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Electroconvulsive Therapy (ECT) Testimony
by Daniel B. Fisher, MD, PhD
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Thank you for the opportunity to share my views on ECT with you today. I base my testimony on my thirty years of practice as a board-certified psychiatrist, my five years of postdoctoral neurochemical research at NIMH, and my 19 years as director of the National Empowerment Center, a federally-funded technical assistance center. I am appalled that ECT has never been required to validate its efficacy or its safety before the FDA. I was heartened to hear that in 2008 the FDA did require such verification. However, today I am dismayed to learn that the manufacturers want the status of their devices downgraded from Class III to Class II, or the same classification as a wheelchair.

In my expert opinion and that of a recent review of the ECT literature by Drs. John Read and Richard Bentall (Read and Bentall, 2010), the short-term gains of ECT do not justify its associated brain damage, memory loss, cognitive deficits and increased risk of death. I therefore recommend:

1. That ECT devices continue to be designated as class III devices and
2. That, in keeping with the approval process of a Class III medical device, their use be suspended unless and until their manufacturers can produce independently obtained evidence with government oversight of meaningful long-term efficacy and minimal short- and long-term risks of memory loss, cognitive deficits, brain damage, and mortality to warrant the resumption of its use.

Three of my cases illustrate the negative aspects of ECT:

Case A: I saw a 19 year-old man in an outpatient clinic. He suffered from major depression and was slow to respond to the antidepressant Prozac. He was admitted to an inpatient facility where the psychiatrist immediately started a series of 8 ECT treatments. Upon discharge, his depression had slightly lifted, but he could no longer recognize his friends. He was so distraught over this side effect of the ECT that he hung himself. This case points out that ECT can actually increase suicidality. Indeed research (Black et al, 1989) has shown that, contrary to claims by its proponents, ECT does not decrease suicidality.

Case B: In my capacity as a consultant to the Massachusetts Rehabilitation Commission, I learned that a 51 year-old woman was experiencing memory loss for recent as well as remote events and confusion which intensified once a month. Upon close questioning, she acknowledged that once a month she was given outpatient ECT. She had been threatened by her doctor that if she told anyone she was undergoing ECT, she would be rehospitalized. She wanted to stop the ECT, but was too frightened to see her doctor alone. She requested that I accompany her. She was then able to tell her doctor she wanted to leave his care. She did so and was successfully switched to an antidepressant with fewer side effects. This case illustrates that ECT causes cognitive deficits and both retrograde and antegrade memory loss. The most detailed studies of memory were carried out by Dr. Irving Janis in 1950-51. He interviewed consumers before, immediately after, and three months following ECT. As a control group he also interviewed consumers, who suffered similar mental health issues but received psychotherapy instead of ECT. Janis found, gross gaps and subtle losses of memory and a general slowness and a great effort in recalling details. In some cases, details returned, but only with great effort and with the help of cues provided by the examinee, (Janis and Astrachan, 1951). Janis reports following half of the shocked patients for 2 1/2 - 3 1/2 months after the end of ECT. He found that in each case most of the instances of amnesia persisted. These side effects were also validated by a proponent of ECT, Dr. Harold Sackheim (Sackheim et al, 2007). In a prospective study of 347 consumers his research team found memory and cognitive deficits immediately and six months after ECT. They concluded, "This study provides the first evidence in a large, prospective sample that adverse cognitive effects can

exist for an extended period and that they characterize routine treatment ECT in community settings."

Case C: Staff of a day treatment facility pointed out that a 65 year-old consumer was becoming progressively disoriented and forgetful. We learned she was receiving maintenance ECT. When I asked her if she had been sufficiently informed of the side effects for her to give informed consent, she said no. The ECT was ordered by her psychiatrist and administered by another psychiatrist, she had not previously met. He had her sign the "consent form" as she was being wheeled in to receive pre-ECT anesthesia. In fact, it is impossible for a consumer to give informed consent when they are not given accurate information. The APA guidelines for ECT inaccurately contend that the memory loss with ECT is minimal. Furthermore, the APA consent form drastically underestimates the mortality associated with ECT. The APA states the risk of death is 1 in 10,000 whereas a numerous studies from 1957 through 2004 give rates between 1 in 648 to 1 in 1630 (Impasto, 1957; Pippard and Ellam, 1981; Nuthall et al, 2004). On average, these studies indicate a 10-fold higher rate of death than suggested by the APA (APA, 2001). The APA also suggests that "brain damage should not be included[in the informed consent process] as a risk of treatment (APA, 2001)."

It appears that the APA task force on ECT chose to overlook the considerable body of evidence that ECT produces brain damage. This evidence was well summarized by neuroscientist, Dr. Peter Sterling: "ECS [ECT] unquestionably damages the brain. The damage is due to a variety of known mechanisms:

1) [ECT] is designed to evoke a grand mal epileptic seizure involving massive excitation of cortical neurons that also deliver excitation to lower brain structures. The seizure causes an acute rise in blood pressure well into the hypertensive range, and this frequently causes small hemorrhages in the brain (Madow, 1956). Wherever a hemorrhage occurs in the brain, nerve cells die — and nerve cells are not replaced.

2) [ECT] ruptures the "blood-brain barrier." This barrier normally prevents many substances in the blood from reaching the brain. This protects the brain, which is our most chemically sensitive organ, from a variety of potential insults. Where this barrier is breached, nerve cells are

exposed to insult and may also die. Rupture of this barrier also leads to brain “edema” (swelling), which, since the brain is enclosed by the rigid skull, leads to local arrest of blood supply, anoxia [lack of oxygen], and neuron death.

3) [ECT] causes neurons to release large quantities of the neurotransmitter, glutamate. This chemical excites further neuronal activity, which releases more glutamate, leading to “excito-toxicity”--neurons literally die due to over activity. Such excito-toxicity has been recognized relatively recently and is now a major topic of research. It is known to accompany seizures and over repeated episodes of [ECT] may be a significant contributor to accumulated brain damage. (Sterling, 2000, 2001).

Indeed the case for brain damage associated with ECT is so compelling that psychiatrists from Walter Freeman, who in 1941 introduced frontal lobotomies to the US, to more recently Dr. Peter Breggin, have concluded that the short-term relief produced by ECT is the direct result of its brain-damaging effects. Dr. Freeman wrote, "The greater the damage the more likely the remission of psychotic symptoms... Maybe it will be shown that a mentally ill patient can think more clearly and more constructively with less brain in actual operation (Freeman, 1941)."

According to Dr. Breggin, the only effects produced by ECT are caused the equivalent of a closed head injury to the brain. He states "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. 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 (Breggin, 1981)."

On a personal note, I was a psychiatric inpatient on three occasions. Whenever I or other patients failed to act “appropriately,” the staff would threaten us with ECT. On one visit my friends brought me a copy of One Flew Over the Cuckoo's Nest. For the remainder of my stay on

the ward I lived in constant fear that I might be given ECT just like Randle McMurphy in the book.

How is it possible that in a democracy with the most advanced constitution of any country, a whole class of people can be subjected to brain disabling procedures without regulation by the government? I can only conclude that being labeled mentally ill means your lose your rights and protection under that constitution. I entreat you to protect these labeled people by regulating these devices as they should be under their Class III designation.

BIO: Daniel Fisher received his AB. from Princeton University, and his Ph.D. in biochemistry from the University of Wisconsin in 1968. He carried out neuchemical research at the NIMH from 1968-1973. He obtained an M.D. from George Washington University in 1976. He carried out his residency in adult psychiatry at Harvard Medical School. He is a board-certified psychiatrist practicing in Cambridge, MA. Dr. Fisher, is the Executive Director of the federally-funded, National Empowerment Center in Lawrence, MA. He helped organize the National Coalition for Mental Health Recovery which is a national voice for consumers. He was a member of the New Freedom Commission for Mental Health, 2002-03.

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A handwritten signature in black ink that reads "Daniel B. Fisher, MD, PhD". The signature is written in a cursive style with a large initial 'D' and 'F'.

Daniel B. Fisher,MD. PhD
Executive Director, NEC