Brain Damage

PROFOUND REGRESSION FOLLOWING TWO ELECTROCONVULSIVE TREATMENTS*

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Relatively little is written in the literature concerning cases of profound, rapid regression after one or two electroconvulsive treatments have been administered. It has been suggested that such regression may be diagnostic of underlying central nervous system pathology. M. M. Gassel (2) reported three cases from the records of 250 consecutive confirmed intracranial meningiomas admitted to the National Hospital for Nervous Diseases, Queen's Square, London England. Electroconvulsive therapy was administered before the correct diagnosis of the space occupying lesion was made. "In each case, it was followed by deterioration in the patient's condition and the appearance of abnormal physical signs." Gassel suggests that there was a "drawn presumption that the deterioration had been accelerated by the electroconvulsive therapy."

Savitsky and Karliner (3) reported stupor in a patient found eventually to have a right temporoparietal glioblastoma after the second electroconvulsive treatment had been administered. The exact mechanism of this deterioration following electroconvulsive therapy is difficult to trace. Gassel suggests that our understanding of "intracranial events in convulsions leaves much to be desired." It is certain that considerable alteration in cellular permeability, vascular tone, blood pressure, blood flow and cerebral spinal fluid pressure are occasioned by both the electrical current as well as the induced convulsion, as suggested by T. C. Fleming (1).

Material is here presented to illustrate the phenomenon of rapid regression following a very abbreviated course of two electroconvulsive treatments.

Case History: A. B. was a 58-year-old Caucasian female who was admitted for the third time to the Neuropsychiatric Institute of the University of Michigan Medical Centre, Ann Arbor, Michigan on September 20, 1963. She presented with complaints of extreme nervousness, agitation, crying spells, and a general lack of interest in her housework. In addition, she had been on glutethimide‡ medication, with a total daily dose of 2 grams.

The patient had first been admitted to the Neuropsychiatric Institute on January 11, 1957, and discharged on April 18, 1957. She was then readmitted on May 4, 1957 and discharged on July 27, 1957. Between July, 1957 and July, 1963, she was not seen at the Neuropsychiatric Institute. On July 8, 1963 her out-patient re-evaluation was precipitated by her excessive ingestion of glutethimide.

Her original symptomatology noted in 1957 was primarily that of depression and agitation. Because of organic heart disease with aortic stenosis and insufficiency as well as possible old posterior myocardial infarction, electroconvulsive therapy was not immediately instituted. She was maintained for several months on phenothiazine with doses up to 1200 milligrams per day of promazine‡‡. However, due to minimal improvement after two months of hospitalization, it was felt that her agitated, depressed state was more of a threat to her impaired cardiac status than the electroconvulsive treatment. Accordingly, five electroconvulsive treatments were administered under suxamethonium‡‡‡, with a profound remission of symptoms. She was asymptomatic at the time of discharge. However, after an interval of three weeks, she began experiencing some somatic complaints of palpitation of the heart and cramping in her abdomen. She then lapsed into prolonged episodes of general agitation and tearfulness. The patient was readmitted to the Neuropsychiatric Institute with the hope that milieu therapy would prove efficacious. Transiently she improved. However, with deterioration of her condition, the patient received nine electroconvulsive treatments with suxamethonium between June 26 and July 15, 1957. There was a considerable amount of 'organicity' noted following electroshock as manifested by memory loss and confusion. She

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‡Doriden.
‡‡Sparine.
‡‡‡Anectine.

apparently did reasonably well between 1957 and 1962.

She was hospitalized in her community hospital in 1962 following a fall and at that time had two *grand mal* seizures in the hospital which were allegedly due to drug withdrawal. She was then seen in the Neuropsychiatric Institute Out-patient Department on July 8, 1963. It was thought that she was on excessive doses of both glutethimide and perphenazine* which resulted in her having some difficulty walking. She also manifested a degree of confusion. The examiner felt that there was evidence of an "organic brain disorder and I am unable to judge whether this is due primarily to her medication or whether there is a large component of beginning arteriosclerotic organic brain deterioration." She was subsequently seen in the Out-patient Department on September 20, 1963. It was felt that she had been unable to reduce her glutethimide intake below 2 grams per day. The mental status examination at that time revealed a rather extreme degree of free floating anxiety. There was no clouding of the sensorium or confusion as noted in the July examination. However, it was deemed impossible to wean her from the glutethimide on an out-patient basis. Because of a poor family living situation where the patient and her invalid husband lived in an isolated circumstance, it was thought that hospitalization was indicated.

After hospitalization, a program of decreasing glutethimide was carried out in an orderly manner without untoward effect. She was begun initially on 500 milligrams of glutethimide, q.i.d. This was lowered to 500 milligrams, b.i.d., 250 milligrams, b.i.d., and then tapered to discontinuation. Perphenazine was decreased from 8 milligrams, t.i.d., to 4 milligrams, t.i.d. The neurological examination during the initial evaluation period was within normal limits. The remainder of the physical examination was unrevealing except for findings in the cardiovascular system and extremities. In the patient's history she was thought to have had rheumatic heart disease since age eight years. She denied shortness of breath but did complain of some angina pectoris for 10 years, with pain radiating down the left arm and responding to nitroglycerin after several minutes. She did manifest 1+ ankle swelling bilaterally. In addition, she had a grade 3 to 4 systolic murmur in the aortic area and a grade 1 diastolic murmur. A-2 was greater than P-2. There was a normal sinus rhythm with a few extra systoles. Blood pressure was 148/80; pulse 70. The internal medicine consultant felt that a low salt and cholesterol diet, in addition to acetazolamide**, 500 milligrams daily, was all that was needed to maintain her cardiac status. Electrocardiogram demonstrated changes compatible with left ventricular hypertrophy with myocardial changes suggestive of old posterior infarction. There had been no significant change from the earlier record in 1957. Chest X-ray demonstrated an enlargement of the heart, due most likely to left ventricular hypertrophy. Electroencephalogram was read as, "There are some minor border-line signs in terms of non-bursting, bi-central theta slowing but the record is not outside of normal limits." Laboratory examination, including alkaline phosphatase, SGP transaminase, and CBC were within normal limits. Urinalysis and stool examinations were unremarkable. Kahn serological reaction was negative.

Her behaviour on the ward following reduction of the glutethimide and perphenazine medication was not grossly dissimilar to the picture seen in 1957. She was extremely agitated, complaining that she was unable to participate in any activities. However, she did respond to firm encouragement. She was rather insistent in her demands for "strong medication to help me." The patient had her first electroconvulsive treatment on October 23, 1963, 120 volts for one-half second with a Model B-24 Medcraft unit. One hour prior to the treatment, she had received 0.4 milligrams of atropine, subcutaneously. Methohexital*** 1%, 170 milligrams, and suxamethonium 45 milligrams, were administered intravenously at the time of the treatment. The patient tolerated the procedure fairly well but became more agitated subsequent to the treatment. She stated that she was 'naked' and exposed herself by raising her dress. The patient was noted to be much less directable, began lying on the floor, and falling immediately upon being helped up. She ate poorly, complained that she could not move and that she was going to die. On October 25, the patient had her second modified electroconvulsive treatment with essentially the same medication regimen. She slept for about two to three hours after this treatment. Subsequently, she was described as being markedly improved, pleasant, conversive with the staff and her family. In a matter of hours, however, she was once again very agitated and angry with the staff. She was obsessed with her 'nudeness'. She steadfastly refused her next scheduled electroconvulsive treatment. She was grossly psychotic with somatic delusions in evidence.

Because of the rapid regression following the two electroconvulsive treatments, an organic component was suspected. Physical examina-

*Trilafon.

**Diural.

***Brevital.

tion was once again unrevealing. She refused to co-operate for a repeat electroencephalogram. Her regression on the ward continued. Initially, she did accept spoon feeding. Ultimately this was refused and a feeding tube was passed and maintained for a week. Her intake improved to the point where the naso-gastric tube could be removed. Urinary and fecal incontinence were progressively in evidence. Following the regressive episode post-EST on October 23, 1963, chlorpromazine† was instituted at a dosage of 50 milligrams, q.i.d. Chloral hydrate, 500 milligrams, was used as a bedtime sedative. On December 3, 1963, the chlorpromazine was increased to 75 milligrams, q.i.d. She was examined by the neurological consultant on the aforementioned date without clinical neurological findings.

On December 5, 1963, a naso-gastric feeding tube was reinserted. Six days later, on December 11, 1963, with the feeding tube in place, the patient was noted to have obvious respiratory distress. She was slightly cyanotic, perspiring, and the pulse was rapid. The tube was immediately removed, airway inserted, and she was suctioned. The impression was that the patient had aspirated and following suctioning, her colour improved markedly. A portable chest roentgenogram was read as normal. A repeat film the next day, on December 13, 1963, likewise showed "no gross pulmonary abnormality." Chlorpromazine was reduced to 25 milligrams b.i.d. and imipramine†† was begun on December 19, 1963. The chlorpromazine was discontinued on December 26, 1963, and the patient was maintained thereafter on 100 milligrams of imipramine daily. During the months of November and December, she was bedridden and efforts were instituted to prevent decubitus ulcers.

The patient was transferred to a state hospital on December 31, 1963. She expired on January 4, 1964, quite suddenly. The clinical diagnosis was one of a myocardial infarction, coronary arteriosclerosis.

†Thorazine.
††Tofranil.

The necropsy report revealed the following pathological diagnoses: "Recent pulmonary arterial emboli. Old myocardial infarct in the posterior wall and septum of the left ventricle. Old rheumatic valvulitis of the aortic and mitral valves. Congestion of the lungs and spleen. Fatty infiltration of the right ventricle and liver. Diffuse colloid goitre. Generalized arteriosclerosis. Ectopic glia in the leptomeninges adjacent to the medulla. Adenofibroma of the breast." Special staining technique of the central nervous system was unrevealing in regard to atypical cellular or matrix formation.

Comment: The aforementioned is a report of a case of profound regression subsequent to two electroconvulsive treatments. In the literature, the consensus seems to suggest central nervous system pathology as being the most frequent concomitant of such convulsive therapy regression. Careful pre-mortem clinical examination and post-mortem gross and microscopic examination fail to support this conclusion in this particular case.

References

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