recall) and 25.8 (recognition).

The patients with Korsakoff's syndrome did not reliably report their own

memory abilities. However, despite the inconsistency in their responses across two test sessions, these patients did underestimate the severity of their impairment on both occasions. In contrast, other amnesic patients appeared capable of accurate and consistent memory self-ratings. This difference between amnesic groups has also been observed with other metamemory tests, given recently to four of the six (non-Korsakoff) amnesic patients in the present study and to six of the seven patients with Korsakoff's syndrome (Shimamura & Squire, 1986). The patients with Korsakoff's syndrome were not able to predict their performance on a subsequent memory test, but the other amnesic patients made accurate predictions.

The difficulty that patients with Korsakoff's syndrome exhibit in reporting their memory abilities is probably not due to diencephalic damage alone, because patient N.A. had good metamemory in the previous study and also reported his memory problems accurately in the present study. The findings for patients with Korsakoff's syndrome may be due to the more widespread neuropathology associated with this patient group, which includes the diencephalic region as well as frontal neocortex (Shimamura, Jernigan, & Squire, in press).

It is interesting to compare the items that elicited reports of memory dysfunction in the memory-impaired groups with the items that did not elicit reports of memory dysfunction. The six items that elicited the lowest average self-rating scores (and that reflected the most severe impairment) for the after-ECT group and the non-Korsakoff amnesic group were items 2, 3, 4, 6, 7 and 11 (Table 2); the six items that elicited the highest self-rating scores for these two patient groups were items 10, 12, 14, 16, 17, and 18. The former items asked about the ability to learn, retain, and recall, especially in the case of new material; and also about the judgment of others. The latter items asked about attention, concentration, immediate memory, and remote memory. Interestingly, these latter items, which were not endorsed by amnesic patients, were nevertheless endorsed by depressed patients about as readily as the other items on the test (see the before-ECT group in Figure 1 and the depressed group in Figure 2). It seems reasonable to suppose that the former items were asking about experiences likely to be associated with amnesia. Note that amnesia most severely affects new learning and memory for the recent past; whereas it typically spares immediate memory functions (including the ability to attend and concentrate), and it typically spares memory for the distant past. In contrast, the latter items ask about experiences likely to be associated with depression (e.g., impaired attention and concentration). Indeed, a sense of impaired attention and concentration might lead to an experience of impaired cognition, in general, and a tendency to endorse all items to a similar degree.

In summary, the findings show that self-rating instruments can distinguish between depression and amnesia, and they can identify those amnesic patients who underestimate their memory problems. This test might have useful application to other populations, where questions arise about the nature of

memory complaints or about the relationship between self-assessment and test performance.

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Neuropsychology and ECT: Past and Future Research Trends

Avraham Calev, D. Phil¹

Abstract

Past research focused on characterizing the cognitive deficits caused by ECT, understanding their causes, and defining ways of ameliorating the deficits. Future research includes the following recommendations:

In characterizing the deficits: more accurately defining the time course to recovery; finding out whether specific memory tasks and specific patients show long-lasting effects; and defining specific components of memory and non-memory deficits (e.g., associative memory, incidental everyday memory, inattention).

In understanding the causes: determining whether seizure activity in certain brain structures is associated with specific cognitive deficits; finding out in which ways electric dose, electrode placement, seizure duration, and seizure threshold interact in causing the deficits; evaluating the effects of mediating variables such as blood pressure rise; and assessing the influence of background variables such as age, sex, and brain abnormality.

In ameliorating the deficits: continuing the search for effective medication; defining ways of reducing the number of treatments (twice weekly ECT, caffeine or thyroxine modified treatment); and manipulating dose in relation to electrode placement.

Introduction

Research on the neuropsychology of electroconvulsive therapy (ECT) evolves around three central issues. These are describing the nature of the neuropsychological adverse effects, understanding the etiology of these effects, and ameliorating them.

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The Nature of the Neuropsychological Effects

It has been known for a long time that ECT adversely affects memory and other cognitive functions (see reviews by Abrams 1992; Calev et al. 1993b; Fink 1979; Price 1982; Sackeim 1992; Squire 1984, 1986). The main findings are summarized below, and needs for further research are stated.

Memory

Immediately after ECT, patients experience memory deficits. These deficits increase as a function of the number of treatments (Squire 1984, 1986), electric stimulus dose (above threshold) (Sackeim et al. 1991), and stimulus waveform [sine wave having more severe effects than brief pulse (Weiner et al. 1986)]. Both anterograde (post-ECT) and retrograde (pre-ECT) memories are affected. Anterograde amnesia is characterized by rapid forgetting of newly learned information. Retrograde amnesia is characterized by a temporal gradient, that is, better memory for the distant than the more recent past (mainly the last year or two) (Calev et al. 1993b; Squire 1984, 1986). Information acquired in the days preceding ECT may be permanently lost (Squire 1984, 1986). Right unilateral ECT spares verbal memory (Squire & Slater 1978). The memory deficits gradually improve, and most studies agree that no deficit is observable 3 to 6 months after ECT (Calev et al. 1993b). Years later, patients who had many ECT treatments during their lifetime do not appear to differ from controls who had no ECT (Devanand et al. 1991). ECT does not seem to appreciably affect procedural or other implicit memories (Squire et al. 1984, 1985) or semantic memory, except for retrieval (Calev et al. 1991b).

There are some questionable findings and specific needs for further investigation:

1. The rate of recovery of these functions has not been fully investigated. For example, with bilateral moderately suprathreshold ECT, Calev and colleagues (1991b) found no rapid forgetting at the 1-month followup. The level of acquisition (learning) was still low and resembled that observed during the pre-ECT depressive state. At the 6-month followup, there was further recovery and the level of acquisition of new information was better than that observed during pre-ECT depression. There are some unknowns that require further research. First,

rapid forgetting may be absent before the 1-month followup point, and the point of normalization still needs to be defined. Second, from the beginning of the second month onwards, encoding and acquisition of new information is better than observed during the depressive pre-ECT stage, and also than that observed 1 month after ECT. It is important to define the point at which this immediate memory performance normalizes. However, it is difficult to define the control group for people who had depression and recovered. The average population seems to be a good reference group, although pre-ECT depressive patients may differ from normals in their premorbid functioning, given the recent pathological brain findings on MRI (Coffey 1993). Third, with different doses and different methods of ECT administration [e.g., age dosing (Abrams 1992), and high dose unilateral or bilateral ECT (Sackeim et al. 1993)], these time points may vary and further work is needed for defining them.

2. There were some disturbing findings of persisting memory deficit 6 months after ECT. Weiner and associates (1986) and Weiner in this volume (1994) report a selective autobiographical memory deficit at the 6-month followup. Similarly, Weeks and colleagues (1980) reported that a subgroup of patients complained they have some cognitive deficits years after treatment. These were confirmed on tests, although this could have been due to factors other than ECT. Such findings, in particular the Weiner groups' results, need replication in view of the importance of defining the time course of deficits and the importance of reassuring and informing the public as to the possible long term effects of ECT.

3. The finding of permanent memory deficits for information acquired days before and during the hospitalization in which ECT was administered, needs substantiation. For example, Calev and associates (1991b) did not observe such a phenomenon. This might have been due to differences in tools and methods of study. A replication by other research groups and a better definition of the phenomenon is important, since this is a worrying qualifier of the deficits.

4. The finding of a temporal gradient in retrograde amnesia (i.e., better memory for the distant than the more recent past), largely comes from Squire's research group (e.g., Cohen & Squire 1981). Our data (Calev et al. 1989, 1991b) did not always confirm it. Squire's data indicate that it is most observable when using a multiple choice recognition remote memory testing format rather than a recall format. It is necessary to replicate these

findings to determine why some tests reveal it and others do not. Calev and co-workers (1991a) and Daniel and associates (1987) found a temporal gradient in the recovery of orientation items in the first hour after ECT; items learned closer to birth were recovered earlier. However, in both studies, this gradient could also be attributed to the degree of overlearning of the information. For example, one's name is learned earlier but also much better overlearned than the current year. The degree of overlearning or depth of encoding, rather than the time of acquisition, may therefore be the cause of the temporal gradient after ECT. The degree of overlearning does not necessarily correlate with the time of learning. Consequently, this may explain why the temporal gradient is not always observed after ECT. A study controlling for overlearning and other confounding factors, such as salience, is needed to define the nature of the temporal gradient in retrograde amnesia after ECT. Such a study could explain why certain pieces of information acquired days before ECT may be permanently lost after ECT: Are they lost because of a low level of overlearning or because of their time of acquisition?

5. There are findings showing that the memory deficit after ECT may have additional undefined characteristics. For example, Calev and colleagues (1991b) found that tasks requiring a high level of mnemonic organization (e.g., recall of unrelated paired associates) show not only rapid forgetting after ECT but also an immediate memory deficit (when compared with the pre-ECT depressive state), whereas tasks that do not require such organization show only rapid forgetting when compared with pre-ECT baseline (e.g., recognition of unrelated paired associates). This associative deficit after ECT needs replication and further investigation in order to better explain the nature of memory deficit caused by ECT. One must clarify whether this finding means, for example, that all effort-demanding tasks (even non-memory tasks) are disturbed by ECT more than simpler tasks. Another understudied area is everyday-life incidental memory after ECT. How and to what extent does ECT affect cognitive functioning in everyday life.

Disorientation

The acute disorientation period after ECT varies with the number of past treatments, specifically, with moderately suprathreshold and low dose ECT disorientation decreases (Calev et al. 1991a; Sackeim et al. 1986),

whereas with earlier methods of ECT administration employing stronger stimulation it increases (Daniel et al. 1987).

Acute disorientation also varies with stimulus waveform [sine wave ECT causes larger effects (hours) than brief pulse ECT (minutes) (Sackeim 1992)]; stimulus intensity above threshold (Sackeim et al. 1986, 1991); seizure duration (Calev et al. 1991a); electrode placement [unilateral ECT causes shorter disorientation than bilateral ECT (Sackeim 1992; Sackeim et al. 1993)]; and background variables such as age (Calev et al. 1991a). Finally, acute disorientation varies with the orientation of subject matter (orientation for *person* recovering before orientation for *place*, and orientation for *time* recovering last (see Daniel et al. 1987). One should note that most of these findings are rather new and have been investigated in only a few studies. Further substantiation of these findings is paramount.

Additionally, there are speculations as to whether disorientation after ECT is an amnesic phenomenon or is caused by other mechanisms such as difficulty in time and space perception, in calculation, or in other cognitive faculties that may be affected by ECT (Calev et al. 1993b; Daniel et al. 1987). It may be wise to study the temporal gradient after ECT (as stated above) and include an orientation component, while controlling for salience and overlearning. This might help to determine whether disorientation after ECT is an amnesic phenomenon, along the same continuum as the temporal gradient in retrograde amnesia, unlike disorientation caused by factors such as spatial and temporal dysfunction in some other conditions.

Except for post-ictal disorientation, patients show a minor degree of disorientation between treatments (Calev et al. 1991a; Daniel et al. 1987), which always increases as a function of the number of treatments, and reaches the proportions of an organic brain syndrome in some, mainly older, patients.

Other Cognitive Tasks

Little attention has been paid to non-memory cognitive effects of ECT (Abrams 1992; Fink 1979; Price 1982). A recent review of the literature at the State University of New York at Stony Brook revealed that this is probably due to the fact that most non-memory studies failed to find a deficit after ECT as compared with the pre-ECT state. However, it would be a mistake to compare pre-ECT to post-ECT performance because depression before ECT also causes such deficits. Tables

1, 2, and 3 clarify this point. Table 1 shows generally no deficit 1 day after ECT (as compared with pre-ECT baseline) when present-day stimulus strength is used.

TABLE 1. Early Subacute Period (7 to 72 Hours).

Area	Study	Effects of ECT
General Intelligence	Squire 1975	No change*
	Calev et al. 1991b	No change*
	Taylor et al. 1985	No change*
	Calev et al., in preparation	No change*
	Lawson et al. 1990	Performance IQ drops nonsignificantly
	McKenna & Pratt 1983	Improvement (on Digit Symbol Subtest of the Weis)
Language	Taylor et al. 1985	Results suggestive that verbal fluency** is adversely affected
	Lerer et al., in press	Verbal fluency** adversely affected
	Taylor & Abrams 1985	No change (on a variety of language tasks)*
	Jones et al. 1988	One of 20 tasks (word fluency, assessing retrieval from semantic memory) affected**
Perceptual and Visuo-Spatial Function	Taylor et al. 1985	No change*
	Taylor & Abrams 1985	No change*
	Calev et al. 1991b	No change*
Motor Function: Manual Dexterity	Taylor & Abrams 1985	No change*
Higher Cognitive and Frontal Function	Taylor et al. 1985	No change*
	Taylor & Abrams 1985	No change*
	Lawson et al. 1990	No change*

* No change can be attributed to ECT.

** Calev et al. 1993b suggest this may be a memory rather than a language problem.

Table 2 shows that this was not true when higher dose sine wave stimulus was used in earlier studies. Table 3 shows that at the late subacute period (i.e., 1 week to 6 months after ECT), cognitive function generally improves and is better than that of both the pre-ECT depressive state and the post-ECT state. This leads to the conclusion that non-memory cognitive function is affected by ECT, and therefore, needs to be addressed in future research. Patients should be informed of these effects of ECT.

There also are some non-memory cognitive findings of theoretical importance for neuropsychology as a field. For example, Sackeim and colleagues (1983, 1992) have repeatedly shown left-sided neglect in attentional cancellation tasks. This was observed with both unilateral and bilateral ECT. Williams and associates (1990) found the return of right ear advantage in dichotic listening tasks, which they found to be disrupted during pre-ECT depression. These findings suggest that depression may to some extent be a right hemisphere disease and that ECT acts to normalize the right hemisphere. These findings are, however, preliminary, and substantiation in future research is important.

The Mechanisms Causing Cognitive Effects After ECT

The physiological effects of ECT that bring about cognitive changes are not well understood. The idea that the temporal location of the electrodes affects the hippocampus and other medial temporal brain locations associated with memory (Squire 1984, 1986) has not

TABLE 2. Early Subacute Period (7 to 72 Hours): Early Studies.

Area	Study	Effects of ECT
Perceptual Tasks	Kahn et al. 1960	Adverse effect
Intelligence Tasks	Fink 1961	Adverse effect
Perceptual & Executive Function	McAndrew et al. 1967	Adverse effect
Language	Fink 1959	Adverse effect
Intelligence, Perceptual & Motor Tasks	Spreche 1963	No change*
Motor Speed, Intelligence & Memory	Scanlon & Matthas 1966	No change*

* No change can be attributed to ECT.

TABLE 3. Late Subacute Period (1 Week to 7 Months).

Area	Study	Effects of ECT
General Intelligence	Small et al. 1986	Improvement
	Malloy et al. 1982	Improvement
	Lawson et al. 1990	Improvement
	Weeks et al. 1980	No change*
Language	Weeks et al. 1980	No change*
Perceptual Function	Malloy et al. 1982	No change (trend for improvement)*
	Small et al. 1986	Improvement
	Williams et al. 1990	Improvement (normalizing auditory discrimination abnormality attributed to depression)
	O'Connor et al. 1984	Improvement (less field dependence relative to depression)
Motor Function: Manual Dexterity	Malloy et al. 1982	Improvement trend
	Small et al. 1986	Improvement trend
	Weeks et al. 1980	Improvement (i.e., normal performance)
Attention and Frontal Function	Malloy et al. 1982	Improvement
	Small et al. 1986	No change (Category test)
	Weeks et al. 1980	Improvement (in mental shifts tests but not in vigilance)
	Shellenberger et al. 1981	No change*

* No change can be attributed to ECT.

been well examined. The idea that pronounced memory effects occur because the hippocampus and other medial temporal brain locations more easily seize during ECT (Sackeim et al. 1991) has not been directly studied. Research evidence supporting these hypotheses is important. Multiple channel EEG and PET scanning during ECT, when they are possible, may be helpful in demonstrating the effects of electric stimuli in the brain.

In addition, the study of other factors related to the causation of deficits is important. It is agreed that

stimulus characteristics affect cognitive deficits after ECT. However, seizure duration and stimulus intensity rarely show correlation with the magnitude of the cognitive adverse effects, such as disorientation (Calev et al. 1991a, 1991b; Sackeim et al. 1987). Sackeim and co-workers (1991, 1993) presented evidence that it is the amount of electrical energy above seizure threshold (rather than energy or seizure duration) that is important in causing cognitive deficit. They found that patients' seizures have not substantially increased with bilateral high dose ECT (2.5 times the threshold), relative to moderately suprathreshold ECT (1.5 times the threshold), but their cognitive adverse effects on disorientation and memory did increase. Long seizures caused by unilateral ECT (which presumably did not generalize as much as bilateral seizures) produced lesser adverse cognitive effects, suggesting that electrode placement and dose interact in producing adverse cognitive effects.

Other factors may mediate cognitive adverse effects of ECT. Some studies gave evidence showing that blood pressure rise is associated with memory deficit. These findings (Hamilton et al. 1979; Zervas et al. 1993a) suggest that the deficit could be reduced if antihypertensive effects are achieved without interfering with other effects of ECT. Thus, the use of medication such as trimethaphan combined with ECT may prove important. These studies, however, are inconclusive in view of other evidence showing no effect of blood pressure rise on cognition (O'Donnell & Webb 1986; Taylor et al. 1985; Webb et al. 1990). Further research may be useful in understanding the mechanism by which ECT affects cognition.

Patient background characteristics also modify the cognitive adverse effects of ECT. Age is fairly consistently reported to be associated with the severity of post-ictal disorientation (Calev et al. 1991a, Sackeim et al. 1987) and there are recent findings suggesting that it may also be associated with memory dysfunction (Zervas et al. 1993b). Sex has sometimes been reported to affect cognitive adverse effects (Sackeim et al. 1987) although this finding was not always replicated (Calev et al. 1991a). Psychosis and low intelligence are sometimes found to adversely affect patients' deficits after ECT (Calev et al. 1991a), but these findings await replication.

In general, most of the reported effects of background variables on cognition are preliminary and await replications. A newly defined background variable that also needs study is the degree of pre-existing brain

abnormalities in pre-ECT depressed patients (Coffey 1993).

Attempts at Ameliorating Cognitive Adverse Effects of ECT

A principal interest to patients and doctors is minimizing the adverse cognitive effects of ECT. One attempted way of minimizing these effects was the use of medication which was reported to improve memory. Table 4 shows that this method was generally unsuccessful. Studies using ACTH, vasopressin, dexamethasone, naloxone, cytidine-5-diphosphate choline, ergoloid mesylates (a vasodilator), and nimodipine also report unsuccessful results. Additionally, a recently completed, large scale study (unpublished) showed primaracetam to be ineffective.

Two reports of improving memory after ECT stand out. Levine and colleagues (1987) reported a beneficial

TABLE 4. Attempts to Improve Memory With Medication After ECT.

Study	Outcome	Treatment
Small et al. 1977	Failed	ACTH4-10
D'Elia & Frederiksen 1980	Failed	ACTH4-10
Ayuzo-Gutierrez et al. 1982	Failed	Cytidine-5-diphosphate Choline
Lerer et al. 1983	Failed	Vasopressin
Horne et al. 1984	Failed	Dexamethasone
Ezzat et al. 1985	Succeeded	Piracetam
Frederiksen et al. 1985	Failed	ACTH4-10
Nasrallah et al. 1985, 1986	Failed	Naloxone
Levine et al. 1987	Succeeded	Physostigmine
Sachs et al. 1989	Failed	Ergoloid mesylates
Mattes et al. 1990	Failed	Vasopressin
Cohen & Swartz 1991	Failed	Nimodipine
Stern et al. 1991	Succeeded*	TRH (T3)
Unpublished 1993	Failed	Primaracetam
Calev et al. 1993a	Succeeded*	Caffeine

* Success could be due to fewer treatments.

effect of physostigmine and Ezzat and co-workers (1985) reported a beneficial effect of piracetam. These two findings require replication in a rigorous way in view of the negative results with similar drugs. The search for promising nootropic medication should not be abandoned in view of the massive failures in finding effective drugs.

Another way of improving memory after ECT is through the use of medication that may reduce the number of ECT treatments. One such attempt was by Stern and co-workers (1991), who used thyroid hormone, T3. The preliminary findings of this study suggest that T3 may increase antidepressant efficacy, reduce the number of treatments, and thus reduce cognitive adverse effects which are reported to increase as a function of the number of treatments in a series (Shapira et al. 1991; Squire 1984, 1986). A similar result was reported by Calev and associates (1993a) using caffeine pretreatment. The antidepressant effect was faster thus reducing the number of treatments and some of the cognitive adverse effects. A third way of reducing the deficits is by using a treatment schedule that minimizes the number of treatments and therefore allows greater spacing between treatments. The twice weekly ECT schedule, compared with the thrice weekly ECT schedule, was reported to reduce cognitive effects without affecting the final antidepressant effect assessed 1 week after 4 weeks of treatment (Lerer et al., in press; Shapira et al. 1991). The only cost was the speed of initial antidepressant response. Again, more research to further investigate and substantiate these findings is necessary.

A third way to get beneficial cognitive results is through the manipulation of stimulus variables: (1) Unilateral ECT, even at high doses appears to cause less adverse cognitive effects than bilateral ECT at lower doses (Sackeim et al. 1993). (2) New attempts at bifrontal electrode placement (see Lawson et al. 1990) have been useful in reducing cognitive effects with no significant cost to antidepressant efficacy. (3) It is established that brief pulse ECT produces less severe effects than sine wave ECT (Sackeim et al. 1991), and that sine wave produces significant effects even on non-memory cognitive tasks other than disorientation (see Tables 1-3). (4) Stimulus dosing is an important variable. Bilateral moderately supratherapeutic ECT appears less adverse than higher dose ECT, and more effective than low dose ECT in its antidepressant effects (Sackeim et al. 1991, 1993). The role of stimulus generalization and seizure duration is currently unclear.

Future research can better clarify the effects of the latter two variables. In addition, the search for a

therapeutically effective dosing-electrode-placement method should continue in order to reduce the cognitive adverse effects of ECT.

Finally an attempt to improve memory after ECT using behavioral techniques, such as decreased stimulation at the post-ictal recovery stage, has been reported (Suefeld et al. 1987). The results were disappointing because only subjective effects were reported. Yet, even if the effects are subjective, this technique may be worthwhile if a cost-benefit analysis suggests that the patient's well being significantly improves. However, since the evidence is unclear, such techniques should not be used clinically until a better understanding is achieved.

Conclusions

The purpose of this review is to identify important future research trends. The trends were identified in three major areas:

1. In characterizing the deficit:

(a) Defining the time course of memory deficits such as rapid forgetting and deficient encoding, in relation to different doses and electrode placements.

(b) Determining persistence of deficits: do autobiographical memory deficits persist longer than 6 months, and are all kinds of information acquired during the immediate pre-ECT period and the ECT course permanently lost? One also should determine whether there is a subgroup of patients that has prolonged memory deficits; this has not been observed thus far using sample averages.

(c) Defining certain components of the memory deficit after ECT: Is associative memory affected upon immediate testing? When is the temporal gradient in retrograde amnesia and on disorientation questionnaires replicable, and is it only replicable if overlearning is confounded with time of acquisition of the to-be-remembered information? It is also important to study the effects on memory function in everyday life including incidental learning.

(d) Replicating and finding out about the nature of non-memory cognitive deficits, such as left visual field neglect (inattention) and right ear advantage achieved after ECT.

2. In understanding the causation of cognitive effects:

(a) Defining physiologically the extent of seizure activity during ECT in different parts of the brain, in relation to brain sites associated with memory.

(b) Determining the effects of electrical dose and seizure duration on cognition while taking seizure threshold into account, and finding out whether there are mediating responses, such as blood pressure rise, that affect cognition.

(c) Determining which background characteristics (such as age, sex, intelligence, brain abnormality, severity of depression, and psychosis) affect the degree of cognitive effects of ECT.

3. In ameliorating the deficits:

(a) The search for effective medication that reduces cognitive adverse effects should continue and drugs that have shown ameliorating effects need replicating results.

(b) A reduction in the number of treatments needed can be a useful way of reducing the deficit after ECT. The findings showing reduced deficits when using thyroxine (T3), caffeine, or a twice weekly rather than a thrice weekly schedule need replication.

(c) Manipulating electric dose and electrode placement should continue in order to find a less cognitively adverse and more effective way of treatment at the same time.

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tients complained that the hyesthetic area became hyperesthetic. There was some improvement also in other types of superficial sensory disturbances. Slight disturbances often recovered completely with 2-3 applications and even severe ones improved. Paresthesias increased, however, in two cases, but subsided in 30 of 45 cases treated. Spontaneous pain in the involved limb subsided in 14 out of 18 cases, and pain provoked by movement diminished in 21 out of 24 cases treated. Deep sensation improved in 43% of 33 cases.

3) Spasticity and contractures were observed to have decreased after 5 to 10 "electrifications." Severe contractures of the involved limbs were seen to improve considerably in a few cases.

4) Speech difficulties improved in 56.9% of 109 cases. Complete recovery occurred in 5.5% (most of these cases were mild ones). Marked improvement occurred in 2.7% (majority of cases were severe).

5) Improvement was also noticed in the emotional and volitional sphere and in the other field where psychogenic symptoms predominated. Anxiety and tension decreased in 29 out of 35 cases; compulsive laughter and crying in 16 out of 18 cases; intellectual impairment disappeared in four cases, and memory defect in two out of four cases. Sleeplessness was reduced in 31 out of 40 cases and headache reduced in 26 out of 27 cases.

Evaluation and results of clinical investigation: No definite influence on the blood pressure, pulse and respiration rate was seen. No significant change was observed in the retinal blood vessels; ECG, venous pressure, capillary resistance, fibrinolysis activity and blood sugar levels were not altered, except for a tendency to eosinopenia after treatment. EEG studies on four patients showed some amplitude difference and a tendency to decrease in two cases. Seventeen ketosteroids and 17 hydroxycorticosteroids were found to increase slightly after the treatment,

with a decrease curve an hour later, and return to increase again in most of the cases. The RPF and GFR were found to decrease slightly at first, then increase gradually towards the end of the electrification. Elevation of blood pressure by the injection of adrenaline was suppressed in 8 of 12 cases, and salivation by pilocarpine was accelerated in 7 of 13 cases. Observations of the EMG records of the paralyzed muscles revealed that spike discharges of maximal voluntary contraction increased remarkably and also that discharges began to appear in paralyzed muscles (after the electrification) which weren't able to elicit any before. By studying the interval diagrams of EMG of paralyzed muscles during maximal effort, it was found that the frequency of the discharge increases and that the tendency of frequency adaptation diminishes.

Following treatment the clinical improvement remained constant in the majority of cases after a follow-up study made three months after the end of the study, with no evidence of side-effects.

Summary

Spinal electrostimulations with rectangular low frequency current was found to relieve many of the symptoms often seen in the hemiplegic patient. It was effective in approximately 80% of the motor and sensory disturbances, and in about 57% of the speech difficulties and also efficient for the improvement of mental symptoms, spasticity and other symptoms due to autonomic nervous disorders. Because of the results it was felt that this treatment should be given a trial on every hemiplegic patient.

The effect of cerebral electrification with this current on hemiplegias was found effective, particularly in the mental sphere, where there was spasticity and motor aphasia. The administration of adenosine triphosphate together with these electrifications was also recognized as valuable in the mitigation of these symptoms.

Retrograde Amnesia Provoked by Electroconvulsive Therapy

TAIEI MIURA, M.D., MORIZI OKADA, M.D.,
and MASAO OKAMOTO, M.D.

Impairment of memory as a complication in electroconvulsive therapy is quite common but cases of retrograde amnesia extending to years before the onset of mental illness are rare. K. Schroder¹ reported in 1940 cases of retrograde amnesia due to convulsive therapy with cardiazol. In the next year Fleischer² studied this problem in greater detail. In Japan Kurosawa reported

a case of retrograde amnesia due to prolonged insulin shock therapy.

In 1959 Suzuki and Yamashita studied similar complications due to electroshock in three schizophrenic patients, two females and one man. Evidence of retrograde amnesia was detected after remission of acute symptomatology. Extension of retrograde amnesia for a span of ten years or more was present in all cases, antedating onset of the schizophrenic illness. However, there was no sign of anterograde amnesia or confabulation

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Miyata, T. 1960

and during the period of clinical observation the retrograde amnesia did not show any tendencies toward regression.

Typical Case History

A similar event has recently been observed in a female patient treated in our institute with electroconvulsive therapy. The patient was a 20-year-old female student, born in a remote province. She had been adopted in early childhood and seemed to have shown a great deal of affection toward her adopted mother but mild hostility toward the adopted father. At high school she showed some interest in boys. Following graduation from high school she entered college in Tokyo. A disappointing love affair provoked a strong emotional reaction with some abnormality in behavior. Following a period of psychomotor agitation, she became abusive, assaultive, tense and overanxious. Sleep and nutrition patterns were disturbed. Electroconvulsive therapy was given (four treatments) together with drug therapy (chlorpromazine). Because of insurgent suicidal ideas, she was admitted on February 27th, 1960 to Sankei Hospital with a diagnosis of psychogenic reaction.

At date of hospitalization she was in a state of psychomotor agitation with evidence of depressive signs and mild disorientation. No hallucinations or disturbance of self consciousness were noticed. Electroconvulsive therapy was administered together with chlorpromazine. Improved after a few days, on March 9, 1960 a Rorschach test was given. There was no evidence of definite psychotic signs in the test, only disturbance in the emotional sphere with conflicts especially of libidinal nature.

The patient became worse after March 10th with a relapse into a catatonic state accentuated by negativistic behavior, delusions and visual and auditory hallucinations. This mental condition lasted for about two months in spite of ECT and drug therapies. By the end of April the patient was considered recovered clinically and in normal contact with the environment. However she complained of loss of memory for past events. This loss of memory extended to a period of two years before the onset of her illness.

At present she evidences no anxiety or preoccupation. She is calm, well balanced and determined to recover her memory. She visits places which should be familiar, scans albums of pictures of past events and repeats conversations just heard.

This patient had already received four ECT's previous to her admission to our hospital. Seven more treatments were given while hospitalized, making a total of 11 treatments. The retrograde amnesia is of some extent, going back to a period two years before the onset of her present illness. The only residues left intact at the present are the names of her school, her mother and one of her teachers. She has no sense of chronological sequence. The most intensive loss of memory for events seems to center in the social sphere and in the college area.

Comment

Since this has been considered a case of retrograde amnesia (all tests for organic impairment or deterioration were negative) the question at

present is: is this retrograde amnesia provoked by electroconvulsive therapy? Was the number of ECT's (11) given sufficient to produce such a disturbance?

In the three cases of Suzuki and Yamashita many more ECT's were given—76, 58 and 25 treatments respectively were administered. If the damage to memory by ECT is concomitant with and contingent upon the number of treatments received, it is unlikely that only 11 ECT's provoked such an extensive degree of retrograde amnesia. It can reasonably be assumed then that the number of ECT's is not the responsible factor.

Does the nature of mental illness treated with ECT tend to produce the retrograde amnesia? Is the psychogenic reaction responsible for the problem in this case. Although acute schizophrenic process was originally suspected, evolution of the clinical symptomatology and the personality structure and behavior, following clinical recovery, indicated a reactive psychotic episode.

Instances of psychogenic amnesia are common but in our present case the retrograde amnesia was noticed after recovery without any evidence of organic damage. This supports the conclusion that this case is one of retrograde amnesia provoked by ECT in spite of the fact that only 11 ECT's were given.

Summary

In this paper the case of a 20-year-old female student with clinical evidence of a reactive confusional state is discussed. After recovery from her psychic disorder which lasted about two and a half months, evidence of retrograde amnesia extending to a span of two years was noticed. Treatment had consisted of ECT associated with drug therapy. The total number of ECT's was only 11. However, because of the lack of evidence of any other etiological factor, this amnesia was considered an aftermath of ECT.

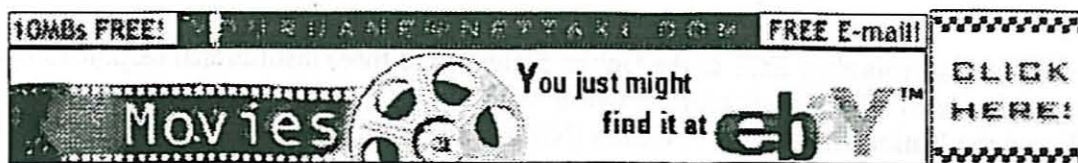
A Note on the Meeting

During July and August, 1960, a representative group of the Eastern Psychiatric Research Association was on a World Tour visiting Tokyo, Hong Kong, Bangkok, New Delhi, and Jerusalem, participating with the local neuropsychiatric societies in scientific meetings.

The first meeting on July 7 and 8 was held in Tokyo with the Kanto Neuropsychiatric Association and the Japan Mental Hospital Association. At that meeting, ten papers were read; five by members of the Eastern Psychiatric Research Association and five by local neuropsychiatrists.

The meeting was held in the spacious Tojo Kaikan Hall under the joint chairmanship of Professor Haruo Akimoto, Tokyo, and Professor William L.

(Continued on page 652.)



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Electroshock: scientific, ethical, and political issues⁽¹⁾

Peter R. Breggin⁽²⁾

Abstract. Electroconvulsive treatment (ECT) is increasingly used in North America and there are attempts to promote its further use world-wide. However, most controlled studies of efficacy in depression indicate that the treatment is no better than placebo with no positive effect on the rate of suicide.

ECT is closed-head electrical injury, typically producing a delirium with global mental dysfunction (an acute organic brain syndrome). Significant irreversible effects from ECT are demonstrated by many studies, including: (1) Inventories of autobiographic and current events memories before and after ECT; (2) Retrospective subjective observations on memory; (3) Autopsy studies of animals and some of humans. ECT causes severe and irreversible brain neuropathology, including cell death. It can wipe out vast amounts of retrograde memory while producing permanent cognitive dysfunction.

Contemporary ECT is more dangerous since the current doses are larger than those employed in earlier clinical and research studies. Elderly women, an especially vulnerable group, are becoming the most common target of ECT. Because of the lopsided risk/benefit ratio, because it is fundamentally traumatic in nature, because so many of the patients are vulnerable and unable to protect themselves, and because advocates of ECT fail to provide informed consent to patients - ECT should be banned.

Introduction

The use of electroconvulsive treatment (ECT), also called electroshock treatment (EST), has been escalating in the United States and Canada. Europe has not yet experienced a significant increase in the usage of ECT or in the controversy surrounding it. However, ECT seems to be used in most European psychiatric centers. With the growing emphasis on biological approaches in psychiatry, as well as the emphasis on cost efficiency, the North American trend will probably begin to take hold in Europe in the near future. In the meanwhile, ECT advocates are making an international effort to encourage the treatment throughout the world. For example, the First European Symposium on ECT was held in Austria in March 1992 and drew representatives from 13 European countries, as well as Russia, Israel, Canada, and the United States [107]. A team of three Americans - Max Fink, Harold Sackeim, and Richard Weiner - made a special presentation to the meeting. Their efforts are central to the current promotional campaign for ECT and their publications will be cited throughout this paper.

The controversy surrounding ECT in the United States led to three institutional responses that have affected the future of ECT throughout the world. First, in 1985 the National Institute of Mental Health (NIMH) and the National Institutes of Health (NIH) held a joint Consensus Conference aimed at establishing some agreement among mainstream professionals about the status of ECT. Second, in 1990 the Food and Drug Administration (FDA) decided for the first time to review the safety of ECT machines. Third, still in 1990, the American Psychiatric Association (APA) issued a lengthy report aimed at convincing the FDA not to require the testing of ECT machines. The APA report was successful in this regard and became the basis for the FDA's final report. The APA's report was also aimed at stifling controversy and protecting psychiatrists from lawsuits being brought by patients claiming brain injury from ECT. The conclusions of the Consensus Conference [63], the Food and Drug Administration [89], and the American Psychiatric Association [8] will be referred to throughout this analysis, often to compare their conclusions to the actual scientific data.

The ECT controversy has also been addressed by various agencies and bodies in Canada [74,128,142] and in England [150], usually in less detail and with reliance on opinions generated in the United States.

Current ECT usage in America and Europe

Nowadays ECT is most commonly recommended for major depression. Some doctors recommend it when other approaches have failed but others quickly resort to it as a treatment of choice. On occasion the treatment is also prescribed for other disorders, especially acute mania.

ECT was originated in Italy in 1938 by Bini and Cerletti who observed the effects of electric current in rendering slaughter house pigs into a state of unconsciousness. That the electrical shocks did not actually kill the pigs led the doctors to try it on human beings [3, p.6; 57]. The first human subject understandably feared that he was indeed about to be slaughtered. When the first shock did not render him unconscious, he beseeched the doctors, "Not again, it's murderous!" [3, p.6]. Cerletti himself [57] translated the victim's plea as "Not another one! It's deadly!"

In the United States, and probably elsewhere, the use of ECT tends to vary from institution to institution. At Johns Hopkins, for example, a biologically oriented psychiatric center, 20% of the inpatients may be on a regimen of ECT at any one time [182, p.9]. Many other hospitals in the US do not even offer ECT.

Probably more than 100,000 patients a year in the United States are electroshocked. The majority are women and many are elderly. In California, for example, two thirds of ECT patients are reported to be women, more than half of whom are 65 or older [165]. Data (1989-1993) from Vermont concerning ECT showed that 77% of ECT patients were female [168]. For all sexes, 58% were at least 65 years old and 20% were at least 80 years old. During this time, one Vermont hospital, Hitchcock Psychiatric, electroshocked 35 women and one man who were 80 and older. Overall, the hospital electroshocked 112 women and 26 men during those 5 years.

Pippard [142] commented "The use of ECT in England has shown a more or less steady decline for many years...." He surveyed ECT in all 35 National Health Service hospitals and five private clinics in the North East Thames and East Anglian Regions. He found that many of the hospitals used older machines and operated them according to the doctors' personal habits rather than "rational strategy" in regard to stimulus settings and other treatment variables. (Wise [183] recently found that 70% of ECT machines in

Britain and Wales remain below standard) Pippard discovered that ECT usage had fallen 55% in North East Thames Region since 1979 while it risen by 20% in the East Anglian region.

Pippard found a wide variation in usage from hospital to hospital, and district to district. In the County of Suffolk in East Anglia "In the year to March 1990, 3580 applications of ECT were given, a rate of 6.50 per 1000 of population. In East Suffolk the rate was 8.32 per 1000." In one of the inner London health districts, few patients other than the elderly received ECT. The overall rate was 0.68/1000 population.

While ECT has been slightly on the decline in Great Britain, successful efforts to escalate its use in the United States are likely to spread abroad. A review by Allan Scott [156], consulting psychiatrist at the Royal Edinburgh Hospital, draws heavily on the American experience and recommends, "Electroconvulsive therapy (ECT) is an effective and important treatment for severe depressive illness and for other depressive illnesses that have not responded to drug treatment." Except to dismiss brain damage from ECT, Scott does not mention any adverse effects, even memory loss. A.G. Hay and Scott [109], in part to counter this author's concerns about ECT-induced brain damage (the British publication of Breggin [39]), presented a single case of a woman who had received a total of 125 treatments over several years. The follow-up evaluation, which showed no mental decline, involved an IQ test and the Clifton Assessment Procedure for the Elderly. The evaluation was conducted by one of the co-authors. This single-case clinical report bears more on the rising enthusiasm for ECT than upon ECT's supposed safety.

Canadian authorities have not published data concerning the use of ECT. However, in reply to inquiries from Don Weitz [104,181], some data has been released. Weitz obtained the estimates for ECT administered in Ontario's general and community psychiatric facilities, and provincial psychiatric hospitals. Outpatient ECT was not included. During the year 1994-1995, 12,865 individual ECT treatments were administered to approximately 1,800-2,000 patients. Payments to all physicians in Ontario in general and community hospitals (not provincial psychiatry hospitals) for the year 1993-1994 showed that almost twice as many women as men received ECT [136]. Women received 6,221 ECTs and men received 3,236. Fifteen youngsters age 15-19 were treated with ECT.

With advancing age, there was a tendency for women to become increasingly overrepresented. The figures for the numbers of individual ECT treatments for women and men in each age group were as follows: age 60-64, 352 women, 342 men; age 65-69, 632 women, 240 men; age 70-74, 655 women, 430 men; 75-79, 592 women, 179 men; 80-84, 318 women, 97 men; 85 and older, 102 women, 94 men.

Stromgren [167] compared electroconvulsive therapy usage in Nordic countries - Sweden, Norway, Denmark, Finland, and Iceland - in 1977 and in 1987. The surveys were sent to departments of psychiatry in each country. The percentage of departments using ECT in 1987 in order of frequency were: Sweden (98%), Denmark (97%), Norway (82%), Iceland (67%), and Finland (57%). Departments that were unlikely to use ECT - child and adolescent, forensic, and drug addiction services - were excluded from the survey.

The number of units using ECT in Nordic countries was unchanged between 1977 and 1987 but there was a slight decrease in the absolute number of treatments given. This small decline was variously attributed to the decreasing numbers of beds, treatment by non-medical professionals, and the increasing use of psychopharmacology.

In the most commonly used diagnostic category in the Nordic countries, endogenous depression, all but 4 of 216 departments used ECT. However, the frequency of use had declined. In 1977, 22% of

departments used ECT frequently (more than 25%) for endogenous depression, but in 1987 only 15% used it frequently. Overall, the report found that "ECT is still regarded as being an important useful treatment" and that during the 1980s, its value "has become obvious to an increasing number of psychiatrists in the Nordic countries".

A survey of 20 general hospitals with psychiatric units and psychiatric hospitals in Barcelona, Spain in 1993 found that 12 of 20 (60%) practiced ECT [24]. Reports from around Europe suggest at least some interest in ECT since the early 1980s, including Belgium [163,164], Germany [73], Poland [58], as well as Israel [49].

In addition to the US, England, and Canada, ECT has generated considerable controversy in Ireland [158] and especially in Australia [16-21,31,120,139,140]. Writing in the Australian and New Zealand Journal of Psychiatry, Durham [70] laments the criticism of ECT, as well as a "distinct prejudice" against the treatment manifested in recent legislation. The controversy surrounding ECT will be addressed in more detail later in this paper.

1. Efficacy studies

1.1. *Is There any basis for the claims?*

Rifkin [149] noted that the claim is frequently made that ECT is more effective and works more rapidly than drugs in the treatment of depression. He located nine controlled studies comparing the two treatments, but they were badly flawed. He could find no conclusive evidence that ECT was better than antidepressant treatment.

Crow and Johnstone [64], in a review of controlled studies of ECT efficacy, found that both ECT and sham ECT were associated with "substantial improvements" and that there was little or no difference between the two. Crow and Johnstone concluded, "Whether electrically induced convulsions exert therapeutic effects in certain types of depression that cannot be achieved by other means has yet to be clearly established" (p.27).

Crow and Johnstone's critical review, which was presented at the largest conference of ECT advocates in recent years, is not cited in either the APA or FDA reports on ECT. Instead, the APA task force's proposal for a "Sample Patient Information Sheet" declares that "ECT is an extremely effective form of treatment" [9, p.160]

At the Consensus Conference on ECT [63], critics and advocates of ECT debated the issue of efficacy. The advocates were unable to come forth with a single controlled study showing that ECT had a positive effect beyond 4 weeks. Many studies showed no effect, and in the positive studies, the improvements were not dramatic. That ECT had no positive effect after 4 weeks confirms the brain-disabling principle (see ahead), since 4 weeks is the approximate time for significant recovery from the most obvious mind-numbing or euphoric effects of the ECT-induced acute organic brain syndrome.

The Consensus Conference panel stated in its report that ECT had no documented positive effect beyond 4 weeks. This is, of course, critical in weighing the risk/benefit ratio.

1.2. Does ECT reduce the risk of suicide?

ECT is frequently justified as treatment of last resort in cases at high risk for suicide. Sackeim [153], for example, claims "When confronted with a psychiatric or medical emergency - for instance, the acute risk of suicide - ECT can save lives" (p.39).

Despite the claims of advocates, research uniformly shows that ECT has no beneficial effect on the suicide rate. In a misleading fashion, the negative studies are cited by the task force report, the FDA report, and others as showing a positive effect. For example, a retrospective study by Avery and Winokur [10] found no improvement in the suicide rate compared to matched controls who had no electroshock treatment: "In the present study, treatment was not shown to affect the suicide rate" (p. 1033). Yet it is presented in the 1990 task force report as supporting the position that ECT results in "a lower incidence of suicide" (p.53). The task force also mentions three other studies as supporting a beneficial effect on suicide. Two of the studies [11,135] specifically found no such beneficial effect. The third [129] did not even deal with suicide.

In two other retrospective studies of relatively large populations of ECT patients and matched controls, ECT had no effect on the suicide rate [13,28]. Overall, there is little or nothing in the literature to suggest that ECT ameliorates suicide, whereas a significant body of literature confirms that it does not. My own clinical experience indicates that ECT increases the suicide risk for many patients. It is well known, for example, that Ernest Hemingway attributed his suicide to despair over ECT ruining his memory and rendering him unable to write [112, p.308].

As they attempt to recover from ECT, patients frequently find that their prior emotional problems have now been complicated by ECT-induced brain damage and dysfunction that will not go away. If their doctors tell them that ECT never causes any permanent difficulties, they become further confused and isolated, creating conditions for suicide.

2. Acute brain dysfunction caused by ECT

2.1. The production of delirium (acute organic brain syndrome)

After one or more treatments, ECT routinely produces delirium or an acute organic brain syndrome. Richard Abrams [3], although an advocate of ECT, has observed that:

... a patient recovering consciousness from ECT might understandably exhibit multiform abnormalities of all aspects of thinking, feeling, and behaving, including disturbed memory, impaired comprehension, automatic movements, a dazed facial expression, and motor restlessness (p.214).

Abrams' accurate description, including the "dazed facial expression", would indicate even to a layperson that the patient has suffered a severe head trauma. The existence of "multiform abnormalities of all aspects of thinking, feeling, and behaving" should raise warning flags about the potential for complete recovery. It should also remind us that not only memory but all mental processes are severely disrupted. The severity of the trauma should signal that it's dangerous to repeat this procedure again and again with the inevitable deterioration of the patient's condition. Finally, in trying to ascertain how ECT "works", it

should direct us, first and foremost, to suspect the traumatic impact on the brain rather than to speculate about the correction of some subtle, undetected biochemical imbalance. This is a treatment that creates abnormalities rather than correcting them.

The acute reaction to routine ECT often reaches the proportions of a neurological catastrophe. Max Fink [82] wrote of ECT:

A more prominent neurological sequel to seizures is the change in mental state and the development of an organic mental syndrome. Although there is a relationship between the number and frequency of seizures and the change in sensorium, an organic psychosis may occur with few treatments (4 citations). The syndrome may include disorientation, amnesia, agnosia, confabulation, aphasia, apraxia, and delirium, the latter being seen principally as the postseizure emergence of delirium (3 citations) (pg. 131).

Fink's description of severe neurological dilapidation amplifies all the issues discussed in regard to Abrams' summary of ECT effects. It would seem extremely unlikely to find a complete recovery in most patients after such a traumatic assault on the brain.

At times, patients are so neurologically impaired following ECT that they will remain prone and apathetic for days at a time, sometimes incontinent of urine and feces, and unable to communicate or to carry out routine self-care. On occasion, the patient's neurological dilapidation from routine ECT will reduce the person to curling up in a fetal position for many hours. In malpractice suits in which I have been a medical expert for plaintiffs, psychiatrists for the defense have claimed that this kind of neurological collapse is normal and harmless following ECT.

A review of the literature by Calev, Gaudino, Squires, Zervas, and Fink [86, p.510] confirms that ECT can acutely disrupt not only memory but "perceptual, language and other cognitive functions", especially if the stimulus intensity is relatively high.

An apparently rare complication is the production of status epilepticus. Scott and Riddle [157] suggest that it may be more frequent than usually estimated because it can occur without obvious motor manifestations following ECT.

A team led by Christina Sobin [160] recorded variations in "orientation recovery" after ECT. The dose of electricity varied from amounts necessary to cause a convulsion (low-dose) to suprathreshold doses (high dose). Recovery after low-dose bilateral ECT (40.0 min recovery time) and after high-dose bilateral ECT (37.2 min) were essentially the same. Recovery from high-dose right unilateral (19.2 min) was much shorter than for either bilateral group and low-dose right unilateral (11.1 min) was even shorter.

Retrograde amnesia as measured by the recovery of autobiographic memories was also worse following bilateral ECT. Two months after one course of ECT, "Longer duration of acute disorientation was also associated with greater persistent retrograde amnesia" (pg. 198). The authors conclude that both the initial disorientation and retrograde amnesia are "overlapping phenomena" -- a function of the same ECT-induced brain dysfunction.

Given that ECT routinely produces acute, global brain dysfunction - and that this dysfunction is obviously associated with persisting retrograde amnesia - there can be no real disagreement about the existence of damaging effects. The only legitimate question is: "How complete is recovery from the initial trauma?"

2.2. ECT as closed-head electrical injury

For more than a decade, neurologists have recognized that relatively minor head trauma - without the delirium, loss of consciousness, and seizures associated with ECT -- frequently produces chronic mental dysfunction and personality deterioration [25]. If a woman came to an emergency room in a confusional state from an accidental electrical shock to the head, perhaps from a short circuit in her kitchen, she would be treated as an acute medical emergency. If the electrical trauma had caused a convulsion, she might be placed on anticonvulsants to prevent a recurrence of seizures. If she developed a headache, stiff neck, and nausea - a triad of symptoms typical of post-ECT patients - she would probably be admitted for observation to the intensive care unit. Yet ECT delivers the same electrical closed-head injury, repeated several times a week, as a means of improving mental function. ECT is electrically induced closed-head injury.

Interestingly, the results of lightning injuries are basically similar to those of ECT and other forms of electrical injury to the head [146]. Obvious impairments of language or consciousness are rare, but "impairments of attention, concentration, verbal memory, and new learning are very frequently identified" (p.279).

The symptoms of mild to severe closed-head injury are listed in detail by J.M. Fisher [87]. They include impairment of every area of mental, emotional, and behavioral function, confirming the multiple adverse effects of ECT on the mind and brain. McClelland et al. [130] describe the postconcussive syndrome in terms of the following:

[The] emergence and variable persistence of a cluster of symptoms following mild head injury. Common to most descriptions are somatic symptoms (headache, dizziness, fatigability) accompanied by psychological symptoms (memory and concentration difficulties, irritability, emotional lability, depression and anxiety).

Between one third and one half experience this symptom cluster over the first few weeks and a "substantial minority" continue to experience it for months or a year or more.

Head-injury victims, including post-ECT patients, frequently develop an organic personality syndrome with shallow affect, poor judgment, irritability, and impulsivity. They seem "changed" or "different" to people around them, much as lobotomy patients often seem to their families. Sometimes they become slightly clumsy, moving awkwardly or dropping things. Often they have "lapses" where they cannot think or cannot voice their thoughts. Sometimes their handwriting deteriorates. Headaches frequently begin with the traumatic treatment and may recur indefinitely.

Many post-ECT patients suffer from irreversible generalized mental dysfunction with apathy, deterioration of social skills, trouble focusing attention, and difficulties in remembering new things. I have evaluated a number who have suffered from dementia, confirmed by neuropsychological testing. Several have developed partial complex seizures or psychomotor epilepsy, permanently abnormal EEGs, and atrophy as measured by brain scans. Many have been deprived of the experience of years of their lives, their professional careers, and their mental ability following ECT [32,33,35,38,39,45].

3. Retrograde amnesia caused by ECT

Memory deficits, retrograde and anterograde, are among the most common early signs of traumatic brain damage, and are seen in virtually all cases of ECT. The controversy surrounds the severity and persistence of these deficits. But neurological experience confirms that patients frequently fail to recover from much less traumatic injury to the brain than that inflicted by ECT.

3.1. Patient self-reports of memory dysfunction

The APA task force report, like the FDA report, disregards all of the relevant research on memory loss, except for Freeman and Kendell's 1986 study, which the task force mentions and then grossly misrepresents. That study asked patients to assess their memory function 6-18 months after ECT. The authors themselves remark that the study was biased toward a low reporting of memory dysfunction because the patients were interviewed by the same doctor who had treated them. Nonetheless, 74% mentioned "memory impairment" as a continuing problem, and "a striking 30% felt that their memory had been permanently affected" ([97]; see also [96] for similar data). In defiance of the facts, the 1990 APA task force cites Freeman and Kendell as indicating "a small minority of patients, however, report persistent deficits".

Squire and Slater's 1983 study [162], also omitted by the APA task force, found that 7 months after the last ECT treatment, patients report an average loss of memory spanning 27 months. Squire, in an oral communication to me at the June 1985 Consensus Conference on ECT, explained that one patient lost the recollection of 10 years of her life. He told me that he felt it was not necessary to report this in his actual publication.

The Consensus Conference on ECT [63] used Squire and Slater's results to conclude that "on average, patients endure memory loss extending from 6 months prior to the treatment to 3 months afterward". These data, while serious enough in themselves, are misleading. The results reported at 7 months following treatment, indicating an average of 27 months of lost memories, are more likely to be accurate. When damage has not healed after two or three months, the brain is not likely to make substantial progress in regaining lost memories. With the passage of more time, there's little likelihood of increased improvement, but much likelihood of a growing tendency to deny the losses.

3.2. Early studies of autobiographic memory loss

Controlled studies by Janis, carried out at Yale University, showed extensive, permanent loss of important personal memories and life history following routine ECT. Janis [115-117] interviewed 19 patients before and after routine ECT, and 11 control patients with similar diagnoses in the same hospitals. The results 1 month post-ECT were striking: every post-ECT patient had significant memory losses. Many were unable to recall 10 to 20 life experiences "which had been available to recall prior to electroshock treatment".

Janis [116] followed up five of the patients at 2 to 3 months later. Most of the lost memories remained obliterated. A later unpublished follow-up by Janis showed that "Some memory impairments persisted for at least one year following the last treatment" (oral communication from Janis to Davies et al. [66]).

Janis [115] confirmed the importance of denial and anosognosia, especially the reality that post-ECT

patients tend to minimize or even confabulate to cover up their memory losses. One patient, for example, in his pre-ECT interview reported that he had been unable to work for several months prior to coming to the hospital. The historical facts were confirmed by the family. But after 12 ECTs, he was unable to recall the period of unemployment. Instead, he confabulated, claiming that he worked right up to his hospitalization. As Janis confirmed, patients often do not complain spontaneously to doctors about their memory loss; they tend to deny it.

The 1990 APA task force report, as well as the FDA report, makes no mention of the Janis studies. Indeed, over the years his work has been repeatedly ignored or misrepresented by ECT advocates. Important reviews commonly read during my psychiatric training actually cited Janis as evidence that ECT did not harm memory (e.g., [12], p.205); for a detailed analysis of distortions in the early pro-ECT literature, see Breggin [32]).

3.3. Recent studies of autobiographic memory loss

In 1986, Weiner et al. attempted to measure the loss of personal subjective recollections following ECT because these are "most consistent with the nature of memory complaints by ECT patients themselves". The memory inventory in the study spanned several years prior to the electroshock treatment. The group found "objective personal memory losses" that lasted throughout the 6-month duration of the study.

In an earlier paper by a team that also included Weiner [65], there was emphasis on the potentially injurious effect on the patient and the patient's family caused by losing autobiographic memories. The authors observed that "autobiographic memory failures, if added across a course of ECT, may produce gross memory gaps that may be disconcerting to a patient and a patient's family, because the patient's sense of continuity with his or her own past may be disrupted" (p.923). Unfortunately, the 1986 paper by Weiner et al. which demonstrates these autobiographic losses shows no such empathic concern for the patients and their families.

In 1989, Avraham Calev and his Israeli colleagues compared the effects of ECT and imipramine on memory and other aspects of cognitive functioning. This method had the advantage of controlling for the diagnosis of major depression. Twenty-six depressed patients were given either 7 ECT (N = 16) or imipramine 200 mg per day for 21 days (N = 10). Bilateral ECT was administered with a MECTA machine using constant current, brief-pulse treatment to ensure that the stimulus intensity was the minimal necessary to produce a seizure. The ECT patients were tested before treatment and then 18-21 h after the seventh ECT. The imipramine patients were tested during the fourth week of treatment. The authors summarize their results: "ECT-treated patients also had a significant and well-characterized impairment in retrograde memory" (p.111).

In the Calev study, using the Famous Events Recall test, ECT patients did "significantly poorer after treatment than before treatment" with a 31% decline in recalled memories compared to baseline (p.115). Since the test did not focus sufficiently on recent events, no amnesic gradient was observed. The retrograde memory of the imipramine patients was unaffected by treatment.

Using the personal memory inventory, "Imipramine-treated patients forgot far less autobiographic information than did ECT-treated patients" (p. 115). A typical retrograde amnesia gradient was observed with more severe forgetting for more recent personal events.

Anterograde memory performance, the recollection of words from one day to another, was found to be

impaired in both treatment groups. Two other tests for anterograde processes showed no change in either treatment group.

In summary, the post-ECT patients showed marked retrograde amnesia for personal and famous events recall while the imipramine patients did not, and both groups showed "relatively mild" anterograde memory problems but the drug group was still taking the medication at the time. The authors believe that their research confirms earlier work demonstrating memory deficits for autobiographic and public events, and "rapid forgetting in verbal retrograde memory", as a result of ECT.

Research conducted by Sobin, Sackeim, and other ECT advocates [160] examined the relationship between stimulus parameters and both acute disorientation and retrograde amnesia. As discussed earlier, they found correlations between the length of initial disorientation following ECT and retrograde amnesia measured two months later. Some of the patients were subjected to crossover treatment with high-dose bilateral ECT. These patients, the authors tell us, had even more severe retrograde amnesia than the others. But instead of investigating and emphasizing this important finding, they excluded these patients from their 2-month follow up data. They report only the data for those patients who received one course of ECT (p.998).

The Sobin et al. study used a structured interview with 281 inquiries to focus on "illnesses, employment history, places of residence, travel and entertainment activities, emotionally significant events, and everyday events in the lives of patients, their families and their friends".

Many of the patients developed irreversible retrograde amnesia that lasted at final testing two months after their last ECT. The table for short-term memory loss (one week after the last treatment) shows that the patients lost large percentages of their autobiographic memories. Following ECT, the four groups of patients lost or distorted the following percentages of their previously recalled memories: low-dose unilateral (29.8% loss), high-dose unilateral (26.8% loss), low-dose bilateral (47% loss) and high-dose bilateral (38.5% loss). These are extraordinary figures reflecting massive losses of retrograde memories of important past personal events.

What percentage of memory did the patients recover at two months? The study provides no relevant charts or tabulations. However, a careful reading of the data discloses massive, irreversible losses. One group of patients, as already noted, received unilateral ECT followed by a crossover to bilateral ECT. Two months after ECT, these patients showed no improvement in their post-ECT retrograde amnesia: "Patients who received one course of ECT showed marked improvement in follow-up amnesia scores compared to short-term amnesia scores... while patients who received crossover treatment were unchanged". In light of the very large losses documented in the chart for all the post-ECT patients, this indicates that these cross-over patients -- with more intensive treatment and even greater memory deficits -- never recovered the large portions of the pre-ECT memories that they lost. The quote also indicates that patients who did regain more of their memories displayed "marked improvement". "Marked improvement" falls short of complete recovery but the authors do not give further information.

Even the relative recovery of the patients who did relatively well is in doubt. The fine-print description of testing procedures explains that patients were not evaluated as failing in their memory at two months post-ECT if they recalled either the memories they reported before ECT or if they recalled instead the distorted memories they produced immediately after ECT: "For long-term testing, patients were credited with consistency if the response at 2-month follow up matched either the baseline (pre-ECT) or the 1-week post-ECT (short-term) response"(p.997)

It makes no rational sense to credit patients with a recovered memory if their 2-month follow up responses match their incorrect, distorted post-ECT memories. Why would the investigators so greatly distort their procedure? Were they trying to cover up marked memory deficits that would have undermined their advocacy of ECT? It is difficult to conceive of any other explanation. The memory losses must have been so great two months post-ECT that the authors, as advocates of ECT, decided to compare their patients' two month post-ECT recollections to their one-week post-ECT memories.

The authors tell us that in general there were significant "magnitudes" of retrograde amnesia at two months but they don't provide us the percentages. They must have been very large. The authors found their results consistent with Weiner et al. [180], using a "shortened version" of the interview, who "observed persistent amnesic effects 6 months after bilateral ECT".

Overall, the studies of autobiographic memory produced by the ECT advocates confirm widespread and potentially devastating losses which they in turn have tried to minimize.

3.4. Autobiographic memory loss from multiple-monitored ECT

One of the newer techniques of ECT - multiple-monitored ECT (MMECT) - employs four electroshocks in one session while recording EEG, EKG, and vital signs. Barry Maletzky, an advocate of the treatment, is one of the few who have asked patients in detail about their memory function following ECT. After pointing out that some psychological testings have failed to confirm cognitive deterioration, Maletzky [126, p.180] observed:

However, if one listens to what patients say who are treated with either conventional ECT or MMECT, subtle cognitive deficits, not easily tested, are discussed. Some patients will mention deficits only if careful inquiry is pursued. Most will not identify these problems even if asked, thus indicating that either they are absent or so subtle as to be imperceivable to the patient.

Maletzky went on to describe a series of 47 MMECT patients who were interviewed 3 to 6 months after ECT treatment. Thirty six percent identified a cognitive problem, including difficulty finding their way around, recalling past events in sequence, and understanding TV shows. In another follow-up by Maletzky using a questionnaire and interviews, 23% reported "long term memory deficits". The problems described by Maletzky's patients extend beyond memory dysfunction to substantial cognitive deficits, such as a math student's loss of his ability to do computations in his head.

3.5. An important review ignores the data

We have already seen how the 1990 APA Task Force report on ECT simply ignored the significant body of literature concerning memory loss from ECT and then misrepresented the one study that it cited. Since then, the most highly quoted review of ECT was written by Devanand et al. (1994). They fail to mention any of the Janis studies. They ignore the follow-up studies indicating that patients frequently experience permanent memory loss, and raise no issues about the improbability of full recovery from a traumatic acute organic brain syndrome. Appearing in the American Journal of Psychiatry amid growing controversy surrounding ECT, the review by Devanand and his colleagues was seemingly intended as an establishment response to criticism. For this reason, I shall continue to examine its conclusions at relevant

points in this paper.

At least some medical reviewers have concluded that the evidence supports the reality that ECT produces persistent retrograde amnesia. R.J. Dolan [69] from the United Kingdom reviewed "Neurologic Side Effects of Psychiatric Treatments". Regarding ECT, he cites the literature and concludes:

Long-term memory impairment is a frequent subjective complaint of patients who have received ECT. An objective basis to these complaints is established. A disturbance of personal biographic memory is seen in a proportion of patients following ECT (p.300).

4. Studies of brain damage from ECT

4.1. Extensive animal research

There is extensive animal research literature confirming brain damage from ECT. The damage is demonstrated in many large animal studies, human autopsy studies, brain wave studies, and an occasional CT scan study. Animal and human autopsy studies show that ECT routinely causes widespread pinpoint hemorrhages and scattered cell death. While the damage can be found throughout the brain, it is often worst in the region beneath the electrodes. Since at least one electrode always lies over the frontal lobe, it is no exaggeration to call ECT an electrical lobotomy.

The original animal studies are from the 1940s and 1950s, but they are still valid. Several of them were elegant by any scientific standard. The model for these studies was conducted by Hans Hartelius on cats and published in 1952 in a book-length publication, "Cerebral Changes Following Electrically Induced Convulsions".

In the double-blind microscopic pathology examination, Hartelius was able to discriminate with error-free accuracy between the eight electroshocked animals and the eight nonshocked animals. The experimental animals showed vascular wall damage, gliosis, and nerve cell abnormalities:

The vessel wall changes found more frequently and more distinctly in the animals subjected to ECT consist of characteristic sac-like dilatations of the perivascular spaces, which in some cases contain histiocytic elements. The glial reaction, of the progressive type, consists of an increase in the number of the small glial elements in the parenchyma and satellitosis beside the nerve cells. The nerve cell changes observed are in the form of various stages of chromophobia, frequently with coincident nuclear hyperchromatism. The arrangement of such cells is mainly focal.

The changes were statistically significant. The abnormalities were found most heavily in the animals given the greater numbers of ECTs, were most dense in the frontal lobe, and were correlated with increased age of the animal (implying increased vulnerability).

Hartelius was cautious in his determination of irreversibility. He required shadow cells and neuronophagia (the removal of dead or diseased nerve cells by phagocytosis). On the basis of these findings, he concluded, "The question whether or not irreversible damage to the nerve cells may occur in association with ECT must therefore be answered in the affirmative".

Hartelius used relatively small doses of ECT - a fraction of that usually administered to contemporary

psychiatric patients. Hartelius' observations supported a number of earlier studies using dogs and monkeys, which also employed doses of electricity below those used in ECT today (comparative currents are reviewed in Breggin [32, pp. 119-122]). Greater electrical energy must be applied in modern ECT in order to overcome the anticonvulsive effects of the sedation given prior to ECT. Patients nowadays are also frequently taking sleeping medications or daytime tranquilizers that further necessitate an increased dose of electricity in order to cause a seizure. Furthermore, modern ECT proponents often advocate and use excessively large doses far above that required to produce a convulsion (see ahead).

Ferraro et al. [76] and Ferraro and Roizin [75] conducted controlled studies of the effect of clinical doses of ECT on *Maccacus rhesus* monkeys. Armando Ferraro was Clinical Professor of Psychiatry at Columbia University and Principal Research Scientist (neuropathology) at the New York State Psychiatric Institute. The conditions of the experiments were highly sophisticated, including the use of regular ECT machines, smaller sized electrodes to fit the monkey heads, restraint to keep the heads from banging, and the minimally necessary dose of electricity to cause a convulsion. They stated, "the intensity and voltage of the current was considered closest to the therapeutic shock used in human beings" [75]. The current dose was as low as 90 V, 102 mA for 0.15 duration - a total energy dose that is well below that routinely used in modern ECT.

In the 1946 study, Ferraro and Roizen administered ECT three times per week in relatively short courses (4-18 in number). After only 4 ECT, one animal had microscopic findings: "here and there in the cerebral cortex there were some areas of rarefaction (cell loss)". After 12 ECT, another showed "small areas of rarefaction as well as satellitosis and neuronophagia". Another, again after 12 ECT, displayed "slight rarefaction of nerve cells and a few acellular areas in the front lobes". In addition to this evidence of cell death, they also found cells in various states of degeneration, loss of myelin sheaths, glial proliferation, dilated blood vessels, microscopic effusions of blood, petechial hemorrhages and other neuropathology which they related to the ECT. The pathology was worse with increasing numbers of ECTs. The overall findings are very consistent with, and perhaps more severe, than those reported by Hartelius in cats.

In the 1949 study, Ferraro et al. gave larger numbers of ECTs (32-100). Many patients receive this number of ECTs, usually over several series. With the fewest electroshocks, they found "moderate nerve cell rarefaction" and "acellular areas". Again damage was proportionate to the current intensity and the number of ECT. Photographs of the microscopic findings are reproduced in both papers.

Studies by Alpers and Hughes [5] on ECT in cats found evidence of subarachnoid hemorrhages and scattered punctate hemorrhages in the brain. They correlated this damage with autopsy findings in two human autopsy cases [6]. In 1946, Alpers reviewed the existing world literature on ECT effects in animals, including additional studies of cell death in dogs [138] and rabbits [110]. Alpers noted that some studies which claim to show little or no effects from ECT in fact indicate cell abnormalities and even cell death.

A variety of mechanisms for ECT-induced brain damage have been proposed, usually related to the intensity and path of the electric current [4,32]. Even very small doses of electrical stimulation - less than the amount that reaches brain tissue during ECT - can produce regional vasospasm, followed by cellular anoxia. Since the blood vessels are constricted, increasing the oxygen content through artificial respiration (modified electroshock) would have little or no positive effect. The exhaustion of brain cells through intense seizures also makes them more vulnerable to damage. More recent studies in rats confirm that seizures induced by minimal currents with indwelling electrodes can produce neuronal loss, especially in the hippocampus of the temporal lobe [56]. The hippocampus plays a critical role in memory.

The studies by Hartelius, by Ferraro et al., and by Alpers and Hughes were definitive. They demonstrated that ECT causes brain damage in monkeys, dogs, and cats, including hemorrhages and cell death. The "controversy" should have ended with these studies, as well as with a number of confirmations from other animal investigations in the 1940s and 1950s [4,32,101]. Instead, the research stopped, and the coverup began.

The Russians have carried out a variety of neuropathology studies on animals subjected to clinical intensities of ECT to determine if there is permanent brain damage. Babayan called for a ban on the treatment in 1985, citing work at the USSR Academy of Medical Sciences as, "convincing proof... pointing to grave changes in the central nervous system, the nerve cells, the glial-tissue apparatus..." (pg. 37). At another institute, studies of the brains of animals led to a "drastic reduction in the use of electroshock therapy in clinical practice" (p. 134). Babayan compares the treatment to lobotomy.

There have been no studies of large animals using modified ECT under clinical conditions. Meldrum and Brierley [132] studied drug-induced (bicuculline) lengthy seizures in baboons and found widespread ischemic (due to lack of blood flow) changes. Meldrum et al. [134] repeated their earlier experiment, now employing modified ECT, and found similar but lesser ischemic changes in neurones. They concluded that modifying the ECT gave some incomplete protection. However, the seizures were very long. Meldrum et al. [133] once again studied the impact of drug-induced (allylglycine) seizures in baboons under modified conditions. They used 13 animals, and in 8 the seizures were brief, recurring 6-63 times in 2 to 11 h. followed by recovery. The short-duration seizures produced no detectable pathology.

Templer [169] reviewed the question of ECT and permanent brain damage. In regard to animal studies, he focused on Hartelius and also pointed out that animals given artificial ventilation (modified ECT) in other studies also had "brain damage of somewhat lesser magnitude".

While few psychiatrists are willing to say in public that ECT causes brain damage, a large survey of the APA membership, conducted with anonymity in the 1970s, showed that 41% of psychiatrists agreed with the statement, "It is likely that ECT produces slight or subtle brain damage". Only 26% responded that it did not [7, p.4].

4.2. How ECT advocates respond to the animal studies

None of the studies using large animals, including Hartelius and Ferraro et al., are included in the 1990 American Psychiatric Association task force report on ECT. Although the report is supposed to be comprehensive, with hundreds of citations from the literature, it somehow manages to fail to mention the most important animal research. The same is true for the 1990 FDA study.

When Deyanand⁽³⁾ and his associates [68] reviewed "Does ECT Alter Brain Structure?", they concluded that animal studies do not prove brain damage. They accomplish this by dismissing the best studies. Hartelius, for example, is criticized for applying a series of four ECTs with each one spaced at 2 h. But there is no reason to assume that this method is more damaging than larger numbers of ECTs spaced over longer intervals. As presently used, multiple-monitored ECT inflicts four electroconvulsive shocks within the space of approximately one hour. In addition, it is extremely misleading to focus on that particular group of subjects within Hartelius' study. One group of animals in the Hartelius study were given one ECT per day for 4 days and others were treated "with clinical frequency" (three per week).

Devanand et al. dismiss Ferraro and Roizen [75] for using a "large number of ECSs (electroconvulsive

shocks) relative to clinical practice. In reality, many patients are given 32 or more treatments, sometimes in one series, more commonly in two or three. Ferraro et al. [76], utilizing small numbers of ECTs, are dismissed on the speculation that the current went through the brain stem.

Devanand and colleagues do not deal with the fact that almost every study using large animals, as summarized in their own table, shows brain damage. My review indicates that even purportedly negative studies, on actual reading, indicate harmful effects [32]. For example, Devanand et al. describe Lidbeck's [125] study in which several dogs developed "minimal perivascular and ischemic changes" [68]. They leave out that in two of the four animals "nerve cells were shrunk and there was a decrease in the number of stainable granules" [125]. Nor do they mention that one of the animals developed blood clots in its brain.

One cannot prove the safety of ECT by criticizing multiple studies that show damage. To be ethical and scientific, ECT advocates must produce carefully conducted, large-animal studies that show no damage. This has not been done. In fact, the only studies that Devanand et al. find acceptable were performed on rats rather than dogs, cats, and primates whose brains are more akin to humans and more sensitive to trauma.

4.3. Brain scans

There has been contradictory evidence of ECT damage in brain-scan studies, most of which have been carried out by staunch advocates of the treatment. Using CT scans, Weinberger et al. [179] found that chronic schizophrenic patients with a history of ECT had more enlargement of their ventricles (cerebral atrophy) than those who had no ECT. Stretching to exonerate ECT, they declare, "Either EST further enlarged the ventricles of the patients treated with it, or it was used with greater frequency in patients who tended to have larger ventricles". In another CT study, Calloway et al. [51] found a correlation between frontal lobe atrophy and ECT in 41 "elderly depressives".

A team led by Coffey et al. [63], using magnetic resonance imaging (MRI), studied 35 patients before and after ECT. The follow-ups were 2 or 3 days after and 6 months after. In five subjects, they found "an apparent increase in subcortical hyperintensity". Coffey, a strong ECT advocate who has performed ECT on many hundreds of patients, dismissed his own finding as "most likely secondary to progression of ongoing cerebrovascular disease during follow up" [62]. I have evaluated several post-ECT patients with very similar MRI findings related to their ECT treatment.

Pande et al. [141] found no MRI pathology in 7 ECT patients. However, the studies were performed 1 week after the last ECT, so that late-maturing pathology would not have been discovered. Bergsholm et al. [23] found no pathology on MRI in 40 patients, with the exception of a 69-year-old man who suffered a dilatation of the left temporal horn, which the authors dismiss as unrelated to ECT.

Devanand et al. [68] reviewed the brain scan literature and found the evidence for brain damage unconvincing. They accept Coffey's unsubstantiated claim that the pathology found in four patients after ECT was due to progressive cerebral vascular disease rather than the more obvious trauma of ECT. They dismiss studies showing damage.

The latest American Psychiatric Association Task Force Report [9] demonstrates the degree to which the possibility of brain damage is now denied or rejected. In regard to the need for acknowledging brain

damage on consent forms, the task force advised:

In light of the available evidence, "brain damage" need not be included as a potential risk.

4.4. Death and autopsy findings

Many deaths were reported in association with ECT in the first few decades of use. An extensive autopsy series indicated that many suffered from trauma to the brain resulting in visible pathology [113]. More recently, advocates for ECT have claimed the death rate is very small or nearly nonexistent; but I have suspected that deaths are simply no longer reported. For example, I have known of deaths of ECT recipients in the Baltimore-Washington area that went unreported.

New data has confirmed the probability of a significant death rate from ECT. A recent law in Texas requires the reporting of death within two weeks after ECT. From June, 1993, through August, 1994, eight deaths were reported among nearly 1,700 patients subjected to electroshock treatment. Controversy surrounds causation, and critics of ECT are attempting to obtain more autopsy details [159].

5. Modified ECT

For the past two to three decades, a modified form of ECT has been commonly (but not exclusively) used in the United States. It involves sedation with a short-acting intravenous barbiturate, followed by muscle paralysis with a curare derivative, and artificial respiration with oxygen to compensate for the paralysis of the patient's breathing musculature. The purpose of these modifications was not, as some advocates claim, to reduce memory loss and brain damage. Muscle paralysis was intended to prevent fractures of the spine and limbs, as well cracked teeth, from severe muscle spasms. The artificial respiration was added to keep the paralyzed patient oxygenated.

The modifications used in contemporary ECT make clear that ECT-induced convulsions are far more severe than the spontaneous convulsions in grand mal epilepsy. Patients with seizures of unknown origin, or with seizures due to brain injury, rarely break their limbs or their vertebrae during the convulsion. The muscle spasms are not intense enough to produce these effects. Yet these fractures were common with unmodified ECT.

ECT advocates commonly claim that recent modifications have made the treatment much safer, and that its negative public image is unfairly based on the older methods. However, the most basic modifications --anesthesia, muscle paralysis, and artificial respiration - are not new at all. I prescribed and administered such modified treatment more than thirty years ago (1963/64) as a resident at Harvard Medical School's main psychiatric teaching facility, the Massachusetts Mental Health Center.

The public's "mistaken" image of ECT is, in reality, based on modern modified ECT, which has been around for a long time. It is actually more dangerous than the older forms. The electric currents must be more intense in order to overcome the anticonvulsant effects of the sedatives that are given during modified ECT [32]. Too frequently, the patient is routinely given a sleeping medication or tranquilizer the night before, further increasing the brain's resistance to having a seizure. In addition, the patient is exposed to the added risk of anesthesia. Other modifications include changes in the type of electrical energy employed and the use of unilateral shocks applied to the non dominant (nonverbal) side of the

brain. However, these modifications remain controversial. Since the APA task force does not exclusively endorse nondominant (unilateral) ECT, the claim that this method is much safer becomes moot. Bilateral ECT continues to be used around the world. Besides, as already described, some ECT advocates give excessive electrical doses -- beyond the dose required to produce a convulsion.

There is no reason to believe that shocking the nonverbal side of the brain is less harmful. As Blakeslee [29] has confirmed, damage and dysfunction on the nonverbal side are more difficult to recognize or to describe (see discussion of anosognosia ahead). But the defects are no less devastating. Injury to the nonverbal side impairs visual memory, spatial relations, musical and artistic abilities, judgment, insight, intuition, and personality. It is ironic that biopsychiatry promotes sacrificing the nonverbal side of the brain, while humanistic psychology is emphasizing its importance to the full development of human potential.

No matter how ECT is modified, one fact is inescapable: evolution has assured that human beings do not easily fall victim to convulsions. Therefore sufficient damage must be inflicted to overcome the brain's protective systems.

6. The brain-disabling principle

6. 1. *Early advocates of "Brain damage as therapy"*

At the time that ECT was first developed, it was thought that convulsions induced by a variety of methods, including insulin coma and stimulant medication, were useful in treating psychiatric disorders, especially schizophrenia. It was often assumed that these treatments had their therapeutic effect by causing significant microscopic brain damage. Some advocates openly called for inducing brain damage and dysfunction (reviewed in detail in [32]). Bini [27], for example, reported that ECT produced "widespread and severe" neuropathology in the brain and that these "alterations" might be responsible for the "transformation" seen in schizophrenic patients after ECT. In the same year, Roy Grinker (in a discussion of Weil et al. [178]) compared ECT to lobotomy and speculated, "Does shock therapy improve schizophrenic patients by structural damage of a less intense but more diffuse type"? In 1941 Walter Freeman wrote an editorial entitled "Brain damaging therapeutics" in which he argued for the basic principle that the major psychiatric treatments, including electroshock and lobotomy, work by disabling brain function. In 1941, Harry Solomon's introduction to Jessner and Ryan's *Shock Treatment in Psychiatry* acknowledged that ECT produces memory loss, brain wave changes, and "cerebral cellular damage and vascular injury". He connected this to the therapeutic effect, specifically the production of euphoria and hypomania. The textbook itself cited evidence for severe brain damage from ECT, including "capillary hemorrhage, ganglion cell changes, consisting of swelling and shrinkage, satellitosis, gliosis and demyelination".

From the very beginning - based on animal studies, human autopsies, and clinical observation - ECT was known to cause brain damage. In fact, the brain damage was considered the principal element of the therapeutic impact. Later, with increasing concern about ECT's bad image, advocates began to deny these well-established observations.

6.2. Fink confirms the brain-disabling principle

Max Fink is a leader in promoting ECT and his attitudes, if sometimes more extreme, reflect those of many others who are leading the current resurgence of ECT in the North America and Europe. A pro-ECT review by another ECT advocate, Weiner, drew from Fink [83] accusations that Weiner "genuflects to avoid criticism" and that "such kowtowing is inappropriate".

Fink, himself a member of the 1978 and 1990 APA task forces, for decades argued and demonstrated scientifically that ECT's "therapeutic" effect is produced by brain dysfunction and damage. He pointed out in his 1974 textbook that "patients become more compliant and acquiescent with treatment" (p. 139). He connected the so-called improvement with "denial", "disorientation", and other signs of traumatic brain injury and an organic brain syndrome (p. 165).

Fink was even more explicit in earlier studies. In 1957, he stated that the basis for improvement from ECT is "craniocerebral trauma". In 1966, Fink cited research indicating that after ECT "the behavioral changes related to the degree of induced trauma..." (p.475). Referring to the multiple abnormalities produced in the brain following ECT, Fink wrote "In these regards, induced convulsions in man are more similar to cerebral trauma than to spontaneous seizures" (p.481). He stated that improvement depends on the development of an abnormal EEG and other changes in the brain and spinal fluid typical of trauma and compared ECT to "cerebral trauma".

Fink cited Tower and McEachem [173], correctly stating that they "concluded that spinal fluid changes in induced convulsions were more like those of craniocerebral trauma than those of spontaneous epilepsy" [80]. He then gave further evidence for this comparison between ECT and traumatic brain injury.

As recently as 1974, Fink continued to propose that ECT has its effect by traumatizing or damaging the brain. He begins his discussion by noting that psychiatric "treatments have been often drastic" and then cites, among other examples, heat and burning, bleeding, water immersion, and craniotomy. He then goes on to present several axioms of ECT, including the connection between the supposed therapeutic effect and traumatic changes in the brain. He speaks directly of the producing "cerebral trauma" reflected in EEG slow wave activity (p. 9). He compares induced convulsions to "craniocerebral trauma" (p. 10). He attributes improvement to the increased use of "denial" by the patient and to the development of "hypomania" -- both signs of profound irrationality caused by brain damage and dysfunction (p.14).

The 1990 task force report, despite Fink's participation, made no such comparisons between head injury and ECT; instead the report dismissed any suggestion that the treatment is significantly traumatic. In depositions in defense of doctors who give ECT, Fink now takes the position that ECT causes no brain damage.

The 1990 APA task force report notes that low-dose unilateral ECT is often less effective than forms of ECT that deliver more electrical energy. This observation tends to confirm the brain-disabling principle that efficacy depends on the degree of damage.

6.3. Sackeim confirms the brain-disabling principle

More recently Sackeim [152] and Sackeim with a team of colleagues [154] have covertly revived the principle that a therapeutic response depends upon the degree of brain damage and dysfunction. Sackeim [152] has found that "Regardless of electrode placement, patients who received high dosage treatments

responded more quickly . . . Critically, we also found that the rate of clinical response was dosage sensitive". As previously noted, the degree of post-ECT disorientation and later retrograde amnesia is also dose sensitive.

The study previously cited by Sobin et al. [160] used the suprathreshold dose (2.5 times) in a group of patients in a crossover study. As already noted, this group suffered from massive retrograde amnesia that did not improve two months after ECT.

I evaluated a case in which a doctor followed Sackeim's published recommendation and gave his patient the increased dosage. The patient suffered severe, irreversible memory loss and chronic mental dysfunction, rendering her permanently unable to work at her previous high intellectual level.

The tendency to increase the electrical dose wholly undermines the promotional campaign aimed at convincing the public that modern electroshock is safer. Sackeim and his colleagues often use bilateral ECT -- the most obviously damaging method -- with a dose of electricity 2.5 times that required to produce a convulsion in the patient. In addition, a growing emphasis on continuation or maintenance ECT will expose increasing numbers of patients to chronic brain trauma and dysfunction (for an example of maintenance ECT, see [147]).

More striking, Sackeim wants to do away with the safety features currently placed on most ECT machines that limit current intensity: "These upper limits result in clinicians resorting to unnecessary and perhaps risky maneuvers.. ." to get higher doses. According to Sackeim, "In my view, a strong argument can be made that the next generation of ECT devices have significantly higher upper output limits, perhaps at least double what is available with the current generation" (pg.235).

In a recent issue of Convulsive Therapy, ECT advocate Charles Kellner [123] quotes a description of shock-induced mental devastation written by survivor "Ellen Wolfe" [184]. Mrs Wolfe describes the "muddles" she gets into reading and her inability to recall even dramatic life events, such as the assassination of President Kennedy. Kellner states that her tragic outcome, "a very severe case", is "likely the result of a series of treatments with high-dose bilateral sine wave ECT" (pg. 133). Without seeming to realize that modern ECT is often more "high-dose" than the older methods, he states that such a tragic outcome is unlikely with contemporary ECT. This view contrasts sharply with his more cautionary words:

Memory is often equated with the very essence of a person's "being". As such, discussions about ECT's effects on memory deserve our most careful consideration (p.34).

6.4. How ECT works: iatrogenic helplessness and denial

ECT provides a prototype for the concept of iatrogenic helplessness and denial [32]. Controlled studies of ECT show that any therapeutic effect evaporates after 4 weeks, the approximate time it takes to recover from the most severe symptoms of the organic brain syndrome or delirium. Except for psychosurgery, ECT provides the most extreme example in which the psychiatrist denies the damage he is doing to the patient, and then utilizes the effects of that damage to produce less emotionally aware, less autonomous, and more manageable patients. As Max Fink used to openly describe, brain damage and the exercise of medical authority push patients into denial about the harm done to them as well as about their still unresolved personal problems.

Consistent with other victims of central nervous system damage, most ECT patients minimize or deny their real losses of mental function. This denial of mental dysfunction in brain-damaged patients is called anosognosia ([88]; also see [36,45]). C.M. Fisher considers anosognosia or denial of dysfunction to be a hallmark of brain injury: "Unawareness accompanies so many neurologic defects that one might invoke anosognosia as a broad principle of cerebral dysfunction in humans". I have pointed out that it should be considered an integral part of the brain-disabling effects of all psychiatric treatments which impair brain function. Brain-disabling treatments reduce the patient's awareness of the mental dysfunction caused by the treatment.

While damage to either side of the brain can produce anosognosia, it seems more common following damage to the nondominant side (in right-handed individuals, the right is usually nondominant). In electroshock treatment, at least one electrode lies over the nondominant side. In contemporary ECT, both electrodes are frequently placed over the nondominant side.

Nondominant electroshock starkly illustrates the principle of iatrogenic helplessness and denial: the doctor damages the brain in such a way as to confound the patient's ability to perceive the resulting dysfunction. Neurologically-informed advocates of ECT are well aware that electroshock patients end up suffering from anosognosia and denial, and therefore cannot fully report the extent of their memory losses and mental dysfunction. Yet these same advocates claim that patients exaggerate their post-ECT problems.

Interviews with family and friends of patients often disclose that they are painfully aware of the damage done to their loved ones. Often, the psychiatrist is the only one who consistently and unequivocally denies the patient's damaged state.

7. Clinical effects on women, children, and the elderly

7.1. ECT women, and memory loss

Women have always constituted the majority of subjects of the most destructive psychiatric treatments, including lobotomy and insulin coma. More recently, as documented earlier in this report, older women have become the major target population for ECT, despite the absence of controlled studies on safety or efficacy in the elderly.

One of the most remarkable reports in the ECT literature was published by Carol Warren [177] who studied 10 women post-ECT, including their family relationships. The study confirms the brain-disabling principle and illustrates how brain damage can be used by relatives to enforce iatrogenic denial and docility. Many of the women thought that the purpose of the ECT was to erase their memory. While some felt it was helpful to forget painful memories, they "uniformly disliked the loss of everyday memory, as well as associated effects such as losing one's train of thought, incoherent speech, or slowness of affect. What specifically was forgotten varied from matters of everyday routine to the existence of one or more of one's children...". Without reporting on the clinical significance of the women's experience, Warren is describing mild to moderate dementia following closed-head injury.

Family members sometimes approved of the memory loss:

Husbands might wish to have their wives forget the emotional troubles, including marital

strife, which precipitated hospitalization. Mr. Karr commented on his wife's long-term memory loss as proof of her successful cure by ECT, saying that her memory was still gone, especially for the period when she felt ill, and that "they did a good job there". These husbands used their wives' memory loss to establish their own definitions of past situations in the marital relationship.... Other relatives, too, found it in their interest to have the expatients forget: thus they could freely redefine past situations without challenge.

Warren described how another husband used his wife's memory loss to manipulate her:

Rita Vick [a pseudonym] had forgotten, after ECT the five of her seven children who had been removed from her custody. One day she found an album in the Vick house and asked her husband "who were all those children?" For fear of upsetting her with renewed thoughts of the custody loss, Mr. Vick told her that they were a neighbor's children.

One woman believed that her mother wanted her to have "the full treatment" to "make me forget all those things that happened", including being molested by her mother's brother.

Three of the ten women lived in dread of ECT for years afterward, but were afraid to express their angry feelings for fear of being sent back to the hospital for involuntary electroshock treatment. In my clinical experience, this is a realistic fear. Doctors frequently respond to complaints about ECT treatment by deciding that the patient is in need of more treatment. Increased exposure to the brain-damaging effects of ECT can almost always be relied on to eventually put an end to the patient's protests ("complaints").

Shock treatment has been used even more blatantly to erase the memories and even the personalities of patients, usually women. H.C. Tien, in the early 1970s, described the use of unmodified ECT to erase the personalities of women, then to "reprogram" them as more suitable wives with their husband's help ("Electroshock: Key Element...", 1972, "From Couch to Coffee Shop...", 1972).

Psychiatrist D. Ewen Cameron at McGill University, in part funded by the CIA, used multiple ECTs to obliterate the minds of his patients and then to reprogram them ([52]; for more details on the Tien and Cameron controversies, see also [32,39]).

7.2. ECT and the elderly

As already noted, elderly women have become the most frequent target of ECT in the United States and Canada. The trend to shock the elderly also seems to be growing in Europe [58,73], and Australia [140].

Urged on by the impetus to cut the costs of medical care in the US, a group led by William Mc Donald [131] has recently advocated maintenance ECT for the elderly. An initial course of ECT was followed by intermittent treatments over a sixth month or more time period. This was not a blind or controlled study; it speaks more to the willing to bombard the brains of the elderly than to the potential value of the treatment.

The elderly, of course, have more fragile brains, and are especially sensitive to biopsychiatric interventions, including relatively low doses of drugs. In addition, many elderly already suffer from memory dysfunction due to a variety of causes, making them especially vulnerable to the worst effects of ECT.

Against all common sense, the APA 1990 task force advises that ECT can be used "regardless of age" (p.15). It cites the successful treatment of a patient aged 102 (pp.71-72). The APA report does warn, however, that "some elderly patients may have an increased likelihood of appreciable memory deficits and confusion during the course of treatment" (p.72). Similarly, Kamholz and Mellow [122] describe the use of ECT for the elderly in glowing terms: "It is increasingly advised as a first-line therapy for severely ill patients who are badly malnourished or who are at risk for suicide". They also recommend it for patients who cannot tolerate antidepressants because of cardiac disease. There is no suggestion that ECT poses a special threat to the vulnerable brain or cardiovascular system of the elderly. Reports like these are spurring on the increased use of ECT in the elderly.

The aged are, in fact, gravely at risk when exposed to any form of head trauma, including electrically induced, closed-head injury from ECT; and there is little evidence that ECT is helpful to them. There are a growing number of reports of special dangers to the elderly that are not mentioned in the APA or the FDA reviews [77,144].

In a curious twist, an article by Burke et al. [48] is listed in the bibliography of the APA report but not cited in the actual discussions of the elderly. Burke and his colleagues found a high rate (35%) of complications among the elderly. They noted, "Common complications in the elderly include severe confusion, falls, and cardiorespiratory problems" (p.516).

In a study involving three times as many women as men, Kroessler and Fogel [12] produced data indicating that ECT can cause a devastating decline in longevity:

This is a longitudinal study of 65 patients who were 80 years old or older at the time they were hospitalized for depression. Thirty-seven were treated with ECT and 28 with medication. Survival after 1,2, and 3 years in the ECT group was 73.0%, 54.1%, and 51.4%, respectively. Survival after 1,2, and 3 years in the non-ECT group was 96.4%, 90.5%, and 75.0%, respectively (p.30).

These are extraordinary findings, indicating a very high increase in mortality in the elderly who receive ECT. The authors, however, argue that the patients receiving ECT were more physically ill and hence at greater risk of dying. They provided no data to explain such a vast difference in mortality. The lethality of ECT was made even more tragically wasteful by its comparative lack of efficacy. Kroessler and Fogel found that ECT patients were much more frequently rehospitalized for depression than non-ECT patients (41% versus 15%). The recurrence rate of depression was more than twice as high among the ECT patients as the non-ECT patients (54.1 % versus 25%). Lasting recovery from depression was much lower in ECT patients (22% versus 71%).

The Kroessler and Fogel study by itself should make any clinician hesitant to recommend ECT for any elderly person. But it shouldn't have been necessary to subject these elderly people to ECT. An earlier study by a team lead by Rogeho Cattan [54], while purporting to support ECT, also demonstrated devastating results.

Cattan's group compared the effects of ECT in 39 patients aged 80 and over (very elderly), and 42 patients age 65 to 80 (elderly). Approximately three-quarters were women. The two groups, the very elderly and the elderly, experienced the following rates of ECT-induced complications: cardiovascular effects, 36% versus 12%; falls, 36% versus 14%, and confusion, 59% versus 45%. In the group aged 80 and over, 77% experienced some untoward event related to ECT. In the younger group, the rate was 62%. The seriousness of the adverse effects endured by these elderly patients is documented in the

report's criteria for recording a cardiac adverse effect:

Cardiovascular events, including cardiac arrhythmias requiring medical treatment, angina, and/or myocardial infarction, persistent hypertension requiring medication or increased dosage of previous antihypertensive medication, hypertensive crisis, development or worsening of congestive heart failure.

In any age group, but especially in the old and the very old, these are extremely serious iatrogenic adverse events.

The outcome evaluation portion of the study was not controlled. Even using highly biased impressions drawn from progress notes, only 13% of the very elderly group were rated as having a "good outcome" at the time of discharge, and only 33% of the elderly group.

The data in the study confirms that ECT is a highly dangerous and relatively ineffective approach to treating depression in the elderly; but the authors conclude otherwise. They declare, "In summary, this study supports ECT as an effective and relatively safe procedure in the older population" (p.758).

Elderly men and women have many reasons -- psychosocial and economic, some of them rooted in the ageist and sexist attitudes of our society -- for feeling depressed. Often, they need improved medical care, social services, family involvement, and loving care from friends and volunteers. Typically they are being prescribed too many medical drugs or they are taking them in an inconsistent a fashion. This often results in drug-induced adverse psychiatric effects, including depression and anxiety. All of their basic needs may require attention. Meanwhile, they do not have the strength to resist a doctor's proposal that they undergo electroshock. There may be no family members available or willing to protect them. Whatever the source of their depression, the elderly do not need more brain cell death, mental dysfunction, and memory deficits.

I have been a consultant or a medical expert in several suits in which psychiatrists have tried to administer electroshock against the will of elderly women who had no family to defend them. Each time, the doctors have backed down or, as in the case of Lucille Austwick, they have lost in court [30, p.19]. However, many other elderly women are getting electroshocked involuntarily without their situation gaining public attention. In addition, in my experience, many seemingly voluntary patients are badgered or misled into taking the treatment.

Electroshock advocates argue that more women than men become depressed and so more women need ECT. But why do more women become depressed? Multiple research studies have now connected depression in women to patriarchal oppression, including outright sex abuse [8]. Warren's study confirmed that ECT can and is used to cover up the sexual abuse of women and girls.

Writing in Australia, where they find older women at increasing risk for getting ECT, Melissa Oxlad and Steve Baldwin [140] summarize:

Older people are an inappropriate population for ECT. Due to behavioural or intellectual impairment/deterioration, older adults often cannot give either valid or informed consent. Older people in institutions are at risk of inadvertent rights abuse. Often, these residents do not have access to an advocacy service. The added medical complications (particularly with cardiac problems) that occur with ageing contraindicate ECT as an appropriate treatment for older people. A range of safer, less invasive treatment alternatives exist, which are more

appropriate for older people (p.39).

Oxlad and Baldwin's review found that a variety of forms of counseling and psychotherapy are helpful to elderly depressed patients.

7.3. ECT and children

The literature leaves the impression that ECT is rarely given to children in the United States, Europe, and elsewhere. However, Baldwin and Oxlad [20] point out that there is a vast under-reporting of this controversial activity. They point to Thompson and Blaine's 1987 estimate that between 500 and 3500 minors are subjected to ECT each year in the United States.

In France, Georges Heuyer and his colleagues [111] subjected a large number of children to ECT during the German occupation (discussed in [148]). Their enthusiasm did not lead to the widespread use of ECT for children in France. However, efforts are being made to revive the treatment for adolescents in France. A team led by D. Cohen [61] reviewed the records of 21 adolescents treated with bilateral ECT in their hospital in Paris. They found a "high rate of relapse at 1 year follow-up" and "adverse effects were frequent", but they nonetheless recommend it. They conclude by lamenting, "Present attitudes in adolescent psychiatry do not favor the use of ECT". In the United States, the history of the use of ECT to treat children is wrapped in scandal. In the 1940s on the psychiatric ward of Bellevue Hospital in New York City, Lauretta Bender subjected large numbers of children to electroshock treatment. In one report, she described 100 cases [22]. Her own estimates of success were glowing, but others involved in the projects described the children as terrified and deteriorating intellectually [59,106]. While she diagnosed many children as schizophrenic, many had developmental and behavioral problems [60].

I have personally evaluated two of Bender's cases, adults who were given electroshock by her as children in the 1940s. One boy, G.R., came from a very chaotic, disturbed family. He was terrified by his father's violence when intoxicated and had been truant at school. There is no indication of any severe psychiatric disorder and he was diagnosed "Primary Behavior Disorder -- Conduct Disturbance". At Bellevue, beginning November 3, 1949, he was subjected to a series of 20 ECTs. As far as I could ascertain from the records, he became aggressive for the first time after ECT treatment. G.R. was soon sent to Rockland State Hospital. In adulthood, he became a convicted multiple murderer.

I was asked by G.R.'s attorney to evaluate him as a medical expert in a post-conviction sentencing trial. A jury was empowered to determine if he should be sentenced to death in the electric chair. I showed the jury old films of electroshock treatment being administered during the 1940s. By implication, I made the point that society, having already electroshocked him as a child, should refrain from electrocuting him as an adult. The jury gave him a life term instead of the death penalty.

Another of Bender's cases, Ted Chabasinski, was removed from his home by a city social worker and sent to Rockland State Hospital as a small child. He, too, was given electroshock by Bender. He grew up to become a reform-minded attorney in Berkeley, California, where he has contributed to the campaign against electroshock (for further details about Chabasinski, see [(39,91)]).

Despite the complete absence of controlled studies, there is once again an active attempt in the United States and elsewhere to encourage the use of ECT with children and adolescents [26,85,86]. Rey and Walter [148] reviewed the literature in all languages and found 396 patients, mostly single case reports,

and no controlled studies. Obviously biased in favor of ECT, they conclude "ECT in the young seems similar in effectiveness and side effects to ECT in adults. However, this conclusion is qualified by the lack of systematic evidence" (p.595).

Walter and Rey [176] surveyed the number of patients younger than age 19 in the Australian state of New South Wales who received ECT between 1990 and 1996. They found that 42 patients age 14 to 18 underwent 450 ECTs. Their retrospective analysis concluded it was "always safe", even though 22% reported "subjective memory problems". Rey and Walter want to encourage the use of ECT in both New Zealand and Australia. Their efforts are being met with substantial resistance, causing concern among ECT advocates that it might be banned [114].

In Australia, psychologist Steve Baldwin and his colleagues, Yvonne Jones and Melissa Oxlad, have published many reports that criticize the use of ECT in children and adolescents [16-21,120,139]. A review of the literature by Baldwin and Oxlad [21] located 217 minors subjected to ECT treatment between 1947 and 1996. They found that ECT was not being used as a "life-saving" treatment. Less than 5% of teenagers and children in their meta-analysis sample were described as having any suicidal ideation.

Baldwin [16] wrote:

ECT administration to one child or adolescent per year is one too many. In the context of a still-developing neurological system, the use of invasive and possibly damaging treatment with an unknown mechanism of action, cannot be justified. The use of electric currents to produce seizures in children and adolescents has no place in the mental health services of the 1990s.

In their publications, Baldwin, Jones, and Oxlad have taken the position that treating children and adolescents with ECT is unethical and that responsible professionals should take actions to prevent it. This is consistent with my own position (see ahead).

ECT for children is also being criticized in England [127] and there are attempts to legislate against it [14,15,148]. Four US states ban ECT on children: Alabama, Colorado, California, and Texas.

8. The controversy

8.1. *The press becomes involved*

Particularly in the United States and Canada in the past several years, controversy has been swirling around ECT in the press. Much of it is due to pro-ECT articles inspired by advocates of the treatment who are trying to expand its usage.

The press has not entirely accepted the promotional claims of ECT advocates. A critical article by Cauchon [55] in USA Today was followed up by a remarkable editorial that declared "the long-term effects can be devastating. They include confusion, memory loss, heart failure, and, in some patients, death" [143].

In an article entitled "Shock therapy: it's back" in the Washington Post, Boodman [30] takes a more promotional stance on ECT. She quotes Max Fink as declaring "ECT is one of God's gifts to mankind".

In defending ECT, Fink has also declared to the media that ECT should be given to "all patients whose condition is severe enough to require hospitalization" (quoted in [71]).

In recent years, advocates of ECT have tried to improve its image. Without additional research, they have rejected the idea that ECT can cause brain damage. Glen Peterson (quoted in [151]), a former Executive Director of the International Psychiatric Association for the Advancement of Electrotherapy, has similarly observed:

The possibility of brain damage is absolutely refuted by brain scans, by neuropsychological studies, by autopsies, by animal studies, and by analysis of cerebrospinal fluid and blood chemicals that leak from damaged cells that aren't detected in ECT patients.

8.2. Initial challenges from within the profession

Psychiatrists seldom criticize their colleagues in a systematic or public fashion. Even the widespread resurgence of lobotomy and other forms of psychosurgery during the 1970s, against which this author mounted a successful international campaign, drew little criticism from medical professionals, including psychiatrists [34,46]. Current attempts to revive psychosurgery in North America and Europe are once again meeting little criticism or resistance from within the medical profession [46].

Some of the most systemic analyses of the scientific issues have come from individuals who have been damaged by electroshock [53,91,93-95].

The first significant challenge to ECT within the medical profession was launched by neurologist John Friedberg [99-102]. His 1976 book for the general public, *Shock Treatment Is Not Good For Your Brain*, was followed by a journal review [101]. Friedberg [102] summarized:

The electrodes, whether applied over the temples or limited to one side of the head, discharge through the very sensitive temporal lobes. The squamous plate of the temporal lobe is the thinnest in the cranium -- thus, where the resistance is lowest, the current is greatest. Just beneath lie the temporal lobes containing the least stable cortex by EEG criteria. On their mesial aspects are found the hippocampal formations, so indispensable to memory that their destruction -- by lobotomy and encephalitis, sclerosis from birth injury or hamartomas, impairment of posterior circulation insufficiency, or loss through thiamine deficiency -- leads to the densest amnesias known to medicine... It is here that the cellular damage caused by ECT wreaks the greatest havoc.

Friedberg's publications were quickly followed by a volume edited by "shock survivor" Leonard Frank [91], and an article by neurologist Robert Grimm. Grimm [105] wrote:

How is it the one group in medicine works to protect patients from fits, while another programs fits as therapy? Can both groups be right? Neurologists are trained from a literature and experience based on clinical and model epilepsy, none of which recommends breaching the intrinsic inhibitory mechanisms of brain with transcortical currents sufficient to trigger a convulsion. Instead, all therapeutic effort is aimed at protecting patients from spontaneous or evoked seizures for a combination of clinical, social, and practical reasons. To those who have had training in the complexity and differentiation of neuronal machinery, it hardly seems wise to drive brain above its convulsive threshold, and to do so crudely and

repeatedly and on schedule. The organ gives every indication, in its acute biochemical and electrical response to ECT, that such evoked seizures are clearly traumatic and that a number of behavioral changes follow as a consequence.

My own critique of electroshock began in the scientific literature with the publication of *Electroshock: Its Brain-Disabling Effects* in 1979 and then continued with a series of reports, reviews, and book chapters [33,35-43,45]. Psychologist Robert Morgan [137] published several editions of *Electroshock: The Case Against* with contributions from representatives of psychiatry, psychology, and neurology, as well as the survivor movement.

Reviews of ECT-induced damage to the brain and mind have continued to be published in professional journals [53,92,169]. Templer and Veleber [170], for example, summarized their review of the literature:

Some human and animal autopsies reveal permanent brain pathology. Some patients have persisting spontaneous seizures after having received ECT. Patients having received many ECTs score lower than control patients on psychological tests of organicity, even when degree of psychosis is controlled for.

A convergence of evidence indicates the importance of the number of ECTs. ... [O]ur position remains that ECT has caused and can cause permanent brain pathology.

Many individual health professionals -- some of whom have already been quoted -- have criticized the treatment from their clinical experience. In 1983, neurologist and electroencephalographer Sidney Sament wrote:

After one session of ECT the symptoms are the same as those of concussion (including retrograde and anterograde amnesia). After a few sessions of ECT the symptoms are those of moderate cerebral contusion, and further enthusiastic use of ECT may result in the patient functioning at a subhuman level. Electroconvulsive therapy in effect may be defined as a controlled type of brain damage produced by electrical means... In all cases the ECT "response" is due to the concussion- type, or more serious, effects of ECT. The patient "forgets" his symptoms because the brain damage destroys memory traces in the brain, and the patient has to pay for this by a reduction in mental capacity of varying degree.

Boyle [31] reviewed the literature and stated:

In conclusion, there is considerable empirical evidence that ECT induces significant and to some extent lasting brain impairment. The studies cited above are but a few which suggest that ECT is potentially a harmful procedure, as indeed are most naturally occurring episodes of brain trauma resulting in concussion, unconsciousness and grand mal epileptic seizures. Accordingly, the continued use of ECT in psychiatry must be questioned very seriously (p.23).

Psychologist Lucy Johnstone [119] wrote about ECT:

Early animal studies provided unequivocal evidence of brain damage, and indeed it was openly admitted by psychiatrists that this was the mechanism of improvement, and that the patient, "secures his readaptation to normal life at the expense of a permanent lowering of functional efficiency". Numerous studies, whose results are not reported in the official journals, confirmed widespread and often permanent

impairment in a range of cognitive abilities, even after standard length courses of ECT ... An appalling abuse is going on in our midst: thousands of people a year are having brain damage inflicted on them in the name of "treatment". Psychologists are in a uniquely favourable position to publicize and protest against this.

8.3. *Survivors of ECT*

Survivors of ECT have become an increasingly active political and moral force. They are members of what is called the psychiatric survivor movement in the United States.⁽⁴⁾

As already noted, some of the most significant scientific reviews have been written by individuals who have been harmed by ECT. In addition to writing and appearing in the media, many who have undergone ECT continue to protest at national psychiatric conventions and electroshock symposia. Some have even chained themselves to the gates and doors of hospitals that carry out electroshock treatment. At Canada's Clarke Institute, for example, they held a candlelight vigil [166]. Most recently, a new organization of several hundred ECT survivors -- the National Association of Electroshock Survivors (NAES) -- has been formed in Texas. It is calling for a ban on ECT. This kind and degree of consumer resistance against a commonly used medical treatment seems unprecedented.

The most dramatic threat to ECT became known as the "Berkeley ban". Ted Chabasinski, who had been subjected to electroshock as a child, organized a grassroots citizens' movement in support of a referendum to ban ECT in Berkeley, California. After the proposition was overwhelmingly approved by the electorate, the psychiatric establishment, led by the APA, intervened and had the ban overturned in court. But the survivors could claim a partial victory -- a "power outage" of 41 days at Herrick Hospital, the city's only ECT facility, in the winter of 1982.

Leonard Frank [95], a survivor of ECT, wrote:

If the body is the temple of the spirit, the brain may be seen as the inner sanctum of the body, the holiest of holy places. To invade, violate, and injure the brain, as electroshock unfailingly does, is a crime against the spirit and a desecration of the soul.

Writing in the British journal, *Openmind*, Jan Wallcraft [175] wrote:

ECT may effectively silence people about their problems... It may fulfill a socially-valued function in reinforcing social norms and returning people to unhappy or abusive situations, or to isolation and poverty without any expenditure on better services or community development. It is easier to numb people and induce forgetfulness than to try to eradicate poverty, provide worthwhile jobs and deal with people's demands to be listened to, understood, loved and valued as part of the community.

Wallcraft had been subjected to ECT at the age of twenty-two [174].

8.4. *Legislation*

Recently California again became the center of public controversy surround electroshock. Inspired by a

coalition of former patients and concerned professionals, Angela Alioto, a member of the San Francisco Board of Supervisors, held hearings on ECT. About two dozen "shock survivors" testified about permanent damage to their brains and minds. Although both sides had ample time to organize, no former ECT patients showed up to offer testimonials in favor of the treatment (40,41,93).

The recommendations of Alioto's committee were adopted by the city's governing body and signed by Mayor Art Agnos on February 20, 1990. The resolution declares the Board of Supervisors' opposition to the "use and financing" of ECT in San Francisco [78]. It also calls for the state legislature to develop more strict requirements for informed consent, including the exposure of potential patients to live or videotaped presentations by critics of the treatment. The resolution, which follows the recommendations made in my testimony at the Alioto hearings, is not legally binding. While the resolution has been an important moral and educational victory for opponents of ECT, its actual impact may be negligible.

More than 30 US states have passed legislation to monitor ECT, set limits on the number of treatments or the age at which it can be given, and require second opinions and informed consent. Four states have banned its use on children, most recently Texas, under NAES leadership. While efforts to require informed consent have proved almost impossible to enforce in the face of psychiatric resistance, they have raised further questions about the use of ECT.

8.5. The Food and Drug Administration (FDA)

In 1979, the FDA classified ECT devices as demonstrating "an unreasonable risk of illness or injury" [89]. This would have required animal testing for safety. However, under pressure from the APA, the FDA gave notice of its intent to reconsider its original decision and to reclassify ECT machines as safe. The APA's most recent Task Force report was timed to come out in the midst of the FDA's political squirming over ECT.

The FDA's final report [89] reads remarkably like the APA's 1990 report. Although no large animal studies have been done with ECT devices since those earlier studies consistently demonstrated brain damage, the FDA panel has now recommended defining ECT devices as safe for depressed patients. It did so ambivalently, recommending that the approval be delayed until the establishment of engineering safety standards for the machines. The approval process continues to be delayed by the lack of approved standards and ECT exists in a kind of FDA limbo which has not discouraged psychiatrists from using it.

Through the Freedom of Information Act, I have obtained and reviewed what the FDA has made available as its complete file on ECT. There are dozens of recommendations from state-funded and private patient rights and advocacy groups to ban ECT, and hundreds more from patients who feel they have been permanently damaged by the treatment. It is astonishing that the FDA has ignored or rejected such an avalanche of official recommendations and personal reports and protests.

In approving the ECT machines as potentially safe, the FDA ignored a most remarkable situation. Before being put on the market, the ECT machines, such as the commonly used MECTA, were not tested for safety on animals or humans. There were no systematic or controlled studies to evaluate their impact on the living brain. The FDA has simply accepted the lobbying campaign of organized psychiatry that ECT is safe and effective.

After hearing evidence presented to the Food and Drug Administration's Respiratory and Nervous System

Device Panel, consumer representative Susan Bartlett Foote [90] reported back to the FDA:

Evidence of the safety and efficacy of ECT devices remains controversial and conflicting. The "new evidence" submitted [by the American Psychiatric Association] petition did not, by any means, eliminate the unanswered or troubling questions surrounding safety and efficacy of the machines.

8.6. The politics of the 1990 APA report

The political nature of the APA task force report (1990) is reflected in the membership of the panel that wrote it. The chairperson, Richard Weiner, was APA's official representative in defense of ECT at the FDA hearings, and has for some time been APA's chief spokesperson on the subject. Two of the other six members are psychiatrist Max Fink and psychologist Harold Sackeim, among the nation's most zealous promoters of the treatment. Together, the three travel the world touting ECT (for example, see [107]). Fink [85,86] is currently pressing to increase the use of electroshock treatment for children and adolescents. Sackeim and his colleagues, as already described, are calling for the use of increased doses of ECT and even for new machines that will greatly escalate the electrical energy delivered into the patient's brain.

By contrast, the task force sought no input from the several patient organizations that oppose the treatment, and none from psychologists, psychiatrists, neurologists, and other professionals who are critical of it.

The APA task force report in its acknowledgments thanks the manufacturers of electroshock machines for their contributions; company advertising handouts are listed as useful sources of public information; and the names, addresses, and phone numbers of these companies are provided in the report. The task force is particularly positive toward Somatics, Inc., whose sole function is to manufacture the electroshock machine. Thymatron, Somatics, Inc., is acknowledged for providing "input into the guidelines". Under "Materials for Patients and Their Families" the task force cites a pamphlet by Richard Abrams and Conrad Swartz and a videotape by Max Fink, both of which are advertising materials for Thymatron and can only be obtained by writing to the manufacturer.

The report nowhere mentions any link between Thymatron and Richard Abrams, who would appear to be the task force's most valued expert. One of Abrams's articles is recommended under "Materials for Patients and Their Families" and another under "Materials for Professionals". Nine of his publications are cited in the report's general bibliography, making him by far the most heavily represented author. Abrams is also listed among those individuals who "provided comment on the draft of the ECT Task Force Report". However, his most interesting affiliation is absent: Abrams owns Somatics, Inc.! In a deposition in which he was a medical expert [67], Abrams acknowledged under questioning that Somatics, Inc., is the source of 50% of his income.⁽⁵⁾ On the book jacket (but not in the text) of the 1997 edition of his book, Abrams now acknowledges that he is president of the shock manufacturing company.

The 1978 APA task force report labeled electroshock treatment as controversial. The 1985 Consensus Conference report stated, "Electroconvulsive therapy is the most controversial treatment in psychiatry" and referred to forty-plus years of dispute surrounding "issues such as efficacy and complications. In the opening sentence of the introduction to Abrams' 1988 book, Fink referred to the "more than 50 years of controversy" surrounding ECT.

By contrast, the 1990 APA task force report says not a word about controversy. ECT is presented as if no one in the profession has ever criticized it. Since a number of psychiatrists have been sued for failing to inform patients about the controversial nature of the treatment, the APA report was in part intended as a step toward cleansing the treatment of controversy.

Psychosurgery remains the only treatment surrounded by more controversy than ECT; but it is used much less frequently [46]. The two treatments are closely related in many ways. Electroshock can be accurately described as closed-head electrical lobotomy.

9. What to tell patients who have been injured by ECT

When I first began to evaluate post-ECT patients early in my career, I was often hesitant to confirm their fears that the treatment had injured their minds and brains. Even though they were telling me that they felt permanently injured, I was reluctant to confirm their personal tragedy. However, I have found over the years that patients who have been iatrogenically injured are almost always grateful to have a doctor confirm their suspicions and fears. Even if we, as physicians, find it painful to inform our patients about iatrogenic injuries, we have a solemn duty to communicate the truth to those who put their trust in us. To do anything else is not only paternalistic, it is dishonest and potentially fraudulent.

Mental health professionals should be advised that it is both ethical and beneficial to acknowledge to patients in a supportive, empathic manner that they have been injured by ECT or by an other medical treatment. Many electroshock survivors have told me that reading my papers and books about ECT, or consulting directly with me, has been a life-affirming experience for them. Instead of reacting with more despair to the confirmation of their ECT-induced brain damage and disability, they have felt understood and empowered for the first time.

10. The need to ban ECT

10.1. The persistent failure to provide informed consent

The 1990 APA task force report became a disillusioning and disappointing watershed for my own reform activities around ECT. I have long argued that ECT is an ineffective, dangerous, anachronistic treatment that should be abandoned by modern psychiatry. Yet, despite the urging of many victims of ECT, I refused for many years to endorse public or legislative efforts to ban it. I believed that the practice of medicine and the rights of patients were better served by insisting on informed consent, while holding liable those psychiatrists who fail to convey to their patients the controversial nature of ECT and its potentially damaging effects. Unfortunately, the 1990 APA report and the APA's political pressuring of the FDA demonstrated organized psychiatry's determination not to inform professionals or patients about the risks of ECT. Despite the disclaimer tucked away on its copyright page, the APA report provides a shield for those who recommend and administer ECT -- an "official" conclusion that there is no serious risk of harm. Doctors who prescribe or recommend ECT now hide behind this report when their injured patients protest to them or bring legal actions.

In the environment created by the APA, informed consent for ECT has become a mirage. Therefore, after much hesitation, I recently endorsed public efforts to ban ECT. The banning of ECT should be supported

by all concerned mental health professionals.

Some patients do feel "helped" by ECT. Often they have been so damaged that they cannot judge their own condition. They suffer from iatrogenic denial and helplessness. But should a treatment be banned when some people believe they are helped by it? In fact, it is commonplace in medicine and psychiatry to withdraw from use treatments and devices that have caused serious harm to a small percentage of people, even though they may have helped a very large percentage. The risk of serious injury to a few outweighs helping many.

In the case of ECT, a large percentage of people are being harmed, and there is very little evidence that many are being helped. There is no evidence that ECT prevents suicide or rescues desperate cases. At best ECT offers a very poor trade-off -- potentially irreversible brain damage and mental dysfunction in exchange for the docility and temporary emotional blunting or euphoria that result from the damage.

If ordinary medical ethics were applied to psychiatry, ECT would have been abandoned or prohibited by the late 1950s based on the original large-animal studies. Prior to trying ECT again on humans, ECT advocates would have been required to conduct newer, similar animal studies to prove that modern ECT is safer. However, the possibility that modern ECT is safer is practically nil, since the doses of electrical energy are uniformly higher today than they were in the animal experiments. Higher doses are required in order to overcome the effects of the anesthesia used to sedate the patients prior to ECT. Often the patients are using sleeping medications or daytime tranquilizers that can also raise the seizure threshold. Furthermore, as we have seen, there is an increasing tendency to advocate and to administer even larger doses of electrical energy -- up to 2.5 times the amount required to produce the seizure. ECT is not safer than it was when brain damage was originally demonstrated in elegant animal studies, and it is not going to become safer in the foreseeable future. It should be banned.

10.2. The personal cost to survivors

It is impossible to find words that are sufficient to communicate the tragic personal cost to many of the patients who undergo ECT. In my own experience, spanning more than thirty years, I have encountered dozens of individuals whose lives have been wrecked by the effects of ECT on their mental function (described, for example, in [32.39.45]). Many have been left with such devastating retrograde amnesia that they can no longer function as professional persons or homemakers. Years of professional training and other key aspects of their lives have been obliterated. Even portions of their past that they can remember may seem remote and alien as if they are watching a movie rather than recalling their own lives. Often they have been impaired in their ongoing ability to focus or pay attention, to concentrate, to make sense out of complex situations, to remember names and places, to learn anything new, to find their way around, and to read and think effectively. Frequently they have become irritable and easily frustrated, emotionally unstable, and shallow in their ability to feel. Often they feel depressed and even suicidal over the loss of their mental function. In short, they have shown all the typical signs of close-head injury, including frontal and temporal lobe dysfunction. Often their families have been irreparably damaged by their inability to function as wage earners, husbands or wives, mothers or fathers. A treatment that can cause such devastation, while producing such limited and questionable results, has no place in the practice of medicine.

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2. Peter R. Breggin, M.D., is director of the International Center for the study of Psychiatry and Psychology (ACSPP), 4628 Chestnut Street, Bethesda, Maryland 20814. Further information about Dr. Breggin, ICSP, electroshock and other psychiatric treatments can be found on two Web sites: www.breggin.com and www.ICSP.org.

3. Devanand is one of the authors in Sackeim et al. [154] calling for the use of intensive ECT using 2.5 times the electrical current required to produce a convulsion.

4. To contact the international psychiatric survivor movement: David Oaks, Publisher, Dendron, P.O. Box 11284, Eugene, Oregon, 97440, E-mail: dendron@efn.org.

5. I was a medical expert for the plaintiff in this case and advised the plaintiff's attorney to question Abrams under oath about his economic involvement in manufacturing ECT machines.

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A CRITICAL REVIEW OF THE CONTROLLED
REAL VERSUS SHAM ECT STUDIES
IN DEPRESSIVE ILLNESS

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SUMMARY

The thirteen published controlled real versus sham ECT studies in depressive illness are critically reviewed. The criticisms of the studies are analysed along a wide range of parameters. The authors conclude that all of the studies can be criticised on both numerous parameters and important parameters. Moreover, irrespective of any criticisms, the reported data at the end of the controlled phase of the studies and subsequent follow-up data, as a body of evidence, does not in the opinion of the authors significantly indicate that real ECT is more effective than sham ECT in treating depressive illness.

INTRODUCTION

ECT was introduced by Cerletti and Bini approximately 50 years ago and since then it has gradually become established as a major physical treatment in psychiatric practice. For many years the use of ECT was heavy and indiscriminate but clinical experience and research subsequently suggested it to be most effective in depressive illness. Indeed the present authors GS and SA employ ECT in their clinical practice and can attest to its effectiveness in saving life in severe depressive illness - associated for example with a refusal to eat or drink. However, despite the consensus that ECT is effective in depressive illness, there has been a fundamental question as to which part of the treatment is therapeutically active. Is it the passage of electrical current through the brain or is it the non-specific placebo effect associated with the ECT procedure that is the crucial therapeutic agent?

Attempts to answer this question have been made through conducting controlled studies, in which one group of patients receive real ECT and a comparison group receive sham ECT (sham ECT involves all the procedures associated with real ECT except the passage of electricity through the head). For a full description of the studies see Tables I, II and III. These controlled 'real' v 'sham' ECT studies can be divided into two historical groups:

1. Those completed between the late 1950s and late 1960s (Ulett et al 1956, Brill et al 1959, Sainz 1959, Harris & Robin 1960, Wilson et al 1963, Fahy et al 1963 and McDonald et al 1966).

NB These studies are hereafter referred to in the text by numbers 1 to 7

respectively. For the precise numbering of each study see any of the tables (I-VI).

2. More recent studies which have been completed between the late 1970s and through the 1980s (Freeman et al 1978, Lambourn & Gill 1978, Johnstone et al 1980, West 1981, Brandon et al 1984 and Gregory et al 1985). NB These studies are referred to in the text as numbers 8 to 13 respectively. For the precise numbering of each study see any of the tables (I-VI).

This paper critically evaluates the thirteen controlled real v sham ECT studies from multiple standpoints e.g. staff/patient blindness, entry requirements for patients and outcome analyses etc. Some of the criticisms point to the possibility of a Type I error: that the null hypothesis was falsely rejected and therefore that any 'positive' result indicating that real was better than sham ECT was less certain. Other criticisms point to the possibility of a Type II error: that the null hypothesis was falsely accepted and therefore that any 'negative result' indicating there was no difference between real and sham ECT was also less certain. The criticisms in this paper (see Tables IV, V and VI) will be divided into the three sections listed below and these sections will then be discussed in turn:

A) 'PRETRIAL' PARAMETERS OF CRITICISM

1. Population used to derive the study sample.
2. Defining the study sample (including the number of patients in the sample).
3. Demographic variables of the sample e.g. age and sex.
4. Parameters related to the depressive illness e.g.

5. Matching of the two groups for pretrial parameters.
6. Random allocation to treatment group.

B) ' DURING THE TRIAL' PARAMETERS OF CRITICISM

1. Patient blindness.
2. Staff blindness.
3. Index treatment heterogeneity.
4. Pharmacological accompaniments of ECT study.
5. Number of ECTs.
6. Frequency of ECT.
7. ECT waveform.
8. Electrode placement.
9. ECT energy.
10. Fit ascertained.
11. Matching (on a variety of parameters during the study).

C) ' END OF TRIAL' PARAMETERS OF CRITICISM

1. Matching of the patients who completed the study.
2. Numerical parameters of the outcome analysis (at the end of the controlled real v sham phase of the study).
3. Numerical parameters of the follow up.

NE In the text 'positive result' means real ECT was found to be more effective than sham ECT and a 'negative result' means real ECT was found to be no more effective than sham ECT.

' PRETRIAL' PARAMETERS OF CRITICISM

1. Population used to derive the study sample

To help ensure that a representative and non-biased sample of patients is selected for a controlled real v sham ECT study, the following factors concerning the wider population from which the sample is drawn (hereafter referred to as 'the population') should be taken into account.

a) Definition of the population. Ideally a study should report how many hospitals have participated in the study and from which geographical area the patients were drawn. Three studies (3, 5 and 11) did not give any definition of the population. Another study (8) did not report either the number of hospitals in the study or the geographical area from which the patients were drawn, but did say the population was patients admitted to 'four acute units'. All of the other studies stated how many hospitals had participated in the study; one hospital for seven studies (1,2,4,6,9,10 and 13), two hospitals for one study (7) and many hospitals in another study which incorporated referrals from all the hospitals in one large geographical area (12). This latter study was the only one to define the geographical area which incorporated the population.

b) The population should not be restricted. Ideally the population size should be large. The population should be drawn from a widespread geographical area and referrals from the population to the study should be from many hospitals. On these ideal grounds, all but

one of the studies (12) could, in strict terms, be criticised either because no definition of the population was reported or because there was no record of the population coming from a widespread geographical area.

- c) All patients in the defined population should be screened for inclusion in the study to help prevent biased sampling. Of the ten studies that attempted to define the population (1,2,4,6 - 10, 12 and 13), two of them (1 and 2) did not report all patients were screened, but the others did.

2. Defining the Study Sample

a) Definition of Depressive Illness

An ideal definition of depression is one that employs specified criteria and is one that has been statistically replicated and accepted by other workers. Eight of the studies did not employ such a definition of depressive illness in their studies (1-7 and 9). All of these studies stated simply that the trial patients had been 'clinically diagnosed' as suffering from depressive illness. Such a definition opens the possibility of deriving a non-representative or even a biased sample. Four of the studies (10,11,12 and 13) employed satisfactory definitions (see Table I). The remaining study (Freeman et al 1978) also used a satisfactory definition, despite the fact that it has not been replicated and is not a definition of depressive illness that has the acceptance of others 282

because specified definitive criteria were employed and the quantitative nature of the definition allows for the possibility of replication (see Table I).

b) Selecting a sample with the best chance of responding to ECT

Ideally, to avoid a Type II error, studies should have endeavoured to select a group of patients with depressive illness who would have been the most likely to respond to ECT. Six of the studies (2-6, 8 and 12) did not try to do this. Five of the studies (5,7,9,11 and 13) did however state, in their inclusion criteria for patients' entry into the study, that the patients selected were those who would normally be treated with ECT. The two remaining studies (1 and 10) went further than this. Ulett et al (1956) stated in their inclusion criteria that only those patients who were 'the most likely to respond to ECT' were included, and Johnstone et al (1980) stated that all patients had to satisfy the 'Newcastle criteria for predicting a good outcome from ECT'.

NB It should be noted that Johnstone et al (1980) and Brandon et al (1984) in their controlled studies found that the best predictor of a greater beneficial response to real over sham ECT was the presence of depressive delusions. Brandon et al also found retardation was a good predictor. No real v sham controlled study to date has selected only those depressive patients with delusions and/or retardation. 283

c) Psychiatric diagnostic categories

Ideally the studies would only have utilised patients from one psychiatric diagnostic category : depressive illness. However, two studies used several diagnostic categories (1 and 2). The first of these studies (Ulett et al) included 'involuntional psychotic reactions', 'schizoaffective disorders' and 'acute schizophrenic reactions : catatonic type (first episode)', as well as depressive illness. The second study (Brill et al) included 'schizoaffective type' depressive reactions and several categories of 'schizophrenic reactions'. NB the results of this latter study were impossible to interpret for the patients with depressive illness, because this data was inextricably bound up with the data for the patients with schizoaffective illness (depressive type) -see below.

d) Number of patients

To help avoid statistical error it is important to study a population of sufficient size. Johnstone et al in their controlled real v sham ECT study noted 'a sample of 70 patients would be adequate to clarify the question of whether or not the convulsion is an important element in the therapeutic efficacy of ECT'. In fact all but one study (Brandon et al, which had 71 patients) failed to incorporate 70 or more patients in the final outcome analysis. Two other studies had 70 patients or thereabouts at the beginning of the study, Johnstone et al had 70 and Gregory et al had 69, but after withdrawals during the study the number of patients remaining at the completion were 62 and 44 respectively. 284

In this paper we have arbitrarily selected for special criticism those studies which had a total population at the completion of the study of less than 30 patients. Seven studies (1-4,6,7 and 11) had in fact less than 30 patients. Of these studies three of them (4,6 and 7) had six or less patients in either the real or the sham group. In view of the low numbers in these three studies, meaningful interpretation of the statistics was considered impossible - see below.

3. Demographic variables of the sample

a) Age

There is evidence that older people have a higher seizure threshold, (Sackheim et al 1987). There is also evidence that the greater the stimuli is above the seizure threshold, the greater the therapeutic response (D'Elia & Perris 1970). Therefore studies which include the more elderly patients would decrease the chances of showing a more beneficial effect of real over sham ECT. All of the 13 studies, to a greater or lesser extent, can be criticised on this parameter and therefore they all increased the risk of a Type II error. Seven of them (1-4,6,7 and 13) gave no information on age. Three studies (5,8 and 9) gave age ranges which included more elderly patients (see Table II). The remaining three studies (10,11 and 12) gave only mean ages without the range of ages or the standard deviations; 49.4 years to 54.4 years was the range of the means over the three studies

b) Sex

There is evidence that females have a lower seizure threshold than males (Sackheim et al 1987). There is also evidence, as noted above, that the greater the electrical stimuli is above the seizure threshold, then the greater the therapeutic response (D'Elia & Perris 1970). Therefore a real v sham ECT study which only included females would help maximise the chances of showing that real ECT was more effective than sham ECT. Slightly less ideal studies would be ones in which there was either a significantly greater number of females to males within the real ECT group, or studies in which there were a greater number of females in the real group compared to the sham group. Only two studies included only females (4 and 6). Five studies had a greater number of females to males in the real group (1,3,8,10 and 12). No study had a significantly greater number of females in the real group compared to the sham group. The six remaining studies (2,5,7,9,11 and 13) did not approach the ideals noted above and therefore could be criticised on this parameter of gender, thus running the risk of making a Type II error.

4. Parameters related to the depressive illness

a) Previous depressive illness

It has been suggested (Fahy et al 1963) that the more episodes of depressive illness a patient or a group have had, the less likely the responsiveness to active treatment e.g. real ECT. (One possible way of understanding this

connection between increased frequency of depressive illness and lack of responsiveness to ECT is to look at the established relationship between an increased frequency of depressive illness and a stressful/unsupportive psychosocial environment - see Brown & Harris 1978 - and the relationship between the latter and relative lack of responsiveness to active treatment). If responsiveness to ECT is decreased in the face of repeated episodes of depressive illness then, to maximise the chances of detecting a 'true therapeutic' effect of ECT (and therefore avoid a Type II error) real v sham ECT studies should only investigate people who are experiencing their first episode of depressive illness. Eight of the thirteen studies did not give any data on history of previous depressive illness (1-4,6,7,11 and 13) and therefore the possibility of their being at risk of the Type II error noted above cannot be excluded. The remaining five studies (5,8,9,10 and 12) did give data on history of previous depressive illness. The proportion of patients with previous depressive illness in all these studies was high (see below). Therefore all of these studies were, because of this, also at risk of making a Type II error. The proportion of patients having previous depressive illness in four of the five studies noted above was as follows:

(25% (Fahy et al in Real ECT Group)

(40% (Fahy et al in Sham ECT Group)

66% (Johnstone et al - 66% of all the patients in the study i.e. real and sham ECT patients combined)

individually)

81% (Lambourn & Gill -81% Real and Sham ECT Groups
individually)

The proportion of patients having previous depressive illness in the fifth study (12) could not be directly determined because the only data provided was mean number of previous admissions to psychiatric hospital (2.6 for the real ECT group and 2.5 for the sham group). However these mean figures suggest that the proportion of patients with previous depressive illness was also high in this study. NB Of the five studies noted above which did present data on previous depressive illness, three of the (5,9 and 10) reported either no or little significant difference between real and sham ECT. These studies, therefore, may not only have been at risk of the Type II error noted above but may have committed it.

b) Previous ECT

If patients have had previous ECT, and therefore have become acquainted with its potential acute side effects such as headache, memory disruption etc., then there is in the opinion of the authors a significant chance that they will be able to determine by the presence or absence of previously experienced acute side effects whether they are receiving real or sham ECT. In other words, patients who have had previous ECT will be more able to break the 'code' of a controlled ECT study, in the process undermining their own

in a controlled study, assuming they had benefited from it in the past, then they would experience a greater than 'normal' positive placebo response. ('Normal' placebo response being the placebo response experienced by a patient who remains fully blind in a double blind controlled study). If on the other hand patients who had had previous ECT were to receive sham ECT, again assuming beneficial response to ECT in the past, then they would experience a less than normal positive placebo response. From these arguments it is therefore clear that if a high proportion of a group of patients entering a real v sham ECT study had received ECT previously, then this could produce a differential placebo response between the real and sham groups with a relatively more beneficial placebo response in the real ECT group. This would therefore tend to skew the results of the study in favour of real ECT. In other words a study with a high proportion of entry patients with previous ECT would be at risk of a Type I error. The ideal real v sham ECT study would therefore include a low proportion of patients with a history of previous ECT. Nevertheless if a high proportion of patients with previous ECT were studied, then possibly the only way in which this Type I error could be avoided would be by minimising ECT side effects and therefore minimising the chances of the patient's blindness being undermined e.g. by using unilateral ECT (which has a relatively low incidence of acute side effects) and/or to use low ECT energy levels (which is also associated with fewer acute side effects).

Only four of the thirteen studies (8,9,10 and 12) produced data on previous ECT. Therefore the remaining nine studies, in theory, may have been at risk of the Type I error noted above. Of the four studies that produced relevant data, those of Freeman et al and Brandon et al had a high proportion of patients with previous ECT (55% and 60% respectively for real and sham groups combined). These studies also concluded that real ECT was better than sham ECT and therefore they may not only have been at risk of the Type I error noted above but may actually have committed it. The third of the four studies (Lambourn & Gill) also had a high proportion of patients with previous ECT (66%) but this study would have tended to avoid the Type I error by employing unilateral ECT with low ECT energy (see above).

The fourth study (Johnstone et al) had a satisfactorily low proportion of patients with previous ECT (21%). It is perhaps noteworthy that the two studies which avoided this Type I error of previous ECT the most (i.e. Lambourn & Gill and Johnstone et al), and and therefore achieved the most satisfactory matching of the placebo response in the real and sham groups, showed either no difference between real and sham ECT (Lambourn & Gill) or only a slight difference between real and sham ECT (Johnstone et al). This perhaps begs the question that, if there is satisfactory matching for the placebo response, is there any difference in effectiveness between real and sham ECT?

c) ECT and/or antidepressant medication (prior to study) for the index episode of depressive illness

If ECT and/or antidepressant medication were given for the index episode of depressive illness, prior to the study, then this might tend to lead to some improvement in the patients. This in turn would reduce the scale of any improvement caused by the electrical component of ECT given during the study, thereby reducing the degree of any difference that might have been found between the real and sham groups. This would produce a risk of a Type II error.

i) 'Prior' ECT. Seven of the nine studies produced no information concerning ECT for the index episode of illness prior to the study (1,3,4,6,10,11 and 12). Therefore the risk of the Type II error noted above for these studies cannot be excluded. Four of the other studies stated that, prior to the study, the patients should not have received ECT for at least a stated period of time; 1 month (McDonald et al), 3 months (Lambourn & Gill and also Freeman et al), 9 months (Brill et al). Despite this, these same four studies did not say that ECT was not given for the current episode of illness. (The current episode of illness may have predated the periods of time given above). Therefore these four studies also cannot be considered to be free of the risk of the Type II error noted above. The only two studies that cannot be criticised on this parameter of 'prior' ECT are those of Fahy et al and Gregory et al, which both categorically stated that no patient had received ECT for the current episode of illness.

ii) 'Prior' antidepressant medication. All of the 13 studies can be criticised with regard to 'prior' antidepressant medication. Seven of the studies (1,2,3,4,6,11 and 13) did not give any information relating to this subject - although the latter study (3) did report that at least one patient had had 'prior' antidepressant medication and three had received lithium. Four other studies reported a relatively high proportion of patients that had received 'prior' antidepressant medication (Lambourn & Gill, 40%; Freeman et al, 45%; Brandon et al, 62%; Johnstone et al, 70%). The remaining two studies (Fahy et al and McDonald et al) did report some avoidance of 'prior' antidepressant medication for the index episode but this avoidance fell short of satisfactory avoidance: Fahy et al (5) reported no patients had received the antidepressant imipramine for the index episode and McDonald et al (7) said no patient had received antidepressant medication for the short period of 'at least two weeks' prior to the commencement of the study.

d) Minor tranquilliser medication (pretrial)

Minor tranquillisers decrease endogenous GABA turnover (probably as a consequence of their facilitating effect on GABA transmission). This reduction in endogenous GABA activity persists after the withdrawal of minor tranquillisers (Schoff, 1987). As GABA activity might be correlated to responsiveness to ECT (see below) pretrial minor tranquillisers may significantly affect the outcome of

a real v sham ECT study. All of the thirteen studies under consideration can be criticised because none of them indicated the proportion of the patients who had received minor tranquillisers pretrial for the index episode of illness.

GABA activity and responsiveness to ECT. A decrease in GABA receptor number lowers seizure threshold (see Sackheim et al 1987). A lowering of seizure threshold may increase the effectiveness of ECT, because a given 'convulsive' ECT stimulus would be more supraliminal (i.e. above the seizure threshold) and there is evidence that the greater an electrical stimulus is above the seizure threshold the greater the therapeutic effect (D'Elia & Perris). Therefore, a decrease in endogenous GABA activity brought about by pretrial minor tranquillisers may increase the effectiveness of ECT by lowering seizure threshold- assuming the minor tranquillisers are subsequently stopped during the ECT study. A converse argument is as follows. GABA receptor numbers are increased by ECT (see Sackheim et al, 1987). It is possible that the effectiveness of ECT may in part be related to this increase in GABA activity. Therefore pretrial minor tranquillisers may paradoxically reduce the effectiveness of ECT by reducing GABA activity caused by ECT - assuming the minor tranquillisers are subsequently stopped during the ECT study. From the above two converse arguments, it is clear that, given current knowledge, it is difficult to predict the precise effect of pretrial minor tranquillisers on a real

v sham ECT study - again assuming the minor tranquillisers are subsequently stopped during the ECT study. To add to the complexity of prediction, the above effects of pretrial minor tranquillisers on a real v sham ECT study would be modified if the drugs were continued during the study.

5. Matching of two groups on pretrial parameters

The essence of a controlled study is that the groups being compared are alike in all parameters that might influence the outcome of the study; these parameters are hereafter referred to as 'non-investigatory parameters'. The investigatory parameter(s) are those that are being investigated e.g. the electrical component in real versus sham ECT studies. The process of trying to make the groups as similar as possible with regard to the non-investigatory parameters is known as 'matching'. If real versus sham ECT studies do not include satisfactory matching of the groups then any difference in outcome between the study groups could be ascribed to either the difference in the non-investigatory parameters and/or the difference in the investigatory parameter, i.e. absence or presence of the electrical component of ECT. In other words poor matching does not allow for a clear interpretation of any results. It is the contention of this paper that all the controlled real versus sham ECT studies have significant limitations in matching. Various parameters of matching are discussed below in relation to the controlled ECT studies.

a) Parameters related to the Index episode of depressive illness

The severity and length of the illness, the presence or absence of contemporary stress and support and whether other types of treatment are used pretrial are all parameters that are very likely to influence the effect of any subsequent treatment of depressive illness, including ECT. As an example of this, a short duration illness, in the absence of adverse psychosocial factors and for which 295

antidepressant drugs have been used is more likely to respond to ECT and less likely to show relapse after treatment during any subsequent follow-up. Listed below are a number of 'matching' parameters related to the index episode of depressive illness.

i) Length of illness. A short period of depressive illness might reasonably be expected to be associated with a greater likelihood of response to any treatment, including ECT (Hobson, 1953) than a long period of depressive illness. None of the studies, with the possible exception of Gregory et al (13), indicated there was matching for length of current illness; Gregory's study simply stated there was satisfactory matching for 'previous illness' but did not specifically say there was matching for the length of current illness.

ii) Severity of illness. This is an important factor to match for, for at least two related reasons: 1) there is evidence that the more severe a depressive illness the greater the likelihood of a response to ECT (Nystrom, 1965) and, 2) the greater the severity of depressive illness the greater the degree of improvement likely to occur, compared to less severe depressive illness, simply because there is more 'room' for improvement. Five of the thirteen studies did not give evidence of satisfactory matching on this parameter (1-5).

iii) Previous ECT (for index illness). ECT given for the index illness prior to the study would also be

brought about by ECT given during the trial. Eight of the studies did not present evidence of satisfactory matching for this parameter (1,3,4,6,7,10,11 and 12).

iv) **Medication (for index illness).** Clearly in a study which is investigating a physical treatment (ECT) for depressive illness, it is important to match for other physical treatments given for the index episode of depressive illness prior to the study. Eleven of the studies did not match satisfactorily for medication, prior to the study; (1-8, 10,11 and 12). Two studies (9 and 13) made a good but not ideal attempt to match for medication. In the first of these studies (9) evidence was presented for satisfactory matching for anti-depressant medication alone. In the second study (13) the authors simply stated that there was a satisfactory matching for 'previous treatment' without specifying whether this included previous medication.

Matching for pretrial anti-depressant medication might be particularly important because pretrial anti-depressants may decrease the scale of any improvement brought about by ECT given during the study. This would therefore increase the risk of a Type II error. Only one study definitely matched for pretrial antidepressant medication (9). In one other study (13) there may have been matching for pretrial antidepressants (see above), but the text of the paper did not allow for a definite opinion on this.

v) **Contemporary stress and contemporary sources of support.**

aetiological factor in depressive illness (Paykel et al 1969) and that some social support can reduce the likelihood of depressive illness (Brown & Harris 1978). The presence of a high degree of stress and the absence of good social support, contemporary with an ECT study, might therefore be expected to reduce the responsiveness to ECT. None of the studies matched for these two parameters.

b) Previous Depressive Illness

This parameter is an important one to match for because it could be argued that the more pronounced a patient's previous history of depressive illness, the less likely they will be to respond to any treatment including ECT and also the less likely there will be a positive placebo response. To match satisfactorily for depressive illness a study should mention that there was matching for the following three components of the 'previous depressive illness' parameter: number of patients with a previous history of depression; number of episodes of depressive illness, and total duration of depressive illness. Ideally a study would produce quantitative evidence of matching for these three components. Nine of the studies did not give any indication of matching for previous depressive illness. Furthermore none of the remaining four studies (8,9,12 and 13), that all attempted matching on this parameter, described satisfactory matching on all the three components of previous depressive illness noted above. One of these four studies (13) said simply patients were matched on 'previous depressive illness'. This study was given the benefit of doubt in

assessing satisfactory matching on the three components noted above for the purposes of the matching table (Table V). Two of the four studies noted above (8 and 9) produced evidence of satisfactory matching for two of the three components: 'number of patients with previous depressive illness' and 'number of episodes of depressive illness'. The final study (12) produced satisfactory matching on one component only: 'the number of episodes of depressive illness'.

c) Previous History of ECT

There are three components to consider when matching for this parameter: number of patients with previous history of ECT; number of ECT treatments; and response to ECT in the past. Matching for previous history of ECT is important for three reasons:

- 1) Patients with a greater frequency of previous ECT might be more able to determine whether ECT was real or sham. This would jeopardize the blind nature of the study and therefore create a potential mismatch in the placebo factor between the two study groups - see above.
- 2) It might also be predicted that patients who had responded well to ECT in the past would be more likely to respond satisfactorily to real ECT in the study.

3) Patients with a greater frequency of previous ECT (and therefore patients more likely to be aware of which treatment they were receiving because of knowledge of side effects etc.), who had also responded well to ECT in the past, would be particularly liable to have a pronounced positive placebo effect from real ECT.

Nine studies (1-7,10 and 11) did not provide matching for any of the three components of the 'previous history of ECT' parameter noted above. Of the remaining four studies (8,9,12 and 13): two matched on only one of the components - 'number of patients with previous ECT' (8 and 11); one of the studies (9) satisfactorily matched for this component and also for 'number of previous ECT treatments'; and the final study (13), although it produced no definite evidence of matching for any of the three components noted above, was given the benefit of doubt for the purpose of the matching table (Table V) because it described adequate matching for 'previous treatment' - although the types of previous treatment were not specified.

d) Age

It is important to match for age for at least one reason: that elderly patients have a higher seizure threshold (see above). Six of the studies provided no data on age to allow a determination of satisfactory matching (1-4,6 and 7). Two studies provided insufficient information to assess the adequacy of matching (10 and 12). The remaining five studies (5,8,9,11 and 13) did provide satisfactory evidence of matching for age.

e) Sex

It is important to match for sex for at least one reason: females have a lower seizure threshold than males (see above). Three studies either provided no or insufficient information to determine whether patients were matched on sex (3,7 and 10). One study provided evidence of less than satisfactory matching on sex (12). The remaining nine studies did provide satisfactory evidence of matching.

6. Random allocation to treatment group

To help eliminate any bias in the placing of patients in the different treatment groups, the allocation of patients should follow a random design. All of the studies had such a design with the exception of Ulett et al, which incorporated the slightly less satisfactory design of 'matched randomness'. In this latter study, each new patient was matched if possible to another patient already allocated to one of the four treatment groups and then this new patient was randomly allocated to one of the three remaining treatment groups; if the new patient could not be satisfactorily matched to a previously allocated patient, then this patient was randomly allocated to one of the four treatment groups.

' DURING THE TRIAL' PARAMETERS OF CRITICISM

1. Patient Blindness

In controlled real versus sham ECT studies it is fundamentally important to ensure patients are unaware of the treatment they are receiving (i.e. real or sham ECT). This is known, of course, as ensuring 'patient blindness'. If the patients are not blind to the treatment received, then the patients receiving the active treatment (real ECT) might be expected to have a more positive placebo response compared to patients receiving sham ECT. This would then increase the chances of a 'positive result', i.e. real ECT being shown to be more effective than sham ECT. In other words studies which do not maintain patient blindness would risk a Type I error.

Two of the studies (2 and 5) clearly did not ensure patient blindness. The remaining 11 studies, from the evidence of their texts, aimed for and felt they had achieved patient blindness. However, it is the contention of the present authors (GS and SA) that these studies did not adequately protect patient blindness because of the intrinsically greater incidence of side effects associated with real ECT compared to sham ECT. If the authors are correct in this assertion then none of the 13 studies can be said to have adequately ensured patient blindness.

The evidence that there are more side effects with real ECT compared to sham ECT is presented below. This is followed by possible mechanisms through which patients might have become aware of the greater incidence of side effects associated with with real ECT.

a) Evidence for greater incidence of side effects with real ECT

A number of side effects can occur immediately after ECT, including the following - headache, confusion (Kendell 1981) and memory disturbance (Kendell 1981 and Weeks et al 1980), nausea, stiffness and aching of the muscles (Kiloh 1962).

In theory any acute side effect of real ECT could be produced by either the passage of electricity and/or the pharmacological accompaniments of ECT i.e. anaesthetic drugs, neuromuscular relaxing drugs and any pre-medication. Arguably the best way of differentiating between side effects produced by the electrical component and the side effects produced by the pharmacological or psychological components of ECT is to look at the differences in side effects produced in real and sham ECT studies. Only one study (Johnstone et al 1980) has done this and they concluded that the electrical component is largely responsible for the acute memory disturbance. Other evidence that the electrical component is involved in ECT side effects is as follows.

The issue of whether ECT produces memory impairment is a complex one. In theory ECT could produce either acute and/or persistent disruption in memory. The following three components of memory could be impaired either acutely or persistently: memory for events immediately preceding and following ECT ('ECT event-related memory'); memory for remote events occurring well before ECT ('remote memory') and general memory function as measured by standard

function etc. ('general memory function'). A discussion on the three components of acute memory disruption and persistent memory disruption is given below.

i) **Acute memory disruption.** It is widely accepted that ECT disrupts 'ECT event-related memory' (see Weeks et al 1980). For acute 'general memory function' the evidence is somewhat contradictory. Johnstone et al (1980) in a controlled study comparing real and sham ECT concluded that ECT caused a disruption in acute general memory function. However, Weeks et al (1980) in a controlled study comparing a combined bilateral and unilateral ECT groups with a group receiving anti-depressant pharmacotherapy did not find evidence in the combined bilateral and unilateral ECT group of a disruption in acute 'general memory function' and indeed reported that overall there appeared to be a small improvement. However, on close analysis it appears that the bilateral group did show a deterioration in memory but this effect was more than offset by an improvement in memory with unilateral ECT. Another study comparing three different types of real ECT, using different treatment energies and waveforms, did not reveal any deterioration in acute 'general memory function' as measured by comparison with pre-ECT memory scores (Warren & Groome 1984); indeed there was a significant improvement in the acute general memory scores. Finally there is also a conflict of evidence concerning the effect of ECT on acute 'remote memory'. Weeks et al (1980) failed to demonstrate impairment but Squire et al (1981) did report impairment in remote

memory at one week after ECT.

ii) **Persistent memory disruption.** There is evidence from the subjective reports of patients who have received ECT that persistent impairment in memory can occur. For example Squire & Chace (1975) reported that six to nine months after bilateral ECT, 63% of patients reported a deterioration in memory compared to 30% of patients who had received unilateral ECT and 17% of controls. In another study (Freeman & Kendall 1980), completed at least one year after ECT, 28% of patients reported their memory had been permanently affected by ECT - mainly by producing permanent gaps around the time of treatment. The objective evidence of ECT induced persistent impairment in memory is less clear cut. Evidence from controlled studies suggests 'general memory functioning' is not impaired (Johnstone et al 1980, Weeks et al 1980, and Warren & Groome 1984). Impairment in remote memory is not apparent for personal events but may be present for public events (Weeks et al 1980 and Squires et al 1980). However, there is evidence of persistent impairment in 'ECT event-related memory', both retrograde and anterograde in type (Squires et al 1980).

Turning away from ECT's effect on memory to another ECT side effect, confusion, there is evidence from bilateral ECT v unilateral ECT studies and also sinusoidal waveform ECT v pulsed waveform ECT studies (e.g. D'Elia et al 1974 and Weaver et al 1975) that post-ECT confusion is, at least in part, caused by the electrical component of ECT.

- b) Mechanisms through which patients might have become aware of the greater incidence of side effects with real ECT compared to sham ECT.

i. Patients who have had a history of previous ECT. These patients, perhaps, would be particularly able to differentiate between real and sham ECT in a controlled study, because of their ability to recognise similarities or dissimilarities in their experience of previous ECT, and their experience of real or sham ECT in the study.

ii. Patients with no previous history of ECT. Patients might well have been informed of the possible side effects of real ECT by the doctors obtaining their consent to receive ECT. Brandon et al (12) in their study, for example, stated that 'written consent to electroconvulsive therapy was obtained by the consultant in the usual way'. The 'usual way' of obtaining consent for ECT includes an explanation of the possible side effects of real ECT. Also patients might have gained knowledge of the side effects of real ECT from other patients on their ward; all the studies investigated in-patients with the exception of Lambourn & Gill's study (9) in which 2/32 patients were outpatients. There is of course no doubt that research in a ward excites a lot of interest and conversation among patients and staff and the patients in the study would have become a focus of interest to other patients. In the patient-patient conversations, resulting from this interest, there would have been a real risk of information being relayed to the patients

in the study regarding the side effects of real ECT. Finally, the sheer presence of a greater incidence of side effects with real ECT would have led to a greater statistical chance of patients with no previous history of ECT guessing which form of ECT (real or sham ECT) they had received.

The essence of this section on patient blindness is that in controlled real v sham ECT studies the presence of increased side effects with real ECT compared to sham ECT enhances the therapeutic effectiveness of real ECT compared to sham ECT by 'disclosing' the trial code to the patients. There is also anecdotal (non-controlled) evidence that a greater incidence of ECT side effects might affect therapeutic response, e.g. in a circular distributed by Ectron, the largest UK manufacturer of ECT machines to psychiatric hospitals throughout the UK in December 1985, it is stated that Ectron's 'early generation' of 'constant current' ECT machines, which were designed to 'achieve minimal side effects', had achieved 'inadequate clinical response' despite the fact that convulsions had been induced. They went on to say that their next generation of constant current machines, which would be designed to deliver more electrical energy (and therefore increase the risk of side effects) should 'ensure a good clinical response'.

2. Staff Blindness to Study Design

In any placebo controlled study of treatment in psychiatric illness it is clearly of central importance that the staff are unaware of whether patients are receiving the active treatment (e.g. real ECT) or the placebo treatment (e.g. sham ECT) i.e. the

staff should be 'blind' to the trial code. If the staff are not 'blind' to the treatment received, then their knowledge of the trial code might be consciously or inadvertently communicated to the patients. This might influence and therefore prejudice the patients' response to the treatment. Furthermore lack of staff blindness might influence the staff's assessment of the patient's response to treatment. There are three groups of staff personnel that need to be 'blind': the doctors responsible for the clinical care of the patients during the study; the nursing staff responsible for the clinical care of the patients during the study, and the staff responsible for completing the formal assessments of the patients to gauge response to treatment. Seven of the studies did not, in the opinion of the authors, provide satisfactory evidence of staff blindness (1,3,4,5,6,7 and 9). Each of these seven studies will now be examined in detail. Two of the studies (3 and 5) did not provide satisfactory evidence for blindness in any of the three staff groups. These studies make no mention of any attempts having been made to achieve staff blindness. It should be noted however that some degree of blindness was at least sought in the assessors in one of these studies (5), in which qualitative assessments by ward staff (who were not described as blind) was coded (and therefore quantified) by two other investigators who were blind to the study design. In two other studies (4 and 6) there was clear and unequivocal evidence of lack of blindness in one of the three staff groups. In Harris & Robbin's study (4) the 'clinicians knew' which patients had received sham ECT and which real ECT. Also in this paper it

was impossible to determine whether the ward nursing staff were blind. In Wilson's study (6) one of the three raters was aware of the study design and the data from this rater was merged with the other raters' data for the purposes of data analysis and not presented separately. In two other studies (1 and 7) there was a lack of unequivocal evidence of staff blindness in at least one of the three staff groups noted above, but there was inferential evidence that blindness might have been achieved. In McDonald's study (7) no unequivocal evidence was presented of blindness in any of the three staff groups noted above, but the statement that 'ECT and simulated ECT were administered by a special team made up of members of staff in no way responsible for the regular care or assessment of the patients' suggests that staff blindness was sought. In Ulett's study (1) no unequivocal evidence of ward nursing staff blindness was presented, but staff blindness was clearly thought of as witnessed by their statement that 'group assignment was made by one of our staff members who had no clinical contact with the patient'. In the remaining study which did not achieve satisfactory staff blindness (9), no information was provided to determine whether the nursing staff were blind. The remaining six studies (2,8,10,11,12 and 13) did provide sufficient and satisfactory evidence of staff blindness. However in one of these studies (11) doubts have been expressed about the extent to which blindness was achieved (Brandon et al 1984). Moreover, in an earlier publication, a research worker associated

randomisation and the administration of one of the assessment scales (West 1981).

3. Index Treatment Heterogeneity

Ideally, real v sham ECT studies should investigate only one index treatment: ECT. This allows for an ideal concentration on a single variable that will affect the outcome of the study : the electrical stimulus, which accompanies real ECT but does not accompany sham ECT. However, two studies had concomitant placebo controlled antidepressant studies intermingled with the real versus sham ECT study (4 and 6). In these studies the real and sham ECT groups received a placebo antidepressant at the same time as ECT. Two additional studies (5 and 7) included a concomitant antidepressant study which was parallel to and not intermingled with the real versus sham ECT study: the real and sham ECT groups did not receive placebo or active antidepressant treatment but additional groups in the study did receive active or placebo antidepressant medication. One further study (1) also included two additional treatment groups: a photoconvulsive group and a subconvulsive photic shock group.

4. Pharmacological Accompaniments of ECT

a) Antidepressant Medication

If antidepressants are given during a real v sham ECT study then this would tend to reduce depressive symptoms. This would in turn reduce the scale of any further improvement in depressive symptoms caused by the electrical stimulus

accompanying real ECT. Therefore if antidepressants are given during a real v sham ECT study then this would reduce the chances of detecting any significant difference between real and sham ECT i.e. there would be a risk of a Type II error. Four of the studies can be criticised on the grounds of not providing any information about antidepressants given during the study (1,2,3 and 6). Two further studies can be criticised because antidepressants were given during the study (8 and 11): in West's study (11) all patients received amitriptyline 50mg nocte and in Freeman et al's study (8) 50% of the sample received antidepressants. The remaining seven studies (4,5,7,9,10,12 and 13) either did not allow antidepressants in the study or in the case of study 13 only allowed them in a small proportion of patients: 4 out of 69.

b) Benzodiazepine/Barbiturate Medication

Benzodiazepines/barbiturates raise the seizure threshold (see Sackheim et al 1987) and limit seizure duration (Sandstromgren et al 1980). Both of these effects may reduce the therapeutic efficacy of real ECT. (For a discussion of the possible relationships between seizure threshold/seizure duration and responsiveness to ECT see Kirstein and Ottosson 1960 and also Robin and De Tissera 1982; note however that Johnstone et al, 1981 have argued that a relationship between seizure duration and therapeutic effect has not been proved). If concomitant benzodiazepine/barbiturate medication reduces the

tend to reduce the degree of any difference between the two forms of ECT and thereby increase the risk of a Type II error. Six of the thirteen studies used anticonvulsants (benzodiazepines and/or barbiturates) during the study (4,5,9,10,12 and 13) and therefore were at risk of this Type II error. Two of these six studies gave 'negative results' (5 and 9) and a third study (10), although showing that real was statistically more therapeutic than sham ECT, gave a result which was less strongly positive than others expected. The results of these latter three studies might therefore have been compromised by the potential Type II error noted above.

5. Number of ECTs

Probably the most commonly used number of ECTs in a clinical course of treatment is between 6 and 8. This was also the most frequently used treatment course among the thirteen controlled studies under discussion; seven of them used between 6 and 8 treatments (5,6,9-13). It has been reported that some depressed patients require a greater number of ECTs. Furthermore Kiloh (1982) has said that the number of ECTs should be continued if necessary beyond 20 before abandoning hope. Therefore it might be argued that to maximise the chances of detecting a beneficial effect of real ECT compared to sham ECT and therefore avoid a Type II error, the number of ECTs should be greater than 6 to 8. Similarly it could be said that studies which employed less than the average number of treatments of 6 to 8 might be particularly at risk of this

less than 6 ECTs. However, despite the fact that using the above line of argument they were at risk of a Type II error, neither committed this error - as their results suggested real ECT was significantly better than sham ECT. The results of the studies which gave the longest courses of ECT (1,2,3 and 7), twelve or more ECTs, did not indicate that their patients showed a greater responsiveness to real ECT than the patients in the remaining nine studies which gave less than twelve treatments.

6. Frequency of ECT

Frequency of ECT administration does not seem to alter responsiveness to ECT. Stromgren (1975) has shown that patients' response to ECT is the same whether it is given two or four times per week. Of the thirteen studies under discussion, eight involved ECT administration twice a week and five involved ECT administration three times per week.

7. ECT Waveform

To avoid the differential placebo effect in real versus sham ECT studies brought about by patients becoming aware of the true nature of the treatment received (either real or sham ECT) by the experience of real ECT side effects, the ECT waveform chosen should give rise to the lowest possible incidence of side effect. Three studies (Valentine et al 1968, Volavka 1972 and Weaver et al 1978) have confirmed that pulsed current gives rise to less post-ictal confusion and memory disturbance than

sinusoidal current. However one study (Warren & Groome 1984) which compared high energy pulse current and low energy pulse current with high energy sinusoidal current did not find any significant difference between the different waveforms in one aspect of memory function: 'acute general memory'.

The only two studies which cannot be criticised on the grounds of using a waveform (sinusoidal) that produced an unnecessarily high rate of real ECT side effects are those of Lambourn & Gill (1978) and West (1981); both of these studies used pulsed current. It could be argued that even the West (1981) study could be criticised for producing an excessively high rate of real ECT side effects because in comparison to the Lambourn & Gill study the former study used more energy than the latter (40 joules compared to 10 joules) and also used bilateral electrode placement (Lambourn & Gill used unilateral placement). Both of these factors have been reported to produce a greater risk of side effects (see Kiloh 1982, Weeks et al 1980 and Kendell 1981).

8. Electrode Placement

To minimise the chances of producing a Type I error it is important, as noted above, to minimise the postulated differential placebo effect caused by a greater incidence of side effects with real ECT compared to sham ECT (see above). To do this the electrode placement which is associated with the least side effects should be used. There is good evidence that unilateral electrode placement is associated with less post-ictal confusion and less memory disturbance than bilateral placement

(see Kendell 1981). None of the studies with the exception of two (Lambourn & Gill 1978 and Gregory et al 1985) used unilateral placement in their study design. These two studies were therefore the only studies at less risk for the potential Type I error discussed above.

If the postulated differential placebo effect brought about by a greater incidence of side effects with real compared to sham ECT is correct, then the following might be predicted: a) Bilateral ECT would be more effective than unilateral ECT because greater side effects are associated with the former and, b) High energy unilateral ECT would be more effective than low energy unilateral ECT because of the greater side effects associated with the former. Analyses of the results of the unilateral ECT studies (9 and 13) confirm these predictions. For example, Lambourn & Gill's unilateral study (9), in contradistinction to other bilateral ECT studies could not find any difference between real and sham ECT. Also in Gregory's paper (13), in which bilateral real ECT was compared to unilateral ECT and sham ECT, the authors said that their results confirmed that unilateral ECT was less effective than bilateral ECT - although the results did not achieve statistical significance. Finally, Gregory's study which used higher energy unilateral ECT than Lambourn & Gill (36 joules compared to 10 joules) further confirmed the predictions above; Gregory's study reported that their higher energy unilateral treatment was significantly more effective than sham ECT but Lambourn & Gill's study could not demonstrate a difference between their lower energy unilateral treatment and sham ECT.

A contrary critical approach to electrode placement, this time attempting to avoid a Type II error rather than a Type I error, would begin with ensuring that the positioning of the electrodes maximises the chances of the postulated 'true' therapeutic effect of real ECT being revealed. There is some evidence (e.g. Gregory et al 1985) that bilateral treatment has a greater 'true' therapeutic effect than unilateral treatment, although of the many studies done in this area the consensus view as expressed for example by Kendell (1981) is that the evidence that bilateral ECT is better than unilateral ECT is only slight. Nevertheless if one accepts at least the possibility that bilateral ECT is more therapeutic than unilateral ECT, independent of associated side effects, then the ideal real v sham studies would incorporate bilateral ECT. If only unilateral ECT is used there could be a risk of a Type II error. Only one of the studies (9) failed to incorporate bilateral ECT. The apparent lack of effectiveness of real ECT in this study could therefore be explained on the basis of this postulated Type II error.

9. Amount of Electrical Energy Used in ECT

In theory it is possible to criticise ECT studies with regard to ECT energy on two apparently disparate levels: too much electrical energy received by the brain or too little. These two levels of criticism will now be considered in turn.

a) Criticisms of Excessive Electrical Energy

Studies which employ greater amounts of electrical energy will increase the risk of ECT induced side effects (Kirstein & Ottasson 1960). This will enhance the postulated

differential placebo response between the real and sham groups (see above). This in turn will tend to increase the risk of a Type I error: falsely concluding that real ECT is better than sham ECT. Seven of the thirteen studies reported details of the amount of ECT energy used (1,8,9,10,11,12 and 13). The studies which utilised the greatest amount of ECT energy were four studies which used 36 or more joules (8,11,12 and 13) - see Table 3. All of these studies concluded that real ECT was more effective than sham ECT. Therefore these four studies might not only have been at risk of a Type I error noted above but may have actually committed it.

b) Criticisms of Too Little Electrical Energy

There is evidence that ECT electrical energy that falls short of producing a convulsion (subliminal energy) is not therapeutic in depressive illness (e.g. Ulett et al 1956). There is also evidence that the greater the electrical energy is above the seizure threshold (i.e. the more supraliminal), the greater the therapeutic response (the basis for this might be a more powerful 'improving' effect of ECT on the hypothetically malfunctioning cerebral chemical/physical system(s) in depressive illness). For example, D'Elia & Perris (1970) found that supraliminal stimuli was more effective than liminal stimuli. Also in a controlled study Robin & Tissera (1982) reported that high energy ECT produced more antidepressant effect than low energy ECT, although both

produced a convulsion. Furthermore, as noted above, the company that has manufactured most ECT machines for use in the UK (Ectron Ltd) have recently recalibrated their earlier generation of constant current ECT machines to a higher energy output 'because the earlier machines, although they were delivering convulsive stimuli, were not achieving satisfactory clinical results'.

Studies, therefore, which use ECT energies below the seizure threshold (subliminal) and to a lesser extent those studies whose ECT energies are 'less' supraliminal, may not maximise the chances of detecting a greater beneficial response with real ECT. Such studies would thus be at risk of a Type II error. None of the 13 studies stated that their ECT energies were subliminal but those studies which did not adequately assess whether a fit was ascertained (see below) might in theory have administered subliminal ECT to at least some of their patients. With regard to studies employing 'less' supraliminal stimuli: of the seven studies which published details of energy used (1,8,9,10,11,12 and 13) the three studies with the least supraliminal stimuli (1,9 and 10) gave significantly less 'positive' results than the four more supraliminal studies (8,11,12 and 13); two of these three studies gave equivocal results (1 and 10) and the other gave a negative result (9). Therefore all of these three studies may have committed the Type II error noted above.

10. Fit Ascertained

There is evidence that for the antidepressant effect of electrical stimulation of the brain to be achieved it is essential that the degree of electrical stimulation is at least sufficient to produce a seizure. For example, Lancaster et al (1958) and Androp (1941) reported that sub-convulsive ECT was less effective than convulsive treatment. Also Ulett et al (1956) demonstrated in a controlled study that sub-convulsive photoshock (light induced brain dysrhythmia) was less effective than convulsive photoshock or convulsive electroshock. Further, Kirstein and Ottasson (1960) claimed that lidocaine, which reduces electrical seizure activity, also reduced the antidepressant effectiveness of ECT. Studies which do not confirm a seizure has occurred may reduce the chances of identifying a therapeutic effect of real ECT i.e. there will be a risk of a Type II error. Seven of the thirteen studies (3,4,5,6,7,8 and 11) can be criticised because they did not record whether the presence of a convulsion was confirmed; they would have been therefore at risk of the Type II error noted above. The remaining six studies did record that at least an attempt was made to determine whether a convulsion had occurred (1,2,9,10,12 and 13). Four of these studies, however, (1,2,9 and 12) did not use a method of determination that is comparable to EEG monitoring e.g. the 'cuff method'. Clinical determination of seizure was used. This method may result in a seizure being confirmed when it has not in reality occurred i.e. there will be an overestimation of seizures (Christensen & Koldbaek 1982). Therefore studies 1,2,9 and 12 may also have committed the Type

II error noted above.

The overestimation of seizures with the clinical determination of seizure can occur because some observers may confuse the motor tonic phase following the electrical stimulus, in the absence of the subsequent clonic phase, with a seizure. Failure to recognise non-occurrence of the clonic phase is common (Pippard & Ellam 1981). It cannot be assumed that the tonic phase means that a seizure has been induced and furthermore it has been reported that the clonic phase is necessary for therapeutic efficacy (Ottoson 1962). Only two studies (10 and 13) used a method to determine seizure that is comparable in sensitivity to EEG monitoring (Fink & Johnston 1984) : the 'Cuff Method'. In this method a cuff is put around the arm that does not receive the muscle relaxant drug and inflated above arterial pressure, before the muscle relaxant is injected.

If a convulsion is achieved by the electroshock, then the arm which has been protected from the muscle relaxant drugs by the cuff will show motor convulsive activity.

In theory, if the convulsion is important/essential for the antidepressant effect of electroshock, this could be for any of the following reasons:

1. Physico-chemical event(s) constituting the convulsion.
2. Physico-chemical event(s) occurring as a direct consequence of the physico-chemical event(s) constituting the convulsion.
3. Physico-chemical event(s) caused by the electroshock but independent of the convulsion, although coincidental in

time. If this is so then this physico-chemical event(s) is probably not limited to the electroshock method of inducing a seizure because non-electrical means of inducing convulsion (e.g. pharmacological or photic) also appear to have anti-depressant properties (Ulett et al 1956; Palmer 1942; Pacella & Bannera 1943; Small et al 1968 and Laurell 1970). NB. This explanation of the antidepressant effect of ECT fits in with the finding that supraliminal ECT is more effective than liminal ECT (see above).

4. It is possible that the presence of a convulsion means that the brain has received sufficient energy to produce significant side effects e.g. confusion, memory disturbance and headache etc. These side effects might cause the patient to believe they are receiving an 'active' treatment. This might in turn induce a positive placebo response which through 'psychological mechanisms' could help promote an antidepressant effect.
5. The postulated positive placebo response accompanying the convulsions' side effects might induce secondary physico-chemical events in the brain and these events might directly alleviate the depressive illness. A precedent for this model is that it has been argued that the placebo factor in analgesic pain relief might be achieved through stimulation of brain endorphin levels (Levine et al 1978).

11. Matching

a) Matching for the Pharmacological Accompaniments of ECT Study

ECT studies should be matched for any drugs given a) immediately prior to ECT (premedication) and b) during the ECT procedure. Four studies (2,4,5 and 6) did not satisfy this matching parameter. Three of these (4,5 and 6) matched for anaesthetic but did not match for neuro-muscular blocking drug; only the real ECT patients received this latter drug. The fourth study (2) did not have satisfactory matching for anaesthetic or neuro-muscular blocking drug. In this study there were three real ECT groups; one of these groups had no anaesthetic drug or neuro-muscular blocking drug, the second group received anaesthetic drug without neuro-muscular blocking drug, and the third received no anaesthetic but did receive the neuro-muscular blocking drug. In this study there were also two sham ECT groups; one group received anaesthetic without neuro-muscular blocking drug and the other group which received no neuro-muscular blocking drug, received the anaesthetic nitrous oxide which was different from the anaesthetic used in all the other groups : thiopentone.

Ideally the amount of medication given pre-ECT and during the ECT procedure should be standardised per unit of body weight. This would permit more ideal matching for dose of drug. Nine of the studies did not attempt this matching (1-8 and 13). However two of the studies did give a standard amount of anaesthetic and neuro-muscular blocking drug per

unit of body weight (10 and 12).

In addition to matching for pre-medication and medication during the ECT procedure it is also important to match for other medication given during the study. Eleven of the studies did not match satisfactorily for medication during the study (1-9, 10 and 12). In one of the remaining studies (13) the tenor of the paper suggests adequate matching although the only drug data that allowed for a quantitative assessment of matching was for benzodiazepine medication. There was statistical matching for dose of benzodiazepines in this study but there may not have been adequate pharmacological matching: the mean daily doses of benzodiazepine equivalent in the real (bilateral) group, the real (unilateral) group and the sham group were 15mg, 11.12 mg and 13.75 mg respectively (for the possible importance of benzodiazepines in ECT studies see test above). The final study (11) did match satisfactorily for medication: all patients received 50mg amitriptyline at night throughout the study, and no other medication.

b) Energy from ECT

If an ECT study employs any ECT machine other than a 'constant current' machine, then satisfactory matching for ECT energy cannot be assumed - see below for an explanation. None of the thirteen studies with the exception of one (11) employed a constant current ECT machine.

The explanation of why a constant current machine is more likely to achieve satisfactory matching for ECT energy now follows. There are two generic types of ECT machines: the constant voltage (c.v.) machines and the more recently introduced constant current (c.c.) machines. Interpatient variability in ECT energy received by the brain, for a given ECT setting, is caused by two factors: variability in energy output from the machine and variability in head resistance; the greater the energy output and the less the head resistance, the greater the energy received by the brain. The c.v. machines produce a constant voltage for a given ECT setting irrespective of the patients' head resistance. The amount of energy output from the machine is directly proportional to the voltage and the time it is applied and inversely proportional to the head resistance. Therefore for a given setting on a c.v. machine (e.g. 150 volts for 3 seconds) a group of patients would receive a variable energy output from this machine, dependent upon their head resistance. (The greater the head resistance, the less the energy output from the machine). Therefore the energy received by the brain from a c.v. machine, on a given setting, varies dependent upon the energy output from the machine (which is dependent upon the head resistance) and the patient's head resistance. The constant current (c.c.) machines, in contrast to the c.v. machines, produce a standard amount of electrical energy output for a given ECT setting, irrespective of head resistance (Mikhail et al 1984). They do this by automatically varying the voltage, at

a given ECT setting, to compensate for differences in head resistance. The only source of interpatient variability in energy received by the brain for a given c.c. setting is therefore variable head resistance and not variable head resistance and variable energy output as with c.v. machines. In other words, for a given mean energy output, c.c. machines produce less variability (and therefore greater clustering around the mean) in energy received by the brain than c.v. machines. This effect in c.c. machines of causing greater clustering around the mean energy would therefore be to move those patients who would have been on the extremes of the 'energy to the brain' variability on the c.v. machines towards the mean.

The constant current machines would therefore tend to eliminate those patients who would have received 'excessive' amounts of energy to the brain from the c.v. machines, on a given setting, because of low head resistance and similarly those patients who would have received 'excessively low' (even though convulsive) amounts of energy because of high head resistance. In other words c.c. ECT machines will produce more satisfactory matching on ECT energy received by the brain in the real and sham groups than c.v. machines.

c) Number of ECTs

Two studies (1 and 7) presented data which does not permit confirmation of satisfactory matching on this parameter. One of these studies (1) stated that all patients received between 12 and 15 ECTs and the other (7) said that 8 was the

'minimum' number of ECTs and 'most' patients received 12 ECTs.

' END OF TRIAL' PARAMETERS OF CRITICISM

1. Matching for Patients who Completed the Study

In all the studies the statistical analysis to determine the relative effectiveness of real and sham ECT was completed on those patients who completed the study. For this statistical analysis to have been meaningful there should have been adequate matching between the two treatments groups (real and sham ECT groups) for those patients who completed the study. Inadequate or unreported matching of the two groups in the 'completor' population would seriously call into question the results of a controlled real v sham ECT study. The matching within the 'completor' population was particularly relevant in seven of the studies (4,5,8,10-13) because the number of patients in the 'completor' population was less than the number in the 'starter' population because some patients were withdrawn. Of these seven studies five of them did not present any data on the completors to determine whether the matching was adequate or of the same degree as the stated matching of the starters (5,7,8,10,12 and 13). One of the other studies (4) matched the completors for gender alone - the 'starter' population was also only matched for gender. The final study (11) was an exception to all the others in that more information was given on matching for the completors than for the starters. In this study matching for the completors was considered adequate.

2. Numerical Parameters of the Outcome Analysis

In this section we will critically discuss the data produced by each study at the time of the final assessment at the end of the controlled (real v sham) phase of the study i.e. we will

critically discuss the 'outcome' analysis. All data produced after this time is discussed under 'Follow up' analysis - see below. A review of the outcome analysis is given in Table VI.

Listed below are criticisms of the numerical aspects of the outcome analysis, for each study, in the numerical order of the studies (1-13). Note that the criticisms below are accorded a score from 1-10, with the exception of the criticism 'small population size' which is accorded no score. These criticism scores, together with the criticism scores from Table IV (which includes 'small population size') and Table V, are added together to derive a 'global criticism score' which is discussed below under 'Conclusions'.

1. Ulett et al

The only criticism was the small sample size (Real n=9, Sham n=9).

2. Brill et al

a) The outcome analysis was done, for both the real group and the sham group, on a combined group consisting of patients with depressive illness together with patients with schizo-affective illness (depressive type). The outcome data from these two diagnostic groups was not separated in the paper. Therefore a separate outcome analysis comparing real and sham ECT for the patients with depressive illness

was not possible.

In the real ECT group, 12 schizo-affective patients were added to 9 patients with depressive illness in the real group and in the sham ECT group 2 schizo-affective patients were added to 7 patients with depressive illness. It might be argued that schizo-affective patients benefit less from ECT than depressive patients. If this is so then the real ECT group in this study may have had less chance of showing a greater therapeutic effect than sham ECT because the real group had a greater proportion of schizo-affective patients than the sham group. A possible 'positive' result from this study (i.e. real ECT better than sham ECT in depressive patients) may therefore have been hidden because of the greater proportion of schizo-affective patients with the real ECT group. NB Contrary to the arguments above, Brill et al stated in their paper that the patients with schizo-affective illness improved more than the patients with depressive illness.

Criticism Score = 8

b) The outcome analysis was completed one month after the controlled trial was completed. Any greater improvement of real over sham ECT, at the end of the controlled trial, might have been lost by this time.

Criticism Score = 4

c) The sample size was small (Real n=9, Sham n=7)

3. Sainz

- a) The sample size was small (Real n=10, Sham n=10).
- b) The only outcome assessment was a two-point global scale (improved/not improved)

Criticism Score = 6

4. Harris & Robin

- a) The very small sample size makes statistical analysis virtually meaningless (Real n=4, Sham n=4).
- b) Although the outcome analysis, using parametric analysis, suggested real ECT was better than sham ECT (see Table V1) the very small number of patients makes the analysis meaningless. For example, if one of the sham patients who showed no improvement had showed only mild improvement (on the four point scale of improvement), the result would not have been significant. Furthermore, non-parametric analysis on the original data outcome, completed by GS and SA using the Wilcoxon Rank Sum Test, revealed no difference between the real and sham ECT.

Criticism Score = 10.

5. Fahy et al

The only measure of outcome used was a global rating scale. The absence of more sophisticated measures of outcome may have lead to a significant difference between the groups being missed.

Criticism Score = 6.

6. Wilson et al

- a) The very small sample size makes statistical analysis virtually meaningless (Real n=6, Sham n=6).
 - b) In the paper a 't' test, which is a parametric statistical test, was used to analyse the results of the Hamilton Rating Scale data. The authors GS and SA completed an 'F' test on the data which was significant; in view of this and the fact that the sample size was less than 30 patients, a non-parametric statistical test would have been the more appropriate statistical tool to use in this study (Pipkin, 1984). The parametric analysis, using the 't' test indicated that real ECT was significantly better than sham ECT but a non-parametric analyses, completed by GS and SA using the Wilcoxon Rank Sum Test, revealed no significant difference between the two groups - see Table VI.
- Criticism Score = 10.

7. McDonald et al

- a) The very small sample size makes statistical analysis virtually meaningless (Real n=12, Sham n=4).
- b) Separate data for the sham ECT group was not produced. Instead, combined data from the sham ECT group together with another group receiving placebo amitriptyline but no ECT (real or sham) was reported. These combined two groups were known as the 'control group'. The real ECT group was compared to this control group. The reported data did not allow a direct comparison of real ECT and sham ECT and therefore it was impossible to interpret the

results of the study with regard to the relative effectiveness of real and sham ECT.

Criticism Score = 10.

8. Freeman et al

a) At the end of the study clinicians were asked to guess which ECT treatment (real or sham) each patient had received. In other words an attempt was made to determine whether the 'trial code had been broken' - in order to test that staff blindness had been maintained during the study. These guesses were then subjected to statistical analysis. This revealed that the clinicians were not able to guess which treatment the patients had had at a level above that expected by chance i.e. the trial code had not been broken. Furthermore in the three other studies which also tested whether the trial code had been broken (Lambourn & Gill, Johnstone et al and Brandon et al) an identical result was obtained.

A criticism of this 'test of code blindness' was that, in the four studies that utilised the test, no specific criteria were noted as being used by the clinicians to determine whether the patients had received real or sham ECT. If the clinicians used, as is likely, the criteria of clinical improvement (i.e. if patients improved they were adjudged to have received real ECT and conversely if they did not improve they were adjudged to have received sham ECT), then this test of code blindness was in effect also a global

outcome assessment of the results of the study i.e. if the trial code was judged to have been broken then real ECT was better than sham ECT and if the trial code had not been broken, then real ECT was no better than sham ECT. The fact that the results of the four studies which used a test of code blindness was that the trial code had not been broken therefore in effect provided evidence that real ECT was no more effective than sham ECT. As the four studies that used the test for code blindness concluded from their other results that real ECT was more effective than sham ECT, and as the results of the test for code blindness argue against this conclusion, then the use of the test of code blindness will be considered to be a criticism of the results of the four studies that used it.

Criticism Score = 5

b) A second but minor criticism is that there was a numerical inconsistency in the number of patients withdrawn from the study. In one table in the paper 7 patients are reported as having been withdrawn from the study, but in another table and in the text 8 patients are reported as being withdrawn.

Criticism Score = 1

9. Lambourn & Gill

The only criticism was criticism of the authors' employment of a 'test for code blindness' (see above).

Criticism Score = 5

10. Johnstone et al

Again, the only criticism was criticism of the authors' employment of a 'test for code blindness' (see above).

Criticism Score = 5

11. West

a) The sample size was comparatively small (n=11 for both real and sham groups).

b) The sham group described by West appeared to be unusually unresponsive in comparison to the sham groups described by the other five real/sham ECT studies of the modern era (8,9,10,12 and 13). In West's study, in contrast to these other studies, there was an absence of significant improvement in the 'sham' group during the controlled phase of the study. Furthermore, all but one of West's 11 sham patients were clinically judged to require a course of real ECT, at the end of the controlled phase. The unusual lack of responsiveness of the sham group may have prejudiced the study to a Type I error.

Criticism Score = 2

c) Others (Brandon et al 1984) have commented on the 'unusually unequivocal result' of this study. At the end of the study all of the 11 real ECT patients were judged to not require further ECT and in complete contrast, all but one of the sham patients were judged to require a course of real ECT.

Criticism Score = 2

d) For the sham group the rating scales employed show an inconsistency in the direction of change, between the

beginning of the study and the end of the study. The Nurses Scale and the Beck Scale indicated improvement (albeit non-significant) whereas the Visual Analogue Scale indicated deterioration.

Criticism Score = 1

12. Brandon et al

a) There is a numerical discrepancy in the number of patients whose rating scale assessments were reported for the beginning of the study. 95 patients were said to have started the study and 18 of these were said to have been withdrawn from the study and their data excluded from the analysis. This therefore should have left data on 77 patients and indeed this number is noted in the text of the paper. However, for the main rating scale used, the H.R.S., data on only 72 patients starting the study is recorded. Therefore five patients (4 real and 1 sham) are not accounted for (77 minus 72) and this discrepancy is not explained.

Criticism Score = 3

b) There are further numerical discrepancies in the number of patients whose rating scale assessments were reported for the end of the study. For the H.R.S. data, of the 77 patients left at the end of the study only data on 71 patients was presented. This therefore left six patients unaccounted for (4 real and 2 sham). For the Global Scale of Improvement (G.S.I.), data on 72 patients was presented; the discrepancy here was therefore 5 patients (4 real and 1 sham).

Criticism Score = 3

c) There is an unexplained and curious inconsistency in the number of sham patients utilised in the three assessment measures at the end of the study: 28 (for the H.R.S.), 29 (for the G.S.I.) and 34 (for the assessment measure 'the number of patients who required further ECT after the 8 ECTs in the study had been given').

Criticism Score = 4

d) One of the outcome assessments was based on the number of patients who subsequently required more ECT than the 8 ECTs given in the study. Significantly more sham patients than real patients required greater than 8 ECTs. However, two of the sham patients received less than four ECTs and their data was not included in the analysis. (NE no other 'real or sham' patients received less than four ECTs). The paper does not indicate why these two patients received less than 4 ECTs e.g. whether they recovered or not. If indeed they had recovered, and if their data had been included in the '8 ECT' assessment, then the significant difference between the real and sham groups disappears - GS and SA analysis.

Criticism Score = 5

e) The level of significance between the real and sham groups on the H.R.S. and the G.S.I. would have been diminished if a) the two sham patients who received fewer than 4 ECTs had not been withdrawn from the study (assuming they improved) and b) if the data on the 6 'missing' patients (4 real and 2 sham for H.R.S. and 4 real and 1 sham

for G.S.I. - see above) had been included (assuming they all showed a poor response).

Criticism Score = 2

f) A further criticism of this study was its utilisation of a 'test for code blindness' (see above).

Criticism Score = 5

g) Data from one of the stated outcome assessments (a self-rating visual analogue scale) was not reported or commented on in the paper.

Criticism Score = 1

13. Gregory et al

a) 69 patients fulfilled the entry criteria for the study. For the Montgomery & Asberg Depression Rating Scale (MADRS), in the only figure presented in the paper, data was analysed for the 44 patients left at the end of the study after 25 had been withdrawn. However, in one of the tables in the paper, MADRS data on a 'completor' population of 60 was analysed. The reason(s) for this difference in the number of patients used as the 'completor' population was not explained but has some relevance to the next criticism (see below).

Criticism Score; given no criticism score because included as part of the next criticism.

b) The MADRS data on the 44 'completor' patients revealed a significant difference between the 'real' ECT groups (bilateral and unilateral) and the sham group of <0.005 .

However, when the other 'completor' population of 60

patients was analysed the significance between the two real groups and the sham group increased to <0.001 . In other words analysis of the 60 patients increased the chances of finding a difference between the real and the sham groups. For the two other rating scales whose data was presented in the study, the Hamilton Rating Scale - HRS and the Psychological Impairments Rating Scale - PIRS the outcome analysis was performed only on the 60 patients. A separate analysis was not presented on the other completor population of 44 patients. For both the HRS and the PIRS there was a significant difference between the real and sham groups (HRS = <0.01 and PIRS = <0.05) but this degree of difference was less than that shown on the MADRS for the same 60 patients ($p = <0.0001$). If the HRS and PIRS data had been analysed for the completor population of 44 patients, and assuming this data had mirrored the MADRS data (i.e. the level of significance for the 44 patients was less than for the 60 patients), then the difference between the real and sham groups would have been less than the figures given above for the 60 patients i.e. not as significant as <0.01 and <0.05 . It is quite possible, therefore, that if the HRS and PIRS data had been analysed for the 44 patients who had actually completed the study, then there would not have been a statistically significant difference between the two real groups and the sham group - assuming a statistically significant difference is a difference at least at the <0.05 level.

3. Numerical Parameters of the Follow-up

After the controlled (real v sham) phase of the study was completed, eight of the studies (1,3,8-13) continued to study the patients in a design which was not controlled i.e. no sham ECT was given. The period over which the patients were studied in this way is known as the 'follow-up' period. During the follow-up patients received real ECT and/or psychoactive medication.

The main purpose of the follow-up was to gather further relevant information to confirm or not whether real ECT given during the controlled phase was better than sham ECT. For the follow-up to maximise the gathering of this relevant information, it should include the following two design features:

a) The patients and staff should continue to be blind to the nature of the treatment (real or sham ECT) received by patients during the controlled phase of the study. If this were not so then knowledge of the controlled phase code could influence the patient's progress during the follow-up and/or the assessment of their progress.

b) ECT (real) only should be given and not further additional psychoactive medication. Additional psychoactive medication would simply add another variable that would make it more difficult to make interpretations regarding the effectiveness of real over sham ECT. If additional psychoactive medication is used, then at least the two groups from the controlled phase (real and sham ECT) should be matched for this medication.

None of the follow-up studies with the exception of Brandon et al (12) attempted to maintain either patient or staff blindness for the controlled phase of the study. Brandon et al attempted to maintain both patient and staff blindness. Furthermore only three of the follow-up studies clearly restricted treatment during the follow-up to real ECT only (2,8 and 11), and avoided psychoactive medication. Three other studies did 'add in' psychoactive medication (9,10 and 13) and two others implied that this was done without clearly saying so (1 and 12). Of the five studies that did "add-in" psychoactive medication, only one gave satisfactory evidence of adequate matching for all psychoactive medication (10); one other study (9) matched for anti-depressant medication alone and another (13) matched for 'other physical treatments to ECT' but only from one month into the follow-up to its completion.

The follow-up studies could, in theory, help to confirm that the real ECT given in the controlled phase of the studies was better than sham ECT by showing the following:

- a) That the patients who received sham ECT in the controlled phase required more real ECT in the follow-up than the patients who received real ECT during the controlled phase - for the same degree of clinical response.
- b) That the total number of ECTs for the sham ECT group (derived by adding together the number of sham ECTs given during the controlled phase and the number of real ECTs given in the follow-up) was greater than the total number of ECTs for the real ECT groups (derived by adding together the number of real ECTs given during the controlled phase and the number of real ECTs given in

the follow-up) - for the same degree of clinical response.

Hereafter this is referred to as the 'total' number of ECTs assessment.

Listed below are criticisms of the follow-up investigations - with the exception of the criticism relating to patient/staff blindness - see above.

NB In this section the labels 'real' and 'sham' ECT refer to the patient's ECT status during the controlled phase of the study.

1. Ulett et al (Study 1)

a) The follow-up analysis was completed on a relatively small proportion of the patients who completed the controlled phase of the study: at 12 weeks follow-up only 22 of the original 42 patients who completed the controlled phase of the study were analysed, and at the 24 weeks follow-up point only 12 of the original 42 patients were analysed.

b) Those patients who received 'no additional treatment' (presumably ECT, although not specified in the paper) during the follow-up were excluded from the follow-up assessment. Furthermore if the patients received 'additional treatment' this was not necessarily because they had deteriorated, as 'additional treatments' were also given 'when it was felt that additional treatments would enable them to maintain the gains they had made'. This unusual way of determining follow-up treatment, and the anomalous method of analysing the follow-up data after those patients who required 'no further treatment' were excluded, suggested to the authors

(GS and SA) that the follow-up data from this study could not be meaningfully interpreted.

c) The authors state that 'additional' treatment was given during the follow-up but do not specify if this included additional psychopharmacological treatment. If it did, this would make it more difficult to interpret the follow-up data - see above.

2. Harris & Robin (Study 4)

The small number of patients indicated in the follow-up (4 in the real and 4 in the sham group) makes statistical analysis meaningless.

3. Freeman et al (Study 8)

a) One of the assessments used in this study to determine the relative effectiveness of real and sham ECT was (total) 'number of ECTs prescribed'. This assessment was based on the number of ECTs prescribed for each patient irrespective of their clinical response i.e. the real and the sham group at the end of the follow-up were not matched for the degree of clinical response. The analysis included data on 2 real patients who showed overall an 'inadequate response' to ECT, and 2 real and 2 sham patients whose treatment was prematurely stopped because of intercurrent hypomanic episodes. This assessment was not therefore a reflection of the total number of ECTs required for significant improvement/recovery. If it had been it would have been a more relevant index of the comparative effectiveness of real ECT compared to sham ECT.

b) Of the 40 patients who entered the follow-up, only two

were excluded from the (total) 'number of ECTs prescribed' analysis. These two patients were patients who had not improved and had refused further treatment (both real patients). They had received a mean of 6 ECTs which was the same as the mean number of ECTs for the remaining 18 real ECT patients. The exclusion of these two patients was somewhat anomalous as the data from other real ECT patients who similarly did not improve (but did not refuse further treatment) was included in the analysis. If the data of these two real patients who refused treatment had been included in the analysis then statistical analysis by GS and SA reveals that the significant difference between the real and sham groups disappears.

c) Although the authors indicated that their data on the (total) number of ECTs prescribed suggest real ECT is better than sham, a separate analysis by others (Crow et al 1978) on the same data suggests the opposite. In Crow's analysis the number of patients in each group that did not satisfactorily respond to treatment, up to the end of the follow-up (4 in real group and 0 in sham group), were statistically compared and this revealed that sham ECT was significantly better than real ECT.

d) Two of the real patients had their ECT stopped because of an 'inadequate response'; none of the sham patients fell into this category. It was not stated how many ECTs these patients had received before being withdrawn (although GS and SA, by back calculation from the presented data, established that the mean number of ECTs for the two

patients was 6) . However, a diagram in the text indicates that at least 2 sham patients received a greater number of ECTs than one of these two withdrawn patients and at least 5 sham patients received a greater number of ECTs than the other withdrawn patient. This suggests the possibility that a greater attempt, with a subsequent greater number of ECTs, was made in the sham patients to achieve a satisfactory response compared to the real patients. If this was so then it would have biased the results in favour of a greater therapeutic effect of real ECT over sham ECT.

e) The text indicates that if sham ECT had no therapeutic effect then the sham patients would have been expected to have received two extra real ECTs than the real ECT patients in the follow-up, because in the controlled phase of the study the real patients received 2 real ECTs and the sham group received no 'real ECT' only 2 sham ECTs. In fact disregarding the many criticisms of the 'total' ECT data noted here, the data on all the patients in the study revealed the sham group on average required only little over 1 more ECT (1.15) than the 'real' group.

f) The difference in the (total) number of ECTs prescribed between the real and the sham group disappears if the data from the one sham patient who received a greater number of ECTs than any other patient in the study (12 ECTs) was excluded from the analysis. Apart from one additional sham patient, the greatest number of treatments given for any other patient was nine.

g) It is unclear from the paper the number of patients

included in the analysis at each of the various follow-up assessment points (see Table VI). It is unclear for example if the data from patients who were withdrawn from the study in between assessment points was included in the subsequent follow-up assessments on the Hamilton Rating Scale and the Visual Analogue Scale. Given the extreme variability in results that can be obtained with this study's data (see above), dependent upon the number of patients whose data is included or excluded, then the absence of data on patient numbers at each follow-up point, allowing others to check the validity of the follow-up data, is a further criticism of the study.

4. Lambourn & Gill (Study 9)

There is some difficulty in interpreting the follow-up data with regard to real/sham relative effectiveness because additional psychopharmacological medication was allowed during the follow-up. However, there was no apparent difference in at least the amount of antidepressant medication given to real and sham groups during the follow-up. Data on other psychoactive medication was not provided.

5. Johnstone et al (Study 10)

As with the Lambourn & Gill study, additional psychoactive medication was allowed during the follow-up. However Johnstone's study, in contradistinction to Lambourn & Gill's study, matched for all psychoactive medication and not just antidepressant medication.

6. West (Study 11)

a) For the sham group there was some mild inconsistency in

the direction of clinical change, as indicated by the rating scales used. For example the Nurses Scale and the Beck Scale data at the beginning of the follow-up, compared to the data at the end of the controlled phase of the study, indicated clinical improvement but Visual Analogue Scale data indicated deterioration.

b) Others have commented on the unusually unequivocal results of this study generally (see above). The unequivocal nature of the results extended to the follow-up, when none of the 11 real patients were judged to need 'follow-up' ECT but 10 of the 11 sham patients did.

c) It is the contention of GS and SA that the clinical decision-making process that determined whether the patients required 'follow-up ECT' was at least questionable. For example statistical analysis by GS and SA on the data at the end of the controlled study, assuming a normal distribution of data as the study employed the Students 't' test, showed that statistically speaking the most improved 3.75 sham patients should have scored between 11 and 19 on the Beck Scale, and the least improved 3.75 real patients should have similarly scored between 11 and 19. This overlap in the scores of over one third of the patients in the real and sham groups (3.75 out of 11) would have made it all but impossible for the clinician to say with necessary clinical precision whether further ECT was indicated or not. Despite this as noted above none of the real patients were judged to require further ECT but 10 of 11 sham patients were.

7. Brandon et al (Study 12)

a) There is an unexplained inconsistency in the number of patients analysed in the follow-up for the two assessment parameters employed; for the Hamilton Rating Scale (HRS) 41 real patients and 28 sham patients were analysed but for the assessment 'need for further continuous ECT' 43 real patients and 34 sham patients were analysed.

b) The follow-up assessment described as the 'need for an additional follow-up course of continuous ECT' was not adequately defined: the specific indications for prescribing further ECT were not defined and neither was the phrase 'a course of continuous ECT' defined. Therefore the data from this assessment method is difficult to interpret.

c) It is unclear from the text whether during the follow-up patients received psycho-active treatments other than ECT e.g. antidepressant medication. The text states for example that during the follow-up: 'consultants had a free choice of treatment methods' and furthermore that 'although continued restriction of treatment choice would have been scientifically desirable, it was not regarded as being acceptable ethically or to the consultants concerned.' If additional antidepressant medication was in fact given, then any difference in the type and amount of this medication between the two treatment groups might have affected the follow-up results.

8. Gregory et al (Study 13)

a) The number of patients analysed for the follow-up was unclear from the text. 44 patients were said to have

completed the controlled phase of the study, after taking withdrawals into account, and 32 patients 'went on to receive further bilateral ECT'. It is unclear whether the 44 patients as a whole were analysed for the follow-up, or only the 32 who received further ECT. NB Indeed it is not clear if all the 32 patients who received further ECT came from the group of 44 patients who completed the controlled phase of the study, or if some of them came from another group of 25 patients who started the controlled phase of the study but were withdrawn from the controlled phase before its completion.

b) The study purported to demonstrate, from the ECT data in the follow-up, that the real ECT given during the controlled phase had been more effective than the sham ECT. However in the opinion of GS and SA this conclusion was subject to a number of reservations/qualifications:

i) The difference in the amount of ECT ('total') required between the real groups and the sham group only extended for the first month of follow-up. There was no difference in number of ECTs given from the first month of follow-up to the end of the follow-up which was six months after the end of the controlled phase of the study.

ii) There was no indication from the text that the real and sham groups were matched for other psycho-active treatments, particularly additional psychopharmacological treatment, during the important first month of follow-up. The interpretation that real ECT was better than sham ECT because from the beginning of the controlled phase of the

study to the end of the first month follow-up the sham patients received more ECT (sham and real ECT) than the real patients (real ECT), must therefore at least be questioned because the real and sham groups may not have been matched for psychopharmacological medication during the first month of the follow-up.

iii) In contradistinction to the first month of follow-up, there was apparent matching for 'other physical treatments' between the end of the first month of follow-up and the end of the follow-up six months after the completion of the controlled phase of the study. Moreover, in contrast to the analyses at the end of the first month of follow-up, there was no difference in the amount of ECT prescribed between the real and sham groups during this period. These facts add weight to the possibility that any difference in amount of ECT prescribed for the real and sham groups up to the end of the first month of follow-up might, at the very least, have been caused by a lack of matching in other physical treatments during this period and not necessarily to any intrinsic differences in effectiveness between real and sham ECT.

iv) No statistics (or data allowing for statistical analysis) were presented demonstrating a significant difference between the real and sham groups in the amount of real ECT given during the six months follow-up. The only relevant data provided was mean number of real ECTs given during this period without relevant standard deviations or standard errors which would allow for statistical analysis.

The statistically significant differences in the number of ECTs reported between the real and sham groups were only for the 'total' number of ECTs given (real and sham) during the period encompassing the controlled phase of the study and the first month of follow-up.

CONCLUSIONS

Electroconvulsive therapy was first introduced as a treatment for depressive illness approximately 50 years ago. Since then, together with antidepressant medication, it has been the mainstay treatment for depressive illness. Despite this long history of use, it is only in recent years that its effectiveness has been the subject of proper critical scientific investigation using controlled trial methodology. It is the authors' opinion that the delay by the scientific community in critically investigating ECT through controlled studies makes it even more important that the controlled studies themselves become the focus of critical attention by others without further undue delay. This paper represents an attempt by the authors to contribute to this critical process. Before summarising the criticisms of the 13 controlled real v sham ECT studies it should be noted that others have reported that double-blind controlled study methodology does not automatically confer objectivity. Mason (1962) in an article in the Lancet commented that the outcome of even double blind trials can be influenced by the ^{beliefs} 'convictions, ^{desires} prejudices and ^{purposes} desires of those who organise them.' More recently a series of articles in the journal Nature this year (1988) have underlined the difficulty in removing the

subjective element from rigorously controlled studies. This series of articles started with the appearance of a paper by Benveniste et al (1988) claiming to have found that human white blood cells respond to a solution of antibodies even when the solution is so dilute that it can no longer contain a single molecule of antibody, giving some theoretical support to homeopathic practice. This work was replicated by others in Israel, Italy and Canada before the journal accepted the article for publication. However, the journal subsequently published a paper by others (Maddox et al 1988) who independently examined the laboratory techniques and data of Benveniste et al and found that subjective bias had unconsciously influenced and indeed determined the results they obtained.

In summary, and in the opinion of the authors GS and SA, of the thirteen controlled studies the reported data results of five of them (and without any reference being made to any criticism of the data results or indeed the study methodology) suggested real ECT was significantly better than sham ECT. However, the data results of the remaining eight studies did not, in the opinion of the authors, show real ECT was significantly better than sham ECT. This difference in number between the 'positive' studies (5) and the 'non-positive' studies (8) was no better than one would expect from chance. (Chi-Square Test = NS)

It could be argued however that the thirteen studies were of differing quality and that it would be more appropriate to restrict this analysis of 'positive' versus 'negative' studies to the better 'quality' studies alone as a body of work. The first step in accomplishing this would be to define the term 'quality' of study. The

authors decided to define 'quality' by devising a 'global criticism' score for each study; the lower the global criticism score, the better the quality. These global criticism scores were established as follows from the parameters of criticism in Table IV (General Criticism Table), Table V (Matching Table) and Table VI - excluding from the latter table criticisms of the follow-up and criticisms of the number of patients in the studies as this was duplicated in Table IV ;

1. All of the parameters of criticism were afforded a score - see below.

2. For the majority of the parameters of criticisms in Tables IV and V (for the exceptions see below), those which recorded no criticism scored 0; those that recorded the possibility of criticism (i.e. 0?) scored 1 and those that recorded a definite criticism (i.e. 1) scored 2.

3. For the remaining criticisms in Tables IV and V, which were in the opinion of the authors more concern-making criticisms, a 'loading value' was made. The range of scores for the 'loaded' parameters of criticism, which had recorded a 'possibility of criticism' (i.e. 0?), was 2-5. The range of values for the 'loaded' parameters of criticism, which had recorded a definite criticism (i.e. 1), was doubled to 4-10.

4. The 'loading values' for the parameters of criticism in Tables IV and V are given below - possible criticism score (0?) only. N.B. The scores below were doubled if there was a definite criticism score, i.e. 1

PARAMETERS OF CRITICISMLOADING VALUES

a) Table IV (General Criticism Table)

i.	'Adequate number of patients'	5
ii.	'Random allocation to treatment group'	3
iii.	'Patient blindness'	5
iv.	'Staff blindness'	5
v.	'Adequate definition of depressive illness'	4
vi.	'Adequate number of ECTs'	3
vii.	'Satisfactory outcome assessments'	4

b) Table V (Matching Table)

i.	'Severity of current episode of depression'	3
ii.	'Medication during trial'	3
iii.	'Energy from ECTs'	2
iv.	'Number of ECTs'	2

5. The individual values for the various criticisms in Table 6 are indicated in the text under 'Outcome Analysis' (see above). Scores vary between 1 and 10.

The 'global criticism' scores for the thirteen studies, in the numerical order given throughout this paper, were respectively: 1) 54; 2) 71; 3) 82; 4) 94; 5) 79; 6) 73; 7) 63; 8) 39; 9) 37; 10) 34; 11) 36; 12) 57, and 13) 24.

From this it can be seen that with one exception the six studies with least criticisms came from the modern era of real versus sham ECT research i.e. studies 8-13; the one exception was Brandon's study (12) which had the 7th lowest score on the 'global criticism' scale (score 57) being marginally displaced by Ulett's study (score 54). However, as Brandon's study was far

more intensive than Ulett's it will be considered as one of the six least criticised studies. Analyses of these six 'least criticised' and therefore 'better quality' studies showed that four of them (8,11,12 and 13) gave a result indicating that real ECT was better than sham ECT, but two of them gave results which did not satisfactorily demonstrate a difference in effectiveness between real and sham ECT (9 and 10). This difference in number between the 'positive' studies (4) and 'non-positive' studies was no better than one would expect from chance. (Chi-Square Test = NS). Moreover it should be noted that these six studies, although having the least criticisms of the thirteen studies, still had substantial criticism scores. The validity of their results must therefore, in the opinion of GS and SA, be at the very least questioned. Furthermore all of these six studies have items of criticism that are particularly noteworthy. For the sake of brevity, single significantly noteworthy criticisms of each of the six studies are noted below:

1. Freeman et al (Study 8)

Only two ECTs (real or sham) were given in the controlled phase of the study.

2. Lambourn & Gill (Study 9)

Low energy unilateral ECT was used and there is evidence that this form of ECT may be significantly less effective than high energy bilateral ECT (see above).

3. Johnstone et al (Study 10)

There was poor evidence of matching between the real and sham groups. The criticism score in Johnstone's study for all the parameters of criticism in the 'matching table'

(Table V) was greater than the similar score for matching in the other five studies with lowest global criticism scores. Furthermore it was one of the highest of all the thirteen studies.

4. West (Study 11)

Potentially significant queries have been raised by others about the degree of staff blindness in this study (see above).

5. Brandon et al (Study 12)

There are a number of potentially very significant criticisms about the numerical aspects of the outcome analysis which casts some doubt on the manner in which the statistical analysis was carried out (see above).

6. Gregory et al (Study 13)

25 of the original 69 patients were withdrawn during the study and the outcome analysis was conducted on the 44 patients who completed the study but although reasonable attention was paid to matching the 69 patients who started the study, no evidence for matching was presented for the 44 patients who completed the study.

With regard to the follow-up data it is the opinion of the authors, GS and SA, that two studies (11 and 13) provided at least reasonably significant evidence that real ECT was better than sham ECT (see above). However, two other studies (9 and 10) provided evidence that real ECT was no better than sham ECT. The results of the remaining four studies (1,3,8 and 12), in the opinion of the authors, cannot be categorised as either 'positive' or 'negative' either because of the

because of the number of criticisms of the follow-up study design and statistical analysis (8 and 12). Moreover it should be noted that even the results of the two 'positive' studies should be considered with caution because neither attempted to maintain patient and staff blindness for the controlled phase of the study (see above).

In conclusion, it is the opinion of the authors that the results of the outcome analyses and follow-up analyses of these controlled studies, irrespective of the many criticisms noted in this paper including criticisms of data analyses, do not offer significant evidence that real ECT is more therapeutically effective than sham ECT in depressive illness. All of the studies have numerous elements worthy of criticism and these have been discussed in detail in the text of the paper. Perhaps the one general item of criticism that has been overlooked by others, and to which the authors GS and SA feel more attention should be paid, is that of patient blindness. With the present design of real versus sham ECT studies, it appears to us to be very difficult to achieve satisfactory patient blindness (see above). If a controlled 'real' v 'sham' ECT study does not have satisfactory patient blindness, then it is probable that the more powerful positive placebo factor will occur in the actively treated group, i.e. the real ECT group. Now the placebo factor is established as a potentially very powerful force (Lancet, 1983) which can have profound pharmacological effects (Wolf, 1956). Therefore, if there is not satisfactory patient blindness, then the response of the real ECT group as the recipient of the more powerful positive placebo factor would be biased in favour of it being more effective than sham ECT. It might be argued by others that an 'ideal' real versus sham ECT

trial, paying attention to all of the possible elements of criticism raised in this paper including patient blindness, could never be accomplished, if only for practical reasons. Therefore it might be argued that too much attention to criticism of ECT studies, of the sort given in this paper, is of doubtful practical value. It is the contention of the authors that psychiatrists as scientists have a duty to criticise irrespective of where these criticisms lead us.

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GRS = Global Rating Scale (the staff that completed the GRS also indicated for some studies).

VAS = Visual Analogue Scale.

HRS = Hamilton Rating Scale.

MADRS = Montgomery & Asberg Depression Rating Scale.

PIRS = Psychological Impairment Rating Scale.

MMPI = Minnesota Multiphasic Personality Inventory.

RS = Rating Scale.

1. The first part of the report is a general
 introduction to the subject of the study.
 2. The second part is a description of the
 methods used in the study.
 3. The third part is a description of the
 results of the study.
 4. The fourth part is a discussion of the
 results of the study.
 5. The fifth part is a conclusion of the
 study.

Legend for Table I (General Description Table I)

- ? = These studies did not provide sufficient information to answer this question.
- * = 'Matched' random allocation (see text).
- ' ' = Direct quotes from the relevant texts.

TABLE 1
(ORIGINAL DESCRIPTION TABLE 1)

Author(s) and study number (1-13)	Year	Population Assessed for Entry into study (and country where study completed)	All patients in population considered for entry	Comparison Groups	Random allocation to comparison groups	Definition of Depressive illness	Other Criteria for Entry into Study
Ulett et al (1)	1956	Regular admissions of patients thought most likely to respond to ECT, to a 23rd and psychiatric unit of a large city hospital over a two year period (USA).	?	Real ECT v Sham ECT NI Also a placebo-comulsive group & sub-comulsive placebo shock group included.	Yes*	Clinically diagnosed as either manic depressive reaction (depressive type), psychotic depressive reaction, psychoneurotic depressive reaction.	1) Those patients thought "most likely to respond to ECT" 2) Two psychiatrists had to agree on the diagnosis. NI. Patients with schizophrenia also included in this study.
Brill et al (2)	1959	In-patients from a veterans administration hospital (USA).	?	Real ECT v Sham ECT NI 3 real ECT groups (ECT only, ECT+medication, ECT+medication & 2 Sham ECT groups (schizophrenic group & others with group) also included.	Yes	Clinically diagnosed as either: 1) Psychotic depression 2) Reactive depression 3) Manic depressive psychosis (depressive type) 4) Involutional depression	No patient had received ECT for at least nine months. NI Schizophrenic "reaction" and schizoaffective reactions (depressive type) also included.
Saluz (3)	1959	Psychiatric admissions (USA).	?	Real ECT v Sham ECT	Yes	Clinically diagnosed as either: 1) Manic-depressive disorders - depressed phase or 2) Involutional depression	NI
Harris & Robin (4)	1960	All admissions to one mental unit at Maxwell Hospital (UK).	Yes	Real ECT (+ placebo phenelzine) v Sham ECT (+placebo phenelzine) NI 2 other groups included: Sham ECT + phenelzine group; phenelzine only group.	Yes	Clinically diagnosed as "depressive reaction" ("patients were a group of severe psychotic depression")	2 clinicians had to independently agree on the diagnosis of depression.
Foley et al (5)	1963	Not stated (UK).	?	Real ECT v Sham ECT NI A 3rd group (imipramine only) included.	Yes	Clinical diagnosis of depression ("moderate severity", "endogenous symptom" & "non-reactive").	1) Only if they were considered, after a weeks observation in hospital, to be in need of ECT. 2) Severe depressions with high suicidal risk were not included. 3) None had received ECT or Imipramine for current episode of illness.
Wilson et al (6)	1963	All new admissions to a hospital (all females) (USA).	Yes	Real ECT (+placebo imipramine) v Sham ECT (+placebo imipramine) NI Also a real ECT (+imipramine) group & a sham ECT (+imipramine) group included.	Yes	Clinically diagnosed as either: 1) Manic depressive (depressed type) or 2) Involutional depression or 3) Depressive Reaction	NI
McDonald et al (7)	1966	All new admissions to two hospitals (Canada).	Yes	Real ECT v Sham ECT NI Also an active amitriptyline group and a placebo amitriptyline group included.	Yes	"Clinical symptoms of depression"	1) All those "in the judgment of the consultant psychiatrist who would be normally treated with ECT". 2) No ECT in the month prior to the study 3) No antidepressants in the two weeks prior to the study.
Freeman et al (8)	1970	All patients from four acute units (UK).	Yes	Real ECT v Sham ECT	Yes	Clinically diagnosed as suffering from primary depression, not less than 15 years in both Hamilton & Beckman's scales.	1) Age 20 - 70 years. 2) No ECT in the past 3 months.
Lawson & Gill (9)	1970	All right handed patients clinically diagnosed as depressive psychosis at Knowle Hospital & referred for ECT (principally in-patients but also 2 out-patients) (UK).	Yes	Real ECT v Sham ECT	Yes	Clinically diagnosed as depressive psychosis.	1) No ECT in the three months prior to the study. 2) Referred for ECT by patient's own consultant psychiatrist. 3) All patients right-handed.
Johnston et al (10)	1980	All patients admitted to Northwick Park Hospital under the care of participating psychiatrists (UK).	Yes	Real ECT v Sham ECT	Yes	1) IHC criteria for depressive illness. 2) Folstein's criteria for primary depressive illness. 3) Newcastle criteria for endogenous depressive illness	1) Age 30 - 69 years. 2) Newcastle criteria for predicting a good outcome from ECT.
Hunt (11)	1981	25 inpatients with primary depressive illness were selected (UK).	?	Real ECT v Sham ECT	Yes	Primary depression (Folstein's criteria).	At least 1 month history of depressive illness.
Brandon et al (12)	1981	All psychiatric inpatients in Leicestershire for whom ECT was prescribed (UK).	Yes	Real ECT v Sham ECT	Yes	Using Present State Examination & Catego Program; 1) Retarded depression or 2) Depression with delirium or 3) Manic depression.	ECT clinically indicated.
Gregory et al (13)	1987	All admissions to Maudsley Psychiatric Hospital who were right-handed and were referred for ECT (UK).	Yes	Blindfold Real ECT v Unblindfold Real ECT v Sham ECT.	Yes	IHC (1965) Criteria for depressive illness.	1) Depressive illness of at least one month duration. 2) Three patients referred on clinical grounds for ECT. 3) No previous ECT for current episode of illness. 4) Right-handed.

Legend for Table II (General Description Table II)


- * = The upper figures represent data for Real ECT group and lower figures represent data for Sham ECT group.
- $\frac{+}{-}$ = Mean data for Real and Sham groups combined. Separate data for the groups not given.
- $\frac{+}{-}$ = Upper figures represent data from bilateral ECT group, middle figures for unilateral real ECT group and lower figures for sham ECT group.
- = Relevant data not recorded in these papers.
-  = Data for completers. Data for starters not given.
- $\frac{+}{-}$ = 4 out of 10 real ECT withdrawals not accounted for in paper and similarly 2 out of 14 sham ECT withdrawals.

TABLE 11
(DEFINITION TABLE 11)

Author(s)	Year	Number of Patients*	Number of Unknowns*	Number of Completed*	Mean Age of Patients* (Age range given in brackets)	Sex of Patients* (M-F)	Number of Patients with previous history of disease*	Number of Patients with history of RZ*	Number of Episodes per week	Total Number of Episodes	D.S.
Mell et al	1956	9 9	0 0	9 9	- -	3(M) 6(F) 3(M) 6(F)	- -	- -	3	12-15	4
Wyllie et al	1959	9 7	0 0	9 7	- -	9(M) 7(M)	- -	- -	3	20	7 (6)
Wyllie	1959	10 10	0 0	10 10	- -	8(M) 12(F) ¹	- -	- -	3	12	4
Wyllie & Hobbs	1960	6 6	2 2	4 4	- -	6(F) 6(F)	- -	- -	2	4	2
Fridy et al	1963	20 20	3 3	17 17	(30-59) (30-60)	10(M) 10(F) 8(M) 12(F)	4 8	- -	2	6	3
Wilson et al	1963	6 6	0 0	6 6	- -	6(F) 6(F)	- -	- -	2	6	3
McDonald et al	1966	12 4	0 0	12 4	- -	- -	- -	- -	3	12 (in most cases; 8 was minimum)	4 c.
Freeman et al	1978	20 20	6 2	14 18	51 (20-70) 50.5 (20-70)	6(M) 14(F) 5(M) 15(F)	14 14	10 12	2	2	1
Lambert & Will	1978	16 16	0 0	16 16	51.4 (39-69) 51.5 (37-66)	7(M) 9(F) 7(M) 9(F)	13 13	11 10	3	6	2
Johnstone et al	1980	35 35	4 4	31 31	49.4 [†]	10(M) 52(F) [†]	46 [†]	15 [†]	2	8	4
West	1981	13 12	2 1	11 11	52 53.3	6(M) 5(F) 7(M) 4(F) [*]	- -	- -	2	6	3
Bracken et al	1981	53 42	10 ¹³ 14 ¹¹	43 28	55.4 53	21(M) 32(F) 13(M) 29(F)	2.6 (mean number of episodes of illness) 2.5	29 28	2	8 (between 4-8, the majority had 8)	4
Gregory et al	1985	23 23 23	9 7 9	14 16 14	- - -	- - -	- - -	- - -	2	6	3

Legend for Table III (General Description Table III)

- = Insufficient information provided to answer this section.
- * = Number of patients who received each group of drugs indicated below. If the number was not recorded in the paper then this is indicated by 'number?'.
- + = 150 volts delivered for three seconds. Assuming the average head resistance was the same as in Freeman's study, (which delivered 400 volts for 1.5 seconds, equivalent to 36 joules), then amount of energy delivered was 27 joules.

THE HISTORY OF THE UNITED STATES

The history of the United States is a story of growth and change. It begins with the first settlers who came to the Americas in search of a new life. They found a land of opportunity, but also a land of challenge. The early years were marked by struggle and hardship, but the spirit of the pioneers was unyielding. They built a nation from scratch, one that was founded on the principles of liberty and justice for all. Over the centuries, the United States has grown from a small colony to a global superpower. It has faced many challenges, but it has always emerged stronger and more united. The history of the United States is a testament to the power of the human spirit and the ability of a nation to overcome adversity.

Author(s)	Year	ECT Technology				ECT Anesthetized	Injury with/without prior to study	Trial Medication*			Outcome Rating Scale
		Machine used	Waveform	Frequency (Hz/sec)	Current (mA)			Anti-depressants	Antipsychotics	Others	
Ulett et al	1956	Mark III B-24	Sinusoidal	-	No	Yes	-	-	-	-	1) Hamilton Psychiatric Scale 2) Global rating for improvement
Drill et al	1959	Electroncraft model 107	Sinusoidal	-	No	Yes	-	-	-	-	1) Lorr Psychiatric Rating Scale (inpatients, doctors and nurses) 2) Psychological tests 3) Global improvement 4) Mean value combining three scales
Salz	1959	-	Sinusoidal	-	No	-	-	-	-	-	'Qualitative Clinical A
Harris & Robin	1960	-	Sinusoidal	-	No	-	-	No	No	Sodium Amytal-bicarbonate (number?)	Global scale of improvement
Foley et al	1963	-	Sinusoidal	-	No	-	-	No	No	Barbiturate hypnotic "meu" (number?)	Global scale of improvement
Hilken et al	1963	-	Sinusoidal	-	No	-	-	-	-	-	1) Hamilton Depression Scale 2) Minnesota Multiphasic Inventory - "D" Scale
McDonald et al	1966	-	Sinusoidal	-	No	-	-	No	-	-	1) Depression Rating Scale 2) Global scale of improvement in depressive symptoms 3) Minnesota Multiphasic Inventory - Depression Psychasthenia Scales 4) Ward observation form
Freeman et al	1970	Electroncraft Mk IV	Sinusoidal Modified (Bilateral) (bilateral)	36	No	-	-	Yes (7 oral; 11 sham)	-	-	1) Hamilton Rating Scale 2) Beck's Self Rating Scale 3) Wakefield Self Rating Scale 4) A visual analogue scale
Lindeman & Gill	1978	Electroncraft Mk IV	Brief Pulse (Bilateral)	10	No	Yes	Yes, for all patients - except for hypotension	No	Yes (number?)	No	1) Hamilton Depression Scale 2) A global scale of improvement
Johnstone et al	1980	Electroncraft Mk IV	Sinusoidal Modified (Bilateral)	27*	No	Yes ("cuff method")	-	No	Yes (all patients had a benzodiazepine hypnotic; some also had additional diazepam)	No	1) Hamilton Depression Scale 2) Leeds Self Rating Scale 3) Nurses Rating Scale (Lundberg)
West	1981	Thymatron	Brief Pulse (Bilateral)	10	Yes	-	-	Yes (50mg amitriptyline twice; all patients)	No	No	1) Beck's Self Rating Scale 2) A Psychiatrists' Visual Scale 3) A Nurses' Global Psych Scale
Bracken et al	1981	Electroncraft Mk IV	Sinusoidal Modified (Bilateral)	36	No	Yes	Yes	No	Yes (number?)	-	1) Hamilton Depression Scale 2) Montgomery & Asberg D Rating Scale 3) Newcastle Depressive Index 4) Leeds Wakefield Irritability, Depression, Anxiety Scale 5) Visual Analogue Scale 6) Spielberg Anxiety Rating Scale
Gregory et al	1985	Electroncraft Mk IV	Sinusoidal Modified (Bilateral)	36	No	Yes ("cuff method")	Yes	Yes (1)	Yes (51)	Neuroleptic (1) Lithium (3)	1) Montgomery & Asberg D Rating Scale 2) Hamilton Depression Scale 3) Psychological Impairment Scale 4) Global Scale of Psychiatric Improvement

LEGEND FOR TABLE IV (GENERAL CRITICISM TABLE)

- = Criticism absent i.e. patient cannot be criticised on this parameter.
- 0 = Study can definitely be criticised on this parameter.
- 0? = No or insufficient data presented in the paper to assess this parameter of criticism.
- = D= Doctors from the patients' ward(s), caring for the patients' clinical needs.
N= Nurses caring for the patients' clinical needs.
A= Those people involved in assessing the patients on the outcome assessments(s).
- 0 = See legend for Table VI under 0.
- = For the relevance and discussion of these parameters of criticism, see text under 'Patient Blindness'.
- x = 'Matched Random' study - see text.
- * = From the texts of these papers (and in the case of West et al the comments of others), GS and SA concluded that the weight of the evidence suggested either the / or 0 indicated, but there was still room for doubt.
- $\frac{1}{1}$ = For definition and discussion of a 'constant current' ECT machine, see text under During Study Parameters :
Matching (ECT Energy Used).

LEGEND FOR TABLE V (MATCHING TABLE)

- ✓ = Matching adequate
- = Text indicates a definite lack of matching
- ? = A lack of data on this point does not allow for an assessment on matching.
- = In these studies patients were not withdrawn.
- + = 'Adequate' means that evidence was presented in the paper that the degree of matching described for the starters was maintained for the completors.
- * = Matching on these parameters not specifically given in the paper's text. However, general statements given, such as 'matched for previous illness' and 'previous treatment'. These general statements are less than ideal in deciding upon matching on the specific parameters given in the matching table, but the authors decided to give this study the benefit of doubt. Similarly the same benefit of doubt was given for a) the age and sex parameters for which matching was described as 'satisfactory' but no quantitative data was presented and, b) medication during trial parameter for which the tenor of the paper suggests matching but does not clearly state this.
- = = Matching for antidepressant medication alone described.

N.B. All matching parameters relate to information given regarding the patients who started the study, with the exception of the parameter "Adequate matching for completors" which relates to the patients who completed the study.

AUTHOR(S) and YEAR	Age	Sex	PREVIOUS HISTORY OF DEPRESSIVE ILLNESS			PREVIOUS HISTORY OF ECT			CURRENT EPISODE OF DEPRESSION (PRIOR TO STUDY)					MEDICATION		MATCHING FROM START OF ECT TREATMENT IN STUDY				
			Number of Patients	Number of Episodes per Patient	Total duration of all Depressive Illness	Number of Patients	Number of ECT's	Response to ECT in the past	Severity	Length	Antide- pressant medication	ECT	Contem- porary stress	Contem- porary support	Pre- trial (for index episode)	During trial	Energy from ECT's	Number of ECT's	Matching for the Pharmacological Accompaniments of ECT procedure.	Adequate* Matching for Completers (in Studies in which some patients were withdrawn during the study).
LEIT et al 1956	02	✓	02	02	02	02	02	02	02	02	02	02	02	02	02	02	02	02	✓	-
SMITH et al 1959	02	✓	02	02	02	02	02	02	02	02	02	✓	02	02	02	02	02	✓	0	-
SALZ 1959	02	02	02	02	02	02	02	02	02	02	02	02	02	02	02	02	02	✓	✓	-
HARRIS & REED 1960	02	✓	02	02	02	02	02	02	02	02	02	02	02	02	02	02	02	✓	0	-
FAHY et al 1963	✓	✓	0	0	02	02	02	02	02	02	✓	✓	02	02	02	02	02	✓	0	-
WELSH et al 1963	02	✓	02	02	02	02	02	02	✓	02	02	02	02	02	02	02	02	✓	0	-
MCDONALD et al 1966	02	02	02	02	02	02	02	02	✓	02	02	02	02	02	02	02	02	02	✓	-
FREEMAN et al 1978	✓	✓	✓	✓	02	✓	02	02	✓	02	✓	✓	02	02	02	02	02	✓	✓	02
LANGLIN & GILL 1978	✓	✓	✓	✓	02	✓	✓	02	✓	02	02	✓	02	02	✓	✓	02	✓	✓	-
JOHNSTON et al 1980	02	02	02	02	02	02	02	02	✓	02	02	02	02	02	02	02	02	✓	✓	02
WEST 1991	✓	✓	02	02	02	02	02	02	✓	02	02	02	02	02	02	✓	✓	✓	✓	-
BRANDON et al 1984	✓	✓	02	✓	02	✓	02	02	✓	02	0	02	02	02	02	02	02	✓	✓	02
GREGORY et al 1985	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	02	02	02	✓	✓	02	✓	✓	02

Legend for Table VI (Outcome/Follow Up Table)

- + = On the basis, solely, of the outcome data presented in the papers, real ECT appeared significantly more effective than sham ECT. This global assessment does not take into account flaws in the derivation/assembly of the outcome data which are noted elsewhere in the text.
 - ? = The outcome data either suggested real ECT was no more effective than sham ECT or provided insufficient / inconclusive information to judge the relative merits of the two forms of ECT. N.B. Studies which included 6 or less patients in either the real or sham ECT groups (i.e. studies 4,6 and 7) were adjudged to have provided insufficient information.
 - ⊙ = Inadequate population size.
 - ✓ = Adequate population size.
 - Δ = Antidepressant treatment (ECT and/or antidepressant medication) given according to clinical needs.
 - ▲ = Upper number is for the real ECT group and the lower number is for the sham ECT group. Where one number is given this is for the real and sham groups combined.
 - * = All significant values noted represent real ECT better than sham ECT. All tests done on those patients still in the studies at the end of the controlled phase or at the end of the follow-up unless otherwise indicated. All significant tests unless otherwise indicated, were conducted between groups (real v sham) and not within groups.
- N.B. The full range of statistical tests used in the studies was as follows: 't' tests were performed by 1,4,8,11 and 13 (the latter for within group analysis only); 'ANOVA' by 2,10,12 and 13; 'Chi Square test' by 2 (Global Rating Scale) and 3 (GS & SA analysis); Wilcoxon Rank Sum Test by 6 (GS & SA analysis) and 9; in study 7, the type of significance test used was not noted; in study 5, a formal significance test was not noted only mean data was noted which did not allow GS and SA to employ a significance test of their own.
- x = The basis of these scales was qualitative assessment by the staff indicated, followed by research workers' quantitative assessment of these qualitative results.
 - = Real ECT v a 'control group' (the control group was a sham ECT group combined with placebo amitriptyline).
 - = Number of ECTs given during controlled phase (real or sham) plus number of ECTs (real) given during the follow up.
 - A = For detailed discussion of the criticisms of the outcome analysis and follow up see text.

GRS = Global Rating Scale (the staff that completed the GRS also indicated for some studies).

VAS = Visual Analogue Scale.

HRS = Hamilton Rating Scale.

MADRS = Montgomery & Asberg Depression Rating Scale.

PIRS = Psychological Impairment Rating Scale.

MMPI = Minnesota Multiphasic Personality Inventory.

RS = Rating Scale.

To: Leonard
From: Diane

Based on
10 Bay Area
women who
underwent ECT
from 1957-61

ELECTROCONVULSIVE THERAPY, THE SELF, AND FAMILY RELATIONS

Carol A.B. Warren

This paper is concerned with the implications of electroconvulsive shock therapy (ECT) for the self and for family relationships. The perspective is interactionist, stressing the meanings of ECT to those who have undergone it: their interpretations of its purposes and effects, and its impact on their lives. This viewpoint, which has similarities to a "consumer" view of medical treatment, contrasts with the medical-model orientation of the psychiatrist ordering shock, and with the organizational perspective of the nurse administering it.

The data for the analysis are intensive interviews with ten women diagnosed as schizophrenic, and with their husbands, during the patient and outpatient phases of their moral careers as mental patients (1). These ten women were among 17 admissions to California's Napa State Hospital in 1957-1961 who were the focus of a large-scale study of mental hospitalization and the family, the "Bay Area" study (2).

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The women were all white, were from lower to lower middle class backgrounds, had at least one child, were currently married, and ranged in age from 26 to 40. All but two were first admissions; all were interviewed at Napa State Hospital, where they stayed for an average of 19 weeks. The interviews began with the week of admission to Napa, and ended up to 100 weeks following release. The mean number of interviews with husbands or wives in the patient and expatient phases of the moral career was about 50 (3). A number of studies dealing directly or indirectly with these data have been published in the decades since the data collection. This paper is part of a larger re-analysis of the data (4).

An additional source of data is reinterviews with the original Bay Area sample in 1972, done by John Clausen and his colleagues at Berkeley. These interviews were one-shot, and took place with either the husband, the wife, or both where available. Although questions about ECT were not systematically asked in the reinterviews (which were focused on the marital relationship and the couple's children); the records include comments about ECT from five of the families.

The intensive interview method is an ideal one for developing an understanding of patients' interpretation of ECT and other therapies, since the interview focuses on verbalized meanings. Similarly, the ethnographic or observational method used by Goffman (1961), Perruci (1974) and other analysts of mental hospitalization and therapy is ideal for developing an understanding of the organizational or social control aspects of hospital life, since these methods focus on everyday life in the mental hospital ward.

The interest of the Bay Area data is both historical and contemporary. While ECT was frequently the therapy of choice in state mental hospitals in the 1950s, it fell into disfavor in the 1960s and 1970s, although not necessarily disuse. Between 40,000 and 50,000 patients yearly were given shock in the United States in the 1970s. In the 1970s and 1980s, private and voluntary "shock shops" sprung up in some metropolitan areas for the "quick and easy" treatment of depression at \$30 and up at a rate of one patient every four minutes (5). Mental hospitals are once more proposing ECT as a useful, and, ironically, "innovative" last resort treatment for the suicidally depressed or catatonically schizophrenic.

ECT's return to favor as a therapeutic practice is occurring not so much in state hospitals—some states, such as Massachusetts, forbid its use in public mental hospitals—but rather in private hospitals and private practices (6). In a survey of the membership of the American Psychiatric Association published in 1981, only 6.2 percent of those members who completed the questionnaire (80 percent of the total membership of 600) reported using ECT in their practices (7); in 1985, however, a study estimated that 16 percent of APA psychiatrists used ECT (8). The greatest growth of ECT use in the early 1980s was in private psychiatric hospitals and psychiatric wings of private general hospitals (9). The impact of ECT on the selves and lives of its consumers, then, is of contemporary as well as historical significance.

THE HISTORY AND PRACTICE OF ECT

The use of electric shock in psychiatric medicine has a long history, predating the scientific and medical models of illness by many centuries:

The use of nonconvulsive electrotherapy as a method for alleviating symptoms through suggestion dates back to Scribonius Largus (c. AD 47), who treated the headaches of the Roman Emperor with an electric eel (10).

The first electroconvulsive treatment for mental illness was, "Probably . . . administered by a French physician, J.B. Leroy, in 1755 on a patient with a psychogenic blindness." (11). The modern use of ECT began in the 1930s in Italy (12). Its use was premised on the claim of a Hungarian asylum superintendent that schizophrenia could not coexist with epilepsy in a human organism. There was no such thing as an epileptic schizophrenic; therefore, his reasoning went, the electroconvulsive (or insulin-coma) inducement of grand mal seizures would cure schizophrenia (13).

There is basically no theory of how or why ECT works, merely a belief on the part of some doctors that it does, and of others that it doesn't (14). Those in favor of the treatment claim that it relieves severe depression and that it is less harmful, in many cases, than alternative treatments such as psychoactive drugs. Opponents of ECT claim that it has never been proven scientifically to be of use, and that it often causes permanent long-term memory loss or even brain damage. Other side effects, agreed upon by proponents and opponents, are headaches, dizziness, loss of appetite, missed menstruation, flat affect or "slap happy" silliness, and short-term memory loss (15). The most problematic ECT side effect of ECT, however, is short-term memory loss (16). Experts and informants disagree over whether full memory finally recurs for all patients or whether it remains patchy, for at least some patients, in the long term (17).

Apart from the side effects, opponents of ECT respond negatively to the procedure itself. Unlike other body-related psychiatric therapies, such as taking pills, ECT is a culturally unfamiliar procedure which seems both strange and horrible to the observer. Friedberg describes the administration of shock as practiced in the early 1960s:

In *bilateral ECT*, the most common technique, electrodes are applied to the patient's temples; in *unilateral ECT* they are placed over the forehead and occipit of one side of the patient's head. An electrolyte paste is used to reduce skin resistance and prevent burns. The voltage necessary to reach seizure threshold and induce a grand mal epileptic seizure—the object of the procedure—ranges from 70 to 150 volts and the current, which varies inversely with impedance, may be up to 1 ampere. The duration of the discharge is preset at .5 to 1 second. As the button is pushed there is an involuntary tonic spasm of the patient's facial musculature. This is followed, after several seconds, by violent shaking, the

grand mal convulsion. . . Most authors refer to the average use of 6 to 10 or 12 treatments of depressive illness and 18 to 25 treatments for schizophrenic illness (18).

One of the Bay Area patients describes ECT (probably bilateral) from an experiential perspective:

(Donna Urey)¹ "And you have been getting shock, you say this morning?"
"Yeah—I got shock this morning."
"What is that like?"
"Uh—it doesn't feel very good."
"Tell me about it, will you."
"Well, it's uh, it's like a blunt thing that hits your head—it doesn't feel very good."
"How long does that go on?"
"Oh just for a while, just for an instant, you know. . . . It's like a big thing, and uh it—takes both sides of your head, it goes *boom* like that, and all of a sudden you feel something, and after that you—don't feel anything."
"Are you conscious after that?"
"No, you're out completely."
"For about how long—have you any idea?"
"For about a half-hour."
"Then what?"
"Then you wake up—then you find you've been under shock."

PERSPECTIVES ON ECT

The medical, organizational and interactionist perspectives on ECT focus on different aspects of the treatment, within different sets of relationships and tasks at hand. The medical model is an organismic one, in which the cure of mental illness is presumed to come from changes in the structure or functioning of the brain. Like other psychiatric treatments, ECT has undergone changes over time, both in its manner of administration and in the disorders for which it is presumed effective. The convulsions and grimaces of the face noted by Friedberg (quoted above) have been eliminated by use of the new combinations of drugs, which have also greatly lessened the risk of fractured vertebrae or coronary arrest (19). Where ECT was used in the 1950s mainly for schizophrenia, and as an initial treatment, today the American Psychiatric Association recommends that its use be restricted to cases of severe depression, with limited indications for schizophrenia, and as a last resort treatment. There is some evidence, however, that even today ECT is used instead of other therapies, rather than as a last resort (20, 21).

The organizational perspective focuses on everyday control of patients on psychiatric wards. ECT can be used by nursing staff to maintain their positions of control over patients by the arousal of fear of ECT or by its sedative effect. As one Napa nurse said to a Bay Area interviewer:

(Eve Low) "Eve is to start ECT on Friday." Another staff member spoke up saying, "Boy, I wish they'd start her on it tonight, she can really be annoying. Wish you could see her at bedtime."

The interactionist perspective is concerned with the meanings of ECT and other treatments to the patient. Although there is no interactionist literature *per se* on ECT, there are numerous autobiographical accounts and case studies on which an analyst may draw. Most of the autobiographical accounts are of patients who have been forced into ECT against their will, and thus are highly critical of the procedure (22). Some of these "railroaded" expatients have joined together in political protests against ECT through patients' rights organizations. Since in both the 1950s and the 1980s about two-thirds of those receiving ECT were women (23, 24, 25), there is also a specifically feminist protest against it (26).

There are few autobiographical or case study sources on patients who volunteer for ECT or who are favorable toward the treatment. In a recent study of 166 patients in a Scottish hospital, 74 percent said the ECT had improved their condition, and 65 percent said that they would be willing to have it again. While 39 percent said it was a frightening procedure, half of these said that it was less frightening than going to a dentist. Almost a third, however, reported a lasting impairment of memory (27).

Neither the political nor the medical assessments of ECT are focused directly on interactionist issues of meaning and social relationships. The political critiques are concerned with ECT in relation to medical (and sometimes marital) dominance, while the clinical studies are concerned with outcome evaluation (28). The analysis in this paper is directed at other issues. How do patients perceive ECT—what do they think is being done to them? And what do they see as the purposes of the persons behind such doings? Do patients interpret ECT medically, organizationally, or in some other way? What, in sum, is the impact of ECT on self and family relationships?

ECT, THE SELF, AND FAMILY RELATIONS

As indicated, and with no pun intended, ECT is a shocking experience. Unlike many general medical procedures, such as pill popping, ECT has no cultural precedent available to consumers from magazines or TV advertisements. While the Bay Area patients often did not bother to discuss or worry about the pills they were receiving from Napa psychiatrists, they never failed to attend to the fact of current or proposed sessions of ECT (29).

Responses to ECT varied both between patients and over time according to the women's self-assessment of feeling better or worse as a result of it. But the most significant experiential feature of ECT, for these women, was the memory

loss attendant upon it. The interpretive work that they and their spouses engaged in, therefore, generally focused on the purposes and effects of memory loss in the context of their psychiatric treatment and of their everyday lives.

Interpretations of ECT

The situation of these hospitalized mental patients was one of uncertainty and lack of information, combined with submission to medical authorities (30). Especially for those wives committed by their husbands, hospitalization also meant arraying medical and spousal authority against them in a sort of conspiratorial betrayal (31, 32). The meaning of ECT, therefore—like the meaning of much of the hospitalization experience—reflected these themes.

The Bay Area women were completely uninformed by the Napa staff about every single detail of their ECT treatment. They were not told what it was for, how often they would get it, what it would be like, or what the expected affects on their memory, physical sensations, or menstrual cycles would be. They relied almost totally on the patient grapevine for information. Other women patients, not staff, were almost always the source of information on such matters as ECT's effect on menstruation.

(Shirley Arlen). "I haven't had a period for three months—but a lot of people that were on shock didn't have their periods right away."

One result of this lack of official information was that a number of women, including Shirley Arlen, spent some time worrying that they might have become pregnant prior to hospitalization.

Another feature of the lack of information in the hospital setting is what I have called, elsewhere, the "therapeutization of the everyday." Goffman (1961) has commented on the fact that staff tend to "symptomize" the behavior of patients, imputing psychiatric meaning to even the most mundane activities. A parallel process is that of patients' "therapeutization" of the behavior of the staff, imputing medical meaning to even the most mundane activities (33). Thus, what doctors interpret as the "side effects" of therapeutic interventions—such as the memory loss attendant upon ECT—are liable to be interpreted by the patient as intended therapeutic effects.

Since the Bay Area patients therapeutized all hospital experiences that flowed in their direction, it is not surprising that the most commonly experienced effect of ECT, the erasure of memory, was construed as the *purpose* of ECT. For example, Shirley Arlen said:

3) "I think the shock treatments are supposed to make you forget—when you do break down or whatever it is you do to get in here—I mean you're pretty sick and I think shock treatment is to make you forget a lot of things that got you sick and the way you felt and everything

like that—I mean it succeeded with me—I can't remember a lot of things—but I'd rather not. There's some things I'd like to but I think it was for the best that I can't remember a lot of things."

Among those who interpreted ECT as intended to erase their memories of their problems, some, like Shirley Arlen, were pleased with this idea. In the 1972 reinterview Shirley Arlen numbered ECT among the treatments that had helped her over the years. Joan Baker, too, wanted to get shock treatment to help her forget, and thus become a "different person":

I asked Mrs. Baker about the idea of getting shock treatments. She said, "I don't care what they do, as long as it helps me—helps me not to be depressed—helps me to be a different person, to like people, I want to forget—I don't know if I can or if I know what I mean when I say it—but my father never liking me as a child made me feel I was a monster, I was different, making me hide in my bedroom."—

A number of women, dimly aware that they had said and done embarrassing things in the prehospital phase, were glad to have forgotten the details.

Other patients inclined to the belief that such forgetfulness would do them harm, by not dealing with their problems consciously. Eve Low said that:

"I did not feel that I wanted shock, because I don't think it is to my advantage to forget the incidents that happened to me as a child because it seemed to me that—ah—those incidents that were buried in my subconscious . . . so terribly unpleasant . . . it caused me to have a complex . . . Well after I remembered these different things, it explained to me why I felt as I did."

It is an irony of shock treatment combined with psychotherapeutic interventions that the one treatment involves an imputed medical authorization to forget, while the other involves the injunction to remember. A number of the patients were perplexed about this issue. Mary Yale, for example, had "Many questions concerning whether she should think about her troubles and feelings and history (her term: "analyze"), or forget them (her term: "repress")."

As indicated medical authority as well as uncertainty was an invariant feature of the hospital situation. In the late 1950s (although not in the 1980s) state mental patients could be given ECT without their consent. Thus, the use of ECT was experienced as coercive medical control. Eve Low discussed the unpleasant effects of shock, and the way in which "forcing" the treatment on her exacerbated her "paranoia":

"I don't believe that I can speak as coherently—I don't think my train of thought is connected. I am more apprehensive. I am more fearful at . . . what will happen to me . . . because . . . until I received shock I had never really been forced to do anything."

Like the feminist critics of shock treatment in the 1970s and 1980s, Eve Low was also concerned with the combined impact of medical and spousal authority in her "treatment":

She went on to say that she'd been getting shock, though against her wish, and that she feels its purpose is to make her forget things, and to change her attitude, including her resentment toward her husband for committing her.

But medical control has subtle as well as overtly coercive aspects. The medical model of mental illness proposes a scientific treatment which is both appropriate and benign. Lidz et al. (49) indicate that patients are persuaded to consent to ECT by psychiatrists who asserted that they could do nothing else for the patients. Patients who are feeling severe distress and who are given no other alterations may agree to ECT and see it as helpful. This seemed to be the case with the Bay Area patients:

(Ruth Quinn) Mrs. Quinn stated that she is afraid of shock treatment but she feels it has helped her a great deal.

(Rita Vick) I asked Mrs. Vick whether she thinks ECT is helping her. She said, "I have noticed some improvement. I can be a little gayer for longer periods."

But reactions to the helpfulness of ECT varied with the patient's feelings in the given situation. In an interview the next week, Rita Vick said:

"I thought the shock treatments would help." (Have they?) "I don't think so. They made me forget some things, but not enough. I haven't had enough, I guess." (Are they supposed to make you forget?) "That's what I heard—that's what everybody tells you—that it's to make you forget."

ECT, Memory and the Self

The self upon which ECT impacted had not only a contemporary dimension—mental patienthood—but also a historical one. The memory loss attendant upon ECT was interpreted by these patients in a context that included the historical self and its network of social relationships, and general cultural values such as the preference for remembering over forgetting. The Bay Area patients' memory losses related to everyday life as well as to their emotional troubles, and were integrated into historical self-conceptions related to personal competence at remembering.

The women were divided on the advisability of forgetting one's difficulties, but uniformly disliked the loss of everyday memory, as well as associated effects such as losing one's train of thought, incoherent speech or slowness of affect. What specifically was forgotten varied from the matters of everyday routine to the existence of one or more of one's children (see below). Donna Urey, two days after her second shock treatment said:

"Ever since I had that shock I can't even remember reading things."

"How does it feel to suddenly be like this?"

"It feels awful. Because usually I can remember pretty much of everything but knowing something and not remembering is pretty terrible."

"When did you first notice it?"

"Right after I got my first shock treatment."

Persons may characterize themselves, or be characterized by others as having "good" or "bad" memories. Donna Urey characterized herself in the interview above as having a good memory for things she had read, and was therefore bothered by the ECT-related loss of memory in that area. In another interview, however, she characterized herself as typically forgetful; the ECT loss of memory, therefore, was just another in a series of "shocking" but normal-for-her forgettings:

(Donna Urey). "How does it feel to have memory sort of—go out on you like this?"

"I don't know, it feels shocking—when I was at home—it happened the same way."

"It did? Can you tell me about what happened at home?"

"If I—if the kids don't remind me of something—then I forget—like if their Daddy tells me to phone them at work, during the day, and if they don't remind me then I forget—"... "Well you know one thing I would be kind of interested in, is if you could kind of collect your impressions of what it's like to be—to suddenly—have some holes in your memory?"

"It's not unusual."

"Not unusual for you?"

"No."

In asking the patients questions about ECT, the interviewers sometimes encountered an interesting research problem: they knew from the records that the women had had ECT, but when they asked about the treatment they discovered that ECT itself had been forgotten. In the 1972 reinterview, Wanda Karr described herself as unable to tell whether or not ECT treatments had affected her memory, since she didn't remember having the treatments:

She remembers only the last ECT, for which she was awake: "I remember the clamps on my head, the sparks as it started, and I was very frightened. Afterwards I woke up with the most terrible headache I ever had. It was like being hit on the head with a bat. It was really an awful experience." I asked if it had affected her memory. She said that immediately after the last one she couldn't remember things, but she doesn't know about the others since she doesn't remember the treatment at all. In talking about memory she said, "You know I can't remember anything about the hospital."

Like many contemporary psychiatric proponents of ECT (34), Mrs. Karr attributed her lack of memory of the hospital to her psychiatric disturbance rather than to the ECT treatments.

There is evidence from the Bay Area interviews that ECT may function repressively—that is, allow the person to forget disturbing events or persons.

Rita Vick, who was illegitimate and who had lost custody of five of her seven children, complained that "I can't remember my children's birthdays or my birthday." After a weekend visit,³ which Mr. Yale described as very tense, the interviewer talked to Mary Yale:

I asked very early about her visit home, and she looked puzzled. I recalled that we had talked last week about her plans to visit home, and she couldn't recall this. She stated flatly that she had not been home over the weekend. Later in the interview she was slightly troubled and doubtful over the questions I had raised about the weekend, and was wondering if it was perhaps possible that she had been home. What she did recall of the weekend was a very vivid nightmare, the first since hospitalization.

The patients may have been aware that their forgetting was at times repressive. Mary Yale said that she was bothered by her loss of memory because "I want to know why I forget those things."

Troubling life-events and relationships commonly forgotten by these women included the existence of their husbands and children, their own names, and their psychiatrists. Elsewhere, I have analyzed these women's resentments of their housewife-mother role, their sense of isolation and lack of identity, and of the combined medical-marital power that facilitated their hospitalization (35).

Forgetting can have a reparative or a disintegrative function for the self and social relations. Repressive forgetting may be useful in restoring a person's or a family's equilibrium following traumatic experiences. The specific impact of forgetting events in the past depends upon the salience of the events to the person in the present; while forgetting traumatic events may be restorative, forgetting mundane events may be traumatic. As the phenomenologists have pointed out, the reality of everyday life is the bedrock upon which we humans build our sense of a secure self in the world. Losing touch with everyday life—with a book read, with a church service attended—can threaten that sense.

Forgetting persons, which was frequent, seems to be a truly interactional difficulty; the image that the patient does not want to project is that of a person unable to carry on routine social interaction. This may be complicated by fears of insulting the other—that s/he is not important enough to be remembered. It is clear that *one* function of remembering someone's name is to demonstrate that one has the social competence necessary to participate in an ongoing social relationship: to the other's name are attached items of the common culture. There are probably other devices that people use in an unaware way which perform this same function, such as recalling an event experienced in common, or making a private joke. One function of the filling-in phenomenon—reminding the ECT expatient of past events—was to aid the forgetter in maintaining a favorable self-image: the image of a competent person.

It is difficult to assess, in everyday life as opposed to experimental settings, the restoration of memory in ECT patients. The ECT patients in the Bay Area

study were embedded in social networks that included husband, children, and other relatives who could and did perform a filling-in role. Thus, the restoration of memory may be in part—or entirely—a process of relearning, after ECT, under the tutelage of others.

ECT and Family Relationships

Memory is not only something experienced by the self, it is also an aspect of social interaction. Thus, the effects of ECT upon memory and the expectation of memory loss were both at issue in the Bay Area women's relationships—especially their family relationships, and especially in the expatient phase of the moral career. In addition, ECT-related memory loss was an issue, at times, in the interview situation.

It appeared to some of the Bay Area interviewers that their respondents used ECT-related memory loss as an excuse to forget. Although difficult to document through ~~other than inference~~, their suspicion was of "purposeful" forgetting and the use of ECT as a rationalizing account:

(Donna Urey). Throughout the interview the effects of ECT were marked in her slowed and somewhat thickened, flattened affect, and her mild confusion. She seemed to be discovering her memory loss only as I asked her for information which she could not remember. When, after a while, I switched to inquiries about her family, she brightened and said with comparative enthusiasm (and perhaps relief). "Now that's something I can tell you about!" . . . Although her memory loss is obvious, there were times when I felt that she was helping this along. This was principally when I was probing about her and her husband's feelings about her working.

The context for producing forgetfulness, as indicated by this example, was not wanting to talk about subjects that were painful, embarrassing, or revealing.

The social production of forgetfulness in order to avoid interview topics is, thus, paradigmatic of the social production of forgetfulness in other social situations. Expatrients who have had ECT can conveniently "forget," and use ECT as an excuse; one Bay Area patient, waiting to be served with a subpoena in a civil case, said that she planned to tell the court that she had had ECT and therefore "couldn't remember a thing." She told the interviewer, however, that she "actually" recalled it all.

But the impact of ECT-related memory loss on family and marital relationships was not confined to the expatrients' production of forgetfulness. Husbands and other relatives could and did use their wives' memory loss as an occasion for purposely not reminding the wives of things that the husbands did not want remembered, or (very rarely) for reminding the wives of events that had not in fact occurred. Although generally couched in the language of doing it for her own good, these interactive memory strategies were related to the relative's relational purposes-at-hand.

All parties use ECT related amnesia (which is assumed) as a rationalization for deception.

Evidently, the memory purposes of husbands and wives could be at odds, with wives wanting to remember and failing to and husbands wanting them to forget and not reminding them—or any logical combination of these stances. The outcome of such divergent relational purposes was conflict over the content of past marital communications; thus, ECT-related memory loss became part of the everyday dynamics of marital interaction for some of the Bay Area families in the hospital and posthospital phases of the moral career, especially in the weeks immediately following release.

Husbands might wish to have their wives forget the emotional troubles, including marital strife, which precipitated hospitalization. Mr. Karr commented on his wife's long-term memory loss as proof of her successful cure by ECT, saying that her memory was still gone, especially for the period when she felt ill, and that "they did a good job there." These husbands used their wives' memory loss to establish their own definitions of past situations in the marital relationship:

(Mr. Karr). Mr. Karr said that Wanda "couldn't remember anything" that happened after Christmas. He feels this is all for the good. "We (that is mama) have decided if she remembers what she did OK, but we're not going to tell her." He doubts (or perhaps I should say hopes) that she will not remember, not that she did anything to be ashamed of, of course. But she "wasn't herself" then.

Other relatives, too, found it in their interest to have the expatients forget; thus they could freely re-define past situations without challenge:

(Eve Low). "Now I am sure that my memory (of being molested, as a child, by her mother's brother) is true, even though my mother, who came down last week, said that it is all nonsense. However, before we left the house last Sunday night, she was explaining to [other relatives] why she wanted me up here, you know, she wants me to have the full treatment she says. I should think that would entail a great deal more than what I've had apparently, but she said that she thought it would make me forget all those things. . . I'm afraid my mother wants me to have more shock so I'll forget all those things that happened. But I don't want this."

Different relatives had different interests in either recalling incidents forgotten because of ECT, or in collaborating with the patient's forgetfulness.

During the post-hospital episode, on the occasion of her mother 'bringing up' embarrassing incidents connected with her psychotic episode, Wanda told her: "Mama, stop telling me those things! I went to the hospital and they made me forget them. Now don't keep bringing them up! You're not doing me any good." When asked if her mother had stopped, Mrs. Karr said, "Well, in her way." Mr. Karr, for his part, expressed pleasure to the research interviewer that electroshock therapy had made his wife forget her hostile outbursts against him in the pre-hospital period.

In one family, the forgetfulness attendant upon ECT treatment had a dampening effect on an extra-marital romance between a Bay Area expatient and a male expatient, thus contributing to the possible repair of a disintegrating marital relationship. Upon the resumption of their contact in the expatient phase of the moral career, these two patients were embarrassed by mutual memory lapses, perhaps as much by their status as reminders than anything else:

(Ruth Quinn) [on her meeting with the male expatient] "it was rather strained at first. I found that there was a great deal he didn't remember. He was in the process of 12 shock treatments when I met him. And when I met him I think I was about two or three weeks off shock. So perhaps I don't remember some of the things but it seems that I do. But he didn't remember half the things that he told me. He didn't remember that I had two children. But he thought I was divorced and was surprised to hear that I'm not divorced."

The original Bay Area researchers noted that ECT can have a positive effect on the restoration of harmonious family relationships once the patient has been restored to the family, citing "the specific effects of electroshock therapy in blurring memories incongruent with the selves the patients and her intimates are reconstituting." (36).

The effect of ECT-related memory loss on family relations was not always counter-disintegrative; at times it had negative implications for the emotional ties between family members. As indicated above, several of the Bay Area patients forgot, after one or more ECT treatments, that they had children. One patient, admitted for post-partum depression, forgot that she had given birth to her child, who was nine months old at the time she was released to resume care of him. Although she had been reminded by others of his existence, she appeared to have lost her affective memory of him as her child:

(Shirley Arlen) "I guess I feel sort of strange with him. In being with him. I don't know. I guess I just feel sort of strange with him. . . I just don't even feel like he's mine, for some reason. . . I think he's nine months now. . . I really don't know. I can't even remember when he was born."

The impact of ECT on family relationships was not confined to the negotiation of memory. ECT also affected marital communication and shared interpretive processes. For some of the couples, ECT provided a convenient rationale for the wife's untoward behavior. For some of the women, the fear of ECT hampered communication with their husbands. while for some of the husbands, fear of their wives' reactions hampered the attempt to repair ECT-related memory deficits.

Both patients and their husbands utilized ECT to explain away a variety of problematic behaviors, including memory loss itself. The range of awareness of memory lapses in these families seemed increased over normal; that is, not

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only were memory lapses explained via ECT that otherwise might have been explained differently (say, tiredness or upset), but many memory lapses that might otherwise not have been explained at all were remarked and categorized via ECT:

Mr. Yale is eager to ask the hospital doctor one question: how long the shock treatment will go on. He has mentioned this on several previous interviews, and the interviewer asks why this particular question is so important. He said it was because of her lack of memory, and "I have the completely unscientific idea that when the shock treatment stops her memory will come back and then she will be well."

Other sorts of undesirable behavior were rationalized by patients or their intimates as a consequence of ECT rather than of renewed emotional disturbance:

Mr. Yale visited Mary on the ward a few days ago and finds her behavior very disturbing. He called his friend . . . tonight and asked him if he thought Mary's reaction was from shock treatment.

Mary Yale "some days I'm not functioning well, not thinking clearly. It's not all the time, not every day. Maybe I want to blame it on shock."

The fear of being rehospitalized and receiving ECT against their will affected at least three of the Bay Area patients throughout the decade following their first admission. Rather than communicating various emotional disturbances and thoughts to their husbands, these women refrained from communication for fear of a resumption of medical-marital control of their lives. Mary Yale, in 1972, said that she had "a dread fear of shock" and was afraid to express her feelings to her husband for fear of reprisal in the form of ECT. She added, "Shock treatment is a helluva way to treat marital problems—the problems involved both of us."

Marital communication can also be affected the other way around. In the expatient phase of the patient's moral career, the Bay Area husbands tended to treat their wives with "kid gloves," refraining from saying or doing things that might "set them off." Sometimes, the husband's kid glove approach conflicted with the wife's search for her past. In one instance, Rita Vick had forgotten, after ECT, the five of her seven children who had been removed from her custody. One day she found an album in the Vick house and asked her husband "who were all those children?" For fear of upsetting her with renewed thoughts of the custody loss, Mr. Vick told her that they were a neighbor's children. Later, when Mrs. Vick discovered through another relative that these were in fact her children, she was "furious" with her husband for lying to her.

DISCUSSION

ger and Kellner (1970) analyze the ways in which marriage creates a stable world of meanings for the participants, while Goffman (1971) notes the "havoc" is wreaked on family life by the symptomatic prepatient member. Hospitalization interrupts both the havoc and its world, while treatments such as ECT intervene between the prehospital and posthospital reality-negotiations of marital partners. In the wake of hospital treatment, the couple "constructs only present reality but reconstructs past reality as well, fabricating a common memory that integrates the recollections of the two individual pasts." When the recollections of one partner are to some degree erased, the amnic (reconstruction of reality) shifts a little, or a lot.

In practical terms, if certain treatments affect not only the self but the marital relationship, then it would seem useful to develop a further perspective on hospitalization in addition to the medical-model, organizational, and political perspectives. This is the interactional perspective on mental health treatments. Treatments are evaluated according to their intrusiveness into the individual's sphere of personal competence and liberty (38), then they should also be evaluated for their intrusiveness into the individual's sphere of relationships in everyday life. And, since ECT is particularly implicated in this aspect of psychiatric treatment, it would seem useful to encourage further research into this aspect of ECT and other highly invasive treatments.

ECT is an intrusive treatment that affects both the social relationship and the sense of self of the mental patient. For some, this invasion is welcomed as a means of forgetting, or, alternatively, as a means of manipulating the marital interpretive world. For others, it is unwelcome. For the majority of Bay Area women and other patients who have undergone ECT, the arbitrariness of the procedures and the loss of memory represent both a loss of continuity in the experience of life, and a loss of control over past, present and future; over body, mind and emotions.

Empirical studies, although sparse and variable by method and by geographical location, indicate a resurgence of ECT in the late 1970s and early 1980s following a decline in the mid-1960s to mid-1970s. In a New York study, Morrissey and his colleagues indicated that there was a 38 percent decrease (from 26,400 to 16,482) in the reported number of ECT treatments between 1962 and 1977, with a decline in the number of patients from 3,035 to 2,194 (39 percent) (39). In California between 1977 and 1983, however, ECT treatments rose from 12,879 to 15,446, an increase of 19.9 percent, while the number of patients rose by 16.9 percent, from 2,422 to 2,831.⁴

The increase in ECT use is in a different type of hospital, and with a different clientele, than in the 1950s. In the 1950s, ECT was utilized mainly in the state hospitals, often on an involuntary basis, and with a clientele that was more poorer and possibly minority (40) than middle class. In the 1980s, on the other

hand, ECT is utilized mainly in private hospitals, with a white, middle class, elderly clientele (41, 42, 43). The only clear commonality throughout the 1950s-1980s is that ECT is, and was, used predominantly (from 60-70 percent) on women (44).

ECT is regaining popularity as a treatment which is fast, inexpensive, and easily reimbursable by third-party insurance payment schemes (45, 46). DRGs should increase this trend (47). Robitscher (1980) comments that ECT fulfills both economic and social control functions for private hospitals, suggesting that an economic model of interpreting therapies is a useful supplement to the medical model. Noting that private, proprietary hospitals sometimes shock up to three quarters of their inpatients, he notes that:

The economics of electroconvulsive therapy show why this treatment modality appeals to the venal. The electroshock machine is inexpensive. The patient who is receiving electroshock is easy to manage, sleeps a great deal, does not need much nursing care, and uses the hospital much as a hotel or motel. Blue Cross, Blue Shield, Medicaid and other third-party plans pay without any questioning (48).

There have been changes since the 1950s not only in the clientele and location of ECT treatments but also in the methods of administration and the informed consent procedures (49). There have also been changes in the structure of marriage, and in the place of women in society. Yet at the same time, the family remains at the center of life's nomic ordering, and ECT continues to affect memory. In the face of the resurgence of this most invasive treatment it would perhaps be wise to attempt a reassessment of its impact on the self and family relationships.

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NOTES

1. The pseudonyms used in this study are those used in earlier analyses of the Bay Area data (see Sampson et al., 1964).
2. This lack of information about ECT in hospital settings has improved considerably since the 1950s (Lidz, 1984).
3. In the 1950s, psychiatric inpatients were allowed weekend visits home under certain conditions.
4. This increase occurred at a time when the California inpatient population was declining steadily year by year (Warren, 1987).

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Final Report

January 2002

Review of Consumers' Perspectives

On

Electro Convulsive Therapy

Service User Research Enterprise (SURE)

Institute of Psychiatry

Commissioned by Department of Health

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Review of consumers' perspectives on ECT

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Review of consumers' perspectives on ECT

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EXECUTIVE SUMMARY

Introduction

In June 2001, the Service User Research Enterprise (SURE) at the Institute of Psychiatry was commissioned to conduct a review of consumers' perspectives on ECT (Electro-convulsive therapy). The review team included clinicians and consumers. Both consumer members of the team have direct experience of ECT. This commission forms part of a wider Review of ECT being undertaken on behalf of the Department of Health.

ECT is a treatment that attracts controversy. There is a consumer viewpoint that is highly critical of ECT. However the Royal College of Psychiatrists' fact sheet on ECT maintains it is a safe and effective treatment that is sometimes life-saving.

Aims

This Review will focus on the following aims:

- Systematically to describe and summarise consumers' perspectives on ECT
- To understand the sources and nature of the controversy about ECT between some consumers and professional bodies representing practitioners of ECT.

The review has the following four themes:

- Persistent memory impairment following ECT (chapter 5)
- Consent and information about ECT (chapter 6)
- Perceived benefits of ECT (chapter 7)
- Emotional responses to the experience of ECT (considered in chapters 5, 6 and 7)

Assembling the material

The following sources of information were used:

- Studies that have tried to ascertain consumers' views about ECT in their own terms.
- 'Testimonies' or first-hand accounts of the experience of receiving ECT, sourced from internet sites, various print media and a video archive
- Advice from an internal Reference Group

Methodological evaluation

35 research studies were identified. Nine were reports by individual consumers, consumer organisations and collaborative works, the remainder were 'attitude' studies conducted by clinical researchers. All the studies were reviewed in terms of their methodology and found

to be very diverse and of variable quality. All the studies start with certain assumptions and values concerning ECT and this leads to both differences in the participant selection and interpretation of data.

Testimony data is now available in a wide variety of formats including a video archive and 'threads' of correspondence sourced from email forums. E-mail forums introduce the arena of group discussion or beliefs into the data. The contexts in which the testimonies are elicited do constrain what can be expressed and how it is expressed. However the different sources each provide checks on the perspectives expressed in the other formats.

Template for analysis of consumer perspectives on ECT

A template for analysing the substantive themes was developed which ensured that no single approach to ascertaining consumers' views on ECT was privileged. Where research studies using a range of methodologies produce similar results, the review makes a statement of the form 'at least, X% of consumers experience Y'. Where different methodological contexts produce different results, conclusions cannot be drawn with confidence. The different strategies used by clinical and consumer research to summarise data are also examined to see how diverse conclusions may be reached on the basis of very similar data.

The quantitative analyses are combined with the testimony data, using a form of qualitative analysis that occupies a middle ground between formal content analysis and discourse analysis. It takes from content analysis the existence of a set of themes decided in advance of examination of the data. At the same time, discourse analytic methods allow the content and detail of themes to emerge from the data and attend to the interactive nature of e-mail forums and interviews.

Combining research and testimony data gives more confidence when similarities of results are disclosed. Conversely, discrepant data from a range of sources must be treated with caution.

Persistent memory loss

In all types of information gathered for the Review it is evident that memory loss is a persistent reported side effect for at least one third of recipients of ECT. For some people, memory loss causes great distress and profoundly affects their sense of identity. Despite a considerable overlap in basic data, clinical and consumer studies tend to be polarised in their conclusions. Clinical research typically refers to a 'minority' or judges the problem to be insignificant or limited whereas consumer research concludes that persistent memory loss is a significant problem for the recipients of ECT.

Neuropsychological assessments of memory loss following ECT used in standard clinical studies tend to measure encoding and retrieval of information learnt after receiving ECT. This is called anterograde memory. Few studies measure the loss of memories which were laid down prior to the ECT (retrograde memory). However, consumers' accounts of memory loss, which correspond across all forms of testimony data, focus on the distress caused by the absence of significant portions of retrograde biographical memory rather than anterograde problems. All attitude research studies report substantial proportions of consumers experiencing this persistent memory loss. Even when levels of depression are controlled in these analyses, significant numbers of people who have received ECT are found to have memory loss into the organic ranges on a number of memory tests.

Information and consent

In almost all research papers, there is a consistent finding that 45-55% of users feel they have been given an inadequate explanation of ECT. There are even higher figures from some consumer-led research. This may be because their sample is drawn from a pool of people who have high expectations about what level of information should be provided about ECT.

It is not clear how more detailed information might impact on consumers' decisions to undergo ECT. In the past information has mainly been provided by treating doctors or leaflets written by professional bodies. More recently the internet has become a new source of information. A short period on the World Wide Web will yield information about ECT from an extremely wide range of sources. There are strong indications from the testimony data that the Internet will have a dramatic impact on whether consumers decide to consent to ECT.

On the question of consent, it is reasonable to conclude that a significant number of patients who sign a form consenting to ECT do so under pressure or in the belief that they cannot refuse. Some clinical papers argue that even though many patients felt they had no choice or where resigned to consenting to ECT they were nevertheless content to leave the decision to their doctor. Other evidence suggests that consumers lack the confidence to contradict the prescriptions of professionals even when they wish to do so.

This Review had difficulty in assessing the effects of legal compulsion because of cross-national differences in sample structures, the nature of the data and the variable proportions of consumers subject to legal compulsion between studies even within the UK.

Perceived benefits of ECT

Methodological variables exert a powerful effect upon consumers' expressed satisfaction with ECT. This conclusion can be drawn with confidence particularly in relation to *when* consumers are asked about or express their view on having the treatment again. In clinical and consumer research studies and in the testimony data, the length of time that has elapsed since treatment is strongly associated with expressed satisfaction. Studies which interview consumers immediately after treatment, especially if they do so in a medical setting, using brief interviews, conducted by the treating doctor, over-estimate to a considerable degree the extent of satisfaction with ECT. Estimates of 80% to 95% of consumers as content with the treatment are, in the judgement of this Review, not valid.

The testimony data makes it clear that 'perceived benefit' from ECT may be both discrepant from and much more complex than clinical concepts of symptom improvement. The testimony data does display a continuum of opinion about perceived benefits. However 61% of those providing testimonies said they would not have ECT again and 43% describe their experience of ECT in extremely negative terms. In addition there is evidence that individuals who would have ECT again will tend to trade-off benefits and risks. For example some consumers say that they are willing to suffer a degree of permanent memory loss in exchange for some relief from depression. Some consumers may be so concerned with the manner in which the treatment was given that whether or not it helped them is not the major issue. There is not a uni-dimensional 'consumer attitude towards ECT' even on the question of whether or not it is helpful.

The testimonies uncovered issues that are never touched upon by clinical research. These include extreme trauma, lying about improvement in order to stop treatment, becoming manic or feeling more suicidal following treatment, the desire to take legal action and the need to seek support and validation from other individuals and organisations.

Conclusions

Consumers who are opposed to ECT are often characterised as a vocal minority. However, this Review found that dissatisfaction with ECT maybe more widespread than is often supposed. Consumers' views are not simple and there is no one 'consumer perspective' on ECT. However, of those providing testimonies, few were equivocal about the treatment, .

Professional failure to acknowledge the different facets of dissatisfaction on the part of recipients of ECT may be a reason for the emergence of organisations providing support and a

forum for those who experience the treatment as negative and coercive.

Future research

It is clear from the review that there are many methodological inadequacies in the studies of ECT. The variation in the complexity of the questions, the timing of the assessments and the professional status of the interviewer need to be considered in the design. Future research questions should include:

- What is the extent of autobiographical memory loss?
- What information on the treatment would potential recipients require in order that they feel adequately informed?
- What trade-offs in benefits and risks are likely to be made by consumers leading to their acceptance or refusal of ECT?
- What are the limits of these trade-offs in relation to compulsion?

The answers to these questions have implications for service provision in relation to information, choice, support, and help for any unwanted effects should a consumer undergo ECT.

CHAPTER 1

INTRODUCTION

BACKGROUND

In June 2001, the Service User Research Enterprise (SURE) at the Institute of Psychiatry was commissioned to conduct a review of consumers' perspectives on ECT (Electro-convulsive therapy). This commission forms part of a wider Review of ECT being undertaken on behalf of the Department of Health.

There exists a psychiatric consensus which holds that ECT is a safe, effective and sometimes life-saving treatment, exemplified by the Royal College of Psychiatrists Factsheet on ECT (see Appendix 6). There is also a clear and sometimes very public consumer viewpoint that is highly critical of ECT. In the context of such polarisation, this Review will focus on describing as comprehensively and systematically as possible what is known about users' views regarding the treatment and attempting to understand the sources of controversy.

AIMS

1. Systematically to describe and summarise consumers' perspectives on ECT.
2. To understand the sources and nature of the controversy about ECT between some consumers and professional bodies representing practitioners of ECT.

To accomplish the above Aims, the following sources of information will be used:

1. Studies that have tried to ascertain consumers' views about ECT in their own terms.
2. First-hand accounts of the experience of receiving ECT, unedited or commented upon by others. These will be called 'Testimonies'.
3. Advice from an internal Reference Group (RG) composed of user representatives of organisations with an interest in ECT (including organisations who are in favour of ECT) and researchers who have conducted work on ECT from the consumer's perspective and/or have expertise in qualitative research.

CONTEXTS

Rise of the user movement and consumer-led research

Consumer groups are engaged in a range of activities all designed to put forward the consumer perspective on mental health services. The number of groups and the scope of their activities have grown rapidly in the last 15 years. A recent survey (SCMH, personal communication) has identified more than 1,000 consumer groups in England. It is

government policy that consumers should be involved in policy development. In this context, the aim of consumer-led research has been to establish users' views on the whole variety of mental health services. Consumer-led research is distinguished from more traditional research in that the researchers are themselves consumers. Consumers also conduct critiques of orthodox research.

Previous research

In 1986, two review papers appeared that described studies on attitudes towards ECT (Freeman, 1986; Freeman and Cheshire, 1986). Freeman (1986) focused particularly on the attitudes of users and included in his review a paper authored by a recipient of the treatment. He identified five studies that had assessed consumer attitudes up to 1986, all of them conducted by clinical researchers.

The present Review has located 21 clinical research studies published since the 1986 reviews and an additional nine reports from consumer or voluntary organisations or consumers collaborating with clinical academics. These consumer studies only began to appear eight years ago and most were not identified by any formal search procedure.

Dimensions of new technology

New technology such as video and the internet have increased the ability of consumers and organisations which represent them to both produce and access digital information. The internet has extended these networks internationally and made it much easier for individuals to become active participants in debates about treatments and services.

Summary

The emergence of consumer-led research and of a mental health consumer movement have changed both the context for a contemporary review of consumers' perspectives on ECT and its actual content. New information technologies have broadened fundamentally consumers' access to information about ECT and enabled them to express their views about their treatment in new formats and in new ways. Both these developments mean that there have been profound changes in the scope of a Review such as this since the initial ones were published in 1986.

STRUCTURE OF THE REPORT

There are two main sections to the report. The first is methodological and begins by describing the strategies used to assemble the material for the review. There follows a methodological discussion of both the research studies and the testimony data. Difficulties

encountered in compiling the research studies made this an essential part of the Review. Without this, it would not be possible to assess the specific findings and results with sufficient clarity. Similarly, the new formats which testimony now takes require further methodological discussion on which to base the subsequent analyses.

The body of the report comprises the main findings. It is a summary and analysis of specific issues or themes concerning ECT of interest to consumers and the DH Review generally. The themes are summarised and analysed by means of a consistent template that is described in chapter 4. They are:

- Persistent memory impairment following ECT
- Consent and information about ECT
- Perceived benefits of ECT

The additional theme of emotional responses to the experience of ECT will be considered in each chapter.

These themes or 'outcomes' are clearly different to those investigated in most clinical research. There is overlap between the two types of research in some specific areas such as the assessment of treatment benefits. But whereas clinical research tends to measure this as changes on symptom rating scales, consumer-led research asks about the consumer's own perception of benefit.

The report concludes with a summary of what can validly be said about consumers' perspectives on ECT and questions that remain open and require further research or review.

CHAPTER 2

ASSEMBLING THE MATERIAL

General inclusion criteria

1. Research participants and all consumers who provide testimony have received ECT.
2. The research ascertains consumer views by asking directly about experience of ECT.
3. Papers have a publication date of 1975 or after.
4. All materials are in the English language

RESEARCH STUDIES

DATABASES

Four initial databases were used to compile a list of studies. Where the database included a facility for mapping search terms onto subject headings, this was used. Free text searches were always used. The databases were: Psychinfo, Medline, Web of Science and the King's Fund.

Search strategies

Main search term	Subsidiary term	Main search term
ECT		Client / Patient Attitudes
(Electro-convulsive therapy)	Memory loss	(Client satisfaction)
(Electro-shock therapy)	Information	(Client rights)
(Shock therapy)	Consent	(Treatment drop-outs)
	Benefit/helpfulness	

Table 2.1: Search strategy for identifying research studies

Each database was searched once with the main terms. Subsidiary terms were added individually.

Scientific databases: search results and problems

The initial searches delivered 16 studies but it was known that some UK research had not been located. The Royal College of Psychiatrists' website journal pages were then searched to identify work that might have appeared in the British Journal of Psychiatry or Psychiatric Bulletin. Photocopies of the articles located were obtained. It became clear that referencing of previous work was extremely patchy in most papers. The bibliographies were therefore

searched by hand to locate additional work. The process led to a final set of 26 clinical research studies. Nearly all the empirical papers take the form of 'attitude studies' using the concepts and techniques of social psychology. There are two review papers and one based on four case histories.

Two sets of studies were ambiguous regarding the inclusion criteria of eliciting consumers' views *in their own terms*. In the first set, Cowley (1985) and Lisanby et al (2000) heavily filter the views expressed by their respondents through additional criteria of the researchers. Although apparently violating inclusion criterion 2, these papers were included on the advice of the Advisory Group.

In the second set, research aiming to educate consumers about ECT (eg. Battersby et al, 1993) assumes that the researcher's views about the treatment are correct and studies consumer views from the perspective that differences between these and the researchers views are misconceptions and reflect a need for education about ECT. Educative research was included on the advice of the Advisory Group.

Subsidiary themes

Adding the subsidiary themes into the main search always reduced the results to zero. Therefore, studies addressing particular themes had to be located by hand.

King's Fund Library

There were nine entries in the King's Fund library under ECT or a synonym. One was a research papers written by social scientists (Rogers and Pilgrim, 1993) and conducted in collaboration with MIND. The others were review or policy documents. Four were from MIND and included quotations from ECT recipients. However, most of these were taken from Rogers and Pilgrim.

Locating consumer-led research

Consumer-led research is rarely published in the peer-reviewed literature. It was anticipated that the King's Fund holdings would include consumer-led work but this was not the case. SURE therefore used its own prior knowledge and contacts to assemble this part of the research database and asked the Reference Group for assistance. Nine consumer-led or collaborative studies were identified.

All but one of the consumer-led studies was conducted in the UK and so there is a possibility of publication-bias here.. Three of them included ECT as part of more general surveys of

consumer views of treatment in psychiatry (Rogers and Pilgrim, 1993; MHF, 1997; MDF, in press). Although, it was not possible to identify the methodology for ECT Anonymous survey, it was included on the advice of the Advisory Group. The US study (Donahue, 2000) is a review of the literature on memory loss following ECT conducted by a consumer who experienced severe memory loss following her own treatment. This paper, the only one picked up through the Medline and Psycinfo searches is, in our view, very thorough.

Research studies - conclusions

The final set of 35 research studies is described in Appendix 1. Some of the difficulties encountered in assembling this material implied at an early stage that it was not a systematic body of work. Papers are not always mapped onto subject headings consistently in the scientific databases. Referencing of previous papers is poor and the emergence of user-led research that does not appear in the peer-reviewed literature makes the picture even more complex.

ASSEMBLING CONSUMER TESTIMONY

Testimony was defined as an individual speaking or writing directly about their own personal experience of ECT. Such testimony might be contained in a variety of source materials: articles in consumer group newsletters, on websites and e-mail discussion forums, as part of oral history archives and so forth. Consumer group campaign literature was generally not admissible. However, if it contained individual testimony as defined above this was included.

There is obviously a problem of selection-bias with material gleaned from the internet. However, contrary to expectations, e-mail forums are very lively and the very anonymity of the internet seems to allow people to contribute as they like. Some are very supportive and some downright angry. We also conducted a positive search for favourable testimonies.

A total of 139 testimonies contained in a variety of media were finally included and analysed for the review. They were assembled in the following way:

Electronic searches

Internet

The Worldwide Web was approached in much the same manner as a consumer or potential consumer of ECT might if they were looking for information about ECT. Searches using the term 'ECT' or 'Shock treatment' were done via the popular search engines YAHOO, Lycos and GOOGLE. The following websites were identified as appearing repeatedly in the results:

www.ect.org

www.members.aol.com/noshock

Testimony appeared on the websites in the form of personal accounts of ECT. Newspaper and magazine articles were also available either as part of the website or via hyperlinks. It became clear that most of these websites were opposed to ECT and so a specific search was undertaken to try to find testimony which was favourable to ECT. This search concentrated on non-consumer sources, such as public health and hospital sites. Some admissible material was located, the main website being:

www.healthplace.com/depression/ect

E-mail forums

The chief sources of testimony from the web sites identified were e-mail forums. These allow people to post e-mail messages that can be read by anyone who accesses the forum. In addition visitors to the forum can post their own messages, creating 'threads' of messages.

Legal testimony

Several websites had links to legal cases in the USA where advocacy groups were attempting to prevent an individual from having ECT without consent. If these contained a consumer's accounts of their own first-hand experience, they were included. Several public health hearings about ECT in North America had consumer depositions that sometimes contained first hand testimony. Legal testimony is clearly not unbiased but it can bring forward issues that do not come to light in other forms of testimony.

Newspaper database

The Proquest Newspaper database contains a complete record of: Daily and Sunday Telegraph, Daily and Sunday Mail, Evening Standard (London), Financial Times, Guardian, Independent (London), Observer and Daily and Sunday Times. It was searched on 13th June 2001, between that date and 1st January 1999 using the search terms 'ECT' and 'Shock Treatment'. This search yielded 21 results but very few articles contained admissible testimony.

Reference group

The Reference Group was asked to suggest possible sources of testimony. This resulted in a large amount of printed material and a further website:

www.mindfreedom.org

By request to other organisations and individuals

A message asking for testimony was posted on *Uksurvivors* egroup, a US notice board and to contacts in Canada. The voluntary organisation, *Mental Health Media* was contacted and asked to supply any testimony held in their library. Sara Dunn, editor of *Open Mind*, *National MIND*'s bi-monthly magazine, was asked to give references for any issues that contained ECT testimony. These networks produced additions to the internet material and two further sources of testimony.

Mental Health Media Testimony Archive

The Mental Health Media Testimony Archive contains 50 four-hour interviews with people who have experience of long-stay psychiatric institutions in all parts of the UK. The interviewers were all themselves consumers. The archive is in the form of videotape, full transcripts and transcript summaries. The material is held at the British Library and by Mental Health Media. The entire archives' transcript summaries were read and 23 that contained substantial references to ECT were selected. The full transcripts for these 23 interviews were then searched on disc and the sections that related to the individual's experience of ECT were extracted.

Miscellaneous testimony

This consists of material from local consumer group newsletters, consumer-authored chapters in books and collections of accounts of ECT. It is not comprehensive as an attempt to extensively cover a literature that is very locally distributed would be impossible. However, it does distil the main points made in a much wider network of accounts according to both the Reference Group and the experience of the authors.

CONCLUSION

In the case of research studies, the aim has been to identify all clinical 'attitude' studies published in the English language and all consumer-led research based in the UK. For testimony, the aim has been to be representative not only of different opinions but of the different formats in which such first-hand accounts are now available. The representation of formats is not the same as the representation of experiences. However, we felt that using the internet might cast light on the forms and content of experience that is being increasingly represented on the world wide web.

CHAPTER 3

METHODOLOGICAL EVALUATION

RESEARCH STUDIES

Appendix 1 lists the 35 research studies referred to in chapter 2 in date order and according to basic aspects of their methodologies. From here on studies will largely be referenced by their number in Appendix 1. A simple inspection of the table makes clear the methodological diversity entailed. The social science literature (Kidder and Judd, 1986) demonstrates that the conditions under which attitudes or beliefs are elicited can influence the resulting data. This chapter will consider methodological issues that may have a bearing on the substantive analyses.

Basic assumptions

There are several studies (eg. 6, 12, 15, 18) where 'correct knowledge' is explicitly defined with reference to a psychiatric consensus or to standard psychiatric textbooks. This is then set against the 'myths' about the treatment that are said to be widespread in society. The commonest hypothesis is that actual experience of the treatment changes attitudes in favour of ECT. This starting point of many studies has an impact on the design and analysis of the research.

The consumer surveys also make assumptions about the 'real' experience of consumers, claiming that this is often at variance with how it is perceived by many clinicians. Consumer surveys are frequently criticised because they are based on anecdote or 'subjective' accounts. However, the scientific work has its own built-in assumptions and values.

Consumer and clinical research

Two comparative studies have examined the responses of interviewees according to the status of the researcher (Clark et al, 1999; Polowycz et al, 1993). Using randomised designs, both found that users were more critical about mental health services when the interviewer was a fellow consumer rather than a professional. Column 7 in Appendix 1 describes the setting where attitudes about ECT were elicited and who, if anybody, conducted the interview. According to the above studies, if satisfaction interviews are conducted by a doctor at the treating hospital, it is more likely that some will be reluctant to express negative views held about their experience.

Sampling and selection bias

Consumer surveys are often criticised on grounds of selection bias. This charge is made as there is no way of knowing who responded to the surveys. It has been suggested (28) that only those with negative experiences will be motivated to complete them as they come from organisations with known doubts or outright hostility towards the treatment. It is impossible to calculate response rates to these surveys as they are typically distributed through membership networks (23, 33, 37) and are only relevant to those with experience of the treatment.

However, clinical research studies may not entirely avoid the problem of selection bias. Some patients have ECT as a relatively routine treatment and maintenance ECT is frequent in some countries. People who have ECT routinely at the same hospital *because they feel it helps*, are more likely to appear in cross-sectional designs which thereby over-select satisfied customers.

Sample structure

Appendix 3 details the demographic characteristics of the study samples as well as UK norms for the use of ECT in the first three months of 1999 (DoH, 1999). There is wide demographic variation in the groups and no sample that mirrors UK norms. This may affect some results and will be referred to where appropriate.

Interval since ECT

In the clinical research, some users are interviewed about their satisfaction with ECT within days of treatment. In other cases, the interval between treatment and interview is several years. This may have consequences for consumers' views about ECT. If treatment is immediately helpful, users are likely to be positive. If the interval between treatment and satisfaction interview is long, return of depression or time to reflect on the experience may diminish favourable attitudes. For the consumer surveys, it is clear (23, 24, 33) that the interval is typically a number of years.

Construction of Questions

There is little attempt to replicate the questions asked of consumers across studies. Since questions vary widely, there is a dilemma about what counts as the 'same question'. Question design is a possible source of bias in studies of any type, but the consumer questions to which we have access (23, 33, 34) are relatively simple and this is true also of some questions reproduced in clinical papers (26, 30). It is in the quasi-experimental designs that the statements put to users are more clearly value-laden (6, 12). For example, Kerr et al (1983)

included the questions: ECT turns patients into zombies. They do not discuss the fact that 19% of their ECT recipients agreed with this statement.

How are attitudes about ECT distributed?

This simple question cannot be answered as the types of quantitative scale vary widely between studies. There are dichotomous scales (6, 26), likert scales (16, 18), semantic differentials (2) and complex interviews (3, 14). The nature of the scale may pre-determine the distribution of responses: dichotomous scales force polarisation. This is significant as it is often suggested that attitudes are polarised or that those with negative experiences are a small minority. Again, attitudes to ECT may not be organised on simple dimensions at all. But may be fluid and contradictory. Research that takes a more qualitative approach (17, 19, 29) suggests that users often qualify or even contradict statements made at one point in time with later ones and that consumer beliefs about treatments are complex.

Analysing data

There is a clear difference between most consumer research and that done by clinicians in the way information is analysed and summarised. Most consumer work adopts the strategy of reporting descriptive statistics on each question and then giving examples of quotations that show what these statistics 'mean' in terms of experiences with ECT. In contrast, clinical research often undertakes statistical analysis of its data. Commonly, the quasi-experimental designs report group differences but not group frequencies of response (12, 15). Although not unusual in scientific writing, the lack of raw data makes it difficult to know how many people depart from the main conclusion drawn. This main conclusion is usually that direct experience of ECT changes attitudes in a positive direction. When some raw data are reported (6, 12, 18), it is clear that a proportion of consumers continue to endorse 'myths' but this is never discussed.

Research studies – summary

Most of the work reviewed here is methodologically flawed. Rather than building on the quite thorough and descriptive early work of Freeman and Kendell, later studies contain a range of methodological pitfalls. Secondly, the different studies cannot be grouped as 'scientific and value-neutral' on the one hand and 'biased and self-selecting' on the other. All start with certain assumptions and values concerning ECT although, again, the degree to which this is done varies. Finally, despite the different assumptions on which clinical and consumer-led research are based, the methodological diversity of the work extends beyond a neat clinical / consumer dichotomy.

TESTIMONY

The testimony material is clearly not filtered through the kind of research protocols that were documented above. However, this does not mean that it is entirely unstructured. The contexts in which the testimonies are elicited do constrain what can be expressed and how it is expressed. It is also apparent that some e-mailers flood the internet with their posts and so the material from the internet cannot be called 'representative'. We have dealt with the existence of repeated e-mailers by counting their contribution once only but this does not resolve the problem of people who read posts but do not contribute.

Email Forums

E-mail testimony is distinguished from research data by its immediacy and interactive nature. It can violate the rules of grammar and narrative by using a short-hand that increases its 'group' quality. The forums use the term 'threads' to refer to series of messages that are like dialogues. The forums are sometimes less spontaneous than 'live' discussions as they are 'moderated' according to the list's ground rules. The moderator may stop very heated exchanges if they contain personal attacks. However, e-mailers are clearly able to criticise others' views due to the anonymity of the format.

The Testimony Archive

Lengthy conversations from the Testimony Archive will clearly result in a different form of information to an e-mail message. Firstly, the Testimony interviews were posed very generally and the interviewer only pursued the topic of ECT if the interviewee raised it. Secondly, although the distinction between 'quantitative' and 'qualitative' techniques of eliciting experience is often over-stated, the format of such a long interview provides scope for reflection on past experience. This 'biographical' approach is used increasingly in the social sciences (Smith, 1994).

Interval since ECT

The testimony data presents the same problem as the research studies. Some individuals in the e-mail forums have only just finished their course of ECT or are even still undergoing treatment. Consumers in the Testimony Archive will usually have experienced ECT years or even decades before. Although their treatment might be deemed out-dated (30), the interview format does not 'freeze' experience in the way other formats do. It also allows scope to reflect on the experience.

Legal depositions

Legal depositions have a legal purpose and in the case of those collected here it is to prevent compulsory ECT or to review its use. They are clearly heavily filtered through the prism of their legal purpose and this will be discussed where relevant

Testimony – summary

The variety of contexts in which testimony data is now available is a methodological strength. The different sources each provide checks on the perspectives expressed in the other formats. E-mail forums particularly introduce the whole arena of group discussion or beliefs into the data. This is currently central in social psychology, the discipline on which the attitude research rests.

CHAPTER 4

TEMPLATE FOR THE ANALYSIS OF CONSUMER PERSPECTIVES ON ECT

The aim of this chapter is to produce a template for analysing the substantive themes outlined in the Introduction. These are: memory loss, information and consent, perceived benefit of the treatment and emotional reactions to ECT. Initial themes were first identified by the Review team and then refined by both the Reference Group and the Advisory Group.

The themes represent issues or 'outcomes' that are priorities for consumers and indicate the different value structures of consumers and clinicians in relation to ECT. The paramount question for clinicians is the effectiveness of the treatment and this clearly matters to users as well. However, the other issues identified may assume equal importance from the perspectives of consumers. Emotional reactions to ECT will also be explored for each of the three themes. In clinical research, this is usually only examined in terms of anxiety about treatment itself. However, in consumer research and testimonies it is clear that other emotions may also be involved, at the time of treatment or subsequently.

Emotional reactions are accessed in qualitative research through analysis of the language in which they are expressed. Free comments, such as testimonies, may simply describe an experience – e.g. persistent memory loss – but may also elaborate that experience in a language that reveals how the person has responded to it emotionally. The analysis of emotional language has been given equal weight to other forms of information in this Review because qualitative research routinely conducts such an examination of its data.

Research evidence

35 studies have been assembled in total but a much smaller number will give basic numerical results on each theme. For the first two themes, the analyses will usually describe the proportion of people whose experience was *negative* or of concern to them. For the theme of perceived benefit the proportion of people who had *positive* experiences will be the focus. Because it is known that the studies vary methodologically, it is likely that they will vary likewise even in very basic findings. So, it will not be possible to 'measure' the extent of negative or positive experiences in any precise fashion. It would not be justified to take an average or mean of all relevant data. However, this is not a counsel of despair – that nothing can be said. Where research studies using a range of methodologies produce similar results, it is reasonable to make a statement of the form 'at least, X% of consumers experience Y'.

Even if descriptive statistics show some consistency across studies, the conclusions drawn about users' experiences of ECT may still differ because of further analysis performed on the data. The different strategies used by clinical and consumer research to summarise data can then be examined to see how diverse conclusions are reached.

Analysing testimony

The quantitative analyses will be combined with the testimony data, using a form of qualitative analysis that occupies a middle ground between formal content analysis and discourse analysis (Bauer and Gaskell, 2000). It takes from content analysis the existence of a set of themes decided in advance of examination of the data. At the same time, discourse analytic methods allow the content and detail of themes to emerge from the data and attend to the interactive nature of e-mail forums and interviews.

The set of key themes are organised as a grid. The themes form the horizontal axis of the grid and the individuals whose experience is represented form the vertical axis. As well as the testimony itself, additional information is noted where this is available. This information includes gender and age as well as the length of time since completion of treatment. Each source of testimony has its own grid so that features of the participants, the internal patterns in the text and the balance of opinion can be examined. This will also enable a comparison, for example, between different e-mail forums (cf. Appendix 4).

The extent to which each theme appears in each grid will be ascertained. This will be done quantitatively in terms both of the proportion of individuals who mention the theme and the balance of negative to positive experiences. For the e-mail forums, if one individual is sending e-mails repeatedly their contributions will be counted only once.

The inter-rater reliability of allocating testimonies to categories in the grids was assessed with a sub-set of 25 testimonies coded independently by two raters. Agreement was high at 83%.

Illustrative quotations from these analyses will be presented for each theme and any sub-themes. These will be chosen to represent views that appear often but not because they are particularly articulate expressions of an experience. Because of the grammatical and narrative structures peculiar to e-mail and also spoken testimony, these quotations will be reproduced exactly as printed on the internet (including transcripts from the Testimony archive). To preserve the flavour of these testimonies, the orthodox 'sic' will not be used.

The dominant theme

Preliminary analysis showed that persistent memory loss was the most frequently mentioned theme in the testimonies and so this comprises the first substantive chapter. The analysis of combinations or concordances of themes will take persistent memory loss as the main topic with which other themes may be combined. Patterns of concordance will be examined for each individual and each grid and summaries presented. Additional qualitative analysis will indicate the emotional tone of reported experience in terms of the content and strength of the language used, as discussed above.

CONCLUSION

This template for analysing the substantive themes does not give an advantage to any one approach to ascertaining consumers' views on ECT. The attempt is to rest with the aims of *describing* the views of consumers and understanding controversy. The extent of methodological diversity uncovered means that it is reasonable to be modest, to ask some basic questions about the 'consumer's perspective on ECT' and supply some provisional answers.

The diversity of conditions under which attitudes towards and experience of ECT have been elicited and expressed will be an occasion for caution but may also sometimes be a strength. If a range of different research designs and formats for testimony reveal consistency of attitudes and values about ECT, then it may be concluded that these are a significant perspective. But if it appears that the findings are contingent on protocols and formats, the conclusion that any one of these findings is significant must be treated with caution.

CHAPTER 5

PERSISTENT MEMORY LOSS

RESEARCH STUDIES

Long-term memory loss was defined as subjective amnesia or gaps in memory still present at least six months after the course of treatment. 20 papers or reports made reference to long-term memory loss following ECT in their abstracts or had a section devoted to this topic. All papers suggested some memory loss associated with ECT but:

- 6 did not meet the six month assessment criterion.
- 4 stated that memory loss was the 'most frequent' side-effect reported but did not give the actual numbers involved.
- 3 reported statistical associations or differences but not the raw data on which these were based.
- Only 7 of the original 20 papers identified included basic numerical information.

These final 7 papers were split into two groups according to the strictness of their definition of memory loss. A strict definition means the words 'permanent' or 'persistent' are included in the question consumers were asked about their memory loss (shown in Figure 5.1). A loose definition includes *any* memory problem reported after 6 months (shown in Figure 5.2). These data show that findings on memory loss are not polarised between clinical research and work carried out in collaboration with consumers. The results for MIND (33) are inside the range of the clinical studies for the strict definition and although in the less strict definition UKAN (23) reports the highest proportion of consumers with memory loss, their figure is less than 10% higher than that of Freeman and Kendell (3), and the Communicate (34) study falls in the mid-range.

The relative consistency of findings means that it is possible to make statements of the form 'at least X% experienced persistent memory loss'. The lowest figure reported is 28.1% (3) and as this is a study that is conducted in a hospital setting by a doctor but where the questions seem to have been straightforward, this may be taken as a lower limit (see Chapter 7 for a discussion of setting effects on perceived benefit).

Figure 5.1: Persistent memory loss (strict definition)

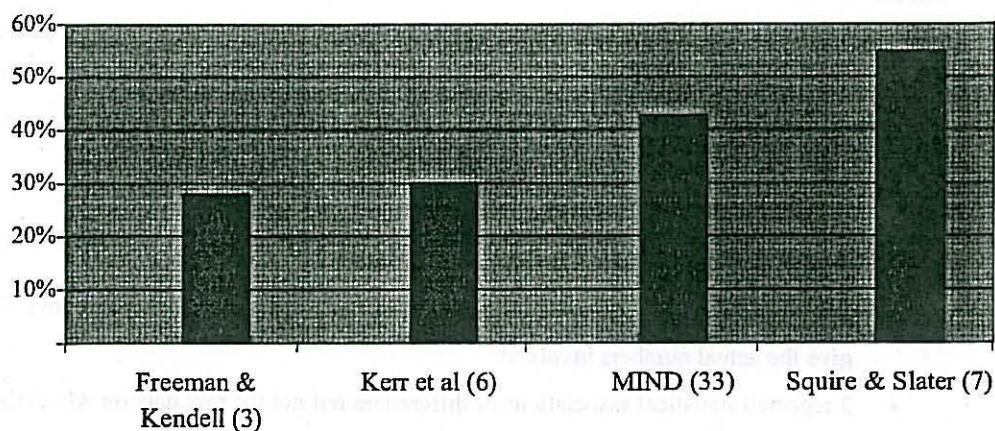
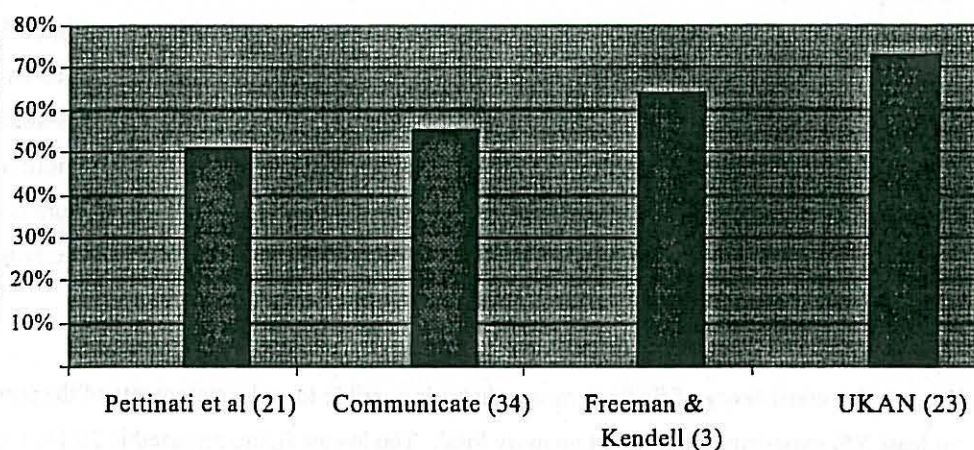


Figure 5.2: Persistent memory loss (loose definition)



Differences of interpretation

Despite the considerable overlap in basic data, clinical and consumer studies tend to be polarised in their conclusions. Clinical research typically refers to a 'minority' (3) or judges the problem to be insignificant (6) or limited (7). Consumer research concludes that persistent memory loss is a significant problem for recipients of ECT.

Consumer research adopts the strategy of illustrating descriptive statistics with direct quotations. These are two examples from the MIND survey:

I can't remember hardly anything about my past life, only very little bits. As for bringing up my three daughters I can't remember a thing. (Woman – Yorkshire)

Several times people have greeted me in social situations as if they were long lost friends and I haven't a clue who they are! My son talks about times I spent with him before I had ECT and I have no recollection of them. (Man – South West)

The different interpretations of clinical researchers might be explained by a focus on group differences rather than absolute values. For example, Kerr et al (6) report that 30% of people with direct experience of ECT endorsed the statement: ECT permanently wipes out large parts of memory. However, this finding is never discussed because the authors focus on the significantly greater number of people with no experience of ECT endorsing this statement.

Some papers explore their data further for the association between memory loss and ECT. In their first study, Freeman and Kendell (3) state that a 'significant minority' of consumers report long-term memory loss but suggest this may be due as much to depression as to the ECT. In a second paper (4), the authors recruited subjects who specifically had complaints about the treatment and found the most common complaint to be persistent memory impairment. The authors are careful to say that none of their subjects held extreme views about ECT or psychiatry. A battery of cognitive tests showed the group did indeed have impairments compared to a control group and for some these were 'well into the organic range on some measures'(p.22) and remained even after controlling for the effects of age, medication and symptoms.

A significant effect on the evaluation of the association between ECT and memory loss is the difficulty of differentiating between memory loss caused by ECT, memory loss due to the depression that it is treating and the maintenance of depression caused by the memory loss itself. This is a particular difficulty when the evidence is made up of correlations and is without any time dimension about the onset of memory loss and depression. There is much evidence that depression is associated with memory difficulties. These are often difficulties in recalling specific autobiographical experiences by substituting categorical memories or "general memories" (Williams, 1996). However, there is little scientific evidence that traditional effective treatments such as anti-depressant medication or cognitive behaviour therapy are associated with increased levels of memory difficulties, particularly long term

memory difficulties. As most controlled studies (e.g. Lasenby et al, 2000) do indicate both personal and impersonal memory loss associated with ECT we can only conclude that this is the most parsimonious interpretation of the results but it is of course possible that this memory loss increases the likely maintenance of residual depression if the forgotten events are part of the personal makeup and identity of the recipient (cf Chen et al, 1999).

The difficulty with statistical analysis is also evident in the strategy used by Squire and Slater (7) whose work probably represents the most thorough analysis of long-term memory impairment. They conclude that there is a period of 'actual' retrograde amnesia for eight months prior to the treatment and of anterograde amnesia for two months afterwards. They then suggest that the experience of actual memory loss leads some users to be doubtful about *all* their memory capacity. This argument has the merit that it suggests practical help for consumers. The estimates are based on complex statistical procedures but are essentially averages. This must mean that memory loss is more serious for some people as Freeman et al (4) and quotations later in this chapter show. Donahue (2000), a consumer who conducted her own review of the literature after profound memory loss following ECT, even refers to herself as an 'outlier'. There appears to be no assessment in the literature of the number of such 'outliers'.

TESTIMONY

The effect of ECT on memory is the most common theme across all sources of testimony. Memory is mentioned in 99 testimonies out of a total of 139.

The issue is important across all age ranges and there is little variation between people who have recently had ECT and people who had ECT 50 years ago. Even when people feel that ECT is an appropriate treatment for them they are often still very concerned with the issue of memory loss. In 13 testimonies, consumers say that ECT has had little or no effect on their long-term memory but 7 of these are writing immediately following treatment.

Widespread memory loss – the erasing of years of memory

Many people describe how several years of their lives and occasionally as much as 15-20 years have been wiped from their memory. They may describe this whether or not they feel helped by ECT.

I had 20 ECT treatments over a 6 month period with positive results...The painful ongoing side effect of the treatment is this though. I have three children and many of the memories of their childhoods are lost. There is no pattern to the memory loss. I don't even realise that I've

forgotten something until my family starts reminiscing about something I have no memory of...then the loss hits all over again.

Megan 38, 20 ECTs over 6 months in 1992 (Healthy place Depression community)

Some consumers experience no long-term effects on their memory:

I never had any long-term effects. I haven't suffered any sort of permanent memory loss, you could lose memories for the, you know, the few hours before you have the shock treatment, but no, I haven't suffered any long-term damage.

Pauline Stott, age 58, several series of ECT (Testimony archive)

Emotional response to memory loss

People's emotional responses to persistent memory loss are a complex and important part of their experience of ECT. The quotation below illustrates the initial disorientation and fear produced by the onset of memory loss.

I came back from the ECT suite and I thought something awful has happened and I'm not having that again. I couldn't remember anybody on the ward, and even when they told me who they were, I couldn't remember them. I struggled to remember the names of my children it was near Christmas, and I was terrified. I was terrified cause I thought.. you know how am I going to sort everything out, because I can't remember anything.

Mo Hutchinson, age 52, nine ECTs in 93 or 94 (Testimony archive)

At this stage some people completely recover their memories except perhaps for a short period immediately prior to treatment. However in the majority of testimonies in which memory loss is a major theme, consumers then start to discover the extent of the damage:

Unless you have taken notes or put a note in an obvious place about where you put them or have an informant willing to tell you what happened to you before your ECT, you are just like a baby, waiting for the never to be realised promise that your memory will come back after a couple of months.

Chris h, age unknown, number of ECTs unknown (ECT.org/ lets talk)

The effects of damage to the memory are present in almost every aspect of people's lives. A language of frustration and humiliation is used to express how the simple tasks of daily living and social interaction become problematic following ECT:

I do remember after that period, and I don't know how long that lasted, going back to work at the National Hospital and the awful situation of, of going into work and not knowing the names of my colleagues, people who I'd been working with for a year, I didn't know who they were. They were familiar to me but I didn't know what their names were and I had to be retrained to do, to do the job I'd been doing for twelve months and, which was, I found, deeply humiliating, deeply humiliating.

Carole Bessington, age 70, 1st series of ECT 1955 (Testimony Archive)

Permanent loss of memories may also directly affect a consumer's sense of personal identity and this may contribute to the maintenance of depression:

Sometimes one fact or piece of information will sound right to me and for a couple of seconds I can hold onto it and fill it as a long lost memory. It is very reassuring and gives me a good feeling. Unfortunately, it passes all too quickly and becomes a very small part of a jigsaw puzzle floating around in the darkness.... Everybody I come in to contact with knows more about me than I do and this makes me feel inadequate, confused, very frustrated and angry about my loss.

Susan Witte, age unknown, 20 ECTs 1987 (Views from off Center 1991)

Information, the role of others and memory loss

When consumers complain of these difficulties to their doctors, they are frequently told that they must be mistaken:

The docs say there is nothing wrong with me. Well something is wrong when you cannot remember events in your child's life or your own.

Kelly, 20 treatments, 1989 (Noshock email forum)

In some testimonies consumers' frustration and anger is further expressed because there is no support available for people experiencing memory loss:

I'm more depressed for having to live with the memory loss and other problems, which began at the time of treatments. (or die with it) IS THERE ANY HELP FOR RECOVERING FROM ECTS??? If so I can't find the link....still need help.

Michel, age unknown, number of ECTs unknown (going mental noshock website)

Effect of ECT on specific skills and abilities

Memory loss and other cognitive impairments may affect some people's ability to return to their former profession or to continue their education.

'Well what is the sense of ruining my head and erasing my memory, which is my capital, and putting me out of business? It was a brilliant cure; but we lost the patient. It's a bum turn. Hotch, terrible

Ernest Hemmingway, two series of ECT in 1962 (Asylum 1995)

I've got 13 GCEs, top grade, but no professional qualifications since ECT I've sat Only one exam, and despite it being 70% project work and continual assessment, I struggled to just pass, well bottom – my memory and impaired concentration can't cope.

Oliver, age 29, number of ECT unknown (going mental no shock web site)

However there are a few consumers who testify that ECT helped them to recover their skills and get back to work:

I have problems remembering things. But these effects are short-lived – I am fine within a fortnight. Since having ECT I have been promoted and continued my writing; I don't feel I have suffered any cognitive impairment.

Rachel Perkins, age 45, three courses of ECT (Open MIND 1997)

THE MEASUREMENT OF MEMORY LOSS

Besides research that directly asks recipients of ECT about their experience and the testimonies assembled here, there is another body of work that assesses memory impairment through formal neuro-psychological testing. This body of work provides the evidence-base for statements on the Royal College of Psychiatrists' leaflet for patients about to undergo ECT that there is no known risk of permanent memory damage (cf. Appendix 6).

When researchers ask patients directly about their memory loss following ECT, they find significant numbers of consumers who report persistent memory impairment. This is so regardless of whether researchers are clinicians or consumers and is confirmed by testimonials. Formal memory researchers, on the other hand, consistently report few memory difficulties following ECT.

This discrepancy is probably largely due to the use of formal measurements of memory being inappropriate to the phenomena that consumers report. Neuropsychological measures of

memory loss tend to concentrate on short-term list learning. These formal measures are also assessments of memory loss called anterograde memory loss i.e. memory difficulties for information which follows the experience of ECT. What most consumers report is memory loss over long periods of time and which is retrograde, i.e. memories prior to the ECT treatment. Forgetting some novel words in a list is clearly totally different to the memory loss that the consumers report. When more appropriate tests are used autobiographical memory is noted to change compared to controls receiving different treatments e.g. Lisanby et al (2000). This is one of the few papers to have similar results to that of consumer testimony data.

CONCLUSION

In all types of information gathered for the Review it is evident that memory loss is a persistent side-effect for at least one third of recipients of ECT. For some, this memory loss profoundly affects their lives and sense of self.

CHAPTER 6

INFORMATION AND CONSENT

INTRODUCTION

The topics of information and consent are treated together because they are routinely linked in the issue of 'informed consent'. However, consent in particular can be over-ridden in psychiatry by legal compulsion and this then becomes a separate issue. This chapter will therefore first deal with the question of information, then that of consent and finally link the two sets of analyses in order to discuss what 'informed consent' means to consumers.

In terms of legal compulsion, this chapter considers only consumers' experiences in the UK. The recipients in the North American research papers were all voluntary patients and those from other UK countries are not reported as to legal status. Some of the UK papers do include consumers who were treated under legal compulsion. Further, legislation to compel patients to receive ECT in countries such as the USA and Australia are state or territory specific and not federal. It is quite beyond the scope of this review to include such variation.

INFORMATION

RESEARCH

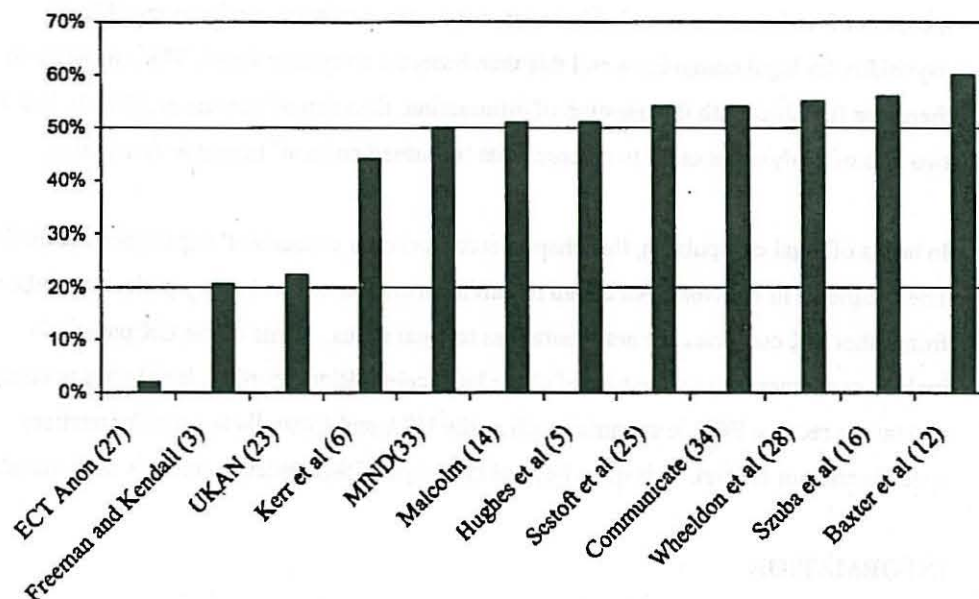
16 papers and reports were identified that included questions on information and/or consent. In 4 cases the data in these papers could not be used either because responses to the questions were not reported (30) or the papers focused on group differences (12, 15, 18).

12 studies asked whether the user felt that they had been given sufficient information about ECT prior to treatment but, as anticipated in chapter 3, it is often difficult to know exactly how the question was phrased. Seven clinical studies asked in a post-treatment interview whether information was adequate and all but one appear to have used terms such as 'adequate information' or 'sufficient information'. The statement reproduced in Kerr et al (6) is: patients are never told what is going on. This is a stronger statement than seems to have been used in the other clinical studies.

Four consumer-led or collaborative surveys ask questions about information. The UKAN (23) and Communicate (34) questions mirror those in the clinical research. MIND's (33) questions are very detailed and the one used here is whether respondents were told why they were being given the treatment. The ECT Anonymous question specifically mentioned full

explanation of the *risks* of ECT. With these differences in mind, figure 6.1 shows what proportion of respondents said they were given sufficient information in each study.

Figure 6.1: Proportion of consumers with adequate information



Although the questions asked were not always directly comparable, 9 of the 12 studies depicted give a consistent picture of 45-55% of consumers reporting that they were not given an adequate explanation prior to treatment. Of these nine studies two involved collaboration with consumers (33,34) and they fall between clinical reports. There is no absolute polarisation between clinical and consumer research on the question of information.

It can be argued that some patients do not want information. The question arises too in physical medicine where patients are argued not to wish to know a serious diagnosis. However, in mental health the issue is bound up with informed consent and against the background of possible legal compulsion.

It might also be argued that due to memory loss for the time around ECT, consumers forget information they have been given. Nonetheless, even the study by Baxter et al (12), which claimed to make very thorough efforts to inform consumers, found that 40% felt ill-informed.

These issues run as differences through the clinical and consumer research. Whilst some clinical attitude studies argue that consumers forget information, other claim that consumers do not want involvement and prefer instead to put their trust in their doctors. MIND on the other hand concludes, on the basis of very similar findings, that consumers are denied even basic information about ECT and that this is unacceptable. We return to this issue in respect of consent below.

Two studies report only one fifth of users saying that they were given sufficient information. One is a clinical report (3) and the other is by UKAN (23). The Freeman and Kendall (3) finding raises interesting questions. Since this paper is scrupulous in reporting its results, it is clear that there were six possible responses to the question about information. 20.6% of the users said they *did have* an adequate explanation about ECT. However, only 49% said they had an *inadequate* one. Nearly all other studies report results in 'yes/no/don't know' form and some only the proportion endorsing a statement (6, 12).

The clinical researchers tend to conclude that patients trust their psychiatrists. An explanation of the UKAN finding, and even more so that from ECT Anonymous, is that members of those organisations do not share this trust. It is also possible that these users have more knowledge about ECT than other groups and therefore have a different standard against which to assess information given by doctors.

OBJECTIVE KNOWLEDGE

Four studies assessed consumers' 'objective knowledge' concerning ECT. There are, of course, problems in defining objective knowledge and also the extent to people wish to know. However, four UK studies did assess 'objective knowledge' about ECT (5, 13, 14 and 20). They assessed how far people knew three things: that anaesthetic is used, an electric current passed through the head and a convulsion or 'fit' is achieved. Table 6.1 shows the findings of these studies.

Study	Percent with full knowledge of ECT
(5) Hughes et al (1981)	7%
(13) Benbow (1988)	12%
(20) Riordan et al (1993)	12%
(14) Malcolm (1989)	16%

Table 6.1: Objective knowledge of ECT amongst consumers in four UK studies

The authors of these studies quote patients making remarks such as 'I should think not!' when asked if they knew a convulsion was involved. In light of Table 6.1, what looks like an absurd figure from ECT Anonymous regarding information becomes more comprehensible. If the vast majority of patients, at least in the UK, do not know what the treatment involves then either it was never explained to them, they forgot what was told them or have even denied the explanation. Consumers who know exactly what the treatment involves are not typical and may have gained this knowledge through membership of groups such as UKAN or ECT Anonymous. If the explanation they were given at the time of treatment was a typical one, most in retrospect will regard it as inadequate.

INFORMATION - CONSUMER-LED RESEARCH AND TESTIMONY

As indicated above, the MIND study asked very detailed questions about information as well as consent. The following quotation is typical.

I felt under a lot of pressure from the staff to go ahead with ECT. I personally don't remember receiving information about how it would work, side effects etc. (Woman - London, ECT in last 6 months)

In the testimony data, there are also examples of complaints about insufficient information that begin to hint at a relationship between lack of information and a feeling of powerlessness.

No one told me what the side effects could be. No one even explained to me what would happen...I didn't even know what the letters ECT stood for. I didn't know and it wasn't explained to me that I would have electrodes attached to my head and that they would put an electric current through my brain.

(Pat Butterfield, age unknown, 12 ECT in 1990 (BBC online 2000))

The quotations from the MIND report were in response to specific questions about adequacy of information. The testimony quotes show that when consumers write or speak more spontaneously about their experiences that this is still a concern. Appendix 4b shows that over 50% of more spontaneous comments about inadequate information were specifically linked to the issue of long-term memory loss. MIND found that consumers were less satisfied with information about side-effects than about the procedure itself.

CONSENT

Legal compulsion

In the first 3 months of 1999 in the UK, 25% of those who received ECT were detained under section. 19% received the treatment under a specific section of the MHA or as an emergency. Consumers may be given compulsory treatment because they are too unwell to sign a consent form or because they simply refuse the treatment. As Appendix 3 shows, no research group has the characteristics of the UK norms although MIND and UKAN approximate them in terms of users on section.

As MIND points out, the difficulty is that an estimate of detention is not an estimate of compulsory ECT in a legal sense. This is because detained patients may consent to ECT and in the three months assessed by the DH in 1999, 29% did so. It is therefore not possible to estimate numerically what percentage of respondents to these consumer studies received ECT under legal compulsion, although a good proportion must have done.

FELT COMPULSION

It is widely reported in the clinical as well as the consumer literature that even where patients sign a consent form for ECT, they often feel that they had no choice but to agree. Malcolm (14) writes: '(many) commented that it was futile to refuse as they would end up getting treatment anyway' (p.163). Johnstone (29) reports that a majority of her respondents either felt so desperate that they would try anything or felt unable to disagree with their psychiatrist.

To estimate what proportion of voluntary consumers felt they had no choice but to have ECT, we focused on questions about whether the user knew they could refuse or felt pressured to have treatment. The statement that Kerr et al (6) put to consumers was: ECT is given if patients don't behave. UKAN asked their respondents if ECT had ever been used as a threat.

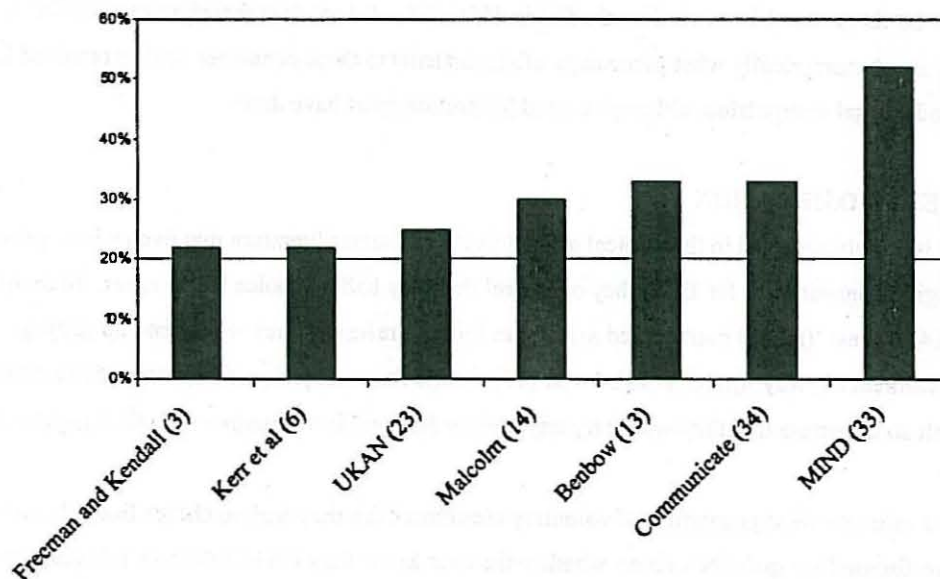
In the case of ECT Anonymous, 87% report that they felt compelled to have the treatment but the report states that this typically included respondents who felt it had been "'oversold' by psychiatrists as 'quick, safe and effective'" (p.4). It would appear this question was much broader than those in the other surveys and for this reason it has not been included in figure 3.

Seven studies asked questions about 'felt compulsion'. In all cases except Freeman and Kendall (3) and Riordan et al (20), the denominator is *consenting* patients. The proportion is of those who were not legally compelled but nevertheless felt they had no choice.

Once again, the methodological diversity of the studies is relevant here, particularly in terms of the different questions asked. Although the figures in 6.1 should be treated with caution in this instance, the consistency of results would seem to make the methodological diversity a strength. It is unusual to get such consistent results from methodologically diverse sources.

Once again, the results do not polarise according to a clinical / consumer division as UKAN is in the mid-range and the Communicate results are identical with those of Benbow. The MIND results are significantly higher but their questionnaire was very detailed on this issue.

Figure 6.2: Proportion of consenting patients who felt they had no choice



It is reasonable to conclude that between one quarter and one third of patients who sign a form consenting to ECT do so under pressure or in the belief that they cannot refuse. According to the UKAN data and that from Kerr et al (6), this pressure can take the form of threat or punishment. These are two statements made by respondents to MIND's survey:

If I had known I had the right to refuse I would have done so. My understanding was that I had no choice in the matter and that they could do it by force. (Woman - Wales, ECT in last year)

I was given no information and had to sign for it after all my medication at night so I was very drugged when I signed the form for my consent. (Woman - Yorkshire, ECT 3-5 years ago)

One respondent to the UKAN questionnaire wrote:

I was told my baby daughter would be put into foster care if I didn't have ECT (even though my husband could have looked after her).

These are not bland accounts of signing or not signing a form. The language signifies fear, an almost total absence of choice and this is apparent too in the testimonies.

TESTIMONY

Emotional Responses to compulsion and felt compulsion

When the issue of consent is mentioned in testimonies it is usually to explain why a person did not feel that they freely gave informed consent. It has already been shown that consumers may believe that they were not given a truthful explanation of the treatment procedure or of long-term side-effects. In the UK particularly, users speak of the threat of compulsion.

...I was asked to give consent for ECT and I refused. I believe it is our human right to do this. A series of persuasions started in a doctor's room. When this failed they asked my daughter saying that if I didn't agree they would transfer me to a hospital a good few miles away. My daughter said she wouldn't be able to visit. What a case for disempowerment. I refused again, and they left a letter on my locker saying I had been sectioned under the mental health act. No explanation was given...

Jean Taylor age 71, 19 ECT in 1989 and 1993 (Mental Health Nursing 1996)

Frequently, consumers feel they did not freely consent as they were so distressed and disorientated at the time that they would have done anything to relieve this. The first respondent below believes the doctor was right to have taken the decision for her:

There was pressure but it was right (Communicate respondent)

I just went along with it but I don't think it was... I mean I would have gone with anything at that stage quite honestly, if they'd ask to do brain surgery or a lobotomy I would have gone with it.

(Premila Trevioli, age 51, two series of ECT 1974 ;Testimony archive)

INFORMED CONSENT

As already discussed, some clinical researchers (13) conclude from their findings that consumers do not want too much information and are happy to let the doctor decide what is best for them. Other authors (3, 14, 20) are unsettled by the degree to which patients will do whatever doctors say, at least according to their own findings.

This trust in doctors was not shared by many of those who responded to the consumer-led surveys. The testimony data also revealed feelings of distrust and betrayal. There are two kinds of data that may further illuminate this question. The first is the qualitative material collected by Johnstone (29) and the second is demographic data that may show differences in satisfaction with consent procedures.

Johnstone specifically recruited participants who felt they had been damaged by ECT and in that sense her study is comparable to Freeman et al (4). Of the 20 consumers she interviewed, 14 said they had signed the consent form. When probed on this, some said they were so desperate they would try anything. However, many also expressed a sense of total powerlessness when faced by a medical professional so confident in their proposed treatment. One woman said:

I was a very compliant young woman, I was frightened of everybody and that was part of the problem.....I wouldn't have known how to object, it wasn't on the horizon. You didn't disagree with doctors, you did what they said (p.74).

It is clearly different to put one's faith in a doctor from a positive sense of trust than to do so because it is not felt possible to disagree with a powerful professional.

DEMOGRAPHIC ISSUES

Age

Malcolm's (1989) sample was almost evenly split between those under and over 65 years of age. Whilst 71% of the younger patients were aware they could refuse ECT, only 27% of those over 65 knew this. Malcolm concludes that the older patients "seemed to think this was a satisfactory state of affairs. Elderly patients appeared to have greater faith in the doctor doing what was in their best interest" (p.164).

Gender

Appendix 2 shows that research studies typically have twice as many women as men. However, very few studies analyse the issues of information and consent according to gender.

Malcolm (1989) reports that men tended to be more aware of the possibility of refusing treatment than women and also to have better objective knowledge of the procedure. MIND and UKAN have an over-representation of men compared to national figures (DoH, 1999) and this, as well as their younger samples, may account for some of the differences discussed in this chapter.

Consumer culture and the internet

Hillard and Folger (2) demonstrated that consumers gain a great deal of information about ECT from each other and explained this with reference to the 'ward culture'. The extension of this culture with the advent of the internet is especially significant for this chapter. In the past a worried consumer would need to make a concerted effort to access information about ECT especially if they were interested in material that questioned the official view. Now a short period on the World Wide Web will yield a wealth of information about ECT from an extremely wide range of sources. There are indications from the e-mail forums that the Internet is set to have a dramatic impact on how consumers actually decide to have ECT.

I'm actually supposed to get ECT this morning but after reading the information on this web-site IM NOT GOING!! I'm scared to death!! I really didn't think the treatments were actually working for me anyways but after some of the info ive read I now realize im not the only person with post ECT problems

Stacey, age 25, receiving 1st series of ECT, 8 or 9 so far (Noshock e mail forum)

In March of 1997 I was truly suicidal for the first time in 30 years of dealing with recurrent depression. I was admitted and my pdoc also recommend ECT. I requested a Medline search of all the articles from our local medical school library and a friend picked them up. I read every single one and decided to go ahead. I had 9 initial treatments (3 a week for 3 weeks) and 4 or 5 monthly maintenance treatments until I had a bad reaction to the anaesthesia. They helped me greatly and I'm very glad I agreed to them.

Madeline, age unknown 14 ECT 1997 (Healthy Place Depression community)

CONCLUSION

The data presented in this chapter show that across research studies at least 50% of users feel they have been given an inadequate explanation of ECT. There is consistency across clinical and most consumer research studies on this issue and confirmation from the testimony data. Where consumer-led research reports very low numbers of people feeling sufficiently informed about ECT, this has to be set in the context of clinical research which demonstrates

that consumers typically are very misinformed about the procedure and that members of 'anti-ECT organisations' may be better informed than most consumers.

It is estimated that some 35% of consenting patients feel pressurised to have the treatment. Clinical researchers tend to conclude that putting trust in one's doctor is the explanation for this. Consumer-led research and testimonies, on the other hand, often use a quite different language – one of threat and manipulation.

It is entirely possible that some consumers have absolute faith in their doctors and others none at all. Some demographic dimensions have been suggested which may affect how likely consumers are to occupy the poles of these positions and how strongly. These arguments will be developed in the chapter on perceived benefits of ECT where the link between perceived benefit and the questions addressed in this chapter will be considered further.

CHAPTER 7

PERCEIVED BENEFIT OF ECT

This chapter more clearly overlaps with the meta-analysis of effectiveness in the review by Carney et al (2001). In their review, however, effectiveness was measured by a change on a symptom rating scale, which is usually but not always completed by an observer. These sorts of ratings may not always correspond with the subjective relief of distress. For example, Hughes et al (1981) report that the same number of people improved according to a clinical measure and according to subjective reports but that in 20% of cases these were not the same individuals. This discrepancy anticipates the possibility that consumers' perspectives on ECT in terms of its helpfulness or otherwise may not correspond to clinical concepts of outcome.

Research

20 research studies asked about the perceived benefit of ECT. There were two main kinds of question: perceived helpfulness and whether the user would agree to ECT again. Figures 7.1 and 7.2 overleaf show the charts for each type of question.

Three studies are not included in the charts because the nature of the question or the context in which it was asked was different to the other studies. Sestoft et al (25) gave ECT and medication as options to their respondents and 29% said they would choose ECT as a first preference. Pettinati et al (21) report that 98% of their consumers would be willing to have ECT again but the results show that half of these would have ECT only if other treatments had been ineffective. Johnstone (29) reports that 45% of very unhappy consumers gained some initial relief from the treatment but said that it did not last and the costs outweighed the benefits.

The first figure shows a gradually rising curve of the proportion of satisfied customers but unlike other charts in this report, the studies are grouped in terms of clinical and consumer research. The second figure shows a similar pattern but it should be noted that the UKAN question was: would you *request* ECT again (italics ours).

The variability between the studies can be considered in light of the issues raised in chapter 3 and this in turn may illuminate the differences between the clinical and consumer research.

Figure 7.1: Helpfulness of ECT

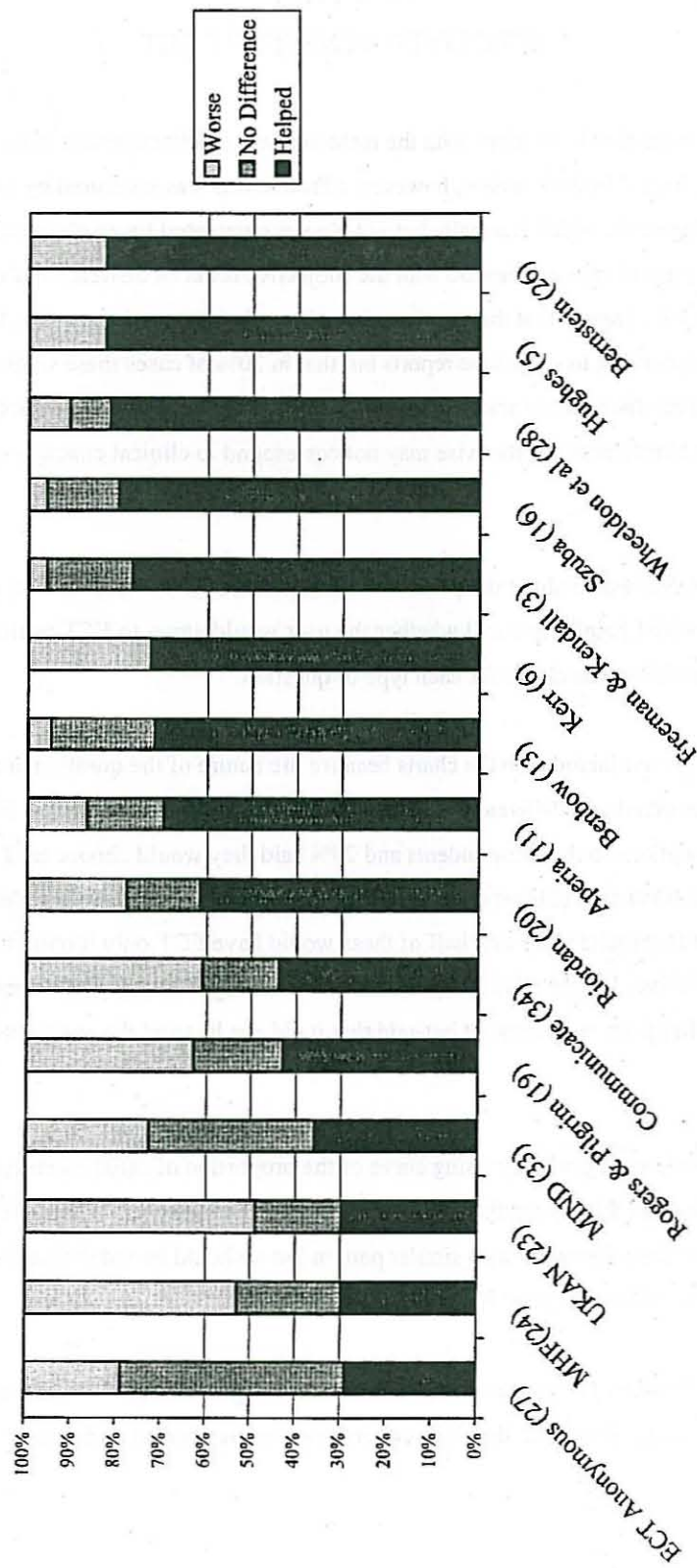
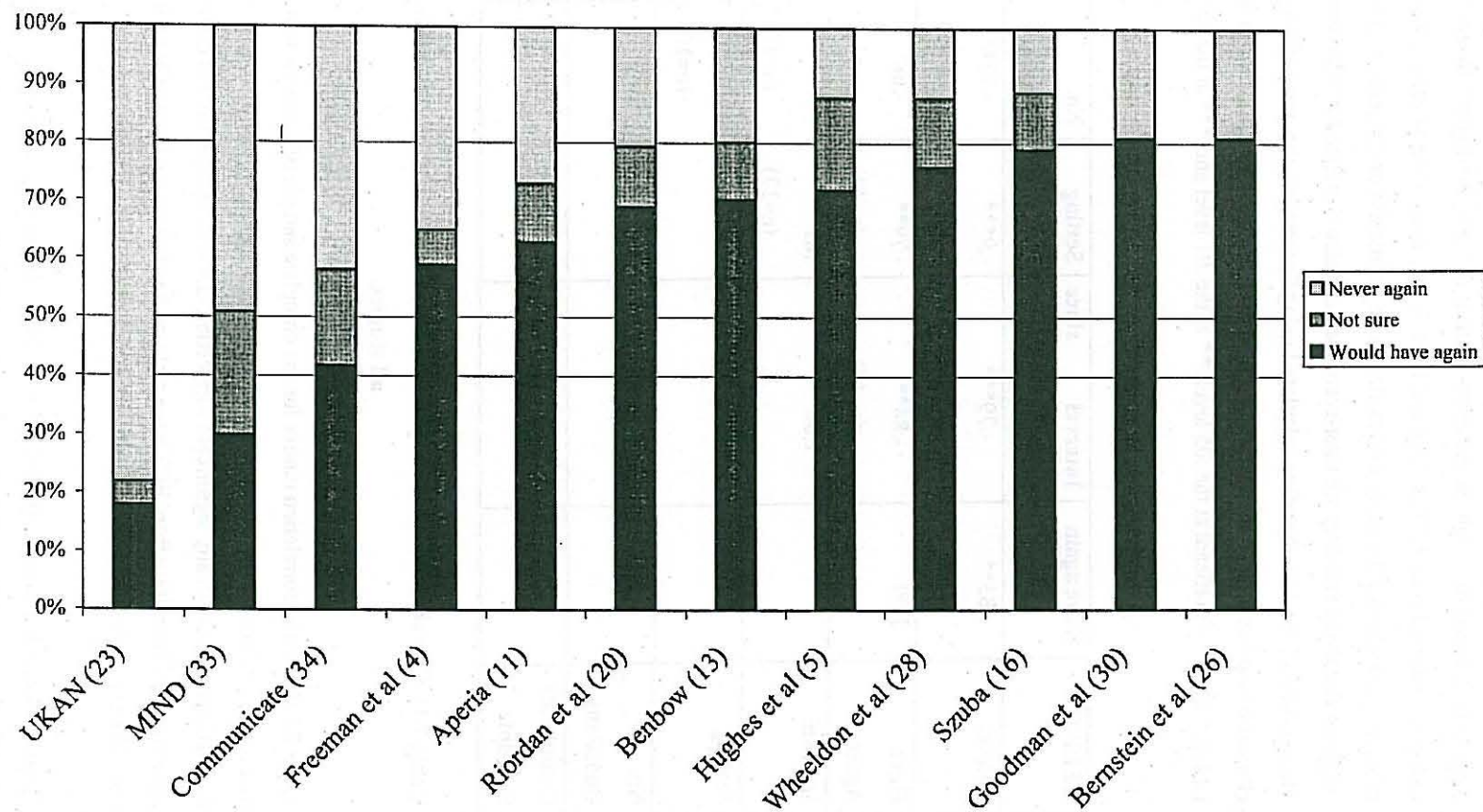


Figure 7.2: Willingness to have ECT again



Methodological correlates of perceived benefit

Appendix 2 shows the results in the charts against five methodological dimensions of the research: interval since ECT, setting and interviewer, number of questions, complexity of schedule and whether the study was carried out by clinicians or consumers. The table suggests that these variables are inter-correlated and also correlated with perceived benefit. These correlations were therefore calculated using Spearman's rho and omitting the clinical/consumer dichotomy as it is already known that this differentiates the results. In the tables, * means significant at the .05 level, ** at the .01 level and *** at the .001 level.

HELPED	Have again	Interval since ECT	Setting	No. Questions	Complexity Schedule
Helped (n=9)	.83**	-.79*** (n=15)	-.9*** (n=16)	-.73** (n=15)	-.4 (n=14)
Have Again	1.00	-.84** (n=10)	-.79** (n=11)	-.68* (n=11)	-.58* (n=11)
Interval		1.00	.81*** (n=17)	.61** (n=16)	.77*** (n=15)
Setting			1.00	.61** (n=16)	.46 (n=16)
No. Questions				1.00	.77*** (n=16)
Complexity Schedule					1.00

Table 7.1: Correlation matrix for perceived benefit and four methodological variables – all studies.

Table 7.1 shows the correlation matrix for the complete sample of research studies, omitting the consumer/clinical dichotomy as this is known to differentiate the studies. Nearly all correlations are statistically significant suggesting that the methods used to elicit users' experiences of benefiting from ECT have a strong bearing on the level of benefit reported and that different studies combine the methodological elements in systematic ways.

Chapter 3 noted that some methodological variables distinguish clinical from consumer research. It is therefore possible that the results in table 7.1 are an artefact of this distinction.

To check for this, the correlations were run again for the clinical research studies only and table 7.2 gives the results.

HELPED	Have again	Interval since ECT	Setting	No. Questions	Complexity Schedule
Helped	.63 (n=7)	-.57 (n=8)	-.48 (n=9)	-.75* (n=9)	-.47 (n=9)
Have Again	1.00	-.84** (n=8)	-.6 (n=9)	-.6 (n=9)	-.95*** (n=9)
Interval		1.00	.57 (n=10)	.46 (n=10)	.73** (n=10)
Setting			1.00	.52 (n=11)	.42 (n=11)
No. Questions				1.00	.5 (n=11)
Complexity Schedule					1.00

Table 7.2: Correlation matrix for perceived benefit and four methodological variables – clinical research studies only.

Although there are fewer significant correlations the pattern of relationships mirrors that in Table 7.1. In the clinical studies, the length of time between treatment and interview and the complexity of the schedule are very highly correlated with each other and with whether the user would agree to have ECT again. If a consumer is interviewed immediately after a course of treatment, on the ward and asked only a few questions then the highest levels of perceived benefit are reported. If interviewed at least six months after treatment with a complex interview, the likelihood of agreeing to ECT again in particular decreases. The complexity of the schedule appears particularly important. A semi-structured interview can be conducted as a conversation and is more likely to elicit thoughtful and complex answers and to engage consumers.

Freeman and Kendell's (3) study illustrates a further effect of interval since treatment on satisfaction. Figure 7.1 gives the answer to a question about how helpful ECT was *at the time*. An additional question concerned the lasting effects of treatment. Only 9% said the effect had

been permanent and 35% that it had lasted a year or more. The transient nature of perceived benefit may affect decisions about the worth of having the treatment again.

The issue of comparing ECT to other treatments is also important and absent in most studies. Even a check-list giving preference options rather than asking about ECT alone can give distinctive results as shown by Sestoft et al (25). Rogers and Pilgrim (19) show that consumers who have received ECT are more likely to regard it as unhelpful than those who are prescribed neuroleptic drugs. Half of Pettinati et al's (21) much-quoted 98% of consumers who would agree to ECT again actually wanted to try other treatments first.

Clinical and consumer research

Only three consumer studies asked about willingness to have ECT again. The highest response was in the Communicate survey and this had the shortest interval between treatment and survey and a simple questionnaire.

Just as there may be a tendency to compliance in hospital settings so critics may be right that consumers are more likely to respond negatively to a questionnaire or interview conducted by other consumers if that is their opinion. However, this is not a matter of 'bias' but a robust research finding as described in chapter 3. Although postal questionnaires are anonymous and so 'impersonal' they do allow the service user to think carefully about how to answer. A consumer could easily mull over a questionnaire from MIND or UKAN before completing it as the time limits for completing the form are usually several weeks. The typical 'please comment' space after each question in these forms also allows the kind of engagement referred to above.

Some clinical schedules are more complex than some consumer ones. However, there is no clinical study in which consumers are interviewed in a place of their choice by a non-medical person. Thus the similar patterns in the two correlation matrices may indicate that the methodological diversity of the complete set of studies is strongly associated with overall differences in the perceived benefit of ECT.

Subject selection

Cross-sectional designs may over-select satisfied customers (see chapter 3). Bernstein et al (1989) report that nearly half their respondents were receiving maintenance ECT and it is difficult to see how users would agree to this unless they found it helpful.

Because this Review had access to the questionnaires from the Communicate study, they were analysed to see whether there was an association between having previous experience of ECT and willingness to have it again. Respondents who said they had had ECT before were more likely to say they would agree to it again (Chi-Square = 4.91; df = 1; $p < 0.05$). Only 4 of the total 17 people who had ECT for the first time said they would definitely have it again, one commenting that she would if she thought it would do any good which it had not this time.

MIND reports that nearly half their respondents had more than one course of ECT. MIND gives no breakdown of the relationship in their sample between having more than one series and satisfaction or having more than one series and compulsion. These would be the important variables to validate the above paragraphs.

Historical and cross-national comparisons

Goodman et al (1999) argue that much of the research reviewed here is out-dated as ECT procedures and patient care have improved in recent years. However, there are no relationships between the date of the clinical research studies and satisfaction rates that cannot be explained either methodologically or demographically. The best data in this respect come from the MIND survey (33) as results are divided according to when the respondents experienced ECT. The evidence is of marginal improvement in the UK at least. However, statistical analyses are not performed so it is unclear whether this is statistically significant.

Consumers in the USA appear to be more satisfied with their treatment than those elsewhere. The exception is the UK study by Wheeldon et al (28). As will be seen, this is not borne out by the internet testimony data. Again the difference may be partly explained in terms of the methodological issues discussed above. There are also demographic differences between the groups studied, including the fact that all US respondents were voluntary patients.

Demographic factors

Appendix 3 shows the demographic features of the samples for each research study considered in this chapter as well as the demographic averages for recipients of ECT in the UK in the first three months of 1999 (DoH, 1999). The figures from each study that approximate those averages are marked in bold in order to show which are the most representative in terms of UK norms. The first point to note is the extreme variability of the samples except in the case of gender. There is a lack of overall conformity to UK norms and no reason to suppose that the populations receiving ECT in other countries are different to the UK.

The UK studies are more likely to include older participants and in that sense be representative. Riordan et al (20) and Malcolm (14) report older consumers to be more likely to trust their doctors and to be satisfied with, or at least resigned to, the treatment. The younger age structure of the consumer-led studies may therefore partially account for lower satisfaction rates in the UK. However, since age is confounded with both occupational status and the country in which the research was conducted, no firm conclusions can be drawn at an international level.

The consumer-led studies which report demographic data (23, 33) have a relative over-representation of men and, since men report lower levels of satisfaction with ECT, this may partly account for lower satisfaction rates in these studies.

Legal and felt compulsion

Wheeldon et al (28) found no difference in satisfaction between patients legally compelled to have ECT and those who consented. However, this is based on 11 formally treated patients, 9 of whom said they found the treatment beneficial. Of all the UK clinical studies that report the legal status of their consumers with respect to ECT, only Malcolm (1989) has a proportion that approximates the national figure. The others are approximately one third of what would be expected given national statistics. Indeed, it is the consumer-led studies (23, 33) and the work of Johnstone (29) that are the more representative in this regard. UKAN reports a negative association between legal compulsion and satisfaction, particularly in the case of men. The MIND report does not state whether there was any relationship between legal compulsion and satisfaction. However, MIND does report that users from black and ethnic minority communities were more likely to be compelled to have ECT and more likely to find the treatment unhelpful, damaging or very damaging.

In terms of felt compulsion, no study analyses its relation with perceived benefit. The Communicate questionnaires were examined and a strict definition was adopted. Feeling compelled to have ECT was defined as saying both that the consumer did not make a fully informed decision and that there was pressure or force to have the treatment. 13 people meet the definition of felt compulsion. Only one of these was treated under a section of the MHA specifically for ECT. Of the 12 remaining, 10 (83%) said they would refuse to have ECT again. This is double the total sample figure of 41%.

TESTIMONY

ECT is a treatment which attracts controversy. The polarisation of opinion about the benefits of ECT expressed in the testimonies reflects this, as does the especially strong language used in many testimonies. People at one end of the continuum of opinion say ECT is a lifesaver and people at the other say ECT is a profoundly damaging treatment. However within the testimonies there is also some graduation of opinion. For example not all those who found ECT unhelpful think it should be banned and many of those who derived benefit accept that others have found it damaging. Out of a total of 139 testimonies, 83 either said they would not want to have ECT again under any circumstances or were very negative about their experience of ECT. 43 people were positive about ECT and/or indicated that they would be prepared to have the treatment again. 14 people said they were unsure whether they would be prepared to have the treatment again or their testimony did not make their view clear.

Different formats of testimony attract contrasting views on ECT. Legally sourced testimony is completely against ECT. However most sources are more equivocal. For example 37% of people contributing to a forum called NOSHOCK said that they would have ECT again. The *Testimony Archive*, which contains accounts by people who may have received less sophisticated forms of ECT in large asylums, has a relatively even spread of responses to ECT: 48% would not have the treatment again, 21% would have it again and 30% are unsure.

ECT Has Benefits

31% of the consumers represented in the testimonies were positive about their experience of ECT.

I know that there is something I can do to stop it [depression] –something that will quickly restore me to my life and self.

Rachel Perkins age 45 3 series of ECT (Open Mind 1999)

We did the ECT and I can truly say that it saved my life. After the first treatment I already felt a difference. I had a total of 6 treatments and I am back to the same person I used to be. I went back to work and I am functioning and performing great. I feel so good and blessed. I feel I owe my life to ECT. It's been 4 months since the treatments and I pray that it doesn't come back. ECT was a miracle for me. It truly saved my life.

Sasha, age 29, 6 ECT (Healthy Place Depression community)

However very forthright and positive testimonies are usually from people who have had ECT very recently or at least within the last six months.

My experience has been great, and I would like to share it in the hope that anyone out there looking for info on this controversial procedure can be that much more informed. Today 2 months after my ECT treatments, which did happen to be BILATERAL, I have no lingering side effects and I feel like the proverbial cloud of depression that has smothered me for at least the last 5 years has been lifted. I am on 'maintenance doses of Effexor and Buspar, and while not 100 % little Miss Mary Sunshine all the time who is? I feel like a normal person.
Layla23 age 21, one series of ECT (ECT.org lets talk)

I can tell you I feel really good and am no longer depressed. That has to say something for this treatment when nothing has ever worked for me before. I do see memory loss as a problem and hope it is a short lived one. I honestly do feel better inside though and I am loving it so far.

Molly age 23, 6 ECT just finished (Health Place Depression community)

Of a total of 139 testimonies, 108 specified a timescale. As Table 7.3 shows, those who have had ECT most recently are more likely to be willing to have it again. This is the same pattern of results as was found in the research studies.

TIMESCALE	Never again	Not sure	Would have again
After 6 months	61	10	17
Within 6 months	4	1	15

(Chi - Square = 12.9 df = 2 p < .01)

Table 7.3: Contingency table of interval since ECT and willingness to have treatment again.

The effect is temporary

No consumer testimony reviewed said that ECT has a long lasting effect and some people refer to the short-lived nature of the benefits:

I finally tried ECT. I was amazed at how good I felt. I had short-term memory loss. I would say that I felt good for about a year. The depression came back and I was back to square one. All I wanted to do was die.

Laura, age unknown, one series ECT (Healthy Place Depression community)

Trade offs

Many people's testimonies describe how they trade off damage to their memory, cognitive ability and personality in order to gain relief from depression:

But I did the ECT treatments and let me tell you; it pulled me out of my suicidal stage. Things began to look brighter in my life. Unfortunately I am now having a relapse. I am starting again my ECT treatments Monday. Yes is it scary, yes it is expensive, yes some people do not benefit, but I have completely benefited by it. The only problem is now is that I need more of it to go on. The only problem that I had during my sessions and even now is the memory loss that I have. I cannot remember things back to 2 years ago.

Shari, age unknown, 15 ECT (going mental no shock web site)

My wife thinks the memory loss is from ECT and doesn't want me ever to have another treatment. My Psychiatrist says it could be from either the ECT or the anxiety of returning to work or a combination of both. I am still an ECT advocate, I don't believe it is a total cure for depression, but perhaps we don't have a total cure for everybody yet. I know from my own experience when you are in so much pain that you want to die in order to stop the pain and a moderately simple procedure is available that can eliminate that pain even if only temporally then I want to use it.

Jim, age unknown, 3 series of ECT (windsofchange website)

It is a common belief expressed in the testimonies that the main benefit of ECT is selective memory loss:

..It just helped me. It helped me get my act together, and get my life together and I forget. Could.. forget the horrible bits, deep down within side of me for a while as well.

Ann Marie Randell age 41, number of ECT unknown (Testimony archive)

Consumer dis-satisfaction is represented as satisfaction

There are indications in several testimonies of how professional and consumer perceptions of the outcome of a course of ECT may seem to be uniform yet differ greatly. It appears that one consequence of the patient/doctor power imbalance might be that some patients are unable to express their true feelings to their psychiatrist:

After every treatment he would say how do you feel and I'd say God I feel awful, just awful. What I finally realised was that as long as I said I felt bad I kept getting them because he thought I needed more treatment. So the next time I said I feel much better now ...He said oh maybe we'd be able to stop these soon. After the next one I said I feel great. And then he stopped them. That was what all the other patients around the hospital taught each other- that if you wanted to stop getting these things, don't say how bad you feel, say how good you feel. Then they will think they're working and leave you alone.

Anita Courtney, age 32, number of ECT unknown (Southern Exposure 1989)

Unsure

9% of people whose testimonies were collected were unsure whether or not they would have ECT again:

Would I undergo ECT again? I have no idea. Where medication does not work, I believe the doctor's judgement that ECT is still the most effective treatment. For people who are sick enough to be considered for ECT- as I was - I believe the benefits justify the potential loss of memory. Losing my memory, my career, my connections to people and places may seem too much to bear, but I see all that as not a huge price to pay for getting better. What I have lost is enormous, but if it is health I have gained, that is obviously far more valuable than what I lost.

Ann Lewis, age 37, 18 ECT, 1999 (Washington Post 2000)

ECT offers no benefits

61% of the consumers whose testimonies were collected said they would not have ECT again. Some people describe their experience of ECT as damaging but not overwhelming so:

I had a series of ECT treatments several years ago and I am still recovering. I would say that the ECT sort of numbed my brain. It's not paralysing to my life because I still work and have a family and my memory is not too bad.

Eric, one series several years ago (noshock e-mail forum).

Others say that they have experienced some benefits but that overall it was a negative experience:

I would never have ECT again, I know it was beneficial in some respects I am no longer depressed; I can have fun now . But I am a totally different person.

Colleen Tate, age 34, Eight ECTs 1991 (Special report 1991)

Some people believe so strongly that ECT damaged them that they are determined never to have it again:

So I..I would never have it again and I...I am quite determined that if I was ever sectioned again, I ...I would never have it again and I..I had somebody, like my solicitor or somebody there, who...who was going to...to make sure that I wasn't given it without my consent... 'cause I just feel that ..it was going to do me damage.

Mo Hutchinson, age 52, nine ECTs in 93 or 94 (Testimony archive)

Within the testimonies some people say that it is ECT that pushed them toward suicide:

It is a barbaric practice. Psychiatrists say it saves lives, but I would say it is more likely to push you towards suicide. It ruined my life and robbed me of my personality and memories.

Beryl Manklow, age 61, 3 or 4 ECT before treatment was terminated by her husband (BBC online 2000)

Several people say that ECT catapulted them into manic or psychotic states, which they had never experienced before.

Two days after the second ECT treatment, I was hospitalised with mania. I was manic for four days: this was my first episode of mania.

Leigh Murry, age unknown, 6 ECT (Healthy place Depression Community)

In 43% of the consumer testimonies reviewed, people describe their experience of ECT in extremely negative terms and said that for them ECT was damaging and traumatic:

I call ECT a rape of the soul. Psychiatrists call it life saving therapy.

Barbara Cody age 59 13 ECT in 1983 (Texas Public Health committee 1995)

ECT equals EVERY CELL TRAUMATISED, I've been tortured in civilised fashion, and all the time have to struggle against the victim mentality its helped produce in me.

Oliver, age 29, one series ECT in 1990s (going mental no shock web site)

Some individuals are so convinced that ECT has damaged them that they are considering taking legal action:

Please help me ..I had ECT 3 years ago and my life has been ruined. What can I do? I want to sue the doctor. Can I ? someone please get back to me.

Annie, age unknown, number of ECT unknown (Healthy place Depression community)

Some people seek validation for their experiences through involvement in campaigns against ECT.

I am finding strength in speaking out against this biased, archaic, barbaric, poorly researched, unscientific and detrimental psychiatric treatment.

Susan A Whal, age unknown, ECT 3 yrs ago (Healthy place Depression community)

CONCLUSION

Despite the evidence that critical consumers are somewhat over-selected by consumer-led research, it has been shown that other methodological variables exert a powerful effect upon consumers' expressed satisfaction with ECT. This conclusion can be drawn with confidence particularly in relation to *when* consumers are asked about or express their view on having the treatment again. In clinical and consumer research studies and in the testimony data, the length of time that has elapsed since treatment is strongly associated with expressed satisfaction. The conclusion is that studies which interview consumers immediately after treatment, especially if they do so in a medical setting using brief interviews, over-estimate to a considerable degree the extent of satisfaction with ECT. Estimates of 80% to 95% of consumers as content with the treatment are, in the judgement of this Review, not valid.

The evidence that legal or felt compulsion affects satisfaction suggests that both do so negatively. There is one clinical study Wheeldon et al (28) that reports that compulsion does not reduce satisfaction rates (which are found to be high) but there is other evidence that compulsion has a negative effect upon consumers' expressed satisfaction with ECT.

The testimony data makes it clear that 'perceived benefit' from ECT may be both discrepant from and much more complex than clinical concepts of symptom improvement. The testimonies covered issues that are never touched upon by clinical research. These include extreme trauma, lying about improvement in order to stop treatment, the desire to take legal action and the need to seek out support and validation from other individuals and organisations. No clinical research paper asks respondents if they became manic or if they felt more suicidal after ECT. Additionally, it is clear that many consumers' views are complex. Individuals will make trade-offs between side-effects and benefit and some may be

so concerned with the manner in which the treatment was given that whether or not it helped them is not the major issue. For these reasons, it is over-simplistic to assume that there is a uni-dimensional 'attitude towards ECT' even on the question of whether or not it is helpful.

ECT is a treatment that provokes strong reactions in those who have experienced it. Despite the variety and complexity of responses, only 9% of those testifying express uncertainty or ambivalence about the treatment. It is common among those convinced of the effectiveness of ECT to characterise consumers who are opposed to it as a small and vocal minority. In the context of the testimony material, people who receive benefit from ECT are in the minority. However, people who clearly feel the treatment works for them do not feel silenced by those who oppose it and a noteworthy feature of the consumer discourse is an awareness of and respect for diversity of opinion.

CONCLUSIONS

1. METHODS

- 1.1. The methodological diversity and variable quality of research studies examining users' 'attitudes' to ECT posed problems in both assembling and drawing conclusions from this material.
- 1.2. The inclusion of the testimony data therefore increased the validity of the conclusions, when it complemented the more robust research evidence.
- 1.3. The strongest methodological correlate of consumer satisfaction with ECT is the interval since treatment. In all data sources, the time elapsed since treatment was negatively associated with satisfaction with ECT.
- 1.4. The testimony data in particular suggests that consumers' beliefs and perspectives on ECT do not take the form of a small set of 'uni-dimensional attitudes'. They should therefore not be measured as if they do take this form.

2. Aim 1 - Systematic Description of Consumers' Views on ECT

- 2.1. There is some evidence of subjective short-term benefit from ECT from all data sources although it is likely that some research studies over-estimate this because of the methods used.
- 2.2. The short-term nature of subjective benefit reduces satisfaction with the treatment in the long-term and the willingness to have it again.
- 2.3. There is conclusive evidence of persistent subjective memory loss for a significant number of consumers.
- 2.4. This memory loss is retrograde and not detected by measures that focus on anterograde amnesia.

- 2.5. All data sources show that approximately one half of consumers feel they were inadequately informed about ECT.
 - 2.6. All data sources show that a significant number of those who sign a consent form do not feel that they freely consented to ECT.
 - 2.7. Memory loss, felt compulsion and inadequate information are not specific to dissatisfied consumers.
 - 2.8. Consumers may respond with feelings of anger about their experience with ECT particularly when this is dismissed or not acknowledged as valid by health care professionals.
3. Aim 2 - Controversy Between Some Consumers and Mainstream Medical Opinion
 - 3.1. Different issues are interpreted differently by consumers and clinicians:
 - 3.1.1 Relief of depression on a symptom rating scale may not correspond with subjective relief.
 - 3.1.2 Persistent memory loss that consumers understand as a side-effect of treatment is, when acknowledged, understood by clinicians as a consequence of other factors including concurrent symptoms, age and alcohol use.
 - 3.1.3 Some research papers conclude that consumers do not wish to be over-burdened with information and wish to leave medical decisions to doctors. What clinicians perceive as trust in the medical profession some consumers experience as powerlessness when confronted with a confident professional.
 - 3.1.4 There are research papers that represent the views of consumers dissatisfied with ECT as insignificant. However, consumer discourse accommodates and allows diversity of opinion.

3.2 The testimony data show that disagreements between consumers and their clinicians about ECT can lead service users to take up a stance against the treatment generally:

3.2.1 Failure to acknowledge the factors under 3.1 on the part of professionals may lead an individual to seek the support of others with similar experiences and of consumer organisations.

3.2.2 In the context of this Review, it is not surprising that organisations should emerge which provide a forum and platform for those consumers who feel that their experience of ECT has been damaging and coercive.

SUGGESTIONS FOR FUTURE RESEARCH

Persistent memory loss

A comprehensive piece of research should be undertaken on persistent memory loss as a consequence of ECT. The main issue arising from the Review has been to differentiate autobiographical memory from anterograde memory, which, while distressing, seems to be less important and more temporary for consumers. Studies need to have long follow-up because losses of memories prior to ECT may only become apparent after a long interval. One approach could be to adopt the lead of the small-scale work of Janis undertaken in 1950 and which has never been repeated. This study adopted a narrative approach to memory, asking individuals to relate their life story in their own terms, highlighting what was important to them and with a limited number of prompts about significant life events. The study would not only collect this observational data on loss of memories but would also ask about subjective distress following the discovery of persistent memory loss. The effects of medication levels and current symptoms in relation to memory loss and levels of subjective distress would also need to be investigated.

Information and consent

A systematic research programme should be undertaken on legislation concerning ECT, the effects of this legislation on consumers' attitudes towards compulsion and on 'felt compulsion' in the absence of legal coercion. This research programme should be cross-national and cross-state and territory for the U.S.A. and Australia. It will need to include non-English speaking countries and will require a team of researchers who are attuned to cultural differences. It should also examine countries, states or territories where there have been major changes to legislation or to central guidance concerning ECT in the last 30 years. Factors which should be investigated include:

- i. The relationship between legal compulsion and beliefs, attitudes and feelings about ECT at a later point in time (at least six months)
- ii. The relationship between felt compulsion and perceived coercion and the general legal context and between felt compulsion and post-treatment attitudes.
- iii. The extent of the belief that patients should have absolute confidence in the medical profession and the relationship of this to the general legal and cultural context.
- iv. For countries or states that have seen major recent changes to legislation or central guidance concerning ECT, the forces that brought this about and the subsequent responses on the part of professionals, consumers and policy-makers.

- v. The relationship between the use of ECT and access to resources, particularly in developing countries but also for poor consumers in developed countries based on insurance systems.
- The difficulty of such a research programme should not be under-estimated. For example, the EPSILON study (British Journal of Psychiatry, Special Issue, 39, 2000) encountered great difficulty in translating instruments between languages/cultures *within* Europe. However, as the UK becomes more integrated into Europe and subject directly to the European Convention on Human Rights, and as Western forms of psychiatry spread to other parts of the world, it would seem that such a programme of research is timely. Care should be taken to avoid Euro-centrism.

Perceived benefit

As the Review has shown, estimates of perceived benefit seem to be related to several different methodological factors, the main one being the timing of the assessment. The effects of this should be more thoroughly investigated and may be transferable to other mental health domains. In particular, first-time recipients of ECT should be interviewed immediately after treatment and then at a 6-9 month interval with other methodological factors found to be important in this review controlled. This could include a comparison between user-interviewers and clinical interviewers in the UK context as previous studies were conducted in North America.

The way in which both new and experienced consumers trade off, or balance, the risks and benefits of ECT should also be examined. This could be done through vignette studies depicting various scenarios associated with ECT which the users would rank or cluster in terms of the risks and benefits. The full range of experiences should be depicted including very negative and very positive experiences on a range of different dimensions. Some of these have been delineated in this report.

Appendix 1: Full corpus of research studies tabulated by methodological variables

Authors	Country and date	Design	Sample size	% Returns	Interval since ECT	Interviewer/ setting	Quantitative/ Qualitative	Author type
1. Gomez	UK 1975	?	96	N/A	Same day	Medic/ same Hospital	Quantitative	Clinical
2. Hillard & Folger	USA 1977	Quasi-Exp.	20 v 10	N/A	Varied	Non-treating doc / Mixed	Quantitative	Clinical
3. Freeman & Kendell	UK 1980	Retrospective	166	89%	Mean 1 year	Medic /Same Hospital	Quantitative / Mixed	Clinical
4. Freeman et al	UK 1980	Survey	26	N/A	Varied	Non-treating doc /Mixed	Mixed	Clinical
5. Hughes et al	UK 1981	Retrospective	72	90%	Mean 19 wks	Med student/ Same hosp.	Quantitative	Clinical
6. Kerr et al	Australia 1982	Quasi-Exp.	88 ECT	52.8%	Varied	Medic/Mixed	Quantitative	Clinical
7. Squire & Slater	UK 1983	Prospective	31 ECT	88%	3 years	Medic, Research	Quantitative	Clinical
8. Cowley	UK 1985	Part of RCT	96 + 23	N/A		Medic/ Same Hospital	Q-sort	Clinical
9. Freeman &Cheshire	N/A 1986	Review	N/A	N/A	N/A	N/A	N/A	Clinical
10. Freeman	N/A 1986	Review	N/A	N/A	N/A	N/A	N/A	Clinical
11. Aperia	Nordic 1986	Prospective	30	86%	2-8 years	Medic/ Same Hospital	Quantitative	Clinical
12. Baxter et al	USA 1986	Quasi-exp.	35+20	75%	During	Same hosp.	Quantitative	Clinical
13.Benbow	UK 1988	Prospective	54	92%	Pre-discharge	Medic/ Same hospital	Quantitative	Clinical
14. Malcolm	UK 1989	Prospective	100	98.5%	Before 1 week	Non-treating doc/ Hospital	Mixed	Clinical
15. Calev et al	USA 1991	Experimental / Time	23	56%	Repeated up to 6 months	Medic/ Mixed	Quantitative	Clinical

16. Szuba et al	USA 1991	? + Quasi-exp.	25 pts 25 rels	56.8%	Before/soon after?	Medic/ Same Hospital	Quantitative	Clinical
17. Fox	USA 1993	Case studies	4	N/A	Years	N/A	Histories	Clinical
18. Battersby et al	Australia 1993	Quasi-Exp.	24 ECT	85%	Varied	Self/ Hospital	Quantitative	Clinical
19. Rogers & Pilgrim	UK 1993	Survey	231	N/A	Varied	Researchers/ Day Care	Mixed	Social Science
20. Riordan et al	UK 1993	Prospective	37	75.5%	Mean 2 months	Non-treating doc/ Mixed	Quantitative/ Mixed?	Clinical
21. Pettinati et al	USA 1994	Quasi-Exp.	78	64%	6 months	Mixed	Quantitative	Clinical
22. Wallcraft	UK 1995	Pilot survey	10	N/A	Varied	Self / Postal	Mixed	Consumer
23. UKAN	UK 1995	Survey	308	N/A	Varied	Self / Postal	Mixed	Consumer
24. Mental Health Fnd'n	UK 1997	Survey	107 ECT	N/A	Varied	Self/ Day Care	Mixed	Vol Org/ Consumer
25. Sestoft et al	Nordic 1998	Retrospective	113	54%	Varied	Self /Postal	Quantitative	Clinical
26. Bernstein et al	US 1998	Survey	52	??	Varied	Mixed	Quantitative	Clinical
27. ECT Anonymous	UK 1998	Survey	200+	N/A	Varied	Self / Postal	Mixed	Consumer
28. Wheeldon et al	UK 1999	Prospective	150	93%	5-10 days	Medic/ Same hospital	Quantitative	Clinical
29. Johnstone	UK 1999	Survey	20	N/A	Varied	Researcher/ Choice	Qualitative	Clinical (Psychol)
30. Goodman et al	USA 1999	Quasi-exp.	24 ECT	45.3%	During + 2 weeks	Researcher/ Mixed	Quantitative	Clinical
31. Lisanby et al	USA 2000	Quasi-Exp.	33 ECT	60%	1 week + 2 months	Medic?/ Same	Quantitative	Clinical
32. Donahue	USA 2000	Review	N/A	N/A	N/A	N/A	N/A	Consumer
33. MIND	UK 2000	Survey	418	N/A	Varied	Self / Postal	Mixed	Vol Org / Consumer
34. Communicate	UK I.p	Survey	45	48.6%	Mean 19 weeks	Mixed	Mixed	Collaborative
35. MDF	UK I.p.	Survey			Varied	Self / Postal	Mixed	Consumer

Appendix 2: Studies tabulated by year, perceived benefit, willingness to have ECT again and five methodological variables (ranked).

Authors	Country and date	% Benefit	% Have Again	Interval since ECT (Note 1)	No. Qs (Note 2)	Complexity Schedule (Note 3)	Setting/Int. (Note 4)	Clinical
Freeman & Kendell	UK 1980a	78	59	3	3	4	2	Yes
Hughes et al	UK 1981	83	72	2	3	2	2	Yes
Kerr et al	Australia 1982	73		2	3	1	3	Yes
Aperia	Nordic 1986	70	63	3	2	3	2	Yes
Benbow	UK 1988	73		1	3	2	1	Yes
Szuba et al	USA 1991	76	72	1	1	2	1	Yes
Rogers & Pilgrim	UK 1993	43		3	3	3	4	
Riordan et al	UK 1993	56	67	2	3	4	3	Yes
Pettinati et al	USA 1994		98	1	1	1	1	Yes
UKAN	UK 1995	30	18	3	3	2	5	
Ment Health Fnd'n	UK 1997	30		3	2	3	5	
Bernstein et al	US 1998	83	79	0	2	1	1	Yes
ECT Anonymous	UK 1998	29		3			5	
Wheeldon et al	UK 1999	81	77	1	1	1	1	Yes
Goodman et al	USA 1999		82	0	3	1	2	Yes
MIND	UK 2000	36		3	3	3	4	
Communicate	UK Forthcoming	44	41	2	3	1	4	
MDF	UK Forthcoming	30		3	3		5	

Note 1: 0 = during course or maintenance ECT; 1 = within 4 weeks or pre-discharge; 2 = 1–6 months; 3 = > 6 months

Note 2: 1 = 5 questions or less; 2 = 6–14 questions; 3 = 15 or more questions

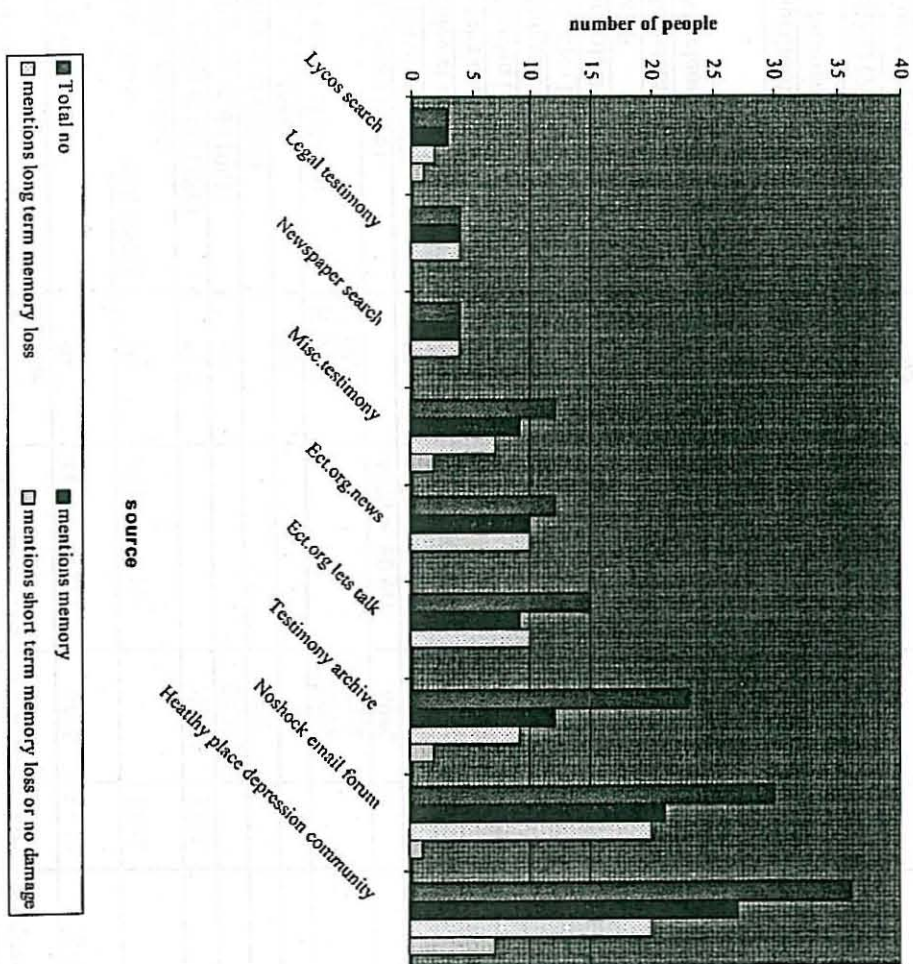
Note 3: 1 = 3-pt likert; 2 = simple likert; 3 = complex likert/ multiple choice; 4 = semi-structured interview

Note 4: 1 = in-patient; 2 = same hospital/treating doctor; 3 = non-treating doctor/at home; 4 = voluntary or collaborative, day care or choice; 5 = source is consumer organisation, choice of setting.

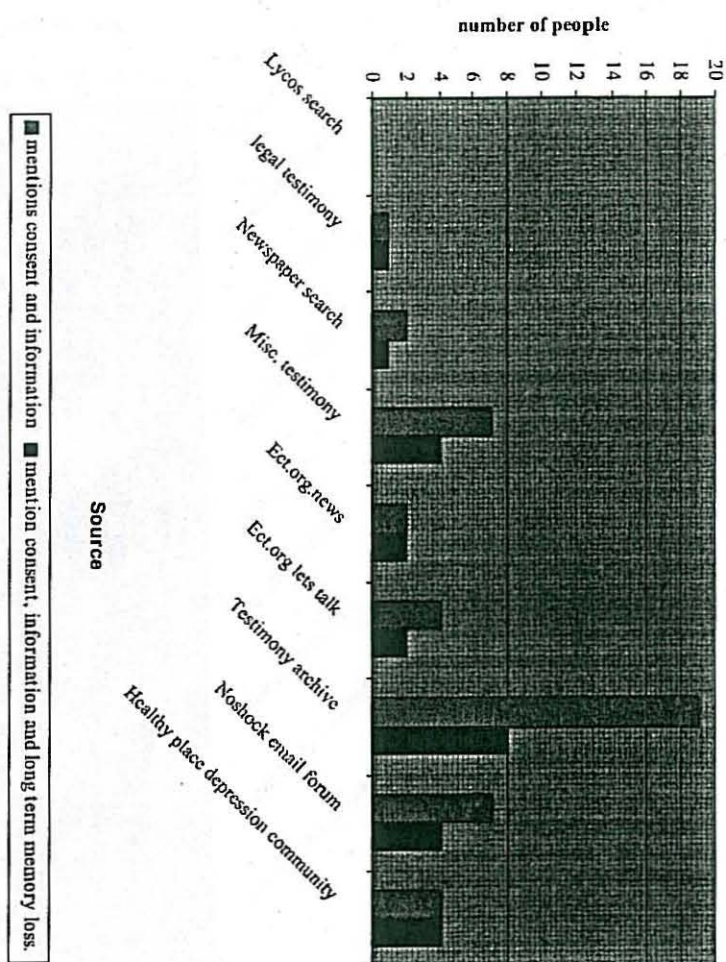
Appendix 3: Demographic Features of Study Samples in Chapter 7 and UK Norms 1999

Study	Age: Mean/ Median	Older consumers	% Female	SES	Sectioned	Legally compelled
UK norms 1999	59	41% > 65 yrs	68%	Low?	25%	19%
Freeman & Kendell (1980) (1976 sample)	50	None > 70 yrs		Med.		
Freeman et al (1980)	51.5		77.3%	Med.		
Hughes et al (1981)	49		59.7%			7%
Kerr et al (1982)			65%			
Aperia (1986)	57					
Baxter et al (1986)	E 39.6 C 60.2		E 57% C 70%			0% 0%
Benbow (1988)		41% > 60 yrs	69%			0%
Malcolm (1989)	68.5	59% > 65 yrs	68%			27%
Calev et al (1991)	48	None > 65 yrs	69.5%	High		0%
Szuba et al (1991)	45.2		72%			0%
Riordan (1993)	53.6	38% > 65 yrs	69.4%		16%	10%
Pettanati et al (1994)	60		66.7%			0%
UKAN (1995)	40	18% > 60 yrs	52.6%		27%	
Sestoft et al (1997)	68		77%			
MDF						
Bernstein et al (1998)						0%
Wheeldon et al (1999)	Inf. 55.2 S3 44.8 S 58 47.8		Inf. 65.5% S3 66.7% S58 72.2%		11%	7%
Goodman et al (1999)	58.3		50%	High		0%
Johnstone (1999)			60%	Low	30%	
MIND (2000)	52	10% > 65 yrs	57%		35%	
Communicate	63					2%

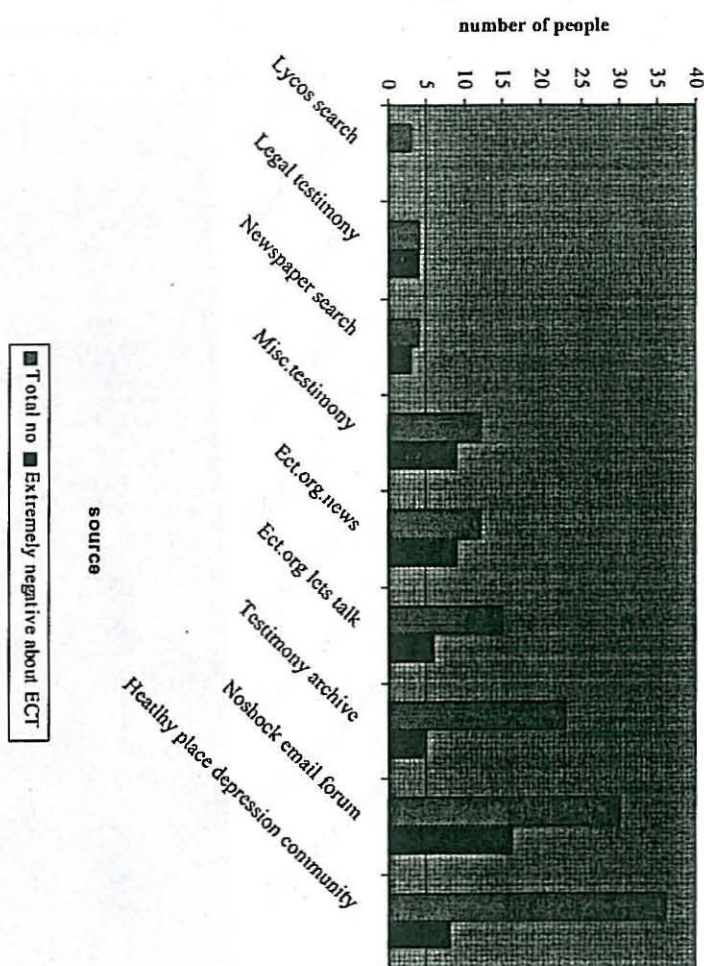
Appendix 4: a) ECT and Memory loss



Appendix 4: b) Consent, information and memory loss



Appendix 4: c) Negativity about ECT



APPENDIX: 5

SAMPLE TESTIMONIES AND ANALYSIS GRID

1) Mental Health Media Testimony Archive.

The following is extracted from the transcript of Pauline Stott's video interview. See Grid (1) for analysis.

'So it was mostly drug treatment but I did also have several courses of ECT, shock treatment as it's normally, as it's normally called. Shock treatment's in disfavour nowadays in a lot of areas. I don't know that it particularly helped me a great deal, but having said that, I have seen people make very good recoveries after ECT so perhaps it was over-used in the sixties, early seventies, but I still think, I personally still think it, it can be a very viable treatment and it can help some people. Really I, when I was in hospital, there used to be literally queues of trolleys going in for ECT. There was a long corridor, the trolleys would be lined up in the corridor then you would be pushed into this long room, the, sort of, the trolley sort of sideways, one after another. The anaesthetist would come round with the, the jabs that they give you before the actual anaesthetic. So, she, this, she were a lovely lady I remember her to this day, so, so cheerful and kind to everybody and she'd come along with her, a dish full of syringes and give you the, the pre, the thing you have before the anaesthetic and then you would be wheeled one by one into the actual room where the treatment was given. You would be anaesthetised in there and that would be it then until you, you woke up, often with a headache, but for myself I never had any, any long term effects. I haven't suffered any sort of permanent memory loss, you could lose memories for the, you know, the few hours before you have the shock treatment, but, no, I haven't suffered any long term damage. It's, it's one of those things at the moment that people are having strong feelings about it's use, but in my experiences of seeing what it's done for other people I do think it has a value, but perhaps, as is happening nowadays, it's, it's used more selectively, perhaps that's the secret.'

'Can you remember when you were first told that you would have ECT, or perhaps you were asked if you wanted it, I don't know.'

'I think it was probably more told than asked cos if you were Sectioned you weren't really asked. But having said that I wasn't unhappy to go along with it because I had seen how it had helped people.'

'You'd already seen, seen it being done to other people before you had the course?'

'Oh yes, yes, yes, so it, no it, I was obviously a bit apprehensive because I wasn't sure exactly what to expect, but because there was so many people on the ward who were having ECT it was done sort of, you know, on a twice weekly rota and you were seeing people go down to the ECT treatment area and coming back from it, you know, sort of, perhaps with a headache and a bit of memory loss but within a couple of hours they seemed fine. So it wasn't a problem and I'm not, I'm not easily scared about medical procedures anyway, so, for, for me, no, it wasn't a problem and I, I certainly wasn't reluctant to, to have it done because it was, it was so much part of the, of the, you know, the mental health treatment plans and the hospital. It was a fact of life, a fact of life and it didn't bother me the thought of having it done because I really hoped it would, it would help. So, yeah, some people have had bad, bad experiences of it, but my own experiences weren't bad and I certainly feel it, it still has a role to play for some people but other people obviously feel differently.'

'It was done quite routinely to most people was it?'

'Well not, lots of, what would happen, people would go into hospital and they would be treated, possibly for some months, well certainly weeks, more likely months with, with the anti-depressant drugs and it was a sort of, I think, you know, they'd have a look round and think well, you know, 'Such a body's not doing very well are they, so we'll try them with a course of ECT' and of course that happened to me also, you know, I had several courses of ECT and I think it was a result of people looking and 'oh well, she's not really doing very well on such and such, such and such a drug so we'll try the ECT', and yeah, it was, it wasn't very selective I don't think, no I don't.'

2)GOING MENTAL NO SHOCK WEBSITE

Web address <http://members.aol.com/noshockectforum.htm>¹

Part one: Front page:

This is the web page that precedes entry into the forum area:

ECT FORUM

Welcome to the ECT Message Board. This board has been created in order to give like-minded people an opportunity to debate one of the most controversial procedures used by psychiatrists; so that people can talk about their experiences; and as a place in which to give and receive advice.

All I ask is that we respect the opinions of others even when we don't agree with them; and that we not criticise or be abusive to people just because their views are different to ours. Messages that are abusive to other contributors will be removed. That said, enjoy the Board.

PART TWO: SAMPLE THREAD

The thread is entitled *Survivor of 9 treatments* and the originating message is from Lisa: User ID: 9823593. for analysis see Grid (2)

Apr 3rd 11:48 PM
I am 39 years old, married, with two daughters. Almost five years ago I underwent 9 E.C.T. treatments! At the advice of my husband and Dr. I have had permanent memory loss. And feel worse than I did, physically and mentally. I am a RN, but due to the memory loss I am not working. I know I have brain damage, even though I have not been properly diagnosed with it, I know it is there. I regret taking the ECT treatments. No one understands in my family, my daughters are 16 and 11 now, but husband has shown no support/compassion to my illness. A supportive, understanding, and experienced voice would help to know that there are others like me.

Barb
User ID: 9823593

Apr 4th 1:25 AM
Hi Lisa
I'm a former teacher and former RN. ECT disabled me. Depression did not stop me from going through nursing school, graduating with ECT disabled me and the damage to my brain is

¹ NB since our download, noshock and ect.org have created much closer links and merged their email forums.

verified.

I was dumb enough to have ECT three times, but the last treatments (out pt, 1983) were essentially the end of who I was.

My testimony is on this site under "Evidence."

You might try to at least get an EEG done, if you have insurance. That may help verify the damage.

There are many "like you" out here. Take care, Lisa, and I'm glad you've posted on the forum.

Barb

Apr 4th 8:23 PM

Thanks for your response Barb. I have read your testimony and other messages on the board. I am an avid reader when I can concentrate. Illness and stress doesn't help that. I would like to hear from others like myself and who are still in the process of trying to raise a family. I've changed a lot and it's difficult for all of us. I can feel pretty guilty at times for the ways emotionally, physically and financially I have affected my husband and daughters. Am hoping to hear from someone. Thank you

May 21st 6:19 PM

Dear Lisa, My heart went out to you as I read your last post. I too am raising a family--oldest 14, youngest 10 months. ECT affected all my relationships with my family. I feel like a stranger in my own body. My kids, bless their hearts, continue to love me. I feel so bad that my love for them has been screwed up but I do my best to be kind and continue to discipline them as appropriate. Also my love for my husband has been stolen. He continues to love me and listen to me but I know the rages on go into are very trying. a lesser man would have left me. sometimes I beg him to get rid of me because now I am such a mess and he deserves far better. I hope I can make good memories with my family. Sometimes I think I probably won't live very long because of how traumatic the experience of the "treatments" was, but I really have no idea. I am trying to take extra good care of myself and my family so hopefully we'll make it thru this nightmare.

Grid (1) Pauline Stott Testimony Archive

	Age Sex	No. ECT	timescale	Dup	Memory (m)	Consent / info (c)	Emotional response (e)	Costs and benefits (c&b)	Open Cat. (o)
Testimony archive Pauline Stott	F58	Several courses	unknown		, but for myself I never had any, any long-term effects. I haven't suffered any sort of permanent memory loss, you could lose memories for the, you know, the few hours before you have the shock treatment, but, no, I haven't suffered any long term damage.	"I think it was probably more told than asked cos if you were sectioned you really weren't asked. But having said that I wasn't unhappy to go along with it because I had seen how it had helped people"	"So it wasn't a problem and I'm not, I'm not easily scared about medical procedures anyway, so, for, for me, no, it wasn't a problem and I, I certainly wasn't reluctant to, to have it done"	"It was a fact of life, a fact of life and it didn't bother me the thought of having it done because I really hoped it would, it would help. So, yeah, some people have had bad, bad experiences of it, but my own experiences weren't bad"	

Grid (2) Survivor of 9 treatments Noshock email forum

Lisa	F39	9	5 yrs ago		"Permanent memory loss"		No one understands in my family – wants support and understanding	Feels worse physically and mentally, not working	
Barb	F	3 series	Last series 1983 as out patient	X	"ECT disabled me and the damage to my brain is verified".			"The last treatments were essentially the end of who I was"	
Lisa				X			ECT affected all my relationships with my family. I feel like a stranger in my own body.		
Hoping	F			X			treatments had profound effect on life and relationships	"sometimes i think i probably won't live very long because of how traumatic the experience of the "treatments" was.	

Appendix 6

The following are extracts from the Royal College of Psychiatrist's Factsheet on ECT that are particularly relevant to the themes covered in this Review. When the Review refers to 'the medical consensus' or 'established medical opinion' it is this published statement of a professional body that is indicated. This is the information most commonly seen by UK consumers and it was current on the Internet at the time of writing.

Royal College of Psychiatrists Factsheet on ECT - extracts

"For most people ECT is a short-acting treatment, but it does act more quickly than drugs. This can be life-saving." (pp1-2)

"Over 8 out of 10 depressed patients who receive ECT respond well to it. In fact, ECT is the most effective treatment for severe depression. People who respond well.....report it makes them feel 'like themselves again' or 'as if life was worth living again'." (p. 3)

"What are the side-effects of ECT?"

Some patients may be confused just after they wake from the treatment and this generally clears up within an hour or so. Your memory of recent events may be upset and dates, names of friends, public events, addresses and telephone numbers may be temporarily forgotten. In most cases this memory loss goes away with a few days or weeks although some patients continue to experience memory problems for several months. As far as we know, ECT does not have any long term effects on your memory or intelligence." (pp.3-4)

"Can I refuse to have ECT?"

You can refuse to have ECT and may withdraw your consent at any time even before the first treatment has been given. The consent form is not a legal document and does not commit you to having the treatment. It is a record that an explanation has been given to you and that you understand to your satisfaction what is going to happen to you.....

Very occasionally a person may become particularly seriously ill with depression. They may be suicidal, convinced they are too wicked to be treated, or even eating and drinking too little to stay alive for much longer. In these circumstances ECT may be given to patients without their consent." (p.4)

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Chronic, treatment-resistant depression and right fronto-striatal atrophy

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Background Treatment-resistant depression (TRD) is relatively common but its neurobiological basis is poorly understood. Fronto-striatal structural brain changes have been reported in patients with depression but their association with treatment resistance and chronicity has not been established.

Method Magnetic resonance images of 20 patients with TRD were compared with images of 20 recovered patients and 20 healthy controls. Images were compared using a voxel-based analysis (VBA) method; the results were validated by conventional volumetric analysis. The clinical associations of magnetic resonance imaging (MRI) changes with illness duration and severity were examined by VBA.

Results Only the TRD group exhibited right fronto-striatal atrophy, and subtle MRI changes in the left hippocampus on VBA. Atrophy was confirmed on volumetric analysis, the degree correlating with the cumulative number of electroconvulsive therapy (ECT) treatments received, suggesting an acquired deficit.

Conclusions This is the first study to demonstrate fronto-striatal atrophy in patients with depression with poor outcome; the atrophy is more marked in those with more severe illness.

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Up to 20% of middle-aged patients with depression attending a psychiatrist follow a treatment-resistant course lasting more than 2 years (Keller *et al.*, 1982; Scott, 1988). Treatment-resistant depression (TRD) is associated with a longer duration of illness before treatment (Keller *et al.*, 1984; Scott, 1988) and a substantially reduced rate of recovery after the first 2 years of illness (Keller *et al.*, 1982, 1984). Such patients are presumably the most likely to show structural brain changes. The most consistent structural findings from studies of mixed-outcome patients with depression are frontal and striatal atrophy (Soares & Mann, 1997), but it has not been established whether or not these changes are related to outcome. Previously we have used voxel-based analysis (VBA) to compare magnetic resonance imaging (MRI) grey matter segments between middle-aged patients with TRD, patients who had recovered from depression and normal healthy volunteers (Shah *et al.*, 1998). Patients with TRD had reduced grey matter density in the frontal and temporal cortex, including the hippocampus. Additionally, reduced hippocampal grey matter density was correlated with verbal memory impairment, implying an association between observed change and function. It is not possible to be sure whether VBA changes represented atrophy or just changes in voxel intensity. This required examination by traditional volumetry and will be described here.

METHOD

Subjects

Twenty patients aged between 21 and 65 years who fulfilled DSM-IV (American Psychiatric Association, 1994) criteria for major depressive disorder (chronic), research diagnostic criteria (RDC; Endicott & Spitzer, 1978) for a primary major depressive disorder and were also treatment

resistant, were recruited from general adult in-patient units and from out-patient clinics in the south-east of Scotland. With this selection we endeavoured to maximise our chances of identifying structural brain changes. All subjects gave written informed consent following a protocol approved by the local research ethics committee prior to participation. Chronicity was defined as meeting DSM-IV diagnostic criteria for a major depressive episode for at least 2 years (American Psychiatric Association, 1994). Treatment resistance was defined as non-responsiveness to at least two treatments from different pharmacological groups employed for at least 4 weeks each at the following doses:

- at least 150 mg of imipramine or an equivalent tricyclic antidepressant;
- at least 60 mg of phenelzine or an equivalent monoamine oxidase inhibitor;
- at least 40 mg of fluoxetine or an equivalent selective serotonin reuptake inhibitor;
- at least six treatments with electroconvulsive therapy (ECT) with seizures lasting longer than 20 s each.

In reality, all patients exceeded the minimum criteria for treatment resistance. Many were on multiple and/or combination treatments. All patients were on a stable medication regime for at least 2 weeks prior to the study. Patients had not received ECT for at least 3 months prior to the study and had no history of intracranial pathology or surgery.

Twenty recovered patients who previously fulfilled DSM-IV criteria for a major depressive disorder and 20 normal healthy volunteers with no lifetime history of psychiatric illness were also examined. Subjects from both of these groups were individually matched with patients with TRD for age, gender, premorbid IQ and years of education. Recovered patients were matched for age of onset and for onset of the index episode with the treatment-resistant group. The recovered group all had had severe illness episodes (details below). Recovery was defined as scoring 5 or less on the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) for at least 3 months prior to the study, and subjects were either medication-free or on stable medication for at least 2 weeks prior to the study.

Clinical assessment

All subjects were interviewed using the lifetime version of the Schedule for Affective Disorders and Schizophrenia (SADS-L; Endicott & Spitzer, 1978). All available psychiatric case notes were reviewed in detail, providing RDC diagnoses and allowing lifetime histories of psychiatric illness and treatment histories to be reconstructed. The total number of hospitalisations, cumulative length of psychiatric hospitalisation and cumulative number of ECT treatments were used as indices of cumulative illness severity. Estimated total lifetime illness duration was derived from psychiatric case-note histories. Healthy volunteers had no lifetime history of significant psychiatric illness, established by the SADS-L interview schedule.

Exclusion criteria were previous manic episodes, other organic cerebral pathology, significant alcohol or substance misuse, head injury associated with significant loss of consciousness, or concurrent use of steroids.

All subjects had standardised neuropsychological and clinical testing within 1 day of each other and within 1 week of the MRL. Symptom severity was measured using the HRSD, the severity of psychomotor retardation was measured using the observer-rated Widlöcher Scale (Widlöcher, 1983) and cerebral dominance was measured with a handedness scale (Annett, 1970). Subjects also performed the revised National Adult Reading Test (Nelson & Willison, 1991), which estimated premorbid IQ.

Magnetic resonance image acquisition

Subjects were imaged within 1 week of clinical assessment. Images were acquired on a 1.0 Tesla Siemens Magnetom SPE system, with subjects undergoing a magnetisation-prepared rapid-acquisition-graded echo (MPRAGE) sequence, acquired perpendicular to a line connecting the anterior and posterior commissure (AC-PC). This yielded high-resolution T1-weighted images with good contrast between white and grey matter (repetition time=10 ms, delay time=500 ms, inversion time=200 ms, flip angle=12°, block size=240 mm, 128 contiguous slices with an effective slice thickness of 1.875 mm).

Voxel-based analysis

Image analysis was performed on a SPARC workstation (Sun Microsystems Europe Inc., Surrey, UK) using ANALYZE software (version 7.5.5, 1995; Biomedical Imaging Resource, Mayo Foundation, Rochester, Minnesota, USA), and SPM'96 software for spatial normalisation and statistical parametric mapping (Wellcome Functional Imaging Laboratory, Institute of Neurology, Queen Square, London, UK), running in MATLAB (version 4.2c, 1994; The Mathworks Inc., Natick, Massachusetts, USA). The technique is fully described by Shah *et al* (1998). An ANCOVA model was applied, removing the global density of each tissue compartment for each subject. Differences between groups were displayed as statistical parametric maps (SPMs), with a 1% threshold probability. Statistical clusters were also projected onto the T1-weighted grey matter density template to facilitate interpretation of the results. Corrected probability values take into account the volume examined, the smoothness of the data, the size of the cluster with $P < 0.01$ and the peak effect (Z value).

Volumetric analysis

Images were analysed using ANALYZE (CNS Software) running on a Unix-based Sun workstation (Sun Microsystems). Images initially were converted to 8-bit images. The threshold voxel intensity between grey matter and cerebrospinal fluid (CSF) was ascertained. Tissue below this threshold (surrounding CSF) and exterior to this rim of CSF (skull, scalp and meninges) was excluded. Meningeal tissue abutting on cerebral tissue was removed manually using an anatomical atlas as a guide. Within ANALYZE, images were corrected for minor degrees of tilt, roll and yaw. Partial volume effects at the exterior edge of cerebral tissue were removed using a 1-bit image template multiplier, re-oriented in an identical manner to the original image using nearest-neighbour interpolation. Total cerebral volume thus remained unchanged after re-orientation.

Landmarks used to delineate cerebral structures

The landmarks defined by Shenton *et al* (1992) and Suddath *et al* (1990) were used as a guide to dissection. The criteria used

are available from the authors upon request. The hippocampus was divided into anterior and posterior portions, using the mamillary bodies as a landmark. No attempt was made to measure amygdala volume separately. Caudate and putamen were measured bilaterally, as was prefrontal tissue, 'posterior frontal' tissue and the temporal lobes.

Segmentation of magnetic resonance images

Two investigators received training to identify landmarks accurately and to segment images reliably into object maps. A third investigator then removed identifying information from the magnetic resonance images. He randomly chose half of the images from each subject group to be mirrored in the midsagittal plane so that the left and right sides were exchanged. The two investigators who segmented the magnetic resonance images were thus blind to left-right orientation and the diagnostic group of the images. Each investigator independently analysed the images from half of the total subjects. Five random images were analysed independently by both investigators, allowing a measurement of interrater reliability. One of the investigators performed a repeat analysis of the same five images 2 months later, producing a measure of intrarater reliability. One-tailed t -tests were used (SPSS for Macintosh, version 4.0) because the direction of change was predicted, and no correction for multiple comparison was used because there were specific hypotheses about the regions expected to show volumetric reductions in patients with TRD. Because the total cerebral tissue volume did not differ between the three groups, controlling for total cerebral volume was not required.

Group demographics and clinical data were compared using univariate analysis of variance and *post hoc* independent t -tests to identify specific group differences. Non-continuous variables were compared using the Mann-Whitney U and χ^2 tests, with correlations made using Spearman non-parametric correlation as appropriate. Data reduction was done with the appropriate programs of SPSS 10.0 for Windows.

RESULTS

The three groups did not differ in age, gender, handedness, years of education or

the total number of years smoking (Table 1). The mean age of onset of the first and most recent and the lifetime number of depressive episodes did not differ between the TRD and recovered groups. The TRD group had longer current episode and total duration of illness and had longer total and a greater number of hospitalizations.

All TRD patients were taking antidepressant drugs. Additionally, twelve patients took regular neuroleptic medication, five took lithium and three took benzodiazepines. Eleven patients in the recovered group were medication-free. Nine of the recovered patients were prescribed antidepressants; one also received neuroleptics and one lithium.

Both the patients with TRD and the recovered patients had, or previously had, melancholic depressive episodes. The TRD group had endogenous symptoms as measured by the Newcastle Scale (Carney *et al*, 1965) and fulfilled the DSM-IV criteria for a depressive episode with melancholic features (American Psychiatric Association, 1994). The recovered patients also previously fulfilled DSM-IV criteria for having depressive episodes with melancholic features.

The TRD group had moderately severe depressive symptoms and psychomotor retardation. Although not clinically depressed, the recovered patients had

significantly more depressive symptoms and more observable motor retardation than the controls.

Voxel-based analysis

The three-group comparison of increases and decreases in each of the three tissue compartments yielded 18 SPMs. Only the TRD group had changes in all three tissue compartments in comparison with the other two groups. Because the areas of differences were virtually identical when comparing the TRD group with controls and recovered patients, and because there was no significant tissue difference between the recovered group and the controls, the TRD group was compared against the pooled recovered and control groups.

Limbic and striatal changes in compartmental densities in TRD

The TRD group had reduced tissue density in the right putamen and a corresponding increase in right lateral ventricular CSF overlying the right striatum, not reaching statistical significance because of the small volumes involved but complementing the volumetric analysis. In addition, a pattern of reciprocal grey matter reductions with overlapping apparent white matter increases was found in bilateral hippocampal and parahippocampal areas, particularly anteriorly, and more markedly on the left

(Fig. 1), upon VBA but not volumetric analysis. Reciprocal changes in grey and white matter, with no increase in CSF, suggest that volumetric change in these areas will not be detected easily.

Cortical change (Fig. 1)

The TRD group had reduced grey matter density in the right superior frontal gyrus, with large reductions in white matter density in the right medial and superior frontal gyri. There were corresponding large CSF increases over the right medial and superior frontal cortex (see Table 2). These changes predict right prefrontal atrophy in volumetric analysis. The large grey matter density reductions in the left superior and medial temporal gyri did not have associated CSF changes, suggesting that this change would not be detected by volumetry. Finally, an unpredicted finding upon VBA was increased grey matter density in the left cuneus, precuneus and lingual gyrus in the TRD group, with a lesser increase in bilateral cerebellar grey density (see Shah *et al*, 1998).

Volumetric object mapping

The TRD patients had less right prefrontal lobe tissue than controls (65.18 cm^3 v. 71.17 cm^3 , $t = -2.34$, $P = 0.012$, effect size = 0.74) and less right caudate tissue than both controls (3.51 v. 3.77 cm^3 ,

Table 1 Group comparisons, with standard deviation (s.d.) in parentheses ($n=20$)

Parameter	Treatment-resistant depression group	Recovered group	Controls	$F_{(1,57)}$ (probability) or t -test (probability for two-group comparisons ¹)	Post-hoc t -test
Mean age	48.9 (9.8)	47.7 (9.9)	49.3 (11.8)	0.89	
Male:female ratio	13:7	13:7	13:7		
Handedness score	13 (16)	16 (13)	19 (5)	0.27	
Years of education	11.7 (2.9)	13.4 (3.4)	13.5 (2.9)	0.12	
NART IQ	107 (12.7)	113 (10.2)	115 (8.7)	0.06	TRD=RD
					TRD < C
HRSD score	20.6 (5.3)	2.6 (1.7)	0.2 (0.7)	<0.0001	TRD > RD > C
Widlöcher score	26.1 (8.6)	2.2 (3.6)	0.3 (0.7)	<0.0001	TRD > RD > C
Total years smoking	20 (13.6)	12 (13.7)	17 (3.7)	0.14	
Age of onset of first episode	38.9 (13.5)	38.2 (10.1)		0.89	
Age of onset of last episode	45.8 (10.1)	44.8 (9.8)		0.76	
Total number of episodes	2.2 (1.4)	2.5 (1.9)		0.52	
Longest duration of episode (weeks)	197 (125)	46 (36)		<0.001	
Lifetime total illness duration (weeks)	263 (133)	76 (58)		<0.001	
Total number of hospitalizations	3.8 (5.3)	1.3 (1.2)		0.05	

NART, National Adult Reading Test; HRSD, Hamilton Rating Scale for Depression.

1. The two patient groups are compared where no data were available for controls (last six parameters).

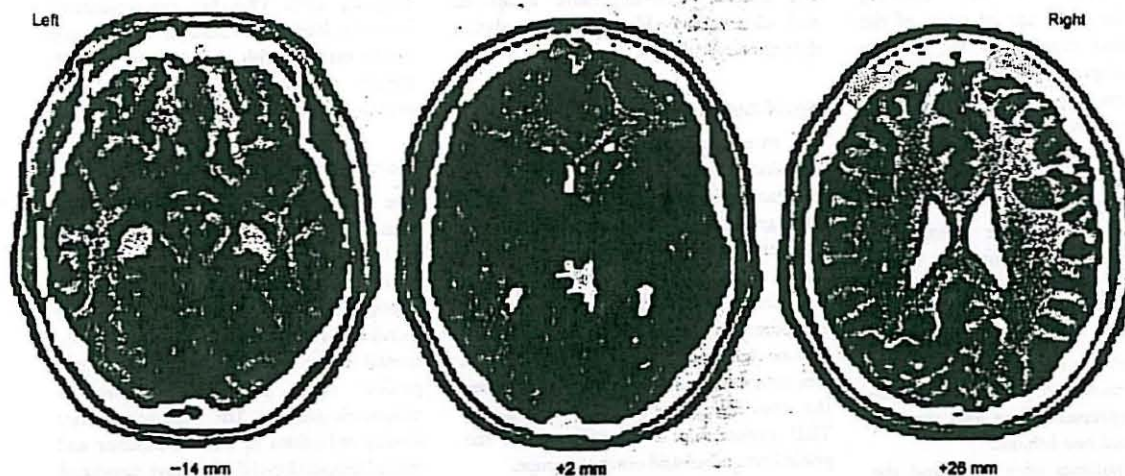


Fig. 1 The Z-maps of tissue changes ($Z > 2.33$, $P < 0.01$) in the treatment-resistant depression group have been colour-coded and then superimposed onto the appropriate transverse section to facilitate interpretation. The labels indicate the distance above or below the transverse plane formed by a line passing from the anterior to the posterior commissures. Blue, reductions in grey matter density; purple, reductions in white matter density; green, increases in grey matter density; yellow, increases in white matter density; orange, increases in CSF; red, blue+yellow.

$t = -1.7$, $P = 0.048$, effect size = 0.54) and recovered patients (3.51 v. 3.80 cm³, $t = -1.77$, $P = 0.04$, effect size = 0.56). Interrater reliability of volumetric object mapping varied from 0.85 (right anterior hippocampus) to 0.99 (posterior frontal tissue), and intrarater reliability from 0.76 (left anterior hippocampus) to 0.98 (posterior frontal tissue).

Correlation of grey matter density in TRD with selected clinical variables

Severity of illness over time was thought to be represented best by the total number of ECT treatments administered, the total duration of hospitalisation and the total number of admissions. Data reduction with the number of ECT treatments in both patient groups, duration of hospitalisation, number of admissions and treatment resistance entered as (dummy) variables in a principal-components analysis with subsequent varimax rotation, resulted in two rotated factors: one represented the two patient groups and accounted for 35% of the overall variance and loading on duration of hospitalisation (0.86) and treatment resistance (-0.83); and the other accounted for the majority of the variance (53%) and loaded on number of ECT treatments (0.96) and number of hospital admissions (0.98). Electroconvulsive therapy at the

Royal Edinburgh Hospital is usually reserved for severely depressed patients requiring admission and for those unresponsive to conventional pharmacotherapy. Although variability in its use between consultants cannot be neglected, the variations observed within the hospital are probably less than those observed nationwide. Nevertheless, the use of ECT as a measure of illness severity clearly has its limitations. As the total number of ECT treatments, total duration of hospitalisation and total number of admissions were highly inter-correlated, we elected to use the total number of ECT treatments as the measure of cumulative illness severity, aware that a significant association of ECT with cortical tissue reductions may have alternative interpretations.

Increasing cumulative ECT correlated extensively with reduced bilateral superior frontal gyri, bilateral superior frontal and inferior parietal gyri, bilateral medial and superior temporal gyri and bilateral caudate grey matter density in the TRD group (Fig. 2) upon VBA. The reduced grey matter density correlations with ECT were unaffected, even after accounting for the current severity of depression (using the HRSD score as a covariate). Thus, neocortical and striatal grey matter reductions appeared to be proportional to the cumulative severity of depression. We repeated the group comparison of grey matter

density between the TRD and recovered groups, controlling for age and the number of ECT treatments administered, and found that only reductions in grey density in the left hippocampus remained. Thus, reduced hippocampal grey matter density in TRD seemed to be unrelated to the cumulative severity (or cumulative ECT received) or duration of illness. Additionally, the estimated total duration of illness did not correlate with neocortical grey density reductions.

DISCUSSION

This study confirms our prediction that patients with TRD exhibit right superior, medio-frontal and striatal atrophy. The degree of atrophy was proportional to a proxy of illness severity over time. It also confirmed that only patients with TRD have hippocampal and rostral anterior cingulate changes, possibly reflecting an altered focal biochemical environment or tissue composition reflected in altered T1 values. No difference was found between controls and patients recovered from depression. The findings of atrophy in right fronto-striatal structures were replicated using the 'gold standard' of conventional volumetry, thus providing validity for the use of SPM'96, a voxel-based approach to analysing high-resolution structural magnetic resonance images.

Table 2 Significant compartmental changes and reciprocal changes in adjacent compartments in patients with treatment-resistant depression compared with the combined control and recovered groups

Compartment density changes	Size of cluster, all Z > 2.33 (uncorrected P, corrected at cluster level)	Coordinates of peak voxels within cluster (uncorrected P)
Left hippocampal grey matter decrease	7.0 cm ³ (P=0.001) Corrected P=0.04	-29, -27, -12 (<0.001) -30, -18, -18 (<0.001) -36, -15, -26 (<0.001)
Reciprocal white matter increase	2.9 cm ³ (P=0.03) Corrected P=NS	-29, -24, -14 (<0.001) -38, -22, -18 (<0.001) -37, -10, -28 (<0.001)
Left superior temporal and precentral grey matter decrease	23.8 cm ³ (P<0.001) Corrected P=0.001	-64, -23, 8 (<0.001) -56, 1, 32 (<0.001) -59, -17, 36 (<0.001)
Right superior and medial frontal white matter decrease	10.7 cm ³ (P<0.001) Corrected P=0.01	31, 50, 4 (<0.001) 36, 41, 10 (<0.001) 18, 50, 24 (<0.001)
Right superior frontal grey matter decrease	2.1 cm ³ (P=0.04) Corrected P=NS	22, 41, 48 (<0.001) 34, 26, 52 (<0.001) 26, 52, 32 (<0.001)
Reciprocal right frontal cerebrospinal fluid increase	1.8 cm ³ (P<0.001) Corrected P=0.007	28, 53, 32 (<0.001) 41, 48, 24 (<0.001) 19, 42, 44 (<0.001)
Increased left frontal cerebrospinal fluid	11.4 cm ³ (P=0.002) Corrected P=0.02	-39, 53, 14 (<0.001) -38, 53, 14 (<0.001) -41, 19, 54 (0.002)
Reciprocal left medial and superior frontal white matter decrease	3 cm ³ (P=0.03) Corrected P=NS	-24, 46, 6 (<0.001) -26, 35, 18 (0.005)

Methods of analysis

Conventional volumetry has a number of limitations. It involves manual segmentation of brain regions, reducing spatial precision and reliability. It assumes functional and structural homogeneity and, because regions are constrained by *a priori* hypotheses, only examines parts of the data set (or image). Only volumetric aspects of the data are examined, making the assumption that MRI reflects structure. Because T1-weighted images are cross-sectional measurements of water distribution and chemistry, they are influenced by tissue characteristics (e.g. fat content), regional metabolism and blood flow. Thus, they may reflect both state-dependent and anatomical differences.

In contrast, VBA detected a range of MRI changes. With atrophy, CSF replaces grey and white matter. Thus, atrophy is represented by reductions in grey and/or white matter together with a corresponding

increase in CSF. If, however, water density decreased without cell loss, then the voxel signals may be brighter. In areas where grey and white matter are in close proximity, brighter voxels have a higher probability of being assigned to the white matter compartment instead of grey, producing reduced grey matter density but an overlapping apparent increased white matter density. Thus, the reciprocal grey and white matter changes seen in the anterior hippocampus, rostral anterior cingulate (Brodmann area 24) and posterior cingulate/precuneus are not likely to be frank atrophy but, rather, indicative of a change in tissue composition.

Fronto-striatal atrophy

Our findings of fronto-striatal atrophy are consistent with previous studies (Husain *et al*, 1991; Coffey *et al*, 1993; Dupont *et al*, 1995). Reduced metabolism in the

rostral anterior cingulate in treatment non-responsive patients has also been shown (Mayberg *et al*, 1997). However, findings of temporal lobe changes have been more equivocal (Coffey *et al*, 1993).

At present, there is no clear hypothesis as to which neuronal systems are involved with the atrophy. However, a brain morphometric study (Rajkowska *et al*, 1999) of patients with major depression found cell atrophy in cortical layers of the rostral orbito-frontal cortex (Brodmann areas 10-47) associated with serotonergic neurons, and in layers associated with dopaminergic and glutaminergic neurons in dorso-lateral prefrontal cortex (Brodmann area 9), extensively connected with striatum. We also found right frontal atrophy in these Brodmann areas. Interestingly, *in vivo* studies have found reduced striatal dopamine release in depression, proportional to the severity of motor slowing (Ebert *et al*, 1994; Shah *et al*, 1997) and that the mood-activating properties of psychostimulants are particularly linked to dopamine (Swerdlow & Koob, 1987). The characteristics of possibly irreversible cognitive deficits in depression also support fronto-striatal involvement, raising the possibility of a 'fronto-striatal dementia' (reviewed in Robbins *et al*, 1992). Thus, it could be speculated that treatment resistance in depression may be related to a loss of dopamine neurons or their function.

Fronto-striatal atrophy, however, is not diagnosis-specific; similar fronto-striatal changes are found in schizophrenia, and may be related to the 'poverty syndrome' characterised by poverty of affect, movement and initiation. At a speculative level, such atrophy may be the final common pathway for severe melancholia, also characterised by poverty of affect, movement and initiation, and for chronic schizophrenia.

Tissue changes in hippocampal and rostral anterior cingulate

Much attention has been paid to hippocampal changes in depression owing to the notion that stress may produce cellular damage. We did not find volumetric change, in contrast to other studies (e.g. Sheline *et al*, 1999), but rather evidence of change in tissue composition, which appeared to be unrelated to illness severity (or to ECT). Our rostral anterior cingulate

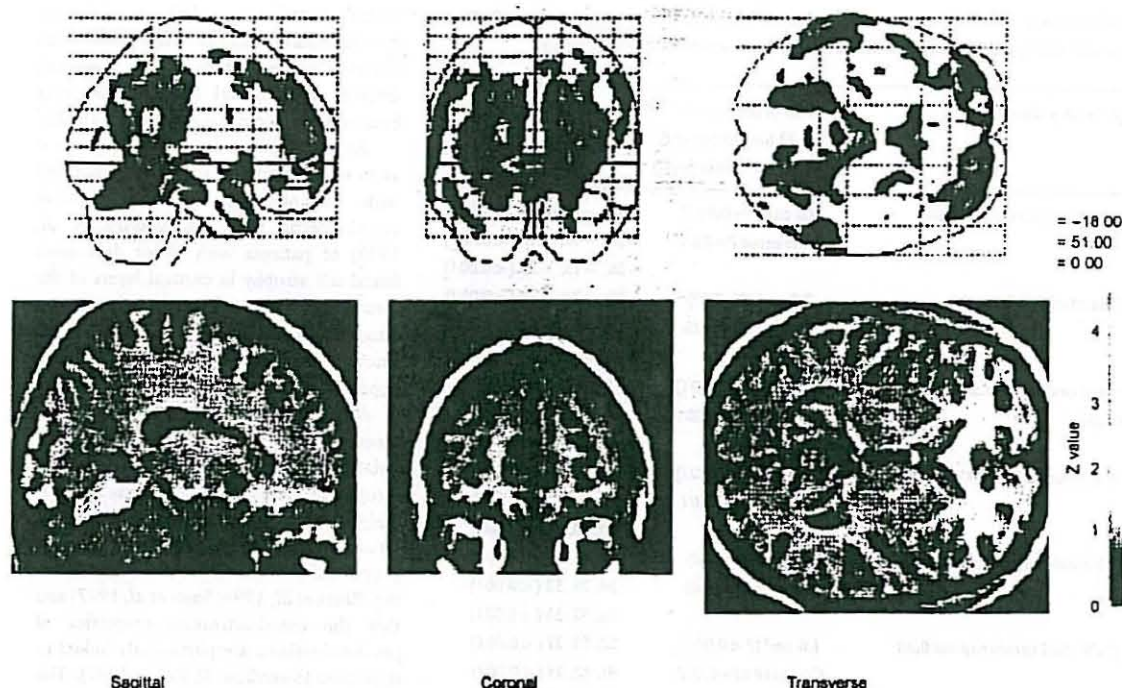


Fig. 2 Negative correlations between grey density and total number of electroconvulsive therapy treatments in patients with treatment-resistant depression ($Z > 2.33$, $P < 0.01$). The upper series represents areas of significant negative correlation viewed in maximum intensity projection (MIP). The lower series illustrates the correlations with grey matter superimposed on three sections taken at the point marked on the (MIP) view.

changes (Brodmann area 24) also agree with Mayberg's (1997) notion of specific metabolic changes in this area in treatment-resistant patients. Our results suggest that this may represent metabolic rather than structural change.

Limitations

Although it could be argued that the differences were the effects of ECT, there is little current evidence that ECT can produce permanent hippocampal or other structural brain changes (Devanand *et al.*, 1994). Because of this, and because the total number of ECT treatments, total duration of in-patient stay and total number of hospitalisations were closely inter-correlated, it seemed reasonable to regard the total number of ECT treatments administered as being an index of cumulative severity. However, the possibility that the findings are ECT-related cannot be discounted. Similarly, all the patients with TRD were medicated,

as were about half of the recovered patients. It was not possible to withdraw medication on these subjects.

Because the study is cross-sectional in design, it is not possible to distinguish between state-dependent, acquired and permanent changes, especially as apparent atrophy on MRI has been found to be partially reversible with illness resolution in conditions such as anorexia nervosa and alcohol dependence. Patients with depression often lose weight, which could produce a general effect. However, it is difficult to see how this would produce specific focal brain changes. The exact time course of these changes in relation to the illness needs to be determined. One possibility is that these brain differences are present prior to illness and confer vulnerability to treatment resistance. However, given that the TRD group had no clinical evidence of premorbid impairment and that the atrophy was more severe in those with the most severe illness, it is more likely that these differences were acquired.

ACKNOWLEDGEMENTS

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CLINICAL IMPLICATIONS

- Patients with treatment-resistant depression had right fronto-striatal atrophy detected with voxel-based and volumetric methods.
- There were also tissue changes, only detected with voxel-based analysis, in the left temporal lobe, left hippocampus, rostral anterior cingulate and posterior cingulate/precuneus.
- The severity of atrophy was greater in those with a longer, more severe illness.

LIMITATIONS

- In this cross-sectional study, we cannot determine whether changes are the causes or consequences of treatment resistance, aggressive treatments such as electroconvulsive therapy or the illness process per se.
- Our treatment-resistant patients were also chronically ill, so the effect of treatment resistance per se may have been confounded by chronicity.
- As in all relatively small studies in this field, independent replication is essential.

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The first part of the document discusses the importance of maintaining accurate records of all transactions. It emphasizes that every entry, no matter how small, should be recorded to ensure the integrity of the financial data. This includes not only sales and purchases but also expenses and income. The document further states that regular audits are essential to verify the accuracy of these records and to identify any discrepancies or errors.

In the second part, the focus shifts to the management of cash flow. It highlights the need for a clear understanding of the company's current financial position, including its assets, liabilities, and equity. The document advises on how to effectively manage cash flow by monitoring incoming and outgoing payments, ensuring that the company has sufficient funds to meet its obligations, and planning for future financial needs.

The third section addresses the issue of budgeting and financial planning. It explains how a well-defined budget can help the company allocate resources efficiently, control costs, and achieve its financial goals. The document provides guidance on how to develop a realistic budget based on historical data and market trends, and how to use it as a tool for decision-making and performance evaluation.

Finally, the document discusses the importance of transparency and communication in financial management. It stresses that all stakeholders, including management, employees, and investors, should have access to accurate and timely financial information. This transparency is crucial for building trust, making informed decisions, and ensuring the long-term success of the organization.

A Long-Term Study of the Effects of Electro-Convulsions on the Structure of the Cerebral Cortex

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Summary. In this study is described the quantitative structure of one circumscribed area of the cerebral cortex 2 months after a series of electro-convulsions. The results indicate a persistent change in the nuclear volume of the cerebral neurons in this area. There was no loss of neurons in the cortex. The changes described are diffuse in the cerebral cortex and not restricted to particular architectonic layers.

Key words: Electro-Convulsions — Cerebral Cortex — Quantitative Cytoarchitectonics.

Introduction

One of the methods used in the study of epilepsy in the animal model is the electroconvulsive method. Roelofs (1967) has stated that electrical current has a great advantage over other methods since all stimulations are comparable within a series. In particular, biochemical research on the influence of electrical stimulation of the brain in short term (Orrego and Lipman, 1967; King *et al.*, 1967) and long term (Pryon and Otis, 1969; Dunn and Giuditta, 1971) studies suggests that in the mouse brain glucose metabolism is disturbed for a considerable period following electroconvulsive treatment.

Inhibition of protein synthesis (Cotman *et al.*, 1971; Sellinger *et al.*, 1972) and a long lasting change in cerebral ribosomes (Wasterlain, 1973) have been described. However, biochemical changes such as the inhibition of brain protein synthesis are only induced if the electroshock (E.S.) levels are above those necessary for eliciting brain seizures (Cotman *et al.*, 1971).

In most cases of epilepsy no loss of cortical nerve cells more severe than that concealed behind the marginal gliosis is found. Cortical lesions are either focal or, more characteristically, affect the third and, less frequently, the second layer in a laminar fashion. The cortex in the deeper parts of the sulci is probably particularly vulnerable. Apparently all lobes may be affected, but in a few cases the temporal lobe has been described as being particularly severely involved (Meyer *et al.*, 1955).

Wasterlain (1973) claimed that rats convulsed electrically between 9 and 18 days of life underwent a reduction in brain proteins and brain RNA, suggesting a reduction in cell size without change in cell number. The largely postmitotic brain of animals shocked later in life (19–28 days) showed no change in the cell number in the brain nor in cell size. It was the aim of this study to extend the above observations by analysis of the effects of electro-convulsions on the cerebral cortex using a quantitative histological method.

Material and Methods

The analysis was carried out in preparations from 6 months old Wistar rats of 200 g obtained from the Central Animal Laboratory of the University of Nijmegen. Two groups of animals were chosen at random. Each animal was placed in an individual cage. After weeks acclimatization the experiment was begun and continued for 3 months; at which time rats were chosen at random from each group and decapitated for histological examination.

The rats of the first group received E.S., the second group served as the control group. E.S. was performed with an Elter E.S. instrument. The two electrodes were attached to the ears of the animals and 50 V were administered for 3 sec. All animals reacted to this dose of electrical activity with a tonic phase of about 30 sec followed by a 60 sec clonic phase. The E.S. treatment was carried out 3 times a week for 4 weeks.

Apart from the E.S. both groups of animals were treated identically. Two months after the last electric shock the animals were sacrificed. The weight of the animals in the control and E.S. groups showed no statistical differences at the beginning or at the end of the experiment ($P > 0.10$). The behaviour of the animals was frequently observed at the end of the trial and no differences were seen.

For histology the Golgi-Cox staining technique (modified according to Van Der Loos, 1959) was used, with cresyl-violet as counter stain. Sections were cut perpendicular to the surface of the cortex to be analysed, and perpendicular to the corpus callosum. Sections of alternating thickness were cut (80 μ and 40 μ). In the 80 μ sections a qualitative and in the 40 μ sections a quantitative analysis was done. The analysis was performed in the first section, from frontal to occipital, in which the corpus callosum was seen. In this way it was always possible to locate the same area with relative ease. The Zeiss Standard GLF, special purpose, microscope (Colon, 1972), made it possible to locate the area in a standardized manner. Going from the uppermost part of the corpus callosum in the slice in an horizontal direction towards the left cortex, the cortical surface is intersected at a specific point. From this point of intersection a column of $40 \times 40 \mu^2$ from pia to white matter was measured, perpendicular to the surface of the cortex. Each neuron located for more than 50% in the column was analysed. For each neuron the depth below the pia was measured, and with the use of a planapochromat 100/1.30 immersion oil objective and a 12.5 wide angle ocular with intraocular device, the length (l) and width (w) of the nucleus was also determined. The nucleus was considered as an ellipsoid of revolution and its volume calculated as $1/6 \pi \cdot h \cdot w^2$. Variations in thickness of the cortex between individual animals makes a standardization procedure for the depth measurements necessary. Layer I can be readily distinguished from the rest of the cortex and contains practically no neurons. Therefore, the standardization refers to the distance from the margin gray/white matter to the margin between layers I and II. This distance was divided into 2000 arbitrary units. A division into logarithmically equidistant depth classes was made in order to describe the results (Table 1).

For each depth class the means of the volumes (with their standard error) and the number of neurons was calculated. The IBM-370 of the University Calculation Centre Nijmegen was employed, using the PL-1 language.

Table 1. Depth levels of the different depth classes expressed in units below layer I

Depth class	Depth class	Depth class	Depth class
1	0—49	8	556—694
2	49—105	9	694—854
3	105—170	10	854—1041
4	170—246	11	1041—1258
5	246—334	12	1258—1510
6	334—437	13	1510—1811
7	437—556	14	1811—2142

Table 2. Mean and standard errors of the nuclear volumes, and the number of neurons, in the different depth classes in the electroconvulsive and control groups

Depth class	Control			E.S.		
	mean	se	n	mean	se	n
1	5.27	0.17	7	5.53	0.10	7
2	5.47	0.24	9	5.43	0.24	9
3	5.07	0.14	13	5.22	0.17	8
4	5.84	0.14	13	5.41	0.09	12
5	5.09	0.12	13	5.82	0.19	9
6	5.88	0.14	15	5.48	0.06	15
7	5.82	0.12	10	5.48	0.11	19
8	5.84	0.09	24	5.55	0.09	24
9	5.48	0.08	29	5.78	0.10	26
10	6.00	0.08	28	5.82	0.11	24
11	5.98	0.08	31	5.77	0.08	30
12	5.86	0.06	45	5.61	0.06	42
13	5.67	0.05	50	5.43	0.06	49
14	5.49	0.09	27	5.49	0.07	31
Overall mean	5.75	0.03	320	5.58	0.03	305

Results

No differences between the cortex of the treated and control group could be found with the qualitative Golgi-Cox and cresyl-violet preparations.

The results of the quantitative analysis are presented in Table 2. The mean nuclear volume, with standard error of the mean, and the numbers of neurons in the different depth classes of both groups is also given in Figs. 1 and 2. With the Student-t-test there is a significant difference ($P < 0.01$) between the overall means of both groups regarding nuclear volume. Using Friedman's m-rankings test, the nuclear volumes of the two groups differed ($P < 0.05$) in the different depth classes, however this test did not reveal any differences in the numbers of neurons in the different depth classes.

Discussion

This study contains an analysis of qualitative and quantitative parameters in a small part of the cerebral cortex of rats 2 months after a series of 12 electroshocks. The results indicate a change in nuclear volume in the cerebral cortex neurons. This change is diffuse throughout the whole cortex in the area of research. The number of neurons did not change. A qualitative analysis of the dendritic structure in this cortex did not give any indication of probable alterations. No differences in the treatment of the animals during the experiment or in their behaviour at the end of the experimental period could be observed. These results

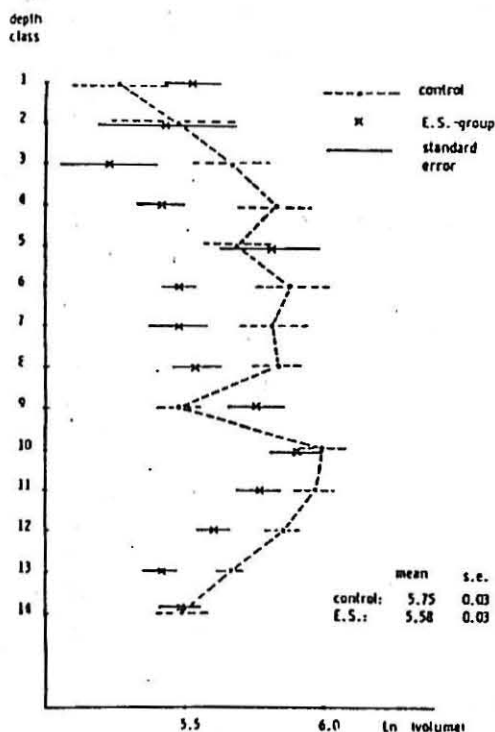


Fig. 1

Fig. 1. Mean and standard error of the nuclear volume in different cortical depth classes in electroconvulsive and control groups

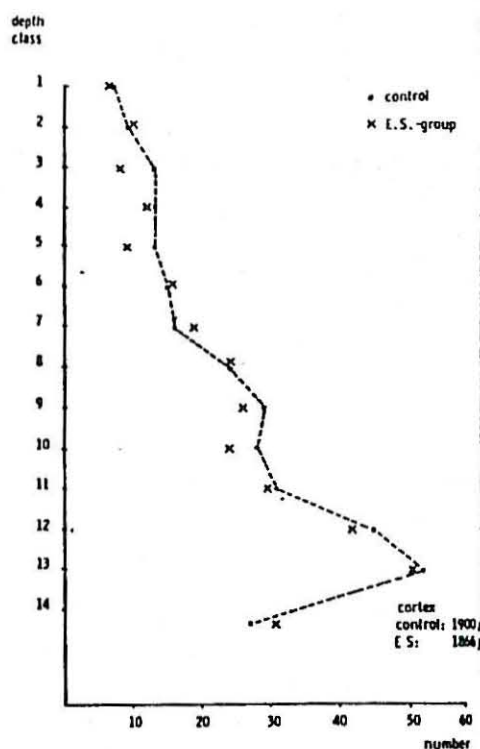


Fig. 2

Fig. 2. The number of neurons in different depth classes ($3 \times 40 \times 40 \mu^2$ below the cortical surface), for electroconvulsive and control groups

indicate a persistent change in the cerebral cortex after convulsive treatment at least in one area: which might be the result of the fact that rats are probably rather resistant toward hypoxia. It is interesting to find that no change in neuronal numbers was found. The most essential changes in the cerebral cortex of some presenile dementias consist essentially of neuron loss in quantities of more than 50% (Colon, 1972b), in contrast to normal ageing in the human where this loss is always less than 50% (Colon, 1972c). Probably neuron loss is not the only factor in deterioration processes as might also be expected in epilepsy. The long lasting change in biochemical properties in convulsive animals, as described in the introduction, seems to be reflected in a persistent decrease in neuronal volume. In contrast to Wasterlain (1973) who suggested that changes in nuclear volume only occurred in mitotic brain, the postmitotic brain also achieves, when shocked at least 12 times, a persistent level of change. This constitutes a serious warning

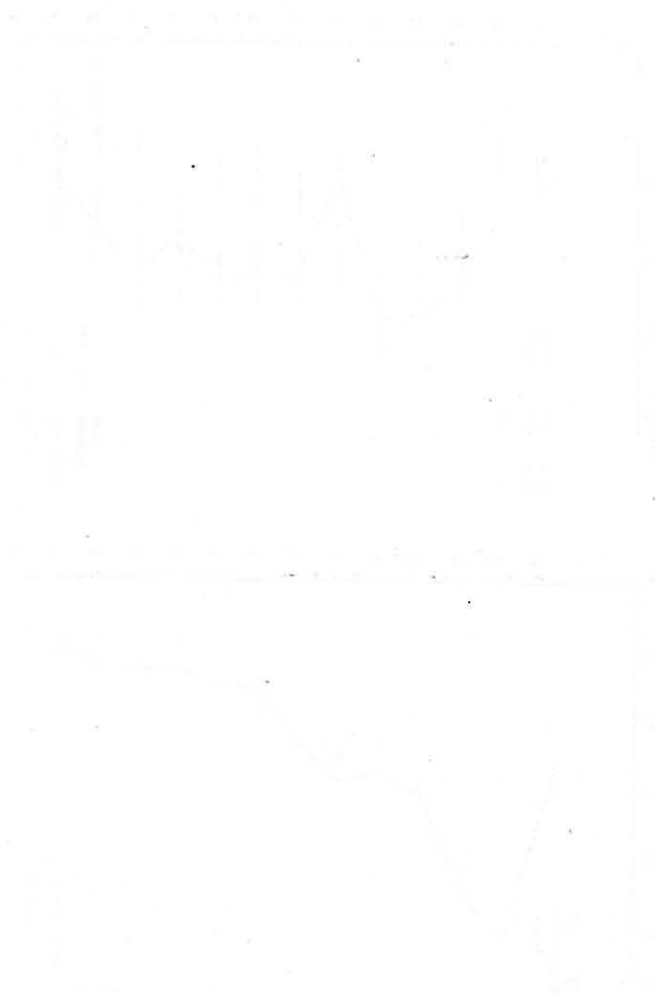
against the use of electroconvulsive therapy and a serious indication for the suppression of epileptic manifestations.

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Electroconvulsive therapy for the depressed elderly
[Review]

Van der Wurff, FB; Stek, ML; Hoogendijk, WL; Beekman, ATF

Date of Most Recent Update: 27-February-2003

Date of Most Recent Substantive Update: 18-February-2003

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Outline

Abstract

Issue protocol first published

Date of last minor update

Issue next stage

Issue review first published

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Types of participants

Types of intervention

Types of outcome measures

Types of studies

Search strategy for identification of studies

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Description of the studies

11/5/03

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 Results
 Discussion
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Implications for practice
 Implications for research

Internal sources of support to the review
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Graphics

Hamilton Depression ...
 Hamilton Depression ...

Abstract

Background: Depressive disorder is a common mental disorder in old age, with serious health consequences such as increased morbidity, disability, and mortality. The frailty of elderly may seriously hamper the efficacy and safety of pharmacotherapy in depressed elderly. Electroconvulsive therapy (ECT) in depressed elderly therefore may be an alternative to treatment with antidepressants.

Objectives: To assess the efficacy and safety of ECT (compared to simulated ECT or antidepressants) in depressed elderly.

Search strategy: We searched the CCDANCTR database, Medline 1966-2000, EMBase 1980-2000, Biological abstracts 1985-2000, Cinahl 1982-2000, Lilacs from 1982 onwards, Psyclit 1887-2000, Sigle 1980-2000. The reference lists of relevant papers were scanned for published reports. Hand searching of the Journal of ECT and the Journal of Geriatric Psychiatry was done. Based on the title of the publication and its abstract, non-eligible citations were excluded.

Selection criteria: Data were independently extracted by at least two reviewers. Randomised, controlled trials on depressed elderly (> 60 years) with or without concomitant with conditions like cerebrovascular disease, dementia of the Alzheimer's type, vascular dementia or Parkinson's disease were included.

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Data collection and analysis: Data were independently extracted by at least two reviewers. For continuous data weighted mean differences (WMD) between groups were calculated.

Main results: Randomised evidence is sparse. Only three trials could be included, one on the efficacy of real ECT versus simulated ECT (O'Leary et al 1994), one on the efficacy of unilateral versus bilateral ECT (Fraser 1980) and the other comparing the efficacy of ECT once a week with ECT three times weekly (Kellner 1992). All had major methodological shortcomings; data were mostly lacking essential information to perform a quantitative analysis. Although the O'Leary study concluded that real ECT was superior over simulated ECT, these conclusions need to be interpreted cautiously. Only results from the second trial (unilateral versus bilateral ECT) could be analysed, not convincingly showing efficacy of unilateral ECT over bilateral ECT, WMD 6.06 (CI -5.20,17.32). Randomised evidence on the efficacy and safety of ECT in depressed elderly with concomitant dementia, cerebrovascular disorders or Parkinson's disease is completely lacking. Possible side-effects could not be adequately examined because the lack of randomised evidence and the methodological shortcomings.

Conclusions: None of the objectives of this review could be adequately tested because of the lack of firm, randomised evidence. Given the specific problems in the treatment of depressed elderly, it is of importance to conduct a well designed randomised controlled trial in which the efficacy of ECT is compared to one or more antidepressants.

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Background

The prevalence of depressive disorders in older people in the community lies around 12.5%. This includes major, minor depressive disorder, and dysthymic disorder (a chronic form of depression). The prevalence of major depressive disorder in people over the age of sixty is estimated around 2% (Beekman 1999). The prevalence of depression in people suffering from dementia, Parkinson's

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disease and cerebrovascular accidents is higher. The burden of depression both to the individual and to society is huge. Depression in the elderly is accompanied by a high (cardiovascular) mortality (Frasure-Smith 1993; Penninx 1999) and a negative effect on well-being and daily functioning (Ormel 1999).

Depression in late life is thought to differ from depression in younger subjects in etiology, presentation, treatment and outcome. Although social, psychological, physical and biological factors interact, depression in the elderly is only partly explained by risk factors like physical health, life-events, social support or personality (Beekman 2000). Biological factors may play an important role in the etiology of late-life depression, and this may be of particular significance in some subgroups of the depressed elderly. It is not clear how these factors interact, but hyperactivity of the hypothalamic-pituitary-adrenocortical (HPA) system and an increased production of immune-mediated cytokines like interleukin-6 (IL-6), may be major biological risk factors in the pathogenesis of depression (Holsboer 1995; Maes 1995; Song 1998). Old age is accompanied by a hyperactivity of the HPA-system independent of the occurrence of depression (Deuschle 1997). Psychosocial stress-factors induce an even stronger HPA-system hyperactivity (Holsboer 1996). The high prevalence of depression in the elderly may thus be explained by the effect of psychosocial stress factors on the HPA-system, which finally lead to depression. Cerebrovascular disorders also are a risk factor of major importance in the pathogenesis of late life depression (Alexopoulos 1997). Lacunar and cortical infarcts occur regularly in elderly with depression. So-called white matter hyperintensities appear often on MRI-scans of depressive elderly (Alexopoulos 1997). These white matter hyperintensities may have a vascular pathogenesis (Pantoni 1995, Pantoni 1997). Depression in older people with concomitant cerebrovascular disorders is therefore sometimes called 'vascular depression' (Alexopoulos 1997).

There are two major forms of biological treatment for late-life depression: treatment with antidepressants and electroconvulsive therapy (ECT). Various antidepressants are now being marketed, and the efficacy these was the topic of a recent systematic review (Wilson 2001). It is generally acknowledged that older, frail depressed patients are particularly prone to the side effects of antidepressants. Cardio-vascular side effects of antidepressants occur more often in older than younger patients (Woodhouse 1992). Many of these drugs also have serious anticholinergic side-effects (Moskowitz 1986), that may seriously affect compliance to treatment. Although approximately 50-60% of patients are thought to improve clinically as a consequence of antidepressant treatment (Schneider 1995), in subgroups of the depressed elderly, the efficacy of antidepressants may be lower. For example, white matter hyperintensities and lacunar infarcts on MRI-scan are thought to markedly affect the outcome of treatment with antidepressants (Simpson 1997, Simpson 1998).

ECT involves the application of an electric current to the head with the aim of inducing a controlled tonic clonic convulsion, and is usually carried out at intervals of days. Although the efficacy of ECT has been established in a considerable number of studies, it is still a controversial treatment. The use of ECT is subject to legal restriction in parts of the world. Some reports suggest that ECT is particularly effective in late-life depression (Flint 1998), and that it is effective in therapy resistant depressive elderly people with

extensive white matter hyperintensities (Coffey 1988). Currently there is no evidence to suggest that ECT causes any kind of brain damage, although temporary cognitive impairment is frequently reported (Devanand 1994; Scott 1995). ECT seems to be a safe procedure, even in elderly with cardiovascular disorders (Rice 1994). ECT is used to treat the depressed elderly more frequently, and its use is declining less rapidly than in the general population (Glen 1999).

ECT may be safer and more effective than antidepressants in the treatment of late life depression. This greater efficacy may be more pronounced in subgroups of the depressed elderly that suffer from co-morbid cerebrovascular disorders, dementia and Parkinson's disease.

Objectives

To perform a systematic review and meta-analysis on the evidence on the efficacy of ECT in late life depression, and to assess the methodological quality and generalizability of the trials in this area.

The primary objectives of this review were:

To test the hypothesis that modified ECT has a greater and/or more rapid antidepressant effect than simulated (sham) ECT, antidepressant drug treatment or non-pharmacological interventions in the early phase (the first six weeks) and longer term (six months post treatment) treatment of late life depression.

The secondary objectives of this review were:

1. To determine whether ECT produces a differential response in depressed, elderly patients with concomitant conditions including:

a. Evidence of cerebrovascular disorders (cortical and lacunar infarcts, and white matter hyperintensities);

b. Dementia of the Alzheimer's type or Vascular dementia;

c. Parkinson's disease

2. To determine the effect of electrode placement (unilateral versus bilateral forms of ECT) and dosage (both the amount of energy supplied and the frequency of ECT-application) on the efficacy of ECT in late life depression

3. To examine the immediate and long-term side effects, and in particular, the cognitive side effects of ECT in the depressed elderly.

Criteria for considering studies for this review

Types of participants

Randomised and non-randomised (observational studies, retrospective studies and case series) evidence was obtained. The review and meta-analysis included randomised, controlled trials only. At present no validated methodology is

available for summarizing non-randomised evidence. The results of non-randomised studies were therefore not summarised in this review. They will be submitted for publication in a narrative review to the International Journal of Geriatric Psychiatry.

Types of intervention

The intervention of interest was unilateral or bilateral ECT, that is, the electrical induction of cerebral seizure activity after the intravenous induction of a brief general anaesthesia and the pre-administration of a skeletal muscle relaxant drug. Trials that evaluated the acute phase treatment of depression were included. Trials that examined the effectiveness of maintenance ECT were excluded.

For the primary objectives trials were included that compared the application of ECT with:

1. "sham-ECT" or "simulate-ECT";
2. treatment with antidepressants;
3. non-pharmacological forms of treatment;
4. any other form of placebo treatment.

For the secondary objectives trials were included that compared the application of:

1. ECT applied in groups of depressed elderly with or without concomitant conditions including Parkinson's disease, dementia of the Alzheimer's type, vascular or multi-infarct dementia and cerebrovascular disorders;
2. Unilateral versus bilateral ECT;
3. ECT applied at different energy levels;
4. ECT applied at different frequencies, for example once or twice weekly.

Types of outcome measures

The primary outcome measure to assess antidepressant efficacy was the reduction in symptoms of depressive illness measured as changes in scores from baseline. It was anticipated that reduction in symptoms would be measured on:

1. continuous symptom scales like the Hamilton Rating Scale for Depression (HRDS), the Montgomery Asberg Depression Rating Scale (MADRS), the Clinical Global Inventory (CGI) which were then analysed as a continuous variable, or
2. clinical global outcome measures such as "recovered", "much improved" or "not improved". For trials in which categorical outcome data were available, these were summarised as the number of people who experienced those outcomes in each

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comparison group and the total number in each group, and were then analysed as a dichotomous variable.

Secondary outcome measures to assess efficacy were if possible determined by:

1. the number of patients in each group who showed significant clinical improvement in the long term (six months post treatment), either measured as a decline in symptoms on continuous scales like the HRDS, MADRS, CGI or dichotomous variables like "recovered", "much improved" or "not-improved";
2. the number of drop-outs;
3. cause specific and non-specific mortality;
4. suicide;
5. functional outcomes like quality of life and cognitive functioning.

The main outcome measures for assessing side effects in controlled trials were:

1. cause specific mortality;
2. Severe somatic complications like myocardial and cerebral disorders;
3. Immediate and long-term cognitive disorders like disorders in attention, orientation and memory measured on the Mini Mental State Examination (MMSE) or any other neuropsychological instruments.

Types of studies

Randomised and non-randomised (observational studies, retrospective studies and case series) evidence was obtained. The review and meta-analysis included randomised, controlled trials only. At present no validated methodology is available for summarizing non-randomised evidence. The results of non-randomised studies were therefore not summarised in this review. They will be submitted for publication in a narrative review to the International Journal of Geriatric Psychiatry.

Search strategy for identification of studies

The search was performed in collaboration with the John Geddes and others, who were kind as enough to share their search results with us. The Geddes' group have conducted a review on the safety and efficacy of ECT for depressive illness, but not specifically in the elderly. Therefore a two-stage search process was conducted for our review.

A. Firstly, we used the search results of Geddes et al. The search strategy was as follows:

1. The Cochrane Controlled Trials Register and the Cochrane Collaboration Depression Anxiety and Neurosis Controlled Trials Register was searched using

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"electroconvuls*" or "electro-convuls*" or "electroshock*" or "electro-shock*" or "convuls*" or "ECT" as search terms.

2. In addition a number of other electronic databases were searched:

The following databases were searched in using the CCDAN-search strategy

Biological abstracts 1985-2000

Cinahl 1982-2000

EMBase 1980-2000

Lilacs from 1982 onwards

Medline 1966-2000

PsycLIT 1887-2000

Signle 1980-2000

2. Hand searching of the reference lists of the handbooks on ECT of Abrams and Fink and the latest APA Guidelines were performed.

3. Hand searching of the Journal of ECT/Convulsive Therapy was done. More than 3000 references resulted from this process, of which John Geddes' group selected just under 600 studies initially satisfying their inclusion criteria. These references were held on Reference Manager Version 9 and this database was shared with us. FvdW searched this database for references in which elderly or aged were used as keywords or appeared in the abstract.

B. Due to the difficulty in locating trials in older people, additional electronic and hand searching was done by the first author (FvdW). In this additional searching, the target was to locate randomised and non-randomised (observational studies, retrospective studies, case series, case reports) evidence on the field of ECT in depressed elderly.

1. The CCDAN and CCTR was searched using "electroconvuls*" or "electro-convuls*" or "electroshock*" or "electro-shock*" or "convuls*" or "ECT" and senil* or geriatr* or older or elder* or late-life or later-life or "late* life" as search terms.

2. The following databases were additionally searched using the CCDAN Trials Register search strategy in combination with search terms on elderly, dementia, cerebrovascular disorders and Parkinson's disease, case reports, case series, retrospective studies:

Biological abstracts 1985-2000

Cinahl 1982-2000

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EMBase 1980-2000

Lilacs from 1982 onwards

Medline 1966-2000

Psyclit 1887-2000

Signle 1980-2000

2. Hand searching of the chapter on "Electroconvulsive Therapy in late-life depression" by Harold Sackeim in the handbook on Clinical Geriatric Psychopharmacology by Carl Salzman was carried out.

3. Hand searching of the Journal of Geriatric Psychiatry was done.

4. Additional hand searching of references cited in all included and excluded trials was carried out to identify any missing studies on the basis of the title.

5. Two experts in the field, H. Sackeim and C.H. Kellner were contacted to ask whether they were aware of any additional eligible studies.

Through this process, more than four hundred citations potentially fulfilling the inclusion criteria for the review were selected. The title, abstracts or full copy of these references were assessed independently by the first two reviewers for inclusion into the review. Following assessment of the references, 160 references that included ECT and depression (with or without dementia, cerebrovascular disorders or Parkinson's disease) in the elderly remained. Of these 160 references, the abstracts or full copy were assessed independently by FvdW and MS. A inter-rater agreement (kappa) of 0.96 was obtained.

Methods of the review

Selection of studies: The first reviewer identified the studies. The first two reviewers independently assessed the relevance of each trial, blind to the decisions made by one-another. Each trial was assessed against pre-set criteria. In cases of disagreement, decisions were reached by consensus through open discussion. Based on the title of the publication and its abstract, and if necessary full copy of the article, irrelevant citations were excluded. Potential studies for inclusion (randomised and non-randomised evidence) were obtained in hard copy. Reasons for exclusion or inclusion were recorded. Reviewers were not blinded for authorship of trials, journals and institutions from which articles came. The four reviewers judged the relevance of the included trials independently. FvdW has a special interest in the topic of depression in the elderly with concomitant cerebrovascular disorders or dementia. The second author (MS) has a special interest in ECT in late life depression. AB and WH are acknowledged experts in the field of late-life depression. The latter two reviewers were not involved in the search and identification phase. They were active in the selection process of included trials and in the editorial phase of the review.

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Data collectionData were extracted from studies using a preset form. Data were entered into RevMan 4.1.1. Where data were missing, the principal investigator of the study was contacted for more information.

Methodological qualities of included studies:Assessment of the methodological quality of each trial was carried out according to the guidelines in the Cochrane Reviewers' Handbook (edition 1999). Concealment of allocation was the main quality criterion for included studies. The adequacy of allocation concealment was judged as adequate (A), unclear (B) or inadequate (C) (for example an open list of random numbers, the use of alphabet or data of birth) or not used (D). Other major aspects on which the methodological quality were assessed, included the blinding of participants and assessors; the adequacy of outcome assessment (intention to treat analysis (ITT), and the adequacy of follow-up assessments. Only trials in category A or B were included in the review.

Data and statistical analysis:We hoped we would be able to find enough studies to be able to analyse data as described in Chapter 8 of the Reviewers' Handbook. Categorical data would be transformed into dichotomous outcomes as described earlier. Relative Risks, Odds ratios and 95% confidence intervals would be calculated for the individual studies and pooled using the fixed effect model of Peto. The random effects method would then be used as a test for the robustness of the findings from the fixed effect analysis. We hoped to present data as 'the Number Needed to Treat' (NNT). In case continuous data were normally distributed, they would be analysed by calculating the weighted mean difference or the standardised mean difference depending on whether the same or different scales had been used for measuring outcome.

Sensitivity analysis:When possible, we wished to investigate the effect of including or excluding studies with methodological shortcomings. We hypothesised that clinical factors, such as the presence of co morbid cerebrovascular disorders would influence the outcome of trials. Therefore, our intention was to perform subgroup-analyses to investigate whether there were differences in response between:

- a. Depressed elderly with or without cerebrovascular disorders (cortical and lacunar infarcts, and white matter hyperintensities);
- b. Depressed elderly with or without Dementia of the Alzheimer's type or vascular dementia;
- c. Depressed elderly with or without Parkinson's disease

Assessment of heterogeneity:If a meta-analysis was possible, the random effects model would be used as a test of the robustness of the findings from the fixed effect analysis.

Description of the studies

For substantive descriptions of studies please see "Included and Excluded

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studies"

General remarks

Randomised evidence on ECT in elderly was sparse. We were only able to select three trials that studied one of the primary or secondary objectives of this review. Only one controlled trial was found that investigated one of the primary objectives of this review, that is, the efficacy of real-ECT to simulated ECT (O'Leary 1994). No controlled trials could be found that examined any of the other primary objectives of this review, for example the efficacy of ECT to antidepressants. Two RCTs were located that investigated one of the secondary objectives of the review, one of which compared the efficacy of unilateral versus bilateral ECT (Fraser 1980) and the other comparing the efficacy of ECT once a week with ECT three times weekly (Kellner 1992). Randomised evidence on the efficacy and safety of ECT in depressed elderly with concomitant dementia, cerebrovascular disorders or Parkinson's disease is lacking completely. One RCT that may meet the inclusion criteria for the review is waiting further assessment (Krystal 2000). It reports on the predictive power of ictal EEG-changes on outcome. Contact has been made with the principal investigator to determine whether data on the outcome of depression in the participants are available. To date no additional information has been received.

Although randomised evidence is sparse and the three included trials had several methodological problems, we decided to present the reader an overview of the different methodological aspects of the studies. In one comparison a quantitative analysis on the data was performed (the effectiveness of unilateral versus bilateral ECT). In case this quantitative analysis was not possible, the major results were summarized as reported by the investigators for each study.

All other identified studies consisted of non-randomised evidence, including observational studies, retrospective studies, case reports and case series. As stated earlier, they will be summarized in a narrative review.

1. Excluded studies One hundred fifty seven possible studies were excluded. One study was an open trial without a control condition. Thirty-three were prospective, mostly naturalistic follow-up studies, but without randomisation or control conditions. Thirty-five were excluded because they were retrospective studies and forty-eight because they concerned case-reports. Another forty studies were excluded, either because they were reviews or because they did not fulfil inclusion criteria (for example they examined the effectiveness of maintenance ECT, or the effectiveness of ECT on the motor symptoms of Parkinson's disease without concomitant depressive disorder, or considered epidemiological aspects of ECT in elderly).

2. Included studies For the primary objective, only one RCT meeting inclusion criteria was found (O'Leary 1994). Some remarks need to be made concerning this study. This study was a post-hoc analysis of data in elderly people who participated in the Nottingham trial on ECT (Gregory 1985). The Nottingham trial is a RCT in which the efficacy of ECT (unilateral and bilateral) was compared with simulated ECT in 69 patients, but not specifically the elderly. The presentation of the data in the O'Leary study was sparse, mean scores in the

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treatment conditions and/or standard deviations for the different comparisons the investigators performed were not available. Therefore we were not able to calculate a standardised mean difference, necessary to perform a quantitative analyses. We contacted the principal investigator in an attempt to obtain the relevant data to be able to perform a quantitative analysis. Because of the post-hoc nature of the report and all the other methodological limitations, which we will describe, the results of this study need to be interpreted with caution. But because of the lack of randomised evidence for the primary objective of this review, we decided to include this study in the review and to provide the reader with its major results. There are a large number of non-randomised studies on the primary and secondary objectives of our review, but as stated before no validated system exists to summarize them and therefore they cannot be included in this review.

For the secondary objectives two RCTs meeting inclusion criteria were found. Fraser 1980 compared the efficacy of unilateral versus bilateral ECT. Kellner 1992 performed a randomised trial on the dosage of ECT comparing the efficacy of ECT weekly with ECT three times weekly. In only one of these studies data could be extracted for inclusion (Fraser 1980). Data in the study by Kellner 1992 were sparse. Means and standard deviations were not reported. We contacted the principal investigator, who informed us that the necessary information to perform a quantitative analysis was not available. Again, because of the lack of randomised evidence, we decided to include both of these trials in the review, but of course the results of this study need to be interpreted with caution because of the methodological problems that we will describe hereafter.

a. Length of included trials The Nottingham trial, from which O'Leary 1994 re-analysed the data, took place between August 1981 and February 1983. More than ten years later O'Leary 1994 performed their analysis on a subgroup of elderly participants. In the Nottingham trial participants were followed up to 6 months. Fraser 1980 and Kellner 1992 did not specify the length of the trial and no relevant follow-up took place.

b. Participants of included trials

All of the studies included participants of 60 years and older. Both sexes were included.

In the Nottingham trial a standardised psychiatric history was taken and all patients were assessed using the MRC and Feighner criteria for major depressive illness and the Present State Examination (PSE). Fraser 1980 operationalised diagnosis according to the Feighner criteria (1972). Kellner 1992 used DSM-III criteria to diagnose depressive illness.

None of the studies used a structured clinical interview to establish diagnosis.

The participants in the Nottingham trial were for the most part referred for ECT-treatment. Fraser 1980 provides no information on preliminary treatment of the participants. The participants in the study by Kellner 1992 were referred for ECT.

More specific information on earlier treatment with antidepressants prior to the episode in which the patients participated in the studies, was missing in all the included studies. In addition, no information is provided on the treatment of possible earlier depressive episodes of the participants. Information on the mean number of admissions is also lacking in all the studies.

As adequate information on somatic and psychiatric co-morbidity and history is important in the treatment of depressed elderly, the lack of data about physical disease and psychiatric co morbidity is a serious limitation. Only Fraser 1980 mention that "mild or moderate senile dementia did not by itself exclude a patient from ECT".

c. Setting of included trials Participants in the studies were inpatients as well as outpatients.

d. Study size of included trials

O'Leary 1994 analysed data on thirty-five patients aged 60 and over from the Nottingham trial (Gregory 1985), in which sixty-nine patients originally participated. Twelve of the elderly participants were withdrawn from the Nottingham trial before completing the six study treatments. Three of these non-completers were withdrawn because they improved before the six study treatments were finished, eight of them because they failed to improve, and one because of physical illness. A separate analysis on the completers and non-completers was performed, but not an intention-to-treat analysis. Some serious doubt is necessary therefore on the value of the results they describe. In the Nottingham trial seven elderly patients completed treatment with simulated ECT, eight patients completed treatment with unilateral and eight with bilateral ECT.

Fraser 1980 initially included thirty-three patients. Four of them dropped out of the study, two of them died, two left treatment "against advice". The authors did not include these drop-outs in their analyses. Kellner 1992 only included fifteen patients. It seems that all of them completed treatment.

e. Interventions of included trials

O'Leary 1994 (based on re-examination of data on elderly in the Nottingham trial) randomised between simulated ECT, unilateral or bilateral ECT. Treatment was twice weekly. Six study treatments were given. An Ectron Duopulse Mark IV machine, waveform 1, was used, delivering energy in units of Joules rather than charge, which made it impossible to quantify the amount of electrical stimulation administered. Bilateral treatment was applied using the bitemporal position and unilateral ECT was administered using the Lancaster position, applied to the right temporo-parietal position (Lancaster 1958). Anaesthesia consisted of methohexitone 70 mg and suxamethonium bromide 50 mg. Patients in the simulated group received the whole ECT procedure, but no shock. Adequacy of seizures was determined by means of the cuff method without further details. Low-dose benzodiazepines were allowed during study.

Fraser 1980 randomised participants between treatment with unilateral or

bilateral ECT. They used an Elektron Duopuls mark 4 machine, delivering 30 to 120 Joules in a unidirectional "chopped" sine waveform, making it impossible to quantify the amount of electrical stimulation administered. Patients were anaesthetised with thiopentone 150-250 mg, and the muscle relaxant was suxamethonium 25-40 mg, doses depending on body weight. The electrode placement was according to the method by Stromgren (1973). No information is provided on the adequacy of seizure induction and the parameters by which this was established. All psychotropic medication was stopped 24 hours before treatment. The number of treatments ranged from 4-11, twice weekly.

Kellner 1992 randomised between two treatment groups. Group 1 underwent ECT once a week for 3 weeks. Group 2 underwent three-times-weekly ECT for 3 weeks. Slow-responders in group 1 were switched to three-times weekly treatment after treatment 3. Slow-responders in group 2 after nine treatments were given ongoing three-times-weekly ECT until clinically well. Bilateral electrode placement was used for all participants. A MECTA SR2 ECT device was used, delivering a brief-pulse, square-wave stimulus. Anaesthesia consisted of methohexital (1 mg/kg) and succinylcholine (0.75 mg/kg). Dose titration was not performed. Stimulus dosing was adjusted to produce a motor seizure >20 s. Motor seizures lasting

f. Outcomes In all studies improvement was measured on internationally validated instruments like the Hamilton Depression Rating scale (HRDS or HAM-D) and the Montgomery Asberg Depression Rating Scale (MADRS). They were all continuous outcome measures. None of the studies used pre-defined criteria for responders or non-responders, and therefore no dichotomous data can be generated.

Primary outcome measures in the Nottingham trial (O'Leary 1994) were scores on continuous outcome scales, the Montgomery & Asberg Depression Rating Scale (MADRS) and the Hamilton Depression Rating Scale (HRDS). Study patients were scored after every two treatments and within two days of the last ECT. Raters were blind for the treatment group. No information is provided on how this blinding was achieved.

In Fraser 1980 scores on the Hamilton Rating Scale for Depression (HAM-D) was the primary outcome. It was measured before treatment, within 24 hours after the fifth treatment and 3 weeks after the last. Fraser 1980 also made comparisons between good outcome and moderate outcome patients. Information on this point is so scarce, that the reviewers have serious doubts on the interpretation of these data. A rater who was blind to the patient's treatment condition measured the depressive symptoms. No information was provided on how this blinding was achieved.

Kellner 1992 measured outcome by means of the Hamilton Rating Scale for Depression (HAM-D), the Beck Depression Inventory (BDI), the Clinical Global Improvement Scale (CGI) and the Brief Psychiatric Rating Scale (BPRS), all of them being continuous outcome scales. A researcher who was blind to the patient's treatment condition rated the depression scales. No information was provided on how this blinding was achieved.

In the Nottingham trial no information on cognitive or other side effects was

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collected.

Fraser 1980 examined cognitive side effects by applying the Wechsler memory scale before treatment, not less than 24 hours after the fifth treatment and 3 weeks after the last treatment. The principal investigator also administered a "simple ad hoc questionnaire" at the moment the second Hamilton test was applied, intended to score potential side-effects such as headache and nausea.

Kellner 1992 examined cognitive side effects by applying the Mini-Mental Status Examination (MMSE) at baseline, weekly on non-ECT days, and 1 week after the last ECT. Thereby, the Wechsler memory Scale-Revised (WMS-R) was administered at baseline and 1 week after the last ECT treatment.

None of the studies provided information on any of the other outcome measures as described in the methodology section of this review.

Missing outcomes

All trials presented findings in graphs or p-values only. They did not provide standard deviations and/or means. Only in Fraser 1980 was it possible to calculate this information from the data provided in the report. Because none of the trials used pre-defined criteria for responders or non-responders, it was impossible to obtain or calculate any dichotomous outcome measures on this point. Information on the efficacy and possible side-effects of ECT on the medium and long term was sparse or lacking completely. In the Nottingham trial data on cognitive side-effects were not collected. None of the trials provided information on cause specific and non-specific morbidity/mortality or suicide rates in the short and longer term. Data on physical co morbidity and mortality/suicide rates were not collected in any of the trials. No trial reported on economic or functional outcomes.

Methodological qualities of included studies

See: Table of included studies

Randomisation

No studies achieved a quality rating of "A" for their descriptions of methods used to randomise. It was not clear exactly how randomisation had occurred in any of the studies, resulting in all of them receiving a rating of "B" (methods used unclear). The information provided in the studies did not rule out that bias could have been introduced.

Blinding to interventions and outcomes

No study clearly described their procedures for blinding of assessors. They all described that assessors were blinded for the procedure under study, but adequate information to determine this with certainty was missing.

Follow-up

In the Nottingham trial participants were followed up until six months after the end of treatment with ECT. O'Leary 1994 mentions that "at 1, 3 and 6 months after the study there were no significant differences in the change in HDRS or MADRS score between the three treatment types". No analysable data were provided on this point, and the statistical procedures on which these conclusions were based were not clear. The number of drop-outs was not reported, neither was it clear how the significant differences had been calculated. Fraser 1980 and Kellner 1992 make no mention of follow-up data. No study analysed their data on an intention-to-treat basis. O'Leary 1994 performed a separate analysis on completers and non-completers.

Because of the lack of information on the adequacy of randomisation, the adequacy of blinding of outcome assessors and all the other limitations, interpretation of the results of the included studies is difficult and necessitates caution.

Results

List of comparison

No study addressing the primary objective of this review was identified. The study by O'Leary 1994 was the only study to examine one of the primary objectives, but the methodological problems and insufficient data prohibited a comparison. Only one study on one of the secondary objectives was available for inclusion in a comparison (Fraser 1980). The trial by Kellner 1992 could not be included in a comparison because the relevant data were missing and could not be supplied. Other well-designed randomised controlled trials evaluating ECT in the depressed elderly were lacking, especially trials in which the efficacy of ECT were compared with antidepressants, or trials in which the efficacy and safety of ECT in subpopulations of depressed elderly was studied. Therefore it was possible to undertake one comparison only, on the efficacy of unilateral versus bilateral ECT, on the basis of the study by Fraser 1980. A comparison in MetaView could be presented for this outcome (see Comparison 2). We decided to give an overview of the major findings as reported by O'Leary 1994 and Kellner 1992 given the fact that no other randomised evidence was available. We provide these findings in Comparisons 1 and 3, without being able to present them in MetaView. Because of the small numbers of studies that found, heterogeneity and sensitivity analyses could not be performed.

Comparison 1 Real ECT versus simulated ECT

One study compared real ECT with simulated ECT (O'Leary 1994). The authors performed an analysis both on the completers and non-completers. No intention to treat analysis was performed. Post hoc analyses of the completers showed that after six study ECT-treatments the mean MADRS scores of the unilateral and bilateral groups differed significantly from the simulated group ($p < 0.05$). Because the investigators had not supplied means and/or standard deviations, a quantitative analysis could not be made on this outcome. A two-way analysis of variance of treatment group and number of ECT-treatments showed a significant interaction between treatment group and number of ECT-treatments ($p < 0.01$). Again, because means in the treatment and control conditions were not provided,

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the analysis we had intended to undertake (change of baseline scores using a fixed effect model) was not possible. The authors of this trial performed a separate analysis on the non-completers. Among the non-completers, there was a significant improvement in the real treatment group compared to the simulated group as measured by the mean and percentage changes in the HRDS and MADRS ($p = 0.02$). In our opinion interpreting results on non-completers without performing an intention to treat analysis necessitates caution.

Comparison 2 Unilateral versus bilateral ECT in elderly patients

Two trials compared unilateral with bilateral ECT (Fraser 1980 and O'Leary 1994). Only continuous data were available. O'Leary did not provide means and/or standard deviations, therefore the comparison could not be included in the quantitative analysis. The analysis was therefore only based on the data supplied by Fraser 1980. Although the estimate suggested superior efficacy of unilateral ECT over bilateral ECT, Fraser (1980) not convincingly demonstrated efficacy of unilateral ECT over bilateral ECT, both after 5 treatments (WMD 6.06 (CI -5.20, 17.32), and after 3 weeks of treatment (WMD -0.37 (-5.02, 4.28). It should be noted that the sample size was small (13 receiving unilateral ECT and 16 bilateral ECT). Fraser 1980 did not provide standard deviations on the outcomes for the cognitive side effects. These outcomes therefore could not be included into the quantitative analysis.

Comparison 3 Once weekly ECT versus three-times weekly ECT

Kellner 1992 compared once-weekly ECT with three-times-weekly ECT. Scores on the HAM-D showed statistically significant improvement in both groups ($p = 0.001$), with HAM-D scores significantly lower in the three-times weekly group compared to the once-weekly ECT-group at week 4 of treatment only ($p = 0.001$). No statistically significant difference was found between pre-treatment and post treatment or group-differences on cognitive measurements. Information on standard deviations was lacking for both the outcomes on the HAM-D and cognitive measurements. No quantitative analysis could be performed because the principal investigator for the Kellner study has informed us that no additional information is available.

Discussion

Methodological considerations and limited data

Given the fact that that older, frail elderly people are particularly prone to the side effects of antidepressants and that ECT in the elderly may be a more effective and safer treatment, it is of importance to have randomised data available on this specific topic. The lack of well conducted randomised prospective trials in which the efficacy and safety of ECT was compared with antidepressants, made it impossible to test the major hypothesis of this review, namely that modified ECT has a greater and/or more rapid antidepressant effect than simulated ECT, antidepressant drug treatment or non-pharmacological interventions. Evidence on the superiority of real over simulated ECT in elderly could only be based on one study, which contained several methodological shortcomings (O'Leary 1994). Ethically, it does not seem justified to employ

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sham ECT in further trials because comparative active treatments are available for depressive disorder in the elderly. Evidence for the superiority of ECT over antidepressants in depressed elderly was simply lacking. There was no clear evidence to support or refute the use of ECT for particular subgroups of depressed elderly, like those with concomitant dementia, cerebrovascular disorders or Parkinson's disease, because randomised evidence on this topic is also lacking. Although a large number of studies appeared on the major topic and subtopics of this review, they mostly consisted of non-randomised studies. Summarizing the results of these non-randomised studies was not the objective of this review.

Only one study on one of the secondary objectives of this review (Fraser 1980) generated data that could be included in the quantitative analyses. It did not convincingly demonstrate the efficacy of unilateral ECT over bilateral ECT. Because of the small number of studies on the efficacy and safety of ECT in elderly, considerable caution must be taken in generalising these findings.

Generalizability

Randomised evidence on the efficacy and safety of ECT in depressed elderly was sparse. The three eligible studies randomised participants without operationally diagnosed disorders. Extensive information on somatic and psychiatric co-morbidity as well as information on previous treatments and depressive episodes was missing. How the participants therefore resemble those seen in general practice is hard to know.

Limited data

Randomised trials on the efficacy and safety of ECT in depressed elderly was sparse. The collection and quality of reporting data was disappointing. In two of the three included trials means and/or standard deviations were not given, or data were presented in graphs that made it almost impossible to extract useful information.

Sensitivity analysis and publication bias

It was not possible to undertake the proposed funnel graph for publication bias or undertake a sensitivity analysis on subgroups of depressed elderly (those with concomitant dementia, cerebrovascular disorders and Parkinson's disease). The absence of any controlled studies that attempted to replicate the included studies was surprising, given the daily use of ECT in depressed elderly. This leads to the question of whether there might be some bias against research in this understudied area.

Conclusions

Implications for practice

The main conclusions from this review are as follows:

1. Randomised evidence on the efficacy and safety of ECT in depressed elderly is

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sparse, based on trials with a limited number of participants and with shortcomings in methodology and in presentation of outcome data. This leads us to conclude that none of the objectives of this review could be adequately tested. These findings are noteworthy given the relatively frequent usage of ECT in elderly people;

2. One trial concludes that real ECT was superior to simulated ECT. Because of the many methodological problems of this study, replication of these findings with a larger number of participants may be justified;
3. The efficacy of unilateral over bilateral ECT or vice versa is not convincingly proved;
4. The superiority of three-weekly ECT over ECT once a week is not convincingly proved;
5. Randomised evidence on the efficacy and safety of ECT in subpopulations of depressed elderly is completely lacking.

Implications for research

1. Given the specific problems in the treatment of depressed elderly, it is of importance to conduct a well designed randomised controlled trial in which the efficacy of ECT is compared to one or more antidepressants or transcranial magnetic stimulation (TMS).
2. In such trials, it is of importance to establish the medium and long term effects of ECT in outcome, morbidity, mortality and economic values.
3. More studies on the safety and efficacy of ECT compared to antidepressants in specific subpopulations of depressed elderly (like those with concomitant dementia, cerebrovascular disorders) need to be performed.
4. Attention should be paid to the presentation of outcome data in future trials.

Internal sources of support to the review

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External sources of support to the review

* College voor Zorgverzekeringen (CVZ), The Netherlands NETHERLANDS

Potential conflict of interest

None

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Contribution of Reviewer(s)

Frits van der Wurff (FvdW) and Max Stek (MS) are the main reviewers, they will perform the review. FvdW will undertake the identification of the studies. The two reviewers will independently assess the relevance of each trial. In case of disagreement whether to include a trial, this will be resolved by discussion and consensus together with the third and fourth authors, Aartjan Beekman and Witte Hoogendijk.

Synopsis

The reviewers examined the effectiveness and safety of electroconvulsive therapy (ECT) in elderly people with a depressive disorder.

The reviewers performed this review because antidepressants in elderly people regularly cause side-effects which hamper the effectiveness of treatment with antidepressant. ECT therefore can be an important alternative to pharmacological treatment of depression in elderly people. The reviewers extensively searched the literature for well-conducted (randomised) studies, both comparing real ECT to simulated ECT and antidepressants. Only three studies were found, which all had serious problems in methodology. Presently, no definite conclusions can be drawn whether or not ECT is more effective than antidepressants. Neither can be definite conclusions drawn on the safety or side effects of ECT in elderly people with a depressive disorder.

Table of comparisons

Fig unilateral versus bilateral ECT/after 5 treatments

Hamilton Depression Rating Scale

Table of comparisons

Fig unilateral versus bilateral ECT/after 3 weeks treatment

Hamilton Depression Rating Scale

Characteristics of included studies

Study: Fraser 1980

Methods: Allocation: Randomized controlled trial, randomisation procedure not described and allocation concealment unclear;

Duration: not specified;

patient and outcome assessor blinding seems appropriate;

no follow-up period; Intention-to-treat analysis not mentioned, possible 4 patients dropped-out, authors seem to have used an end-point analysis;

Participants: Diagnosis: Depressive illness, based on Feigner criteria;

Age 64 - 86 years;

N = 33, 5 participants dropped-out;

Sex: 8 M, 25 F;

Inclusion criteria: depression of a least a month duration, dementia was not an exclusion criterium, other inclusion criteria not specified.

Interventions: Unilateral versus bilateral ECT; no information is provided on the adequacy of seizures or the method by which seizures were induced;

unilateral ECT n = 13

bilateral ECT n = 16

Outcomes: Mood change: Hamilton Rating Scale (HAM-D) and the Nursus' Observation scale for Inpatient evaluation;

Cognitive side-effects:

the Wechsler Memory Scale

Other side-effects by questionnaire

Notes:

Allocation concealment: B

Study: Kellner 1992

Methods: Allocation: Randomized controlled trial, randomisation procedure not described and allocation concealment unclear;

Duration: not described;

No follow-up period;

Patient blinding: not possible;

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Outcome blinding: procedure not described, bias seems possible, cannot be excluded;

Intention to treat analysis not mentioned, none of the participants dropped out of treatment.

Participants: Diagnosis: DSM-III criteria for major depression;

Age 53-87 years;

participants had been referred for ECT treatment, no information is provided on earlier antidepressant treatment in any of the participants;

N = 15;

Sex: 11M and 4F

Interventions: ECT once a week versus three-times-weekly ECT;

bilateral treatment in all participants;

method of seizure induction adequately described

Outcomes: Mood change: Hamilton Rating Scale for Depression (HAM-D), the Beck Depression Inventory (BDI), the Clinical Global Improvement Scale (CGI) and the Brief Psychiatric Rating Scale (BPRS)

Cognitive side effects: Mini Mental Status Examination (MMSE) and the Wechsler Memory Scale-Revised (WMS-R)

Notes:

Allocation concealment: B

Study: O'Leary 1994

Methods: Allocation: Randomized controlled trial,

Duration: not described;

Follow-up period: six month;

Patient blinding adequate;

Outcome blinding: seems adequate;

Intention to treat analysis not mentioned, 12 of the participants dropped out of treatment.

Participants: Diagnosis: DSM-III criteria for major depression;

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Age 60 - 85 years;

participants had been referred for ECT treatment, no information is provided on earlier antidepressant treatment in any of the participants;

N = 35;

Sex: not provided

Interventions: Sham ECT, versus unilateral or bilateral ECT; maximum number of study ECT-treatment 6;

method of seizure induction described

Outcomes: Mood change: Hamilton Depression Rating Scale (HDRS), Montgomery Asberg Depression Rating Scale (MADRS)

Side effects: no information provided.

Notes: Reexamination of outcome data from the Nottingham ECT trial from 1985

Allocation concealment: B

Characteristics of excluded studies

Study: Alexopoulos 1984

Reason for exclusion: not a randomized controlled trial - retrospective chart review

Study: Alexopoulos 1989

Reason for exclusion: not a randomized controlled trial - review

Study: Allen 1982

Reason for exclusion: not a randomized controlled trial - case report

Study: Allman 1987

Reason for exclusion: not a randomized controlled trial - case report

Study: Andersen 1987

Reason for exclusion: not a randomized controlled trial on depression in Parkinson's disease

Study: Asnis 1977

Reason for exclusion: not a randomized controlled trial - case report

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Study: Atre-Vaidya 1988

Reason for exclusion: not a randomized controlled trial - case report

Study: Avery 1976

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Babigian 1984

Reason for exclusion: not a randomized controlled trial - epidemiologic follow-up study

Study: Ball 1995

Reason for exclusion: not a randomized controlled trial - retrospective chart review

Study: Balldin 1980

Reason for exclusion: not a randomized controlled trial on depression in Parkinson's disease

Study: Barnes 1997

Reason for exclusion: not a randomized controlled trial - case report on maintenance ECT

Study: Beale 1996

Reason for exclusion: not a randomized controlled trial - case report on maintenance ECT

Study: Benbow 1987

Reason for exclusion: not a randomized controlled trial - retrospective chart review

Study: Benbow 1988

Reason for exclusion:

not a randomized controlled trial - letter

Study: Benbow 1989

Reason for exclusion: not a randomized controlled trial - traditional review

Study: Blackburn 1994

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Reason for exclusion: not a randomized controlled trial - case report

Study: Bracken 1987

Reason for exclusion: not a randomized controlled trial - case report

Study: Brodaty 2000

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Brodaty 2001

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Burd 1998

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Burke 1985

Reason for exclusion: not a randomized controlled trial - retrospective chart review

Study: Burke 1987

Reason for exclusion: not a randomized controlled trial - retrospective chart review

Study: Burke 1988

Reason for exclusion: not a randomized controlled trial - case report

Study: Calloway 1981

Reason for exclusion: not a randomized controlled trial - retrospective chart review

Study: Casey 1994

Reason for exclusion: not a randomized controlled trial - traditional review

Study: Casey 1996

Reason for exclusion: not a randomized controlled trial - retrospective chart review

Study: Cattan 1990

Reason for exclusion: not a randomized controlled trial - retrospective chart review

Study: Chacko 1983

Reason for exclusion: not a randomized controlled trial - case report

Study: Coffey 1987

Reason for exclusion: not a randomized controlled trial - case report

Study: Coffey 1988a

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Coffey 1988b

Reason for exclusion: not a randomized controlled trial - retrospective chart review

Study: Coffey 1989

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Currier 1992

Reason for exclusion: not a randomized controlled trial - retrospective chart review

Study: Cybulska 1997

Reason for exclusion: not a randomized controlled trial - case report

Study: D'Mello

Reason for exclusion: not a randomized controlled trial - case report

Study: Devanand 1994

Reason for exclusion: not a randomized controlled trial - review

Study: Dighe-Deo 1998

Reason for exclusion: not a randomized controlled trial - case report

Study: Douyon 1989

Reason for exclusion: not a randomized controlled trial - case report

Study: Drop 1988

Reason for exclusion: not a randomized controlled trial - case report

Study: Dubin 1992

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Reason for exclusion: maintenance ECT, not a randomized controlled trial

Study: Duncan 1990

Reason for exclusion: maintenance ECT, not a randomized controlled trial

Study: Dysken 1976

Reason for exclusion: not a randomized controlled trial, case report

Study: Ehrenberg 1955

Reason for exclusion: not a randomized controlled trial, not modified ECT

Study: Erman 1979

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Faber 1991

Reason for exclusion: traditional review on Parkinson's disease and ECT

Study: Fall 1999

Reason for exclusion: not a randomized controlled trial, case report

Study: Figiel 1989

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Figiel 1990a

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Figiel 1990b

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Figiel 1991

Reason for exclusion: not a randomized controlled trial, case report

Study: Flaherty 1984

Reason for exclusion: not a randomized controlled trial, case report

Study: Flint 1997

Reason for exclusion: not a randomized trial, open study

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Study: Frances 1989

Reason for exclusion: not a randomized controlled trial, case report

Study: Fraser 1978

Reason for exclusion: not a randomized controlled trial, case reports

Study: Fu 1999

Reason for exclusion: not a randomised trial on the efficacy of ECT in depressed elderly

Study: Gallinek 1947

Reason for exclusion: Not modified ECT

Study: Gaspar 1982

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Godber 1983

Reason for exclusion: letter

Study: Godber 1987

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Gormley 1998

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Greenberg 1992

Reason for exclusion: traditional review

Study: Greenwald 1989

Reason for exclusion: not a randomized controlled trial on the outcome of ECT in depression

Study: Guttmacher 1989

Reason for exclusion: not a randomized controlled trial, case series

Study: Hay 2001

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Heshe 1978

Reason for exclusion: mixed unipolar and bipolar group, no separate analysis of unipolar depressive elderly possible

Study: Hickie 1995

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Holcomb 1983

Reason for exclusion: not a randomized controlled trial, case report

Study: Hunt 1998

Reason for exclusion: not a randomized controlled trial, case report

Study: Hussar 1968

Reason for exclusion: not a randomized controlled trial, case report

Study: Jenike 1983

Reason for exclusion: not a randomized controlled trial, traditional review

Study: Jenike 1989

Reason for exclusion: not a randomized controlled trial, traditional review

Study: Karlinsky 1984

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Kelly 2000

Reason for exclusion: not a randomized controlled trial, traditional review

Study: Kelsey 1995

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Kramer 1986

Reason for exclusion: not a randomized controlled trial, case report

Study: Kramer 1987

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Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Krause 1988

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Kroessler 1993

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Krystal 2000

Reason for exclusion: randomized trial on the predictive power of Ictal EEG Indices on respons of unilateral or bilateral ECT. No outcome measures of depression provided. Therefore, study excluded. Authors have been contacted to provide additional information.

Study: Lambourn 1978

Reason for exclusion: although randomisation took place, no reliable extraction on data in elderly possible

Study: Lebensohn 1975

Reason for exclusion: not a randomized controlled trial, case report

Study: Levy 1983

Reason for exclusion: not a randomized controlled trial, case report

Study: Liang 1988

Reason for exclusion: not a randomized controlled trial, case report

Study: Liberzon 1991

Reason for exclusion: not a randomized controlled trial, case report

Study: Lipman 1993

Reason for exclusion: not a randomized controlled trial, follow-up study

Study: Loo 1991

Reason for exclusion: not a randomized controlled trial, case report

Study: Lovell 1948

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Reason for exclusion: not a randomized controlled trial, traditional review

Study: Magni 1988

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Malcolm 1989

Reason for exclusion: not a randomized controlled trial, letter

Study: Mandel 1980

Reason for exclusion: not a randomized controlled trial, case report

Study: Manly 2000

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Martin 1992

Reason for exclusion: not a randomized controlled trial, case series

Study: Mattingly 1991

Reason for exclusion: not a randomized controlled trial, case report

Study: Meyers 1985

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Mielke 1984

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Morris 1991

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Mulsant 1991

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Murray 1986

Reason for exclusion: not a randomized controlled trial, retrospective chart review

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Study: Nelson 1989

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Nelson 1991

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: O' Connor 2001

Reason for exclusion: not a randomized controlled trial on the efficacy of ECT, randomisation on maintenance ECT versus pharmacotherapy

Study: O'Leary 1996

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: O'Shea 1987

Reason for exclusion: not a randomized controlled trial, case report

Study: Palmer 1990

Reason for exclusion: not specifically on elderly with a depression

Study: Pande 1990

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Petrides 1996

Reason for exclusion: not a randomized controlled trial, case report

Study: Pettinati 1984

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Philibert 1995

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Price 1989

Reason for exclusion: not a randomized controlled trial, traditional review

Study: Rao 2000

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Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Regestein 1980

Reason for exclusion: not a randomized controlled trial, case report

Study: Reynolds 1987

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Rice 1994

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Rosen 1992

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Rubin 1993

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Ruxin 1994

Reason for exclusion: not a randomized controlled trial, case reports

Study: Salaris 2000

Reason for exclusion: not a randomized controlled trial, case report

Study: Salzman 1982

Reason for exclusion: not a randomized controlled trial, traditional review

Study: Scott 1990

Reason for exclusion: not a randomized controlled trial, letter

Study: Scott 1991

Reason for exclusion: not a randomized controlled trial, letter

Study: Simpson 1998

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Smith 2000

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Reason for exclusion: not a randomized controlled trial, case report

Study: Sommer 1989

Reason for exclusion: not a randomized controlled trial on the efficacy of ECT in depressed elderly

Study: Spear 1997

Reason for exclusion: not a randomized controlled trial, case report

Study: Stack 1988

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Stern 1997

Reason for exclusion: not a randomized controlled trial, case report

Study: Stoudemire 1990

Reason for exclusion: not a trial on the efficacy or side effects of ECT in depressed elderly, naturalistic study

Study: Stoudemire 1991

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Stoudemire 1993

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Stoudemire 1994

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Stoudemire 1995

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Strain 1971

Reason for exclusion: not a randomized controlled trial, case report

Study: Swett 1977

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Tancer

Reason for exclusion: not a randomized controlled trial, case report

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Study: Tew 1999

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Thienhaus 1990

Reason for exclusion: study of the efficacy of maintenance ECT

Study: Tomac 1997

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Van Marwijk 1988

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Van Waarde 2001

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Ware 1990

Reason for exclusion: not a randomized controlled trial, case report

Study: Weiner 1982

Reason for exclusion: not a randomized controlled trial, traditional review

Study: Weisberg 1991

Reason for exclusion: not a randomized controlled trial, case report

Study: Wesson 1997

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: West 1999

Reason for exclusion: not a randomized controlled trial, case report

Study: Wetterling 1998

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Wijeratne 1999

Reason for exclusion: not a randomized controlled trial, case report

Study: Wilkinson 1993

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Williams 1997

Reason for exclusion: not a randomized control trial, naturalistic study

Study: Wolff 1954

Reason for exclusion: no modified ECT

Study: Yesavage 1980

Reason for exclusion: not a randomized controlled trial, retrospective chart review

Study: Young 1985

Reason for exclusion: not a randomized controlled trial, case report

Study: Yudofsky 1979

Reason for exclusion: not a randomized controlled trial, case report

Study: Zorumski 1988

Reason for exclusion: not a randomized controlled trial, review

Study: Zubenko 1994

Reason for exclusion: not a randomized controlled trial, naturalistic study

Study: Zvil 1992

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THE NORTHWICK PARK ELECTROCONVULSIVE THERAPY TRIAL

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62 completed
How many in each group?
Summary 70 patients with endogenous depression, defined by strict criteria, who fulfilled the Newcastle indications for electroconvulsive therapy (ECT) were randomly allocated either to a course of eight simulated ECTs or to a course of eight real ECTs. The improvement in terms of psychiatrists' ratings in the group of patients given real ECT was significantly greater ($p < 0.01$) than that in those given simulated ECT, but the difference between the two groups was small in relation to the considerable improvement of both groups over the 4-week treatment period. No differences were found between the two groups at one-month and six-month follow-up. The therapeutic benefits of electrically induced convulsions in depression were of lesser magnitude and were more transient than has sometimes been claimed. In the real-ECT group memory was impaired during treatment but memory tests revealed no difference between the groups at six-month follow-up.

Introduction

ELECTROCONVULSIVE therapy (ECT), introduced by Cerletti and Bini¹ over 40 years ago, has been a major treatment of severe depressive illness. The antidepressant efficacy of the whole procedure is well established^{2,3} but, although the convulsion is often held to be a critical element, the evidence that this is so is slender. Cronholm and Ottosson⁴ found that 46 depressed patients treated with electrically induced convulsions improved more than 23 patients in whom the convulsions were shortened with lignocaine, but the treatments were not allocated at random. Robin and Harris⁵ reported that a group of 15 depressed patients treated with ECT and placebo tablets did significantly better than 16 patients treated with "pseudo-ECT" (an anaesthetic with no shock) and imipramine. Other workers,^{6,7} treating diagnostically mixed groups of patients, found that the addition of electrically induced convulsions

offered no advantage over the administration of anaesthesia alone. This issue has lately attracted attention. Freeman et al.⁸ presented data which suggested that a course of bilaterally applied ECT which began with two pseudo-ECTs effected significantly slower improvement than a course which included real ECTs from the first treatment. On the other hand, Lambourn and Gill⁹ reported that depressed patients improved as quickly with six sessions of pseudo-ECT as with six real unilateral treatments.

The present study concerned 70 patients diagnosed by well-defined criteria as having endogenous depression and randomly allocated either to eight real or to eight pseudo ECTs. The mental states of the patients were assessed, by psychiatrists who were unaware of the treatment allocation, throughout the course of treatment and again one month and six months after completion of the course.

In addition to the question of the role of the convulsion in the efficacy of ECT, possible short and long term effects of the convulsion upon memory are of considerable interest. This topic has received substantial study¹⁰ but the effects of ECT have been hard to disentangle from those of change in mood.¹¹ The present study provided an opportunity for clarifying this issue. Memory was tested before and during the study and at the six-month follow-up.

Method

From the results of previous studies of depressed patients with the Hamilton¹² depression scale it was calculated that a sample of 70 patients would be adequate to clarify the question of whether or not the convulsion is an important element in the therapeutic efficacy of ECT. The patients were selected from those aged 30–69 years who required inpatient treatment for depressive illness and were admitted to Northwick Park Hospital under the care of the participating psychiatrists. After admission, the Present State Examination¹³ was conducted and the following criteria for inclusion in the trial were applied: the MRC³ criteria for depressive illness (modified by the extension of the age range to 30–69 years); the Feighner¹⁴ criteria for primary depressive illness; the Newcastle¹⁵ criteria for endogenous depressive illness; and the Newcastle¹⁵ criteria for predicting a good outcome from ECT. Patients who fulfilled these criteria were asked for consent to ECT and inclusion in the trial. Relatives' consent was also obtained. In those who consented the risk of anaesthesia was assessed by the anaesthetist concerned and if this was thought to be increased the patient was excluded. Patients were entered in the trial until the target figure of 70 cases was reached. During this time 109 depressed patients of appropriate age were admitted. Of the 39 who were not included 2 were detained under the Mental Health Act, 6 were regarded as poor anaesthetic risks, 4 refused ECT, 8 refused to participate in the trial, and in 19 the characteristics of the illness did not meet the criteria.

Having been included in the trial patients were allocated, for the purpose of randomisation, to separate groups according to the

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Leeds =
self
assessment

presence or absence of delusions, agitation, or retardation. The allocation of the treatments was known only to the psychiatrist who administered the ECT and to the anaesthetist. Neither of these doctors was involved in the care or assessment of the patients. The assessments used were the Hamilton¹² depression ratings, the Leeds¹⁶ scales for depression, and the nurses' rating scales devised by Bunney and Hamburg.¹⁷ The Hamilton ratings were conducted by one of the two consultant psychiatrists and were done before the course of ECT, weekly throughout the course, and one month and six months after completion of the course. The patients were asked to complete Leeds¹⁶ scales at the same time. The treatments were given on Tuesdays and Fridays. Patients who began their course of ECT on a Tuesday were rated on five consecutive Mondays and those who began their course on a Friday were rated on five consecutive Thursdays. The mean time between admission and the start of ECT was 9.4 days. After the first four treatments a third psychiatrist independently assessed the advisability of continuing with the study. All the patients were treated as inpatients in the research ward of the psychiatric unit at Northwick Park Hospital.

Both groups of patients received methohexitone 1.5 mg/kg, atropine 0.6 mg, and suxamethonium 0.5 mg/kg. No electricity was passed in the simulated ECT group but in the real ECT group electrodes were placed in the bifrontal position and a current of 150 V duopulse wave form 1 was passed for 3 s. To allow confirmation that a convulsion had taken place a sphygmomanometer cuff inflated above arterial pressure was applied to one arm. In this way the convulsion could be observed unmodified by muscle relaxants. No other antidepressant treatment was given. All patients had a benzodiazepine hypnotic every night during the trial and if additional sedation was necessary diazepam was prescribed.

Memory tests were conducted by psychologists who, like the clinical investigators, remained blind to the allocation of the treatments until all the six-month follow-up assessments had been completed. This selection of tests was intended to assess both the immediate and the possible long-term effects of ECT.

After the eighth treatment the Hamilton¹² and Leeds¹⁶ ratings were completed and the patients were then treated as the consultants in charge of them thought fit.

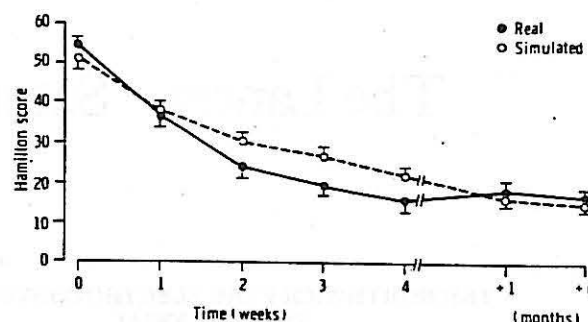
Results

General

The sample consisted of 52 females and 18 males of mean age 49.4 years. 46 patients had had definite previous episodes of depressive illness and 7 had had definite previous episodes of mania. 15 patients had received ECT for a previous episode. 49 patients had had antidepressants prescribed for the index episode before admission to the trial. 62 patients completed the full course of eight treatments. Of the 8 patients who did not, 4 (3 on simulated ECT, 1 on real) were withdrawn because of failure to progress; 1 on real ECT was withdrawn because he had a minor vascular incident involving his retina; 2 (1 on real ECT and 1 on simulated) withdrew consent to ECT; and 1 (on real ECT) became manic. Of the 62 patients who finished the course 18 (8 on real ECT and 10 on simulated) were given benzodiazepines, mainly either as diazepam 5 mg regularly thrice daily or as diazepam 10 mg in occasional doses to relieve distress. Improvement scores were similar in patients with and without diazepam. The only other psychotropic medication was a benzodiazepine hypnotic prescribed for all patients. No antidepressant medication was given during the trial. 57 of the 62 patients who completed the course were seen one month and six months later.

Outcome Assessed by Clinical Ratings

Patients in both groups improved considerably during the course of the treatment but the improvement was greater in



Severity of depression in relation to the course of ECT and one and six months thereafter.

the real-ECT group. The advantage of real over simulated ECT was not retained and at the one-month and six-month follow-ups the Hamilton¹² scores of the two groups were almost the same (see figure). The Leeds¹⁶ self ratings showed similar trends but these were never significant, and this was also true of the ratings by nurses using the Bunney and Hamburg¹⁷ score.

The effects of ECT were assessed by analysis of variance at the end of treatment and one month and six months after completion of treatment. The tests ask whether the two groups (real or simulated ECT) differ significantly in terms of mean improvement at each time of assessment. At the end of week 4—i.e., after eight treatments—this effect is significant ($F [1,54] = 7.8, p < 0.01$) but at one month ($F [1,51] = 0.1, NS$) and six months ($F [1,49] = 0.4, NS$) there was no difference between the groups.

The patients' treatment ceased to be determined by the rules of the trial after the rating which immediately followed the course of real/simulated ECT. When the one-month and six-month ratings were done the patients had been on various treatments as dictated by their clinical condition. The treatments given to the two groups of patients during this time were very similar (table 1) and it is not possible to

TABLE 1—TREATMENT IN MONTH AFTER COMPLETION OF COURSE OF TRIAL ECT

Treatment	No. of patients receiving treatment	
	Real	Simulated
ECT	1	2
Tricyclics	16	13
Other antidepressants	1	4
Anxiolytics	9	7
Lithium	1	2
Nil	3	2

attribute the loss of the advantage of real ECT to differences in the subsequent treatment of the two groups. In order to check the blindness of the study the investigators guessed which treatment they thought the patients were having. The accuracy of these guesses was no better than that predicted by chance.

Results of the Memory Tests

These will be dealt with in detail in another publication and are only referred to briefly here. Memory deficits were clearly demonstrated in the real ECT group during the course of the

TABLE II—RESULTS OF MEMORY TESTS BEFORE, DURING, AND AFTER THE COURSE OF ECT

62/31+31? Doesn't say 1319
After 57 total

Test	No. of patients		Before treatment		During/after treatment		6 mo follow-up	
	Real ECT	Simulated ECT	Real ECT	Simulated ECT	Real ECT	Simulated ECT	Real ECT	Simulated ECT
Subjective memory (% of patients complaining)	18	9	70	68	62	38	38	26
Concentration	16	20	62	80	67	56	50	32
15 min vigilance task (% of patients missing signals)	20	19	76	74	57	32*	—	—
Word list recall (words out of 20)	20	19	7.2	8.0	6.5	8.3	8.7	—
Non-verbal recall score (complex line drawing)	17	23	—	—	9.1	20.2†	—	—
Learning labels for faces: learning score	29	28	—	—	2.6	4.6‡	5.0	5.2
Long-term memory (sentence comprehension time)	26	27	—	—	2.4	3.6§	—	—
Long-term memory (discrimination of names from the past)	14	12	2.16	2.08	—	—	2.08	2.09

* $\chi^2 = 2.8$ ($p < 0.10$); † $F(1,36) = 16.1$ ($p < 0.01$); ‡ $F(1,53) = 5.2$ ($p < 0.05$); § $F(1,45) = 6.4$ ($p < 0.05$)

treatment but there was no evidence of persisting memory impairment at the six-month follow-up (table II). For this comparison patients who received ECT at any time other than during the trial were excluded.

Discussion

Although significant differences were established for only one of the three methods of rating used, the findings show that electrically induced convulsions do enhance the rate of recovery from an episode of depression. The findings differ in important respects from those of both Freeman et al.⁸ and Lambourn and Gill,⁹ although in terms of initial Hamilton¹² scores the patients in these studies had depression of similar severity to those participating in ours. Thus in a series of 70 patients treated with a course of eight bilateral real or simulated ECT over 4 weeks we found a significant effect of the convulsion while Lambourn and Gill,⁹ using six unilaterally applied real or simulated treatments given over 3 weeks in 32 patients, found none. On the other hand Freeman et al.,⁸ studying a group of 40 patients, reported that significant improvements could be detected on some rating scales after two real as compared with two simulated ECTs. In our larger series no significant difference was found after two treatments. In the study of Freeman et al.⁸ clinicians seem to have been able to detect group differences at the end of the course of treatment and gave significantly more ECT to the group of patients in which the initial two treatments were simulated. By contrast, the clinicians responsible for our patients (who, unlike those in the study of Freeman et al.,⁸ received no antidepressants during the 4-week course of the treatment trial) gave similar amounts of antidepressant therapy to the two groups of patients after the trial period (table I).

While our trial reveals a significant advantage ($p < 0.01$) for the electrically induced convulsions, the size of the difference between the two groups is not large. The group receiving real ECT showed a mean improvement of 38.1 (SE 3.0) points on the Hamilton scale over the 4 weeks of the trial while those receiving simulated ECT showed a mean improvement of 28 (SE 2.7) points.

The most striking finding is that the differences which were present at the end of the course of eight treatments had disappeared one month later and were undetectable also at six

months (figure). Although the use of antidepressant treatments (including ECT) during the follow-up period was not restricted by the trial design there were negligible differences between the groups in the extent to which such treatments were considered necessary. The findings of this study offer no support for the view that the benefits of repeated convulsions are substantial and long-lasting; they indicate that the benefits lie in speed of response rather than in long-term outcome. Because both our treatment groups had general anaesthesia we cannot assess the extent to which this, either by pharmacological or by psychological mechanisms, may contribute to the previously reported advantages of the ECT procedure over other antidepressant regimens.^{2,3} Moreover, since no antidepressant-drug treated group was included, we cannot assess the question of whether optimal drug treatment can achieve as rapid a response as that seen with eight episodes of anaesthesia either with or without a convulsion.

Although these results indicate that electrically induced convulsions offer less benefit in terms of antidepressant effect than has sometimes been thought, they also indicate that such convulsions are not as harmful as has been supposed. Subjective accounts have indicated that some patients experience persisting impairments,¹⁸ but in our wide-ranging series of tests no differences were established at six months between the real and the simulated ECT groups, who had at that time very similar ratings on the Hamilton¹² scale. We thus found no evidence that a series of eight electrically induced convulsions gives rise to lasting memory impairment but we can make no comment of the effect of larger amounts of ECT.

In conclusion, our findings suggest that the antidepressant effects of repeated electrically induced convulsions are of relatively short duration. No lasting effects upon memory were detected. 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.

This trial was conducted under the auspices and according to the rules of the Ethical Committee of Northwick Park Hospital and with the advice of the Drug Trials in Psychiatry Subcommittee of the Medical Research Council under the chairmanship of Prof. M. Shepherd. We thank sisters I. Crichlow and G. Andrews, charge nurses M. Howell and C. Morris, staff nurse V. Palmer, and the nursing staff of Eastlake Ward, Northwick Park Hospital, who

NO
meaningful
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figures
given
for
comparison

contributed so much to the conduct of this trial, Dr A. Davenport and Dr D. White for anaesthetic assistance, and Dr J. F. McMillan, Dr D. G. C. Owens, and Dr J. S. Price.

Requests for reprints should be addressed to E.C.J.

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ALLOGENEIC BONE MARROW TRANSPLANTATION USING STEM CELLS FRACTIONATED BY LECTINS: VI, IN VITRO ANALYSIS OF HUMAN AND MONKEY BONE MARROW CELLS FRACTIONATED BY SHEEP RED BLOOD CELLS AND SOYBEAN AGGLUTININ

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Summary A procedure was developed for the isolation from human bone marrow of a cell fraction enriched for haematopoietic precursors and depleted of T lymphocytes. T cells are eliminated from bone marrow by rosetting with sheep red blood cells, followed by differential agglutination of residual T lymphocytes in the non-rosetting population by the lectin, soybean agglutinin. The fraction unagglutinated by the lectin contains a high proportion of colony-forming cells and non detectable T cell alloreactivity in vitro. Similar results were obtained with monkey bone-marrow cells, suggesting that monkeys can be used for evaluation of this fractionation technique for bone-marrow transplantation across histocompatibility barriers.

Introduction

In the past decade, marrow transplantation has emerged as a promising curative approach to the treatment of lethal

congenital immunodeficiencies,¹ aplastic anaemia,² and leukaemia.³ At present, a marrow transplant can be used only for the minority of patients who have a suitable donor, matched for determinants of the HLA system, since transplants from HLA-histoincompatible donors commonly induce lethal graft versus host disease (GvHD).⁴ However, results in rodents suggest that lethal GvHD can be avoided if, before administration, the histoincompatible marrow graft is depleted of alloreactive T lymphocytes.⁵⁻⁸

In earlier work, Reisner et al. showed that the plant lectin soybean agglutinin (SBA) binds to the B lymphocytes and haematopoietic precursors in the marrow and spleen of the mouse, and does not bind to T lymphocytes in these tissues.^{8,9} Marrow or spleen cells could thus be fractionated by agglutination with SBA and differential sedimentation of agglutinated cells on 5% albumin.^{9,10} The agglutinated T lymphocyte depleted fraction can be dispersed by washing with D-galactose, the specific sugar inhibitor of SBA. Reisner et al. subsequently showed that this cell fraction could be used to reconstitute haematopoietic function in lethally irradiated H-2 incompatible mice, without GvHD.⁹

We record here the SBA lectin-binding characteristics of lymphoid and haematopoietic elements from human and cynomolgus monkey marrow.

Materials and Methods

Human Bone-marrow (BM) Cells

BM cells from healthy volunteers were obtained by aspiration from the iliac crest as previously described.¹¹

Monkey BM Cells

BM cells from cynomolgus monkeys (*Macaca fascicularis*) were obtained either by aspiration from the femora, tibiae, and iliac crests of living animals or by flushing of marrow from the long bones of killed animals.

Isolation of BM Mononuclear Cells

Mononuclear cells were isolated from the heparinised marrow cell suspension by density gradient centrifugation over 'Ficoll-Hypaque' (Lymphoprep, Nygaard, Oslo, Norway) as previously described.¹²

Lectin

SBA was purchased from Vector Laboratories, Burlingame, California.

Separation of BM Mononuclear Cells by SBA

Ficoll-isolated BM mononuclear cells (2×10^8 cells/ml, 0.5 ml) were incubated in polystyrene tubes (17×100 mm) with SBA (0.5 ml, 2 mg/ml phosphate buffered saline (PBS)) for 5 min at room temperature. The cells were then gently layered on top of a solution of bovine serum albumin (5% w/v in PBS, 8 ml) in a 15 ml conical bottom plastic tube. After 10 min at room temperature the agglutinated cells sedimented while the unagglutinated cells remained at the interface with the bovine serum albumin solution. Top and bottom fractions were removed separately and transferred to 15 ml conical bottom plastic tubes. The cells were then suspended in 10 ml of D-galactose (0.2 mol/l) in PBS. After 10 min at room temperature the cells were collected by centrifugation (200 g, 5 min) and washed once more with D-galactose and then twice with PBS.

Fractionation of Human BM Cells by Sheep Red Blood Cells (SRBC) and SBA (fig. 1)

Immediately after aspiration, the bone-marrow cell suspension is mixed with 'Hetastarch' (McGraw Laboratories, Irvine, California,



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Teacher
Student
Occupational Therapist
Shorthand typist
Taxi driver
Careworker
Office Clerk
Typist
Audit Nursing
Management
Nurse (Ex NHS)
Housewife
Legal secretary
Retail store supervisor
Poultry Farmer
Housewife
Schoolgirl
Expense Analyst
Diagnostic radiographer
State Regd. Nurse
Teacher
Social Worker
Commercial Artist
Student
Journalist
Plumber
A level student
Prison Officer
Chef
Labourer
Student
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Student
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Catering assistant (ret.)
Librarian
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Banker
Pro photographer
Nurse
Unemployed
Nurse
Soleman
BT Engineer
Typist
Local government clerk
Apprentice painter
Secretary
Local government officer
Student
Post Grad. Res. Student
Technician
Labourer
Nurse (Ex NHS)
Driver
Refined
Insurance collector
Housewife

What do a nuclear physicist, a chef, a Royal Marine commando, a plumber and 7 nurses have in common?

Answer: they are all linked by the same experience - *powerful electric shocks through their brains that left a legacy of profound after-effects.*

And these are just a sample from literally *hundreds* in our files - all shocked, not accidentally, but *deliberately*, by doctors who assured them it was a safe and effective way to cure their mental problems.

They have never met each other, yet again and again they describe similar effects that have disrupted their lives, ended their careers and left them with dull and blunted minds.

They ask: *why do I feel like a robot?... why have I no feelings any more?... why am I so fatigued all the time?... why can't I remember day to day things like I used to?... are others as scared as I am of visiting the doctor?*

After first hearing of us, many spent weeks gathering the courage to ring and tell their story. Some spoke for hours, relieved beyond measure to find at last someone who understood and *believed* what they were saying. Many felt they had been "mentally raped".

Just what exactly are they saying?

In September 1998, to determine just what had happened to all these victims of Electroconvulsive Therapy (ECT), we send out over 500 detailed questionnaires. In particular, we included a list of the most-reported symptoms and asked victims to indicate any they had developed AFTER their shock treatment.

We didn't expect a very high response. It is a great credit to these fragile and damaged victims that so far over 200 have replied - with only a handful of anonymous ones. **Better still - almost half of them have agreed to 'come out' and tell their story in public!** (see back page).

Survivor-group **The US-Wales Network** have since sent out a further 600 forms to their membership. Other groups are doing the same. What we've seen so far is just the tip of a scandalous iceberg.

When listing questionnaire results it is easy to lose, amongst the numbers, the humanity of what is behind them. Please, when you read them, try to keep clearly in your mind that these figures equate *directly* with real human lives - with lost hopes and dreams and ambitions and loves.

We can't show the personal details that might fully bring this home to you. But a person's occupation can offer a glimpse into the reality of their life. Take a look at the list in the left margin. It goes on page after page. It is a catalogue of destroyed lives; and it's just one part of what being ECT-shocked can mean. Somewhere down its long length there is indeed a nuclear physicist, a chef, a Royal Marine Commando, a plumber and seven nurses. Amongst a host of varied occupations there are also seven teachers, seven secretaries, 16 housewives, 18 students **and five children**, for goodness sake, one as young as thirteen years.

Think of them when you read the mere numbers in the following pages.

What was the illness for which you were given Shock Treatment?

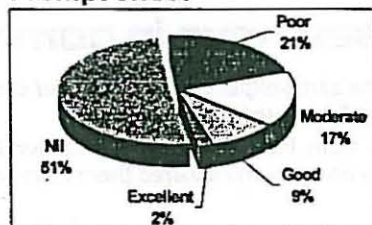
We expected this to be mainly depression. However, although two-thirds did indeed answer 'Depression' or 'Nervous Breakdown', 13% had been given it for schizophrenia and the remainder for a wide variety of often inappropriate reasons such as "Asthma" or "Epilepsy" or "Pain in joints" and even "sexual abuse".

How effective was the Shock Treatment against this condition?

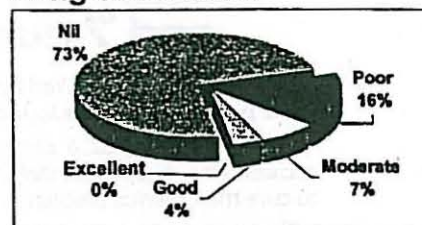
As, you can see, about half our respondents were able to report that ECT initially had some effect, although a large proportion of even these deemed it to be "poor".

However, when it came to the long-term result, almost three-quarters reported that the treatment had been completely ineffective, and most of the rest now felt it had been "poor".

Prompt effect



Long-term effect

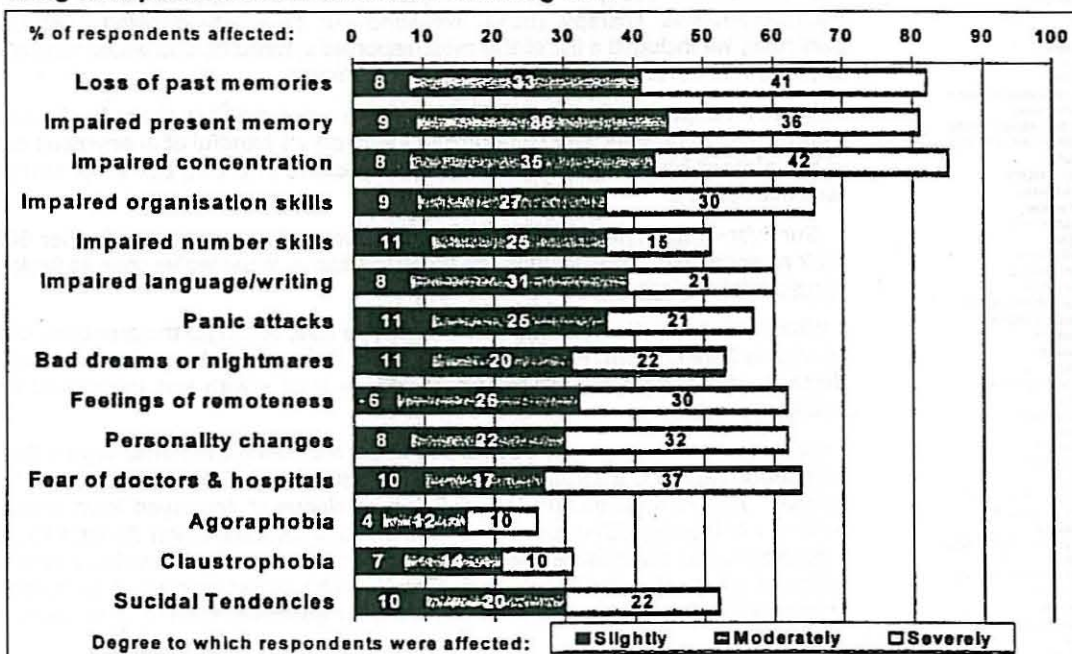


What conditions have you developed SINCE your ECT?

Whatever the effect of the treatment on the original condition, ALL our respondents found the after-effects widespread and devastating. Against a list of previously-reported symptoms, we asked them to mark A, B or C (or nothing) according to how severely they had been affected.

We knew to expect a high incidence of the first few conditions shown below, but we were utterly unprepared for the sheer scale **and severity** of the damage that each individual reported. Almost everyone reported a host of symptoms. And their forms **bristled** with Bs and Cs.

Long-term/permanent mental effects following ECT.



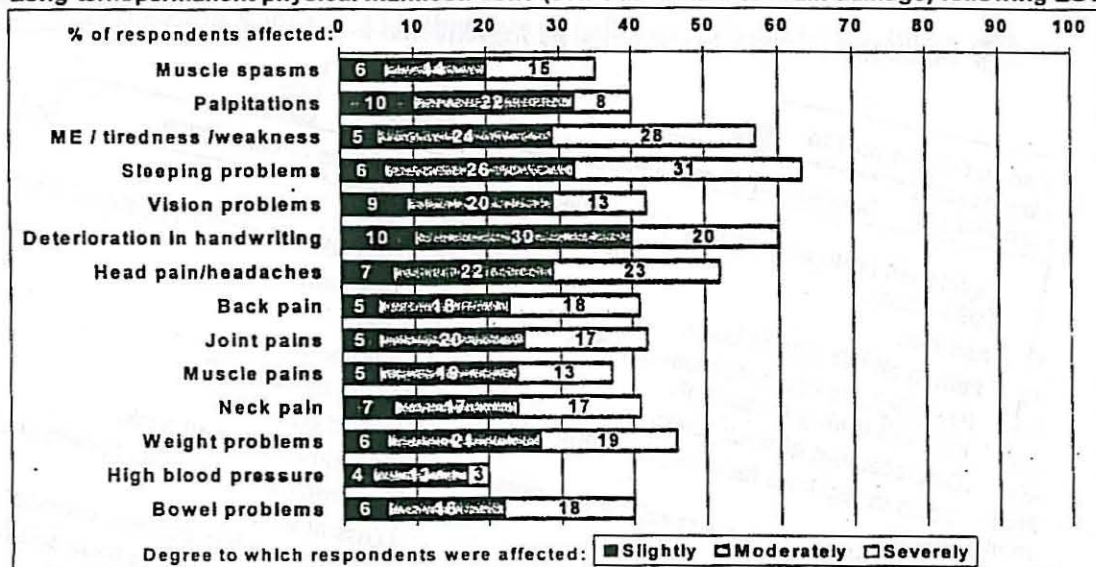
In the early days of ECT, despite rubber gags and the like, broken jaws and spinal or back injuries were fairly common, due to the violent convulsions caused by the shock.

Nowadays, powerful "muscle-relaxant" drugs are used, which paralyse the entire body. Despite this, it is evident from the disturbing results below that widespread physical damage is still occurring to a serious degree and which is all too often permanent.

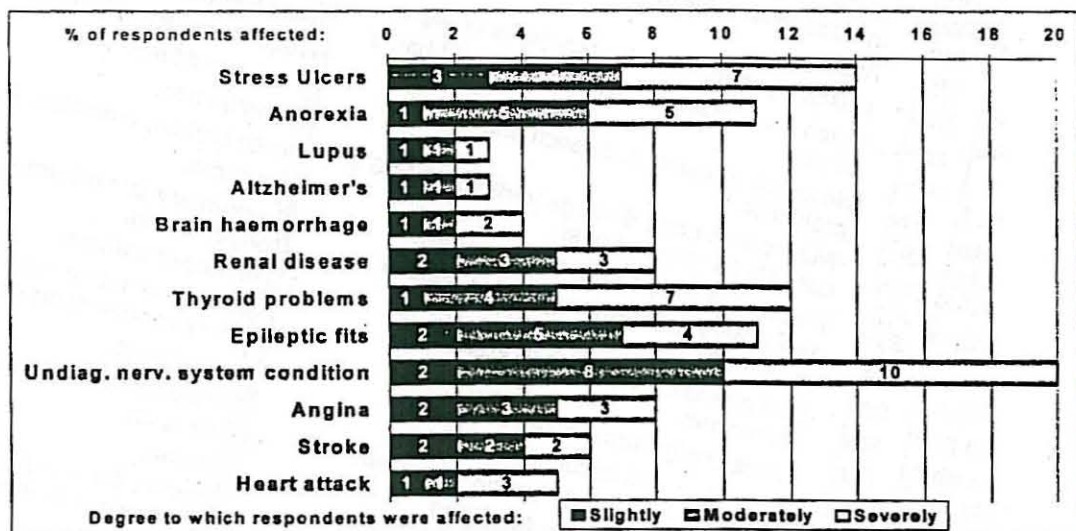
Deterioration in hand-writing, muscle-spasms (and others) are really "brain damage" problems, but are included here because, having physical manifestations, they can be easily shown to be real.

The high percentage of respondents reporting weight problems may be quite significant - the BBC2 *Horizon* series recently showed a connection between brain damage and weight gain.

Long-term/permanent physical manifestations (some indicative of brain damage) following ECT.



Below are the categories which were reported by only a relatively small number of respondents. Even so, there are some disturbing results here that are worth looking into further.



We asked respondents to tell us of any conditions they'd developed which weren't included in our list. Six people reported asthma, which may well be linked with repeated anaesthesia.

Many people reported a wide variety of distressing conditions - see Insert.



**QUESTIONNAIRE
RESULTS
MARCH '99**

14 Western Ave Riddlesden Kelghley West Yorks BD20 5DJ
ECT Anonymous
Phone: 01535 661493 Fax: 01535 602736
email: ect.anon@ndirect.co.uk

Additional information supplied by respondents about their problems following ECT.

Had to relearn how to
town; can't
familiar pla

Breakdown of marriage, lost custody of two of

Developed brain abnormality (diagnosed in
1991-scan).

Asthma.

Pain in all left side of body.

Paraplegic as result of brain damage
incurred during treatment.

Displacement of neck joints.

Pains in legs and feet despite months of
drugs.

Right/left confusion - spacial awareness
problems.

Visual memory wrecked.

Loss of taste.

Difficulty recognising familiar faces; noises
in head before going to sleep;

Asthma.

Muscle spasms - involuntary jerks of legs.

Given brain scan and then asked if I'd had a
stroke.

No sense of balance. Speech and hearing
problems.

Widespread brain damage verified by EEG's.
Lost 15-20 years of past.

Fracture of jaw; spinal degeneration.

Jaw & neck problems - damage caused
during ECT.

Lost one knee cap during ECT due to
dislocation.

Large benign tumour on neck.

Allergies - bad circulation.

Almost totally blind - retina damaged.

Possibly epileptic fits (awaiting
confirmation).

Noises in head before going to sleep;
difficulty following simple books and films.

Phobia, Loss of confidence.

Epilepsy.

Deterioration of sight - registered blind.

Incontinent.

Vertigo.

Clumsiness.

Sexual dysfunction.

Loss of creativity.

Neuralgia, face and neck.

Became docile, remote, speech shaky.

Loss of smell.

Confusion when outside, difficulty shopping.

Tingling sensations in head and face.

Damage to teeth during ECT.

Passing out for no reason.

Hand tremors.

Frequent severe migraines.

Noise sensitivity.

Incontinence.

Teeth broken, crumbled. (letter attached).

Dizziness.

Involuntary jaw movements at night causing
tongue biting.

Developed asthma.

Leaving out letters when spelling words;

Lack of coordination; confusion.

Hearing loss.

Incontinence; loss of speech; require full
time care.

Asthma.

Couldn't draw anymore, couldn't think
visually; fear of travel.

Losing balance.

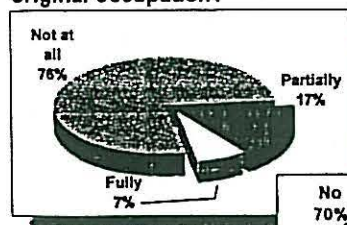
Problem remembering events in early life.

Unable to put things in chronological order.

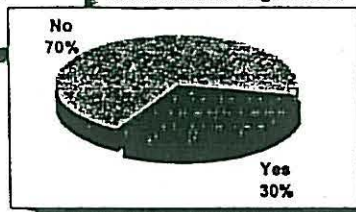
Burns on head after treatment.

● Were you able to return to your original occupation?

Were you able to return to your original occupation?



Could you take up other, less demanding work?



Our respondents answered a resounding **"NO!"** to this question. Take another look at that list in the left margin of page one, and imagine it running on, page after page, not only for all our 200+ respondents, but for the hundreds more still in our files who didn't reply, and the thousands more who've never heard of us. Then look at the bald statistics illustrated here and try to imagine the wholesale destruction of peoples' lives that ECT is **routinely** causing.

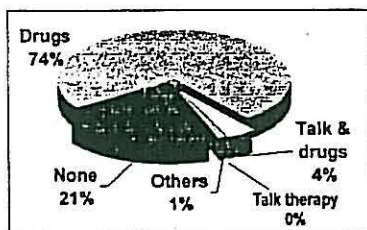
Writer Ernest Hemingway said, after receiving forced Shock Treatment:

"What is the sense of ruining my head and erasing my memory, which is my capital, and putting me out of business? It was a brilliant cure but we lost the patient."

38 years later we're still putting people out of business and losing patients.

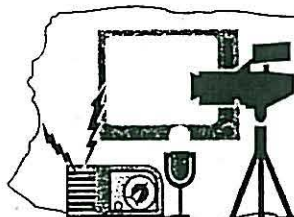
● Did you feel you were pressured to have ECT?... and were the risks FULLY explained to you?

Our correspondents have often complained that they were pressured to have ECT, sometimes under the threat of sectioning, but more typically by its being 'oversold' by psychiatrists as "quick, safe and effective". In reply to our questions on this aspect of ECT, 87% felt they had been pressured, and an even more decisive 98% felt that the risks had not been properly explained to them.



We were astonished to find that 11% had been **outpatients** (and were thus probably not ill enough to have needed such an extreme therapy), and a sobering 24% **were forced to have it** under Section 58 of the Mental Health Act. For only 24% of the voluntary patients was a relative or other supporter involved in making the decision.

This chart (left) shows the alternatives offered before resorting to ECT. Despite RCP recommendations that ECT is used only as a last resort, a disturbing 21% were offered it first. The extreme paucity of any kind of talk therapy speaks for itself. (no pun intended).



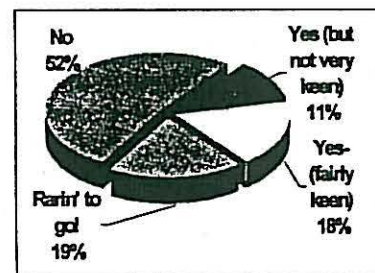
Would YOU be prepared to appear on TV?

One of our main functions is to publicise the truth about ECT as widely as possible, and in this respect it is vital we gain the maximum media coverage. A single television or radio program or newspaper article can do more than years of sending out information packs. In the three years since *ECT Anonymous* was first set up our survivors have appeared on *News at Ten*, *Look North*, *Here and Now*, *Over the Edge*, *North of Westminster*, *Kilroy*, *The Cook Report*, *Radio 4's All in the Mind*, *Radio Wales*, *Midlands Radio*, and in many newspaper and magazine articles.

This was the last and perhaps in many ways the most important question we asked. Only by victims standing up and telling their stories loudly and publicly will we ever hope to bring about the kind of outrage needed to change things. We were thus astonished and delighted to find that almost **HALF** our respondents were prepared to do just that, to "come out" and tell their stories **publicly**.

So, you media people out there, we now have a list of over 100 people ready to talk. More are being added daily (the *US Wales* results are starting to come in). Over 40 are ready to shout it from the rooftops!

You want personal stories? We're ready when you are.



QUESTIONNAIRE RESULTS

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20 Valley Creek Road
Norristown, PA 19401
July 17, 1990

Dear Sir:

Because of the severe brain damage and furthering disability that I received from ECT (Electroconvulsive Therapy), I am sincerely requesting that FDA's proposal to reclassify this deadly device from Class III to Class II be disapproved.

The confusion, memory loss, (in my case, much of it permanent, is a great price to pay and still remain as a functioning individual in a family and place of work. Although functioning while in a severe depression comes to a halt, the super-imposed damage of ECT does not make things better but worse. The consent form used by the APA which states that there is only temporary memory loss in and around the time of the treatments is a total lie. The only thing I could remember was the treatments and the daze that followed me from day to day as I could not remember who I was, where I was, or what I would ever do now in this condition. This is NO SMALL MATTER. The American public is not as uneducated as they were at one time in medical matters. However, I fear that enough education is not available to those considering such a drastic measure for themselves or for a loved one. Temporary memory loss does not exist. The loss is frequently over a period of previous years as well as the loss of the ability to process information that is necessary for daily living. Our brain is our computer and with it damaged, we are limited in our capabilities. The damage from ECT can be extreme and completely disabling, to a degree inconceivable except by those who have undergone this horror. Ridiculously enough, though there are support groups in this country for just about everything, there is none for those who need help after the ravages of ECT. This is no doubt because of the denial by the psychiatrists that it does little or no damage and is no doubt due to the person's "depression". That is one easy way to push away the real facts. Ernest Hemingway, who was treated for depression by electroshock "therapy", later committed suicide because he could not remember how to write, and that was his livelihood.

Are you aware that the FDA files are filled with reports from persons just as myself? All of us want to be HEARD. This is an archaic treatment and needs further investigation--but not on humans, or for that matter, animals either. It holds no place in this world of modern science. It is a quick "cure" just as a good blow to the head would cause a person to forget his troubles until the daze left, perhaps days, weeks, or months later. It is no different than the punch-drunk affect exhibited by the boxer after he has been knocked out.

After 5 years, my brain is beginning to heal to some extent,

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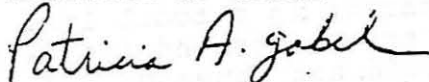
but the horror of ECT will never leave. Please consider these facts and do not lay the path for future victims of the "monster machine, the ECT device."

Stop ECT before it stops the lives of more of our American public. How do we know how many of those who were shocked have never been able to get out of institutions because of ECT, because of the further damage to body and soul. Please keep in mind that the business of psychiatry and the medical profession is to heal, and not to destroy.

Thank you for your consideration in reading this letter. Though I am certain you are a busy administrator, I would so appreciate a reply.

Sincerely

Patricia A. Gabel

A handwritten signature in cursive script that reads "Patricia A. Gabel". The signature is written in dark ink and is positioned below the typed name.

Enclosure: James B. MacRae, Jr.

NETS MANAGER

90 JUN 14 1990

2345 Balsam Drive
Boulder, Colorado 80304
May 30, 1990

Mr. James Benson
Food & Drug Administration
40 Dockets Management Branch (HFA-305)
5600 Fishers Lane (Room 4-62)
Rockville, Maryland 20857

Dear Mr. Benson,

I am writing to urge you to use your influence to keep Electroshock Therapy in Class III regulation.

I have had an unusually severe, 15½ year major depression, unrelenting and resistant to psychotherapy and all available medications. I was urged to have ECT by several psychiatrists and, believing I would be helped, I agreed. I was a patient at University Hospital, Denver, Colorado from early January to March 22, 1989. I was given 12 treatments and allowed to come home. A few days later, I was so desperate to get relief from anxiety and depression that I nearly died by a suicide attempt. I was asked to sign permission for further ECT within less than 24 hours after I overdosed on medication. I was in no mental state to be capable of making a decision to have further electroshock treatments so I blindly signed permission. I had 20 electroshock treatments and I regret very much my decision to have any of the treatments.

I had to retire from part-time work as a para-professional in a local high school and I doubt I will ever be able to work again. I spent 11 months in psychiatric hospitals and at home. I have forgotten how to read, could not concentrate on anything, felt very disappointed in life and still feel suicidal. I often don't remember people who speak to me, much of my past life is gone from my memory, I have cognitive thinking problems, fear being in any social situations, cannot spell, cannot remember factual information and live a rather hermit-like existence.

I believe I have permanent brain damage as a result of ECT and I do not think I will ever again be as intelligent a person as I was before the electroshock treatments. I feel betrayed by the doctors who persisted with the treatments when I obviously did not improve with the first 2 treatments. I also think that the profit motive to the treatments is a strong factor in doctors promoting and promising improvement to patients who are desperate to get relief from severe depressive illness. The administering psychiatrist profits and the anesthesiologists also profit charging \$150-\$200 a treatment, perhaps as many as 10 or 15 patients a day.

I do not personally know anyone who has improved from electroshock treatments and have heard of only one through my present therapist. Most people have to return for "maintenance" treatments as often as once a month.

I hope you will consider seriously keeping this dangerous, brain breaking treatment in the Class III category as it should be considered a dangerous, damaging treatment for mental illness, in my opinion.

Thank you.

Sincerely,

Mrs. Theresa G. Blumen

May 20, 1983

ADMINISTRATIVE
1983 JUN -2 PM 1:53

Dockets Management Branch
Food and Drug Administration
5600 Fishers Lane
Room 4-62
Rockville, Maryland 20857

Re: DOCKET NO. 82P-0316
RECLASSIFICATION of ELECTROCONVULSIVE THERAPY DEVICE

My personal experience as a patient, shock treatment is intrusive therapy. I saw my physician once when admitted. To my knowledge, I never saw or spoke to my doctor after entry into the hospital. Blue Cross, Blue Shield pays for a quick fix. I had no after-care followup in the community. The experience of going back to work was horrendous. I could not remember names of fellow employees, code numbers for the computer department was wiped out of my mind. Fortunately, my job was protected by the union regulations. The episode of a mental exhaustion happened on the job.

Before this hospitalization, I was attending business school for accounting. All that I learned was wiped out of my mind. My vocal studies were brought to an abrupt halt. My repertoire of music was wiped out of my mind. There has been much written about memory loss. Followups of each and every patient who have had shock treatment should be a matter of necessity. A Pet-Scan or Cat-Scan would be a necessary, cautionary followup procedure. Pretesting with the Cat or Pet-Scan to have a view of the brain before and after treatment.

PLEASE DO NOT CHANGE SHOCK FROM CLASS 111 to CLASS 11.

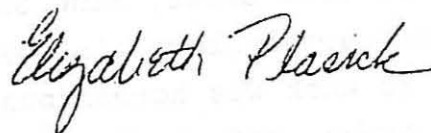
X-Rays indicate that I have a split spine. There are difficulties with sitting, lifting, standing, lying in one position is painful. I have had problems holding a job. Rehabilitation should be included after shock treatment. Currently I am counseling at a university for investigative search for causes for memory loss.

STATE HOSPITALS IN MICHIGAN DO NOT USE SHOCK. They claim
to be more progressive than the private hospitals.

There also has been abuse of shock treatment. Much has been
written about this.

Please send me the documented evidence that the FDA has on
the safety of shock.

Citizens for Action in Mental Health would like to be informed
of further action on this matter. Please inform for further hearings.



Elizabeth Plasick
Citizens for Action in
Mental Health
1019 Karendale Avenue
Portage, Michigan
49002

Enc: 1

November 2, 1990

Food and Drug Administration
Dockets Management Branch (HFA-305)
Room 4-62
5600 Fishers Lane
Rockville, MD 20857

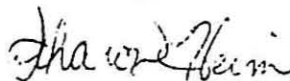
Re: Proposal to Reclassify the ECT Device, as published in the Federal Register of September 5, 1990

I am writing to urge the F.D.A. not to reclassify the ECT device, but to keep it in Class III where it has been since medical devices were so classified. This would ensure that performance standards are carried out and that consumers are protected from the harm that is a result of use of the ECT device.

I write from personal experience, which is why I feel very strongly about this issue. I was 18 years old, in my first year of college, when ECT was administered to me for treatment of depression. My doctor informed me that I would experience some short term memory loss, but reassured me that my memory would return to normal within six weeks following the end of the treatments. Upon discharge from the hospital, he pronounced me cured, and urged me to return to college. I had to drop out of school when I realized that I could not remember what I had studied before entering the hospital, and I was totally unable to absorb new information. I suffered for many months from a complete inability to concentrate, and was not able to even read a newspaper or magazine. I have been left with permanent memory loss of events that preceded the ECT by several months. I continue to have difficulty concentrating for extended periods of time, which I believe is a result of the ECT.

I believe that stringent regulations are necessary to protect patients from being easily pressured into consenting to ECT. In my case, there was no "informed consent" because I wasn't properly informed of the risks involved. Patients suffering from depression are especially vulnerable to believing that ECT will provide a quick cure. Doctors must be responsible to not use ECT as an easy solution in a difficult situation, at the risk of their patients. ECT does not provide a magic cure, and the price paid is too great. I therefore urge the F.D.A to not reclassify the device.

Sincerely,



Sharon Heim
1870 Beacon St. #5-3
Brookline, MA 02146

8-11-0316

102 W. 79th St.
Apt 20
NYC, NY 10024
Feb. 4, 1988

Dear Mr. Villforth,

Now 56 years old, I spent almost an entire lifetime to date 'shadow boxing' with some sort of built-in mystifying symptoms which not only I had but also my mother and one brother. This is known by the professional medical community by diagnosis, mostly spoken behind closed doors in the past, as a 'schizophrenic family' - i.e. more than one case of this syndrome of symptoms.

Today my mother is dead; the brother is dead also. However I myself set out on a journey from young adulthood to the present to find the answers to the veritable holocaust which struck our family, and I ~~needed it~~.

I spent 25 years, on and off, with psychiatry. These years, with the drugs and ECT, were horrendous. I can assure you that these modes of treatment cure nothing. In truth, these treatments, particularly the ECT, complicate the picture enormously. Because ill persons become dependent on drugs and are permanently damaged by ECT, myself included.

The receipt of ECT might better be described as the ending of life for most persons who receive same. The reason is that on top of either unsolved problems or untreated unknown illness etiology is superimposed permanent damage to the brain.

Thus along with either emotional disturbance and/or physical severe health problems, one goes home to cope with the reality that one's brain is no longer trustworthy. One reaches to the mind to seize some well known fact or skill and most often one gets back, in nerve response - a blank. Or one takes much longer to be able to draw back some recall. Or whole years are missing forever. Or areas of education and/or skills can be totally erased from the mind and memory. My natural 'gift' was that of a styles-playing actress. Any chance of having my own profession died with ECT as I no longer have 'near' memory. I know persons who have had just about every skill, knowledge or memory area destroyed - who had to learn to tie shoe laces again - to add the simple arithmetic to pay bills - who had lost the treasured ability to sing - who could no longer paint (this one was a magnificent artist) - who had to close a desk and go ~~over~~ ^{home permanently} as all of the professional knowledge was gone - and on and on.

Mr. Villforth, is this true or not: a human being is in essence the sum total of experiences in life, ^{as he existed in the world} skills, knowledge, acquaintances, friends? * When these can be destroyed instantly just by the flip of a button, what is left? Do you claim that this individual is really still left living as that individual - or, in essence did that individual truthfully have to be killed to some ~~to~~ degree in order to allegedly 'cure' to some degree? Is killing to cure really an answer the FDA can accept?

* One woman asked what the man who showed up to see her after ECT. It was her husband. She no longer knew her own husband.

After the nightmare of the 30 years post-ECT, I sat in a self help meeting and was astounded to hear a young woman sitting next to me testify to my exact type of experience with ECT 30 years earlier. ^{in the same hospital!} In her case, her budding career as a writer was totally destroyed. In essence, at 24 years of age, she now has no future whatsoever. (~~Ironically~~ ECT supposedly 'cures' depression - ^{when one discovers it is permanent} ~~no, it increases depression 300%~~)

To my horror, I suddenly realized that all of the damage and agony of post-ECT was still simply unknown or perhaps more accurately, unacknowledged three decades later by people who seem to want to battle to the death to continue this dark-ages treatment.

My twenty-five years with psychiatry finally caused me to conclude that psychiatry isn't even an authentic specialty of Medicine. It is rather a fraud. It is an attempt to convince society and Medicine that there is such a thing as the possibility of creating a hybrid specialty of 'psychology-~~and~~-organic medicine.' Psychology is not a medical field. It is a field of academia - extremely subjective, speculative and untestable. It has no place in Medicine whatsoever.

Psychiatry is in Medicine to meet specialized needs at this time for society. But being needed at a specific time by society does not make the so-called specialty of medicine a valid and authentic specialty of medicine. *It is not an authentic Specialty.*

This is why psychiatry needs locks, bars, straitjackets, prisons, and the harsh damaging treatments it inflicts - because in truth it is a hoax as a specialty of medicine, *I am firmly convinced.*

I feel that since you were given the responsibility to make medical treatment safe for patients that you should meet that public trust. You originally found ECT a highly hazardous treatment from your study of it. You should therefore meet the responsibility to keep this alleged 'treatment' in Class III.

The last 12 years: The last 12 years I discovered orthonomolecular medicine. I feel it was just in time as I am firmly convinced I was approaching death after 25 years of psychiatry.

The orthonomolecular method immediately began reversing my hellish symptoms and I have been on the upward path ever since. However what I lived for - the theater - will never be - as ECT permanently damaged my memory.

Yours truly,

June Barrett

Jan 2 1988

Dear Mr. Villforth:

I understand that the FDA is undecided whether to take back the notice of intent regarding Electric Shock Treatments.

In 1968 I had 19 shock treatments. We found out later that they were probably unnecessary and that I had severe thyroid and female hormone deficiencies.

Needless to say, the Electric Shocks didn't help my hormone deficiencies.

They did wreck my life however! I suffer severe memory loss which has never returned. It comes 8 to 10 years!

I also have a very deep inability to learn and comprehend things and this has led to problems with my own self understanding. It also has affected my relations with my own family and other people too.

Mr Villforth, I urge you NOT to make E.C.T. such an easy to use treatment.

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My husband once asked a doctor who praised E.C.T. why he didn't

try 6 or 8 himself and then try
to work his medical practice. He
couldn't reply.

Maybe they are useful to some
people but all of the 8 or 10 people
that I know that had them say that
they didn't know what they were
getting into and regretted having
them.

Thank you for your consideration.

Sincerely

Dorothy Cumette

X

1137 Miramar Plaza, #41
Sunnyvale, CA 94086
November 28, 1987

Frank E. Young, M.D., Ph.D.
Commissioner, Food & Drug Administration
C/O Dockets Management Branch (HFA-305)
5600 Fishers Lane (Room 4-62)
Rockville, MD 20857

RE: Docket No. 82P-0316 (ECT)

Dear Sir:

Please add my name to those strongly urging you to thoroughly investigate the safety of Electroconvulsive Therapy.

From personal experience, I can vouch for the fact that ECT does permanent damage which inhibits the brain's facility for memory (particularly short-term), and concentration.

I know many others who have had ECT and there is not one who is positive about it. It is hard to imagine how such a barbaric form of "treatment" has been allowed to continue!

Sincerely,

Edith G. Harris

Edith G. Harris

EGH/egh

ADONIS 0000 0000 0000 0000
1983 APR 28 AM 10:55

5300 Konrad Dr.
Temple Hills, Md.
20748
April 21, 1983

Dockets Management Branch
Food and Drug Administration
5600 Fishers Lane Room 4-62
Rockville, MD 20857

Docket No. 82P-0316

Gentlemen:

This is to inform you of my opposition to the reclassification of ECT devices from the high risk category of Class III to the low risk Class II of medical devices.

I know of no equipment devised for use on humans that is more profoundly damaging to the individual's brain function.

From personal experience I can verify that the 20 ECT's I had, left permanent brain damage in the form of amnesia. This covered the preceding years before the ECT's and still exists. In addition, I have suffered impaired retention for 24 years. Thirdly, my college education has gone down the drain as I'm unable to perform my duties as a teacher since 12 years past.

May I add, I did not sign, nor did I want the horrible hell the trauma of those shocks left on me until this day when I still suffer from nightmares of them. Finally, four friends and two relatives who also had ECT are now dead, one having died during the seizure.

Betty C. Scoleri

Betty C. Scoleri

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OF THE
CONGRESS
WASHINGTON, D. C. 20540

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THE
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OF THE
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WASHINGTON, D. C. 20540

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X John Villforth, Director (HFZ-1)
Center for Devices and Radiological Health
Food and Drug Administration
5600 Fishers Lane
Rockville, Md 20857

Dear Sir,

Being one of those persons who has permanent "brain damage", apparently the result of ECT, I respectfully request and urge you to insist that the FDA rescind their "notice of intent" of 1983 to reclassify the ECT device.

How is it possible for APA to consider ECT as being safe when they conduct no follow-up of the long term mental and emotional conditions of the many persons who were "treated" by use of ECT without truthfully informed consent?

Thank you for your consideration.

Sincerely

A

C00325-

Branson L. Morris Jr.
107 Shelby St.
Auburndale, Fl. 33823

82P-0316

X
Joy Rose
414 S. Olive St.,
Media, Pa., 19063

January 27, 1988

John Villforth, Director (HFZ-1)
Center for Devices and Radiological Health
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20857

Dear Mr. Villforth:

As one who has suffered the results of shock "treatment",
(There are parts of my memory that are forever lost to me), I
urge you to reconsider rescinding the "Notice of intent" to
reclassify ECT devices.

As I testified before the American Psychiatric Association:
"If shock "therapy" is so safe and so affective, then why is the
psychiatric association fighting so hard to get the FDA from
testing it for safety?"

I have offered my brain to be cat-scanned to see if I am
right about the permanent damage I feel I have sustained. This
~~has~~ been rejected because of lack of funds. I am only hoping that
you will ^{let it} be classified in Class III until such time that there
~~are~~ funds to test it for safety. I only hope to keep others from
suffering as I have.

Very truly Yours,

Joy Rose

Joy Rose

82P-0316

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C06311

85 Douglas Road
Glastonbury, Ct. 06033
January 20, 1987

Mr. John Villforth, Director
Center for Devices and
Radiological Health
Food and Drug Administration
5600 Fishers Lane
Rockville, Maryland 20857

Re: ECT CLASSIFICATION

Dear Mr. Villforth:

I implore you, please rescind the 1983 "notice of intent" to reclassify ECT devices to Class II status. The American Psychiatric Association is asking you to gamble with the lives of psychiatric patients by reclassifying the devices without actual scientific evidence that they cause no permanent harm.

I am one of many ECT patients who cannot help but suspect that ECT caused brain damage and until there are longitudinal and cross sectional studies successfully carried out which prove otherwise, my opinion will remain the same. I would like to think that someday, thanks to the FDA, the truth about ECT will be known, whatever that truth may ultimately be. I would like to think that my brief description of my experience with ECT will aid you and your colleagues in your pursuit of a truly informed decision. I know that many of us who joined the Committee for Truth in Psychiatry are writing to you; I hope you will realize that all of us, past and future ECT patients, are depending on you and your associates.

Mr. Villforth, I can write a pretty good letter, I think, but in this case it's what I can't do that counts. I am constantly reminded of what I can't do...although I could do it once. And what is "it"? I can't remember new information with the ease I could before ECT. Distractions and interruptions seriously interfere with information retention...any new bit of information may "cancel out" the bit that preceded it. My auditory and visual memory seem to function episodically...enough so that I know they exist and remember how well they functioned before ECT.

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82P-0316

How have these deficits, which developed immediately after ECT, affected my life?

1. When I returned to my 6th grade teaching job after ECT I could not remember how to teach, I could not remember my own or others' instructions/directions/information, I could not function with the interruptions that are a unavoidable part of a teacher's life. Therefore, in 1968, 5 months after ECT, I attempted suicide. Rehospitalization was for 8 months.
2. For two and a half years I worked in a kitchen. The loss in income was dramatic but worse was the total loss of confidence and the perception that I was a complete failure.
3. When I dared to take a college course in the hopes that I could rebuild my financial security (seven years later) I had to memorize information because even multiple readings of the same material yielded next to nothing recalled. I programmed my course material on file cards, sentence by sentence, and quizzed myself - studying about 20 hours a week minimum, often more, while working full time as a secretary.
4. In September of 1987, after taking one course a semester for 4 years at a school of social work, I matriculated and started full time. However, because there no longer was time to memorize information, because the information was complex and often theoretical, and because I found it hard to remember instructions on my field placement - I finished the semester (after withdrawing from 3 of four courses) and withdrew from school. I am very fortunate that I survived the subsequent depression.
5. Today I interviewed for 3 jobs. At one the employer asked, "Why aren't you teaching? Teachers make such good salaries." Indeed why, why am I not making the \$40,000 I would be making if I'd remained in teaching like my roommate who started the same year I did in the very same school system. Why am I instead praying that I'll find a job that pays me at least what I earned in my previous job - \$16,000. Why am I likely to settle for less if it will make few demands on my memory. I'm sure I need not answer "why."

I survived the "psychotic depression" that put me in the hospital in 1968. However, I don't know how I would have survived the aftereffects of the ECT if I had not had endless love and support from family and friends. I say aftereffects of ECT because I can't imagine what else could have had such a disastrous effect on my ability to learn and to remember. I only know I had a high "B" average in college, I remembered ideas better than facts, I read material rather than memorize, I was not a slave to my studies. One year six months later functioning like that was just a bitter memory.

Did it have to be like this for me? Was ECT necessary? Was it harmful? Were there alternatives to ECT that could have been used, or tested, first (besides 3 weeks of drug therapy)?

Did it have to be like this for others who have similar stories to relate?

If ECT is harmful, can we save some people from unnecessary suffering by determining that it is so and using it when truly there is no alternative.

And if it must be used in spite of its damaging effects, can we not develop cognitive retraining programs to help people adapt to their new deficits.

I know in the first page of this letter I referred to my "brief" description of my experience. Is this brief? It is for someone who feels she struggles with the consequences of ECT still -- after 20 years.

This is not a request that you begin an investigation of ECT devices immediately. It is an earnest appeal that you rescind the "notice of intent." Those of us who have "come out" to protest a reclassification may be just the tip of an iceberg. I know three people whose lives changed after ECT, who suffered considerable career "dislocation," but you will never hear from them. Who else won't the FDA hear from?

I do sincerely appreciate the time you or your staff have spent reading this letter.

Sincerely,

Pam Maccabee

Pam Maccabee

cc: American Psychiatric
Association

X

Truth In Psychiatry

Marjorie E. Faeder
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January 21, 1988

John Villforth, Director (HFZ-1)
Center for Devices and Radiological Health
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20857

Dear Mr. Villforth:

I am requesting that as a senior official of the FDA you recommend that the "Notice of Intent" to reclassify the Electroconvulsive Treatment (ECT) device be rescinded. I am also requesting that the device remain in Class III until a complete and unbiased investigation has been made as legally required.

I commend the FDA for the original Class III classification, and my feeling is that the American Psychiatric Association has been unreasonable to ask your agency to vouch for ECT's unproved safety without a proper investigation.

Our small Truth in Psychiatry group, each member of which has been brain damaged in varying degrees, is only a very small sample of the hundreds and thousands of patients who have suffered longterm memory loss as well as other side effects of ECT.

My feeling is that our "anecdotal complaints," as snidely referred to by psychiatrists at FDA hearings, are not given enough consideration, and that because we are considered "ex-mental patients" we are not credible. One psychiatrist wrote patronizingly in The Washington Post Magazine that we do not sound like brain damaged people. He saw only what he wanted to see, unwilling to dig deeper to verify our losses.

It took me five years of hard work and frustration to restore my reading comprehension to the college level. And I had been a Reading Specialist. For some twenty years I could not play the piano; only in the last three months has some of that skill started to return. I doubt that I'll ever reach the level I had before I was subjected to ECT.

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It has taken me the past twenty years, and will take me the rest of my life to approximate the education I lost. My career as an Intelligence Officer for the Federal Government was lost forever.

The effects of ECT not only ruined my life, but it nearly destroyed my family and my marriage. I find that a large percentage of patients suffer a divorce because they no longer function well. Many others I have dealt with have had to be supported by public means, avoidable burdens to local, state, and the Federal governments.

Our patient group is not seeking a ban on ECT because this is a democracy and there are those who say that it benefitted them, that in spite of their recognized faulty memories they would have it again. We have such members in our group. However, we do advocate that there should be a proper and truthful consent so that the patient really knows the potential danger.

While the immediate goals of our Committee are to rescind the "Notice of Intent" and to require a meaningful consent procedure, my personal belief is that an investigation is in order to prove that ECT is indeed beneficial and not brain damaging. How can I feel differently when that so-called therapy has wrecked a major portion of my life?

Respectfully yours,

Marjorie E. Faeder
National Representative C711

Marjorie E. (Mrs Gustav S.) Faeder