

Brain Damage
ECT - brain damage

Exacerbation of Organic Brain Disease By Electroconvulsive Treatment

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Electroconvulsive treatment (ECT) is considered a safe procedure, although major complications do occasionally occur. Fractures of vertebral or long bones were once common, but with the use of muscle relaxants such accidents are now rare. One of the more serious complications is the abrupt presentation of organic neurologic disease during the course of treatment. It is possible that some instances of exacerbation of organic brain disease during ECT are due to chance, or to the fact that there are many patients with progressive organic brain disease in all large psychiatric hospitals.

The following six cases were seen in one psychiatric hospital (Dorothea Dix) during a three-year period. This paper suggests that ECT may directly accentuate underlying organic disease. Some mechanisms of such deleterious effects of ECT are reviewed.

Clinical Material

Case 1

A 41-year-old white man was committed by court for petty larceny and then gradually displayed confusion, a clumsy gait, and a severe memory defect. The tentative initial diagnosis was chronic brain syndrome of undetermined type. There had been a gradual loss of energy and ability to work. Examination of his mental status revealed confusion and defective memory for recent events.

Presumably because of the progressive mental decline, he received ECT for a possible depression. Immediately after the first treatment he became rigid, comatose, and was decerebrate for more than 24 hours. Deviation of the head to the right and a mild right hemiparesis then developed, followed by gradual improvement over the next week.

Spinal puncture and examination of fluid shortly after ECT revealed a protein content of 135 mg/100 ml, 21 lymphocytes, strongly positive tests for syphilis, and an abnormal colloidal gold curve. Plans for arteriography or ventriculography were cancelled when the serum Wasserman test was reported shortly after the ECT. The patient gradually improved while receiving penicillin therapy. Several months later there

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was a mild residual defect in memory, but otherwise no abnormalities were noted on neurologic examination.

Case 2

A 47-year-old white woman was admitted to the alcoholic service in a poor nutritional condition and with multiple bruises and abrasions on the body. She was treated with paraldehyde and evidenced no confusion or convulsions during her hospital stay. She was soon allowed a trial visit home, from which she returned two months later both intoxicated and confused.

Two days after readmission she was agitated and depressed and was therefore given one electroconvulsive treatment. Immediately after this treatment she was unresponsive and semicomatose. There were no focal neurologic abnormalities, although the right optic disc was indistinct.

After two days of observation the coma was more profound and the reflexes on the left side became hyperactive. A right carotid arteriogram revealed the anterior cerebral artery to be shifted to the left. A large subdural hematoma was evacuated, and the patient immediately became responsive and alert.

Case 3

A 44-year-old man was admitted because of depression. Several hours after his first electroconvulsive treatment he was observed to have right hemiparesis and he remained semiconscious for several days. Retinal artery pressures at this time revealed marked inequality with a decreased carotid pulsation on the left. Electroencephalograms showed diffuse slowing over the entire left hemisphere. Bilateral carotid arteriograms done some months after the onset of hemiplegia revealed complete obstruction of the left carotid artery with cross-over filling of the left anterior cerebral artery from an injection of the right internal carotid. The hemiplegia improved following physical therapy. The aphasia almost totally cleared, but the patient remained confused and continued to be institutionalized.

Case 4

A 49-year-old woman had had a right radical mastectomy for carcinoma one year prior to admission to the state hospital because of depression. For three months before admission she had had mild headaches. A month before admission she noted discomfort in her right shoulder. She had one generalized seizure immediately prior to admission to the hospital.

At admission no abnormalities on general physical examination were recorded, although she was noted to be somnolent and withdrawn. She received one ECT and immediately became unresponsive. On examination several hours after treatment there was severe

bilateral papilledema with hemorrhages. The lower left side of her face was weak, and left hemiparesis was obvious. Her neck was stiff. The pineal artery was shifted to the left, and bilateral carotid arteriograms revealed a tumor stain in the right parietal region. Brain scan with radioisotopes confirmed the lesion, which was found to be a metastatic carcinoma at operation. The patient died several days after craniotomy.

Case 5

A 57-year-old registered nurse had undergone excision of a carcinoma of the colon five years prior to admission for treatment of depression. Two months before this admission an exploratory laparotomy with readjustment of the colostomy wound had revealed no sign of recurrence of the cancer. She had had several previous depressions and had once attempted suicide. ECT and antidepressant drugs had afforded moderate relief of the depressive episodes.

Several days prior to another course of ECT she complained of difficulty in writing and had a dull headache. Immediately following the first treatment her right side became weak. On examination three hours later she had moderate hemiparesis, but was able to follow commands well. There was mild dysphasia and drowsiness. The cranial nerves were generally unremarkable with the exception of weakness of the lower portion of the right side of her face. Deep tendon reflexes were increased on the right, and an abnormal plantar response and a Hoffman reflex were elicited on the same side. Lumbar puncture and examination of spinal fluid revealed normal pressure and 80 mg of protein per 100 ml. A left carotid arteriogram done the day she became hemiparetic revealed a single left frontal mass. Surgery was not attempted. Autopsy disclosed several metastatic brain lesions, the largest of which was in the left frontal region.

Case 6

A 20-year-old man was transferred from prison because of persistent pain in the neck and head considered to be functional in origin. He was indifferent, depressed, and hostile, and a course of ECT was started. Following the sixth treatment he became stuporous and was noted to have severe bilateral papilledema with hemorrhages in both eyegrounds. The left arm was weak and the plantar reflex was abnormal on the left. The left lower part of the face sagged and his neck was slightly stiff. Bilateral carotid arteriograms revealed an upward and forward displacement of the right middle cerebral artery. Ventriculograms and exploratory surgery exposed a sarcoma of the cerebellum. He died of respiratory failure several days later.

Discussion

The physiologic effects of electroshock in each of the cases reported may not have been identical, but the clinical result was similarly disastrous in each instance. In 5 of the 6 cases the unfavorable effect appeared after the first treatment.

In the first case the patient undoubtedly had active syphilis at the time of the treatment. Paretics have received ECT without immediate incident¹ but at a time when the disease was quiescent. Although the complication is seen infrequently in modern times, it was once well recognized that patients with tertiary syphilis can suddenly become hemiplegic, probably because of the severe (Heubner's) endarteritis which can be associated with lues. Older textbooks state that patients with syphilis who have seizures may manifest a marked increase in their disability after each seizure. The period of anoxia and increased metabolic demand that may accompany the convulsion increases the effects of a partial ischemia.

The patients with brain tumor may have had a postictal effect similar to Todd's paralysis. Although Todd's paralysis has been attributed to cortical "exhaustion," mechanisms such as extreme or inappropriate cortical inhibition are probably more significant.^{2,3} It can be postulated that marked functional neurophysiologic changes have occurred in the brain substance adjoining the tumor of patients who do poorly following convulsions. It is also probable that a seizure briefly increases the cerebral edema around a neoplasm, perhaps by inducing changes in vascular permeability.

Cole and Spatz⁴ have emphasized that seizures are not rare in patients with chronic subdural hematoma, but such seizures presumably relate more to underlying cortical damage than to the effect of hematoma itself. There is no evidence in the literature that seizures in patients with subdural hematomas are likely to produce clinical worsening, and there is no clear explanation of why ECT in patients with subdural hematomas would produce such worsening of symptoms as was noted in Case 2. Minor systemic electrolyte changes are known to occur during a course of electric shock therapy, and these may lead to further accumulation of fluid in a subdural hematoma that is already large. Friable granulation tissue in the membrane of a subdural clot might bleed during a convulsion and thereby add to the total mass. It would seem most likely, however, that in

Case 2 the brain was already tightly compressed by the hematoma, and the electrically induced seizure produced an increase in the diffuse cerebral edema beneath the subdural mass.

ECT can occasionally be hazardous in more diffuse types of organic brain disease. Senile patients with minimal neurologic findings but with a history suggestive of cerebrovascular disease in the past sometimes have prolonged confusion after ECT. Any procedure such as cerebral arteriography may be more hazardous in aged patients, and it is not surprising that an induced seizure added to the vascular changes of age can produce a similarly high incidence after ECT if there has been previous brain trauma. Patients with diffuse vascular disorders such as lupus erythematosus could be expected to respond poorly to ECT, and we have seen two such cases.

In our institution one case of spontaneous subarachnoid hemorrhage was noted immediately after ECT. The patient refused arteriography, and the cause of bleeding is unknown. Since spontaneous subarachnoid hemorrhage occurs in many situations, it is hard to relate the sudden intracranial bleeding solely to electroconvulsive treatment.

Physiologic changes associated with convulsions and ECT

ECT is more than just a convulsion, since premedication and the direct effect of the electric current on brain or heart are also involved. Wilson and Gottlieb⁷ report differences in the degree and quality of postictal confusion depending on the placement of the electrodes, with current to the left hemisphere producing more verbal disability. Very high voltage and a misplacement of electrodes is capable of producing unequivocal histologic change in the brain, according to Alexander and Lowenbach⁸. Furthermore, ECT can be associated with anoxia related to medication or aspiration, and such anoxia may of itself produce brain damage. Nevertheless, the conspicuous physiologic effect of ECT remains the phenomenon of the convulsion.

For 40 years Spielmeier⁷ and others have,

emphasized that a seizure can occasionally produce brain damage, although most current neurologists feel that this does not happen unless significant anoxia occurs. Certainly almost all deaths which have followed seizures were associated with organic brain disease, status epilepticus, or asphyxiation. Although the normal brain is not permanently injured by a single convulsion, with experimental ECT there are changes in sensitivity to shock which relate to the intensity and duration of electrical stimulation, and Pollack and his group⁹ reported that the threshold of seizure activity may rise with successive treatments. In addition to such functional and probably temporary neurophysiologic changes occurring during therapy, it is possible that more permanent changes can occur in neuronal association patterns after repeated ECT.

There are surprisingly few data regarding the metabolic effects of convulsions. As early as Gowers' classic text,⁹ in 1885, albuminuria and other minor urinary changes were mentioned, but a detailed study of the metabolism of serum and spinal fluid during convulsions has not been done. It is conceivable that a depletion of local metabolites or of local sodium stores may be one factor in the exaggerated neurologic signs which may follow convulsions. Sargant and Slater¹⁰ stated that generalized metabolic deficiencies may become manifest during ECT, and Walsh¹¹ similarly suggested that when a latent vitamin deficiency is present, a convulsion can be permanently destructive to brain function.

Ottosson and Rendahl¹² stated that a seizure also increases the cerebrospinal fluid pressure and produces marked changes in vascular permeability within the cranium. It seems likely that cerebral edema, which is probably responsible for the worsening of patients with organic brain disease following ECT, is itself precipitated by changes in vascular permeability.

Summary and Conclusions

There are at least three general ways by which ECT can produce a sudden neurologic change in patients with organic brain dis-

ease. Tenuous local metabolism or nutrition may be disturbed by the increased demands at the time of a seizure, severe functional neurophysiologic impairment may occur, or cerebral edema may be produced in an area of diffuse or focal disease. The factor of edema is considered most significant in the type of cases reported here.

The six cases reported here document facts already apparent to most physicians. It is hoped that they serve to re-emphasize the need for caution in the use of electroconvulsive treatment when organic brain disease is suspected. At a time when a high percentage of patients in public or private psychiatric hospitals have organic brain disease, electric shock must be used only when the diagnosis is reasonably clear and the indications are unequivocal.

Furthermore, if a patient who has received ECT fails to improve, or if his post-shock confusion is unduly prolonged, a brain tumor or other underlying organic disease must be suspected. The patient should receive a careful medical, psychiatric, and neurologic examination whenever the response to ECT is atypical.

There are certain situations in which ECT is legitimately used to confirm a diagnosis of functional disease. For example, intractable pain of the lower half of the face or bizarre headache will at times respond dramatically to ECT. When administered ju-

diciously, ECT can serve as a confirmatory diagnostic tool in psychiatry. Unfortunately, this procedure can also conspicuously expose or exaggerate organic brain disease.

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Weak nerves are the constant companions of inactivity. Nothing but exercise and open air can brace and strengthen the nerves, or prevent the endless train of diseases which proceed from a relaxed state of these organs. We seldom hear the active or labourious complain of nervous diseases; these are reserved for the sons of ease and affluence. Many have been completely cured of these disorders by being reduced from a state of opulence, to labour for their daily bread. This plainly points out the sources from whence nervous diseases flow, and the means by which they can be prevented.—William Buchan: *Domestic Medicine, or a Treatise on the Prevention and Cure of Diseases by Regimen and Simple Medicines, etc.*, Philadelphia, Richard Folwell, 1799, p 73.