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Electroconvulsive Therapy and Brain Damage: Survey of the Evidence From a Philosophical Promontory

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In a combination of literature review and theoretical article, the author analyzes a broad variety of scientific and real-world evidence that iatrogenic brain damage results from electroconvulsive therapy (ECT). The author critically reevaluates the evidence using knowledge of basic biology and logic, and, to a lesser extent, the author makes ethical observations and legal implications. Despite many scientific and governmental authorities having concluded that ECT does not cause brain damage, there is significant evidence that ECT has indeed caused brain damage in some patients, both historically and recently, and evidence that it always causes some form or degree of brain damage.

Keywords: electroconvulsive therapy (ECT); electroshock; shock therapy; brain injury; philosophy of science; iatrogenesis

Electroconvulsive therapy (ECT; electroshock) is a psychiatric treatment that involves using electricity to create an artificial seizure in a patient, for which some evidence shows relief of psychiatric symptoms, in some patients. The safety issue of whether ECT causes brain damage has been studied and written about extensively. Major psychiatric and medical authorities have asserted or concluded that ECT does not cause brain damage, and they have cited numerous studies that purport to support that conclusion. Among them is a 2011 U.S. Food and Drug Administration (FDA) systematic review prepared for the Neurological Devices Panel (FDA, 2011). In Section 4.7, Neuropathological Changes, the agency's review cites several studies giving evidence against the hypothesis of ECT-induced brain damage, finding little scientific evidence of neuropathology caused by ECT (pp. 33–35). The review even states, "There is no evidence to suggest that ECT causes brain damage" (p. 37).

However, important reasons remain to dispute these claims. This article will examine scientific, logical, and real-world evidence of the physical effects of ECT on the brain. It will evaluate whether this evidence supports the assertion that ECT never causes brain damage and whether some ECT-induced brain changes may have been misinterpreted.

This article will not focus on the effects on memory and cognition or on whether ECT is effectual or moral, although it will briefly touch on those issues as they relate to the central question. The opinion of this author is that all human thought is biased, and this article will make no pretense of neutrality but will strive to make a scrupulous evaluation of the evidence. By no means will it be all-encompassing, but rather a contemplative reconsideration of selected evidence from unique considerations by means of critical thinking and some elementary philosophy and biology.

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CHANGES IN BRAIN STRUCTURE

Multitudinous studies have examined whether ECT changes brain structure. Some scientific studies and reviews find no evidence of structural brain damage (Coffey et al., 1991; Weiner, 1984), supporting assertions of other prominent experts that ECT is safe and does not cause brain damage.

In recent years, the media has often parroted these claims, with one example being an article on the website of *Discover* magazine by popular science writer Carl Zimmer. The article calls ECT's "safety questions put to rest" (Zimmer, 2012, para. 10). An article for *The Atlantic* by Dan Hurley, another popular science writer, contains an interview with psychiatrist Sarah Hollingsworth Lisanby and gives a mostly glowing depiction of the treatment in its contemporary form, with no mention of brain damage (Hurley, 2015). On March 17, 2016, Lisanby, in her professional role with the National Institute of Mental Health (NIMH), held a lengthy question-and-answer session about ECT through Facebook, in which she stated in answer to one question, "Research shows that ECT does not cause measurable brain damage" (NIMH, 2016). More interestingly, on January 17, 2017, Lisanby went on *The Dr. Oz Show* with celebrity doctor Mehmet Oz to further promote ECT (Sugarman, 2017). In a 2016 article for Boston Globe Media's health news site *STAT*, psychiatry volunteer Kate G. Farber and financially conflicted psychiatrist and ECT specialist Charles H. Kellner write, "So where does the [ECT] 'controversy' come from? Mainly from a combination of outdated information and popular culture" (Farber & Kellner, 2016, para. 3). Ironically, not only is this article contributing to a pop culture understanding of ECT, but this statement is also a gross mischaracterization of the arguments of anti-ECT activists. It is an example of the straw man fallacy, the logical fallacy of misrepresenting an opponent's argument as a weaker argument (straw man) than the one the opponent has actually given (Aikin & Casey, 2011; E. D. Cohen, 2009, pp. 383–386; Lewiński, 2011). Kellner and Farber also assert, "Severe memory loss is a rare side effect, while claims of brain damage are unfounded." They never address the actual basis of those claims.

Structural Brain Damage

Contrary to these straw man arguments, some studies have indicated detrimental structural changes to the human brain. Jim Gottstein, president of the Law Project for Psychiatric Rights apparently identifies some in a formal comment (Gottstein, 2016) responding to the FDA's public dockets for a proposed order on ECT, to reclassify the ECT device under specific conditions (FDA, 2015a).

A 2002 study identified by Gottstein showed a correlation between ECT treatment and right fronto-striatal atrophy (P. J. Shah, Glabus, Goodwin, & Ebmeier, 2002). This study compared magnetic resonance images of 20 patients with "treatment-resistant depression" (TRD), 20 "recovered" patients, and 20 "healthy" controls. Those categorized in the TRD group had not received ECT for at least 3 months prior to the study and lacked any history of intracranial pathology or surgery (p. 434). The researchers elected to use total number of ECT treatments as a measure of cumulative illness severity (p. 437). They found,

> Increasing cumulative ECT correlated extensively with reduced bilateral superior frontal gyri, bilateral superior frontal and inferior parietal gyri, bilateral medial and superior temporal gyri and bilateral caudate grey matter density in the TRD group (Fig. 2) upon VBA [voxel-based analysis].

> The reduced grey matter density correlations with ECT were unaffected, even after accounting for the current severity of depression (using the HRSD score [Hamilton Rating Scale for Depression] as a covariate).

In the "Discussion" section, they state,

> Although it could be argued that the differences were the effects of ECT, there is little current evidence that ECT can produce permanent hippocampal or other structural brain changes (Devanand et al., 1994). Because of this, and because the total number of ECT treatments, total duration of in-patient stay and total number of hospitalisations were closely inter-correlated, it seemed reasonable to regard the total number of ECT treatments administered as being an index of cumulative severity. However, the possibility that the findings are ECT-related cannot be discounted. Similarly, all the patients with TRD were medicated, as were about half of the recovered patients. It was not possible to withdraw medication on these subjects. (p. 439)

So, although the authors were inclined to attribute the regional brain atrophy to depression, they admitted that ECT was used as the marker of illness severity, making the perceived correlation between brain atrophy and depression more accurately a correlation between brain atrophy and ECT. Also, potentially confounding variables of medication were not controlled.

Gottstein cites additional studies that he believes show correlations between ECT and detrimental structural brain changes, such as those of Weinberger, Torrey, Neophytides, and Wyatt (1979); Calloway, Dolan, Jacoby, and Levy (1981); Dolan, Calloway, Thacker, and Mann (1986); Figiel, Coffey, Djang, Hoffman, and Doraiswamy (1990); and Diehl et al. (1994). This author was unable to obtain full copies of those articles but did read the abstracts. Only having access to the abstracts, it's impossible to examine the rigor of these studies' methodology or analysis. Therefore, this author chooses not to personally speculate about the soundness of Gottstein's evaluations, whereas recognizing that Gottstein raises important concerns about these studies that should be thoroughly vetted.

It is interesting to note that despite being 154 pages in length, perhaps making it the longest literature review on ECT done by a U.S. government agency, *none* of these studies are addressed in the *FDA Executive Summary Prepared for the January 27–28, 2011 meeting of the Neurological Devices Panel*. This 2011 FDA review states in Section 4.7 that a search was conducted via PubMed to review the literature regarding neuropathological changes associated with ECT and that studies were evaluated for scientific rigor by a neuroscientist (p. 33). Yet, that search failed to identify any of these studies cited by Gottstein.

Even if reevaluations of the methodology, data, and/or analyses showed all of these studies' implications of ECT-induced brain injury to be scientifically unsound, one must remember the history of medical science's blunders and reversals on such things as lobotomy (Diefenbach, Diefenbach, Baumeister, & West, 1999), smoking (HemOnc Today, 2009), homosexuality (Drescher, 2015), and eugenics (A. S. Cohen, 2016; Denhoed, 2016). There is also the wisdom of understanding the logical fallacy of the *argumentum ad ignorantiam*, the argument from ignorance or appeal to ignorance. Put simply, this is the fallacy of concluding that a proposition is false merely because it has not yet been proven true or vice versa (Lander University, 2009; E. D. Cohen, 2009, pp. 355–358; Walton, 1999). An antidote to this fallacy is the folk wisdom that absence of proof is not proof of absence.

Structural Brain Growth

Then there are the studies that indicate beneficial structural brain changes, growth of regions of the brain.

Sartorius et al. (2016) examined magnetic resonance imaging (MRI) of 18 patients diagnosed with a major depressive episode who were administered a mean number of 11.3 ± 4.8 individual ECT sessions (p. 508). They found that ECT led to increased gray matter, especially in the hippocampus and amygdala, as well as increased cortical thickness. The authors go so far as to declare, "Our data add further evidence to the hypothesis that ECT enables plasticity falsifying older ideas of ECT induced 'brain damaging'" (p. 507).

Other studies that show brain growth include ones by Nobuhara et al. (2004); Tendolkar et al. (2013); Nordanskog, Larsson, Larsson, and Johanson (2014); Bouckaert et al. (2016); Joshi et al. (2016).

Assuming these studies are sound, it sounds comforting, even encouraging—scientific evidence that rather than ECT causing brain damage, it causes brain growth.

But, as an old saying goes, things are not always what they seem, and here is an example of the need for critical thinking especially when it comes to scientific data. Collecting data takes one set of skills. Interpreting data beyond mere statistics takes another.

Let us temporarily suppose that the data and analyses in these studies showing brain growth are correct—that ECT induces growth or regeneration in a diseased brain. Even if this is true, that regeneration needs to be put into context. What is the nature of the event that precipitates the regeneration?

In animals, damage to tissue by physical injury or the entry of pathogens triggers an inflammatory response (Campbell & Reece, 2005, pp. 901–902, 948, Glossary; Talaro, 2008, pp. 432–433, Glossary). It stimulates immune reactivity, works to block the spread of infectious agents, and aids in tissue repair. Signs and symptoms of inflammation include fever, pain, soreness, and swelling. Plants also have immune systems, which have not only differences but also similarities to animal immune systems (Jones & Dangl, 2006).

If a person cuts off a branch of a tree, then in months, weeks, perhaps even days, one may see sap flow around the wound, if the tree is resilient enough to overcome the damage. Often, there will be visible tissue growth around the site of damage, part of the organism's homeostatic responses to mitigate the damage or infection. If researchers inflict injuries in a laboratory animal, similar healing mechanisms will be triggered in the body of the animal, and some animals have remarkable powers of regeneration.

Yet, it would not necessarily be right to say that cutting off the limb of the tree is beneficial to the tree. And in most cases, it would be a poor argument to say that inflicting injuries in the laboratory animal is therapeutic to the animal, despite the animal's ability to regenerate. Each time damage is inflicted in an organism with a competent immune system, it triggers the organism's automatic repair response, but the cause of that response is still damage. Some plants and animals can regenerate from these injuries over and over again, but each time, energy is expended and there are other physiological costs.

When a plant or animal is damaged, it may not always be resilient enough to overcome the damage, even when the automatic repair response is triggered. If the injury is severe, oftentimes, the body's natural repair mechanisms will be unable to completely restore the damaged tissue to its previous functional state. If the damage is frequent, then despite the repeated triggering of the organism's natural healing responses, the damage may become debilitating, outweighing the regeneration.

It may be counterintuitive, but what if abnormal brain growth is actually a medical sign of brain injury? What if this regeneration is actually evidence of the brain attempting to heal itself from physical damage?

We might not have enough information to answer those questions, but at least, we can conclude this: Tissue damage (A) and tissue growth (B) are not mutually exclusive. The presence of B does not prove the absence of A. Therefore, Sartorius et al. (2016) are committing a logical overreach when they state, "Our data add further evidence to the hypothesis that ECT enables plasticity falsifying older ideas of ECT induced 'brain damaging,'" specifically the part about their data "falsifying" the alternative, as others who engage in this reasoning. (The syllogism: *Either A or B is true, but not both. B is true. Therefore, A is false.*) This type of logical fallacy is called the fallacy of the alternative disjunct or affirming a disjunct (E. D. Cohen, 2009, pp. 60–64; Curtis, 2014; Green, 1998), along with several other names. Not all exclusive disjunctive syllogisms are illogical, but this one is, because the two conditions are not exclusive, so the first premise is false. In psychology, this may be an example of confirmation bias, a cognitive bias explained in "Cognitive Biases and Statistical Biases" section of this article.

Yet, further considerations are that form is not the same thing as function, and quantity is not the same thing as quality. Although several diseases and types of injury are associated with loss of brain volume (general or localized) or reduction of brain cells, higher brain volume or higher number of brain cells does not always mean increased quality of neurocognitive functioning. An example of this is the findings of a 2011 study by Kanai, Dong, Bahrami, and Rees, which examined the relationship between distractibility and the brain's frontal and parietal lobes, using Cognitive Failures Questionnaire (CFQ) scores and MRI brain scans of a sample of 145 "healthy" adults. The study found that higher gray matter density of the left superior parietal lobe (SPL) had a positive correlation with distractibility, indicating that more gray matter in this area causes more difficulty in concentration. From the "Discussion" section,

> We found that the gray matter density of the left SPL was larger among highly distractible individuals. This positive correlation is interesting, because it has previously been implicitly assumed that a larger cortical volume or greater gray matter density is associated with better performance. Here, we show that greater gray matter density in adults can be associated with poorer performance. (p. 6625)

They go on to cite research showing reduction of gray matter in the cortex as the human brain matures because of pruning of neurons to increase efficiency, as well as studies that they believe show smaller cortical volume is associated with better performance in specific tasks. If this is true, then although increasing gray matter in parts of the brain may be beneficial, increases in certain other parts may signify cognitive harm, even brain damage. The timing and methods of the change also may be important.

Neurogenesis or Neurodegeneration

Neurogenesis is the creation of neurons or nervous tissue. Neurodegeneration is the reverse. Again, the prevailing line of thought, as expressed by Sartorius et al. (2016), is this: Neurogenesis (B) occurs in ECT, therefore neurodegeneration (A) does not occur. (*Either only A is true or only B. B is true. Therefore, not A.*) The logical fallacy of affirming a disjunct. Again, it is possible that in ECT, A may be the precursor to B, or even that

A follows B. And, if so, it would still be uncertain if the advantages acquired by the neurogenesis outweigh the disadvantages given rise to by the neuronal necrosis, or how the relationship between them changes with time.

There are several studies on whether ECT causes neurogenesis or neurodegeneration.

Electroconvulsive shock (ECS) is an animal analog of ECT. The 2011 *FDA Executive Summary* on ECT notes that studies in murine rodents have had mixed results (p. 35). Of those cited, some have shown neurogenesis and/or synaptogenesis (Chen, Madsen, Wegener, & Nyengaard, 2009; Hellsten, Wennström, Bengzon, Mohapel, & Tingström, 2004; Madsen et al., 2000; Malberg, Eisch, Nestler, & Duman, 2000; Vaidya, Siuciak, Du, & Duman, 1999). Others have shown neurodegeneration (Cardoso et al., 2008; Lukoyanov, Sá, Madeira, & Paula-Barbosa, 2004; Zarubenko, Yakovlev, Stepanichev, & Gulyaeva, 2005). In their article, Zarubenko et al. (2005) state, "Cell death may be the stimulus for increased neurogenesis as a compensatory mechanism in a variety of lesions" (p. 718). Despite the scientific evidence on both sides, and having found only a small difference of only five studies showing growth to only three showing damage out of a small total of only eight cited, the 2011 *FDA Executive Summary* describes the studies showing neurogenesis or synaptogenesis as "many instances" (p. 35), while describing the studies showing synapse loss and/or neuronal death as "a handful of studies." This improper description does not even take into account the sample sizes or other methodological differences of the studies, a hint of confirmation bias.

Perera et al. (2007) examined the effects of ECS in a small sample of bonnet macaque monkeys (*Macaca radiata*) and found cellular proliferation in the dentate gyrus of the hippocampus of those treated with ECS. Bromodeoxyuridine (BrdU) labeling suggested that many of the cells proliferating after ECS survived for at least 1 month (p. 4896).

Many other animal studies are cited in a lengthy review by Reisner (2003), with mixed findings. This part of the review is under the section "Brain Damage and ECT," subsection "Animal Studies" (pp. 207–208). Because the FDA's cited studies have already been noted here and animal studies have limited relevance to ECT in humans, this author will leave the descriptions of those others to Reisner's article.

Let us reconsider the findings of the studies showing neurogenesis. It sounds like a good thing. But are any old neurons destroyed in the process of creating the new neurons? How long do the new neurons last? If and when the new neurons die, is the brain left in a better or worse state than when it had old neurons that were lost?

Some of the sources cited so far already provide evidence to help answer these questions.

Perera et al. (2007) used the Gallyas suppressed silver staining technique to check for hippocampal apoptosis (programmed cell death), necrosis (cell death by injury or disease), and other cell death. The stain found no evidence of cellular death (p. 4896). However, it is important to note the study's limitations. The sample consisted of six monkeys treated with ECS, six treated with anesthesia only (sham), and two untreated monkeys (p. 4895), that is, a small and unbalanced sample. It is stated later that the experiment was repeated with a doubled sample size showing "continued absence of pathology," but the source is an unpublished observation (p. 4898). Real and sham ECS were administered over a duration of only 4 weeks, that is, a short-term duration. The researchers used only one staining technique to check for cellular damage, that is, because it was only one technique, it doesn't rule out the possibility of other techniques finding damage, or the possibility that something went wrong with the Gallyas stain. Of note, this author did not find any description of a positive control being used for the Gallyas stain. Yet, the researchers jump to this premature conclusion: "The increased proliferation with ECS was not a response to

cell damage" (p. 4896). It was an animal study using ECS in a nonhuman primate, that is, it used a treatment that is not quite the same as ECT in humans. The article states, "There were no signs of acute or chronic distress or significant weight loss in any of the animals" (p. 4895). However, no objective, statistical measure is given to support this anecdotal claim of the monkeys' normative health. There was no long-term checkup to confirm that the proliferated cells continued to survive in live animals. There could not be because the monkeys were sacrificed 3 days to 4 weeks after the 4-week ECS regimen.

About 1 year after the study's publication, study coauthor Sarah Lisanby was sent a warning letter by Timothy A. Ulatowski, the director of the Office of Compliance in the Center for Devices and Radiological Health of the FDA (Ulatowski, 2008). The Office of Compliance "takes targeted, risk-based compliance actions that address significant violations of device-related laws" (FDA, 2015b). The letter to Lisanby begins:

> This Warning Letter is to inform you of objectionable conditions observed during the Food and Drug Administration (FDA) inspection conducted at your clinical site from May 19, 2008 to June 9, 2008, by an investigator from the FDA New York District Office and a Compliance Officer from the FDA Center for Devices and Radiological Health, Rockville, Maryland.

Ulatowski's letter notes, "As a clinical investigator, you failed to conduct the investigation in accordance with the investigational plan," citing numerous violations of research protocols. Yet, by 2016, Lisanby had become director of the Division of Translational Research (DTR) at the NIMH (Cuthbert, 2016), hired by Thomas Insel, who was director of NIMH at the time (Hurley, 2015).

Reisner's (2003) review cites not only animal studies on potential brain damage but also many human studies (pp. 208–214). Reisner describes several other studies and reviews with apparent examples of brain damage in some patients who had older forms of ECT (Friedberg, 1977; Madow, 1956; Riese, 1948; Templer & Veleber, 1982). Reisner writes, "Poor physical health prior to ECT appears to be a common factor among patients who displayed brain pathology postmortem" (p. 209). He also writes, "Most of the post-mortem brain damage cases occurred before the use of anesthesia and hyperoxygenation, which have a protective effect" (p. 208). In the review's Conclusions Regarding Brain Damage in Humans, it is written, "Advocates of ECT suggest that there is no conclusive evidence that ECT causes permanent brain damage in humans. To a large extent, this is true" (p. 214). But Reisner also states, "It may be premature to rule out the possibility of ECT causing permanent brain damage or dysfunction in a minority of patients" (p. 215).

Let us return to more recent research. The 2014 observational study by Nordanskog et al. (2014) had a sample size of only 12 patients, and no negative controls, but it found that the patients' total mean hippocampal volume had increased from 6,014 mm^3 1 week before ECT (Assessment Point 1, A1) to 6,296 mm^3 1 week after ECT (A2). Yet, the study also found that after 6 months (A3) the total mean volume had decreased to 6,010, and after 12 months (A4), the mean volume was only 5,841 mm^3 (p. 305, Table 1). The authors describe the average hippocampal volume as having "returned to baseline" by A3 and they go on to state, "There were no further significant changes after 1 year (A3 vs. A4), and no significant differences between A1 and A3 or between A1 and A4" (p. 307). This analysis contradicts their numeric data, which show that the average total hippocampal volume initially increased by approximately 5% (rounded to the integer), but by the 12-month follow-up point it had decreased by approximately 3% (rounded to the integer) from its

original measurement. (Regardless of whether this decrease is statistically significant, it would seem to bear general significance to many people.) These calculations of the data are by this author and are not written in Nordanskog and colleagues' article, which, aside from displaying the numeric data, makes no worded recognition of the patients' net loss of hippocampal volume at the final follow-up point (another possible example of ECT researchers' confirmation bias). Although reduction in volume is not proof of loss of neurons alone, the long-term decrease in hippocampal volume is imperfect evidence of a long-term decrease in hippocampal neurons, and imperfect evidence of long-term brain atrophy, which could also explain the 2002 finding by P. J. Shah et al. of a correlation between ECT and reduced gray matter, despite many research findings of an initial short-term increase. And although Kanai et al. (2011) found that smaller volume in one part of the brain is associated with better performance on some tasks because of natural pruning of neurons through age, it is highly improbable that any beneficial long-term reduction in brain volume could be caused by ECT (a crude and artificial process) without also causing some form of brain damage.

Furthermore, ECT researchers appear to have not adequately and thoroughly addressed the possibility that the short-term increases in brain volume found in studies like this may not mostly be caused by regeneration or beneficial growth, but rather may mostly be caused by swelling because of electrical brain injury. In neurology, swelling is known to occur in at least some traumatic brain injuries (Galgano et al., 2016, p. 2; S. Shah & Kimberly, 2016; Werner & Engelhard, 2007). Nordanskog et al. (2014) do address the possibility of the short-term increase being a "transient oedema," noting that they found no evidence of this in T2-weighted fluid attenuation inversion recovery (FLAIR) images and citing one reference (p. 309). But Reisner's review, for example, identifies many cases of pathology in animals in the historical scientific literature and some in humans, even if it is a minority of human cases. And then there's the simple fact that Nordanskog et al. overlooked the long-term decrease in hippocampal volume in their own data.

In addition, the study by Nordanskog et al. (2014) measured the severity of the patients' depression using the Montgomery–Åsberg Depression Rating Scale (MADRS), and the data showed no correlation between hippocampal volume and an antidepressant effect. This contradicts other studies' indications that ECT's short-term increases in hippocampal volume are a cause of any therapeutic effect. The study also measured the patient's cognitive functioning using a neuropsychological test battery and found no statistical relationship between hippocampal volume and cognitive performance.

There are some additional important considerations about what happened during this study. Although 20 patients had been recruited, only 12 completed the study to A2, 10 to A3, and 7 to A4 (p. 305). Some patients refused to participate fully to completion, one went into a manic state, and one died before A3 (p. 305).

CHANGES IN SUBCELLULAR BRAIN CHEMISTRY

Changes in brain chemistry below the level of the cell give additional evidence for the issue of ECT-induced brain damage.

Growth factors are defined as proteins necessary for the growth and normal development of certain cells and which stimulate proliferation and differentiation in nearby cells (Campbell & Reece, 2005, Glossary). Neurotrophic factors (NTFs) are defined as

molecules that enhance the growth and survival potential of neurons (Nature Publishing Group, 2016). Neurotrophins are conceived of as molecules which are both growth factors and a type of NTFs and which regulate neural development, survival, function, and plasticity (Chua, n.d.; E. J. Huang & Reichardt, 2001).

The neurotrophin or neurotrophic hypothesis proposes that repetitive neuronal activity enhances the expression, secretion, and actions of neurotrophins to modify synaptic transmission and connectivity, providing a connection between neuronal activity and synaptic plasticity, and implicating neurotrophins as having a key role in psychiatric disorders (Lang, Jockers-Scherübl, & Hellweg, 2004). Some studies and reviews show increases in neurotrophins or NTFs in the brains of animals after ECS and of patients after ECT, particularly brain-derived neurotrophic factor or BDNF (Sartorius et al., 2009; Taylor, 2008). This has further led researchers to the conclusion that ECT has beneficial physical effects on the brain.

But again, this change needs to be put into context, the proverbial forest through the trees. If the mechanism by which ECT works involves brain injury, then the enhancement in neurotrophins may not be enough to substantially restore the brain from the damage. Even if ECT does not cause brain damage, what can logically be concluded from the increases in neurotrophins?

Buttenschøn et al. (2015) looked at the roles of BDNF and vascular endothelial growth factor A (VEGF) in the pathophysiology of depression. The researchers investigated longitudinal associations between depression scores and serum levels of these NTFs during antidepressant treatment in 90 individuals with depression of at least moderate severity. They found no baseline or longitudinal correlations between depression scores and serum levels of BDNF or VEGF. They write, "Our results do not support the neurotrophic model of depression, since a significant decrease in serum BDNF and VEGF levels after 12 weeks of antidepressant treatment was observed." Buttenschøn et al. note that their study's finding is in agreement with three studies, but in disagreement with an equal number of others. Yet, there are differences in experimental design. To measure levels of the NTFs in blood serum, Buttenschøn et al. used the Quantikine Human VEGF or BDNF Immunoassay by R&D Systems, Inc., whereas they state that most of the other studies used the commercial ELISA kit from Promega. And they write that most of the other studies have small samples (between 10 and 56 depressed patients).

In neuroscience, the role of BDNF is being shown to be complex, with continuing debate about how this neurotrophin affects epilepsy and other health conditions. Is it a good thing? Is it a bad thing? Is it good sometimes and bad others? It appears that context matters.

In a 2013 commentary on the research of the role of BDNF in epilepsy, Scharfman writes,

> A central question remains: are the effects of BDNF causal, i.e. do they contribute to epileptogenesis, or are they compensatory, and protect the brain from epilepsy? Today there is still no clear answer, although Gill *et al.* (2013) provide an important piece of evidence that, at least in the hippocampus, BDNF contributes to epileptogenesis. Moreover, they show that the effects are due to a facilitation of the abnormal rewiring of brain circuitry after injury, and the promotion of hyperexcitability. . . . Indeed, it has been suggested that BDNF is a 'double-edged sword' (Binder *et al.*, 2001). (p. 3552)

There may be times when BDNF becomes "too much of a good thing," to use the very words of Binder, Croll, Gall, and Scharfman (2001). In fact, one study of transgenic mice

genetically engineered to overexpress BDNF indicated that "overexpression of brain-derived neurotrophic factor in the brain can interfere with normal brain function by causing learning impairments and increased excitability. The results also support the hypothesis that excess brain-derived neurotrophic factor could be pro-convulsant in the limbic system" (Croll et al., 1999, p. 1491). A second study of transgenic mice engineered for BDNF overexpression demonstrated that "chronic BDNF overexpression in the central nervous system (CNS) causes learning deficits and short-term memory impairments, both in spatial and instrumental learning tasks" (Cunha et al., 2009). Another study analyzed genetic variation and plasma levels of BDNF in rats and in U.S. Army Special Operations soldiers deployed during the Iraq and Afghanistan wars (Zhang et al., 2014). Stressed rats and soldiers who had probable posttraumatic stress disorder (PTSD) had allelic differences in the *BDNF* gene (Val66Met) associated with increased BDNF expression, indicating that "stress results in BDNF overexpression in the hippocampus" and suggesting "a BDNF-related compensatory mechanism" (p. 10).

Thus, increases in BDNF after ECT may actually be a medical sign of brain injury or impairment or trauma.

CHANGES IN ELECTRICAL FUNCTIONING

There is some evidence that ECT can disrupt the normal healthy electrical activity of the human brain. A recent study by a small group of neurology researchers identifies five patients who received maintenance ECT and developed "florid temporal epileptiform abnormalities on electroencephalography (EEG)" and other seizure activity, even though they had no history of epilepsy (Bryson, Gardner, Wilson, Rolfe, & Archer, 2016). The abnormalities ceased after cessation of ECT. The researchers hypothesize, "Maintenance ECT may predispose to epilepsy with a seizure focus in the temporal lobe."

Bryson and colleagues' (2016) article was met with skepticism (Asadi-Pooya, 2017). However, they logically defended their work, noting that prior research that did not find an association between ECT and epilepsy involved patients tending to have received a much smaller number of ECT treatments and/or no EEG was recorded (Archer, Bryson, Gardner, Wilson, & Rolfe, 2017). They also report that they "have observed temporal lobe epileptiform discharges in several other patients."

Reisner's (2003) review also identifies several older studies that found EEG abnormalities after ECT (pp. 210–211). This author was not able to find records of most of those. On a side note, although Reisner's review is extremely comprehensive in its list of studies summarized, he often gives uncritical acceptance of study conclusions, especially of more recent research, and he glosses over the significance of the many historical findings of pathology in his own conclusions.

BRAIN DAMAGE IN AUTOGENOUS SEIZURES

When ECT is performed as intended for inducing a therapeutic effect, a seizure is always involved. Therefore, a pertinent question is whether naturally occurring seizures cause brain damage. There is disagreement over this issue among neurology experts, although seizure duration and frequency are believed to affect whether brain damage occurs.

A 2015 Web page on the site of Epilepsy Action, which is described as "the UK's leading epilepsy organisation," states that seizures (either single or in clusters) that last longer than 30 minutes can cause damage to the brain. This is the duration the organization uses as a standard for *status epilepticus*, although other definitions have been noted (Lawson & Yeager, 2016). Several dictionary definitions do not apply any minimum time frame, instead defining the term similarly to that of Merriam-Webster, "A single prolonged seizure or a series of seizures without intervening full recovery of consciousness" (Status epilepticus, n.d.). The seizure duration in present-day forms of ECT in the United States is often less than 60 s (Mayo Clinic Staff, 2015), which implies that ECT seizures do not necessarily cause brain damage.

But some neurology experts have expressed beliefs that autogenous seizures are harmful to the human brain (Bronen, 2000; Carroll, 2003; Childs, 2008; Young & Jordan, 1998).

Carroll's (2003) news article for *The New York Times* states "mounting evidence now suggests that repeated seizures can indeed harm the brain—or, in rare cases, even lead to death." Among the neurology professors Carroll interviewed are Marc Dichter, Timothy Pedley, and Thomas P. Sutula, who all speak with great concern about the dangers of epilepsy being minimized. They speak of the syndrome Sudep (sudden unexpected death in epilepsy patients), damage to the hippocampus, swelling of the hippocampus, loss of neurons, and permanent changes that increase the probability of having a seizure again.

In the article by Childs (2008), Orrin Devinsky, director of the epilepsy program at New York University, is quoted as saying, "Recurrent seizures injure the brain, and the memory centers are particularly vulnerable."

Bronen, a doctor at Yale University School of Medicine, writes, "A number of experimental animal and clinical imaging studies support the idea that seizures by themselves cause brain damage," (p. 1782) specifying hippocampal damage, decreases in gray matter, metabolic dysfunction, swelling of neurons and glial cells, and more.

Doctors Young and Jordan write that nonconvulsive seizures damage the brain, citing sources on cognitive impairment, recurring seizures, and neuronal death. Generally, modern American ECT uses only nonconvulsive seizures because the treatment is modified to prevent full-body convulsions. "Unmodified" ECT is ECT without anesthesia or muscle relaxants and is still practiced in other parts of the world, where reports of abuse and public controversy have ensued even in recent years (Lewis, 2014; Shukla, 2000).

Referring back to the changes in subcellular brain chemistry following ECT, it should be repeated that increased expression of BDNF has been recorded after general seizure activity, meaning production of the NTF is increased (Binder et al., 2001). If one is to believe that an increase in NTFs after ECT is a sign that the treatment is beneficial to the brain, then it is logically consistent to also believe that an increase in NTFs after autogenous seizures is a sign that naturally occurring seizures are beneficial. This would contradict the opinions of Devinsky, Dichter, Pedley, Sutula, Young, and Jordan, and other experts who consider autogenous seizures to be harmful. If one does not accept that proposition as true, yet one still believes that the increase in NTFs after ECT is a sign of benefit to health, then one must be prepared to answer this question: How is the increase of NTFs after ECT any better than the increase of NTFs after autogenous seizures?

CLEAR CASES OF BRAIN DAMAGE

There are a few cases of patients with clear brain damage subsequent to ECT. Considering older cases, Reisner (2003) refers to a 1963 case of evident brain damage in a 23-year-old man (p. 209), cited originally by McKegney and Panzetta (1963). The patient was described as "in apparently good physical health" but with catatonic schizophrenic symptoms (McKegney & Panzetta, 1963). It is interesting to note the historically dated, questionable diagnoses given to this man, such as a "character disorder" of "passive dependent and homosexual type" (p. 398). (As noted earlier in this article, homosexuality is one example of medicine's blunders and reversals because the trait was for several years considered a mental disorder by the majority of psychiatry.) On the same page, the article states, "He first manifested evidence of brain damage immediately following his first ECT and died after a progressively deteriorating course of 2 months." This was despite the apparent use of anesthesia and oxygenation, alterations to modern ECT intended to prevent full-body convulsions and anoxia (inadequate oxygen in body tissue).

There is also a case of death and brain damage from 2015, which wasn't widely publicized until 2016 (Armstrong, 2016; Prince, 2016; Silfverskiold, 2016). Seventy-one-year-old Elsie Tindle, an involuntary patient with a learning disability who was suffering depression and downward physical health, was forced to have ECT at Sunderland Royal Hospital in Sunderland, England. After only three ECT treatments from February to March 2015, she had an unexpected spontaneous seizure, ending in her demise on April 4, 2015. The inquest was presided over by Sunderland Coroner's Court HM Senior Coroner Derek Winter, whereas the postmortem examination was done by Home Office pathologist Nigel Cooper. Cooper "concluded the formal cause of death as anoxic-ischemic brain damage, due to status epilepticus, due to electroconvulsive therapy" (Prince, 2016; Silfverskiold, 2016). This case especially raises questions of what other interventions could have been tried before the drastically invasive and often traumatizing procedure of involuntary ECT (Breeding, 2016; Burstow, 2006; Froede & Baldwin, 1999; Johnstone, 1999; Spikol, 2006), which this author has also personally experienced. Ironically and illogically, despite Tindle's death, the jury ruled that her involuntary ECT was "lawful and necessary" (Armstrong, 2016; Prince, 2016) as though the only options were ECT or no treatment. This is a logical fallacy concerning ethics similar to the fallacy of the alternative disjunct concerning physical truth. The fallacy has complex, somewhat varied forms (Tomić, 2013). But put simply, the false dilemma fallacy is belief in a false dichotomy or false set of alternatives (Boone, 1999, p. 6), the fallacy of thinking there are only two options, or not considering all of the choices available in an ethical dilemma (Texas State University, Department of Philosophy, n.d.). Treatments for depression include several classes of oral and injectable medication, some with long-term effectiveness, and some for short-term emergencies; other technologies such as transcranial magnetic stimulation (TMS), cranial electrotherapy stimulation (CES), transcranial direct current stimulation (tDCS), and low-field magnetic stimulation (LFMS); nutritional and micronutrient therapies; clinical social work, peer support, various talk therapies, emotional regulation techniques, and psychosocial interventions; temporary physical restraints; and intravenous medication or nourishment. There are also novel treatments under investigation, such as precision-medicine biologics meant to replace missing brain proteins (Hamill, 2016; Pan et al., 2017). In addition, sham ECT (anesthesia only) may be effectual for many patients because some research has shown (Ross, 2006). (See "Placebo and Nocebo Effects" and "Anesthesia,

Muscle Relaxants, and Postelectroconvulsive Therapy Medication" sections for hypotheses about potential confounding by placebo effects and the effects of anesthetics and other drugs used during or after ECT.)

CONFOUNDING VARIABLES

A confounding variable (confounder) is a variable in research that can interfere with truthful assessment of the relationship between an independent variable and a dependent variable, such as ECT and any measure of brain health or dysfunction. There are various confounders that could interfere with research on whether ECT causes brain damage.

Brain Scan Inaccuracy

The accuracy of tools and methods used to perform brain scans has come into question in recent years. For instance, one study showed that the technique of spatial smoothing could corrupt analysis of functional magnetic resonance imaging (FMRI) data (McClure, 2013; Sacchet & Knutson, 2013). Stanford News reporter Max McClure writes, "It's easy to forget that these brain images aren't real snapshots of brain activity. Instead, each picture is the result of many layers of analysis and interpretation, far removed from raw data."

Placebo and Nocebo Effects

A placebo is a substance that would normally have no pharmacological effect, which in medical research is used as a negative control to contrast with the effects of a biologically active treatment. The placebo effect occurs when a patient has a beneficial reaction to a placebo, lessening symptoms of illness or reducing side effects. The effect is directly related to the patient's positive expectations. The nocebo effect is the opposite of the placebo effect, in which the control treatment causes a detrimental reaction because of the patient's negative expectations.

It may be difficult to believe that brain changes after ECT could be significantly influenced by placebo and nocebo effects. However, placebo and nocebo effects, both conscious and unconscious, have been implicated in a wide variety of neurological changes (Beauregard, 2009; Colloca & Benedetti, 2016; Wager & Atlas, 2015). Thus, ECT patients with positive expectations may be more likely to experience a placebo effect, whereas patients with negative expectations may be more likely to experience a nocebo effect. Whether ECT is performed voluntarily or against the will of the patient (involuntary) could directly and greatly affect the chances of either effect. A previous theoretical article lays out the hypothesis that involuntary psychiatric treatment is more likely to cause nocebo effects, while diminishing the chance of placebo effects (Meynen & Swaab, 2011).

Cognitive Reserve

Cognitive reserve is a hypothetical phenomenon by which differences in cognitive processes or neural networks allow some people to cope better than others with brain damage (Stern, 2009). Cognitive reserve may have acted as a confounder in ECT research such as the 2014 study by Nordanskog et al.

Anesthesia, Muscle Relaxants, and Postelectroconvulsive Therapy Medication

Modified ECT uses anesthetics and muscle relaxants to put the patient to sleep and to prevent full-body convulsions. The anesthetics for ECT include etomidate, ketamine, propofol, and thiopental (Hoyer, Kranaster, Janke, & Sartorius, 2014). It is probable that at least some patients acquire a therapeutic effect from one or more of the drugs alone, and they may feel relaxed from being put to sleep and the subsequent sedation even after they awaken. The medications might even contribute to changes in brain size or number of neurons. This hypothesis is supported by recent research on ketamine as a medication for depression (Coyle & Laws, 2015; Fond et al., 2014) and by animal studies showing that ketamine can alter neurogenesis in the brain (Clarke et al., 2017; Dong, Rovnaghi, & Anand, 2012; H. Huang et al., 2015; Keilhoff, Bernstein, Becker, Grecksch, & Wolf, 2004; Winkelheide et al., 2009). Furthermore, it is probable that psychiatric and other medications taken after ECT cause brain changes that temporarily counteract or superficially mask the changes caused by ECT, causing another form of confounding.

Cognitive Biases and Statistical Biases

A cognitive bias is a systematic error in judgment and decision-making, which can be caused by cognitive limitations, motivation, and/or environmental adaptations (Wilke & Mata, 2012). The cognitive biases of researchers and medical professionals almost certainly play roles in their interpretations of scientific evidence on whether ECT causes brain damage, as well as in how they formulate studies on this question.

Confirmation bias, mentioned previously in this article, is the cognitive bias of tending to acquire or interpret new information in a way that confirms one's preconceptions or hypotheses, instead of disconfirming them (Allahverdyan & Galstyan, 2014; Hallihan & Shu, 2013; Wilke & Mata, 2012).

Statistics are sometimes marred by biased sampling methods that produce results that systematically differ from the truth about the population.

One major example is selection bias, a tendency on the part of the sampling procedure to exclude or include a certain type of unit (Aliaga & Gunderson, 2006, p. 90). In research on ECT, there is always a possibility that researchers select test subjects in a way that leads to biased statistics, either making the treatment seem safer and more effective than reality or vice versa.

Another statistical bias is nonresponse bias, distortion because a large number of units (in this case, ECT patients) do not respond or refuse to respond, and the nonresponders tend to be different from the responders (Aliaga & Gunderson, 2006, p. 90). A related statistical bias is response bias, distortion that can arise because the wording of a question or the behavior of the interviewer affects the truthfulness of the responses received (Aliaga & Gunderson, 2006, p. 90). The ways that ECT patients are asked about possible cognitive impairment caused by the treatment, as well as the level of trust in the patient–professional relationship, could result in response bias and/or nonresponse bias that distorts research on whether ECT causes brain injury, when cognitive impairment is used as a measure of it. In particular, patients lacking trust may be less likely to be forthcoming and truthful in their responses, even when given the opportunity to share their subjective experiences of the treatment.

Bias blind spot is the cognitive bias of lacking awareness of one's biases (Shaw et al., 2016, pp. 1–2). When this blind spot occurs in ECT researchers, it may solidify and reinforce confounding in their research.

THE 2011 U.S. FOOD AND DRUG ADMINISTRATION *EXECUTIVE SUMMARY* ON ELECTROCONVULSIVE THERAPY HAS SIGNS OF BIAS

The aforementioned includes scientific, real-world, and logical evidence that ECT causes brain damage. The 2011 FDA review of ECT is in some ways superficial and even contains indications of confirmation bias and selection bias. The report's statement that "there is no evidence to suggest that ECT causes brain damage" (p. 37) is clearly false because it dismisses or disregards so much. Although this author has opined that all human thought is biased, the report's deviations from objective evaluation are unacceptable coming from a governmental agency with oversight on medical laws and regulations. Instead of merely excluding the unmentioned studies with important findings from its report, the FDA should have explained why those studies were deemed lacking in rigor or reliability. What standards were used to define scientific rigor and to exclude certain research as lacking in rigor or completely lacking in value? But perhaps the FDA failed to mention those studies not just because of bias, but because of simple ignorance. Moreover, although the FDA report has detailed sections on memory and cognitive adverse events, neuropathological findings, and death, the report almost completely ignores the long history of negative patient accounts showing that ECT, especially involuntary ECT, is often dehumanizing and psychologically traumatic (Breeding, 2016; Burstow, 2006; Froede & Baldwin, 1999; Johnstone, 1999; Spikol, 2006). Such accounts also indicate that patients with negative experiences may be less likely to report those experiences and more likely to be invalidated, ignored, and dismissed by psychiatric professionals.

"DISSENTING" EXPERTS

There have been neurologists, psychiatrists, psychologists, and related health professionals who have opined that ECT causes brain damage. They stand in contrast to the apparent predominant opinion of the present day, but their views are not based on flights of fancy and they should not be dismissed without serious consideration.

A 1977 article by neurologist John Friedberg, cited by Reisner and earlier here, asserts that ECT results in neuropathology and "brain disease." Friedberg also had a book published in 1976 that attacked electroshock as dangerous, cruel, and ineffectual, and presented interviews with patients expressing serious complaints. Friedberg passed away in 2012. An obituary for Friedberg notes he had trained at Yale University and University of Rochester School of Medicine. He was "well known for his courageous opposition to abuse by the psychiatric establishment" (San Francisco Chronicle, 2012).

In a 2000 letter in *Nature*, neuroscience professor Peter Sterling disputes the insistence of financially conflicted psychiatrist Max Fink that brain damage from ECT is a merely a myth promoted by antipsychiatrists. Sterling states,

> ECT is used as an experimental tool by neuroscientists, as it releases massive quantities of glutamate, whose release following stroke causes significant neuronal death. Indeed, observers describe people who have had many ECT treatments as "punch drunk"—resembling boxers who have sustained chronic brain damage. . . . It is a good bet that history will view ECT as one of what neuroscientist and author Elliot S. Valenstein calls the "great and desperate cures"—and its promoters as kin to the promotors of lobotomy. (p. 242)

In a 2002 article, philosopher and psychology professor James Giles (PhD in Philosophical Psychology) lays out the history and philosophy of ECT, ideas about it from proponents and critics, and accounts for the long list of known side effects, including long-term cognitive damage, summarizes evidence of brain damage, and describes the severe problems with research on ECT. The result is an unsettling sense of inhumane treatment that enables psychiatrists to exert power and control, with Giles concluding that ECT is qualified for the "title of the blood-letting of our time" (p. 82).

In a 2003 op-ed, psychiatrist Loren Mosher and professor of social work David Cohen write that ECT is not safe and not necessary. They cite the words of electroshock survivors Leonard Roy Frank, Thomas Hsu, and Jackie Mishra, who in turn call ECT brain-damaging, violating, and impairing. The article notes that Mosher was formerly chief of the Center for Studies of Schizophrenia at the NIMH and served as first editor-in-chief of *Schizophrenia Bulletin*, whereas Cohen received the 2003 Eliot Freidson Award from the American Sociological Association for outstanding publication in medical sociology.

In a lengthy 2010 review of the research on ECT, psychology professors John Read and Richard Bentall found "strong evidence (summarized here) of persistent and, for some, permanent brain dysfunction" (p. 333). They also write that in the 1940s it was commonly accepted that ECT "works" by causing brain damage and that in the 1940s and 1950s many autopsies showed evidence of brain damage (p. 344). Cited studies and reviews include that of Alpers (1946). This author could not find database records of the others, although a Web search indicated Read and Bentall are not the only ones to cite them. Read and Bentall refer to more recent studies they believe indicate brain damage, including the study by Calloway et al. (1981) and a 2001 study by Rami-Gonzalez et al. whose aim was "to review different subtypes of memory dysfunction associated with ECT from a neuropsychological perspective."

Psychiatrist Peter Breggin has multitudinous times written that ECT is brain-damaging (Breggin, 1998, 2007, 2010, 2011; van Daalen-Smith, Adam, Breggin, & LeFrançois, 2014). Breggin has received research grants from Harvard Medical School and NIMH and has testified as a medical expert in more than 90 cases since 1987 (Breggin, n.d.). One was the 2007 case *Salters v. Palmetto Health Alliance*, in which former nurse practitioner Peggy Salters won $625,177 in damages for cognitive injuries sustained from ECT. This was found to be caused by the medical negligence of Dr. Eric Lewkowiez, who referred her for ECT but failed to report her increasing memory difficulties to the ECT specialist, resulting in Salters becoming "completely unable to function" at work or home (Anderson, Kittredge, & Short, 2007).

RELEVANCE TO LAW

In United States law, burden of proof is the obligation of a party in a legal case to provide evidence that will convince the court or jury in support of a contention. It dictates the degree of certainty that judges or juries must have to decide in favor of a party (Bergman, 2015). Ascending standards of burden of proof include "preponderance of the evidence," "clear and convincing evidence," and "beyond a reasonable doubt."

In judicial cases, a burden of proof must be satisfied to answer legal questions of fact, which are distinct from questions of law in that they may be determined without reference to any legal rule or standard (Brown, 1943).

Prima facie is an adjective and legal term meaning plain, clear, or self-evident; strong enough to establish as fact unless disproven or rebutted (Legal Information Institute, n.d.); or presenting sufficient evidence at first glance for a plaintiff to win a case (NOLO, 2016).

The evidence reviewed here shows a near certainty of brain damage in some individual cases and indicates a rational likelihood that all ECT causes some degree of brain damage. Therefore, it can be reasonably concluded that the claim that ECT never causes brain damage does not meet the standard of proof as defined by clear and convincing evidence. There is not even a preponderance of evidence to prove the claim. The contention cannot legally be established as a fact by *prima facie* evidence because that evidence would be overturned by the evidence against it. Rather, there is clear and convincing evidence that ECT has caused brain damage in some individuals.

Furthermore, placebo and nocebo effects in ECT may affect legal liability if a patient were to sue their treatment providers for medical malpractice over an ECT-induced brain injury. An article in *Psychological Injury and Law* discusses how contextual factors can influence a patient's expectations about his or her recovery from a brain injury and how both the accident and negligence-related nocebo effects can create legal liability (Vanderploeg, Belanger, & Kaufmann, 2014). Thus, poor care of a patient before, during, and/or after ECT may add to legal liability and increase the chances of winning a case on the partial basis of establishing brain damage. Involuntary ECT could add to this liability.

SUMMARY AND CONCLUSION

Psychiatric authorities and governmental agencies, including the FDA, have concluded ECT does not cause brain damage. However, the research on this issue has been deeply flawed, with researchers sometimes analyzing data using erroneous reasoning and drawing hasty conclusions, often reflecting researchers' confirmation bias and at times even logical fallacies.

When examining changes in brain structure after ECT, researchers have been inclined to attribute brain atrophy to depression rather than ECT. Several brain imaging studies have found increases in brain volume after ECT, but they have not adequately considered and answered the possibility that such volume increases may be caused by swelling from electrical brain injury, rather than beneficial brain growth or regeneration, with at least one study's analysis engaging in the logical fallacy of affirming a disjunct.

Nonpsychiatric neuroscience research indicates that neurons in the human brain undergo pruning with age, such that less gray matter in the cortex is actually associated with better cognitive performance. This has implications for ECT research because researchers have often concluded that the increase in brain volume found after ECT must be beneficial, rather than detrimental. Quantity of brain tissue may not be a good indicator of quality of neurocognitive functioning.

Several studies have found some type of neurogenesis occurs after ECT, but at least a few have found neurodegeneration, and it is unclear if the neurogenesis found is actually beneficial to health or a sign of injury. Many historical animal studies, and a few human studies, found signs of pathology, although ECT was often performed differently or unmodified at the times of those studies. A modern interventional nonhuman primate study that found neurogenesis and no sign of cell death after ECT used a very small sample, a short

duration of treatment, and its analysis shows some confirmation bias. One of the study authors, who now works at NIMH, was later sent a warning letter by a department of the FDA for violations of research protocols.

A poorly designed recent observational study with a small sample of patients found a short-term increase in hippocampal volume after ECT, like several other studies, but the authors overlooked how their data showed that after 12 months mean hippocampal volume had decreased slightly from its original measurement. This provides evidence of long-term brain damage.

Some research has found an increase in neurotrophins or NTF after ECT and after ECS, an animal analog of ECT, especially BDNF. Scientists have hastily concluded this is a sign of therapeutic benefit. However, several neuroscience studies show that the role of BDNF is complex. Research on the neurotrophic hypothesis that depression and other psychiatric disorders are caused by low quantities of neurotrophins has had mixed findings. Research with rodents suggests that overexpression of BDNF, rather than being beneficial to health, causes cognitive impairment, may be epileptogenic, may be a response to seizures in general, and at least one human study indicates that increased expression of BDNF is a response to trauma. This suggests that increases of this NTF after ECT are actually a sign of harm.

A recent study by a small group of neurology researchers recorded epileptiform abnormalities on EEG in five patients who received maintenance ECT, and there are older studies with similar findings.

Although neurology experts have expressed disagreement over whether autogenous seizures alone cause brain damage, many have indeed stated that seizures damage or injure the brain.

There is strong evidence bordering on proof that ECT has caused brain damage in some individuals, including a recent case in which an involuntary patient died from a spontaneous seizure, and the pathologist concluded the cause of death as brain damage because of status epilepticus, caused by ECT. This case also raises ethical questions because it shows how psychiatric professionals often do not consider all available treatment options but rush to ECT, violating the dignity of the patient and endangering patient safety.

There are many variables that likely confound research on whether ECT causes brain damage, including brain scan inaccuracy; placebo and nocebo effects; cognitive reserve; anesthesia, muscle relaxants, and post-ECT medication; and cognitive biases and statistical biases.

A lengthy 2011 FDA review of ECT has signs of selection bias because multiple studies that could indicate brain damage were omitted. The FDA report also contains some language indicating confirmation bias in its evaluation of the studies cited. The report's statement that "there is no evidence to suggest that ECT causes brain damage" is false.

Although the present consensus among psychiatrists seems to be that ECT does not cause brain damage, there have been several dissenting professionals from neurology, psychiatry, psychology, and related health fields. Their observations and arguments have been at times compelling.

From a standpoint of legal burden of proof, there is clear and convincing evidence that ECT has caused brain damage in some individuals. Legal liability may be increased when poor care of an ECT patient leads to nocebo effects.

This author concludes that ECT likely always causes some form or degree of brain damage. The primary mechanism by which it works probably involves brain damage,

with resulting memory loss and cognitive impairment the means by which it relieves depression, disabling the overall functioning of the patient in ways that may outweigh whatever psychiatric disability the patient had, or continues to have if the treatment is ineffectual. Beneficial physical effects are more likely because of placebo effect and anesthetics than because of electricity or seizure. Involuntary ECT likely often works as torture and social control, which this author has experienced. Even voluntary ECT with perceived informed consent can be traumatizing. Patients who have positive subjective experiences of the treatment are probably unaware of much of the damage and disability inflicted in them and/or accept it because of social conditioning and iatrogenic anosognosia.

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Christopher James Dubey had just graduated with a bachelor degree in 2005 when unrecognized SSRI withdrawal and difficult circumstances pushed him to attempt suicide multiple times. He was shortly forced by probate court order to have electroshock against his will at the Institute of Living in Hartford, Connecticut, from 2005–2006. Traumatized by this, he became involved in psychiatric survivor issues and wrote a novel entitled *Assignment Yggdrasil*, which features multiple characters forced to have electroshock. While studying for an associate degree in biotechnology, he was a volunteer research assistant in the Department of Biology at Wesleyan University, working in an entomology laboratory. Before graduation, he won an academic award for biotechnology. He currently is a book reviewer for the website IndieReader.

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