1960). For this reason continued treatment with antidepressant drugs for a period of time should be advocated in many cases. **How long do the effects of ECT last?** Efficacy duration is an unresolved issue, but since many studies (some just cited above) show that within 3–6 months one-third to about one-half of the patients have relapsed, it might be conjectured that the effect on the brain of a series of induced seizures would be limited to a few months. If this is true then it means that a patient suffering from an endogenous depression (which usually lasts 6–8 months if left untreated) who is given ECT soon after the onset of the illness may belong to the relapse group, whereas a patient who has been suffering for several months with or without unsuccessful drug treatment may profit more definitively from ECT because of the time of the treatment relative to the onset of the illness.

**Is there evidence for such a hypothesis?** I find support for a hypothesis of a time-limited effect of ECT in three studies of different parameters.

1. In a study carried out in Copenhagen (Heshe, Röder & Thelgaard 1978) very careful neuropsychological examinations were performed in a study of patients undergoing ECT with either bilateral or unilateral electrode placement. In both groups it was found that declining cognitive disturbances could be measured up to about 3 months after the last treatment.

2. Electrophysiological studies carried out by Small (1974) show that EEG changes such as admixture of slow-wave activity and bursts of spikes following successful convulsive therapy could be demonstrated with declining intensity up to 3 months after the last seizure, whereupon the EEG became completely normal.

These two studies involving behavioral and neurophysiological changes in patients seem to show the same time course for unwanted disturbances, and they also seem to support the idea that some of the biological changes that may be assumed to be responsible for wanted and unwanted effects are quite parallel.

3. In a study by Bolwig and Jørgensen (1981) in which specific proteins were examined after repeated electroconvulsions, a significant increase was found in both Synaptin, which is assumed to regulate endo-exocytosis in the synaptic cleft, and the synaptic membrane protein D2, which is assumed to be involved in the “recognition” process during brain development. The authors suggest that these changes reflect an increase in the number of synaptic vesicles and a remodeling within the central nervous system that justifies the term electrostimulation. The findings were significantly higher in the hypothalamus than in other regions, and it was noteworthy that three months after the last seizure values returned to normal. The relevance of these experimental findings in a discussion of basic mechanisms of ECT is supported by the finding of Jørgensen, Bock, Bech & Rafaelsen (1972) that in the cerebrospinal fluid of patients undergoing antidepressant therapy the amount of the protein D2 was increased after the remission of the depressive illness.

**Where does ECT work?** The importance of deep brain structures has already been well emphasized in the target article. In one of the studies cited (Bolwig, Hertz & Westergaard 1977) it was found that the increased blood-brain barrier permeability to large molecules was a phenomenon taking place mainly in deep brain structures despite the fact that it was the increased cerebral blood flow during seizures that turned out to be the determining factor for this effect. In contrast, an increase of cerebral blood flow induced by increasing blood pressure alone does not lead to the same finding, suggesting a rather specific effect on central areas of the brain when flow and permeability increases are induced by seizures.

Weiner’s paper clearly reflects the reasons for encouraging interest in an understanding of basic mechanisms of ECT, a treatment modality that is unlikely to be replaced in the near future by other types of treatment in the therapy for severe depressive illness and for certain other conditions mentioned by Weiner. As has been indicated by Fink (1979), the treatment of delirious conditions with ECT is rare in the United States whereas it is rather common in Denmark (Heshe & Röder 1975; Kramp & Bolwig 1981).

It is highly gratifying that a treatment modality that has been so strongly criticized, mostly on irrational grounds, has now received the thorough and careful evaluation provided by the target article.

**Electroshock therapy and brain damage: The acute organic brain syndrome as treatment**

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The psychiatric literature is wondrous to behold and even more wondrous to review; from that vast body of research and opinion one can cull a mass of unqualified support for the efficacy and harmlessness of every imaginable assault upon the brain: classical prefrontal lobotomy, carbon dioxide asphyxiation, insulin coma, total body freezing, and poisoning with a variety of neurotoxins, such as arsenic and cyanide (for reviews, see Breggin 1979; 1981a; 1981b; 1983). Even when the treatments begin to fall into disrepute, as with classical prefrontal lobotomy and insulin coma, the reviews that appear in the literature will be almost uniformly positive to the bitter end.

In this light, it is certainly no surprise that a proponent of electroshock, Dr. Richard Weiner, should be able to use the literature in defense of ECT. More surprising, perhaps, this same strongly promotional ECT literature can be reviewed by a critic of the treatment who finds ample evidence for the dangerousness and destructiveness of the treatment (Breggin 1979; 1981a). The evidence consists of human and animal autopsy studies, animal behavioral and biochemical studies, human brain-wave research, psychological testing, and multiple clinical reports. In many instances, such as the animal autopsy literature, the studies reviewed may be the same, but the analyses and deductions are diametrically opposed. Short of reading the entire literature for themselves how, then, are intelligent, scientifically minded individuals to make up their own minds? They can start with common sense, an elementary knowledge of psychology and neurology, and most important, a genuine interest and concern for the actual experience of the patient undergoing the treatment.

From the viewpoint of the patient undergoing the treatment, there is one overriding fact about every form of convulsive therapy: the production of an acute organic brain syndrome. A series of artificially induced convulsions produces to one degree or another a generalized dysfunction of the brain and mind, characterized by disorientation, disruption of memory functions, impairment of intellectual functions and judgment, and emotional lability, varying from apathy to euphoria. Curiously enough, even attempts to alleviate depression by self-medication, such as sniffing glue (toluene intoxication) or drinking alcohol can produce symptoms of generalized central nervous system dysfunction.

It is therefore wrong and misleading to ask whether ECT can produce serious brain damage. It always produces serious brain damage as manifested in the acute organic brain syndrome. The question should be, Is it safe to assume that many or most patients experience a complete recovery from this trauma? Similarly, it is misleading to seek a subtle biochemical mechanism to explain the action of ECT (or any other trauma to the brain). We should ask ourselves more directly, How does an acute organic brain syndrome give the appearance of an improvement?

In regard to recovery from damage, my review of the literature suggests that the electrical current is the main culprit in producing the damage. It follows the path of least resistance throughout the brain, the vascular tree, producing vasospasm,
blanching, breakdown of the blood-brain barrier with the extra- 
vacation of toxic substances, petechial hemorrhages around the 
small blood vessels, glial reactions, and cell death (see Breggin 
1979 for a detailed review).

That patients frequently complain about memory dysfunction 
long after ECT is well known. Weiner confirms that testing also 
demonstrates a loss of personal memories. That psychological 
tests for memory and other intellectual functions are frequently 
negative is irrelevant, since the tests are not used anywhere else 
in medicine or neurology to prove an absence of pathology. 
Rudimentary neurology tells us that a negative psychological 
test cannot rule out even a gross lesion in the brain, let alone 
subtle but widespread damage, such as that found in chronic 
drug intoxication or ECT.

What is the improvement seen following ECT? It is the direct 
effect of the acute organic brain syndrome, which not only 
blunts patients’ memory and awareness of their problems, but 
produces a corresponding artificial apathy or euphoria. In so-
called retarded patients, the euphoria will be taken as an 
 improvement, and in agitated patients, the apathy will be seen 
as an improvement. The “nurses” or occupational therapists’ 
notes on the ward, however, will show that the patient is no 
longer able to focus attention, remember everyday details, or 
complete complex tasks. Why doesn’t the “cure”? Because 
the gross effects gradually subside, and as the patients’ brain 
function approximates normal again, their problems again be-
come apparent.

Is there hope for newer variations in the technology of the 
treatment? No, because the treatment “works” by means of the 
trauma. If unilateral ECT causes less trauma, as some propos-
ants advocate, then it will often be given in longer courses to 
produce the equivalent trauma. In reality, the most important 
modern modification, the use of anesthesia, raises the seizure 
threshold, requiring more intense or more prolonged doses of the 
offending electrical current. A review of the literature 
confirms that modern clinical ECT uses a larger dose of electro-
cal energy than the premodified era (Breggin 1979). Further-
more, the appearance of reduced damage in unilateral or non-
dominant ECT is misleading. Damage to the nondominant side 
produces less verbal memory disability, but more visual memo-
ry disability. More ironically, nondominant damage, as any 
textbook of neurology will confirm, tends to produce a greater 
degree of denial of symptoms on the part of the patient (this 
particular form of confabulation is called anosognosia). Non-
dominant ECT may even be more damaging, since it focuses 
the energy in a more localized area, producing more severe local 
trauma as manifested in transient neurological signs on the 
opposite side and focal brain-wave abnormalities on the same 
side (Breggin 1979).

ECT is an irrational and often brutal treatment. The psychi-
atric and medical professions ought to place a self-imposed ban 
on the therapy. Lacking such self-restraint, the public will 
continue to protest and even to take action to halt the treatment.

Note: See “Best ECT Resources” for the citations.

Possible brain damage by electroconvulsive therapy: Memory impairment and cultural 
resistance

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Weiner’s target article provides a thorough review of the state of 
the art of electroconvulsive therapy (ECT). He suggests investiga-
tive scientific questions aimed at improving our understand-
ing of the memory impairments said to be associated with ECT. 
A persistent theme is that memory studies yield contradictory 
results. The following conceptualization might account for this; 
it is based upon similar reasoning applied to studies of failing 
memory in the elderly (Cherkin & Riege 1983). The fundament-
al point is that current research strategy attempts to use one or 
another unitary hypothesis to deal with a multifactorial phe-
nomenon, rich in interactions. Memory is a many-splendored 
thing. On the one hand, it can survive severe insults to the 
brain, such as ECT, epileptic seizures, prolonged unconscious-
ness, and deep anesthesia. (As an extreme example, consider 
the fact that chicks treated with carbon dioxide under near-
lethal conditions, leading to an isoelectric EEG for 3 minutes, 
recover and retain the memory of pre-CO2 training; Porter 
1974.) On the other hand, simply elevating the level of free L-
proline in the brain, shortly after training chicks in the same 
paradigm, causes amnesia with only transitory minor changes 
in the EEG and behavior (Gerbrandt, Eckardt, Davis & Cherkin 
1977) and without inhibiting brain protein synthesis (Cherkin, 
Bennett & Davis 1981). 

There are myriad alterations in the brain that can affect 
memory processing, in addition to those listed by Weiner. ECT 
triggers a wide array of these alterations, as primary or second-
ary effects. Any one of these could interact with any other. 
 Drugs acting on the cholinergic system interact to a remarkable 
extent on their effects on memory, both enhancing and ances-
tic, with mutual potentiation of 20-fold or more (Flood, Smith & Cherkin, in press). If two cholinomimetics (e.g. arecoline 
and oxotremorine), or two anticholinesterases (e.g. édrophonium 
and phystostigmine) can interact with each other so powerfully, 
interactions between different neural systems can be expected 
to be the rule, not the exception. If this is correct, then 
uncontrolled interaction would be expected to lead to the 
contradictory results observed in apparently similar memory 
experiments.

Weiner refers to studies by Zornetzer (1974) as helping to 
clarify the confusion concerning the effect of ECS and ECT 
stimulus-intensity factors upon memory. Earlier studies (Cher-
kin 1969) using a chick model that bears many similarities to 
mammalian models, support Zornetzer’s findings. In brief, full 
clonic-tonic motor convulsions, the classical criterion for a 
maximal convulsive treatment required to cause full retrograde 
amnesia, proved to be misleading because such convulsions 
could be followed by no retrograde amnesia, or partial amnesia, 
or total amnesia (Cherkin 1969). The reliable criterion turned 
out to be the total number of 200-µV spikes resulting from the 
treatment—whether the convulsive stimulus was fluoroethyl (Indo-
klon®) (Herz, Spooner & Cherkin 1970), or intracranial ECS 
(Gerbrandt, Herzog & Cherkin 1973), or pentyleneetetrazole 
(unpublished data).

A major problem that limits the appropriate therapeutic 
application of ECT in the United States is cultural resistance 
because of widespread public misunderstanding. The reality is 
that ECT of acute endogenous depression is the most effective 
and most rapid therapy in the entire armamentarium of psychia-
try. The typical public view, however, is that ECT is a bad 
procedure which should be constrained by legislation because it 
“messes with the brain,” alters personality, is painful, converts 
defenseless patients into zombies, is not well worked out, and is 
used to line the pockets of mercenary physicians. These false 
perceptions will not be altered by the detailed studies of ECT 
parameters so ably reviewed by Weiner and so necessary for 
further perfecting clinical ECT. Today’s evidence is already 
overwhelming that ECT does not mess up the brain, is not 
painful, and does not create zombies. The challenge is clear –
there is a professional imperative to enlighten the public, 
patients, and concerned legislators. To change a cultural preju-
dice is not easy; the task would be simplified by initially limiting 
general support of ECT for the “routine treatment of depression 
in patients refractory to drug therapy, with an upper limit of 12 
ECT treatments in a given course. Such limitations would 
exclude fewer than 18% of today’s ECTs. Treatment of schizo-