

DEATH FROM ELECTRICAL CONVULSION THERAPY.

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TEN years have elapsed since the introduction by Meduna in 1934 of convulsion therapy, and five since Cerletti and Bini first described the use of electricity as a convulsant. A not unreasonable early prejudice finds a later echo in the comment of Critchley (1943) that convulsion therapy "is crude, dangerous and repellent to anyone who holds the central nervous system in respect."

Nevertheless, the experience of the decade has established that convulsion therapy comes near to providing a specific treatment for the affective psychoses. Batt (1943) quotes a recovery-rate of 87 per cent. from a series of a hundred depressives of different types; Fitzgerald (1943) reports 78 per cent. recovered out of 150 patients suffering from depressive states. It is a remarkable advance that a type of case in which the outlook was formerly so problematical can now be offered with some confidence the prospect of restoration in a matter of weeks.

Successive observers have described the advantages of electrical convulsion therapy (E.C.T.) over other forms of induced convulsion. Greater freedom from complications, of which the chief are fractures and dislocations, and a lower mortality are among the conclusions of the recent comprehensive survey by Cook (1944). Kolb and Vogel (1942), who are responsible for the most considerable American analysis, record 4 deaths in 7,207 electrically treated cases, a rate of 0.05 per cent., which compares with the figure of 0.1 per cent. for cardiazol cases.

No similar series has appeared from English hospitals, and as the deaths that have occurred have been so few, no individual experience can for a long time be large enough to give a valid rate, nor do the published figures of treated cases amount as yet to a total in any way comparable with that of Kolb and Vogel.

As a step therefore towards assessing the position for this country, it was thought to be of value to record the deaths that have already occurred. Only one of these (Batt, 1943) has been published, and an otherwise fairly voluminous literature is thus short of one important particular.

E.C.T. is carried out in public and private mental hospitals, in out-patient departments and in service establishments. Cases from the first group will form by far the largest number for obvious reasons, and any deaths in this group are automatically reported to the Board of Control, who were good enough to inform me of four of these. The cases here recorded therefore would appear to be the only known civilian deaths that have as yet resulted from E.C.T. in England and Wales. Two have been personally observed. Two more have been most kindly communicated by the medical superintendents concerned. The fifth has been published. A sixth was reported to the Board of Control, but is to form part of a later compilation and no details are available.

CASES.

(1) A. C. H.—, male, aged 46, admitted 23.ix.41. Complaining of depression, insomnia and widespread pains.

History.—"Shellshock and gas" in last war. "Graves' disease" 1920. Unemployed for last 21 years.

On examination.—Poor physique, pallid and with a number of physical complaints, none of which were based on demonstrable physical disease. No exophthalmos, no thyroid enlargement, no signs of thyrotoxicosis.

Course.—No response to simple psychotherapy and no spontaneous improvement at six months.

E.C.T.—31.iii.42, 11.30 a.m.: 150 volts, 0.2 second. Satisfactory convulsion without unusual features. Normal recovery over subsequent half hour. Walked back to his own ward for midday meal, only complaining of slight malaise, as is not infrequent in the immediate post-convulsive phase. At about 4.30 p.m. he was noticed to be rather restless, but he made no further complaint, nor were his manner and appearance in any way altered. At 5.30 p.m. he complained of dyspnoea, and died in his chair at about 5.40 p.m. There was then seen to be some fullness of the lower neck anteriorly, and this area later showed a subcutaneous deep shadow.

Post-mortem, 1.iv.42.—Brain and cord showed no abnormality beyond a cystic choroid plexus. On opening the neck and thorax an extensive spreading haemorrhage occupied the region of both thyroid lobes, reaching posteriorly and across the isthmus; further spread downwards into mediastinum, surrounding trachea, oesophagus and great vessels. Right lobe of thyroid showed some recognizable thyroid tissue, much disorganized; left lobe entirely disorganized by blood-clot macroscopically. Retrosternal mass of clot contained some glandular tissue, much disorganized and unrecognizable by naked eye. Epiglottis, arytaenoids and larynx showed much jelly-like oedema, with injection of trachea but no froth or vomitus. No petechiae on epicardium or liver. Heart showed increase of subepicardial fat, but otherwise normal. No other relevant findings.

Sections of thyroid lobes: Normal thyroid tissue, without alveolar proliferation or colloid excess; widespread haemorrhages. The retrosternal mass consisted microscopically of clearly recognizable thymus and blood-clot.

It was not possible to identify the source of the bleeding, though it can hardly have been other than in the thyroid venous plexus. So also it remains conjecture that the onset of the bleeding was in the congestive phase of the convulsion, though no other explanation is tenable. In the absence of signs of asphyxia, death would appear to have been caused by mediastinal shock before the closure of the glottis by oedema could occur.

(2) M. A. D.—, housewife, married, aged 52, admitted 11.xii.40. Involutional depression.

History.—Agitated depression of four years' standing.

On examination.—Small, poorly nourished woman; no important physical findings. Deeply depressed, agitated, self-absorbed and almost entirely inaccessible.

Course.—No spontaneous improvement in further two years—a total of six years in hospital. Not confined to bed.

E.C.T.—15.iv.42: 120 volts, 0.15 second. Satisfactory convulsion with no unusual features. Recovery normal. Afterwards access was very slightly improved and she answered questions in a whispered voice.

17.iv.42: 120 volts, 0.15 second. No convulsion. Short interval and 150 volts, 0.2 second. Satisfactory convulsion with no unusual features. Recovered normally, but then complained of inability to walk and pain in the right hip. X-ray showed fracture of floor of right acetabulum, with medial displacement of an elongated fragment. Femoral neck intact. Treated by extension in abduction. Mentally she showed further improvement and was more accessible, though still deeply depressed. Subsequently her condition deteriorated and she developed signs of pulmonary tuberculosis. She died on 21.x.42, six months after *E.C.T.* and the injury.

Post-mortem, 22.x.42.—Confluent tuberculous foci in both lungs, most pronounced in the left upper lobe. Fracture of right acetabulum; the head of the femur could be palpated from inside the true pelvis.

Cause of death: Pulmonary tuberculosis; fracture of pelvis.

(3) B. C. M. H.—, male, aged 62, admitted 26.x.42. Anxious hypochondriacal depression. Ideas of suicide, of venereal disease, of punishment for his venereal disease.

History.—First attack of depression.

On examination.—Peripheral arteries soft. B.P. 185/90. Slight enlargement of the left ventricle; the heart-sounds of poor quality. No organic neurological signs. Blood W.R. negative. Urine 1010, acid, haze of albumen, pus-cells. 23.xi.42: Urine clear.

E.C.T.—11.i.43: Potentiometer 65, 0.25 second. Patient's resistance 4,000. No fit. Four further doses of electricity were given up to and including 21.i.43, with potentiometer at 95, time 0.35 second. Patient's resistance always high (3,000–5,000 ohms). No convulsion.

25.i.43: Potentiometer 95, 0.4 second, 5,000 ohms. A strong convulsion, the first and last, at 11.15 a.m., 5 seconds after the passage of the current. Condition during and after the fit not abnormal. He regained consciousness at 11.45 a.m. "Half an hour after the fit he displayed some excitement and restlessness, sat up in bed and collapsed; he became grey in colour, his pulse was imperceptible, his breathing stopped; the pupils were moderately dilated and did not react to light. Artificial respiration was started at once, heat was applied, the limbs were massaged and bandaged. He was given 1.0 c.c. of coramine, followed by 1.0 c.c. of adrenalin, and finally strophanthin into the heart. For a few seconds after this the pulse could be felt at the wrist. Artificial respiration was continued for 1½ hours without effect.

Post-mortem.—Pia generally thickened and oedematous, haemorrhagic staining in the frontal region and, on the right side only, over the parietal and occipital regions. Basal arteries healthy except for some thickening of the right internal carotid and a patch of sclerosis at the lower end of the basilar. Apart from a degree of cortical atrophy there were no other notable changes.

"Heart: Somewhat globular in shape. Marked relative preponderance of the left ventricle.

All chambers empty. Valves healthy on the whole, though the mitral and aortic were more opaque than usual. Heart muscle was extremely soft, the thumb penetrating it easily.

"Aorta: Almost free from atheromatous changes. Pericardium showed a broad band-like adhesion about an inch long and nearly as wide between the posterior surface of the heart and the parietal pericardium.

"The remaining organs, including kidneys and bladder, showed no prominent abnormality."

Cause of death: Syncope due to myocardial degeneration.

(4) E. S. C. M. H—, female, aged 74.

History.—Manic-depressive illness of long standing.

E.C.T.—Following the third treatment with E.C.T. she was found to have a fracture of the neck of the right femur. Subsequent satisfactory progress, but on 31.xii.43 she showed signs of uraemia—blood urea 102 mgm. per cent.—and pulmonary congestion, and she died on 1.i.44.

Certified cause of death: (1) (a) Uraemia; (b) chronic nephritis. (2) Bronchopneumonia, cardiovascular degeneration, fracture of neck of right femur.

(5) (Quoted from Batt, 1943.) Female, aged 54. Manic-depressive. Six days after the second E.C.T. convulsion she developed bronchopneumonia and died four days later. Post-mortem showed bronchopneumonia and chronic nephritis.

COMMENTARY.

Only in two of these cases is death directly referable to the convulsion, and of these Case 1 belongs to the group of rare and unpredictable vascular catastrophes. Haemorrhage into the thyroid is itself uncommon, and a fatal thyroid apoplexy exceedingly rare. Ryan (1942) quotes two comparable cases. In one a spontaneous haemorrhage ruptured through the capsule and infiltrated the fascial and muscle planes of the neck, spreading into the mediastinum as in the present case. The second recalls the mode of onset of Case 1. A man, aged 55, straining at defaecation, had sudden difficulty in swallowing, became dyspnoeic, unconscious, and died. A similar uncharted hazard is offered by the cerebral aneurysm.

Case 3, on the other hand, represents a group of cases where physical signs point definitely to an incalculable risk which it is only possible to cover by an attempt to induce the mildest type of convulsion. Where the threshold is normally high this is hardly possible. Nevertheless it is certain from personal experience and from many accounts in the literature that, though such fatalities could be reduced even further, it would be at the cost of excluding a number of poor risks who would otherwise benefit. Undoubtedly a policy of ruthless selection could achieve a very high rate of cure with a very low fatality- and complication-rate. Such a policy would ignore the constant risk to life from intercurrent infection or suicide, to which a chronic depression predisposes, as also the fruitlessness of a life maintained on such terms.

Cases 2 and 4 have much in common, though Case 2 also illustrates the possibility which has already been well established of lighting up old tuberculous foci by convulsion therapy of all forms. The diametrically opposed views of those who restrain and of those who avoid restraint show how little as yet the risk of fracture can be guarded against, and the occurrence in these two cases after the successful negotiation of one and two previous convulsions illustrates the element of chance in the development of a breaking strain.

Case 5 is admitted by the author to show no more than a temporal association, but he points out that if no proof of a relation can be adduced, there is none to exclude it.

SUMMARY.

1. A record is given of six cases known to have died following electrical convulsion therapy.
2. Of these deaths, two arose directly out of the convulsion, three were associated deaths, and in one case the relation is not known.
3. These are believed to be the only civilian deaths from E.C.T. which have been met with in England and Wales.

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