The Journal of Mind and Behavior
Winter and Spring 1994, Volume 15, Numbers 1 and 2
Pages 177–198
ISSN 0271–0137
ISBN 0-930195-07-8

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

Douglas G. Cameron

World Association of Electroshock Survivors

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

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

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

Requests for reprints should be sent to Douglas G. Cameron, World Association of Electroshock Survivors, P.O. Box 343, San Marcos, Texas 78667.

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 Unpublished conversations with many of Janis' patients six months or one year later (Davies, Detre, and Egger, 1971) led him to conclude the memory loss was long-term,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

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

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

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

---

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

[2]Only Squire, Slater, and Miller (1981, p. 95) have repeated the Janis prospective study. Even after two years, and even with reminder cues, 50% of the ECT recipients in this study still could not recall specific autobiographical events spontaneously recalled before ECT. This does not preclude the possibility that autobiographical events which could be "remembered" after two years, might simply have been re-learned rather than recalled.

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

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

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

---

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

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

Misinformation from the ECT Manufacturers

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

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

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

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

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

Misinformation from the American Psychiatric Association

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

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

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

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

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

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

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

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

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

The Myth of Convulsive Therapy

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

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

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

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

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

This "non-convulsive therapy" failed to elicit the "therapeutic" effect (Impastato, 1952). However, in his quest to determine the ideal electrical dosage, Wilcox discovered that the strength of an electrically induced grand mal seizure did not depend upon any more electricity than that required to induce the seizure (Alexander, 1953, p. 64; Sulzbach, Tillotson, Guillemin, and Sutherland, 1943, p. 521). This meant that "adequate" convulsions could be induced with much lower dosages of electricity than had previously been used, and that the Cerletti–Bini devices were utilizing much more electricity than needed to induce such convulsions (Friedman, 1942, p. 218). Cerletti and Bini's device, then, was not an *electroconvulsive* device, but an electroshock device.

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

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

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

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

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

---

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

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

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

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

First Brief Pulse

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

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

Brief Pulse Fails

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

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

[9]Eventually, with the introduction of informed consent, all unmodified EST (without exception terrifying to recipients) was replaced with anesthetized EST. Fear associated even with modified EST continues to baffle practitioners today (Fox, 1993).

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

As early as 1948 then, it was known that, *even with potent seizures*, the "anti-depressant" effect at low electrical dosages was simply not satisfactory.[10] Liberson (1946, p. 755) must have understood that electricity was the true "therapeutic" agent, but rather than publish findings showing von Meduna's convulsion theory weakened considerably, he focused instead on making his BP ECT device "work." After calling for more and more treatments, he recommended longer doses of BP ECT (Liberson, 1945b), eventually marketing a machine which allowed the current to flow between the temples for a full five seconds (compared to between 0.5 and one second previously) [see Weiner, 1988, p. 59, Figure 6]. The Liberson device could no longer be called an ECT, but was now an EST device. Next, although Liberson had already increased the wave length duration from 0.3 to between 0.5 and one millisecond[11] (Weiner, 1988, p. 57), his newer BP model offered adjustable wave lengths from between 1.5 to two milliseconds. The current was eventually stepped up to between 200 and 300 milliamps and, finally, Liberson returned to AC — doubling the original power.

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

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

---

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

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

The Wilcox–Reiter Device

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

Even so, by 1953, it was apparent that the Wilcox–Reiter ECT "electrostimulators" also began to decline in popularity and could not compete with the more powerful Cerletti–Bini style American EST machines (i.e., Radha, Lectra, and Medcraft). In December 1956, at the Second Divisional Meeting of the APA in Montreal, Canada, psychiatrist David Impastato[12] and his colleagues made this announcement:

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

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

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

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

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

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

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

The Case for Consumer Misinformation

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

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

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

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

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

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

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

This compensation is accomplished in the following ways:

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

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

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

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

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

Thus modern BP devices are made to *equal* the charge[14] of SW devices in every consideration with respect to percent of energy utilized. In addition, they *surpass* the "older" SW machines in energy output (joules), or actual power emitted.[15] The following electrical features account for this increase:

---

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

[15]Using a straight-forward mathematical formula, the power of the new brief pulse devices can be verified by calculating joules (or the more familiar "watts" as in a lightbulb), the measure of actual energy emitted (voltage is potential energy or power). All four companies (e.g., MECTA, 1993, p. 13) do list their devices as 100 joule maximums in all 4 brochures, but the manufacturers' calculations are based on a typical resistance of 220 ohms (ohms are the measure of resistance, here, of the skull and brain, to current flow). However, the true maximum joules or watts for all modern day BP devices is much higher than the estimate reported by

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

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

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

Conclusion

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

---

the manufacturers. For SW devices, the formula is: joules = volts x current x duration, or joules = current squared x impedance x duration. For BP devices, the formula is : joules = volts x current x (hz x 2) x wave length x duration, or joules = current squared x impedance x (hz x 2) x wave length x duration. All four manufacturers utilize the latter in lieu of the former formulas, deriving the 100 joule maximums for their BP machines. Utilizing the former formulas, however, which give us non-theoretical amounts, we find that the Thymatron DG BP is capable of emitting 250 joules or watts of electricity; the MECTA SR/JR BP models, 256 joules; the Medcraft B–25 BP, 273 joules; and the Elcot device even more. Compare these energy emissions with the following typical analogy: the standard SW device can light up a 60 watt light bulb for up to one second. (Modern SW devices can light up a 100 watt light bulb for up to one second.) Modern BP devices can light up the same 60 watt light bulb for up to four seconds.

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

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

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

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

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

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

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

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

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

---

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

[17]This is best exemplified through unilateral ECT. Originally utilized by Wilcox and Friedman to induce the most minimal stimulus threshold seizures possible (Alexander, 1953, p. 62; Liberson, 1948, p. 32), unilateral ECT is used by modern manufacturers to induce the highest electrical dosages possible (Abrams and Swartz, 1988, pp. 28–29) in order to achieve efficacy.

References

Abrams, R., and Swartz, C. (1988). *ECT instruction manual.* Lake Bluff, Illinois: Somatics, Inc.

Abrams, R., and Swartz, C. (1990). *The technique of ECT* [video]. Lake Bluff, Illinois: Somatics, Inc.

Aird, R.B., Strait, L.A., and Pace, J.W. (1956). Current pathway and neurophysiologic effects of electrically induced convulsions. *Journal of Nervous and Mental Disease, 123,* 505–512.

Alexander, L. (1953). *Treatment of mental disorder.* Philadelphia: W.B. Saunders.

Alexander, L. (1955). Modified electroconvulsive therapy with unidirectional current. *Diseases of the Nervous System, 16,* 1–4.

Alexander, L., and Löwenbach, H. (1944). Experimental studies on electro-shock treatment. *Journal of Neuropathology and Experimental Neurology, 3,* 139–171.

American Psychiatric Association. (1990). *The practice of electroconvulsive therapy: Recommendations for treatment, training, and privileging. Task Force on ECT.* Washington, D.C.: American Psychiatric Association.

American Psychiatric Association. (1992). *American Psychiatric Association fact sheet: Electroconvulsive therapy (ECT)* [three page flyer]. Washington, D.C.: American Psychiatric Association.

Aubrey, K. vs Johns Hopkins Hospital. (1991). Health Claims Arbitration Office, Dec. 3, 1991, Rockville Maryland, p. 140.

Bender, L. (1947). One hundred cases of childhood schizophrenia treated with electric shock. *Transactions of the American Neurological Association, 762,* 165–168.

Breggin, P.R. (1979). *Electroshock: Its brain disabling effects.* New York: Springer.

Breggin, P.R. (1990). Brain damage, dementia and persistent cognitive dysfunction associated with neuroleptic drugs. *Journal of Mind and Behavior, 11,* 425–463.

Breggin, P.R. (1991). *Toxic psychiatry: Why therapy, empathy, and love must replace the drugs, electroshock, and biochemical theories of the "new psychiatry."* New York: St. Martin's Press.

Breggin, P.R. (1992). The return of ECT. *Readings: A Journal of the American Orthopsychiatric Association, 7,* 12–17.

Brody, M.P. (1944). Prolonged memory defects following electrotherapy. *Journal of Mental Science, 90,* 777–779.

Brunschwig, L., Strain J., and Bidder, T.G. (1971). Issues in the assessment of post-ECT memory changes. *British Journal of Psychiatry, 119,* 73–74.

Cameron, D.G. (1979). *How to survive being committed to a mental hospital.* New York: Vantage Press.

Cameron, D.G. (1991). (Ed.). *Electroshock treatment: 100 cases of permanent memory loss, Vol. 1.* Unpublished manuscript, World Association of Electroshock Survivors, P.O. Box 343, San Marcos, Texas 78667.

Cameron, D.G. (1994). *Passage of the strictest ECT legislation in America: The story behind it and how it was accomplished.* Unpublished manuscript, World Association of Electroshock Survivors, P.O. Box 343, San Marcos, Texas 78667.

Chabasinski, T. (1978). The other half. In L. Frank (Ed.), *The history of shock treatment* (pp. 26–27). San Francisco: Frank.

Coffey, C.E. (1993). Structural brain imaging and ECT. In C.E. Coffee (Ed.), *The clinical science of electroconvulsive therapy* (pp. 73–92). Washington, D.C.: American Psychiatric Press.

Cohen, D. (1988). Social work and psychotropic drug treatments. *Social Service Review, 62,* 576–599.

Cook, L.C. (1940). Has fear any therapeutic significance in convulsion? *Journal of Mental Science, 40,* 414.

Corsellis, J.M., and Meyer, A. (1954). Histological changes in the brain after uncomplicated electroconvulsant treatments. *Journal of Mental Science, 100,* 375–383.

Cronholm, B., and Ottosson, J.O. (1963). Ultrabrief stimulus techniques in ECT. II. Comparative studies of therapeutic effects and memory disturbances in treatment of endogenous depression with the Elther ES apparatus and Siemens konvulsator III. *Journal of Nervous and Mental Diseases, 137,* 268–276.

Daniel, W.F., Weiner, R.D., and Crovitz, H.F. (1983). Autobiographical amnesia with ECT: An analysis of the role of stimulus waveform, electrode placement, stimulus energy, and seizure length. *Biological Psychiatry, 18*, 121–126.

Deakin, J.F.W. (1983). Antidepressant effects of electroconvulsive therapy: Current or seizure? *British Medical Journal, 286*, 1083.

Davies, R.K., Detre T.P., and Egger, M.D. (1971). Electroconvulsive therapy instruments: Should they be reevaluated? *Archives of General Psychiatry, 25*, 97–99.

Delmas–Marsalet, P.L. (1942). L'électro-choc par courant continu. *Comptes rendus des Séances. Société de Biologie et de ses Filiales et Associées (Paris), 136*, 551–553.

Dunn, A., Giuditta A., Wilson, J.E., and Glassman, E. (1974). The effect of electroshock on brain RNA and protein synthesis and its possible relationship to behavioral effects. In M. Fink, S. Kety, and J. McGaugh (Eds.), *Psychobiology of convulsive therapy* (pp. 185–197). New York: Wiley.

Ebaugh, F., Barnacle, C., and Neuburger, K. (1943). Fatalities following electroconvulsive therapy. *Archives of Neurological Psychiatry, 49*, 107–117.

Echlin, F.A. (1942). Vasospasm and forced cerebral ischemia. *Archives of Neurological Psychiatry, 47*, 77–96.

Essman, W.B. (1968). Electroshock-induced retrograde amnesia in seizure-protracted mice. *Psychological Reports, 22*, 929–935.

Fink, M. (1979). *Convulsive therapy: Theory and practice*. New York: Raven Press.

Fink, M. (1986). *Informed ECT for health professionals* [video]. Lake Bluff, Illinois: Somatics, Inc.

Foderaro, L. (1993, July 19). With reforms in treatment, shock therapy loses shock. *New York Times*, p. A16.

Food and Drug Administration. (1990, September 5). Neurological devices: Proposed rule to reclassify the electroconvulsive therapy device for use in treating severe depression. *Federal Register*, Part III, 21 CFR Part 882.

Fox, H.A. (1993). Patients' fear of and objection to electroconvulsive therapy. *Hospital and Community Psychiatry, 44*, 357–360.

Frank, L.R. (1978). (Ed.). *The history of shock treatment*. San Francisco: Frank.

Freeman, C.P.L., and Kendell, R.E. (1980). ECT: I. Patients' experiences and attitudes. *British Journal of Psychiatry, 137*, 8–16.

Friedman, E. (1942). Unidirectional electrostimulated convulsive therapy. *American Journal of Psychiatry, 99*, 218–223.

Friedman, E., Wilcox, P.H., and Reiter, R.M. (1942). Electrostimulated convulsive doses in intact humans by means of unidirectional currents. *Journal of Nervous and Mental Disease, 96*, 56–63.

Geyer, H. (1960). Evaluation of hydroxyzine prior to electroshock therapy. *Diseases of the Nervous System, 21*, 1–4.

Glenn, M., and Weiner, R. (1983). *Electroconvulsive therapy, a programmed text* (pp. 33–34). Washington, D.C.: American Psychiatric Press.

Gordon, D. (1980). Ruptured spleen in electric convulsion therapy. *British Medical Journal, 280*, 1426.

Gordon, D. (1982). Electroconvulsive therapy with minimal hazard. *British Journal of Psychiatry, 141*, 12–18.

Grunhaus, L. (1988). *Electroconvulsive therapy: E.C.T. The treatment, the questions, the answers* [video]. Ann Arbor, Michigan: University of Michigan Medical Center. [Distributed through Lake Oswego, Oregon: MECTA Corporation].

Hartelius, H. (1952). Cerebral changes following electrically induced convulsions: An experimental study on cats. *Acta Psychiatrica and Neurologica Scandinavica, 77*, 1–128.

Heilbrunn, G., and Weil, A. (1942). Pathologic changes in the central nervous system in experimental electric shock. *Archives of Neurological Psychiatry, 47*, 918–927.

Hirsch, C.S., and Martin, D.L. (1971). Unexpected death in young epileptics. *Neurology, 21*, 682–690.

Hudson, W. (1978). NAPA battles shock. In L. Frank (Ed.), *The history of shock treatment* (pp. 146–152). San Francisco: Frank.

Impastato, D. (1952). Electrically produced unilateral convulsions. *Diseases of the Nervous System, 13*, pages unavailable.

Impastato, D. (1957). Prevention of fatalities in electroshock therapy. *Diseases of the Nervous System, 18,* 34–75.
Impastato, D., Berg, S., and Gabriel, A.R. (1957). The Molac–II — an alternating current electroshock therapy machine incorporating a new principle. *Journal of Nervous and Mental Disease, 125,* 380–384.
Impastato, D., Frosh, J., and Robertiello, R. (1951). Improved electro-convulsive therapy with low amperage unidirectional currents. *Diseases of the Nervous System, 12,* pages unavailable.
Janis, I. (1950). Psychologic effects of electric convulsive treatments (I, II, III, post treatment amnesia). *The Journal of Nervous and Mental Disease, 3,* 360–397; 469–489.
Kellner, C.H. (1994, February 2). *Electroshock with Susan Spencer* [television interview]. In C. Lasiewicz [Producer], *48 Hours*. New York: CBS.
Liberson, W.T. (1945a). Study of word association processes, part II: Reactions to "average emotional" and "average neutral" words in normal and abnormal populations. Effect of electric convulsive therapy. *Digest of Neurology and Psychiatry, 13,* pages unknown.
Liberson, W.T. (1945b). Time factors in electric convulsive therapy. *Yale Journal of Biology and Medicine, 17,* 571–578.
Liberson, W.T. (1946). Physiological basis of electric convulsive therapy. *Connecticut State Medical Journal, 10,* 754–756.
Liberson, W.T. (1948). Brief stimulus therapy. *American Journal of Psychiatry, 105,* 28–39.
Liberson, W.T. (1949). Review of psychiatric progress, 1948. Electroencephalography. *American Journal of Psychiatry, 105,* 503–505.
Liberson, W.T., and Wilcox, P.H. (1945). Comparison of "brief pulse" technique with Friedman–Wilcox–Reiter technique. *Digest of Neurology and Psychiatry, 8,* 292–302.
Maletzky, B. (1987). *Dual channel — JR/SR models* [video]. Lake Oswego, Oregon: MECTA Corporation.
Malitz, S., Sackheim, H.A., and Decina, P. (1979). ECT in the clinical treatment of major affective disorders: Clinical and basic research issues. *Psychiatric Journal of the University of Ottawa, 7,* 126–134.
McGaugh, J.L., and Alpern, H.P. (1966). Effects of electroshock on memory: Amnesia without convulsions. *Science, 152,* 665–666.
McKegney, F.P., and Panzetta, A.F. (1963). An unusual fatal outcome of electroconvulsive therapy. *American Journal of Psychiatry, 120,* 398–400.
MECTA Corporation. (1987). *Health information network for hospitals and professionals* [video]. Lake Oswego, Oregon: MECTA Corporation.
MECTA Corporation. (1993). *MECTA Instruction Manual, SR and JR Models*. Lake Oswego, Oregon: MECTA Corporation.
Medcraft Corporation. (1984). *Medcraft B24III, Electroconvulsive Therapy Instruement* [flyer]. Darien, Connecticut: Medcraft Corporation.
Mowbray, R.M. (1959). Historical aspects of electric convulsant therapy. *Scottish Medical Journal, 4,* 375.
Page, L.G.M., and Russell, R.J. (1948). Intensified electrical convulsion therapy in the treatment of mental disorder. *Lancet, 1,* 597–598.
Proctor, L.D., and Goodwin, B.A. (1943). Comparative electroencephalo-graphic observations following electroshock therapy using raw 60 cycle alternating and unidirectional fluctuating current EEG. *American Journal of Psychiatry,* 99, 525–530.
Quandt, J., and Sommer, H. (1966). Zur frage der hirngewebsschadigungen nach electrischer krampfbehandlung. *Zeitschrift für Neurologie und Psychiatrie, 34,* 513.
Reed, K. (1988). Electroconvulsive therapy: A clinical discussion. *Psychiatric Medicine,* 6, 29.
Robie, T. (1955). Safe electric treatment of melancholia. *The Journal of the Medical Society of New Jersey, 52,* 82–87.
Robin, A., and De Tissera, S. (1982). A double blind controlled comparison of the therapeutic effects of low and high energy electro-convulsive therapies. *British Journal of Psychiatry, 141,* 357–366.
Sackheim, H.A. (1987). Seizure threshold in electroconvulsive therapy. *Archives of General Psychiatry, 44,* 355–360.
Sackheim, H.A. (1991). Are ECT devices underpowered? *Convulsive Therapy, 1,* 233–236.

Sackheim, H.A. (1992). *An adequate treatment* [audio tape]. (Lecture delivered in April, Mariott Marquis Hotel, New York City, Advanced Clinical Issues in ECT, The Certificate Course.)

Sackheim, H.A., Decina, P., Prohovnik, I., Portnoy S., Kanzler, M., and Malitz, S. (1986). Dosage, seizure threshold, and the antidepressant efficacy of electroconvulsive therapy. In S. Malitz and H.A. Sackheim (Eds.), *Electroconvulsive therapy: Clinical and basic research* (pp. 398–410). New York: New York Academy of Sciences, .

Sakel, M. (1956). Sakel shock treatment. In A. Sackler (Ed.), *The great physiodynamic therapies in psychiatry: An historical perspective* (pp. 13–14). New York: Hoeber–Harper.

Salzman, L. (1947). An evaluation of shock therapy. *American Journal of Psychiatry, 103*, 676.

Small, I.F. (1974). Inhalant convulsive therapy. In M. Fink, S. Kety, and J.L. McGaugh (Eds.), *Psychobiology of convulsive therapy* (pp. 65–77). New York: Wiley.

Somatics, Inc. (1993). *Caution* [flyer]. Lake Bluff, Illinois: Somatics, Inc.

Squire, L. (1986). Memory functions as affected by electroconvulsive therapy. *Annals of the New York Academy of Sciences, 462*, 307–313.

Squire, L.R., and Chace, P.M. (1975). Memory functions six to nine months after electroconvulsive therapy. *Archives of General Psychiatry, 32*, 1557–1564.

Squire, L., and Slater, P.C. (1983). Electroconvulsive therapy and complaints of memory dysfunction: A prospective three-year follow-up study. *British Journal of Psychiatry, 142*, 1–8.

Squire, L., Slater, P.C., and Miller, P.L. (1981). Retrograde amnesia and bilateral electroconvulsive therapy: Long-term follow-up. *Archives of General Psychiatry, 38*, 89–95.

Squire, L., and Zouzounis, J.A. (1986). ECT and memory: Brief pulse versus sine wave. *American Journal of Psychiatry, 143*, 596–601.

Sulzbach, W., Tillotson, K.J., Guillemin, V., and Sutherland, G.F. (1943). A consideration of some experiences with electric shock treatment in mental diseases, with special regard to various psychosomatic phenomena and to certain electrotechnical factors. *American Journal of Psychiatry, 99*, 519–524.

Sutherland, J.M., Tait, H., and Eadie, M.J. (1974). *The epilepsies*. London: Churchill Livingston.

Texas Department of Mental Health and Mental Retardation. (1993). Texas informed consent form for ECT requiring a signature for each individual treatment. *Disclosure and Consent for Electroconvulsive Therapy*. Austin, Texas: Texas Department of Mental Health and Mental Retardation.

Texas Medical Disclosure Panel. (1993). Informed consent, medical treatment and surgical procedure established by the Texas Medical Disclosure Panel, 601.1; *Procedures Requiring Full Disclosure* (List A), Section 13; *Nervous System Treatments and Procedures*.

von Meduna, L. (1938). General discussion of the cardiazol therapy. *American Journal of Psychiatry, 94*, 46.

Weiner, R.D. (1988). The first ECT devices. In M. Fink (Ed.), *Convulsive therapy* (pp. 50–61). New York: Raven Press, Ltd.

Weiner, R.D., Rogers, H.J., and Davidson, J.R.T. (1986a). Effects of stimulus parameters on cognitive side effects, in electroconvulsive therapy. In S. Malitz and H.A. Sackheim (Eds.), *ECT: Clinical and basic research issues* (pp. 315–325). New York: Annals of the New York Academy of Sciences.

Weiner, R.D., Rogers, H.J., and Davidson, J.R.T. (1986b). Effects of ECT upon brain electrical activity, in electroconvulsive therapy. In S. Malitz and H.A. Sackheim (Eds.), *ECT: Clinical and basic research issues* (pp. 270–281). New York: Annals of the New York Academy of Sciences.

Wilcox, P.H. (1946). Brain facilitation, not brain destruction, the aim in electroshock therapy. *Diseases of the Nervous System, 7*, 201–204.

Wilcox, P.H. (1972). Electrostimulation for promoting brain reorganization. *Diseases of the Nervous System, 33*, 326–327.

Will, O.A., Rehfeldt, F.L., and Newmann, M.H. (1948). A fatality in electroshock therapy: Report of a case and review of certain previously described cases. *Journal of Nervous and Mental Disease, 107*, 105–126.