DEATHS ASSOCIATED WITH ELECTROPLEXY

By

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Introduction and Problem

A RECENT court case (16) exposed many of the problems that confront the psychiatrist called upon to perform electroplexy.

In particular it revealed that in 1957, nearly 20 years after E.C.T. had first been used for the treatment of mental disorder, no uniform technique had yet been evolved. The medical witnesses were unable to agree on the use of muscle relaxants or mechanical restraint, but were agreed that there were certain risks attendant upon the use of electroplexy, especially the risk of fractures and the risk to life. It was argued that had the plaintiff received a relaxant drug he might not have sustained bilateral acetabular fractures, but might have suffered an even worse disaster, namely death.

As a result of this controversy, it seemed important to enquire into the death rate associated with electroplexy, and to examine the causes of death, noting especially any possible relationship between death and particular E.C.T. techniques.

(a) *The Death Rate from Electroplexy.* This has been variously estimated by different American authorities, and the results are shown in Table I.

TABLE I

	Number of Deaths	Patient Fatality Rate	Number of Patients Treated	Treatment Fatality Rate
		Per cent.		
Kolb and Vogel (1942) ..	4	0·05	7,207	—
Impastato and Alamansi (1943)	8	0·8	11,000	—
Alexander *et al.* (1956) ..	5	0·1	5,325	1 death in 14,000 treatments
Gaitz *et al.* (1956)	3	0·4	700	1 death in 2,600 treatments

It will be appreciated that no complete data exist regarding the total number of treatments carried out during any particular period of time, so that at the best the above figures relating to patient and treatment mortality rates can only be rough approximations. Oatman (14) stressed that the actual mortality rate may be very different from that suggested by the above figures, because of sampling errors, and because *fatalities* are not always reported. Alexander *et al.* (1) were, however, to some extent aware of this discrepancy, and suggested that the risk of death lies between 1 per 3,000 patients and 1 per 300 patients.

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Impastato (8) pointed out that most of the statistics make no allowance for the death rates of patients of different ages. He demonstrated that in 1,000 unselected patients, the most frequent age group receiving E.C.T. was between 30–40, and that the majority of 153 previously reported E.C.T. fatalities had occurred in the age group 50–60. He estimated that the death rate is approximately 0·5 per cent. per patient, in patients over 60, and decreases to 0·033 per cent.–0·025 per cent. in patients between 20 and 30 years of age, but that in "poor risk" patients both these death rates should probably *be doubled.*

(b) *The causes of death* in association with E.C.T. have been tabulated by Impastato (8) based upon a study of 214 previously reported cases together with 40 other cases. He also gives a full bibliography, making further review of the literature unnecessary.

Deaths can be classified into the following four groups:

1. Cardiovascular deaths
2. Cerebral deaths
3. Respiratory deaths
4. Other causes of death including some of the rarities, endocrine disease (Graves' disease, adrenal haemorrhage, etc.), exhaustion, genito-urinary causes (lower nephron nephrosis, uraemia), gastro-intestinal causes (peritonitis, strangulated hernia, rupture of the liver, etc.), leukaemia, death from pre-medication before E.C.T., and cause unknown.

(c) *Possible Relationship of Fatality Rate to E.C.T. Technique.* In Maclay's (13) series, 28 out of 62 E.C.T. deaths had received a relaxant and other pre-medication, but he stressed that the variety of relaxants and other drugs used made it impossible to ascertain whether any combination was more dangerous than another. Nevertheless he was of the opinion that relaxants might not be an "unmixed blessing" and should be used cautiously in cases with cardiac complications. Kalinowsky and Hoch (11) also believed that the risk was greater with modified than unmodified E.C.T., and it has been stressed that muscle relaxants themselves carry a risk which will be increased when they are followed by E.C.T.

The Questionnaires

In order to ascertain which E.C.T. techniques were in current practice, the first questionnaire was sent to 13 teaching hospitals in London and 42 mental hospitals in June, 1957. The results have been fully reported elsewhere (3) but are briefly summarized below. Questionnaire II was sent in March, 1958, to the same hospitals that were contacted by Questionnaire I. There was nearly a full response to this second questionnaire, which suggests that the subject matter aroused considerable interest.

The following information was requested:

1. A rough estimate of the total number of E.C.T. treatments carried out in the hospital (and associated out-patients' departments) during the years 1956 and 1957. This referred to the number of individual treatments and not courses of E.C.T.

2. The number of deaths that were considered to have been associated with E.C.T. during this period. For the purposes of this investigation only those deaths that had occurred within three days of the last treatment were acceptable, for in cases where this period is longer, it becomes increasingly difficult to incriminate E.C.T. as the direct cause of death.

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If there had been any deaths, the hospital was requested to supply full details under the following headings:

Name
Hospital Number
Age
Sex
Psychiatric Diagnosis
Physical Diagnosis
Number of Treatments before Death
Interval between last Treatment and Death
Technique used
Cause of Death
Post-mortem Findings
Any other Remarks

Information about the total number of patients treated was not requested. It was felt that the treatment fatality rate would be a more useful index of the overall position than the fatality rate per patient. It is well known that some patients may die after the first treatment, and that others may have several hundred E.C.T.s before death.

Results

Replies to Questionnaire I showed that no uniform technique of E.C.T. administration was practised by psychiatrists in this country. A survey of the previous literature also revealed no unanimity of opinion.

Briefly, E.C.T. was given modified with thiopentone and Scoline at 60 per cent. of hospitals, and muscle relaxants were used at 85 per cent. Anaesthetists were present for E.C.T. sessions at rather less than 50 per cent. of mental hospitals; at the others treatment was usually given single-handed by the psychiatric registrar, even though thiopentone and muscle relaxants were being used.

Replies to Questionnaire II gave the following results:

1. The number of E.C.T. treatments per annum carried out at a large (2,000 bedded) mental hospital and supporting out-patient clinics ranges from 400 to 8,000.
2. At teaching hospitals in London, the number of E.C.T.s given to in-patients and out-patients ranges from 100 to 2,000 per annum.
3. The total number of treatments carried out during the two-year period at 42 hospitals replying to item I of the circular was approximately 259,000.
4. The number of deaths associated with E.C.T. from all the replies received during this period was 9 (occurring within 3 days).
5. Therefore the incidence of death due to E.C.T. is approximately 1 death in 28,000 treatments, giving a treatment fatality rate of ·0036 per cent.

It is emphasized that the treatment fatality rate may be even lower than this, because some hospitals that failed to provide data concerning the total number of treatments carried out, were able to state that no deaths had occurred, hence the number of deaths is drawn from a slightly larger number of hospitals than those who supplied information about the amount of E.C.T. given.

The fatality rate from E.C.T. taken from over a quarter of a million treatments is very low, far lower than has been recorded by previous authors drawing upon smaller samples.

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Discussion on the Deaths Reported in this Series

Brief descriptions of patients who died following electroplexy are given at the end of this article. Emphasis has been placed upon the circumstances of death together with other relevant findings. The salient points are summarized in Table II.

(a) *Age.* The average age of the series was 67 years. Eight of the patients were aged 60 and over. This indicates that age is an important consideration and must be carefully associated with any figures relating to the E.C.T. fatality rate. This point has been emphasized by Impastato (8) and Eyres (6). Ehrenburg and Gullingsrud (5) reported four deaths in 112 patients aged between 65 and 83 years, though other authorities have reported that E.C.T. can safely be given to elderly patients, and that the incidence of fractures following E.C.T. in feeble senile patients is less than would have occurred during a corresponding period of time from other causes.

(b) *Previous Physical Health.* In only three cases was there no evidence of pre-existing physical diseases. Case I had suffered from a previous coronary thrombosis, and had mild hypertension, dying of left ventricular failure. Case 2 had hypertension, arterio-sclerosis and auricular fibrillation, and gave a history of a recent cerebral thrombosis, yet died of cardiac causes. Case 4 was obese with hypertension and sustained a coronary thrombosis after E.C.T. Case 5 gave a history of cerebral thrombosis 6 months previously and died after a cerebral haemorrhage. Case 6 had suffered for one year with progressive muscular atrophy. Case 7 was obese with hypertension and chronic bronchitis, and died of acute heart failure. These patients all had known physical diseases but had withstood several treatments before death. Muscle relaxants would seem inappropriate in Case 6, who had progressive muscular atrophy. Pre-existing extensive cardiovascular disease could have been clearly diagnosed in many of these cases before they received E.C.T., though only two of the series were considered "poor risk" patients.

(c) *Number of Treatments before Death.* Case 5 died after the first treatment of a second course. Cases 6 and 8 died after "enormous quantities" of E.C.T. had been given. These findings are at variance with those of Maclay (13) who reported that half his series of patients died after the first convulsion.

(d) *The Interval between the last Treatment and Death.* As might have been expected there seemed to be a close association between the mode of death, and the interval that followed the last treatment. Those patients who died of cardiac failure or "shock following E.C.T." either did not recover or died within two hours of the treatment, whereas death from coronary thrombosis or cerebral haemorrhage took place between 30 hours and two days following the treatment.

(e) *Tranquillizers.* Half of the patients in this series were receiving tranquillizing drugs, usually chlorpromazine, at the time of death. It is interesting that all these patients died suddenly of shock or cardiac failure following E.C.T. Weiss (15) reported a patient who suffered a disturbing vasomotor collapse after treatment with E.C.T. while receiving chlorpromazine (450 mg. daily). A marked fall in blood pressure occurred immediately following the treatment, which was not present after subsequent treatments when chlorpromazine was omitted. Weiss postulated that the hypotensive action of chlorpromazine was in this case intensified by E.C.T. Kalinowsky (10) referred to an increase in fatalities among patients undergoing treatment with reserpine and chlorpromazine during E.C.T., and recommended suspending administration of

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TABLE II

Case No.	Initials	Age	Sex	Psychiatric Diagnosis	Physical Diagnosis	No. of Treatments Before Death	Interval	Record of Tranquillizers	Technique	Cause of Death
1	E.S.W.	76	F	Senile Psychosis	Hypertension Previous Coronary Thrombosis	4	2 hours	—	Thiopentone and Scoline	Left ventricular failure secondary to hypertension.
2	F.W.	70	M	Agitated Depression	Residual Hemiplegia Arterio-sclerosis	3	Minutes	Chlorpromazine 50 mg. t.d.s.	Scoline	Hypertensive cardiac failure accelerated by E.C.T.
3	E.S.	74	M	Depression	—	6	Did not recover	—	Thiopentone Brevidil E.	Myocardial syncope. Coronary artery disease. Emphysema.
4	E.A.L.	73	F	Senile Depression	Obesity Hypertension	3	30 hours	—	Thiopentone and Scoline	Coronary thrombosis.
5	H.A.F.	60	M	Involutional Depression	Previous Cerebral Thrombosis	9 1st of 2nd Course	30 hours	—	Thiopentone and Scoline	Cerebral haemorrhage. Purulent broncho-pneumonia.
6	E.C.	62	F	Recurrent Depression	Progressive Muscular Atrophy	Very many	2 days	—	Thiopentone and Scoline	Coronary thrombosis. Progressive muscular atrophy.
7	H.E.B.	60	M	Recurrent Depression	Obesity Hypertension Chronic Bronchitis	5	20 minutes	Chlorpromazine 50 mg. t.d.s.	Thiopentone and Scoline	Acute cardiac failure due to E.C.T. Fatty infiltration of myocardium.
8	M.E.H.	50	F	Catatonic Schizophrenia	—	332	A few minutes	Chlorpromazine 100 mg. t.d.s.	Unmodified	Death due to shock following E.C.T.
9	M.M.	67	F	Schizophrenia	—	5	Did not recover	Chlorpromazine 100 mg. t.d.s.	Unmodified	Cardiac failure. Aortic stenosis.

these drugs for a few days before commencing treatment. Ayd (2) however, took the opposite view, after treating 75 patients without incident suffering from anxiety states with combined chlorpromazine-E.C.T., and regarded this drug as ideal for combination with electroplexy, maintaining that chlorpromazine, acting as a sympathetic depressant, counteracts the excessive sympathetic stimulation that so often accompanies E.C.T. It is possible that the presence of chlorpromazine in the tissues may potentiate the respiratory depression encountered in some patients receiving thiopentone and muscle relaxants, and that unmodified E.C.T. might be a safer procedure in patients receiving this drug.

(f) *E.C.T. Techniques.* It will be observed that only 2 out of the 9 patients who died had received unmodified E.C.T. The majority had received thiopentone and scoline, though Case 2 received scoline alone, and in Case 3, E.C.T. had been modified with thiopentone and suxethonium (Brevidil E). These findings do not in themselves indicate a higher death rate in persons receiving modified E.C.T., but merely reflect the present popularity of modifying techniques. (See results of Questionnaire I.) It is probably a common experience among those who give electroplexy in any quantity to encounter patients who have well tolerated large numbers of unmodified treatments, but whose condition has given rise to alarm following the first E.C.T. modified with a muscle relaxant with or without an anaesthetic. Maclay (13) reported a number of instances of death following the first modified treatment.

Some of these patients may exhibit hypersensitivity to succinylcholine and develop respiratory paralysis, which has been reported on a number of occasions, and recently suxethonium (Brevidil E) has been shown to be not immune from this complication (4). A low serum pseudo-cholinesterase is often found to accompany respiratory difficulties in these patients.

The dangerous properties of muscle relaxants have been stressed by other authors, notably Impastato (8) who reported 5 patients who died following premedication with muscle relaxants *before* they had received E.C.T., and suggested that many of the fatalities attributed to E.C.T. are in fact due to the relaxants.

(g) *Cause of Death and Autopsy Findings.* In the present series there were 2 deaths from coronary thrombosis and one from cerebral haemorrhage which were all subsequently confirmed at autopsy. The remaining cases died fairly suddenly and shortly after treatment, and the cause of death was usually recorded as "acute cardiac failure" or "shock following E.C.T.". In these cases one is at once struck by the meagre autopsy findings, death being presumably due to a severe functional disturbance of delicately balanced physiological mechanisms necessary for life. In some of these patients however there was autopsy evidence of myocardial disease and coronary atheroma which might have contributed to death on some future occasion, and it is in these cases that one may justifiably refer to death as being "accelerated by E.C.T.". The predominant mode of death is cardiovascular—a fact which has been stressed by previous authors.

General Conclusions

1. The risk of death due to E.C.T. is very small and this study shows that it may be even less than previous writers have suggested. It is not, however, an entirely innocuous procedure, to be given without careful consideration of the patient's physical state and psychiatric disorder.

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2. The occasional death following E.C.T. necessitates a careful physical examination, with special reference to the cardiovascular system, for all patients, especially those in middle and later life. The day to day physical health of these patients should be carefully checked between treatments.

3. Very great care should be shown to patients in whom technical difficulties have been encountered during previous treatments, and attention directed to changing techniques, generally in the direction of modified to unmodified E.C.T. Additional medication should be reduced to the barest minimum. Electrical modification of the convulsion (i.e. by the Ectonus technique) may be safer than chemical modification in these patients.

4. Nevertheless, there is no evidence from our findings to show that any particular E.C.T. technique carries a lesser risk to life than others.

Appendix

Case 1. E.S.W., a female patient aged 76, was admitted on 4 June, 1957 suffering from a senile paranoid psychosis. She had exhibited numerous bizarre unsystematized paranoid delusions for an indefinite period, but her mental condition had deteriorated following treatment for a coronary thrombosis a few weeks previously at another hospital, and she had recently become violent and refused her food. Her physique was described as poor, and she was cyanosed with dorsal kyphoscoliosis and marked emphysema. Her blood pressure was 160/90 with radial arterio-sclerosis. In spite of the physical findings E.C.T. was considered a "justifiable risk".

She received no drugs apart from parentrovite injections after admission. E.C.T., using a current of 30 Joules, modified by thiopentone 0·15 gm. and scoline 35 mg. was given uneventfully at three-day intervals from 26 June. Following the fourth treatment on 5 July, respirations returned, but after 5 minutes they became laboured, and she developed bronchospasm with moist sounds in the chest. She was very restless and did not appear to recover consciousness, showing no response to intravenous injections of aminophylline and nikethamide. She died 2 hours after treatment.

The cause of death confirmed at post-mortem was given as left ventricular failure secondary to hypertension. There was gross pulmonary oedema but this was not regarded as the prime cause of death. There was little atheroma, and the coronary vessels were patent. There were no other lesions of note.

Case 2. F.W., a male patient aged 70, was admitted on 26 January, 1956, suffering from an agitated depression, having made a suicidal attempt by cutting his wrists. He had led an active life as a gardener, but had become depressed following a mild left cerebral thrombosis two months previously, which had reduced his activities. On admission he was emaciated and had a residual hemiparesis. The blood pressure was 160/90 and there was much thickening and tortuosity of the peripheral arteries. The pulse showed an irregular irregularity, but there was no evidence of congestive heart failure. He was seen by a consultant physician and E.C.T. was considered a "justifiable risk". Mentally he was severely depressed and pre-occupied, with ideas of unworthiness, and expressed numerous bizarre hypochondriacal delusions. He was put on chlorpromazine 50 mg. t.d.s. and received two E.C.T.s well modified with 25 mg. scoline on 10 and 14 February, recovery from which was uneventful. Following his third treatment by this technique on 21 February, he became shocked and cyanosed and his pulse was imperceptible at the wrist. Artificial respiration and injections of nikethamide were given without effect, and he died after a few minutes without regaining consciousness. The cause of death ascertained at autopsy was hypertensive cardiac failure accelerated by E.C.T. At the subsequent inquest the verdict was given as "death by misadventure".

Case 3. E.S., a male patient aged 74, was admitted on 8 January, 1957, suffering from a depressive illness with suicidal pre-occupation. A general dealer, he had enjoyed good mental health until some 10 months previously when he developed facial pain for which no cause could be found, and he imagined he had cancer.

On admission he was in poor physical health, but there was no evidence of systemic disease. His blood pressure was 140/80. He received no medication apart from fluids and parenteral vitamins.

On 10 and 11 January he received E.C.T. modified by thiopentone 0·5 gm. and Brevidil E 50 mg. following which it was noted that he took rather a long time to recover. Atropine gr. 1/100 was given ½ hour before all treatments. On 12 January he was given E.C.T. modified with scoline alone without thiopentone, and recovery was reported as being much quicker. Two further treatments were given on 13 and 15 January but the techniques were not recorded. By 20 January his depression and agitation seemed worse than ever, and he received a further E.C.T. (the 6th) modified with thiopentone 0·5 G and Brevedil E 50 mg. Oxygen was administered both before and after the shock, following upon which he breathed stertorously three times and suddenly expired.

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The post-mortem showed myocardial fibrosis, and there was marked narrowing of the descending branch of the left coronary artery due to atheromatous ulceration. The liver was congested but there was no other pathology of note. The cause of death was recorded as cardiac failure due to coronary artery disease and emphysema, and was regarded as accelerated by E.C.T.

Case 4. E.A.L., a female patient aged 73, was admitted on 25 June, 1957 suffering from a senile depressive illness. She was unable to sleep without sedatives, and then woke early and was unable to get off again. She had lost self-confidence and her interest in her friends and her home. She was self-reproachful with suicidal pre-occupation. Her physical health was satisfactory though she was obese and suffered from hypertension, blood pressure 210/110.

She was sedated with sodium amytal gr. 1½ t.d.s. and gr. 3 nocte, but received no other medication. She was given E.C.T., modified with thiopentone and scoline on 1 and 4 July, with no after effects, and some improvement in her mental state was noted afterwards. On 11 July she received a further treatment from which she made a good recovery. Twenty-four hours later she complained of indigestion after supper which was relieved by an antacid mixture. Shortly afterwards she slipped while getting out of her bed, and had to be helped back. She appeared to go off to sleep, but soon afterwards her respirations became stertorous, and she died suddenly at 1.0 a.m. on 13 July. At autopsy the cause of death was shown to be a coronary thrombosis. It was however commented that there did not appear to be any connection between the E.C.T. and the coronary thrombosis.

Case 5. H.A.F., a male patient aged 60, was admitted on 15 January, 1957, suffering from an involutional depression with hypochondriasis. A haulage contractor, he was described as obtuse and mentally retarded, being excessively pre-occupied with his loss of appetite, and imagined that his bowels were "blocked up".

Three years previously he had been treated for a similar depressive illness with 8 E.C.T.s modified with thiopentone and scoline, and had shown a satisfactory response. Six months before admission there was a history of mild confusion and he was thought to have had a cerebral thrombosis at the time.

Physical examination on admission was entirely normal. In spite of the history of a previous cerebral thrombosis, it was decided to give him further E.C.T., particularly as he had shown such a good response to this treatment previously. On 21 January he was given a further treatment modified with thiopentone and scoline, and appeared to recover normally afterwards, and was fully orientated. The following morning he fell out of bed and appeared to have sustained a cerebral haemorrhage from which he died the next day. The interval between E.C.T. and death was 30 hours. The cause of death confirmed at autopsy was cerebral haemorrhage and bronchopneumonia. The pathologists imagined that the latter had been present before the E.C.T. had been given. Extensive atheroma was also present.

Case 6. E.C., a female patient aged 62, was admitted on the sixth occasion in March, 1955 suffering from recurrent melancholia. She complained of insomnia and had much suicidal pre-occupation. She gave a history of having made a number of suicidal attempts previously by various methods. She was first admitted in 1946, and improved temporarily following courses of unmodified electroplexy given without incident. On her last admission her physical condition was satisfactory, though she was mildly hypertensive, blood pressure 150/100.

A year before her death she began to develop weakness and wasting of the lower motor neurone type of her hands, forearms and legs, without sensory changes, which was diagnosed as progressive muscular atrophy. This condition progressed relentlessly, and she was eventually unable to walk, falling several times a day, due to weakness and lack of muscle tone. She became incontinent of urine. She also had bilateral oedema of the ankles which was considered to be due to her neurological condition and not of cardiac origin.

She received no medication during her last admission except sodium amytal given nocte. Since 1956 she had received 23 E.C.T.s modified with thiopentone 0·5 gm. and scoline 50 mg. On her 24th treatment by this technique, she failed to come round and remained cyanosed. A marked tracheal tug was noticed. Following intravenous injections of nikethamide, her condition improved after half an hour, but some 3 hours after E.C.T., she became comatose and her blood pressure fell to 110/60, and she was thought to have had a coronary thrombosis. The following day she appeared better and was fully orientated. Though still slightly cyanosed she was able to sit up in bed and took her food well. The next day, 2 days after treatment, she collapsed and died suddenly. The cause of death confirmed by autopsy was coronary thrombosis. Coronary atheroma and generalized arterio-sclerosis were also noted, together with progressive muscular atrophy.

Case 7. H.E.B., a male patient aged 60, was admitted on 30 November, 1957 suffering from a depressive illness. He had had numerous less severe bouts of depression since 1930, and a year previously had undergone a prostatectomy following which he had never regained his previous health. A clerical worker, he had been depressed with cancerphobia for 5 weeks, and had been unable to sleep properly or concentrate. He repeatedly wished that he was dead, and on the morning of admission made an unprovoked attack on his wife, stating that he wanted to put her out of her misery.

Of pyknic build, he was in fair physical health, apart from a history of chronic bronchitis. His blood pressure was 170/100.

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On admission he was put on chlorpromazine 50 mg. t.d.s. which was reduced to 25 mg. t.d.s. on 8 December. He received 5 E.C.T.s all modified with thiopentone 0·25 gm. and scoline 50 mg., atropine gr. 1/100 being given ½ hour previously. No incidents were reported after the first four treatments, but following the fifth on 12 December, he became cyanosed in the recovery room and was given further oxygen. He died after some 20 minutes had elapsed, and showed no response to oxygen, intravenous and intracardiac nikethamide and artificial respiration.

Autopsy showed moderate hypertensive cardiac hypertrophy, with coronary atheroma and fatty infiltration of the right ventricle. The cause of death was recorded as acute cardiac failure due to electroplexy.

Case 8. M.E.H., a female patient aged 50 at the time of death, was admitted in September, 1937 under certificate suffering from catatonic schizophrenia, having made a suicidal attempt by taking poison and inhaling coal gas. In 1941 her condition deteriorated, and she made frequent unprovoked attacks of violence upon other patients and the nursing staff. She received courses of E.C.T., in 1943, 1946, 1947 and 1948, resulting in temporary behavioural improvement, but unfortunately she tended to relapse, and during these phases became dangerous to persons around her. Maintenance E.C.T. was therefore given up to the date of her death, at monthly, and more recently at fortnightly intervals, when she was also receiving chlorpromazine 100 mg. t.d.s. Between 1943 and her death a total of 332 treatments had been given, and at the time of death she appeared to be in good physical health.

On 14 October, 1957, E.C.T. was given unmodified (as it had been throughout). She came round from the convulsion and was given oxygen. She appeared to be quite comfortable for 2 minutes and was on the point of regaining consciousness when she suddenly collapsed, death occurring in spite of intravenous nikethamide and artificial respiration. The circumstances associated with death were reported to the Coroner who returned the following verdict: "Death due to shock following E.C.T.—accidental death". At autopsy very little abnormal was found. The heart was completely normal and the coronary arteries free from atheroma.

Case 9. M.M., a female patient aged 67, was admitted in December, 1957 suffering from schizophrenia. There was a previous history of episodes of "religious mania", but recently she had been found wandering by neighbours, shouting and referring to herself as "Her Majesty" and had been refusing her food.

On admision her physical health was poor, but there was no evidence of systemic disease. She was very resistant and negativistic. Treatment with chlorpromazine was commenced with a dose of 50 mg. t.d.s. increased to 100 mg. t.d.s. after a few days. She was also given daily injections of parentrovite.

She received four unmodified E.C.T. treatments on alternate days shortly after admission without any untoward effects, though little improvement was noted in her mental state. During the night following one of these treatments she had an attack of what was described as "bronchial asthma" but this subsided after an injection of adrenaline.

On 28 December she received her 5th E.C.T. after which she took only a few breaths. Oxygen, nikethamide, and artificial respiration were given in an attempt to resuscitate her but she died after 10 minutes. All electroplexy had been given unmodified. The cause of death ascertained at post-mortem was cardiac failure due to coronary atheroma and aortic stenosis. The latter had not been detected during life.

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