

# Electroshock: scientific, ethical, and political issues \*

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**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

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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 over-represented. 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) (p. 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” (p. 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.

Psychiatrists who prescribe or administer ECT almost never seriously consider the memory deficits of their patients. In case after case that I have evaluated for clinical or forensic purposes, I have been the first doctor to take the symptoms seriously, let alone to take a complete inventory of memory losses and ongoing mental difficulties. I have previously outlined a method for evaluating memory deficits from ECT [32].

#### *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  $2\frac{1}{2}$  to  $3\frac{1}{2}$  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., [121, 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 crossover 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 imperceptible 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 0.1 s 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. . .” (p. 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 Devanand<sup>1</sup> 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

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<sup>1</sup>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.

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 nondominant (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 McEachern [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" (p. 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" (p. 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. 134).

#### 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 re-define 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 McDonald [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 [124] 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 Rogelio Cattán [54], while purporting to support ECT, also demonstrated devastating results.

Cattán'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 as 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, Laretta 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.<sup>2</sup>

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.

<sup>2</sup>To contact the international psychiatric survivor movement: David Oaks, Publisher, *Dendron*, P.O. Box 11284, Eugene, Oregon, 97440. E-mail: dendron@efn.org.

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.<sup>3</sup> 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.

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<sup>3</sup>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.

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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