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7 Attorneys for Plaintiffs JOSE RIERA;
MICHELLE HIMES; DIANE SCURRAH;
8 DEBORAH CHASE; MARCIA BENJAMIN;
and DANIEL BENJAMIN
9

10 **UNITED STATES DISTRICT COURT**
11 **CENTRAL DISTRICT OF CALIFORNIA**
12

13 JOSE RIERA; MICHELLE HIMES;
14 DIANE SCURRAH; DEBORAH
CHASE; MARCIA BENJAMIN and
15 DANIEL BENJAMIN, individually,
and on behalf of all others similarly
16 situated,

17 Plaintiffs,

18 v.

19 MECTA CORPORATION; SOMATICS,
20 LLC; and DOES 1 through 10, inclusive,

21 Defendants.
22

Case No.: 2:17-cv-06686 RGK(PJWx)

**DECLARATION OF PETER
BREGGIN, MD IN SUPORT
CLASS CERTIFICATION**

23 I, Peter Breggin, declare under penalty of perjury as follows:

24 1. I am a medical doctor (physician) with a specialty in psychiatry. I am licensed to
25 practice medicine in New York State and since 2002 I have an active practice of psychiatry in
26 Ithaca, New York. I also have inactive licenses in Virginia, Maryland, and Washington DC, the
27 area where I practiced until 2002.

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1 2. I graduated from Harvard University with honors in 1958 and Case Western Reserve
2 School of Medicine in 1962, where I conducted four years of psychopharmacology lab research
3 with controlled animal trials under a grant from the National Institute of Mental Health, resulting
4 in the first two published papers in the field of psychopharmacology. In 1963, I earned the
5 highest grade in the country on the psychiatry portion of the National Board of Medical
6 Examiners used to qualify for medical licenses. I completed a mixed internship in medicine and
7 psychiatry at the State University of New York Upstate Medical Center (SUNY). I completed my
8 first year of residency at Harvard's main teaching hospital, working in the Massachusetts Mental
9 Health Center, and a teaching fellowship at Harvard Medical School. I finished my second and
10 third year of psychiatric residence at SUNY. Following that I was a full-time Consultant with
11 the National Institute of Mental Health (NIMH) in Washington, DC while a commissioned officer
12 in the U.S. Public Health Service (1966-1968).

13 3. Throughout my career, I have taught as a faculty member or adjunct professor at
14 multiple universities, including the University of Maryland (1968-1970), Washington School of
15 Psychiatry (1968-1972), George Mason University (1990-1996), Johns Hopkins University
16 (1996-1999), and the State University of New York at Oswego (2007-2008, 2010-2014).

17 4. From 1998 to 2002, I was the Founder and Editor-in-Chief of *Ethical Human*
18 *Sciences and Services: An International Journal of Critical Inquiry* (now titled *Ethical Human*
19 *Psychology and Psychiatry*). I currently serve as an editorial consultant to numerous other
20 publications, including the *International Journal of Risk and Safety in Medicine*.

21 5. I have written numerous publications on the practice of electroconvulsive therapy
22 ("ECT"). A true and correct copy of my Resume is attached as Exhibit A which includes my
23 complete bibliography. Dating back to 1979, I wrote the medical book, *Electroshock: Its Brain-*
24 *Disabling Effects* (New York: Springer), which remains the only medical textbook that focuses on
25 the harms caused by ECT. Since then I have written many medical articles on electroshock
26 treatment, including "Electroshock Therapy and Brain Damage: The Acute Organic Brain
27 Syndrome as Treatment" in *Behavior and Brain Sciences* (1984), "Neuropathology and Cognitive
28 Dysfunction from ECT" in *Psychopharmacology Bulletin* (1986), "Electroshock: Scientific,

1 ethical, and political issues” in *International Journal of Risk & Safety In Medicine* (1998), “The
2 FDA should test the safety of ECT machines” in *International Journal of Risk & Safety in*
3 *Medicine* (2010) and “The Utmost Discretion: How Presumed Prudence Leaves Children
4 Susceptible to Electroshock” in *Children & Society* (2014).

5 6. I have also written many books chapters on ECT and have discussed it in detail in a
6 series of my medical textbooks, most recently, *Brain-Disabling Treatments in Psychiatry: Drugs,*
7 *Electroshock and the Role of the FDA, Second Edition* (New York: Springer Publishing
8 Company, 2008).

9 7. In 1985, the National Institutes of Health (NIH) invited me to be the scientific
10 presenter on the subject of "Neuropathology and Cognitive Dysfunction from ECT" at its
11 *Consensus Development Conference on Electroconvulsive Therapy, June 10-12, 1985.*
12 Consensus Conferences are significant scientific and media events in which acknowledged, well-
13 known experts make presentations on controversial topics and a panel without conflicts of interest
14 renders a consensus from the presentations. The Consensus Conference final statement regarding
15 ECT were published in *JAMA* (“Consensus Conference: Electroconvulsive Therapy,” *Journal of*
16 *the American Medical Association, No. 15, October 1986.*). My scientific presentation, along with
17 others, was individually published (“Neuropathology and Cognitive Dysfunction from ECT” in
18 *Psychopharmacology Bulletin*, 1986).

19 8. Electroconvulsive therapy is the practice of inducing a grand mal motor seizure through
20 application of electricity to the head and brain. It began in 1938, when Ugo Cerletti and Lucio
21 Bini observed the shocking pigs to render them manageable before slaughter. It has been in
22 widespread use across the States, including California for decades.

23 9. ECT is primarily used on patients seeking treatment for major depression. It is also
24 liberally prescribed for a broad range of psychiatric conditions including bipolar disorder,
25 schizophrenia and catatonia. I believe the practice has become more widespread since 1979,
26 when I estimated that 100,000 people received ECT per year in the United States. A report by the
27 California Department of Mental Health indicates that over 18,000 people underwent ECT
28 treatment in California in 2001 alone. While there is no formal record of the exact number of

1 patients who undergo ECT in California each year, my estimate is that it would amount to several
2 thousand per year, perhaps tens of thousands.

3 10. Early in my career, I administered ECT and supervised a ward upon which ECT was
4 performed. Throughout my career I have observed the effects of ECT. Based upon all my
5 education, experience, training and study of ECT to date, it is my opinion, as to a reasonable
6 medical certainty, that ECT inherently causes damage to the brain, causing symptoms such as
7 severe permanent memory loss, cognitive impairment, and apathy and indifference towards
8 oneself and others.

9 11. Prior to 1979, the psychiatric community acknowledged that the purpose of ECT was
10 to damage the brain. In 1979, the year that the FDA first ordered the submission of all safety and
11 effectiveness data relating to ECT treatment, I published my aforementioned medical textbook,
12 *Electroshock: Its Brain-Disabling Effects*. In the book, I quoted from the scientific literature the
13 statements of many leading advocates of ECT that brain damage was the intended effect of ECT.
14 Around this time, because of the negative publicity, the dialogue surrounding ECT shifted away
15 from brain damage, and ECT proponents instead began to assert that ECT is a way of correcting
16 chemical imbalances in the brain. There is no scientific foundation for this recent claim that ECT
17 corrects biochemical imbalances. In fact, by causing widespread dysfunction and harm
18 throughout the brain ECT causes biochemical imbalances, as well as other pathological results.

19 12. ECT universally damages the brains of patients who receive it, and the mechanism
20 of trauma is identical among all ECT victims. Some patients are fortunate enough to escape
21 grossly obvious dysfunctionality, enduring relatively minor cognitive impairment and loss of
22 memory for the days, weeks or months surrounding the treatment. Other patients will experience
23 severe memory losses covering prior decades, as well as continuing memory dysfunction and
24 over all cognitive dysfunction with emotional apathy, disinterest or blunting. Although the
25 degree of harm varies, the nature of the harm caused by ECT is consistently the same, specifically
26 including: (1) retrograde memory loss (past memories injured or destroyed) with the worst losses
27 nearer to the ECT treatments; (2) especially severe memory loss surrounding the ECT itself; (3)
28 anterograde memory loss (a broad term referring to persisting memory and cognitive

1 dysfunction); and (4) degrees of apathy or disinterest.

2 13. The reason that all ECT patients endure similar injuries is that the treatments
3 attempt to provide a suitable amount of current to the brain to produce a seizure. The current and
4 the seizures then produce most of the harm, including through the breakdown of the blood brain
5 barrier, hypertension, anoxia, exhaustion of energy sources, heat injury, and electrical injury.

6 14. **The result in all cases without exception is a concussive-like traumatic brain**
7 **injury from every single effective treatment.** The immediate result of this injury is a total
8 disruption of the brain's electrical pattern, driving the recording needle on the EEG strip into a
9 series of explosive, jagged peaks. This is often followed by flat-lining, with a straight line on the
10 EEG indicating that the brain has temporarily stopped functioning, at least in respect to this gross
11 measurement of activity. If the ECT treatment proceeds routinely, the patient is immediately
12 driven into a comatose state. Recovery from the coma then requires several minutes or more in a
13 specialized recovery room under constant supervision. The individual then awakens in a
14 confused state, usually with apathy, and with no memory of what has happened. As the ECTs
15 increase in number, the patient typically awakens from the coma with increasing amounts of brain
16 dysfunction and injury, often with headaches and nausea. There can be no legitimate doubt that
17 ECT damages the brain and mind—no more than there can be about repeated blows on the head
18 that render an individual comatose and then confused and disoriented on awakening. The only
19 question is how much recovery occurs—and anyone who claims that such repeated assaults on
20 the brain are harmless is ignoring the fact that repeated severe traumatic injuries to the brain that
21 cause coma will inevitably leave persistent negative aftereffects to the brain and mind.

22 15. No mechanism of action by which ECT “treats” depression has been identified or
23 proven to this day by the advocates of the treatment; but there is considerable evidence that the
24 apathy and disinterest caused by the treatment is mistaken for improvement by some patients,
25 families and physicians.

26 16. Some ECT advocates claim that ECT reduces the risk of suicide. This is an easy
27 claim to test, because the endpoint, suicide, can be easily measured and recorded. Yet there is no
28 sound scientific evidence that ECT reduces the risk of suicide while there is some evidence that it

1 increases the risk, probably because of the despair patients feel when they realize they have been
2 harmed.

3 17. The “newer” and allegedly “modified” forms of ECT are not different or less
4 harmful than the original form, as both apply enough electricity to the head of a patient to induce
5 a major motor seizure. It is impossible to induce a major motor seizure through application of
6 electricity to the cranium without causing traumatic brain injury. Indeed, contemporary ECT is
7 more damaging to the brain because it requires much higher energy doses in order to produce a
8 seizure in patients who given prior sedatives for sleep or anxiety, and then anesthesia during the
9 ECT treatments. Sedatives and anesthesia increase the seizure threshold, requiring these more
10 traumatic doses of electricity. In previous years 200 milliamps of electrical current were
11 commonly used in humans as well as in animal experiments to produce seizures as a part of ECT,
12 while today the doses produced by the machines are over 1,000 milliamps.

13 18. The clinical markers of brain damage and chronic traumatic encephalopathy
14 resulting from ECT include pinpoint hemorrhages, neurogenesis, scattered cell death in the
15 regions beneath the electrodes, vascular wall damage, gliosis, nerve cell abnormalities, dilated
16 blood vessels, and other markers. Brain damage caused by ECT to an individual patient can
17 sometimes be documented by brain scans, electroencephalograms, and autopsy studies. The most
18 sensitive methods for detecting the extent of brain damage from any cause, including ECT, are a
19 clinical interview by an experienced and well-informed clinician who involves the family and
20 neuropsychiatric testing by an experienced and well-informed psychologist. It is my opinion,
21 that the application of a large enough electric current to induce a grand mall or generalized
22 seizure with unconsciousness causes brain injury is well supported by the medical community and
23 findings developed over a significant time in scientifically reliable publications. The following
24 publications confirm pathology damage in the brain or memory and cognitive dysfunction to
25 indicate an underlying physical damage:

26 Alpers, B. (1946). The brain changes associated with electrical shock treatment. A
27 critical review. *Journal-Lancet*, 66, 363-369.

28 Alpers, B. & Hughes, J. (1942a). The brain changes in electrically induced convulsions

in cats. *Archives of Neurology and Psychiatry*, 47, 385-398.

1 Alpers, B. & Hughes, J. (1942b). The brain changes in electrically induced
2 convulsions in the human. *Journal of Neuropathology and Experimental*
3 *Neurology*, 1, 173-180.

4 Babayan, E. (1985). The structure of psychiatry in the Soviet Union. New York:
5 International Universities Press.

6 Barrera S, Lewis N, Pacella B, et al. (1942). Brain changes associated with electrically
7 induced seizures. Trans Amer Neurol Assoc. Richmond, Va., William Byrd Press,
8 pp 31-35

9 Boyle, G. (1986, November). Concussion of the brain with electroconvulsive shock
10 therapy (ECT): An appropriate treatment for depression and suicidal ideation?
11 *Australian Clinical Psychology*, XX, pp. 21-27.

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

13 Breggin, P. (1980). Brain-disabling therapies. Chapter 23 in Valenstein E (ed.), *The*
14 *Psychosurgery Debate: Scientific, Legal and Ethical Perspectives* (pp. 467-505).
15 San Francisco, WH Freeman.

16 Breggin, P. (1981). Disabling the brain with electroshock. M. Dongier and & E.
17 Wittkower (Eds.), *Divergent Views in Psychiatry* (pp. 247-271). Hagerstown, MD:
18 Harper & Row.

19 Breggin, P. (1986). Neuropathology and cognitive dysfunction from ECT. [Presented
20 at the Consensus Development Conference on Electroconvulsive Therapy,
21 sponsored by NIMH and NIH, 1985.] *Psychopharmacology Bulletin*, 22, 476-479.

22 Breggin, Peter. Brain Disabling Treatments in Psychiatry. Chapter 8, "Electroshock for
23 Depression." New York: Springer Publishing Company, 1997.

24 Breggin, P. (2007). ECT damages the brain: Disturbing news for patients and shock
25 doctors alike. *Ethical Human Psychology and Psychiatry*, 9, 83-86.

26 Breggin, Peter. Brain-Disabling Treatments in Psychiatry: Drugs, Electroshock, and
27 the Psychopharmaceutical Complex. Second Edition, Chapter 9, "Electroshock for
28 Depression", Springer Publishing Company, New York, 2008.

- 1 Breggin, P. (2010). The FDA should test the safety of ECT machines. *International*
2 *Journal of Risk & Safety in Medicine*, 22, 89-92.
- 3 Breggin, P. (2014). For joint authorship, see van Daalen-Smith, et al. (2014).
- 4 Cameron, D. G. (1994, Winter/Spring). ECT: Sham statistics, the myth of convulsive
5 therapy and the case for consumer misinformation. *Journal of Mind and Behavior*,
6 15, 177–198.
- 7 Cerletti U: Old and new information about electroshock. *Am. J. Psychiatry*, 107:87-
8 94,1950
- 9 Cerletti U: Electroshock therapy. *JGin Exper Psychopath* 15:191-217, 1954
- 10 Cerletti U: Electroshock therapy, in *The Great Physiodynamic Therapies in Psychiatry:*
11 *An Historical Reappraisal*. Ed Sackle AM, et al. New York, Hoeber-Harper, 1956.
12 Reprinted in *The Age of Madness*, Ed Szasz TS. Garden City, NY, Anchor
13 Press/Doubleday, 1973
- 14 Cerletti U, Bini L: L'electroshock: le alterazioni istopatologiche del sistema nervoso in
15 seguito all'. *E S Riv Sper Freniatr ecc* 64,1940
- 16 Consensus Conference: on Electroconvulsive Therapy. (1985). *Journal of the*
17 *American Medical Association*, 245, 2103–2108.
- 18 Daniel, W., Crovitz, H., Weiner, R., and & Rogers, H. (1982). The effects of ECT
19 modifications on autobiographical and verbal memory. *Biological Psychiatry*, 17,
20 919–924.
- 21 Ferraro A, Roizen L (1949). Cerebral morphologic changes in monkeys subjected to a
22 large number of electrically induced convulsions. *Am J Psychiatry* 106:278-284.
- 23 Ferraro A, Roizen L, Helford M. (1946). Morphologic changes in the brain of monkeys
24 following electrically induced convulsions. *Neuropathol Exp Neural* 5:285-308.
- 25 Fink, M. (1957). A unified theory of the action of the physiodynamic therapies.
26 *Journal of Hillside Hospital*, 6, 197–206.
- 27 Fink, M. (1966). Cholinergic aspects of convulsive therapy. *Journal of Nervous and*
28 *Mental Disease*, 142, 475–484.

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- 1 Fink, M. (1974). Induced seizures and human behavior. In M. Fink, S. Kety, J.
2 McGaugh, and & T. Williams (Eds.), *Psychobiology of convulsive therapy*. New
3 York: John Wiley.
- 4 Frank, L. (1990). Electroshock: Death, brain damage, memory loss, and brainwashing.
5 *Journal of Mind and Behavior, 11*, 489–512.
- 6 Friedberg, J. (1977). Shock treatment, brain damage, and memory loss: A neurological
7 perspective. *American Journal of Psychiatry, 134*, 1010–1014.
- 8 Halpern L, Peyser E. (1953). The effect of various convulsive procedures on the cranial
9 vessels of the dog angiographically visualized. *J. Neuropathol. Exp. Neural*
10 *12:277-282.*
- 11 Hartelius, H. (1952). Cerebral changes following electrically induced convulsions. *Acta*
12 *Psychiatrica Neurologica Scandinavica, 77*(Suppl. XX), 1–128.
- 13 Hartelius, Book Review of Hartelius, 1952 (1953) *AMA Archives of Neurology 7* (5),
14 685-686.
- 15 Heilbrunn G, Weil, A. (1942). Pathologic changes in the central nervous system in
16 experimental electric shock. *Arch. Neurol. Psychiatry 47:918-927.*
- 17 Impastato D. (1957). Prevention of fatalities in electroshock therapy. *Dis. Nerv. Syst.*
18 *18*(Sec 2):34-75, 1957.
- 19 Janis, I. L. (1948). Memory loss following electroconvulsive treatments. *Journal of*
20 *Personality, 17*, 29–32.
- 21 Janis, I. L. (1950). Psychological effects of electric convulsive treatments. *Journal of*
22 *Nervous and Mental Disease, 111*, 359–397, 469–489.
- 23 Janis, I. L., and & Astrachan, M. (1951). The effect of electroconvulsive treatments on
24 memory efficiency. *Journal of Abnormal Psychology, 46*, 501–511.
- 25 Kahn, R., Fink, M., and & Weinstein, E. (1956). Relation of amobarbital test to clinical
26 improvement in electroshock. *Archives of Neurology and Psychiatry, 76*, 23–29.
- 27 Maletzky, B. M. (1981). Multiple-monitored electroconvulsive therapy. *Boca Raton,*
28 *FL: CRC Press.*

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- 1 Meldrum, B. S., and & Brierley, J. B. (1973, January). Prolonged epileptic seizures in
2 primates: Ischemic cell change and its relation to ictal physiological events.
3 *Archives of Neurology*, 28, 10–17.
- 4 Meldrum, B. S., Horton, R. W., and & Brierley, J. B. (1974). Epileptic brain damage in
5 adolescent baboons following seizures induced by allylglycine. *Brain*, 97, 407–
6 418.
- 7 Meldrum, B. S., Vigouroux, R. A., and & Brierley, J. B. (1973). Systematic factors and
8 epileptic brain damage: Prolonged seizures in paralyzed, artificially ventilated
9 baboons. *Archives of Neurology*, 29, 82–87.
- 10 Neuberger, K., Whitehead, H., Rutledge, E. & Ebaugh, F. (1942). Pathologic changes
11 in the brains of dogs given repeated electric shocks. *American Journal of Medical
12 Science*, 204, 381-387.
- 13 Pettinati, H., and & Bonner, K. (1984). Cognitive functioning in depressed geriatric
14 patients with a history of ECT. *American Journal of Psychiatry*, 141, 49–52.
- 15 Sackeim, H., Prudic, J., Devanand, D., Kiersky, J., Fitzsimons, L., Moody, B.,
16 McElhiney, M., Coleman, E., and Settembrino, Jet al. (1993). Effects of stimulus
17 intensity and electrode placement on the efficacy and cognitive effects of
18 electroconvulsive therapy. *New England Journal of Medicine*, 328, 839–846.
- 19 Sackeim, H., Prudic, J., Fuller, R., Keilp, J., Lavori, P., &and Olfson, M. (2007). The
20 cognitive effects of electroconvulsive therapy in community settings.
21 *Neuropsychopharmacology*, 32, 244–254.
- 22 Squire, L., and & Slater, P. (1983). Electroconvulsive therapy and complaints of
23 memory dysfunction: A prospective three-year follow-up study. *British Journal of
24 Psychiatry*, 142, 1–8.
- 25 Templer, D. (1992). ECT and permanent brain damage. In D. I. Templer, L. C.
26 Hartlage, and & W. G. Cannon (Eds.), Preventable brain damage (pp. 72–79). *New
27 York: Springer*.
- 28 Templer, D., Hartlage, L., & and Cannon, W. (Eds.). (1992). (Eds.). Preventable brain
damage. *New York: Springer*.

Templer, D., and Veleber, D. (1982). Can ECT permanently harm the brain? *Clinical Neuropsychology*, 4,(2), 62–66.

van Daalen-Smith, C.; Adam, S.; **Breggin, P.**; and LeFrançois, B. (2014) The Utmost Discretion: How Presumed Prudence Leaves Children Susceptible to Electroshock. *Children & Society*, 28, 205-217.

19. Memory impairment such as that observed after ECT does not occur naturally, nor is it caused by depression or other psychiatric disorders. The memory loss follows the typical, expected pattern following a discrete, traumatic physical injury to the brain and in fact is similar in its clinical effects to other forms of injury to the head and brain. The possible exception is that ECT seems to produce an especially drastic impact upon personal memories of one's experiences in life, such as family celebrations, holidays, work accomplishments, and educational experiences. For this reason, the harm caused by ECT is particularly destructive to personal identity.

20. Based upon my active involvement in this industry, my experience, training and review of all relevant materials including the nature of the "informed consent" that is generally discussed in the medical communities that offer ECT treatment, physicians that administer ECT do not generally acknowledge or advise of any risk of brain damage, permanent memory loss, or the loss of self that ECT victims frequently report. This is often very discouraging to patients who do not understand why their cognitive abilities have been so severely affected following ECT. Many health professionals tell patients injured by ECT that it is harmless and that their perceived dysfunction in the brain and mind is a "mental illness."

21. The psychiatric profession is keenly influenced by device manufacturers' research and required FDA reporting. The Manufacturer and User Facility Device Experience ("MAUDE") database houses medical device reports submitted to the FDA by mandatory reporters and serves as a primary source of information for psychiatrists and other medical professionals to rely on in evaluating and informing patients of the relative risk and safety of utilizing medical devices.

22. If the manufacturers fully performed their reporting and testing requirements, the psychiatric community would be informed of all risks of ECT through the required mandatory

1 reporting of any adverse events required to be reported and/or addressed by manufacturers in the
2 MAUDE database.

3 23. If the ECT device manufacturers had reported upon any adverse events associated
4 with the administration of ECT in the use of their devices to the FDA as required so that they
5 appear within the MAUDE database, the psychiatric community would have utilized the MAUDE
6 database reporting as an avenue to become informed of such untoward events. Such reporting
7 associated with ECT provides the medical community as a whole with information regarding the
8 risks of utilizing the ECT procedures and in informing our patients of known risks, the dangers
9 and the inherent damages known to be universally caused by ECT. Had there been reporting over
10 the years as required, physicians administering ECT would have been apprised of the grave
11 dangers inherent in ECT in time to prevent injury.

12 24. I have served as the expert witness in numerous actions where adverse events, such
13 as brain damage, have occurred as a result of ECT. Despite these actions where adverse events
14 were alleged and did occur, I am not aware that the manufacturers of ECT devices, including
15 MECTA Corporation and Somatics, LLC investigated or reported to the FDA those adverse
16 events and understand they have continued to manufacture, sell and distribute their ECT
17 machines. I am not aware of any reporting of any such known adverse ECT events reported by
18 any ECT manufacturer within the FDA's MAUDE database.

19 25. In the previous litigation actions that I have been involved addressing the injuries
20 caused by ECT, the defense has often portrayed the individual plaintiffs' injuries as stand-alone
21 events, rather than the remarkably uniform result of an invariably injurious psychiatric practice
22 that has repeated itself continuously over the years that ECT has been utilized in the psychiatric
23 community.

24 26. I believe ECT is still available as a treatment methodology and remaining on the
25 market today because of the substantial influence and power of the psychiatric lobby which gains
26 from and supports ECT. Based upon my experience and involvement, it is not uncommon for
27 psychiatrists to typically charge whatever the insurance will cover for a session of ECT. In
28 addition, anesthesiologists and the facility, as well as others, are all compensated from an ECT

1 practice where hospitals charge considerably for the procedure. The proceeds from ECT,
2 typically paid by Medicare, are often sufficient to support the profitability of individual
3 psychiatrists and the entire psychiatric department at healthcare facilities.

4 27. Typical consent forms that patients sign before receiving ECT are routinely and
5 uniformly inadequate by not disclosing the known risks of long-term damage that occurs from
6 ECT. Typical consent forms provided to most ECT patients that I have reviewed, including the
7 standard APA consent forms, do not inform the patient that ECT inherently damages the brain,
8 nor do they warn of the risk of permanent memory loss and the probable long-term cognitive
9 impairment that can occur. These consent forms generally warn only of risks such as nausea,
10 headaches, and short-term memory loss which would not discourage patients and their families
11 from ECT treatment.

12 28. The adverse events that have occurred following the administration of ECT over the
13 past several decades have clearly demonstrated that the certainty of damage to the brain from
14 ECT, the risk of permanent memory loss and the probable long term cognitive impairment are
15 risks that should have been disclosed to any patient receiving ECT. Had Defendants populated the
16 MAUDE database with reports of reasonably known adverse events by filing adverse event
17 reports with the FDA as required, the treating psychiatrists of members of the putative class
18 would have been in a position to warn members of the putative class of the latent dangers inherent
19 in ECT treatment in time to prevent their injuries.

20 29. All of the information I have provided here is documented in my dozens of peer-
21 reviewed articles and scientific books. I also provide the profession and the public with a free
22 ECT Resource Center on my website, www.breggin.com which contains more than a hundred
23 scientific documents, including my entire book, *Electroshock: Its Brain-Disabling Effects*. The
24 Resource center can also be reached directly at www.123ECT.com.

25 I declare under penalty of perjury the foregoing is true and correct. Executed this ___ day
26 of December, 2017 at Ithaca, New York.

27 _____
Peter Breggin, M.D.