DEATH AFTER E.C.T.

DEAR SIR,

It is worthy of record to note that a heavily-built Jamaican woman, aged 33 years, died after E.C.T. treatment for agitated melancholia. She had been terrified that the devil was after her, and she was full of guilt.

She had previously responded to twenty treatments given in July/August, 1966. Leave home was allowed in August, but she returned to hospital after two days, when sixteen E.C.T. treatments were given. Leave was again granted in September, and patient was discharged October, 1966, having had a total of 36 E.C.T.

A relapse in February, 1967, was treated with six E.C.T.; four and a half hours after the last treatment she was found dead.

The treatment had been given about 10.15 a.m.; there had been slight delay in resumption of respiration, and an airway had been inserted and oxygen applied by manual pressure on the rubber bag. Two hours later she asked for a drink and was given a beaker of water. She was seen to be asleep, breathing normally, at 3.30 p.m.; at 4.40 p.m. she was found to be dead.

At the inquest the verdict was “misadventure”, and the cause of death was returned as “asphyxia which resulted from the patient turning on to her back following the electro-therapy and her tongue obstructing the air passage”.

I believe this is the first record of death following E.C.T. in this manner.

The only medication had been an intramuscular injection of Largactil four days previously. Her blood pressure in June, 1966, had been 160/90. There was no evidence of her having had any spontaneous fit.

At post-mortem, the brain was congested, otherwise normal. All organs of the body were healthy but congested. There was no evidence of coronary disease or pulmonary embolism. The lungs were dotted with petechiae, but not the conjunctivae. Stomach contained a little clear fluid, bowels little contents.

The body was thick-set and short-necked. It was estimated at being 11 stone and she was 5 ft. 4 in. in height.

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HYDROXOCORTICOSTEROIDS IN DEPRESSIVE STATES

DEAR SIR,

In their recent paper Hullin et al. (1967) imply that the reduced tolerance to glucose which is often found in depressive disorders may be caused by increased secretion of glucocorticoids. However the main finding in the paper by me which they quote (Pryce, 1964) was the absence of any relationship between glucose utilization and 17-hydroxycorticosteroid excretion in depression. This is not surprising, since as they themselves comment, the variations in plasma glucocorticoids in depression are small; moreover glucocorticoids are only weak hyperglycaemic agents, and in depression more potent agents associated with carbohydrate deficiency and age are not infrequently present.

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REFERENCES


REHABILITATION OF THE YOUNG SCHIZOPHRENIC PATIENT

DEAR SIR,

Dr. Durrant’s article in the Journal for June, 1967, is important. Those who are interested in the subject may wish to refer to previous work. I therefore append a short list of references.

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