

## ***Shock Therapy- Schlock History***

**By Richard A. Warner**

*Shock Therapy, A History of Electroconvulsive Treatment in Mental Illness*  
By Edward Shorter and David Healy  
Rutgers University Press

Note: Citations from the book are referenced by page number, in parenthesis.

Psychiatric treatments\* tend to follow a common historical path. Initially each one is hailed as an important advance and tales of amazing “cures” appear in the media. Early signs of harmful effects are denied by treatment proponents. Then researchers start finding damaging side effects, patients begin complaining, investigators uncover hidden data, effectiveness begins to be questioned, and regulatory agencies eventually issue warnings. Finally it becomes evident that the treatment does little to change the condition it has been claimed to alleviate and that, in fact, it can result in a great deal of harm.

Harvard psychiatrist Joseph Glenmullen, referring to a similar pattern with psychiatric drugs, called it “the cycle from miracle to disaster” (Glenmullen, 2000, p. 12). We saw it in the 50’s when Walter Freeman toured the nation lobotomizing patients with an ice pick, while stories in the popular media, including *Time*, *Life*, and the *Saturday Evening Post*, extolled his crude mutilations (Valenstein, 1986, pp. 156 –193), and again in the 50’s and 60’s when it became apparent that antipsychotic drugs were causing permanent irreversible brain damage but the warnings were ignored (Brown and Funk, 1986).

We saw it in 1976, when the Drug Abuse Warning Network (DAWN), a federal statistics-gathering organization, identified Valium as “the ‘number one’ abused drug in emergency rooms across the nation” and “the first or second most abused drug by 19 of the 23 reporting cities”

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\*An unfortunate aspect of writing about mental health issues is that one is forced to use words which imply a reality that has never been proven to exist. Words like treatment, therapy, patient, and mental health act to subliminally indoctrinate the reader to see life in medical terms, that is, to 1) view people with mental and emotional problems as sick and 2) view what is done to them as curative or palliative. Regarding the first premise, Elliot Valenstein, Professor Emeritus of Psychology and Neuroscience at the University of Michigan, has written “... no biochemical, anatomical, or functional signs have been found that reliably distinguish the brains of mental patients” (Valenstein, 1998, p. 125). Harvard psychiatrist Joseph Glenmullen agrees: “We do not have proof either of the cause or the physiology for any psychiatric diagnosis” (Glenmullen, 2000, p. 193). In 2005, the president of the American Psychiatric Association, Steven Sharfstein, admitted, “We do not have a clean cut lab test” for chemical imbalances. (Sharfstein, 2005). As regards the second premise, psychiatrists admit they offer no cures (Satcher, 1999; The Menninger Clinic, 2007; Callahan, D., 1999) and a large body of evidence suggests that what they do to individuals actually creates illness and hinders recovery (Whitaker, 2002 & 2005).

(*Journal of the American Medical Association (JAMA)*, 1976, p. 432). The DAWN report had covered the period from April 1974 to April 1975. Only 7 months earlier (September, 1973) a review in *JAMA* had praised Valium because “tolerance, abuse and abstinence are very rare” (Blackwell, 1973, p. 1640).

The pattern was repeated in 1990’s when mounting evidence that the new Prozac-type antidepressant drugs caused extreme agitation and suicidal ideation was dismissed by a Food and Drug Administration (FDA) panel with deep conflicts of interest, including one psychiatrist who, at the time of the hearing, had \$200,000 in pending grants from Eli Lilly, the maker the principal drug the committee was investigating, Prozac (Breggin, P, 1994, pp. 170-174). In 2001, researchers at Yale University reported that mania and psychosis induced by antidepressants accounted for 8.1% of all general psychiatric hospital admissions over one 14-month period (Preda, MacClean, Mazure, Bowers, 2001). 70% of the cases had been taking one of the newer antidepressants (Prozac, Luvox, Paxil, Zoloft. These drugs are generally classed as SSRIs - selective serotonin reuptake inhibitors) prior to admission. In 2004, the FDA finally ordered that black box warnings be placed on SSRI antidepressants after finding they doubled the risk of suicidal behavior or thinking for children and adolescents (Food and Drug Administration, 2004). That finding was later extended to young adults up to 25 years of age (Vedantum, 2006).

There are countless other examples. Together they remind us that the evaluation of psychiatric practice demands a good measure of skepticism and a willingness to question research and look behind the marketing façade that the field presents to the public and to itself.

So when a book about shock therapy – presented as a *history* - ignores the historic patterns that have characterized shock and other psychiatric treatments and instead approaches the subject matter with a distinctly credulous frame of mind, you can’t help but wonder if the work isn’t being guided by an unseen agenda.

It’s not that authors Edward Shorter and David Healy are unaware of psychiatry’s troublesome M.O. Indeed, five pages from the end of their new book, *Shock Therapy, A History of Electroconvulsive Treatment in Mental Illness*, they launch a remarkably withering attack against the profession they spend the first 293 pages indulging. “[F]ar from current psychiatric practice being evidence-based and rational,” they write, “it is as ideological as it has ever been” (294). Shorter and Healy refer to the 21st century thinking of psychiatry as a “bio-babble” that, “within a few years ... will almost certainly seem as vacuous as Freudian notions about libido” (296). “Psychiatric thought,” they say, “far from having developed since the 1960’s, has arguably atrophied” and “there is probably no other branch of medicine where the outcomes for a core disease are steadily worsening” (295). A more succinctly punishing dismissal of psychiatry has seldom been heard.

Pronouncements like these are not unexpected coming from Healy, who, in a recent article on the marketing of bipolar disorder, excoriated clinicians, drug companies, academics and even parents, suggesting that the lot of them were suffering from a “variation on Munchhausen’s syndrome, where some significant other wants the individual to be ill, and these significant others derive some gain from these proxy illnesses” (Healy and Le Noury, 2007, p. 219). Even Shorter, who began his 1997 book, *A History of Psychiatry*, by gushing that biological psychiatry in the

20<sup>th</sup> century was “a smashing success” (Shorter, 1997, p. vii), has been willing to acknowledge some of the problems inherent in psychiatry’s symbiotic relationship with the pharmaceutical industry (Shorter and Tyrer, 2003, Shorter, 1997).

Unfortunately, both authors have abandoned critical thought in this surprisingly shallow shout-out to shock. For Healy, who has long been an astute critic of psychiatric dissembling, this is a particularly disappointing effort. Make no mistake: psychiatry’s empty posturing is no less visible with convulsive “treatments” than it is with its other procedures. But S & H were apparently not the least bit interested in seeing it.

### **Insulin shock**

Healy and Shorter waste no time in sharing their enthusiasm for shock. By page four we’ve already been told six times that electroconvulsive therapy is “effective.” The second chapter’s treatment of Manfred Sakel, who developed insulin coma therapy (ICT), illustrates how far S & H are willing to go to paint convulsive therapies in a positive light. The authors refer to Sakel’s “typical tendency to exaggerate his achievements” (14), his mania (20), his “increasingly obsessional and grandiose” behavior (20), his lying that he was the chief physician of a sanatorium when he was not (14), his claims that he carried out animal research when there was no evidence he did (14), and his willingness to switch his theoretical position on the importance of convulsions to ICT so that he could claim credit for having created convulsive therapy (17). They note that other clinics were unable to confirm Sakel’s results (18). The retired director of one mental hospital calls the treatment “useless” (21). “The whole subject remains quite obscure today,” say the authors (14).

Surely all this is reason enough to doubt Sakel’s reporting. And yet Shorter and Healy conclude that Sakel was the “originator of an effective active treatment in psychiatry” (21). He may have originated something. But an “effective active treatment”? Based on what data? The authors offer us only two anecdotes, one by Sakel and one by a clinician with whom he worked, and a Sakel study whose results other clinics were unable to replicate.

In chapter four the authors return to insulin shock with more “impressive” data: the 1938 report of a statistician who found that insulin shock therapy (IST) raised the recovery rate in schizophrenics from 4 percent in untreated cases to 13 percent in treated cases (58), and a 1941 study by T.D. Rivers and Earl D. Bond, who

... followed up eighty-two insulin shock patients who had been treated rather timidly ... and seventy-one patients who had been treated aggressively in the years 1939-1940. Of those treated timidly, only 32 percent remained well two years later; of those treated aggressively, 61 percent. Of the entire sample of 153 patients, only 17% were still well at four years, about comparable to untreated controls (58).

Here the authors' zeal clearly got the best of them. Regarding the aggressively treated group, Table 1 of Rivers' study presents the data as follows:

Of 71 cases	63%	were recovered or much improved at the end of treatment
“ 53	“ 62%	“ “ “ “ “ “ “ “ “ “ “ 6 months
“ 42	“ 57%	“ “ “ “ “ “ “ “ “ “ “ 1 year
“ 18	“ 61%	“ “ “ “ “ “ “ “ “ “ “ 2 years

(Source: Rivers, Bond, 1941, p. 383)

It's apparent that only 18 of the 71 cases were followed up at two years. All we know for certain is that 11 (61% of 18) of the original 71 cases, only 15 percent, were not relapsed at 2 years. Moreover, Rivers and Bond cautioned that, "In the four year cases we deal with such a small number (23) that we cannot put much reliance on the percentage of recovery" (Rivers and Bond, 1941, p. 383). If the recovery percentage was unreliable for 23 cases, then certainly the follow-up results for 18 cases was even more so.

Similarly, it wasn't 17% of 153 patients given IST who were well at four years. Table 1 states, "Of 23 cases 17% were recovered or much improved at the end of 4 years" (Rivers and Bond, 1941, p. 383). Only 23 of 153 patients were followed up at four years and, as noted above, Rivers and Bond themselves questioned the four year statistic. We know only that 4 (17% of 23) of the original 153 cases, less than 3 percent, were well at four years.

### **Insulin shock's brief reign**

Shorter and Healy tell us that the reign of IST was brief because it was replaced by ECT and chlorpromazine (Thorazine) (59). A 1953 Harold Bourne study in *The Lancet* ("The Insulin Myth") "landed a staggering punch" (59). Two other studies – one that found no difference between insulin treatment and "deep sleep narcosis induced by barbiturates (a weakly effective therapy)" (59) and another that "found insulin no more effective than chlorpromazine" (59) – sealed IST's demise.

We are left with two puzzling questions. Why was a treatment with allegedly impressive data to support it so staggered by a three and a half page review in the *Lancet* and why did psychiatrists abandon it?

The authors offer no satisfying answers. We are told that Bourne's piece provoked "replies of outrage from such experienced researchers as Pullar-Strecker, William Sargent, Linford Rees, and Willy Mayer-Gross, who knew from years of experience that insulin was effective" (59). In the case of Pullar-Strecker it was more of a polite, British, "have another cup of tea?" sort of outrage. In his reply letter to *The Lancet* he wrote, "As the one who introduced insulin therapy into this country almost twenty years ago I should like to comment on Dr. Bourne's very sound article last week" (Pullar-Strecker, 1953, p. 1047).

The principal objection of all the researchers was to Bourne's contention that ECT was more effective than insulin in treating schizophrenia. Pullar-Stecker wrote, "The uninterrupted recovery under insulin, now in its twelfth year, of *even a single case* of 'hopeless schizophrenia' ... should be enough to convince anyone that insulin is the superior treatment here" (Pullar-Stecker, 1952, p. 1047 – emphasis added). Sargant had similar complaints: "Having used both insulin and E.C.T. in the treatment of schizophrenia since 1937, I have still not the slightest doubt of the clinical superiority of insulin in many patients.... Insulin is certainly not a specific treatment for schizophrenia, but the best we have available at present for many patients in the early stages" (Sargant, 1953, p. 1051). Mayer-Gross wrote, "...it [insulin] is the only treatment that holds out some hope of shortening and relieving schizophrenia...." (Mayer-Gross, W., 1953, p. 1153).

"...even a single case..." "...best available..." "...some hope..." – not exactly ringing endorsements. Moreover, none of these critics effectively challenged Bourne's conclusion that studies of insulin in the treatment of acute schizophrenia were "riddled with sources of fallacy" (Bourne, Harold, 1953, 965) and that "Insulin offers the schizophrenic no long-term benefits; at best it hastens improvements that would occur anyhow with simple care in hospital..." (Bourne, H., 1953, p. 967).

Additional data would have helped put the whole question of IST's purported effectiveness for schizophrenia in perspective. For example, in a recent review of *Shock Therapy* in *The New England Journal of Medicine*, Arthur Rifkin wrote that "the evidence for the benefit of ECT in schizophrenia is scarce" (Rifkin, 2008, p. 206). The recent CATIE (Clinical Antipsychotic Trials of Intervention Effectiveness) study of antipsychotic drugs found that three-quarters of the patients "discontinued their assigned treatment owing to inefficacy or intolerable side effects or for other reasons" (Lieberman, Stroup, McEvoy, 2005, p. 1209). Drugs like chlorpromazine (Thorazine) are known to cause permanent brain damage (Cahn, Pol, Lems, 2002; Madsen, Keiding, Karle, 1998; Gur, Maany, Mosley, 1998). They produce a chemical lobotomy (Breggin, 1991, 56), offer no cure, result in much higher rates of relapse (Whitaker, 2005), and shift outcomes away from recovery and toward chronic illness (Whitaker, 2002; Whitaker, 2005).

In other words, insulin shock, a supposedly effective treatment for schizophrenia with impressive data supporting it, was replaced by two treatments, one of which, ECT, has never been shown to be effective and another, chlorpromazine, which is brain damaging and hinders recovery. It doesn't make sense. Do psychiatrists give up treatments they find effective simply due to a few critical reviews? Not according to Mayer-Gross, who argued that insulin-coma therapy "has been severely and authoritatively criticized for many years.... Nevertheless, most clinicians have refused to abandon the method..." (Mayer-Gross, 1953, p. 1153).

### **The inept magician**

It is ultimately unimportant whether insulin shock was more or less effective than the treatments that took its place. Healy and Shorter want the reader to believe that convulsive treatments have always been unfairly maligned, so as to set the stage for their argument that electric shock is just the most recent of such procedures to get a bad rap.

But there is a much more important point to be realized here - and it's one that can hardly be overemphasized. When psychiatrists talk about "effectiveness," the word has to be taken with several grains of salt and a cup and a half of sugar. The bar is set so low that any activity that manages to crawl over it will probably get psychiatry's giddy stamp of approval. (In ECT research, for example, "effectiveness" is typically defined in terms of a response to treatment that lasts one week (Prudic, Sackeim, Devanand, 1990; Prudic, Haskett, Julsant, et al., 1996; Sackeim H., Prudic J., Devanand D.P., et al, 2000).

It's not that subsequent treatments are necessarily any better than the ones that preceded them. Indeed, if Short and Healy are right, and mental health outcomes "are steadily worsening," one might justifiably conclude that treatments are getting worse.\* But since their predecessors were so dismal, it is easy to assume that the new therapies must be better and it takes a while to discover how ineffective they are. Like an inept magician, the psychiatrist stumbles from one bad trick to the next, hoping to hold the crowd's attention, and the audience stays, thinking that things will get better.

Thus the challenge to the psychiatric industry is to convince the public that things are improving when the data suggest just the opposite. Anecdotes are a primary means by which this is accomplished and anecdotes also form the essential thread of Healy and Shorter's fabric. As a reviewer for *The New England Journal of Medicine* wrote, the authors "present many anecdotal paeans to ECT instead of information about randomized controlled trials" (Rifkin, 2008, p. 205).

S & H have a ready reply, which they use as something of a shield in defending ECT against charges that it damages memory. "This is a history, not a chapter in a task force report, and hence it is not our goal to establish whether ECT causes memory problems beyond the relatively short-term difficulties that everyone agrees can arise for some people immediately after treatment" (214).

Their statement is at once disingenuous, yet very revealing. To claim that it's not your goal to establish whether ECT causes memory problems, i.e., to imply that you are taking a "neutral" stance, when your entire work argues only one side of the debate - this is the stuff of clever editorials, not legitimate historical analysis. A history of shock treatment that excuses itself from tackling one of the key issues that makes ECT so controversial - memory loss - and instead substitutes "anecdotal paeans," a few cherry picked studies, and a multitude of frivolous rationales explaining why ECT has received bad press (flower children, Scientologists, the intellectual class, the "premium" our era has placed on memory, spiritual leaders protecting their turf, and cinematic dramatization, to name a few) - such a "history" hardly deserves the title.

Moreover, task force reports are ostensibly based on a current body of research. Healy and Shorter are here confessing, albeit in sideways fashion, that the only proper arena for sorting out

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\*Additional evidence of this comes from a recent 8-state report on individuals who received inpatient or outpatient services from public mental health agencies. They died, on average, nearly 30 years earlier than the general population. The authors of the study noted that this figure used to be 10 years. (National Association of State Mental Health Program Directors, 2006)

the question of memory impairment is the scientific literature, not anecdotes. Of course the same is true when it comes to sorting out other aspects of ECT practice, such as effectiveness and brain damage. In other words, Healy and Shorter are, in effect, admitting that when readers reach the end of their book, they will still not have any substantial answers to the most important questions surrounding shock treatment.

### **Politics and the code of silence**

This is not to say that the scientific literature holds all the answers - far from it. The literature often serves more of a PR function than anything else. "Psychiatry," as Thomas Szasz has pointed out, "is politics – has always been politics. It is politics pure and simple" (Szasz, 2006). We saw this recently in a study published in *The New England Journal of Medicine (NEJM)* that looked at publishing bias related to 12 antidepressant drugs. The researchers analyzed 74 FDA registered antidepressant studies and found that only half (38 = 51%) had positive outcomes. But while 94% of those positive studies found their way into print, only 14% of those with negative or uncertain results were published. According to the *NEJM*, "Not only were positive results more likely to be published, but studies that were not positive, in our opinion, were often published in a way that conveyed a positive outcome" (Turner, Matthews, Linardatos, et al., 2008, p. 256).

Similar tricks abound in the literature on electroconvulsive therapy. Take, for example, the American Psychiatric Association's 1990 task force report on ECT, which devoted 2 out of 124 pages to "Cognitive Side Effects" and only a few sentences to memory loss. The task force cited one 1986 study, by Freeman and Kendall, in claiming, "A small minority of patients, however, report persistent deficits" (American Psychiatric Association, 1991, p. 61) Compare this with what Freeman and Kendall actually wrote: "We were surprised by the large number who complained of memory impairment. Many of them did so spontaneously without being prompted, and a striking 30% felt that their memory had been permanently affected" (Freeman and Kendall, 1986, p. 351). In fact, the researchers noted that these patients had been interviewed by psychiatrists in the hospital where they had been treated and therefore it was possible that there were "a significant number of people in the midground who felt more upset by ECT than they were prepared to tell us" (Freeman and Kendall, 1986, p. 351).

And that's not all. As Healy and Shorter report, the chair of the 1990 task force committee was Duke University psychiatrist Richard Weiner (who they interviewed for the book (xi)). His task force cited three studies in arguing that memory was not persistently impaired by ECT. Weiner himself had been critical of one of those studies in a 1984 paper (Weiner, 1984) and Weiner's own 1986 study found "provocative evidence for what amounts to objective personal memory losses lasting at least six months with BL [bilateral] but not with UL[unilateral] ECT ..." (Weiner, 1986, p. 321). The results, said Weiner, suggested that the cause of the memory loss was "organic rather than functional" (Weiner, 1986, p. 321). The 1990 task force did not even mention the findings of *its own chair* in both these studies!

Discrepancies like this run throughout the ECT literature but none of them make it into Shorter and Healy's book. Their reporting on the memory loss question matches the ECT task force reports in deceptiveness. In their one-paragraph treatment of the "empirical evidence" they refer

to the work of well-known shock researcher, UCLA psychologist Larry Squire. In 1984, they tell us, Squire “finally concluded ... ‘despite repeated efforts it has not yet been possible to demonstrate persisting memory impairment following a standard course of treatment.’ It would be hard to imagine a more definitive statement” (244).

Well, yes, but it would be easy to imagine a less definitive one – if you were aware of Squire’s 1986 study, in which he found “a persisting impairment was present” in autobiographical memory loss of ECT patients. Squire wrote,

It is not yet clear how to evaluate the finding that at seven months after treatment persons who had received bilateral ECT occasionally failed to recognize as familiar even remote events that had occurred many years ago. Specifically, 5 of the 10 persons in our sample denied familiarity to a total of 18 remote events that they had reported as facts before ECT, seven months earlier (Squire, 1986, p. 310).

Healy and Squire skip Squires’ 1986 findings and instead focus on a 1988 *New York Times* story which reported Squire’s expert witness testimony in a bribery-conspiracy trial. Presumably their view is that newspaper reporting of paid trial testimony provides more objective evidence of shock treatment’s impact on memory than actual research. (They may actually have a point there.)

At trial Squire dismissed ECT’s effects as “spotty memory loss going back months before treatment.” “Spotty memory loss” is a favorite euphemism of memory loss apologists. It doesn’t seem to matter how big the spots are. It’s like saying the bombing of Dresden produced spotty building loss. Healy and Squire conveniently leave out (well, it’s at the back of the book, if you read footnotes) the title of the *Times* report, “Experts Say Treatments Affect Recall,” and the story’s paragraph four:

Electroshock therapy is the only one of Ms. Gabel's treatments that is certain to have produced some memory loss, experts said. Even then, the degree of loss depends on how many treatments she had and how much electricity was delivered to her brain. (Kolata, 1988, p. B3)

It was somewhat odd that Healy and Shorter made Squire the star witness in their defense of ECT on the memory loss charge. Squire, as they point out, is a psychologist and the opinions of psychologists regarding memory loss, we are repeatedly warned, are not to be trusted: “Dipping through the professional literature of psychology on ECT is like boring into a steam tunnel” (241).

The psychologist who has generated the most steam is Harold Sackheim, a researcher at the New York State Psychiatric Institute who, the authors concede, is eminently qualified to reach the boiling point. They quote Weiner, who said, “Harold Sackeim is really the top researcher in ECT” (243). In 2000, ECT’s top researcher wrote, “Virtually all patients experience some degree of persistent and, likely, permanent retrograde amnesia.... There are some people, we don’t know how many, going to lose five years of their life” (241).

Needless to say, verdicts like this demand a rhetorical response. “Was Sackeim’s work,” write the authors, “an aberrant deviation from the mainline of ECT thinking? Or did it represent the



triumph of reason in a field sundered by partisanship?” (241) It’s the psychologists versus the psychiatrists and in this melodrama the outcome is a foregone conclusion. In response to Sackeim’s findings, write S & H, “the psychiatrists stood by flabbergasted. First of all, none of the veteran ECT-specialists had ever seen anything like the devastating, permanent abolition of memory of which some anti-ECT activists complained” (243).

Actually many of them did see it. They just weren’t talking about it. In a 2004 trial deposition Sackeim described a conference at which he had spoken. (He doesn’t give the date but says the third APA task force on ECT was being written at the time, so it was around 1999 or 2000. His statement is worth quoting at length:

There was an opportunity, almost a watershed moment for the field in California when a couple hundred practitioners in a couple-day course on ECT -- I was lecturing on the issue of cognition and lecturing about long-term consequences. And one of the very famous people in the field got up, said this doesn't happen, just doesn't happen.

And there was a confrontation where I asked the audience, had they seen it, do they have patients who they believe generally have long-term negative effects. And two-thirds raised their hands. And some other very well known people in the field said, yes, that happens.

And so that was the first time publicly that the field itself said no to the position that it can't happen (Sackeim, 2004, p. 147).

That doesn’t sound like “flabbergasm.” It sounds like psychiatrists knowing what they are doing and making sure no one else does. It took 60 years before they had an “opportunity” to admit to each other what they all knew: ECT was causing permanent memory damage.

*Shock Therapy* also avoids any discussion of the ongoing pattern in ECT research of refusing to design studies that could actually reveal the extent of ECT’s impact on memory and cognitive function. In a January 2007 study published in *Neuropsychopharmacology*, Sackeim and Prudic confessed, “Despite ongoing controversy, there has never been a large-scale, prospective study of the cognitive effects of electroconvulsive therapy” (Sackeim, Prudic, Fuller, 2007, p. 244). Never! In 70 years! Isn’t that something S and H could have ferreted out? Among the numerous revealing findings of this study were the following:

At both the short- and long-term time points, patients treated with BL [BL = bilateral – with the electrodes placed on opposite sides of the person’s head] ECT had greater amnesia for autobiographical events, and the extent of this amnesia was directly related to the number of BL ECT treatments received (Sackeim, Prudic, Fuller, 2007, p. 252).

The average decrement in AMI-SF [a measure of autobiographical memory deficits] scores in patients treated exclusively with BL ECT was 3.4 and 2.8 times the amount of forgetting seen in the healthy comparison groups at the post-ECT and 6-month time points, respectively, suggesting that the deficits were substantial (Sackeim, Prudic, Fuller, 2007, p. 252).

Other measures of cognitive deficit following ECT were equally apparent. The researchers found,

Compared to baseline performance, at the postECT time point the total patient sample showed deficits in the mMMS, sensitivity of the CPT, delayed recall of the BSRT, delayed reproduction on the CFT... (Sackeim, Prudic, Fuller, 2007, p. 248). [The mMMS (modified Mini-Mental State exam) measures global cognitive status. The CPT (Continuous Performance Test) is a measure of attention. ECT victims frequently complain of inability to focus attention. The Buschke Selective Reminding Test (BSRT) and Complex Figure Test (CFT) measure learning and memory ability.]

This study provides the first evidence in a large, prospective sample that the adverse cognitive effects can persist for an extended period, and that they characterize routine treatment with ECT in community settings (Sackeim, Prudic, Fuller, 2007, p. 253).

Sackeim and Prudic's 2007 study was not available for Shorter and Healy. But it doesn't take that much digging to uncover how shallow the research on ECT has been and how much the field has avoided the type of research that would have revealed the effects of which patients continually complain. Even Sackeim and Prudic's 2007 study, as Linda Andre has noted in a detailed analysis available at [ect.org](http://ect.org), was designed to minimize the damaging effects that might be found (Andre, L., 2007). Sackeim and Prudic failed to report the results of several of their tests. This is common in the ECT literature. Research is designed to produce favorable outcomes and when damaging effects are found they are likely not to be reported (Warner, 2006). Shorter and Healy follow the same script.

## **Effectiveness and relapse**

Regarding ECT's effectiveness, H & S are typically chatty - and selective when it comes to the research literature. "The adage among its practitioners was that you were doing something wrong if any fewer than 80 percent of your patients showed improvement" (95). The 80% figure is a popular one in the shocker's guild. It sounds good – not too timid, but not too ambitious either. The American Psychiatric Association website states that "ECT will produce a substantial improvement in at least 80 percent of patients" (APA, 2008). They say this despite the fact that a 2004 study of ECT's effectiveness in a community setting found, "In contrast to the 70%-90% remission rates expected with ECT, remission rates, depending on criteria, were 30.3%-46.7%" (Prudic, Olfson, Marcus, et al., 2004, p. 301). (ECT practitioners would appear to be much better at adages than actual ECT.) S & H make no mention of this study.

It gets worse. Remission in this study was measured, on average, within 3 days of ECT termination, "with 318 of 347 patients (91.6%) evaluated within 10 days" (Prudic, Olfson, Marcus, et al., 2004, p. 304). According to the study, "A longer interval to assessment was associated with less improvement and lower rates of response and remission" (Prudic, Olfson, Marcus, et al., 2004, p. 304). The study found, "...on average, 10 days after ECT, patients had lost 40% of the improvement that accrued over the ECT course" (Prudic, Olfson, Marcus, et al., 2004, p. 304). Thus, if *all* the patients had been evaluated at 10 days post-ECT, remission rates would certainly have been well below 30%.

This might seem respectable, but it's not. A 30% *placebo response* is common in drug studies (Walsh, Seidman, Sysko, and Gould, 2002). A 2002 study in the *Journal of the American Medical*

*Association* reported that 31.9% of the placebo treated depressed patients had a full response (Hypericum Depression Trial Study Group (HDTSG), 2002, p. 1807). (Full response required a HAM-D\* total score of 8 or less). In his review of the placebo-controlled literature on ECT, psychiatrist Colin Ross found, “No study demonstrated a significant difference between real and placebo (sham) ECT at one month post-treatment” (Ross, 2006, p. 17). [In the sham condition, the patient is anesthetized and electrodes are attached to the head but no current is delivered. Neither the patients nor the evaluating psychiatrists know who received real ECT.]

This brings us to the question of relapse, a major problem with ECT that S & H essentially ignore, noting only that one author admitted that “many patients relapsed after treatment” (240). The 2004 Prudic community study cited above found that after 6 months, only 22.5% of patients met the moderate remission criteria and 16.1% met the strict criteria (Prudic, Olfson, Marcus, et al., 2004, p. 310). The median time to relapse for those who met the moderate remission criteria was 8.6 weeks (Prudic, Olfson, Marcus, et al., 2004, p. 310).

All adages aside, this was not really news. By 1990 it was known that the typical ECT candidate – one who had failed several trials of antidepressant drugs – only responded at a 40% - 50% rate (Prudic, Sackeim, Devanand, 1990) – and then soon relapsed (Sackeim, 1990), resulting in an effective response rate close to the 16%-22% Prudic and her colleagues found in 2004.

Shorter and Healy’s failure to address the actual facts concerning effectiveness and relapse is significant. A 2001 study by Sackeim published in the *Journal of the American Medical Association* concluded that, “... almost universal relapse should be expected without effective continuation therapy” (Sackeim, 2001, p. 1305).

### **Doing their own thing**

Another major area of neglect in *Shock Therapy* concerns ECT technique itself, which is widely varied. According to S & H, “In the late 1980’s ... it became the norm to limit the amount of electricity used to doses only marginally in excess of the seizure threshold” (205). This is simply untrue.

The APA itself recommends doses for bilateral ECT that are 1.5 to 2.5 times the amount needed to produce a 25 second seizure. But psychiatrists do their own thing when it comes to ECT dosing. In a 1991 paper, three renowned shock researchers admitted that, “some patients may receive at each and every treatment an electrical dose that is grossly in excess of their threshold (e.g., by up to 4000%)” and, “the practice of using electrical intensities far in excess of that needed to produce seizures undoubtedly contributes to adverse cognitive side effects” (Sackeim, Devanand, Prudic, 1991, pp. 806-807).

In the same paper, the authors wrote, “Commonly, clinicians do not know the degree to which they are exceeding seizure threshold when setting electrical intensity. For some patients with low thresholds, even relatively moderate settings on the device will produce a stimulus that may

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\* HAM-D stands for the Hamilton Rating Scale for Depression. Maximum score on the 17 item scale is 52 points. Higher scores indicate more severe depression.

exceed threshold by 500% or 1000%” (Sackeim, Devanand, Prudic, 1991, p. 810). The formulas that ECT practitioners use to estimate threshold, according to the APA’s own 2001 task force report on ECT, “account for only 40% or less of the variability in initial seizure threshold” (American Psychiatric Association, 2001, p. 160).

In 2002 Rasmussen estimated that the arbitrary nature of threshold estimation may mean that “On a busy ECT service, this may amount to over 100 per year potentially treated with an inadvisable electrical dose” (Rasmussen, 2002, pp. 210-211).

The corollary to the arbitrary nature of ECT practice is that research studies, which limit themselves to the APA recommended doses, grossly underestimate the memory damage that is produced by psychiatrists practicing ECT in the field. Sackeim and Prudic’s 2007 study is the one partial exception to this rule, although as Andre has noted, it too is intentionally flawed. It’s like testing a drug for side effects at a dosage of 25 mg when you know the drug will be commonly prescribed at doses exceeding 100-200 mg. It’s easy to say there is no evidence of something as long as no genuine attempt to collect evidence has occurred.

## **ECT and brain damage**

Healy and Shorter also briefly address the question of ECT caused brain damage, which they call a “half-century old myth” (135). They refer to a 1991 MRI study by Edward Coffey as a “definitive” answer to this question (135). It wasn’t. How do we know? Edward Coffey said so. Three years after his 1991 study Coffey wrote, “Clearly, prospective studies that compare pre- and post-ECT imaging data are required to determine whether ECT causes changes in brain structure” (Coffey, 1994, p. 478). These studies have never been done.

There were numerous flaws in Coffey’s study (Warner, 2006, pp. 32-35). However, Coffey did find brain changes in 5 of 29 subjects; and while he suggested that these effects were “most likely secondary to progression of ongoing cerebrovascular disease during follow-up” (Coffey, 1991, p. 1013), he was cautious in interpreting his results:

Although we failed to find evidence of changes in brain volume after ECT, this does not mean that such changes did not or could not have occurred, since even modern brain imaging technologies have certain inherent limitations (Coffey, 1991, p. 1018).

... it is possible that changes in neuronal density could have occurred that were not detectable with the methods available in this study (Coffey, 1991, p. 1018).

... it is possible that subtle atrophic [shrinking in size] effects in a particular structure may be obscured when that structure is combined with other adjacent structures in the same region (Coffey, 1991, p. 1018).

Still, it is difficult to interpret with certainty worsening of brain MR imaging abnormalities after ECT in the absence of control data describing the natural course of such brain abnormalities (Coffey, 1991, p. 1019).

Shorter and Healy don't mention any of this. It goes without saying that the many studies that do provide evidence of ECT-induced brain damage are also not mentioned, including 1) a 1988 Coffey study, in which he states that one explanation of his evidence was that "...ECT may have caused cerebral atrophy..." (Coffey, 1988, p. 704); 2) A 1990 Andreasen study which found a correlation between the number of previous ECT treatments and increased lateral ventricular volumes (loss of brain tissue) measured by MRI (Andreasen et al, 1990); and 3) a CT scan study which found that patients treated with ECT showed more brain tissue loss (Dolan, 1986). This last study noted, "There was no evidence of association between the presence of these changes and a family history of depression, the duration of depressive illness, the age of onset of illness, the course of illness or exposure to psychotropic medication" (Dolan, 1986, p. 778). There was also no association with age or alcohol use in Dolan's study.

S and H prefer to give us a six-week study of 12 adolescent (most ECT patients are not adolescents) monkeys, only three of which were given ECT, not at the high seizure threshold multiples that characterize ECT in community practice, but at 2.5 times an individual monkey's seizure threshold. Though the study's authors found no brain lesions in the ECT-treated monkeys, they conceded that "the group was too small for us to detect subtle, quantitative changes" and "many more animals would be needed to rule out lesions that occur infrequently" (Dwork et al., 2004, p. 576). But for Healy and Shorter three dead monkeys is sufficient to put the matter to rest and perpetuate the real myth – that there is no evidence of ECT caused brain damage.

## **A final word**

One might compare *Shock Therapy* to a paper doily where the holes take up more area than the paper. In reviewing such a work one is forced to choose a few of the holes to discuss and leave the vast majority untouched. The ECT literature is mostly ignored and the voices of patients are mostly silent and, if heard, quickly dismissed. History, as they say, is written by the victors.

It could be that the influence of longtime ECT advocate Max Fink had something to do with the slant of this book. S & H, while acknowledging that, "This book owes a special debt to Max Fink" and that Fink "gave the manuscript a critical reading," felt compelled to say, "This is in no sense Max's book..." (xi). Well, it is in one sense. The authors write that their research was supported in part by a grant from the Scion Natural Science Association (xi). They leave out the amount, \$33,900 (Department of Family and Community Medicine, University of Toronto, 2008, 6), and the fact that Scion is Fink's private foundation (Spikol, L, 2007). Fink once offered reporters a chance to interview patients after they had undergone ECT – for \$40,000 each, with \$25,000 going to him and \$15,000 to the patient (Breggin, 1991, p. 188). He narrated shock videos for shock machine maker Somatics (Breggin, 1991, p. 192). Shock has long been Fink's cash cow and he's apparently quite willing to lie to keep it that way, making false statements about shock victim Marilyn Rice (Spikol, L, 2007) and misleading the public about the effect of shock treatment on the brain (Breggin, 1991, p. 199).

Healy and Shorter's work does serve one useful purpose. It confirms that psychiatry is indeed "as ideological as it has ever been." But those who are searching for the truth about shock treatment will not find it here.

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[http://www.nasmhpd.org/nasmhpd\\_collections/collection4/meeting\\_presentations/Summer%202006%20cmmish/NASMHPD%20Morbidity%20and%20Mortality%20Slides%20071006.pdf](http://www.nasmhpd.org/nasmhpd_collections/collection4/meeting_presentations/Summer%202006%20cmmish/NASMHPD%20Morbidity%20and%20Mortality%20Slides%20071006.pdf)

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