Shock Treatment, Brain Damage, and Memory Loss: A Neurological Perspective

BY JOHN FRIEDBERG, M.D.

The author reviews reports of neuropathology resulting from electroconvulsive therapy in experimental animals and humans. Although findings of petechial hemorrhage, gliosis, and neuronal loss were well established in the decade following the introduction of ECT, they have been generally ignored since then. ECT produces characteristic EEG changes and severe retrograde amnesia, as well as other more subtle effects on memory and learning. The author concludes that ECT results in brain disease and questions whether doctors should offer brain damage to their patients.

A 32-YEAR-OLD WOMAN who had received 21 ECT treatments stated 5 years later,

> One of the results of the whole thing is that I have no memory of what happened in the year to year and a half prior to my shock treatments. The doctor assured me that it was going to come back and it never has. I don't remember a bloody thing. I couldn't even find my way around the town I lived in for three years. If I walked into a building I didn't even know where I was. I could barely find my way around my own house. I could sew and knit before, but afterward I could no more comprehend a pattern to sew than the man in the moon. (1, p. 22)

By 1928, 10 years before the introduction of electroconvulsive therapy, it was known that accidental death by cardiac arrest could result from as little as 70 to 80 milliamperes in the human (2). It was also known in this early period that voltage applied to the head, as in legal electrocution, produced hemorrhage and rupture of cranial contents. Ugo Cerletti (3) demonstrated that electricity in the range of 100 volts and 200 milliamperes is rarely fatal when the current path is confined to the head, but does evoke a grand mal seizure marked by a stereotyped succession of events. A tetanic muscular contraction, the "electric spasm," is followed after a latency of seconds by unconsciousness, a high voltage paroxysmal spike and sharp-wave discharge, and a clonic convulsion. Upon recovery of consciousness the subject is left with a transient acute brain syndrome, a high likelihood of permanent brain damage, and greater retrograde amnesia than is seen in any other form of head injury.

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BRAIN DAMAGE IN EXPERIMENTAL ANIMALS

Before examining the premise that ECT damages human brains, a brief discussion of the lesions produced in animals by electrically induced convulsions is worthwhile. The many reports on this subject indicate that petechial hemorrhages scattered throughout both white and gray matter and concentrated in the path of the current are the most consistent finding. If animals are sacrificed after a delay of days or weeks following a convulsive series, hemosiderin pigment in phagocytes remains as evidence of vascular insult. Proliferation of glial cells, neuronal changes, and dropout are also commonly reported.

In 1938, the year of the first use of ECT on a human being, Lucio Bini, Cerletti's collaborator, reported "widespread and severe" brain damage in dogs with mouth to rectum electrode placement (4). At least seven subsequent animal studies employing conventional cranial electrodes supported his findings (5–11). These culminated in the exhaustive controlled experiment by Hans Hartelius in 1952 (12). This researcher found discernible vascular, glial, and neuronal changes in cats subjected to a maximum of 16 shocks. The animals were not paralyzed but were protected from physical injury during the seizure. Damage was slight but consistent, and the author concluded: "The question of whether or not irreversible damage to the nerve cells may occur in association with ECT must therefore be answered in the affirmative." Furthermore, by examination of unlabeled slides alone Hartelius was able to correctly recognize 8 of 8 slides from shocked animals as well as 8 of 8 controls. Although he considered many of the vascular and glial changes to be reversible, there was no mistaking the brain of a shocked animal for that of a control.

Since that time, ECT in humans has been modified through the use of oxygen and muscle paralysis to reduce the incidence of bone fractures. Although it is believed that these modifications also reduce brain

damage, there are no animal studies to support this idea. On the contrary, recent work in England by Meldrum and associates (13, 14) on status epilepticus in primates suggests that the overexcited neuron by itself may be an important factor in seizure damage, especially in the hippocampus.

HUMAN BRAIN DAMAGE

Let us turn now to the neuropathological findings in humans who died during or shortly after ECT. As in lower animals, bleeding is the most frequent nonspecific tissue response to injury and the one seen most often after electric shock. The first autopsy study in this country revealed brain damage identical to that seen in experimental animals. Alpers and Hughes (15) described the brains of 2 women who had received 62 and 6 shocks, respectively. The first woman's seizures had been suppressed by curare. Both brains showed hemorrhagic lesions around small blood vessels, rarefaction of tissue, and gliosis.

Throughout the 1940s similar reports continued to call attention to brain changes after ECT, including cases in which oxygen and curare had been administered (16). In 1948 Riese (17) added 2 more autopsy studies to the growing list and commented, "In all observations of sudden death after electric shock reported so far, petechial hemorrhages, cellular changes and some glial proliferation stand out prominently, as an almost constant whole."

Pathologists were especially interested in cases that discriminated between the direct effect of electricity and the mechanical and hypoxic effects secondary to convulsive motor activity. In 1953 Larsen reported on a 45-year-old man who had been given 4 electroshocks in the course of 5 days. The ECT did not induce any convulsions. The subject died from pneumonia 36 hours after the fourth electroshock. At autopsy fresh subarachnoid hemorrhage was found in the upper part of the left motor region—"at the site where an electrode had been applied" (18).

In 1957 Impastato summarized 254 electroshock fatalities. Brain damage was the leading cause of death in persons under 40 years of age, and nearly one-fifth of all cerebral deaths were hemorrhagic (19).

Some physicians were alarmed by the evidence of human brain damage. In 1959 Allen reported 18 cases in which he had found signs and symptoms of neurological sequelae following ECT. He concluded, "It is probable that some damage, which may be reversible but is often irreversible, is inseparable from this form of treatment," and called for "more serious consideration" of the entire procedure (20).

In 1963 McKegney and associates (21) reported the case of a 23-year-old man who became comatose 15 minutes after a single shock. The significance of this case was twofold: first, a complete physical and neurological examination was reportedly normal prior to ECT, and second, the ECT technique was contemporary and impeccable. The patient had received .6 mg of atropine, 16 mg of succinylcholine (Anectine), and forced oxygenation pre- and post-shock. ECT parameters were conventional, i.e., 130 volts for .3 seconds. Four days later a brain biopsy showed diffuse degeneration of neurons with hyperplasia of astrocytes. The young man never regained consciousness and at autopsy 2 months later evidence of old hemorrhage was found in the brain. This was the last detailed report in the English-language literature.

The damaging effects of ECT on the brain are thoroughly documented. All told, there have been 21 reports of neuropathology in humans (22–36). It is interesting that, despite the importance of a negative finding, there has not been a single detailed report of a normal human brain after shock.

ELECTROENCEPHALOGRAPHIC EFFECTS OF ECT

Like other insults to the brain, ECT produces EEG abnormalities. Diffuse slowing in the delta and theta range, increased voltage, and dysrhythmic activity are seen in all patients immediately following a series of bilateral ECT and, according to Blaurock and associates (37), may persist more than 6 months in 30% of the cases. Such slowing suggests damage to the thalamus.

Sutherland and associates (38) showed that the side of the brain shocked with unilateral ECT could be predicted by double-blind assessment of EEG tracings.

The seizure thresholds of the hippocampus and other temporal lobe structures are the lowest in the brain; considerable interest has centered recently around "kindling," or seizure induction by subthreshold stimulation of these areas in animals (39). The induction of a permanent epileptic disorder following ECT in humans was first reported in 1942 and other reports followed (40).

MEMORY LOSS

ECT is a common cause of severe retrograde amnesia, i.e., destruction of memories of events prior to an injury. The potency of ECT as an amnestic exceeds that of severe closed head injury with coma. It is surpassed only by prolonged deficiency of thiamine pyrophosphate, bilateral temporal lobectomy, and the accelerated dementias, such as Alzheimer's.

After ECT it takes 5 to 10 minutes just to remember who you are, where you are, and what day it is. In the first weeks after a full course, retrograde and, to a lesser extent, anterograde amnesia are evident to the casual observer. But as time passes compensation occurs. As in other forms of brain injury, the subject is often oblivious to the residual deficit. Unless specific memories essential to daily living are discovered to be unavailable the victim may never know for sure the extent of memory loss. Unless sensitive tests for spon-

taneous recall of personal preshock data are employed, no one else will know either.

The memory loss following ECT generally follows Ribot's law for all pathological amnesias: the new dies before the old. This, of course, is the opposite of normal forgetting. Squire, however, has shown that the loss may extend to items learned more than 30 years before (41).

The effect of ECT on memory was common knowledge within a few years of its introduction. There were reports of persons who forgot they had children (42, 43), although most amnesias involved humbler matters, such as the woman who forgot how to cook familiar dishes (44) and another who couldn't remember her own clothing and demanded to know who had put the unfamiliar dresses in her closet (45). Some doctors dismissed these sequelae as trivial or transient, although one psychiatrist remarked that psychotherapy was useless in patients undergoing ECT because they couldn't remember "either the analyst or the content of the analytic sessions from one day to the next" (46).

Numerous such case reports finally led to a definitive study of the effects of ECT on memory by Irving Janis in 1950 (47). He found that all 19 subjects in a controlled prospective investigation had significant memory loss 4 weeks after ECT, compared to negligible losses among control subjects. He also noted that these losses may involve events of early childhood dating back 20 to 40 years, with the more recently encoded memories being the most vulnerable. Patient E, for example, a 38-year-old woman, had told Janis in an interview prior to ECT that thyroid medication had caused heart palpitations and panic which led to her admission to the psychiatric hospital. When asked after a course of 10 shocks if she had ever taken thyroid she responded, "I don't think so."

In the late 1940s, when the enthusiasm for ECT seemed to have passed its peak (48), Lancaster and associates (49) advocated the use of unilateral nondominant ECT in treating patients who earn their livelihood with retained knowledge. In this variant the current path and most of the damage is confined to the nonverbal side of the brain, usually the right hemisphere. This exploits the well-known neurological phenomenon of anosognosia, or denial, that is associated with right-hemisphere lesions—victims can't verbalize their difficulties. They complain less. Cohen and associates (50), however, using design-completion tests, proved that shock to the right hemisphere produces its own kind of memory loss—visual and spatial. Inglis found in 1970 (51) that the effects of unilateral ECT were comparable to those of right and left temporal lobectomy, with identical impairment of memory and learning.

Recently there has been a good deal of human experimentation in a futile effort to find electrode placements that eliminate amnesia. As the use of ECT has shifted from state hospitals to private practice, the literature has focused more and more on memory loss. Although some studies have purported to show improvement of learning ability after ECT, not one used sham ECT as a control and few used any controls at all.[1]

In regard to more general intellectual ability, a study in 1973 (54) showed that the performance on the Bender Gestalt perceptual motor test of 20 institutionalized subjects who had received 50 or more ECT treatments 10 to 15 years before testing was significantly impaired compared to the performance of 20 carefully matched control subjects who had not received ECT. The authors inferred that ECT had caused permanent brain damage.

MECHANISM OF ACTION OF ECT

The mechanism of action of ECT can now be summarized on the basis of evidence accumulated since its introduction. Penfield and Perot showed in the 1950s that memory traces may be evoked by direct electrical stimulation of the temporal lobe cortex, and nowhere else (55). Scoville and Milner (56) discovered that bilateral hippocampal resection utterly abolished the ability to remember any new material, resulting in a catastrophic inability to learn. From numerous studies of the neuropathology of the amnestic-confabulatory syndrome of Korsakoff it is known that the mammillary bodies, the dorsal median nuclei of the thalamus, and the gray matter surrounding the third ventricle and aqueduct are essential to the general memory process. All of these critical brain structures are just beneath the thin squamous plate of the temporal bone, within seven centimeters of the electrodes, in the direct path and highest density of the current during ECT.

CONCLUSIONS

From a neurological point of view ECT is a method of producing amnesia by selectively damaging the temporal lobes and the structures within them. When it was first introduced it was only one of several methods of producing brain damage employed in psychiatry, including insulin coma (1927), camphor and pentylenetetrazol (Metrazol) injections (1933), and prefrontal lobotomy (1935). It is the only such method from that era still used on a large scale. It is highly unlikely that ECT, if critically examined, would be found acceptable by today's standards of safety.

From a neurological point of view ECT produces a form of brain disease, with an estimated incidence of new cases in the range of 100,000 per year (57). Many psychiatrists are unaware that ECT causes brain damage and memory loss because numerous authorities and a leading psychiatric textbook (58) deny these

[1] Sham ECT, an essential control technique, has been employed in only two studies, which were tests of efficacy, not tests of memory loss. Neither study showed any superiority of ECT over the control treatment (52, 53).

facts. Others, who know of its effects, argue that the interruption of unpleasant states of mind is worth the damage. Some are beginning to give the client a truly informed choice, although most state laws still allow ECT to be imposed if the doctor feels that "good cause" exists.

Assuming free and fully informed consent, it is well to reaffirm the individual's right to pursue happiness through brain damage if he or she so chooses. But we might ask outselves whether we, as doctors sworn to the Hippocratic Oath, should be offering it.

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Current Perspectives on ECT: A Discussion

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MY COMMENTS about each of the preceding papers on ECT reflect my personal views and not those of the former Massachusetts Task Force on ECT (1) or the American Psychiatric Association's Task Force on ECT. Space limitations preclude a complete discussion of each of the papers, but I will attempt to address at least some of their important or controversial points.

ECT: A NEUROLOGICAL PERSPECTIVE

In my view, Dr. Friedberg has weakened his position by the manner in which he has gathered the evidence to support it. The questions he raises are relevant; they have been asked by many others. However, he has attempted to answer them with data that have been carelessly culled from the literature and frequently reported inaccurately.

Dr. Friedberg's evidence against ECT is arranged in four main sections. He reports on the neuropathological findings in experimental animals subjected to electrically induced convulsions, and in humans who have died during a course of ECT or within weeks or months afterward. He then reports studies and subjective accounts of memory loss and, finally, EEG changes. He concludes that "from a neurological point of view ECT is a method of producing amnesia by selectively damaging the temporal lobes and the structures within them." He states that "ECT produces a form of brain disease" of epidemic proportions and asks why doctors, who are sworn to the Hippocratic Oath, are offering it.

Dr. Friedberg has listed 58 references in his article. I have examined 25 of these and selected those which seemed especially relevant to his argument.

In presenting his evidence for brain damage in animals exposed to electrical currents, Dr. Friedberg states: "In 1938, the year of the first use of ECT on a human being, Lucio Bini, Cerletti's collaborator, reported 'widespread and severe' brain damage in dogs. At least seven subsequent animal studies confirmed his findings." The following quotation is from the Bini reference:

> We have so far employed exclusively the method of Viale, which consists of passing the street current (120 volts) for a very short time (1/15 to 1/20 second) through the entire body of the animal with one of two electrodes (carbons from a voltaic arc) in the mouth and the other in the rectum.
>
> With this method we succeeded in producing constantly typical epileptic attacks in dogs. During the passage of the current the animal howls and has a violent tonic spasm with opisthotonus which lasts several seconds after the circuit is opened. Then there appear frequent and violent generalized tonic and clonic convulsions with foaming at the mouth, biting of the tongue, and incontinence of urine and feces. The duration of this second phase varies from 1–2 minutes. There follows a comatose state with complete muscular relaxation, absence of corneal and pupillary reflexes and stertorous breathing. In a short time the

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