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**Disabling the Brain with
Electroshock**

Electroconvulsive therapy (ECT) or electroshock therapy (EST) are synonyms for a psychiatric treatment in which electricity is applied to the head and passed through the brain to produce a *grand mal* or major convulsion. The seizure brought about by the electric stimulus closely resembles, but is more rigorous or strenuous than that found in idiopathic epilepsy or in epilepsy following a wide variety of insults to the brain.

The treatment was invented by Bini and Cerletti in Italy in 1938, and reached the United States in 1940. It won quick acceptance throughout the world, and by the mid-1940s tens of thousands of patients in the United States, and hundreds of thousands around the world, had been subjected to it.¹¹

In the beginning ECT was used in state mental hospitals, often as an undisguised means of subduing and controlling large numbers of difficult, uncooperative, and unruly patients.¹¹ It was also widely considered to be a treatment for schizophrenia. In recent years, more conservative advocates have limited its use to severe psychotic depressions, but it continues to be used for a wide variety of purposes. Those psychiatrists who favor the treatment are in general agreement that it greatly benefits individuals suffering from psychotic depressions and that it has no permanent ill effects. They also tend to agree that the beneficial effect is "empirical," that is, without known scientific or theoretical explanation. In 1978 Lothar Kalinowsky, the world's best known advocate of ECT, indicated his continued support of a statement originally made in an earlier textbook with Paul Hoch: "At present we can only say that we are treating empirically disorders whose etiology is unknown, with methods whose action is also shrouded in mystery." Kalinowsky continues to feel "proud" of that statement, declaring in an interview, "Today we are in 1978—30 years later—and exactly the same is true."⁵⁴

In 1979 I published *Electroshock: Its Brain-Disabling Effects* in which I attempted to remove the shroud of mystery for the first time with a complete review of the literature concerning brain damage following ECT, and with the

presentation of six new cases.¹¹ I proposed the brain-disabling hypothesis which directly links the brain and mind-disabling effects of ECT to its allegedly beneficial effects (see Chapter 6).^{11,12} This present discussion draws heavily upon *Electroshock: Its Brain-Disabling Effects*, and the broad references I make to the literature are detailed in that source.*

EPIDEMIOLOGY OF MODERN ECT

ECT is in far greater use than most laymen and professionals realize. When I first began investigating the matter, the response was similar to the one I received following my initial disclosures about the resurgence of psychosurgery. Most people were hard pressed to believe that the treatment was widely supported or utilized within the profession. At first I could only pursue a hunch—supported by an increasing number of articles and professional panel discussions—that ECT, along with psychosurgery, was enjoying a renaissance. Then two studies confirmed my concerns. The first was a 1973–1974 survey of psychiatric facilities in Massachusetts that, when extrapolated to the nation, provided a rate of ECT usage approaching 100,000 patients per year.³⁸ The second was a 1975 survey by the National Institute of Mental Health which indicated a minimum national rate of 60,000 persons each year, but that grossly underestimated the true figure due to numerous sampling inadequacies.⁷⁷ I have discussed these studies at length elsewhere.¹¹

These two studies, combined with a recent review of treatment methods in New York City hospitals, revealed why so many informed individuals believed that ECT was approaching the twilight of its existence.⁷ In a ratio of more than 20:1, ECT is carried out in private profit-making psychiatric hospitals rather than in more easily scrutinized academic and public facilities. Another large percentage of such treatments occur in psychiatric wards in general hospitals. Many private hospitals turned out to be treating up to 50% or more of their admissions with ECT, while some larger state facilities and academically oriented centers were no longer using ECT at all.

The usage of ECT also varies enormously from psychiatrist to psychiatrist. Some physicians seem to treat most of their patients with ECT, while others never utilize it.¹¹ The result of this situation is that the patient's likelihood of receiving ECT is based almost wholly upon the hospital and the psychiatrist who treats him, rather than upon the nature of his problem. This not only has profound implications for the so-called empirical basis of the treatment, but also for the problem of informed consent. How many patients receiving ECT realize that the treatment is not only controversial, but that its prescription is deter-

*Where I could find no advantage to rewriting my thoughts, I have occasionally borrowed the exact language from *Electroshock: Its Brain-Disabling Effects*. See reference list for full bibliographical data—PB

mined more by the personal preferences of the treating physician than by any commonly accepted standards within the profession?*

Data collected from the epidemiologic studies confirmed that psychiatrists and facilities that use ECT tend to prescribe it for a far broader spectrum of diagnoses than is commonly considered appropriate within the profession. In particular, large numbers of individuals labeled "neurotic" and "schizophrenic" are given ECT, although depression constitutes the largest percentage of patients. The preponderance of females in sex distribution of the patients was also revealing, varying from 2.35:1 in Massachusetts to 2:1 in the more faulty national survey. This too raises serious questions about the basis for prescribing the treatment.¹¹

ECT CONTROVERSY

In 1973 and 1975, a young neurologist, John Friedberg took a public stand against ECT, and helped to generate a national controversy.²⁸⁻³¹ His efforts were aided by a handful of other physicians and by many individuals representing organizations of former psychiatric inmates.^{10-13,27,35} But while public debate of the issue was a new phenomenon, acrimonious controversy about ECT within the confines of the profession was almost as old as the treatment itself. By 1947 the prestigious Group for the Advancement of Psychiatry published a unanimous report showing grave concern over the "promiscuous and indiscriminate use of electro-shock therapy." It condemned "its indiscriminate administration to patients in any and all diagnostic categories," "its immediate use to the exclusion of adequate psychotherapeutic attempts," and "its use as the sole therapeutic agent, to the neglect of a complete psychiatric program."³⁷ The Group for the Advancement of psychiatry noted that "complications and hazards" of ECT had been "minimized by some workers," and, in a move that may be unprecedented in medicine or psychiatry, it concluded that⁴¹ abuses in the use of electro-shock therapy are sufficiently widespread and dangerous to justify consideration of a campaign of professional education in the limitations of this technique, and perhaps even to justify instituting certain measures of control.

In the following years, the abuses did not abate.⁴¹ Instead criticism abated, the advocates of ECT presented an increasingly laundered version of its effects, and the treatment gradually went *sub rosa* into the confines of private profit-making hospitals and psychiatric services in general hospitals. Indeed, only 3 years after the first Group for the Advancement of Psychiatry report, the Group was pressured by ECT advocates to publish a revised version.³⁸ Concern was still voiced about "widespread abuses" and about the danger that "electroshock nullifies attempts at psychotherapy," but more severe criticism was relegated to a minority report, and the call for education and controls was dropped. The

report declared that its revisions were based on uncited "clinical" and "scientific" evidence, but the review that follows will indicate that the 3-year span from 1947 to 1950 actually witnessed the publication of some of the most convincing evidence that ECT produces permanent brain damage and mental disability.¹¹

One method of whitewashing ECT has been the profession's total reliance upon outspoken, highly promotional ECT advocates as the sole source of information and criticism. In *Electroshock: Its Brain-Disabling Effects* I described in detail how these advocates have systematically withheld pertinent studies from the profession and systematically misrepresented other relevant research. But these individuals have not been alone in their efforts to hide the facts about ECT. A 1976 survey of psychiatrists by the American Psychiatric Association was conducted with a questionnaire that made it impossible for the generation of serious criticism.⁶ The questions themselves showed the bias of the Association and its ECT authorities; the most negative item the psychiatrist can check states, "It is likely that ECT produces slight or subtle brain damage." A remarkable 41% agreed with this statement, in marked contrast to the typical ECT advocate who sees no harm whatsoever in the treatment. The possibility of reporting brain-damage-related deaths is drastically compromised when the questionnaire asks, "How many ECT deaths have occurred among your patients during, or within 24 hours of, ECT?" Brain death from ECT frequently follows days and even weeks of coma.* Finally, only psychiatrists who use ECT are authorized to complete the lengthy Section II, which seeks information about the possibility of memory loss. This excludes the reports of individuals like myself—or any critic of ECT—who has given up the treatment precisely because of the brain and mind disability he has witnessed. The questionnaire reflects a prevailing attitude of self-protection within the field of psychiatry that has created a situation in which a relatively small group of psychiatrists—perhaps 4,000 in number—are able to freely administer a treatment that has fallen into disrepute and disuse in many hospitals and among many psychiatrists.¹¹

The widespread abuses cited by the report of the Group for the Advancement of Psychiatry may be as commonplace today as they were 3 decades ago. As the epidemiologic data suggest, psychiatrists working in private profit-making facilities often use ECT as the *routine* psychiatric treatment for many or most of their patients, and they utilize it across the diagnostic categories, especially for women. Recent disclosures in Massachusetts were so sensational that the state legislature threatened to impose legislative controls, one private hospital was forced to close, and the state Department of Mental Health decided to calm the waters by imposing minimal ineffectual regulations of its own.^{17-19,40} In California, shortly afterward, another public outcry led to legislation setting standards for informed consent and for judicial procedures to determine the capacity to give consent.⁵³ My own forensic experience confirms that many private psychiatric hospitals give ECT as their main treatment, often with little justification even by textbook standards. I have come across several recent cases in which individuals were given 100 or more treatments at well-known private psychiatric hospitals.

In two cases litigation followed; a review of the charts and the depositions of the psychiatrists in these cases indicated that resentment of involuntary treatment (labeled "paranoia") was the main indicator for the diagnosis of "mental illness," and the main justification for instituting ECT. A long series of ECT were administered until the patients stopped complaining about the treatment, but in their resentment they continued these complaints until rendered euphoric, incoherent, and helplessly dependent. In effect, ECT had been used to enforce submission to the involuntary treatment.

Basic Issue: Reversibility of Acute Organic Brain Syndrome

There can be no doubt that ECT in all its forms produces brain damage and mental dysfunction. This is because all forms of ECT are administered in sufficient intensity and duration to produce an acute organic brain syndrome, characterized by the classic symptoms of disorientation to time, place, and person; mental deterioration in all intellectual spheres such as abstract reasoning, judgment, and insight; emotional lability with extremes of apathy or euphoria; and overall childlike helplessness. As in many other insults to the brain, the patient is often left with severe memory loss, most drastic for the time of the trauma, but reaching back into the past as well. Persistent headaches, nightmares, and generalized malaise may also develop.¹¹ That these reactions reflect organic brain damage cannot be seriously questioned. Indeed, it seems unconscionable that so many psychiatrists have blithely assumed that these effects are reversible, and have placed the burden of proof on their critics. The basic issue, then, is *not*, Does ECT produce brain damage and dysfunction? The basic issue is, Does the individual fully recover from the brain damage and dysfunction produced by routine ECT?

The introduction of modified ECT more than 2 decades ago has not changed the basic issues. As I have documented in detail, all commonly used modifications of ECT produce an acute organic brain syndrome, and all are typically applied until that syndrome reaches florid proportions. Sutherland and co-workers for example, have documented and compared the acute organic brain syndrome following bilateral and unilateral modified ECT, and Kafi and colleagues have measured these deficits on relatively insensitive tests 24 hours after six modified ECTs.^{48,76} Indeed, as Kalinowsky has noted, modifications that seem to temper the acute organic brain syndrome are given in greater numbers until the same effect is achieved.⁵⁴

Recently it has been claimed that the most frequently used modifications of ECT—muscle paralysis with a neuromuscular blocking agent, anesthesia with a barbiturate, and artificial respiration—produce less brain damage and dysfunction. This was never the purpose of these modifications, which were introduced wholly in order to reduce bone fractures due to muscular contractions. These modifications add the danger of anesthesia to the already existing hazards of ECT, and may produce a higher morbidity and mortality rate.¹¹ The anesthetic actually raises the seizure threshold, requiring an increased intensity of the

electric current to produce a seizure. The American Psychiatric Association survey indicated that most psychiatrists continue to employ the same sine wave current used in the earliest Bini and Cerletti machines, as well as in the machines used in demonstrating brain damage in animals.^{6,11} A review of current intensities used in modern studies shows them to be higher than in earlier years.¹¹ Since the current passing through the brain is a major cause of brain damage and mental dysfunction, modified ECT may be more likely to produce severe, lasting effects.¹¹

Brain- and Mind-Disabling Hypothesis

The brain- and mind-disabling hypothesis states that the more potent somatic therapies in psychiatry, that is, the major tranquilizers, lithium, ECT, and psychosurgery, produce brain damage and dysfunction, and that this damage and dysfunction is the primary, clinical or so-called beneficial effect.^{11,12} The individual subjected to the dysfunction becomes less able and more helpless, ultimately becoming more docile, tractable, and most importantly, more suggestible or easy to influence.* As with any brain-damaged person, the post-ECT patient will tend to deny both his personal problems and his brain dysfunction; the cooperation between physician and patient in this mutual hoax I have labeled *iatrogenic denial* (see elaboration in Chapt. 7). Surprisingly, perhaps, Fink himself came very close to stating the brain disabling hypothesis when he observed that improvement is correlated with various parameters of brain dysfunction, such as an abnormal electroencephalogram (EEG), and with an attitude of denial on the part of the patient.^{11,26}

Individual reactions to brain damage and dysfunction may also determine whether or not the patient is considered to be improved. A reaction of apathy to the damage may lead to a judgment of "improved" if the individual has previously been hostile, rebellious, manic, uncooperative, or restless and overactive. A reaction of euphoria to the damage may be called an improvement if the individual has been previously depressed, sluggish, and uncommunicative. The memory loss characteristic of ECT may also be considered an improvement if the individual no longer "knows" or "reports" on his concerns or bad recollections. Overall, not all patients will be judged improved after they have been afflicted with brain damage and dysfunction, but only those patients who become less troublesome to themselves or to others, usually as a result of their increased docility, suggestibility, or manageability, and sometimes because of their euphoria.

The brain-disabling hypothesis refers to the effects of the treatment upon *normal brain tissue and normal brain function*. ECT, psychosurgery, the major

*My emphasis in this analysis focuses upon the loss of autonomy or self-determination through brain-disabling therapies. For an analysis of the importance of autonomy and self-determination in human life see P. Breggin: *The Psychology of Freedom*. Buffalo, Prometheus, 1980—PB

tranquilizers, and lithium all impair and sometimes destroy the function of normal brain tissue. The argument that they specifically alter the function of abnormal or diseased tissue is not only unproven, it is irrelevant. Even if patients treated with these agents do have abnormal brain tissue or brain function, the overriding and predominant effect of the agents upon the central nervous system is destructive. In the case of ECT, this destructiveness is most obvious in its manifestation as an acute organic brain syndrome with residual effects, including amnesia. When organic impairment exists prior to the treatment (e.g., deterioration due to age, brain tumor, or intoxication) the existing impairment will be further aggravated by the treatment. Under such conditions, less intensive treatment (e.g., fewer ECTs) will produce a more intense effect due to the cumulative compromises of brain function.¹¹ Thus, an unusually spectacular reaction to ECT immediately brings up the suspicion that the patient is already suffering from a brain disease. For this reason, the brain- and mind-disabling hypothesis is independent of any controversy concerning the nature of so-called mental illness. If a true organic disorder, such as a biochemical imbalance, does underlie some psychiatric conditions, then the major somatic therapies can only add further organic impairment to the already existing disorder.

The brain-disabling hypothesis has been criticized on the grounds that some ECT patients show an immediate and dramatic improvement after one or two treatments, before the development of gross signs of brain dysfunction. This same observation can be made in regard to all the so-called shock treatments throughout history, from purely psychological methods such as the snake pit or threats of mutilation, to more physiologically damaging methods such as sustained dunking and insulin coma. There are many possible explanations for rapid "cures." Depressed persons notoriously respond to almost any new stimulus in their lives, from a new loved one to a moral "kick in the pants."* This is not to say that such interventions will typically affect very depressed people, but surely they sometimes do. More surely, depressed people often respond to life-threatening exigencies or other grave challenges. ECT may at times mobilize a person, if only to escape the ECT. But there is a more subtle issue that relates directly to the brain-disabling hypothesis. Euphoria, including denial and confabulation, is often one of the *earliest* signs of brain damage and dysfunction. It makes its presence known, on many occasions, before more gross or devastating symptoms. This is precisely why people turn to alcoholic, drug, or nitrous oxide intoxication, glue sniffing, and other brain-disabling self-treatments.¹² The euphoric effect is quickly achieved, and it often masks the more debilitating later effects, sometimes with fatal results. My own experience with ECT indicates that the euphoric "high" develops early in the treatment, while resentment and then apathy typically follow some time after. The sequence however is mixed and very variable.

Six Cases of Brain Dysfunction Following Modified ECT

The literature on mental dysfunction following ECT divides the subject into two separate disabilities: retrograde amnesia and anterograde amnesia. Retrograde amnesia is a typical finding after trauma to the brain, and consists of a gradient of memory loss greatest for the period of time surrounding the trauma and diminishing into the past. Anterograde amnesia is really a euphemism, for it suggests an isolated mental defect consisting of difficulties recalling new material following the trauma. The mental processes for remembering or recalling new material are intimately bound up with perception, recognition, learning, abstract reasoning, and nearly every other higher mental process, even concentration and attention. Due to the highly integrated neurophysiology of the brain, a defect in anterograde memory function is likely to reflect a much broader or more general intellectual defect. Therefore, I will speak of *anterograde mental dysfunction* when describing the ongoing or current intellectual losses of post-ECT patients.

The six cases, previously presented in greater detail in *Electroshock: Its Brain-Disabling Effects*, reflect my overall impression that ECT usually causes some degree of significant permanent mental dysfunction, and often causes severe, lasting disability, including anterograde mental dysfunction. The six include three men and three women, ranging in age from 18 to 50 at the time of ECT.

The treatments varied in number from 6 to more than 100 *modified* ECTs. Each person displayed an acute organic brain syndrome during and after treatment. The physicians' progress notes often focus upon memory loss and confusion as the main signs of the brain syndrome, but nursing notes, occupational therapy reports, and other evaluations graphically describe the individual's progressive deterioration into a state of relative helplessness and dependency. Following the termination of ECT, there was a gradual reduction of most of the severe signs of brain damage and dysfunction, leaving a residual amnesia for events surrounding and prior to the ECT in all six cases, plus other lasting symptoms in at least four cases.

The least severe permanent aftereffects among the six occurred in a patient in a short-course group, and involved nearly complete amnesia for all experiences over the 3- to 4- month period prior to ECT, plus a sense of unfamiliarity or alienation for events that occurred earlier than that, spanning several more months. Each of the other five persons had a much more severe permanent loss, which blotted out most experiences during at least the 1 year prior to ECT. Four patients, two with short courses, had difficulty remembering most of what had happened in the 2 or 3 years prior to ECT, with lesser but significant losses reaching back 5 to 10 years, including major events such as important vacations, a wedding day, family gatherings, and educational and professional experiences.

In four cases, previously clear memories of childhood seemed less familiar, with definite losses of occasional important recollections, such as houses lived in before age 10 or elementary schools attended. As anticipated, the most extreme memory losses were those closely related to the hospitalization, and none of the

patients had a clear recollection for that period of time. Where ECT had been given over a period of months, this accounted for blotting out a substantial portion of their lives. Patients who later read their hospital records were introduced to an era that was wholly unknown to them.

Four patients lost portions of their professional knowledge spanning several years, including three who felt permanently professionally impaired. Five of six experienced devastating losses in regard to persons well known to them prior to ECT. Each lost almost all sense of inner self-thoughts, feelings, and personal conflicts for the period of most severe amnesia, resulting in a sense of alienation from self or of emptiness. This involved several months in the most mild case and several years in three cases, two of whom had short courses of ECT.

The global and devastating nature of the amnesia was frequently missed by clinical observers, even during the time of most severe disability, in the hospital following ECT. Comments in the hospital charts typically noted that the person could no longer identify "what was upsetting him so much before he had his treatment," as if it were assumed (but not validated) that he could recall other things from the same period of time. Or it might be reported that a woman had forgotten data pertaining to her family or household, without acknowledging existing losses in regard to the same person's professional life. The selectivity was due to a combination of observer bias and patients' tendencies to focus on one or another deficit. I tried to overcome this by developing a global memory inventory.¹¹

Anterograde mental dysfunction is more subtle and more complex, and hence more difficult to evaluate than retrograde amnesia. In every case, the patients were also more reluctant to talk about these ongoing or current disabilities. Obviously each person experienced considerable anterograde dysfunction during the period of recovery from ECT, but there was great variation in the degree of deficit reported on long-term follow-up.

A person with a short course of ECT who felt mentally recovered did display a remarkable degree of continuing intelligence, but this had to be weighed against a past history of mental brilliance. Five years prior to ECT, the patient had received a negative neurologic evaluation, including skull films and an EEG, and immediately prior to ECT findings on history and physical examination were normal. During and after ECT, the patient developed a severe acute organic brain syndrome, mild euphoria, and lasting retrograde amnesia. When evaluated 2 to 3 years after ECT because of persisting complaints of retrograde amnesia, objective signs of brain damage were found: on neurologic exam, a *snout* reflex and Hoffmann reflex, and astereognosis and clumsiness of the left hand; on computerized axial tomography (CAT scan), right temporal lobe atrophy and ventricular enlargement to two and one-half times normal;* and on EEG, temporal lobe abnormalities. Psychological testing revealed a loss in abstract

reasoning and right hemisphere deficits in the area of nonverbal memory and concepts. The highly experienced professor of psychology who conducted the tests concluded that these defects represented "a devastating loss on the phenomenological level" despite a persisting high intelligence quotient.

While the retrograde amnesias were extremely upsetting to these persons, the anterograde mental problems were far more disturbing. The sense of being "defective" and the personal cost in terms of a full, rich life in the future were far greater. An abstract presentation of the case histories cannot communicate the anguish experienced by these patients in regard to their ECT treatment and their subsequent mental losses. For most of the patients, the inability to recall names and faces was the cause of some of their most humiliating experiences. They would meet individuals in the market or at work, and after receiving a warm welcome as if from an old friend, they would be unable to recognize the person. Sometimes old friends would visit, but except for a faint recognition, the relationship was effectively wiped out. There was also enormous shame and upset over the inability to carry out routine affairs, such as finding the way to familiar shops, or recalling a favorite recipe. The loss of important life experiences, such as wedding day or graduation, cut the person off from his or her identity, and turned conversations about the past into humiliating interchanges. Beyond that, the loss of ongoing mental function, such as mental alacrity and emotional responsiveness, created a ghastly sense of hopelessness about the future.

Clinical and Research Reports Confirming Permanent Mental Dysfunction

Considering the frequency with which patients complain of severe amnesia and other mental dysfunction following ECT, it is astonishing that so few attempts have been made to systematically evaluate these symptoms. Without a great deal of sophistication or effort, it would be possible to take memory inventories for real-life experiences from patients before ECT, and to compare the availability of these recollections after treatment. The relative absence of such studies is especially inexcusable in a profession whose major concern is supposed to be the psychological status or condition of its patients.

Those authoritative sources that do suggest the possibility of mental dysfunction following ECT usually limit their concern to retrograde amnesia. This too is a surprising oversight. Since the treatment produces an acute organic brain syndrome with gross disruption of all mental function, there must be a presumption that the treatment may produce permanent ill effects in general intellectual function.

Contrary to the prevailing opinion within the profession, there are many anecdotal and clinical reports in the literature that warn against the danger of permanent, severe retrograde amnesia following ECT, and there are a few significant systematic studies as well. I have reviewed them in great detail.¹¹ Here I can only present the highlights.

Mental Dysfunction after Unmodified ECT

One of the most detailed clinical follow-ups of long-term retrograde amnesia was published by Brody, who selected five patients who were reportedly doing well following their treatment.¹⁴ Some of them were carrying on routine life activities. As in my six cases, all five displayed serious retrograde amnesia, as well as despair. One of his patients, who was still aware of gross losses after eighteen months, reported, "I have met one or two people who seem to know all about me and I cannot remember any thing about them: I look silly at them and get frightened meeting people."¹⁵

A 48-year-old woman with only 15 treatments complained of anterograde defects as well as retrograde amnesia. "I cannot seem to remember but it comes back later on. It takes me a long time to remember. My memory seems slower. It lets me down over just small things that I am doing like posting a letter."¹⁶

She had shown some improvement for 6 months, then none over the following 2 years. She was similar to still another case reported by Brody whose condition improved for 9 months and then leveled off with persistent memory defects.

Like most psychiatrists involved in ECT research, Brody is unwilling to condemn it in principle. But he did warn that these memory problems caused considerable "mental strain," and that they implied "permanent, or semi-permanent, damage to the brain." He proposed that ECT should not be given to people working in professions requiring a high degree of mental and memory dexterity. Such a viewpoint belittles the spiritual or personal loss of individuals who value their minds regardless of their job requirements but it is an attitude that is unhappily repeated throughout the ECT literature into modern times.¹⁷

Two of the more dramatic reports in the unmodified ECT literature concern physicians given ECT. Watkins, Stainbrook and Lowenbach in 1941 reported the devastating effect of one subconvulsive electroshock (400 ma for 0.165 sec) administered to a 25-year-old physician as an experiment. Forty-one minutes after the shock, he "had lost the ability of recalling recent as well as past events, and could not retain information."¹⁸ He was euphoric 3 hours later, and forgot his regular room assignment 4 hours later. Fourteen hours later he awakened with a "queer" feeling and suffering an indescribable "feeling of unreality." Over the following days, most of his memory gradually returned, but events prior to shock and for 15 hours after the shock remained "completely blotted out." Throughout this, his outward behavior seemed normal to casual observers.

A report by Bersot describes similar dramatic effects upon a physician after two ECTs.¹⁹ For several months afterward he showed a variety of symptoms, including mild euphoria, mental fatigue, difficulty with memory, and a remoteness from the past. Again, his social conduct remained relatively normal during this period. He felt that he became himself again about 1 month after the two treatments.

The damaging effects of ECT can be seen in exaggerated form following intensive or regressive ECT in which the individual is given ECT at the rate of one or more a day until a state of neurologic dilapidation sets in, including dementia, the appearance of primitive neurologic reflexes, mutism, inconti-

nence, and the inability to carry out routine self-maintenance activities, such as eating or dressing. Memory is usually so obliterated that the patient is unaware that he has received any treatment at all. Stengel evaluated the lasting effects of this form of treatment. In one case: "She had forgotten not only the events of her whole previous life, but also much that she had learned from childhood. Everything seemed new to her. She inquired about the significance and the names of familiar objects like a child of three."⁷² This woman's amnesia, 4 years later, had gradually "shrunk to a period of three years."

Stengel found the name "annihilation therapy" (coined by Cerletti) unfortunately appropriate, and he compared its effects to that of head injury with "severe traumatic interference with brain functions." He declared that it did much harm and little good, and should be abandoned. But intensive, regressive, or annihilation therapy is enjoying a resurgence at the present time.^{1,9,23}

The literature on unmodified ECT also contains some systematic research studies of retrograde amnesia. The most carefully conducted study by I.L. Janis produced such devastating findings that in itself should have produced a halt in the clinical use of ECT.⁴⁴⁻⁴⁶ Instead it was largely ignored.

Janis studied 19 patients given routine ECT in psychiatric hospitals, and interviewed them before and after their treatments concerning significant events in their past. He also interviewed 11 control patients with similar diagnoses from the same hospitals. The posttreatment interview was administered 4 weeks after the termination of ECT and was designed to test the recall of personal data the patient had produced prior to treatment. Great care was taken to retest in a gradual and ultimately thorough manner, eventually presenting a portion of former recollections to see if these memories could be restimulated after they were lost. The patients' ability to recall past events (as I described) was devastated in the ECT group, but almost untouched in the control groups.

It was found that every one of the 19 patients in the electroshock group displayed definite retroactive amnesias, as of approximately four weeks after the termination of ECT. For each case it was possible to verify many of the forgotten events as actual occurrences, on the basis of independent sources of information in the patient's case history records. Many of the patients were unable to recall from 10 to 20 life experiences which had been available to recall prior to electroshock.⁴⁴

As in the cases with modified ECT I reported, some losses reached back into childhood. "It should be mentioned that the amnesias are by no means limited to events of the recent past, although experiences during the six months prior to treatment are more likely to be forgotten than those which had occurred in earlier periods. Occasionally the amnesias involve events of early childhood that date back from 20-40 years."

Some patients lost *total recall* for a period of several months prior to ECT, exactly as the cases in my presentation. More tragically, in the five cases he was able to follow up beyond 4 weeks, almost all the memory gaps remained. At 2½ to 3½ months, "it was found that the patients were still amnesic for almost all of the personal experiences which they had been unable to recall in the earlier post-treatment interview. This finding reinforces the conclusions that the post-ECT amnesias persist well beyond the usual period during which there is

recovery from the transient organic effects of ECT." Indeed, Janis continued to find memory losses at least 1 year-ECT.⁴⁴⁻⁴⁶

Janis also carried out word association tests and found "deviant" and irrational responses as well as "defective reproductions," indicating decreased ability to recall after ECT.⁴⁵ Thus he found anterograde as well as retrograde mental dysfunction.

Schwartzman and Termansen also studied the long-range effects of "de-patterning" or intensive ECT as developed by Cameron.⁴⁶ Their 1967 publication dealt with patients who had been treated with unmodified ECT between 1956 and 1963, and provided very long-term follow-ups. They found a 6-month to 10-year retrograde amnesia reported by 60% of their patients, and a dependency for recall reported by 63%. They concluded that these and other findings argued against the continuation of this form of treatment.

Stieper and co-workers also dealt specifically with the complaint of lost personal memories, noting that "in personal interviews, post-shock patients most frequently express concern over their personal memory defects, rather than impersonal defects."⁴⁷ They studied a control group and 15 patients who had received between 5 and 25 treatments, using a personal inventory of 20 recent and 20 remote items. They found a statistically significant loss of "personal and current information" and found that "items which appeared to be most affected were those involving their prehospitalization personal adjustments: jobs held previous to entering the hospital and recent illnesses." Patients frequently responded "I don't know," when asked about recent memories.

A variety of other studies confirm lasting mental dysfunction following unmodified ECT, including two modern follow-ups by Goldman and associates and Templar and colleagues, the latter concluding "the ECT patients' inferior Bender-Gestalt performance does suggest that ECT causes permanent brain damage."^{48,49}

In my book I have examined systematically how well-known advocates of ECT, such as Lothar Kalnowsky and Paul Hoch, distorted or simply ignored key studies indicating irreversible mental dysfunction following unmodified ECT. I have also examined how the studies they cite to support the harmlessness of ECT are often irrelevant to the subject, or in many cases, actually confirm the dangerousness of the treatment.¹¹

Mental Dysfunction after Modified ECT

There are fewer good studies on any aspect of ECT in the modern modified literature than in the earlier literature. This is one reason why I have presented my own six cases of irreversible mental dysfunction following modified ECT. Indeed, most of the modern studies of memory function following ECT use tests of unknown validity and measure variables that relate little or not at all to true retrograde amnesia. The all-important Janis study has not been repeated.

The team led by Squire has been most egregiously guilty of using tests of unknown validity to prove the harmlessness of ECT. But when Squire and Chace asked patients to assess their own memory loss 6 to 9 months after routine modified ECT, they had startling results: amnesia was reported by 63% of patients receiving bilateral ECT and by 30% of patients receiving unilateral

ECT.⁷⁰ Squire elaborated on these findings in 1977, reporting that 37 of 55 subjects receiving short courses of bilateral ECT "indicated that their memory was not as good as it used to be."⁶⁹ These results were obtained despite the exclusion from the study of memory loss around the time of hospitalization. In still another study Squire and associates modified their tests and found that memory for "temporal order is remarkably affected by ECT" for a period covering 1 to 7 years prior to treatment.⁷¹ Their follow-ups came 6 to 25 days after 5 to 18 ECTs.

The team including Strain, Bidder, and Brunschwig developed a Personal Data sheet to test recall for recent and remote memory after modified ECT.⁷⁵ They found significant memory losses that remained stable 10 days after six bilateral or unilateral ECTs. While they do not have longer follow-ups, in personal communications to me Brunschwig made clear that long-term clinical evaluation indicated that memory did not completely clear in many cases.¹¹ In the last report of their experimental results, they observed that "impaired memory persisted after treatment even among patients strongly motivated to regain normal functioning."¹⁵

Small, Sharpley, and Small have also studied long-term memory after modified ECT, and found memory defects on psychological testing 60 to 90 days after ECT.⁶⁷ In 1974 Small followed up patients 2 to 5 years after treatment and found that "more than half the patients considered their memory to be worse" and that a number "complained of persistent memory defects for several years after convulsive therapy."⁶⁶

Teuber, Corckin, and Twitchell confirmed that psychosurgery and modified ECT in combination can produce very severe retrograde and anterograde dysfunction.^{11,70} Global, serious losses in mental function were documented on a variety of objective tests, and attributed wholly to ECT, although a scrutiny of the data suggested a combined effect of ECT plus psychosurgery.¹¹ Of importance here, psychosurgery alone did not produce these drastic mental deficits.

Turning from experimental to clinical reports, the literature continues to be sparser in modern times. However, many authorities have continued to argue that ECT should not be given to individuals whose jobs require memory and intellects.¹¹ An autobiographic report by "a practicing psychiatrist" was published in 1965 in an attempt to prove the harmlessness of ECT, but a careful reading discloses that the psychiatrist admits to severe retrograde amnesia following a short course of ECT.⁶¹ He was forced to painstakingly relearn his own filing system as well as a very familiar subway system. When prompted on past memories, he found the memories retained an unreal quality—as reported by my six cases.

Neurologist John Friedberg published rich verbatim reports of memory loss following both unmodified and modified ECT.²⁰ His sources are biased—individuals specifically asked to contact him concerning brain damage from ECT—but the reports parallel others in the literature as well as my own cases.

Perhaps because of renewed concern about ECT, two detailed case studies have been recently published documenting severe, persistent mental dysfunction following ECT. Regestein and co-workers describe the case of a woman with "prolonged, reversible dementia" following long-term ECT.⁶² Her mental state 29 weeks later is, unfortunately, not described in detail, but the symptoms were

not entirely reversible. She is noted to have "a tendency to perseverate." In another recent report Elmore and Sugarman describe three cases of flagrant psychoses developing in response to ECT.²² This phenomenon has been reported by Kalinowsky and Hippus, and I have witnessed it myself.⁴⁰ Instead of recognizing this as an extreme manifestation of the acute organic brain syndrome, clinicians tend to describe it as the "unmasking" of the patient's "psychosis."¹¹

Are the Patients Lying?

At times authorities on ECT claim that patients never complain of memory loss after ECT, and at other times they admit to these complaints and attempts to invalidate them as the ravings of "neurotics" or patients who have failed to respond to the treatment. Perhaps one of the most self-serving and yet revealing statements in the entire ECT literature was made by Kalinowsky and Hoch in the 1952 edition of their textbook: "All patients who remain unimproved after ECT are inclined to complain bitterly about their memory difficulties."⁵⁰

The authors say that *all* patients who remain unimproved complain about amnesia, and furthermore, they admit that they complain bitterly. How then can Kalinowsky and Hoch argue on the same page that "no evidence has been brought forward to indicate that permanent mental sequelae are caused by the treatment"? Not only must they ignore the various studies demonstrating memory losses, they must discount the patients as "neurotics."^{11*}

I have examined in great detail this issue of whether or not the patients are lying.¹¹ Here I want to reaffirm a well-known clinical observation: individuals who suffer brain-disabling assaults tend to underestimate their losses rather than to exaggerate them. This confabulation is a defensive attempt to cover up the frightening and shameful disabilities they experience. This is equally true with regard to post-ECT patients. Their personal reports almost invariably underplay the degree of their losses.

There is a considerably greater reason to believe that the patients are not lying. Animal research, human autopsy studies, and human EEG studies all confirm permanent brain damage and mental dysfunction in many cases following ECT. The summary that follows draws upon the review presented in *Electroshock: Its Brain-Disabling Effects*.¹¹

Animal Research Demonstrating Irreversible Brain Damage following ECT

There is a series of studies that confirm diffuse brain damage in animals following ECT. The most common findings are petechial or pinpoint hemorrhages throughout the brain and surrounding blood vessels, as well as areas of gliosis and neuronal degeneration, with patches of cell death (ghost cells and neuronophagia). In addition, occasionally larger hemorrhages and edema of the

brain are found. The findings are also seen on human autopsies, and can be explained by diffuse passage of the electric current down the vascular tree. The skull acts as a resistor, and the build-up of electric charge then breaks through at various points of high conductivity. Because of the placement of the electrodes, the greatest damage is found over the anterior temporal lobes and frontal lobes, but it is by no means restricted to these areas.¹¹

The most thorough study of diffuse brain damage is contained in a book-length monograph by Hartelius, who reviews the literature, making many of the points I have summarized in the previous paragraph.⁴¹ Hartelius used small sized electrodes (though this turned out to be of no importance), the least possible current intensity needed to produce convulsions in cats, a variety of current types, and protection of the head against trauma. Indeed, as in most animal studies, the conditions were far less traumatic than those in routine ECT. Of great importance, the study was double blind, and the pathologist had no idea which animals had been shocked.

The results were striking. The pathologist made no mistakes in identifying which animals had or had not been given ECT. He found changes in the vessel walls, nerve cell deterioration, and glial reaction scarring in animals who had received as few as four ECTs. In slides taken from throughout the brains of the shocked animals, he also found a statistically significant number of irreversible changes in the form of cell death. The great majority of these irreversible changes were found in animals given 11 to 16 ECTs. Hartelius concludes, "On the basis of the present results, the question of whether or not irreversible changes may occur after ECT must be answered in the affirmative."⁴¹

Hartelius' findings should have been no surprise. Despite the myth that Bini and Cerletti proved the harmlessness of ECT before using it on human beings, an actual review of their early reports demonstrates that they found widespread and irreversible changes following the use of ECT in animals.^{11,41} By the time Jessner and Ryan's *Shock Treatment in Psychiatry* was published in 1941, the statement could already be made that "a great deal of evidence has been accumulated which indicates that brain damage is possible with this form of shock therapy."⁴⁷ In 1941 Heilbrunn and Liebert examined biopsies of rabbit brains after ECT and found that even one treatment could produce serious effects in the form of the cellular outline loss and cellular deterioration.⁴² Alpers and Hughes administered ECT to cats under conditions mimicking clinical usage and found frequent hemorrhages.⁵ Neuberger and associates administered ECT to dogs and found similar findings to those of Hartelius, including ghostlike cells.⁵⁸

In 1946 in an elegant study, Ferraro, Roizen, and Helford gave clinical doses of ECT to monkeys and found "possibly some permanent slight structural damage," and despite their bias toward ECT, they admitted that "such damage no matter how slight may ultimately become permanent."²⁵ Their actual slides show extensive damage, including definitive areas of cell death with cell rarification, acellular areas, and neuronophagia, along with the typical petechial hemorrhages. This study used controls, small electrodes, routine numbers of ECT, and current intensities lower than those in typical use today, and in other ways tried to respond to criticism of earlier studies. In 1949 Ferraro and Roizen published a second study, using greater numbers of ECTs. They found cell death propor-

tional to the number of ECTs administered, but present even in the cases where fewer numbers of ECTs were given.^{24,32-74}

Unhappily, most of these studies have been ignored in pro-ECT literature, or actually misrepresented.¹¹ Indeed, pro-ECT animal studies that try to suggest that ECT is harmless usually demonstrate severe cellular changes and hemorrhages. (For a detailed review, see reference 11.)

A number of studies have demonstrated the mechanism of ECT damage, which is largely related to the passage of the electric current. Even small doses of ECT visualized by angiography³⁹ or through craniotomy²¹ produce severe vasoconstriction and blanching. The permeability of the blood-brain barrier breaks down so that dyes extravasate into the tissue and hemorrhages may occur.² The hippocampal area, which is crucial to memory, is particularly susceptible to anoxia and damage. It lies directly under the electrodes, which accounts for the relative severity of memory loss.¹¹

Studies that show that any general anoxia due to ECT is reduced by recent modifications do not bear on the question of brain damage, because destructive changes can occur without an overall anoxia of the brain, just as any trauma may cause serious damage without producing general anoxia. Indeed, subconvulsive shocks have produced serious mental aftereffects, and in animals shocks without convulsion have also produced amnesia and behavioral changes due to central nervous system (CNS) damage.^{11*} Epileptics who become anoxic may suffer some brain damage, but this is debatable; the damage is certainly not as demonstrable as the damage following ECT, so that anoxia is again ruled out as the culprit. The main source of the trauma appears to be the stimulus used to produce the convulsion, with the convulsion itself compounding the insult.

A wide variety of animal research focuses on the biochemical changes associated with electric stimulation and ECT, and most experienced researchers agree with Dunn and colleagues that permanent retrograde amnesia does take place and is probably produced by the action of the electric current (even in subconvulsive doses) in disrupting protein synthesis.^{11,20} In 1974, for example, the experienced researcher McCaugh concluded that ECT produces permanent retrograde amnesia in animals. "Although some investigators have reported finding that memory impairment produced by ECT is only temporary, most studies investigating this problem have found that the RA [retrograde amnesia] is permanent, at least over intervals of time ranging from 12 hours to one month. Overall there is little evidence to support the view that ECT produces only temporary RA."⁶⁵ I have reviewed this and related data on animal biochemical and behavioral studies, many of recent origin, in *Electroshock: Its Brain-Disabling Effects*.

Human Autopsy Studies Following Modified and Unmodified ECT

Many textbooks and authoritative reviews state that the death rate following ECT is very low, and that therefore there are few autopsy studies in the

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literature. In reality, some studies report a death rate as high as 1:1,000 in the total ECT population, with rates as high as 1:200 in selected groups, such as the aged.^{41,49} There are many dozens of autopsy reports in the literature showing a high proportion of brain death following ECT with results that often mimic those found in animal experiments with ECT. Occasionally, the results of animal research and human autopsies are directly compared. However, nearly all these studies are omitted from the textbooks and authoritative reviews. By 1948 Otto Will and his associates⁴⁴ at St. Elizabeth's in Washington, D.C., were able to review 33 cases from the literature, and a new one of their own.⁴⁴ Of the 16 cases where the brains were examined, 50% showed brain changes attributable to ECT, many of them severe. Many individual case reports followed over the years, culminating in Impastato's 1957 review of 214 fatalities from the literature, plus 40 new cases.⁴³ Many of the patients died of *modified* ECT, and many show a variety of severe forms of central nervous system damage associated with ECT. Although a staunch defender of ECT, Impastato voiced concern over its dangers, and his study has been largely expurgated from textbooks and reviews. While the literature on ECT in general became sparse after the 1950s, cases of death with brain pathology have continued to be reported.^{41, 53, 52, 56}

Human Brain Wave and Neurologic Studies

Despite constant claims in the authoritative textbooks that ECT produces no permanent changes in the brain waves, dozens of research projects confirm long-lasting and permanent abnormal waves in a significant percentage of patients. The findings vary from profound dysrhythmias with massive, irregular high-voltage slow waves to an increase in high-voltage slow waves. Often these studies compare the changes with those of chronic epilepsy, intoxication with associated brain pathology, and other severe conditions. Although Kalinowsky himself in all his publications claims that no abnormal brain wave patterns remain after ECT, a study in which he himself was involved showed that after 13 to 22 treatments, abnormalities of brain wave patterns typically remained from 2 to 6 months, and 30% remained abnormal at the conclusion of the study 6 months after discontinuation of the treatments. In a later publication from the same project, Pacella found continuing abnormalities months later, and also warned that the disappearance of abnormalities by no means meant that the pathology within the brain had disappeared or healed, since the EEG is a notoriously insensitive test.⁵⁹

Many similar reports were made over the years, culminating in a major report by Mosovitch and Katzenelbogen in 1948, which at the conclusion of a 10-month study, left no doubt about massive, irreversible EEG changes in many patients, including 15% of patients receiving 16 to 42 treatments.⁶⁷ Katzenelbogen found the abnormalities associated clinically with memory defects.⁴¹

There are numerous EEG studies confirming the same changes following modified ECT.⁴¹ Unilateral ECT, for example, may produce even greater changes on the side of the head to which both electrodes are attached, again confirming that the current is the major source of damage. Among the several studies cited in *Electroshock: Its Brain-Disabling Effects*, Matsuda found persis-

tent EEG changes 30 days after ECT, Turkek after 2 weeks, and Valentine and associates after 10 days, when final testing was terminated in each study.^{51,60,62} Volavka and colleagues reviewed the subject of ECT and brain wave changes and concluded that "EEG changes may persist for several months after the termination of a course of treatment" in both bilateral and unilateral modified ECT.⁶² Roth and Garside have concluded that "long courses of treatment are almost invariably associated with an obvious and sustained change in the electrical activity of the brain," which they believe to be "continuous and lasting."⁶⁴ They compare the damage to that produced by lobotomy.

Serious neurologic complications have also been reported in the literature on both unmodified and modified ECT, although most are limited to articles in which death resulted. I.M. Allen was among the first physicians to report on neurologic impairments following ECT and to relate them in a systematic manner to animal research, human autopsy studies, and EEG reports.^{3,4} He gives many vivid clinical descriptions of permanent mental dysfunction as well. His work, which is almost wholly expurgated from authoritative texts and reviews, is well worth reviewing. In addition, occasional reports of neurologic disorders following ECT continue to appear in the modern literature on modified ECT, including Strain and Bidder and Reinhart.^{11,63,74}

Efficacy of ECT

The efficacy of ECT in depression is frequently reported to be as high as 90%. ECT has also been seen by some advocates as an absolutely indispensable treatment for severely suicidal patients. It is said that even if the treatment produces brain damage, psychiatry has nothing with which to replace it. The argument that ECT is indispensable is easily dismissed. The data reviewed in this article, and more deeply in *Electroshock: Its Brain-Disabling Effects*, clearly demonstrate that many hospitals, both private and public, never employ the treatment under any conditions. Unless it can be demonstrated that these hospitals experience an increased suicide rate, the argument for the indispensability of ECT for this purpose falls apart. Since a variety of studies on ECT and suicide have utterly failed to show that ECT can reduce the suicide rate¹¹ there seems little basis to believe that hospitals in which ECT is *not* used will turn out to have higher suicide rates. In addition, the argument that ECT is the best treatment for depression also falls apart on examination of the literature. One of the most highly touted studies merely shows that women benefit from ECT while men do not, while the other shows that placebo does nearly as well.^{16,34} Considering the hundreds of attempts to prove the efficacy of ECT, even using the standards proposed by the advocates—such as discharge from the hospital—there is no evidence that ECT is helpful.

On the other hand, ECT does have a potent effect upon people, and I believe that those psychiatrists who use the treatment are indeed gratified by the results. As I have described in the brain-disabling hypothesis earlier in this paper, in Chapter 7 on psychosurgery, and elsewhere, ECT produces severe mental disability, and this disability plays an active part in the psychiatrist's

judgment that the patient is improved.^{11,12} The patient may wish to leave the hospital simply because he is terrified of what is being done to him. When he asks for discharge and receives it, he may be viewed by his psychiatrist as "improved." The patient may become apathetic or euphoric, and this may be evaluated as an improvement in comparison to the depression. The patient typically becomes more "cooperative," that is, docile, suggestible, and generally easier to handle. Fink himself has postulated and proven that patients who tend to use denial (including euphoria) as a defense mechanism are rated as improved after ECT.²⁶ Most ECT cures are a product of what I call *iatrogenic denial* (see Chapt. 7). Memory loss may also provide the patient a measure of relief, until he realizes how severe and irreparable it is.

Amid all the controversy about ECT, very few patients have come forth to praise the treatment, while many have come forth to complain bitterly about it.¹¹ Of course, some patients will want ECT, just as some individuals will want to destroy their mental function by sniffing glue, abusing alcohol, or ingesting dangerous drugs. The surprising fact is this: despite the great tendency in human nature to seek out self-destructive methods of achieving mental oblivion, few individuals actively seek ECT for themselves. There are no organizations of former ECT patients rising up to defend the treatment, while many organizations of former ECT patients are raising their voices to the media in protest.²⁷

Recommendations

ECT is a dangerous, destructive intervention whose sole effect is the production of brain damage and dysfunction. It has no theoretical or scientific rationale, but can be understood in terms of the brain- and mind-disabling hypothesis. It produces a disabled, helpless, highly suggestible individual who for a time at least, is less troublesome to others, and sometimes, to himself. If ECT were subjected to the kind of scrutiny to which new and experimental drugs are now subjected, it would never be approved for research or clinical usage. In my own opinion, it is time to stop its use in human beings for either research or clinical purposes.*

Many hospitals and many individual psychiatrists no longer use ECT. It is my firm hope that the research I have summarized in this chapter and produced in full in *Electroshock: Its Brain-Disabling Effects* will encourage greater numbers of hospitals and doctors to stop using the treatment. However, a large group of pro-ECT psychiatrists have banded together in a formal organization whose purpose is to improve the public and professional image of ECT, and the major psychiatric organization, The American Psychiatric Association, has shown every inclination to continue to support the treatment.¹¹ Any major changes must originate from outside the profession.

Changes from outside the profession can take many forms. First, a public already wary of the treatment can gain still greater caution through the educational efforts of psychiatrists, organizations of former psychiatric inmates, and public interest groups. Second, legislation such as that recently passed in California can affirm the patient's right to be fully informed of the effects of the treatment, and also confirm that the psychiatrist himself cannot take responsibility for giving ECT to a patient deemed incapable of giving informed consent. Third, malpractice suits can be brought against psychiatrists who have not fulfilled the requirements of informed consent, either because they have coerced their patients into taking ECT or because they have failed to describe its damaging effects, its controversial nature, and its unproven efficacy. Fourth, by legislative action the treatment can be banned on involuntary mental patients, prisoners, children, and incompetents.¹¹ I also believe that the treatment is sufficiently dangerous and unproven to justify its removal from insurance company lists of acceptable treatments.

However, I am not in favor wholly of outlawing ECT for voluntary patients by legislative fiat. On practical grounds, when the government gets into the business of outlawing treatments, it invariably attacks those treatments that are innovative or lack establishment support, while it supports those treatments that the prevailing authorities find in their own self-interest. On theoretical or moral grounds, I do not believe that the government has the right to tell physicians what treatments they may prescribe, nor voluntary, competent patients what treatments they may seek. Instead, patients and doctors alike should be permitted to join in voluntary contractual agreements concerning any form of treatment. The patient's protection is his right to refuse any treatment, and his right to be informed of its dangerous effects and unproven efficacy.¹¹ We are a long way from the achievement of this ideal situation, but there is no way to hurry it along by government bans. Liberty remains the greatest principle upon which to base individual rights and well-being. I believe that in time ECT will be abandoned through a combination of scientific, rational discourse, and the willingness of injured patients to take their cases to court. Legislation that affirms the patient's right to refuse any and all treatment must be the cornerstone of any defense against oppressive, destructive therapies.

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