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# What Is Brain Damage and Does Electroconvulsive Therapy Cause It?

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**Abstract:** Surveys show public misperceptions and confusion about brain damage and electroconvulsive therapy (ECT). Fictional movies have misrepresented ECT to suggest brain damage and to ridicule mental illness and psychiatric patients. “Brain damage” has become a colloquial expression without consistent meaning. In contrast, brain injury is the medical term for destruction of brain cells, such as from kinetic impact (concussion), hypoxia, or infection. Studies of both high-resolution magnetic resonance imaging (MRI) and enzyme assays find that causes of brain injury are accompanied by observable structural changes on MRI and elevated blood and cerebrospinal fluid levels of brain enzymes that leak from injured brain cells. Concussion is also followed by intracerebral bleeding, progressive brain atrophy, diffuse axonal injury, cranial nerve injury, and 2–4 fold increased risk for dementia. In contrast, there is no evidence that ECT produces any of these. Studies of ECT patients find no brain edema, structural change persisting 6 months, or elevated levels of leaked brain enzymes. Statistical comparisons between brain injury and ECT effects indicate no similarity ( $P < 0.00000001$ ). Moreover, the kinetic, thermal, and electrical effects of ECT are far below levels that could possibly cause harm. This robust evidence shows that there is no basis to claim that ECT causes brain injury.

**Key Words:** ECT, electroconvulsive, therapy, brain damage, brain injury, MRI, brain enzymes, concussion, traumatic brain injury, chronic traumatic encephalopathy

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## THE BASIC QUESTION AND METHODS OF INVESTIGATION

“Brain damage,” pain, cognitive difficulties, and simple fear are common negative public perceptions about ECT.<sup>1</sup> Large rapid clinical improvement during a course of ECT in many patients suggests that ECT produces changes in the brain; is damage part of this? What specifically is brain damage and does the ECT procedure cause it, separate from anesthesia concerns (e.g., hypoxia) and co-occurring medical conditions (e.g., coronary vascular illness)?

Google and PubMed searches on the terms “brain damage,” “brain injury,” “traumatic brain injury,” “MRI ECT,” “ECT brain injury,” and “ECT dementia” and then PubMed searches on “ECT” with each specific brain cell enzyme produced the information used. All studies found reporting verifiable observable brain structure or body fluid level data on these topics were reviewed.

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## WHAT DO PHYSICIANS REFER TO BY “BRAIN DAMAGE”

The expression “brain damage” is not used in publications by neurologists or neurosurgeons in countries where English is the official language; rather, the term brain injury is used. “Brain damage” is occasionally used by other physicians indistinguishably from brain injury. As used in the neurological literature, brain injury is a detrimental change in brain structure beyond behavioral and psychological changes and complaints. Because behavioral and psychological effects can occur without brain injury, they are not evidence of it, and the focus here is on brain measurements.

Brain injuries are commonly grouped into traumatic brain injuries (TBIs) and nontraumatic injuries, such as from hypoxia, blood flow interruption, infection, tumor, or aneurysm. Causes of TBI noted include falls, collisions, assaults, and sports injuries, producing an external force with kinetic energy that causes skull damage (e.g., from a weapon) or brain movement inside the skull.<sup>2</sup> Every cause and type of brain injury mentioned produces observable structural brain changes at least sometimes.

## WHAT DO THE PUBLIC AND NONPHYSICIANS REFER TO BY “BRAIN DAMAGE?”

Brain damage is a colloquial expression without consistent or specific meaning. In publications addressed to the public, its usage sometimes includes unverified—even unverifiable—beliefs. The idea of “brain damage” has been used to provoke fear about ECT, combining absence of knowledge about brain physiology and structure, how the nervous system works, serious brain illness, and the nature of electricity. For example, few people can identify what voltage is, beyond something about electrons and wall sockets.

As a brain-related illustration, a survey found that a high proportion of the population has flatly incorrect ideas about the sequelae of TBI, and even health professionals who do not specialize in brain injury hold similar misconceptions.<sup>3</sup> In this survey, 56% of respondents had at least some college education, 17% had experienced brain injury, and nearly half knew people with brain injury. However, 40% of respondents endorsed that sometimes a second head impact can help remember forgotten items, a misconception presumably provoked by fictional television and movies.

Analogously, fictional movies and television mislead audiences by adversely depicting ECT and its consequences, as in “One Flew Over the Cuckoo’s Nest (1975)” and “Death Wish Two (1982),” leaving the impression ECT causes permanent harm to the brain.<sup>4</sup> “Cuckoo’s” impact is so strong that recent movie portrayals, as in “Don’t Worry Darling (2022),” provoke conditioned fear reactions with minimal detail. Fictional comedy movies and TV programs have misrepresented ECT to suggest brain damage, and to comically ridicule mental illness and psychiatric patients, as in “The Beverly Hillbillies (1993),” “Strange Brew (1983)” and “Mad TV (May 30, 2009),” and similarly in Pink Floyd’s famous song “Brain Damage (Dark Side of the Moon, 1973):”



**FIGURE 1.** CCHR Placard Outside 2019 Annual APA Meeting.

Besides movie makers, antipsychiatry organizations such as CCHR (“Citizens Commission” a Scientology organization affiliate) have long directed threatening claims about ECT to the public (Fig. 1). As an example, outside the main meeting hall of the 2019 annual meeting of the American Psychiatric Association, demonstrators carried color printed placards displaying in large bold font “ELECTROSHOCK TREATMENT. A FANCY WORD FOR ‘KILL BRAIN CELLS’” with the organization name CCHR Citizens Commission on Human Rights. (See photo). Referring to its downloadable video “Therapy or Torture: The Truth about Electroshock,” the CCHR website<sup>5</sup> states, “It hits the head with the force of a 40-pound cinder block dropped seven and a half feet. It’s been described by patients as a grenade going off in your body. It’s called electroconvulsive therapy.” This video presents a sequence of similarly threatening and distorted claims.

On its website, Scientology published a report of a CCHR exhibit, “Psychiatry: An Industry of Death” stating “ECT...victims are subjected to as much as 460 volts of electricity to the brain.” (<https://www.scientologynews.org/press-releases/citizens-group->

exposes-psychiatric-crimes.html, caution: this site attempts to install viruses). Likewise, British psychologist nonphysician John Read<sup>6</sup> wrote “brain cells receive electrical signals of a fraction of a volt. Subjecting them to 150 V inevitably causes damage, similar to traumatic brain injury.” Please consider what 150 volts and 460 volts mean, then reconsider that a point of voltage represents no power or energy without the presence of a second location of differing voltage for electrons to flow between. In comparison, the voltage difference across the cell body of a human brain neuron during the ECT stimulus is calculated as 0.006 volts<sup>7</sup> and (by a different method) as less than 0.070 volts.<sup>8</sup>

In a litigation against psychiatrists, Dr. Peter Breggin mentioned working as a hired consultant (“expert witness”) for plaintiffs in about 80 lawsuits, and he claimed ECT causes brain damage.<sup>9</sup> In that litigation, the plaintiff’s brain images were interpreted as normal before and after ECT. The jury rejected claims against the defendant psychiatrist, noting plaintiff attorneys’ inability to identify that ECT caused harm. Breggin has authored numerous books attacking psychiatry, e.g., “Toxic Psychiatry,” “Medication Madness,” “Brain-Disabling Treatments in Psychiatry.” Previously, in a 1993 CBS network television program “Eye on America,” Breggin said to the camera “We have a long history in Psychiatry of damaging peoples’ brains and claiming it helps.” This same program shows a large banner displaying “SHOCK TREATMENT CAUSES BRAIN DAMAGE CITIZENS COMMISSION ON HUMAN RIGHTS (800)869-CCHR.”

Because the expression “brain damage” has no specific meaning, claims about it also have no specific meaning or implications. In contrast, reports about brain injury can be understood because it has specific meaning.

In a public internet survey of 1091 individuals who screened positive for depression, many respondents stated they are frightened about ECT and concerned that it could cause brain damage or pain. A large proportion were not aware that scientific evidence for ECT is robust and it is a standard therapy for severe and treatment-resistant depression.<sup>1</sup>

## BRAIN INJURY FROM TBI

Internet search for “brain damage” produces results mostly concerning TBI related to common injuries from sports, vehicle collisions, or falls. It may be surprising that TBI is far more likely following rotational motion, i.e., shear, than from straight line impact.<sup>10</sup> Moderate to severe concussion can immediately cause bleeding inside the brain and then produce brain tissue loss of about 5% per year as atrophy, sometimes generalized and sometimes focal. This generalized atrophy is typically seen as widening of cortical sulci, ventricular enlargement, and cortical thinning.<sup>11</sup>

Other examples of structural brain changes after TBI include onset of epileptic (seizure) disorder, contusion (bruising, swelling), hematoma (bleed inside the skull), cranial nerve injury that occurred together with TBI,<sup>12,13</sup> and visible neurological signs of impaired motoric actions (e.g., paralysis) or reflexes (e.g., spasticity) associated with specific brain regions or illness.

Of concussion patients studied, 181 of 251 (72.1%) showed magnetic resonance imaging (MRI) signs of diffuse axonal injury within the first month of experiencing concussion with loss of consciousness. These signs were visible on direct inspection of the MRI. Patients with diffuse axonal injury showed significantly worse functioning and neurocognitive outcomes compared to other patients, with personality changes, aggression, and major depressive disorder.<sup>14</sup> Chronic traumatic encephalopathy produced visible atrophy of several frontal and temporal regions of brain cortex, including orbital-frontal, dorsolateral frontal, superior frontal, anterior

temporal, and medial temporal regions, as well as larger brain lateral and third ventricles.<sup>15</sup>

Dementia is increased 2- to 4-fold following moderate or worse TBI, according to a survey of publications.<sup>16</sup> In a prospective longitudinal study of 208 patients with TBI, intracranial hemorrhage and hospitalization for at least 3 days was followed by increased incidence of dementia.<sup>17</sup>

### BRAIN INJURY FROM TEMPERATURE ELEVATION

In its “Final Order on ECT” of December 2018,<sup>18</sup> the US FDA noted that direct brain injury from the ECT electrical stimulus would require temperature elevation in the brain to injurious levels. This does not occur because most of the electrical current is dispersed through the scalp and shielded by the skull, and is thereby short-circuited between the electrodes. Worst case calculation shows deep tissue temperature elevation is less than 0.092°C,<sup>19</sup> recently confirmed by measurements of scalp temperatures corresponding to strong current shunting.<sup>7</sup>

Moreover, the hippocampus—the brain center for learning and memory—is distant from the location of ECT electrodes, about as far as a brain region can be. Temperature injury in the hippocampus cannot occur in the absence of temperature injury closer to the electrodes, and this does not happen.

### MRI STUDIES OF BRAIN STRUCTURE BEFORE AND AFTER ECT

Early prospective brain imaging studies of ECT, using conventional MRI, showed no evidence of change in size of brain cortex, hippocampus, or ventricles between before and after a course of ECT.<sup>20–22</sup> One of the earliest nonconventional prospective MRI studies used diffusion-weighted imaging, chosen for sensitivity to localized tissue changes from impaired energy metabolism, as from ischemia or prolonged seizure.<sup>23</sup> Diffusion-weighted imaging showed no abnormalities after ECT.

More recent studies used high-resolution MRI scans to examine the hippocampus, a deep brain area involved with memory function, and other limbic areas. These limbic regions are about as distant from the scalp and ECT electrodes as a brain region can be, so direct effects of the ECT electrical current are less likely than elsewhere in the brain. As direct effects on brain structure do not occur closer to the electrodes, there should be no direct effects of ECT current on the hippocampus and other deep brain areas.

In one survey, 31 of 32 MRI brain-imaging studies of a total of 467 patients and 285 controls found no evidence of brain injury after ECT.<sup>24</sup> More recent studies by this survey's authors found that all structural brain changes fade by 6 months after ECT; therefore, no detrimental changes persist. Specifically, after MRI brain imaging in 18 severely depressed patients showed increased thickness in 26 cortical regions 6 days after ECT, imaging repeated 6 months later found all brain regions had returned to baseline thickness.<sup>25</sup> In a later MRI study of 22 severely depressed patients, the same investigators<sup>26</sup> similarly observed initial increase in volumes but no change between baseline and 6 months after ECT in both right and left hippocampal dentate gyri, and in the volumes of 20 other major hippocampal regions.

Nordanskog et al<sup>27</sup> observed that the volume of the hippocampus is the same 6 months after ECT as it was before ECT. Measuring hippocampal volumes in 23 elderly patients, Bouckaert et al likewise found no difference between 6 months after ECT and before ECT.<sup>28</sup>

Jehna et al<sup>29</sup> examined 12 depressed patients before, shortly after, and 10 to 36 months after ECT. Before ECT patients had less global brain volume, white matter, and peripheral gray matter than healthy controls. After ECT, patients showed increased brain

volumes but at 10- to 36-month follow-up, these increases had faded to nonsignificant.

Studying MRI data in nine patients, Gyger et al<sup>30</sup> observed that water content does not increase with hippocampal volume after ECT, so there is no evidence that brain volume increase after ECT is edema (swelling injury). Likewise, using data from ultra-high field MRI measurements on 21 patients before and after 10 ECT sessions, Nuninga et al<sup>31</sup> observed that edema is not part of hippocampal volume increases that accompany ECT.

### LEAKAGE FROM BRAIN CELLS AFTER ECT

Besides MRI changes, brain injury can be detected by changes in blood substances such as enzymes that leak from inside brain cells through damaged cell walls. Several study reports describe that blood and cerebrospinal fluid (CSF) concentrations of substances that show elevations after brain injury are not increased by ECT.

The brain isozyme (BB) of creatine phosphokinase (CPK) is released rapidly into the circulation following stroke or transient ischemic attack,<sup>32</sup> seizure with prolonged loss of consciousness, and traumatic brain injuries whether mild, moderate, or severe—including in amateur boxers. CPK-BB is also elevated in neurological diseases and malignancy. Blood samples taken shortly before ECT and 1, 2, and 6 hours following bilateral ECT in a series of 31 patients of age 23 to 70 showed no alteration in serum CPK-BB concentrations, using a sensitive radioimmunoassay.<sup>33</sup>

CSF concentrations of three established markers of neuronal and glial generation were measured in nine patients before and after a course of six ECTs. These markers are CSF-tau protein, CSF neurofilament, and CSF-100 beta protein, and none showed significant change with ECT. Moreover, the CSF/serum albumin ratio was unchanged; this ratio reflects blood-brain barrier dysfunction. The authors concluded that there is no biochemical evidence of neuronal or glial injury or blood brain barrier dysfunction following ECT.<sup>34</sup> Serum concentrations of S100B protein measured in 22 depressed patients treated with ECT showed no change from baseline to after the ECT course or 6 months later.<sup>35</sup> Likewise, S-100 protein levels were unchanged throughout courses of ECT in 19 patients.<sup>36</sup>

Likewise, measurements of serum neuron-specific enolase concentrations, a marker of neuronal injury that increases in the bloodstream after generalized epileptic seizures and TBI, showed no increase with ECT treatment. One study of seven patients assayed serum enolase concentrations twice before ECT and at 16 different times after the first ECT, and again once before and after later ECTs, finding no variations among these concentrations.<sup>37</sup> Another study examined 14 patients before and along the course of bilateral ECT, finding no elevation in enolase concentration.<sup>38</sup> Later studies found no changes in serum enolase concentration after ECT in 10 patients<sup>39</sup> and in a different group of 19 patients.<sup>36</sup>

Elevation of serum neurofilament light concentrations (sNfL) correspond to neuronal axonal injury in neurodegenerative dementias and in multiple sclerosis.<sup>21</sup> In clinical practice, neurologists judged sNfL as useful in confirming or excluding neurodegeneration in 59 of 109 patients, suggesting 53% of patients with neurodegeneration showed abnormalities.<sup>40</sup> In two series of 15 patients each, concentrations of sNfL drawn both 24 hours and 7 days after ECT remain unchanged from baseline, and these concentrations did not differ from healthy controls.<sup>41,42</sup>

### HISTOLOGICAL EXAMINATION

No cell death or injury occurred the brains of laboratory monkeys after the electroconvulsive shock procedure,<sup>43</sup> the

laboratory animal model of ECT. Several reports describe brain autopsy examination of elderly patients who had received large numbers of ECT treatments. An 89-year-old woman with recurrent episodes of mania and documented history of more than 1250 bilateral ECT treatments showed no signs of brain injury on microscopic (histological) and macroscopic (gross anatomical) examination.<sup>44</sup> Postmortem brain examination of a 92-year-old woman after 91 ECT treatments showed no pathological changes that could be attributed to ECT, including in the hippocampus. Moreover, her cognition was intact as shown by perfect score on Mini-Mental State Exam 6 days before death.<sup>45</sup> Likewise, post-mortem examination of an 84-year-old man after 422 ECT treatments showed no pathological changes attributable to ECT. Examination included thickness and deep structures, such as basal ganglia, thalami, ventricles, hippocampus, amygdala, and brainstem.<sup>46</sup>

Microscopic postmortem examination of hippocampus from 12 patients who had received ECT for depression within 5 years of death revealed no substantial cell loss, structural changes, or signs of inflammation, and likewise for age-matched non-ECT patients and healthy controls<sup>47</sup>; all groups showed similar mild age-related changes. Patients included a 100 year old who received over 176 ECTs since age 90 and a 58 year-old who received more than 69 ECTs. Several studies cited above report brain growth and cell additions during the first few months after ECT,<sup>25,26</sup> pointing away from injury and demonstrating that the adult brain remains capable of growth, i.e., mitosis.

### DEMENTIA ONSET

There is no evidence of increase in dementia incidence in patients who received ECT. After an average of 34 years of follow-up on 1089 consecutive depressed inpatients treated with ECT, dementia was not more common than in 3011 matched depressed inpatients who did not receive ECT,<sup>48</sup> and age had no effect. Another follow-up study of 5901 patients 2.4 to 7.8 years after ECT reported that in those younger than 70 years, ECT was not associated with greater incidence of dementia than in age-matched patients not given ECT. For patients at least 70 years old, ECT was associated with significantly lower rate of dementia.<sup>49</sup>

### DISTINCTIONS BETWEEN TBI AND ECT

The kinetic energy of an object is calculated by the formula  $1/2$  of mass times velocity squared. This represents the ability of an impact to produce changes. High kinetic energy requires rapid physical movement but there is only slight to no physical movement during routine modern ECT, which is given under anesthesia, so produces virtually no kinetic energy. In ECT, there is no physical force that can produce skull injury or brain movement. Moreover, TBI is far more likely following rotational motion (i.e., shear) than a straight impact,<sup>10</sup> and there is no rotational motion during ECT. Before the ECT electricity is applied, patients are so deeply paralyzed they cannot breathe, and an anesthetist must provide ventilation.

In contrast to studies of TBI consequences, studies of patients before and after ECT have found no evidence of brain shrinkage, diffuse axonal brain injury, bleeding into the brain, cranial nerve injury, onset of epileptic seizure disorder, or signs of impaired motor actions or reflexes. Accordingly, ECT is not followed by any sign of brain injuries that accompany TBI.

The studies of MRI brain imaging cited above include all known reports of imaging both pre-ECT and again at least 6 months afterward; these examined a total of 87 patients, none of whom showed persisting brain changes. The higher incidence of brain changes after TBI shows that it is a different condition. Statistically comparing 0 of 87 ECT patients against 181 of 251 traumatic brain

injuries with MRI abnormalities,<sup>14</sup> Fisher's exact test shows negligible chance ( $P < 0.00000001$ )—as close to zero as statistical testing permits—that TBI and ECT are similar. To illustrate the strength of these results, consider the imaginary circumstance that brain abnormalities appear post-ECT in 5 of 87 ECT patients from undiagnosed deteriorative conditions such as head injury from a fall during the course of ECT treatment. Still, Fisher exact statistical test continues to show the same negligible chance,  $P < 0.00000001$ , of TBI and ECT similarity.

The studies of brain cell leakage cited above examined a total of 142 patients after ECT, and none showed cell leakage. Studies of blood markers after TBI show significant enzyme elevations but only one study found identified the incidence of these elevations.<sup>50</sup> It reported that sNfL in all of 14 boxers were elevated and higher than controls, with no overlap. Those experiencing severe head impact showed higher sNfL levels. Likewise, sNfL in all of 28 concussed professional hockey players were higher than controls with no overlap.<sup>50</sup> This totals sNfL elevation in 42 of 42 concussions. Statistically comparing 0 of 142 ECT patients with 42 of 42 concussed athletes, Fisher exact test shows what amounts to no chance ( $P < 0.00000001$ )—as low as statistical testing can find—that TBI and ECT are similar. Even these statistical results are understated; consider the imaginary circumstance that five athletes with concussions do not show sNfL elevations but 5 of 30 ECT patients do. Again, Fisher exact statistical test yields the same result, no chance  $P < 0.00000001$  that TBI and ECT are similar. Likewise, comparing 0 of 30 ECT patients showing sNfL elevations with 59 of 109 neurodegeneration patients<sup>40</sup> again reveals no chance ( $P < 0.00000001$ ) of similarity.

As further contrast between ECT and TBI, as noted above, TBI is followed by increased incidence of dementia but ECT is not. Moreover, ECT is not followed by structural brain changes or brain cell enzyme leakage as accompany interruption of blood flow or oxygen to the brain, brain toxin exposure, brain infection, or brain bleeds or tumors, and there is no logical rationale for ECT to produce these. Thereby, and including concussion, ECT has nothing in common with events, diseases, or agents that produce brain injury.

### LIMITATIONS

The analyses are limited by the data available, technology, and ethical considerations. The TBI data were not from randomized controlled studies, but deliberately exposing subjects to TBI is not ethical. The MRI imaging resolution and brain enzyme measurements are limited by the current state of technology; new different or more detailed measurements may produce other evidence. This study was limited to measurements of verifiable physical evidence indicative of brain structure. Cognitive performance and complaints and other subjective symptoms were not considered relevant and were not examined.

### CONCLUSIONS

Numerous studies show that all effects of ECT on brain structure are only temporary, disappearing within 6 months. Separately, there is no evidence of enzyme leakage from brain cells after ECT. Accordingly, there is no evidence of permanent structural changes or brain cell injury following ECT. In contrast, all known causes of brain injury produce persisting visible structural deteriorative brain changes and brain cell enzyme leakage in at least some people, and statistically significant differences from them show that ECT is not like any of them. Moreover, the sensitivity of identifying structural brain deteriorative changes is high enough to find they accompany major depression itself and also exposure to anti-psychotic drugs commonly used in treating major depression.

Claims that ECT causes “brain damage” or brain injury are not consistent with scientific understanding or knowledge, and dissemination of such claims amounts to misleading provocation of fear and alarm.

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